DOI-10.21304/2014.0104.00044

Intracranial Pressure and its Monitoring: A review

Suresh Panda, Rakshay Shetty
Rainbow Childrens Hospital, Hyderabad

Received:18-Oct-2014/Accepted:25-Oct-2014/Published online:15-Nov-2014

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.[3] 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.

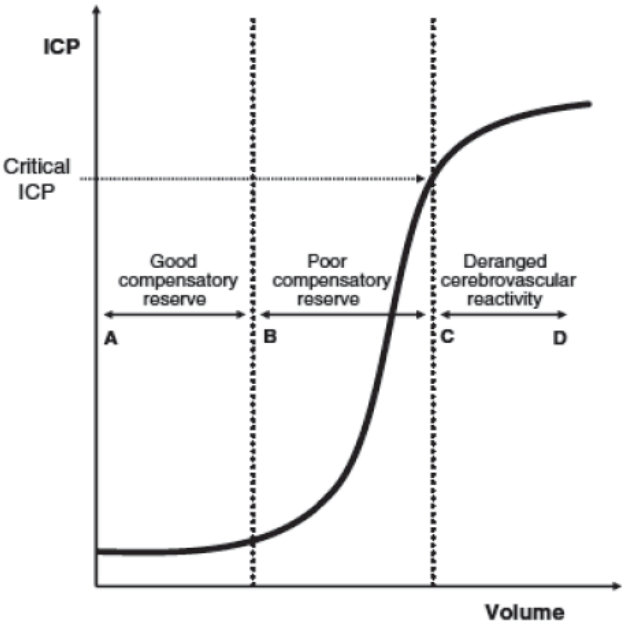

Figure 1. Intracranial pressure (ICP) volume curve. The curve has three parts: a flat part representing good compensatory reserve (A-B), an exponential part representing reduced compensatory reserve (B-C) and a final flat part representing terminal derangement of cerebrovascular responses at high ICP (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, [16] particularly if the period of intracranial hypertension is prolonged.[17] 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 ow

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[2], whereas CPP elevation above the upper limit of autoregulation can be associated with hyperemia, exacerbation of vasogenic edema, and increased ICP[3]. 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.

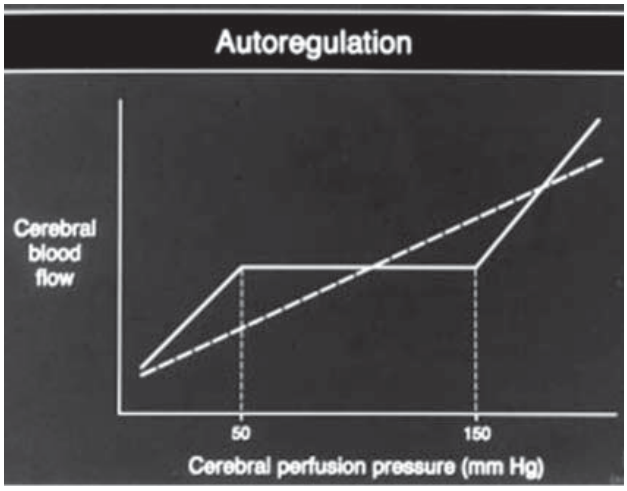

Fig 2: Cerebral autoregulation curve. In the normal relationship (solid line), with CBF held constant across a wide range of CPP (50±150 mmHg). In disease states (e.g., vasospasm, ischemia, intracranial mass lesion), cerebral blood flow may become pressure passive (dotted line).

Normal and Pathologic ICP

Table 2: Normal intracranial pressure values

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

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, [9] whereas after TBI, treatment is indicated when ICP exceeds 20 mm Hg.[10] 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 upto 8-yr-of-age and 20 mm Hg in older children and teenagers.[11]

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.[12] In the injured brain, there may be intraparenchymal pressure gradients between the supra and infra-tentorial compartments[13] and bilateral monitoring has revealed differential pressures across the midline in the presence of hematomas[14] and also in the absence of space-occupying lesions.[15]

Causes of Raised Intracranial Pressure

Table 3

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

Methods of ICP Monitoring

ICP cannot be reliably estimated from any specific clinical feature or computed tomography (CT) finding and must actually be measured.

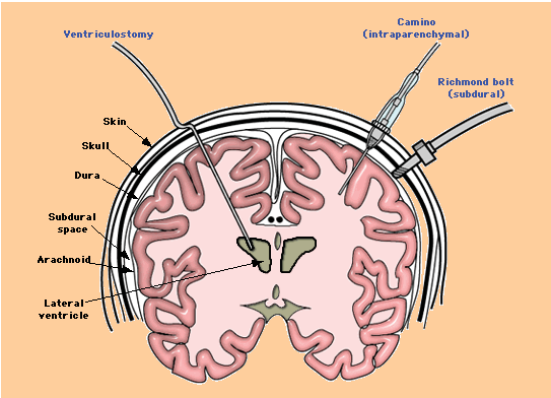

Figure 3: Intracranial pressure monitors ventriculostomy allows both ICP monitoring and therapeutic drainage of cerebrospinal fluid (CSF). Subdural and intraparenchymal monitors cannot be used to drain CSF.

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[33]. 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[34].

Epidural Transducers

These devices (the Gaeltec device) are inserted deep into the inner table of the skull and superficial to the dura[35]. In most of these devices, pressure is transduced by an optical sensor. They have a low infection rate (approximately 1%)[29], 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)[38,39] 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[36]. 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[37]

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 5: Indications of ICP Monitoring

Severe head injury
Intracerebral hemorrhage
Subarachnoid hemorrhage
Hydrocephalus
Stroke
Central nervous system infections
Hepatic encephalopathy

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.[40,41] 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.

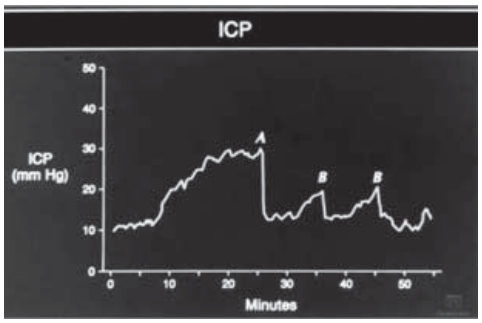

FIG 4: Pathologic ICP elevations. (A) Lundberg A (plateau) waves. (B) Lundberg B waves. (Reprinted from Mayer SA. Management of increased intracranial pressure. In: Wijdicks EFM, Diringer MN, Bolton CF, et al. Continuum: Critical Care. Minneapolis, MN: American Academy of Neurology, 1997:47±61.)

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[43]. 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[44]. There is also evidence that time-bound repetition of CT scans does not contribute to patient management[45]. In addition to the lack of benefit, transporting a critically ill patient for investigation increases the risk for the patient and imposes 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,[46] subarachnoid hemorrhage and severe head injury.[47]

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[43,48,49]. There is also data to show that even transient, controllable rises in ICP indicate a worse prognosis in head injury[46].

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[42], 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.

Conflict of Interest: None **Source of Funding:** None

References

1. Marmarou A, Anderson RL, Ward JD, Choi SC, Young HF, Eisenberg HM, Foulkes MA, Marshall LF, Jane JA. Impact of ICPinstability and hypotension on outcome in patients with severe head trauma. J Neurosurg 1991; 75: S59-66
2. Marmarou A. Increased intracranial pressure in head injury andinfluence of blood volume. J Neurotrauma 1992; 9: S327-32
3. Czosnyka M. Association between arterial and intracranialpressures. Br J Neurosurg 2000; 14:127-8
4. Marmarou A, Fatouros PP, Barzo P, Portella G, Yoshihara M,Tsuji O, Yamamoto T, Laine F, Signoretti S, Ward JD, BullockMR, Young HF. Contribution of edema and cerebral blood volume to traumatic brain swelling in head-injured patients. JNeurosurg 2000;93:183-93
5. Unterberg AW, Stover J, Kress B, Kiening KL. Edema and braintrauma. Neuroscience 2004;129:1021–96. Marmarou A. Pathophysiology of traumatic brain edema: currentconcepts. ActaNeurochirSuppl 2003;86:7–10
7. Marmarou A, Signoretti S, Aygok G, Fatouros P, PortellaG. Traumatic brain edema in diffuse and focal injury: cellular orvasogenic? ActaNeurochirSuppl 2006;96:24–9
8. Albeck MJ, Skak C, Nielsen PR, Olsen KS, Borgesen SE, GjerrisF. Age dependency of resistance to cerebrospinal fluid outflow.JNeurosurg 1998;89:275–8
9. Czosnyka M, Pickard JD. Monitoring and interpretation ofintracranial pressure. J NeurolNeurosurg Psychiatry 2004; 75:813-21
10. The Brain Trauma Foundation. The American Association ofNeurological Surgeons. The Joint Section on NeurotraumaandCritical Care. Intracranial pressure thresholds. J Neurotrauma 2007;24:S55–8
11. Mazzola CA, Adelson PD. Critical care management of head trauma in children. Crit Care Med 2002;30:S393–401
12. Wolfla CE, Luerssen TG, Bowman RM. Regional brain tissue pressure gradients created by expanding extradural temporal mass lesion. J Neurosurg 1997;86:505–10
13. Rosenwasser RH, KleinerLI, Krzeminski JP, Buchheit WA. Intracranial pressure monitoring in the posterior fossa: a preliminary report. J Neurosurg 1989;71:503–5
14. Chambers IR, Kane PJ, Signorini DF, Jenkins A, MendelowAD. Bilateral ICP monitoring: its importance in detecting the severityof secondary insults. ActaNeurochirSuppl 1998;71:42–3
15. Mindermann T, Gratzl O. Interhemispheric pressure gradientsin severe head trauma in humans. ActaNeurochirSuppl1998; 71:56-8
16. Balestreri M, Czosnyka M, Hutchinson P, Steiner LA, Hiler M, Smielewski P, Pickard JD. Impact of intracranial pressure and cerebral perfusion pressure on severe disability and mortality after head injury. Neurocrit Care 2006;4:8–13
17. Saul TG, Ducker TB. Effect of intracranial pressure monitoring and aggressive treatment on mortality in severe head injury.J Neurosurg 1982; 56:498–503
18. The Brain Trauma Foundation. The American Association of Neurological Surgeons. The Joint Section on Neurotrauma and Critical Care. Intracranial pressure monitoring technology.J Neurotrauma 2007; 24: S45–54
19. Citerio G, Andrews PJ. Intracranial pressure. Part two: clinical applications and technology. Intensive Care Med 2004; 30:1882–5
20. Steiner LA, Andrews PJ. Monitoring the injured brain: ICP and CBF. Br J Anaesth 2006;97:26–38
21. Zhong J, Dujovny M, Park HK, Perez E, Perlin AR, Diaz FG. Advances in ICP monitoring techniques.Neurol Res 2003; 25:339-50
22. Lozier AP, Sciacca RR, Romagnoli MF, Connolly ES Jr. Ventriculostomy-related infections: a critical review of the literature.Neurosurgery 2002; 51:170–81
23. Mayhall CG, Archer NH, Lamb VA, Spadora AC, Baggett JW,Ward JD, Narayan RK. Ventriculostomy-related infections.Aprospective epidemiologic study. N Engl J Med 1984; 310:553-9
24. Lo CH, Spelman D, Bailey M, Cooper DJ, Rosenfeld JV, BrecknellJE. External ventricular drain infections are

independent ofdrain duration: an argument against elective revision. J Neurosurg 2007; 106: 378-83 246 Intracranial Pressure Monitoring ANESTHESIA & ANALGESIA
25. Birch AA, Eynon CA, Schley D. Erroneous intracranial pressure measurements from simultaneous pressure monitoring and ventricular drainage catheters. Neurocrit Care 2006; 5:51-4
26. Zabramski JM, Whiting D, Darouiche RO, Horner TG, Olson J, Robertson C, Hamilton AJ. Efficacy of antimicrobialimpregnatedexternal ventricular drain catheters: a prospective,randomized, controlled trial. J Neurosurg 2003;98:725–30
27. Kaufmann AM, Lye T, Redekop G, Brevner A, Hamilton M,Kozey M, Easton D. Infection rates in standard vs. hydrogelcoated ventricular catheters. Can J NeurolSci 2004; 31:506-10
28. Ostrup RC, Luerssen TG, Marshall LF, et al. Continuousmonitoring of intracranial pressure with a miniaturized fiberoptic device. J Neurosurg1987; 67:206±209
29. Gopinath SP, Robertson CS, Contant CF, et al. Clinical evaluationof a miniature strain-gauge transducer for monitoring intracranial pressure. Neurosurgery 1995; 36: 1137±1140
30. Levin A. The use of a fiberoptic intracranial pressure monitorin clinical practice. Neurosurgery 1977;1:266±271
31. Sundbarg G, Nordstrom CH, Messeter K, et al. A comparison of intraparenchymatous and intraventricular pressure recording in clinical practice. J Neurosurg1987; 67:841±845
32. Yau Y-H, Piper IR, Clutton RE, et al. Experimental evaluationof the Spielberger intracranial pressure and intracranialcompliance monitor. Technicalnote. J Neurosurg 2000; 93: 1072±1077
33. Vries JK, Becker DP, Yang HF.A subarachnoid screw formonitoring intracranial pressure. J Neurosurg 1973; 39: 416±419
34. Miller JD, Bobo H, Kapp JP. Inaccurate pressure readingsfrom subarachnoid bolts. Neurosurgery 1986;19:253±255
35. Ream AK, Silverberg GD, Corbin SD, et al. Epidural measurement of intracranial pressure. Neurosurgery 1979; 5: 36±43
36. Hassler W, Steinmetz H, Gawlowski J. Transcranial Dopplerultrasonography in raised intracranial pressure and in intracranial circulatory arrest. J Neurosurg 1988;68:745±751
37. Mayer SA, Thomas CE, Diamond BE. Asymmetry of intracranialhemodynamics as an indicator of mass effect in acuteintracerebral hemorrhage: a transcranial Doppler study. Stroke 1996;27:1788±1792
38. Schmidt B, KlingelhoÈfer J, Schwarze JJ, et al. Noninvasiveprediction of intracranial pressure curves using transcranialDoppler ultrasonography and blood pressure curves. Stroke1997; 28: 2465±2472
39. Pranevicius O, Bertasius K, Pranevicius K, et al. Non-invasivedynamic assessment of the elasticity of intracranialstructures. ActaNeurol Scand 1992; 86:512 ±516
40. Czosnyka M, Smielewski P, Piechnik S, et al. Critical closingpressureincerebrovascularcirculation. J Neurol Neurosurg Psychiatry 1999; 66: 606±611
41. Lundberg N, Troupp H, Lorin H. Continuous recording of theventricular-fluid pressure in patients with severe acute traumaticbrain injury. A preliminary report. J Neurosurg 1965; 22: 581–90
42. The Brain Trauma Foundation. The American Association of Neurological Surgeons.The Joint Section on Neurotrauma and Critical Care.Indications for intracranial pressure monitoring. J Neurotrauma 2000; 17(6-7): 479-91
43. Narayan RK, Kishore PR, Becker DP, Ward JD, Enas GG, Greenberg RP, Domingues Da Silva A, Lipper MH, Choi SC, Mayhall CG, Lutz HA 3rd, Young HF. Intracranial pressure: to monitor or not to monitor? A review of our experience with severe head injury.J Neurosurg. 1982; 56(5):650-9.
44. Miller MT, Pasquale M, Kurek S, White J, Martin P, Bannon K, Wasser T, Li M. Initial head computed tomographic scan characteristics have a linear relationship with initial intracranial pressure after trauma. J Trauma. 2004; 56(5):967-72.
45. Kaups KL, Davis JW, Parks SN. Routinely repeated computed tomography after blunt head trauma: does it benefit patients? J Trauma. 2004; 56(3):475-80.
46. Cho DY, Chen TC, Lee HC. Ultra-early decompressivecraniectomy for malignant middle cerebral artery infarction.Surg Neurol. 2003; 60(3):227-32.
47. Strege RJ, Lang EW, Stark AM, Scheffner H, Fritsch MJ, Barth H, Mehdorn HM. Cerebral edema leading to decompressivecraniectomy: an assessment of the preceding clinical and neuromonitoring trends. Neurol Res. 2003; 25(5):510-5.
48. Becker DP, Miller JD, Ward JD, Greenberg RP, Young HF, Sakalas R. The outcome from severe head injury with early diagnosis and intensive management.J Neurosurg. 1977; 47(4):491-502.
49. Heuer GG, Smith MJ, Elliott JP, Winn HR, LeRoux PD. Relationship between intracranial pressure and other clinical variables in patients with aneurysmal subarachnoid hemorrhage. J Neurosurg. 2004; 101(3):408-16.
50. Stuart GG, Merry GS, Smith JA, Yelland JD. Severe head injury managed without intracranial pressure monitoring. J Neurosurg. 1983; 59(4): 601-5.