Intracranial Pressure and its Monitoring: A review

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Evidence suggests that the mortality and morbidity of acquired brain injury could be reduced if clinicians used an aggressive intracranial pressure guided approach to care. Despite nearly 50 years of evidence that intracranial pressure monitoring benefits patient care, only about half of the patients who could benefit are monitored. Some clinicians express concerns regarding risks such as bleeding, infections, and inaccuracy of the technology. Others cite cost as the reason. This article discusses the risks and benefits of intracranial pressure monitoring and the current state of evidence of why patients should be monitored.

Keywords: intracranial pressure, cerebral perfusion pressure, monitoring devices, drift, infections, hemorrhage

Background
Elevated intracranial pressure (ICP) is seen in head trauma, hydrocephalus, intracranial tumors, metabolic encephalopathy, intracranial bleeds and CNS infections. Increased ICP is an important cause of secondary brain injury, and its degree and duration is associated with outcome after TBI.1,2 Intractable intracranial hypertension can lead to death or devastating neurological damage either by reducing cerebral perfusion pressure (CPP) and causing cerebral ischemia or by compressing and causing herniation of the brainstem or other vital structures. Prompt recognition is crucial in order to intervene appropriately. The association between the severity of intracranial hypertension and poor outcome after severe head injury is well recognized. Outcomes tend to be good in patients with normal ICP, whereas those with elevated ICP are much more likely to have an unfavorable outcome. Elevated ICP carries a mortality rate of around 20%.

The rapid recognition of elevated ICP is therefore of obvious and paramount importance so that it can be monitored and so that therapies directed at lowering ICP can be initiated. Continuous ICP monitoring is important both for assessing the efficacy of therapeutic measures and for evaluating the evolution of brain injury.

Although some investigators have questioned invasive ICP monitoring in improving patient outcomes, numerous retrospective have favored the use of this technique.

The goal of ICP monitoring is to ensure maintenance of optimal CPP. The ICP also forms a basis for medical or surgical intervention in cases of increased ICP in cases of intractable ICP elevation that do not respond to conservative management.

ICP monitoring may be discontinued when the ICP remains in the normal range within 48-72 hours of withdrawal of ICP therapy or if the patient’s neurological condition improves to the point where he or she is following commands.

Pathophysiology
Physiology of ICP was described by Professors Munroe and Kellie in the 1820s. In essence, they noted that, in adults, the brain is enclosed in a rigid case of bone and that the volume of its contents must remain constant if ICP is to remain constant. The intracranial compartment consists of brain approximately 83%, cerebrospinal fluid (CSF) approximately 11%, and blood approximately 6%.

Under normal conditions there are two main components of ICP namely CSF and vasogenic. The former is derived from the circulation of CSF and is responsible for baseline ICP. It may be deranged in pathologic states, causing an increase in ICP, because of resistance to CSF flow between intracerebral compartments secondary to brain swelling or expansion of intracranial mass lesions, or because CSF outflow is obstructed.
The vasogenic component of ICP is associated with continuous, small fluctuations of cerebral blood volume (CBV). Vasogenic increases in ICP may be caused by high PaCO₂, increase in cerebral metabolism, and cerebral hyperemia. An increase in the volume of one of the components of the intracranial cavity (e.g., brain) requires a compensatory reduction in another (e.g., CSF) to maintain a constant pressure. Brain tissue is essentially incompressible, so any increase in ICP due to brain swelling initially results in extrusion of CSF and (mainly venous) blood from the intracranial cavity, a phenomenon known as "spatial compensation." CSF plays the largest role in spatial compensation because it can be expelled from the intracranial cavity into the reservoir of the spinal theca.

**Figure 1.** Intracranial pressure (ICP) volume curve. The curve has three parts: a flat part representing good compensatory reserve (A–B), an exponential part representing reduced compensatory reserve (B–C) and a final flat part representing terminal derangement of cerebrovascular responses at high ICP (C–D).

The relationship between ICP and intracranial volume is described by the pressure-volume curve that comprises of three parts (Fig. 1). The first part of the curve is flat because compensatory reserves are adequate and ICP remains low despite increases in intracerebral volume (A–B in Fig. 1). When these compensatory mechanisms become exhausted, the pressure-volume curve turns rapidly upwards in an exponential fashion. Intracranial compliance is now critically reduced and a small increase in intracerebral volume causes a substantial increase in ICP (B–C in Fig. 1). At high levels of ICP, the curve plateaus as the capacity of cerebral arterioles to dilate in response to a reduction in CPP become exhausted. The high brain tissue pressure results in collapse of these dysfunctional vessels as cerebrovascular responses become terminally disrupted (C–D in Fig. 1).

Increased ICP causes a critical reduction in CPP and CBF and may lead to secondary ischemic cerebral injury. A number of studies have shown that high ICP is strongly associated with poor outcome, particularly if the period of intracranial hypertension is prolonged. Increased ICP can also cause actual shift of brain substance resulting in structural damage to the brain and to herniation through the tentorial hiatus or foramen magnum. The latter results in pressure on the brainstem causing bradycardia and hypertension (the classic Cushing reflex) and, if untreated, respiratory depression and death.

**Interaction with blood pressure and cerebral blood flow**

Cerebral perfusion pressure (CPP), defined as the mean arterial pressure (MAP) minus ICP, is a critical determinant of cerebral blood flow (CBF) and plays an important role in ICP management. Normally CBF is "autoregulated" at a constant level over a wide range of CPPs (from 50 to 150 mmHg in adults) (Fig 2).

Pressure auto regulation of this type is mediated by changes in arteriolar diameter and cerebrovascular resistance. The auto regulatory curve is shifted to the left in children and shifted to the right in patients with chronic hypertension. In pathologic states with impaired auto regulation, such as TBI and subarachnoid hemorrhage, CBF may approximate a linear relationship with CPP, which creates a smaller range of optimal CPP (Fig 2). Reduction of CPP below the lower limit of autoregulation can
lead to ischemia, whereas CPP elevation above the upper limit of autoregulation can be associated with hyperemia, exacerbation of vasogenic edema, and increased ICP. Although the optimal CPP for any particular patient may vary, as a rule of thumb CPP should be maintained above 70 mmHg to avert ischemia and below 110 mmHg to avoid breakthrough hyperperfusion in adults.

CBF also depends upon Paco2 and Pao2 level. In general, the cerebral vessels are less responsive to changes in PaO2 than to those in PaCO2. Arteriolar diameter and CBF progressively increase as PaCO2 rises from 20 to 80 mmHg, whereas hypoxemia leads to vasodilatation and increased CBF only when PaO2 falls below 50 mmHg.

![Cerebral autoregulation curve. In the normal relationship (solid line), with CBF held constant across a wide range of CPP (50±150 mmHg).](image)

**Normal and Pathologic ICP**

**Table 2: Normal intracranial pressure values**

<table>
<thead>
<tr>
<th>Age group</th>
<th>Normal range (mm Hg)</th>
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<tbody>
<tr>
<td>Adults</td>
<td>&lt;10–15</td>
</tr>
<tr>
<td>Children</td>
<td>3–7</td>
</tr>
<tr>
<td>Term infants</td>
<td>1.5–6</td>
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The definition of intracranial hypertension depends on the specific pathology and age, although ICP >15 mm Hg is generally considered to be abnormal. However, treatment is instituted at different levels depending on the pathology. For example, ICP <15 mm Hg warrants treatment in a patient with hydrocephalus, whereas after TBI, treatment is indicated when ICP exceeds 20 mm Hg. Thresholds vary in children and it has been recommended that treatment should be initiated during TBI management when ICP exceeds 15 mm Hg in infants, 18 mm Hg in children up to 8-yr-of-age and 20 mm Hg in older children and teenagers. ICP is not evenly distributed in pathologic states because CSF does not circulate freely and intracranial CSF volume may be low because of brain swelling. The assumption of one, uniform, ICP is therefore questionable and intraparenchymal pressure may not be indicative of “real” ICP, i.e., ventricular CSF pressure. In the injured brain, there may be intraparenchymal pressure gradients between the supra and infra-tentorial compartments and bilateral monitoring has revealed differential pressures across the midline in the presence of hematomas and also in the absence of space-occupying lesions.

### Causes of Raised Intracranial Pressure

**Table 3**

<table>
<thead>
<tr>
<th>Pathological process</th>
<th>Examples</th>
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<tbody>
<tr>
<td>Localized mass lesions</td>
<td>Traumatic hematomas (extradural, subdural, intracerebral) neoplasm (glioma, meningioma, metastasis) Abscess Focal edema secondary to trauma, infarction, tumor</td>
</tr>
<tr>
<td>Disturbance of CSF circulation</td>
<td>Obstructive hydrocephalus, Communicating hydrocephalus</td>
</tr>
<tr>
<td>Obstruction to major venous sinuses</td>
<td>Depressed fractures overlying major venous sinuses Cerebral venous thrombosis</td>
</tr>
<tr>
<td>Diffuse brain edema or swelling</td>
<td>Encephalitis, meningitis, diffuse head injury, subarachnoid hemorrhage, Reye’s syndrome, lead encephalopathy, water intoxication, near drowning</td>
</tr>
<tr>
<td>Idiopathic</td>
<td>Benign intracranial hypertension</td>
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### Methods of ICP Monitoring

ICP cannot be reliably estimated from any specific clinical feature or computed tomography (CT) finding and must actually be measured.
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Different methods of monitoring ICP are depicted in figure 3. Intraventricular catheter and intraparenchymal micro transducer systems are the most common monitoring devices used in practice. Subarachnoid and epidural devices have much lower accuracy\(^{18,19}\) and are now rarely used. Measurement of lumbar CSF pressure does not provide a reliable estimate of ICP and may be dangerous in the presence of increased intracranial hypertension.\(^{20}\) Advantages and disadvantages of each monitoring method are summarized in Table 4.

**Intraventricular Catheters**

The “gold standard” technique for ICP monitoring is a catheter inserted into the lateral ventricle, usually via a small right frontal burr hole. This can be connected to a standard pressure transducer via a fluid filled catheter. The reference point for the transducer is the foramen of Munroe, although, in practical terms, the external auditory meatus is often used. Ventricular catheters measure global ICP and have the additional advantages of allowing periodic external calibration, therapeutic drainage of CSF, and administration of drugs (e.g., antibiotics).\(^{20,21}\) However, placement of the catheter may be difficult if there is ventricular effacement or displacement due to brain swelling or intracranial mass lesions. The use of intraventricular Catheters is complicated by infection in up to 11% of cases.\(^{22,23}\) This is a serious complication resulting in significant morbidity and mortality. The risk of infection increases after 5 days\(^{23}\) and this has been presumed to be related to retrograde colonization of the catheter. However, recent data suggest that CSF infection is also likely to be acquired during introduction of the catheter in a significant number of cases.\(^{24}\) Intraventricular catheters may become blocked, especially in the presence of subarachnoid blood or increased CSF protein.\(^{25}\) Although the patency of catheters can often be restored by gentle flushing, repeated attempts significantly increase the risk of infection.\(^{23}\)

Regular microbiological analysis of CSF samples to permit early diagnosis of ventriculitis is recommended by some, whereas others believe that routine sampling may actually predispose to higher infection rates because of the repeated opening of the closed drainage system. The use of antibiotic-impregnated catheters is associated with a lower infection rate, \(^{26}\) although catheters coated with hydrogel to impede bacterial adherence are not associated with reduced infection rates.\(^{27}\)

**Intraparenchymal Pressure Transducers**

The pressure transducer in these disposable devices is incorporated into the tip of a thin fiber optic cable (the Camino device) or within a strain-gauge micro sensor at the tip of a flexible catheter (the Codman device). These catheters can be placed into either the brain parenchyma or the ventricle via a small burr hole and screw\(^{28,29}\). With intraparenchymal placement, the infection rate is exceedingly low (approximately 1%)\(^{30}\). When combined with a ventricular catheter, the system allows simultaneous CSF drainage and continuous ICP measurement. These devices only need to be calibrated once prior to insertion, and the accuracy of ICP measurements is generally superior to those provided by subarachnoid bolts or epidural transducers\(^{31}\). A new version of the Codman monitor also provides measurements of brain temperature. A third intraparenchymal monitor recently approved by U.S. Food and Drug Administration (FDA) (the Spielberg device) features a small air filled balloon at the tip of a flexible catheter; it has the advantage of providing measurements of intracranial compliance (calculated as a pressure/volume index) as well as ICP.\(^{32}\)
Subarachnoid Bolts
This is another fluid-coupled system which connects the intracranial space to an external transducer at the bedside via saline-filled tubing\(^3\). The subarachnoid bolt is actually a hollow screw that is inserted via a burr hole. The dura at the base of the bolt is perforated with a spinal needle, allowing the subarachnoid CSF to fill the bolt. Pressure tubing is then connected to establish communication with a pressure monitoring system. Although the infection risk is low, these devices are prone to error, including underestimation of ICP, screw displacement, and occlusion by debris\(^3\).

Epidural Transducers
These devices (the Gaeltec device) are inserted deep into the inner table of the skull and superficial to the dura\(^3\). In most of these devices, pressure is transduced by an optical sensor. They have a low infection rate (approximately 1\%)\(^2\), but are prone to malfunction, displacement, and baseline drift that can exceed 5±10 mmHg after more than a few days of use. Much of the inaccuracy results from having the relatively inelastic dura between the sensor tip and the subarachnoid space.

Noninvasive ICP Monitoring
At present there is no noninvasive method that can provide accurate continuous online measurement of ICP. However, optic nerve sheath diameter (OPNSD)\(^3\)\(^8\)\(^9\) and transcranial Doppler (TCD) Ultrasonography are promising modalities. TCD measures the velocity of blood flow in the basal cerebral arteries, shows characteristic changes with increasing ICP\(^6\). As CPP falls, diastolic velocity decreases and pulsatility increases, reflecting increased distal vascular resistance to flow. Though this finding is specific for severe intracranial hypertension, TCD is not sensitive to mild to moderate ICP elevations. Lateralized asymmetries in TCD pulsatility correlate with lesion volume in intracerebral hemorrhage, and are believed to reflect compartmentalized ICP gradients\(^3\)\(^7\).

Indication
The most common use of continuous ICP monitoring is in the management of severe closed head injury. Bullock and 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 indications summarized in Table 5.

<table>
<thead>
<tr>
<th>Table 5: Indications of ICP Monitoring</th>
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<tr>
<td>Severe head injury</td>
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<tr>
<td>Intracerebral hemorrhage</td>
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<tr>
<td>Subarachnoid hemorrhage</td>
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<tr>
<td>Hydrocephalus</td>
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<tr>
<td>Stroke</td>
</tr>
<tr>
<td>Central nervous system infections</td>
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<tr>
<td>Hepatic encephalopathy</td>
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Contraindication
No absolute contraindications. However, caution should be exercised in following conditions
- Coagulation defects
- Anticoagulant therapy
- Scalp infection

Complications
- Intracranial infection
- Intracerebral hemorrhage
- Air leakage into the ventricle or subarachnoid space
- CSF leakage
- Over drainage of CSF leading to ventricular collapse and herniation
- Loss of monitoring or drainage capabilities due to the occlusion of the catheter with brain tissue or blood
- Inappropriate therapy because of erroneous ICP readings due to dampened waveforms, electromechanical failure, or operator error (i.e. inappropriate leveling)

Analysis of ICP Wave Form
Four major waveforms are of clinical importance: normal, A, B, and C.
Normal: Normal ICP waves have a steep upward systolic slope followed by a downward diastolic slope with a dicrotic notch. In most cases, this waveform occurs continuously and indicates that the ICP is between 0 and 15 mm Hg.

“A” waves or “plateau” waves
These are steep increases in ICP from baseline to peaks of 50–80 mm Hg that persist for 5–20 min. These waves are always pathologic and may be associated with early signs of brain herniation, such as bradycardia and hypertension. They occur in patients with intact autoregulation and reduced intracranial compliance and represent reflex, phasic vasodilatation in response to reduced cerebral perfusion. The development of plateau waves leads to a vicious cycle, with reductions in CPP predisposing to the development of more plateau waves, further reductions in CPP and irreversible cerebral ischemia.

“B” waves
These are rhythmic oscillations occurring at 0.5–2 waves/min with peak ICP increasing to around 20–30 mm Hg above baseline. They are related to changes in vascular tone, probably due to vasomotor instability when CPP is at the lower limit of pressure autoregulation.

“C” waves
These are oscillations occurring with a frequency of 4–8/min and are of much smaller amplitude than B waves, peaking at 20 mm Hg. They occur synchronously with ABP, reflect changes in systemic vasomotor tone, and are of no pathologic significance.

Why Monitor ICP?
Four lines of evidence support the use of ICP monitoring in children with severe TBI.
1. Frequently reported high incidence of intracranial hypertension.
2. A widely reported association of intracranial hypertension and poor neurological outcome.
3. Concordance of protocol driven intracranial hypertension and best reported clinical outcome.
4. Improved outcome associated with successful ICP lowering therapies

There is a large body of evidence to indicate that ICP monitoring is of benefit to the patient by the following ways:

1. Early detection of developing pathology
Patients at high risk of developing raised ICP usually are drowsy or sedated and ventilated, and the first clinical indication of an increase of edema or a hematoma might be signs of herniation. By alerting the medical team prior to this deterioration, monitoring enables early intervention and improved outcome. Intervention when a small rise in IC occurs has also been shown to prevent later profound intracranial hypertension. A management protocol based solely on repeated CT scans is economically not feasible for most of our patients, and has been shown to be less accurate than actual monitoring. There is also evidence that time-bound repetition of CT scans does not contribute to patient management. In addition to the lack of benefit, transporting a critically ill patient for investigation increases the risk for the patient and impose a logistical strain on the ICU. ICP monitoring can indicate the need for a repeat imaging and avoid routine protocol-based investigation. ICP monitoring should never be at the expense of clinical examination.

2. Limit avoidable therapy
Empirical therapy for presumed raised intracranial  

pressure runs the risk of inflicting unnecessary iatrogenic complications on patients who either had only mild or no intracranial hypertension. These include unnecessary prolongation of ventilation, brain ischemia induced by hyperventilation, fluid-electrolyte imbalance induced by mannitol and diuretics and even at times unnecessary surgery.

3. Cerebral perfusion pressure
The CPP can be calculated only if the ICP is measured. The importance of maintaining an adequate CPP has been discussed earlier in this review.

4. Safety factor
ICP monitoring can help in revealing shortcomings in other treatment modalities like head positioning, adequacy of sedation, analgesia or paralysis, and even draws attention to other abnormalities such as hyponatremia. Most raised ICP alarms are in fact due to one of these causes, and therefore the monitoring provides an additional layer of safety for the patient.

5. Decision on surgery
The decision to operate on the brain when the clinical and radiological features are ambiguous is extremely difficult. Knowledge of the ICP can help in decision-making regarding surgery in these cases. ICP monitoring also provides essential information for the timing of decompressive craniectomies in stroke, subarachnoid hemorrhage and severe head injury.

6. CSF drainage
The use of an intraventricular catheter to monitor ICP also provides the option of venting CSF, which directly lowers the pressure without any of the systemic effects associated with all other means of ICP control.

7. Prognostication
Refractory raised pressure intuitively indicates a bad prognosis which has been demonstrated in all studies from the 1970s to the present. There is also data to show that even transient, controllable rises in ICP indicate a worse prognosis in head injury.

Opinions against ICP monitoring
The arguments against ICP monitoring are generally negative and much fewer than those of proponents of monitoring.

1. Lack of evidence
There has not been a randomized controlled trial on the efficacy of ICP monitoring in improving outcome, and there most likely will never be one because the utility of monitoring is so widely accepted that a trial where ICP is not monitored for a group of patients is considered unethical. Even if a trial were to be attempted, the sample size required to prove the benefit would be over 750 patients, which would be logistically and financially extremely difficult.

2. Outcome without monitoring
Recent Trial on Intracranial Pressure Monitoring in Traumatic Brain Injury by Randall M, Chesnut et al showed no significant between-group difference in the primary outcome, a composite measure based on percentile performance across 21 measures of functional and cognitive status (score, 56 in the pressure-monitoring group vs. 53 in the imaging–clinical examination group; P = 0.49). Six-month mortality was 39% in the Pressure-monitoring group and 41% in the imaging–clinical examination group (P = 0.60). The median length of stay in the ICU was similar in the two groups (12 days in the pressure-monitoring group and 9 days in the imaging–clinical examination group; P = 0.25), although the number of days of brain-specific treatments (e.g., administration of hyperosmolar fluids and the use of hyperventilation) in the ICU was higher in the imaging–clinical examination group than in the pressure-monitoring group (4.8 vs. 3.4, P = 0.002). The distribution of serious adverse events was similar in the two groups.

3. Clinical deterioration
Temporal lobe hematomas and swelling can theoretically cause uncal herniation and brainstem
compression without raising the ICP to alarm threshold values – there is a report of herniation taking place at an ICP of 18 mmHg.

4. Choice of patients to monitor
The debate on which patients will benefit from ICP monitoring is nowhere near settled, the closest approach to agreement being with regard to trauma. There is not sufficient data on other disease conditions for the establishment of guidelines.

Conclusion
ICP monitoring is safe, has relatively low complications rates, and has been shown to improve patient outcomes by giving the clinician a tool to evaluate both the patient and the effectiveness of treatment. Proper training for the clinician who is inserting the device, whether neurosurgeon or non-neurosurgeon, will minimize complication and facilitate accurate information being obtained. It is important to remember the strengths and limitations of each system when choosing the placement of the monitor (intraparenchymal, intraventricular, or surface monitor), the technology (fluid-filled versus advanced technology), and as well as cost. With proper education of all staff regarding the care, management, and troubleshooting of ICP, monitoring ICP will enhance the care we give our brain-injured patients.

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