Review Article

Intracranial pressure monitoring: Vital information ignored

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Abstract

Though there is no Class I evidence for the benefit of intracranial pressure (ICP) monitoring, the bulk of the published literature supports its use when indicated. This review deals with the pathophysiology of raised ICP, evidence for and against monitoring, and basic guidelines for monitoring. It is unfortunate that ICP monitoring is not routinely performed in most of the centres in India due to the popular perception of it being risky, technologically complex and expensive. This article is an attempt to provide all the essential information on this complex topic without going into excessive detail, in the hope that ICP monitoring will be more widely used in India.

Key Words: Intracranial pressure monitoring, Cerebral perfusion pressure, Indications, Complications

Introduction

Intracranial pressure (ICP) has been systematically measured only for the last half a century, but the concept of raised pressure has been known for centuries, and was measured manometrically by Quincke in 1897. The seminal publication on ICP monitoring was by Guillame and Janny[1] in 1951. This study unfortunately did not gain the publicity it deserved as it was published only in French. The first widely read paper on systematic ICP monitoring was by Lundberg in 1960,[2] in which he acknowledged Janny's earlier work. Subsequently there have been numerous important publications on the incidence, pathophysiology and influence of raised ICP on outcome from various intracranial pathologies, but the next major impetus towards increasing the incidence of routine ICP monitoring was the publication of the Brain Trauma Foundation guidelines in 1995 and their updates in 2000.[3-5]

ICP monitoring has been used in subarachnoid hemorrhage, hydrocephalus, brain tumours, infarctions, nontraumatic intracerebral hemorrhage, Reye's syndrome and various intracranial infections for characterization, prognostication and treatment, but the most prominent use is in the field of head trauma. Since the preponderance of available literature deals with its use in trauma, the greater part of this review will inevitably deal with head injury.

Pathophysiology of raised intracranial pressure

The fundamental Munro Kelly concept is that the intracranial cavity is a closed and rigid compartment with three components: brain, blood and CSF, and that increase in any one component can be achieved only at the expense of another. Thus in the event of a growing mass lesion in the brain, the initial response would be a decrease in the volume of CSF and blood (mainly from the venous sinuses), and once this compensatory mechanism failed, the ICP would begin to rise signifi-

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cantly. This is the principle that underlies all the causes of raised ICP, most of which are multifactorial.

For example, in traumatic brain injury all the three components contribute to the rise in ICP to various degrees:

- Brain edema due to
  - Cellular damage
  - Blood-brain barrier damage
- Contusions and hematomas
- Increased blood volume due to vascular injury and consequent loss of tone
- CSF outflow resistance in the presence of significant traumatic subarachnoid hemorrhage

This is a very simplified outline of the complex mechanisms of intracranial hypertension, and there are elaborate mathematical and physical models to explain the intricate interplay of the various factors involved. The interested reader is referred to an excellent chapter on this subject by Marmarou.[6]

Cerebral perfusion pressure (CPP) and jugular venous saturation (SjvO2)

The concept of adequate cerebral perfusion as a means of improving outcome is a relatively recent development. The term CPP has been in use since at least 1972,[7] and is the difference between the mean arterial pressure and the ICP, mathematically expressed as:

CPP = MAP – ICP

The importance of the CPP in the treatment and outcome of head injury seems obvious now, but until two decades ago the basic drive of treatment was solely aimed at reducing brain edema by dehydration - “bring down the ICP and don’t worry too much about the blood pressure or volume unless the patient is actually hypotensive”. The relationship between blood pressure and cerebral blood flow was well known, but until Graham et al[8] demonstrated the high incidence of ischemic damage in fatal head injuries the influence of an inadequate CPP on outcome from injury was not completely understood. Rosner[8] is one of the strongest proponents of CPP based management.

The level of CPP to be maintained was conventionally accepted as 70 mmHg.[9] However some recent studies[10] suggest that a lower value of 60 mmHg might be sufficient as a compromise between adequate cerebral perfusion and the pulmonary and other systemic complications induced by attempts to maintain a higher CPP. In the author’s institution the practice is to tailor the CPP to the age of the patient - lower the target to 60 mmHg in the elderly who might not tolerate the cardiovascular stress necessary to maintain a CPP of 70 mmHg.

The degree of importance attached to ICP and CPP values differs among studies. Some authors claim that as long as an adequate CPP is maintained the ICP is of no concern.[11] Other large trials demonstrate that as long as the CPP is over 60 mmHg an ICP over 20 mmHg is the most powerful predictor of neurological deterioration.[12] The answer is probably closer to the latter opinion - raised ICP itself can have deleterious effects on brain tissue which are not entirely due to decreased perfusion, and therefore vigorous attempts must be made to control it while ensuring adequate perfusion.

Continuous jugular venous oxygen saturation monitoring gives us an idea of the degree of oxygen extraction by the brain, with a lower saturation indicating higher extraction and therefore indirectly decreased perfusion. Though there are numerous criticisms of SjvO2 including its inability to detect localized ischemia, episodes of desaturation have a strong correlation with outcome, independent of other variables. In a study by Gopinath et al[13] where an episode of desaturation was defined as a SjvO2 value less than 50% for 10 minutes or more, the percentage of patients with a poor neurological outcome was 90% with multiple episodes of desaturation and 74% in patients with one desaturation, compared to 55% in patients with no episodes of desaturation. Early detection of desaturation and manipulation of the CPP could prevent ischemia.

Why monitor ICP?

Lesions causing raised ICP can cause damage by tissue deformation, tissue shift causing brainstem or vascular compromise and by impairment of CPP with resultant ischemia. There has never been a prospective, randomized, controlled trial to prove the efficacy (or the lack of it) of ICP monitoring in improving outcome, and it seems unlikely that a randomized trial will ever be done due to the fact that ICP monitoring in severe head injury has come to be regarded as the standard of care. The Brain Trauma Foundation’s evidence based recommen-

dations for ICP monitoring are only at the level of a guideline and not a standard.[3] However there is a large body of evidence to indicate that it is of benefit to the patient.

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 cerebral edema or hematoma formation might be the signs of herniation. By alerting the medical team prior to this deterioration, monitoring enables an early intervention and improved outcome. Intervention when a small rise in ICP occurs has also been shown to prevent later profound intracranial hypertension.[14]

A management protocol solely based on repeated CT scans is economically not feasible for most of our patients, and has been shown to be less accurate than actual monitoring.[15] There is also evidence that time-bound repetition of CT scans does not contribute to patient management.[16] In addition to the lack of benefit, transporting a critically ill patient for investigation increases the risk for the patient and imposes a logistic strain on the ICU staff. ICP monitoring can indicate the need for a repeat imaging and avoid routine protocol-based investigation. At the author's institution the protocol requires repeat imaging for all patients who have an ICP >20 mmHg in spite of all measures short of paralysis, and the incidence of lesions requiring surgical evacuation in these patients is almost 50% (unpublished data). ICP monitoring should never be at the expense of clinical examination.

2. Limit avoidable therapy: Empirical therapy for presumed raised ICP 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 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.

4. Safety factor: ICP monitoring helps 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 level 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,[17] subarachnoid hemorrhage and severe head injury.[18]

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.[14,19,20] There is also data to show that even transient, controllable rises in ICP indicate a worse prognosis in head injury.[21]

8. Outcome: As mentioned earlier, there is no definite proof by way of a clinical trial that ICP monitoring improves outcome, but there are numerous reports indicating that patients who have their ICP monitored tend to have better outcomes. A complete discussion of this data is not within the scope of this review, and the reader is referred to a few prominent papers by Marshall et al,[12] Saul and Ducker[22] and Ghajar et al.[23] There is also the confounding factor that units that monitor ICP are those that generally provide more intensive care to their patients.

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: No randomized controlled trials exists demonstrating 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,[3] which would be logistically and financially difficult.

2. Outcome without monitoring: There have been studies demonstrating that the outcome for severe head injuries is as good without ICP monitoring.[24]

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 not yet settled, the closest approach to agreement being with regard to trauma. There is insufficient data on other disease conditions for the establishment of guidelines.

Monitoring techniques

There are several techniques available for monitoring that vary in accuracy, ease of use and cost. These have been ranked by the Brain Trauma Foundation based on their accuracy, stability and ability to drain CSF as follows:[5]

- Intraventricular devices – fluid-coupled catheter with an external strain gauge or catheter tip pressure transducer
- Parenchymal catheter tip pressure transducer devices
- Subdural devices – catheter tip pressure transducer or fluid-coupled catheter with an external strain gauge
- Subarachnoid fluid-coupled device with an external strain gauge
- Epidural devices

The reference standard for ICP monitoring is still the intraventricular device,[5] and it is necessary for all other systems to be compared against it for accuracy. Of the few centres in India that routinely monitor ICP, the majority use subarachnoid bolts that are comparatively less accurate and do not offer the therapeutic option of CSF drainage. The ability to drain CSF is an important additional treatment alternative in the control of raised ICP that is available only with ventricular catheters. It permits direct lowering of ICP without the systemic effects of all other therapeutic measures.

The Brain Trauma Foundation analysis of costs of various systems of ICP monitoring makes the statement that "generally, fluid-coupled ICP systems were less than half the cost of other systems".[5] The author has reported on an intraventricular monitoring device using disposables available in any ICU which also permits CSF drainage, at a total expense to the patient of less than Rs. 500.[25] The only other device with comparable accuracy is the catheter tip transducer device, which if used as a parenchymal device does not permit CSF drainage. It also requires the purchase of a dedicated monitor, and the disposables cost more than Rs. 4,000.

Patient selection

ICP monitoring is not a risk-free procedure, so the choice of patients to monitor will depend on the experience of the institution and the estimation of risk versus benefit for the particular patient. The incidence of raised ICP in mild head injury is only 3% and moderate head injury about 10-20%. Therefore, routine monitoring is not recommended in these groups of patients, though the physician may decide to monitor individual cases based on the clinical and radiological features. The incidence of raised ICP in severe head injury has been reported at over 50%[14,26] and therefore these patients are suitable for routine ICP monitoring. However a subgroup of these patients with normal CT scans have an incidence of raised ICP of only 13% unless they have any two of the following three risk factors: age >40 years, systolic BP <90 mmHg or motor posturing.[14] The other groups of patients in whom there is some data on ICP monitoring are subarachnoid hemorrhage[20,27] and hepatic failure,[28] though indications are not as well defined as those for trauma.

Treatment threshold

Normal ICP is 0-10 mmHg, and may be lower in children. Once the decision has been made to monitor the ICP, the level at which intervention is necessary has to be determined. The values at which various authors recommend treatment vary from 15-25 mmHg. Saul and Ducker demonstrated a significant improvement in out-

come after lowering the threshold from 25 mmHg to 15 mmHg.[22] Marmarou demonstrated that the strongest association between the outcome measured by the Glasgow Outcome Score and ICP was at a value of 20 mmHg,[21] and this is the generally accepted value at which interventions are started.

Complications of ICP monitoring

The potential complications of ICP monitoring include malposition, malfunction, infection and hemorrhage. The incidence of each of these varies with the type of monitoring being done and the experience of the personnel performing the monitoring. There is a definite learning curve for establishing and maintaining ICP monitoring, as well as for the optimal utilization of the information thus obtained.

1. Malposition: This is most commonly seen with intraventricular devices, where the catheter either misses the ventricle or is inserted too far into the ventricle. The subarachnoid bolt will under-read ICP if the dura is not properly opened, and similarly all other devices have their own need for a correct technique of insertion.

2. Malfunction: This is the commonest complication, occurring in different ways for different types of monitors. If too much CSF is drained, the ventricles collapse around the intraventricular catheters and they get blocked. Parenchymal catheters had a major problem of drifting zero point and they cannot be re-zeroed like the ventricular catheters, resulting in greater inaccuracy with length of monitoring. This problem has largely been solved now. The subarachnoid bolt can get plugged by brain tissue following which it will underestimate the ICP. These problems are also a matter of experience - at the author's institution more than 50% of the intraventricular catheters would be blocked within 48 hours (in the initial phase), but now the phenomenon is rare.

3. Infection: Infection in relation to ICP monitoring generally refers to a positive culture of CSF or the device, and reported infection rates vary widely and with the type of device. Intraventricular device infections range from 0%[29] to 10.5%.[30] There is a general consensus that the duration of monitoring has a direct relationship to incidence of infections and that the infection rate climbs steeply after 5 days,[31] though this can be mitigated by subcutaneous tunneling of the device. The Brain Trauma Foundation's section on the choice of monitoring technology[5] states "though these studies document increasing bacterial colonization of all ICP devices over time, clinically significant intracranial infections are uncommon".

4. Hemorrhage: The limited published reports of hemorrhage rates also vary, with an average incidence of 1.1% reported for intraventricular devices.[14,29] The incidence is approximately 2.8% for parenchymal devices[32] and 0% in the scarce literature on complications due to other monitoring devices.

Advanced analysis of ICP

The conventional application of ICP monitoring is the utilization of the mean pressure for decision making and manipulation of the ICP and CPP. In addition to the actual number alone, the utility of the ICP waveforms has been a subject of intense study. These analyses are used to derive additional information on brain compliance and the reactivity of the cerebrovascular bed. Decreasing brain compliance gives a warning of impending ICP rise, enabling earlier intervention.

The correlation coefficient between amplitude and mean pressure[33] (RAP coefficient) is useful in deriving the compensatory reserve in the intracranial cavity for further rises in volume. A RAP coefficient close to 0 indicates a good pressure-volume compensatory reserve at low ICP, where a change in volume produces no or very little change of the pressure. When RAP rises to +1 it indicates a shift to the right towards the steep part of the pressure-volume curve. Here compensatory reserve is low; therefore any further rise in volume may produce a rapid increase in ICP. The pressure volume index is another good indicator of intracranial compliance.[34] Computerised analysis of the ICP waveform using a derived high frequency centroid can provide information on compliance without manipulation of intracranial volume.[35]

Computer analysis of slow waves in ABP and ICP is able to provide a continuous index of cerebrovascular reactivity to changes in arterial pressure. This pressure-reactivity index (PRx) has been shown to be good indicator of prognosis.[36] Advancing technology that enables us to detect increases or potential increases in ICP at an earlier stage will help us maintain ICP control, with

resulting benefit to the patients.

Tailpiece

The standard question asked by proponents of ICP monitoring is "Would you treat arterial hypertension without measuring the blood pressure?" We measure the blood pressure to detect the need for and effect of treatment, as all therapies used are known to have side effects and one would not want to unnecessarily expose the patient to this risk. Admittedly the process of measuring ICP is not as easy or risk-free as the measurement of blood pressure, but with indications and techniques for ICP measurement becoming increasingly refined, exposure of patients to the risk of undertreatment or unnecessary empirical treatment of raised ICP is not an acceptable solution. The lack of Class I evidence does not mean that it has been proved to be of no benefit. Patients who are at significant risk of developing intracranial hypertension should have ICP monitoring to ensure appropriate management.

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