REVIEW ARTICLE

Intracranial pressure monitoring

Mary Abraham, Vasudha Singhal

Abstract

Brain specific monitoring enables detection and prevention of secondary cerebral insults, especially in the injured brain, thereby preventing permanent neurological damage. Intracranial pressure (ICP) monitoring is widely used in various neurological, neurosurgical and even medical conditions, both intraoperatively and in critical care, to improve patient outcome. It is especially useful in patients with traumatic brain injury, as a robust predictor of cerebral perfusion, and can help to guide therapy and assess long-term prognosis. Intraventricular catheters remain the gold standard for ICP monitoring, as they are the most reliable, accurate and cost-effective, and allow therapeutic cerebrospinal fluid drainage. Newer fibreoptic catheter tip and microchip transducer techniques have revolutionised ICP monitoring, with their ease of insertion in patients with narrow ventricles, and reduced risk of infection and haemorrhage. Furthermore, non-invasive methods of ICP monitoring, such as transcranial Doppler, optic nerve sheath diameter, etc., have emerged as promising techniques for screening patients with raised ICP in settings where invasive techniques are either not feasible (patients with severe coagulopathy) or not available (setups without access to a neurosurgeon). Therefore, ICP monitoring, as a part of multi-modality neuromonitoring, is a useful tool in the armamentarium of the neuro-intensivist in decreasing morbidity and mortality of critically ill neurological patients.

Key words: Brain, intracranial pressure, neuromonitoring

INTRACRANIAL PRESSURE MONITORING

The concept of intracranial pressure (ICP) being a function of the volume and compliance of each component of the intracranial compartment was proposed by the Monroe - Kellie doctrine centuries ago.[1-3] To keep it in simple terms, the skull is a semi-rigid box, consisting of the brain, blood and cerebrospinal fluid (CSF), lightly stuffed inside to create a mildly positive pressure, known as the ICP. These components exist in a state of volume equilibrium, such that a change in the volume of one component should be compensated by the change in the volume of the other; otherwise, an increase in ICP will ensue. Metabolic requirements, as well as cardiac and respiratory variations, modulate the cerebral blood flow (CBF), thereby providing capacitance in the system. Once this capacitance is exhausted, there is a sharp increase in the ICP resulting in intracranial hypertension that may lead to devastating neurological damage and even death. Prompt recognition of raised ICP and initiation of therapy targeted at reducing the ICP is therefore of paramount importance to prevent brain damage.

PATHO-PHYSIOLOGY OF INTRACRANIAL PRESSURE

The main purpose of monitoring ICP is to monitor the cerebral perfusion pressure (CPP), which is the pressure differential across the arteriovenous bed in the brain.[4,5] CPP is calculated by the formula:

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Access this article online	
Quick Response Code:	Website: www.jnaccjournal.org
	DOI: 10.4103/2348-0548.165039

How to cite this article: Abraham M, Singhal V. Intracranial pressure monitoring. J Neuroanaesthesiol Crit Care 2015;2:193-203.

CPP = MAP (mean arterial blood pressure) − ICP

Furthermore, the CBF is determined by the relation:

CBF = CPP/CVR; where CVR is the cerebrovascular resistance.

The cerebrovascular resistance is kept constant over a wide range of CPP via constriction and dilatation of the microvascular networks. A constant CBF is thus maintained in the face of physiological and pathological swings in mean arterial pressure (MAP). However, this buffer system, called the autoregulation, is only effective between a MAP of 60 and 150 mm Hg. The auto-regulatory reserve is interpreted as the difference between current mean CPP and the lower limit of autoregulation (a threshold of 60 mm Hg). Policies to therapeutically maintain a high CPP are controversial, especially in an injured brain, as autoregulation can be impaired. In such a scenario, the cerebral vessels are non-reactive, and an increase in CPP may result in hyperaemia, increase in vasogenic oedema and a secondary rise in ICP. Therefore, it becomes important to maintain MAP within specific limits in order to prevent steep rises in ICP.

Intracranial compliance

ICP is also determined by the intracranial compliance, which is the relationship between ICP and cerebral blood volume. A mono-exponential curve depicts the relation between the ICP and volume [Figure 1]. Note that at low pressures the ratio of dP/dV (elastance) is small, while at high pressure the ratio is large. Thus, initial increases in intracranial volume (up to 30 cm^3) are well compensated by displacement of CSF from the cranial to the spinal compartment, and to a lesser extent, by extrusion of venous blood from the cranium. Once these compensatory mechanisms are exhausted, further increases in intracranial volume can lead to a sustained elevation of ICP and catastrophic brain herniation. The slope of the curve is also dependent on which intracranial constituent is increasing. If it is blood or CSF, both of which are poorly compressible, then the slope is steeper. If it is brain parenchyma, as from a tumour, the curve is less steep as the tissue is compressible.

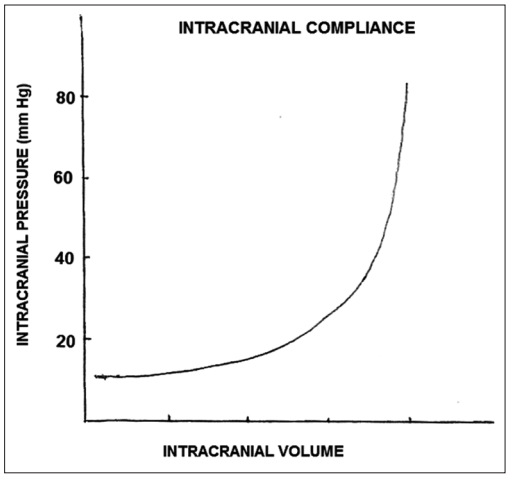

Figure 1: Intracranial compliance curve. Initial increases in intracranial volume (up to 30 cm^3) are well compensated, while further increases result in sustained elevations in intracranial pressure and catastrophic brain herniation

Ryder *et al*. studied the pressure-volume relationship by injecting fluid into the CSF space. The degree of the change in pressure resulting from an injection of a known volume permitted characterisation of the ICP-volume dependence.[6]

Normal intracranial pressure

The cranial biomechanical state is a term characterising the haemodynamic and hydrodynamic states of the craniospinal system and is a function of the total CBF, intracranial compliance, and ICP. This changes with normal activities (e.g., change in body posture), with aging, and with head trauma and diseases (e.g., strokes, intracranial haemorrhages, hydrocephalus, Chiari Malformations, etc.). The normal ICP in adults and older children is 10–15 mm Hg, in young children 3–7 mm Hg, and in full term infants 1.5–6 mm Hg.[7,8] Pressures above 15–20 mm Hg are considered pathological and warrant treatment.[9,10] Severe, uncontrolled hypertension with ICP >40 mm Hg may lead to catastrophic brain herniation and eventually death.

The goal of ICP monitoring is to ensure maintenance of an optimal CPP. It also forms the basis of all intervention, whether medical or surgical, including the use of osmotic agents (mannitol/3% saline), ventriculostomy procedures such as external ventricular drainage (EVD), barbiturate coma, or decompressive craniectomy in severe, intractable ICP elevation not responding to conservative management.

ICP monitoring is, therefore, important to confirm or exclude intracranial hypertension, so as to initiate ICP lowering measures at the earliest, and to assess whether these measures are effective. This assumes particular importance when the patient is sedated and paralysed, and conventional neurological assessment is not possible. Also, if ICP is not elevated, potentially dangerous treatment can be avoided.

DYNAMICS OF INTRACRANIAL PRESSURE

Intracranial pressure waveforms

ICP monitoring not only allows measurement of ICP at a given point of time but also provides information about intracranial dynamics and brain compliance from the waveform assessment. Information which can be derived

from ICP and its waveforms, includes – CPP, regulation of CBF and volume, CSF absorption capacity, brain compensatory reserve, and content of vasogenic events. Some of these parameters allow prediction of prognosis of survival following head injury and optimisation of 'CPP-guided therapy'.

Normal ICP trace is pulsatile and reflects cardiac and respiratory cycles [Figure 2]. The respiratory wave reflects changes in intrathoracic pressure with respiration. Its amplitude varies between 2 and 10 mm Hg. This respiratory variation diminishes and eventually disappears with rising ICP.

The pulse component of normal ICP waveform generally consists of three peaks, which correlate with the arterial pressure waveform occurring with each cardiac cycle [Figure 3]. These waveforms are generally 1–4 mm Hg in amplitude.

- The P1 wave, or the percussion wave, correlates with the arterial pulse transmitted through the choroid plexus into the CSF.
- The P2 wave, or the tidal wave, represents cerebral compliance and can be thought of as a 'reflection' of the arterial pulse wave bouncing off the springy brain parenchyma.
- The P3 wave, or the dicrotic wave, correlates with the closure of the aortic valve, which makes the trough prior to P3 the equivalent of the dicrotic notch.

The pulsatile nature of the CSF flow has been studied by many investigators since 1943, when the pulsatility of CSF pressure was first linked to vascular factors, and since then, the specific relationship and origin of CSF pulsation has been the subject of debate.[11] It was first suggested by some that CSF pulsations arise primarily from the large arteries of the brain,[12] while others suggested that it arises entirely from the pulsation of the choroid plexus. It was also found that CSF pressure in the lumbar subarachnoid space is attenuated compared to that in the ventricles and attributed this to the elasticity of the dural envelope.[13]

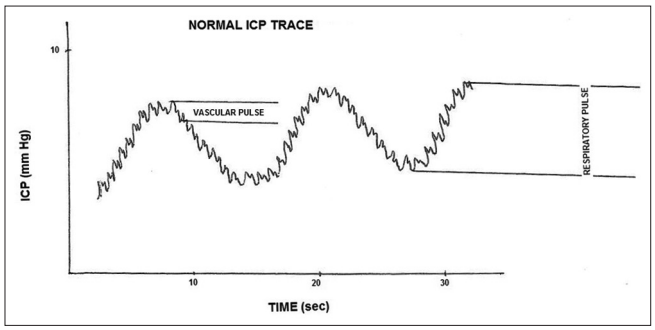

Figure 2: Normal intracranial pressure trace showing a respiratory and a cardiac component

Hamer *et al*. categorised the ICP pulse wave under normal and pathophysiological conditions. These authors found that in normal conditions the systolic portion of the intracranial CSF pulse wave is primarily arterial in origin, and the descending portion is similar to the superior vena cava pulse pressure. When ICP is raised, the descending part of the CSF pulse wave resembles the arterial pulse, and when central venous pressure is high, the pulsations assume a venous shape. Based on these observations, these authors concluded that the morphology of the CSF pulsations could be regarded as an index of the state of the intracranial elastance or compliance.[14]

As ICP increases above the resting level, the amplitude of the cardiac pulse component increases while the relative magnitude of the respiratory component may decrease. When the ICP is raised, the waveform assumes an arterialised shape (increase in P2). Visual inspection of waveforms can provide information about decreased intracranial compliance and altered intracranial dynamics – increased waveform amplitude, elevated P2, rounding of waveform, and appearance of plateau waves – all signify that intervention to decrease ICP is warranted [Figure 4].

ICP monitoring also helps in assessing the intracranial compliance, reflected by the slope of the pressure-volume curve at that point of time. Greater the change in ICP after the withdrawal of 1 ml of CSF (≥5 mm Hg), poorer is patient's intracranial compliance.[15]

Lundberg waves

When the ICP is increased, and the intracranial compliance is decreased, pathological waves appear.[16] Lundberg described these as A, B and C waves:

- Lundberg A waves, or plateau waves, are characteristic of conditions that lead to a markedly reduced intracranial compliance. These waves have an amplitude of 50–100 mm Hg and occur for a

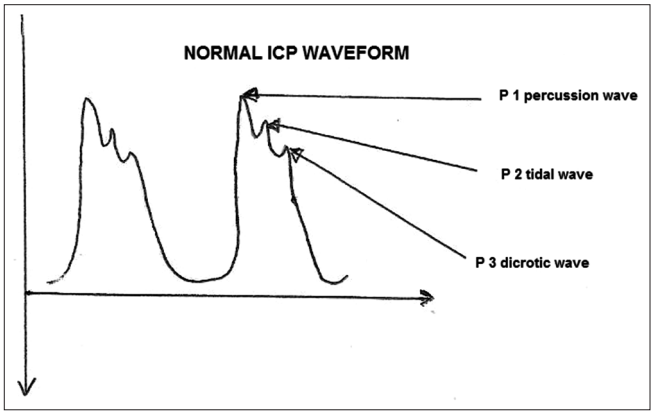

Figure 3: Intracranial pressure waveform reflecting three peaks – P1 (correlating with the arterial pulse); P2 (relating to the cerebral compliance); P3 (corresponding to aortic valve closure)

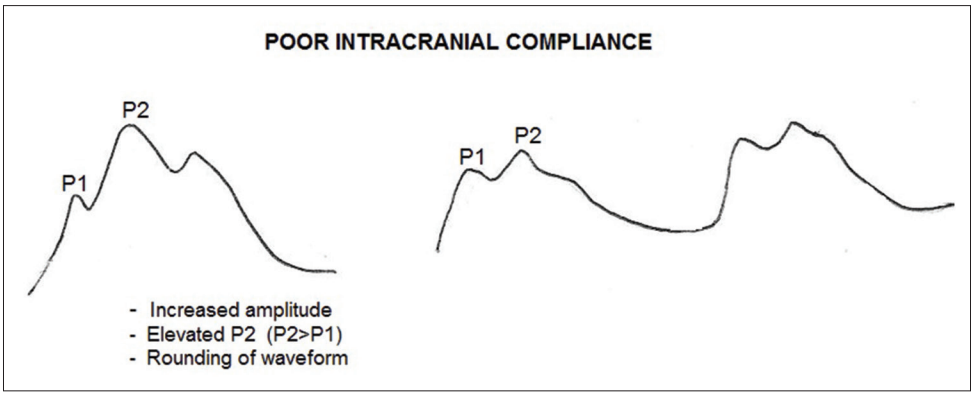

Figure 4: Schematic diagram of the intracranial pressure waveform in intracranial hypertension

duration of 5–10 min [Figure 5]. They are indicative of a very low CPP and ischaemia, and when present, are an ominous sign for the development of brain herniation if ICP is left untreated

- Lundberg B waves are rhythmic oscillations, sharply peaked, occurring every 1–2 min. ICP increases in a crescendo manner to 20–30 mm Hg from a variable baseline, and are not sustained [Figure 6]. These reflect vasomotor changes and are associated with an unstable ICP[17,18]
- Lundberg C waves correspond to Traube-Hering-Meyer fluctuations in arterial pressure brought about by oscillations in baroreceptor and chemoreceptor reflex control systems. They have been documented in healthy adults and have no clinical significance.

INDICATIONS OF INTRACRANIAL PRESSURE MONITORING

ICP monitoring is widely used in various neurological, neurosurgical and even medical conditions, such as hepatic encephalopathy. ICP monitoring is useful, if not essential, in head injury, poor grade subarachnoid haemorrhage, stroke, intracerebral haematoma, meningitis, acute liver failure, hydrocephalus, benign intracranial hypertension, craniosynostosis, etc. In hydrocephalus, CSF dynamic tests aid diagnosis and subsequent monitoring of shunt function.[8]

Perioperative ICP monitoring may be indicated in patients who have undergone resection of large brain tumours with mass effect, or arteriovenous malformations, and are at an increased risk of developing cerebral oedema and perfusion pressure breakthrough, in whom a clinical neurological examination is not possible.

The most common indication for ICP monitoring is traumatic brain injury (TBI).[19] The Brain Trauma Foundation guidelines (2007) support ICP monitoring in the following conditions:

- Salvageable patients with severe TBI with a Glasgow Coma Scale score (GCS) between 3 and 8 after resuscitation, and an abnormal computed tomography (CT) scan, that is, one showing haematomas, contusion, swelling, herniation, or compressed basal cisterns (Level II evidence)
- In patients with a GCS of 3–8 but a normal CT scan,

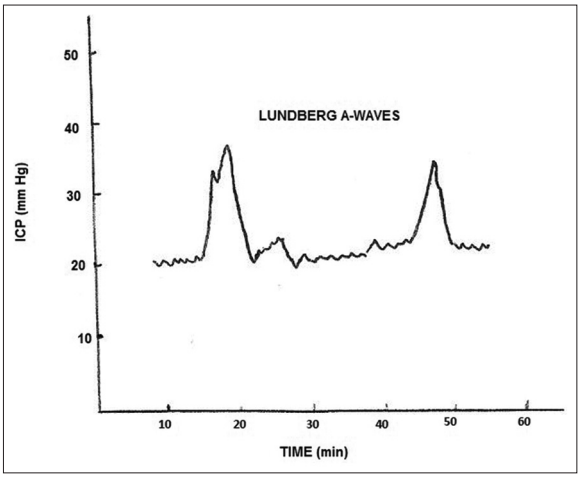

Figure 5: Schematic diagram depicting Lundberg A waves (intermittent sharp peaks)

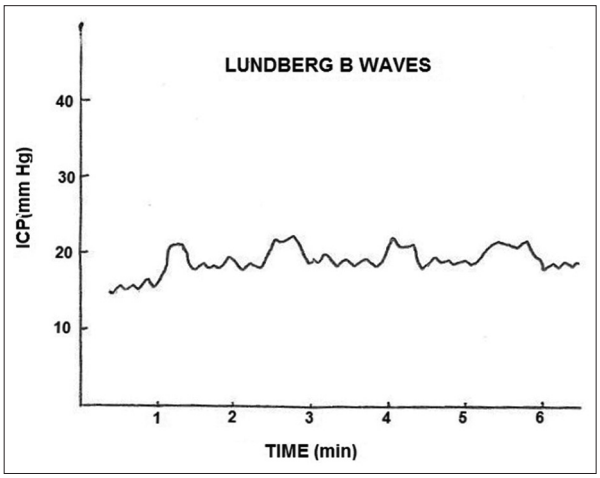

Figure 6: Schematic diagram showing Lundberg B waves

ICP should be monitored if two or more of the following conditions are present:

- Age over 40 years
- Unilateral or bilateral motor posturing, or
- Systolic blood pressure under 90 mm Hg (Level III evidence).

However, a recent multi-centric controlled trial, (BEST TRIP trial), revealed that for patients with severe TBI, care focused on maintaining ICP at 20 mm Hg or less done with intraparenchymal ICP monitoring, was not superior to care based on imaging and clinical examination.[20]

METHODS OF INTRACRANIAL PRESSURE MONITORING

ICP varies moment to moment and hence, single pressure recordings of ICP may miss fluctuations in ICP. At the same time, continuous recording of data may not be feasible. Marmarou *et al*. found that recording the ICP at the end of an hour ('end hour recording') was a good estimate of the ICP for the entire hour and this was used for data entry in research projects and visual scanning of the chart outputs for routine cases.[21]

NON-INVASIVE METHODS OF INTRACRANIAL PRESSURE MONITORING

The most accurate way to reliably diagnose elevated ICP is via a direct measurement approach with an invasive intracranial ICP monitor. But because of the invasiveness of these monitoring techniques (which require insertion of an ICP sensor into the ventricles or brain parenchyma), additional risks that they may pose to the patient (e.g., haemorrhage and infection), high costs associated with ICP sensor implantation, and limited access to trained personnel, that is, a neurosurgeon/neuro-intensivist in suburban locations, invasive monitoring may not always be done. Alternative methods to assess ICP non-invasively have, therefore, been sought. A detailed clinical examination along with imaging modalities (e.g., CT head and magnetic resonance imaging [MRI]) forms the basis for all ICP monitoring techniques.

Clinical examination

A thorough neurological evaluation to diagnose elevated ICP in patients remains the time-tested method of ICP monitoring. Symptoms such as headache, nausea, and vomiting may be early signs of raised intracranial tension. Patients are frequently assessed for deterioration in the level of consciousness (the Glasgow Coma Score), pupillary reactivity, and development of papilloedema[22] on fundoscopic examination. Vital signs monitoring may also help in diagnosing intracranial hypertension (Cushing's triad – bradycardia, hypertension and respiratory depression).

Imaging modalities

Non-contrast computed tomography scan

This is the fastest and the most cost-effective method to evaluate raised ICP and associated pathology. The presence of mass lesions, intracranial bleed or hydrocephalus, as a cause of intracranial hypertension, may be ruled out by means of a non-contrast CT head. Findings suggestive of a high ICP include cerebral oedema, midline shift, effacement of basal cisterns, loss of grey-white differentiation, and loss of normal gyri and sulci pattern.[23]

Magnetic resonance imaging

This modality is costly and time consuming, and hence not the first line investigation in the acute care setting. However, advances in dynamic MRI now enable visualisation and quantification of CSF flow dynamics which, in turn, has led to the development of a newer non-invasive method for measurement of intracranial compliance and pressure, the MR-ICP method. This MRI-based method of measuring ICP integrates human neurophysiology and fluid dynamic principles with dynamic MRI techniques to measure intracranial elastance (inverse of compliance) and ICP. It utilises the small fluctuations in intracranial volume and pressure that occur with each heartbeat. The intracranial elastance, that is, a change in pressure due to a small change in volume, is derived from the ratio of these changes. A mean ICP value is then derived from the linear relationship between ICP and elastance.[24]

The role of MRI-based ICP measurement is different from that of invasive techniques of ICP measurement. While invasive monitoring provides continuous ICP measurements, the MRI study provides a single time point measurement and therefore it serves only as a diagnostic test. However, there are several clinical settings in which a 'snapshot' of ICP may be beneficial.

Transcranial Doppler ultrasonography

This technique applies ultrasound to detect the velocity of blood flow through the major intracranial vessels, most commonly the middle cerebral artery. Elevated ICP can be estimated from the transcranial Doppler (TCD) measurements because it impedes the CBF and consequently decreases the blood flow velocity. Besides the mean velocity, pulsatility index (which is the difference between peak systolic and end diastolic velocity, divided by mean flow velocity), and slopes of the TCD waveforms have been correlated with ICP.[25-27] The estimates, however, have a margin of error of ± 10–15 mm Hg.[28] Besides, the technique requires training, and inter- and intra-observer variations may be seen.[29] Furthermore, it may be difficult in 10–15% patients who do not have an adequate bone window.

Tympanic membrane displacement

Because the CSF and perilymph communicate through the cochlear aqueduct, an increase in ICP is directly transmitted to the footplate of the stapes, changing its initial position and thereby affecting the direction and magnitude of the outwards displacement of the eardrum in response to a sound. This forms the basis for the tympanic membrane displacement (TMD) technique. Inwards displacement (negative peak pressure on an audiogram) is suggestive of high, and outwards of normal or low ICP.[30] Accuracy of TMD estimates of ICP are of the order of ± 15 mm Hg, which is not sufficient for a reliable quantitative assessment of ICP in clinical practice.[31] Moreover, the assumption that the pressure of perilymph is equal to ICP does not hold if the patency of the cochlear aqueduct is compromised, which is often the case in elderly subjects.

Optic nerve sheath diameter

The optic nerve is a part of the central nervous system and the space between the optic nerve and its sheath is a continuation of the subarachnoid space, filled with CSF, whose pressure is equal to the ICP. In cases of increased ICP, the diameter of the sheath increases, and the blood flow through the central retinal vein that courses through the sheath gets impeded (causing papilloedema). The diameter of the nerve sheath can be conveniently measured using a transocular ultrasound. This technique of ICP measurement is cheap and efficient and takes around 5 min for measurement. It has been validated as a screening method for identification of patients with raised ICP requiring treatment in several large studies, with optic nerve sheath diameter (ONSD) >5 mm corresponding to an ICP of 20 mm Hg or higher.[32,33] However, the ultrasonography technique requires training and has inter- and intra-observer variability.[34] Furthermore, conditions such as tumours, inflammation, Grave's disease and sarcoidosis, may alter the ONSD. Also, lesions of the orbit or optic nerve in patients with head injury may preclude nerve sheath diameter measurement.

ONSD is a promising technique for screening patients with raised ICP in settings where invasive ICP monitoring techniques are either not feasible (patients with severe coagulopathy) or not available (setups without access to a neurosurgeon).

INVASIVE METHODS OF INTRACRANIAL PRESSURE MONITORING

Several different invasive methods to measure ICP exist. The routine direct measurement of ICP by directly monitoring the fluid pressure in the ventricle has been credited to Lundberg way back in 1960.[35] The introduction of the subarachnoid screw bolt in 1973 was the next milestone in ICP monitoring,[36] which further led to the development of the newer techniques. Each type of device, depending on the intracranial location and method of pressure transduction, has its advantages and disadvantages.

Modern ICP monitors can be classified on the basis of pressure transduction method into, strain gauge or fibreoptic technology based monitors. The strain gauge pressure transduction can be external (intraventricular drains), or internal (catheter tip microchip). Another classification may be into – fluid coupled devices (connected to an external strain gauge), or non-fluid coupled devices (fibreoptic or catheter tip microstrain gauge).

Lumbar CSF pressure is very seldom measured in neuro-intensive care. This form of assessment of craniospinal dynamics is more often used in hydrocephalus[37] and benign intracranial hypertension.[38]

Current ICP monitors can be placed either in intraventricular, intraparenchymal, subarachnoid, subdural, or epidural locations. The intraventricular and the intraparenchymal catheter tipped microtransducers are the most commonly used.

Intraventricular devices

Invasive monitoring using the EVD technique, where a catheter is placed into one of the lateral ventricles through a burr hole, is considered the gold standard of ICP monitoring, and is the standard against which newer monitors are calibrated.[39-42] It is a fluid coupled device connected to an external strain gauge, and can be re-calibrated *in vivo* against an external reference at any time. The reference point for an external transducer should be the foramen of Monroe, 2 cm above the pterion on surface marking. However, most researchers take the external auditory meatus as the reference point for convenience. The EVD needs repositioning of transducer level with each change in head position.

Direct intraventricular ICP monitoring is the most reliable method in current use, and has the advantage of minimal expense with maximum accuracy. In addition to monitoring the ICP, it can also be used for therapeutic CSF drainage intermittently to control the ICP, and to instil medications intrathecally, e.g. antibiotic administration in ventriculitis, or instillation of thrombolytics in case of intraventricular haemorrhage or clotting at the proximal catheter.

This technique however, has its own set of pitfalls. It is the most invasive of all available techniques, as the ventriculostomy penetrates the meninges and brain, with the risk of bacterial transmission through the fluid coupling.[43,44] The documented risk of infection

varies between <1% and 27% in various studies, the mean EVD-associated infection rate being 8–9%. Strict adherence to aseptic technique while insertion, use of antibiotic impregnated catheters, subcutaneous tunnelling of the catheter, sterile occlusive dressing over the incision points, minimal manipulation, flushing or accessing of the CSF drainage tubing – have all been known to reduce infection rates.[45-47] Currently, catheters impregnated with silver nanoparticles are also being used to reduce the risk of catheter associated ventriculitis, but larger, multi-centric studies are needed to draw any firm conclusions on the same.[48] Routine change of EVD catheters, or antibiotic prophylaxis is not recommended at present.

Another disadvantage of the intraventricular method is that EVD placement may be difficult in young patients with compressed or slit-like ventricles, due to cerebral oedema.[49] The ICP waveform may be dampened in these cases, and the values recorded may be low due to artefact effect.

Caution should be exercised for overshunting, especially in patients with poor grade SAH (subarachnoid haemorrhage) who need an EVD for the relief of acute hydrocephalus, where over-drainage may lead to aneurysmal rebleed.[50] Also, CSF overdrainage in patients with large unilateral mass lesions may lead to hemispheric shifts and herniation.[51,52]

The potential risks of haemorrhage, misplacement, and obstruction of the catheter, are other complications of intraventricular catheter placements. The rate of clinically significant haemorrhages, (i.e., the ones causing neurological deficits, requiring surgical intervention, or resulting in fatality) have been reported to be 0.91–1.2% for studies using post-procedure CT scans.[53,54]

Optimal placement of the EVD is in the ipsilateral lateral ventricle anterior to the foramen of Monro, or into the top of the third ventricle, but avoiding the choroid plexus in the bottom of the third ventricle. The right hemisphere, which is non-dominant in 90% of the population, is the preferred site. Malplacements, either intraparenchymal or extraventricular, have been reported in various retrospective studies.[55] Injury of the basal ganglia can occur with ill directed or over enthusiastic attempts at ventricular cannulation.

Careful attention needs to be taken to assess for kinks in the catheter, and the drainage tubing to look for air bubbles, blood clots, or debris that could be blocking the free-flow of CSF or causing a dampened waveform.[56,57]

Subarachnoid devices

The subarachnoid bolt (or Richmond screw) is a hollow screw that goes into the skull abutting the dura.[36] The dura is perforated to fill the bolt with CSF, which is then connected to a closed fluid filled tubing and a pressure transducer. These devices may be quickly and easily placed, without invading the brain, and thus have lower infection rates. But, they are prone to errors such as ICP underestimation, misplacement of the screw, and occlusion by debris.[58] Their popularity has therefore declined in the recent years.

Epidural/subdural devices

They are an attractive option, as they are the least invasive, and can be easily and quickly placed. But, their questionable accuracy and reliability, coupled with the increasing baseline drift over time, makes them less popular choices as compared to intraventricular or intraparenchymal devices.[59]

Pneumatic sensor (Spiegelberg Brain Pressure Monitor)

This is a fluid filled catheter transducer system, which uses a distal air filled balloon tipped catheter to measure ICP [Figure 7].[60] The monitor connects to an internal strain gauge transducer used to measure the internal balloon pressure. The Spiegelberg monitor is capable of automatic zero drift correction *in vivo* which obviates the problem of re-zeroing and drift, but a disadvantage with this monitor is that its limited bandwidth makes most of the methods used for ICP waveform analysis impossible. It is a relatively low cost monitor (as compared to the contemporary microtransducers) classically used for epidural and subdural pressure measurements. A newer version of the monitoring system, using a double lumen intraventricular catheter system, also has the capacity of measuring intracranial compliance, simultaneously permitting CSF drainage as a treatment option [Figure 8]. Studies show that the Spiegelberg air-pouch ICP/compliance monitor provides ICP and compliance data that are very similar to those obtained using both gold standard methods and intraparenchymal ICP monitors over a range of pathological ICPs.[61,62]

Intraparenchymal devices

This group of devices are non-fluid coupled devices, and can be divided into two subtypes:

Figure 7: Spiegelberg brain pressure monitor

- Fibreoptic devices, e.g., the Camino ICP monitor, the Innerspace ICP monitor
- Strain gauge devices, e.g., the Codman MicroSensor, the Raumedic Neurovent P ICP sensor, and the Pressio sensor.

These ICP microtransducers can be used at any location – intraventricular, epidural, subdural or intraparenchymal; but those measuring ICP intraparenchymally, usually placed in the right frontal region at a depth of approximately 2 cm, are the most widely used. The microtransducers are accurate and a close correlation is observed between the ICP measured by them and that using the intraventricular drainage devices.[63] These can be easily transported, and their recordings are independent of patient positioning. Measurement artefacts, and damping of pressure waveform, a drawback seen with external transducers, are improved by the microtransducers. They are non-fluid coupled, therefore do not require irrigation and have a low risk of infection. The incidence of clinically significant haemorrhage is also negligible.

The disadvantages associated with the intraparenchymal monitors are: (i) They cannot be re-calibrated *in vivo*; (ii) they measure localised pressure, which may not be reflective of global ICP; (iii) therapeutic CSF drainage is not possible; and (iv) they may be subject to drift when used for long periods.

Fibreoptic catheter tip transducers (Camino intracranial pressure monitor)

Fibreoptic catheter tip transducer transmits light via a fibreoptic cable towards a displaceable diaphragm. Light is reflected off the diaphragm and the change in light intensity is interpreted in terms of pressure. It requires a dedicated microprocessor-driven amplifier to provide a numerical value and/or continuous waveform display. The system is costly and the catheters are disposable. Moreover, these catheters cannot be re-calibrated once inserted, and a drift (daily baseline drift of 0.3 mm Hg) occurs if the monitoring is to be continued for longer than 5 days. Also, these catheters are fragile, and if they are acutely bent during insertion or maintenance, or if the patient is restless, they can get damaged.[64-66]

Implanted microchip transducers (Codman sensors)

These consist of a miniature solid state pressure transducer, mounted on a titanium case, at the end of a 100 cm flexible nylon tube. The transducer tip contains a silicon microchip with diffuse piezoelectric strain gauges [Figures 9 and 10]. The microsensor monitors ICP at the source – intraparenchymal, intraventricular or subdural, and the information is relayed electronically, rather than through a hydrostatic system or fibreoptics. The Codman microsensor is accurate, and stable, with a

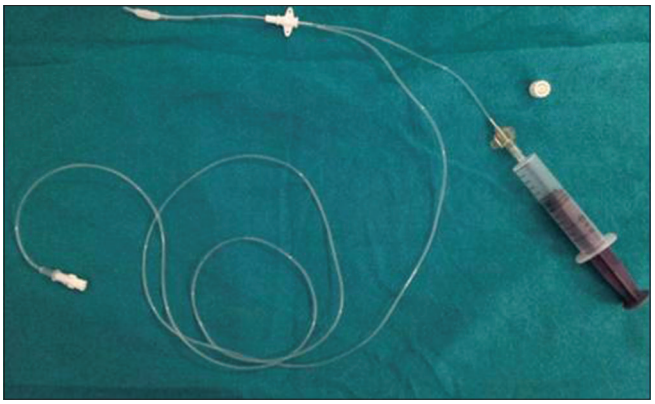

Figure 8: Spiegelberg brain pressure monitor and catheter for cerebrospinal fluid withdrawal. Note the distal balloon tipped catheter to measure intracranial pressure

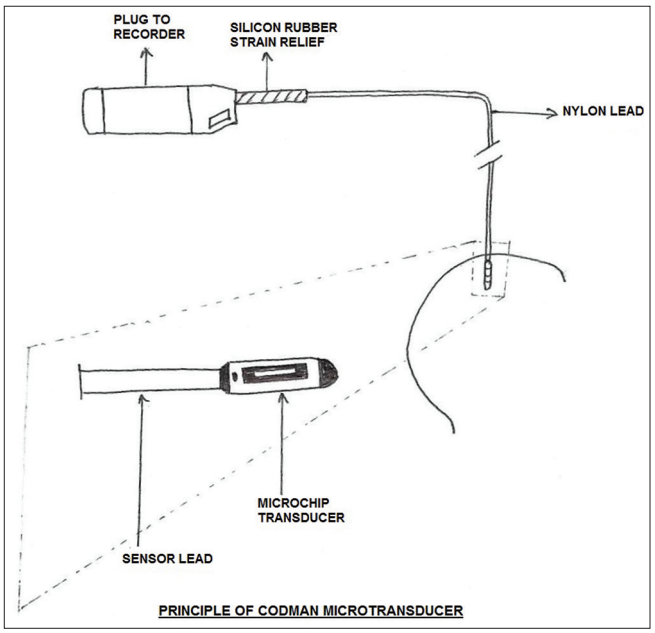

Figure 9: Principle of Codman microsensor. The Codman device has a pressure transducer, mounted on a titanium case, at the end of a 100 cm flexible nylon tube. The transducer tip contains a silicon microchip with diffuse piezoelectric strain gauges to measure the intracranial pressure

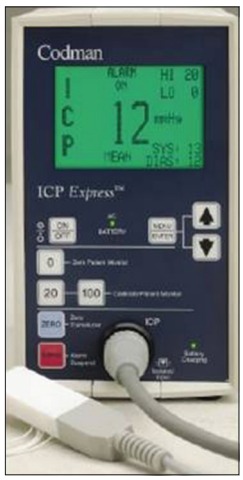

Figure 10: Codman microsensor

daily drift of − 0.13–0.11 mm Hg per day. When this is incorporated into a ventricular catheter, the system allows simultaneous drainage of CSF as well as ICP recording. It is flexible, and can be tunnelled beneath the scalp, preventing it from being easily broken. Its small size (a nominal outer diameter of 0.7 mm for the nylon vent tube and 1.2 mm for the transducer case) is an additional advantage, particularly for use in paediatric population. The absence of a fluid column precludes dampening by blood clots, debris, or air bubbles, and makes it less prone to infections. Also, the microsensor at the tip eliminates the need for constant realignment of the transducer with the patient's head and repeated re-zeroing.[67-69]

Which system to use?

This depends on several variables which include the underlying pathology, availability of trained personnel and costs. Direct intraventricular ICP monitoring is preferred if access to the ventricles is required, besides being reliable and cost-effective. However, this is not always feasible, especially in patients with severe head injury due to the presence of slit-like ventricles. In such situations, bedside ICP monitoring can be done using fibreoptic methods (Camino or InnerSpace) or implantable transducers (Codman) which can be inserted intraparenchymally. These three devices were compared for long-term and temperature zero drifts, frequency response characteristics, and the measurement error, by Czosnyka *et al*. These authors found that all three devices scored satisfactorily, with the Codman device scoring best overall.[70]

The Association for Advancement of Medical Instrumentation, in association with the Neurosurgery committee has developed the American Standard for ICP monitoring devices. According to their standards, an ICP device should have the following specifications:

- Pressure range from 0 to 100 mm Hg;
- Accuracy of ± 2 mm Hg within the range of 0–20 mm Hg
- Maximum error of 10% in the range of 20–100 mm Hg.[57]

To summarise, an optimal ICP monitoring device should be reliable and accurate, cost-effective, and associated with minimal patient morbidity. In the current state of technology, intraventricular catheters connected to an external strain gauge are considered the gold standard in ICP monitoring. Fibreoptic and microchip transducers have similar reliability and accuracy, but at a higher cost. The intraparenchymal probes are however, preferred if the patient has cerebral oedema or the ventricles are slit-like. Clinically significant infections or haemorrhage associated with ICP devices causing patient morbidity are rare and should not deter the decision to monitor ICP. Subarachnoid or subdural fluid coupled devices or epidural ICP devices are currently less accurate.

Is intracranial pressure monitoring useful?

ICP data can be used to predict outcome and worsening intracranial pathology, calculate and manage CPP, allow therapeutic CSF drainage with ventricular ICP monitoring and restrict potentially deleterious ICP reduction therapies. ICP is a robust predictor of outcome from TBI and threshold values for treatment are recommended based on this evidence (ICP threshold 20–25 mm Hg). ICP data are thus useful in assessing prognosis and guiding therapy, and there is an improvement in outcome in those patients who respond to ICP lowering therapies.[71-73]

An important consideration which needs to be made in patients with TBI is the danger of localised elevations of ICP due to compartmentalised pressure gradients caused by mass lesions. Care should therefore be exercised in the evaluation of patients where ICP and clinical symptoms differ markedly.[74,75]

CONCLUSION

ICP monitoring is a robust brain monitoring modality which can be used to predict outcome and guide treatment. Intraventricular catheters remain the gold standard for ICP monitoring, as they are the most reliable, accurate and cost-effective, and allow therapeutic CSF drainage. In the present day scenario, additional neuromonitoring techniques should supplement ICP in the critical care setting to increase patient safety, by more accurately guiding treatment options in terms of type, aggressiveness and duration of therapeutic strategies.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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