Chapter 3. Indications for intracranial pressure monitoring

I. RECOMMENDATIONS

Strength of Recommendations: Weak.

Quality of Evidence: Low, from poor and moderate-quality class III studies.

A. Level I

There are insufficient data to support a level I recommendation for this topic.

B. Level II

There are insufficient data to support a level II recommendation for this topic.

C. Level III

Use of intracranial pressure (ICP) monitoring may be considered in infants and children with severe traumatic brain injury (TBI).

II. EVIDENCE TABLE (see Table 1)

III. OVERVIEW

Secondary injury to the brain after severe TBI occurs, in part, as a result of reduced perfusion of surviving neural tissue, resulting in reduced oxygen and metabolite delivery and reduced clearance of metabolic waste and toxins. Secondary injury also occurs as the result of cerebral herniation syndromes, resulting in focal ischemic injury and brain stem compression along with other mechanisms. Intracranial hypertension represents a key pathophysiological variable in each of these secondary injury mechanisms (1–3).

Since the late 1970s, significant improvements in both survival and functional outcome after severe TBI have been achieved using intensive care management protocols that center on the measurement of ICP and medical and surgical treatment of intracranial hypertension (4). A study by Tilford and colleagues (5) demonstrated that an intensive care unit with higher incidence of ICP monitoring in severely brain-injured children, plus certain medical interventions, had a trend toward lower mortality than two other pediatric intensive care units. Similarly, a study by Tilford and colleagues (4) demonstrated improved outcomes after severe TBI in an era during which the overall rates of ICP monitoring in these patients increased. Attempts to evaluate the independent benefit of direct ICP measurement to improve outcomes, *per se*, are confounded by the numerous therapeutic interventions that have been simultaneously introduced and have not been subjected individually to controlled trials. These confounders include protocol-driven prehospital care, tracheal intubation and oxygenation, aggressive treatment of systemic hypotension and hypovolemia, osmolar treatment of cerebral edema, rapid cranial computed tomography (CT) imaging to detect mass lesions, improved enteral and parenteral nutrition, among others.

Several studies demonstrate an association between intracranial hypertension and/or systemic hypotension and poor outcome after severe TBI (6–8). It is less clear, however, whether intracranial hypertension or reduced cerebral perfusion secondary to intracranial hypertension is the primary mechanism of secondary injury. Cerebral perfusion pressure (equals mean arterial pressure minus ICP) is the simplest correlate of global cerebral perfusion (9–12). The relative value of ICP monitoring as a means of evaluating and manipulating cerebral perfusion pressure, vs. avoidance of cerebral herniation events, is also unclear (13).

The lack of controlled trials on ICP monitoring limited the strength of the recommendations contained in the first edition of the *Guidelines for the Management of Severe TBI in Children* (14). This dearth of strong evidence is associated with mixed adoption of guidelines-directed management in the United States and abroad (15–17). In a 2007 survey of U.S. neurosurgeons and nonneurosurgeons caring for such patients, Dean et al (15) found approximately 60% agreement and conformity with guidelines recommendations. In the United Kingdom, only 59% of children presenting with severe TBI underwent ICP monitoring with only half of clinical units caring for such children using monitoring technology (16, 17). The use of monitoring in children <2 yrs of age with severe TBI may be even less likely. A study by Keenan et al (18) observed use of ICP monitoring in only 33% of patients in this young age group at multiple centers in the state of North Carolina. There is also significant variability in the incidence of using various interventions for the treatment of intracranial hypertension at different centers (5).

IV. PROCESS

For this update, MEDLINE was searched from 1996 through 2010 (Appendix B for search strategy), and results were supplemented with literature recommended by peers or identified from references lists. Of 36 potentially relevant studies, seven studies were added to the existing table and used as evidence for this topic.

V. SCIENTIFIC FOUNDATION

Two moderate and 14 poor-quality class III studies met the inclusion criteria for this topic and provide evidence to support the recommendation (9, 19–33).

Are Children With Severe TBI at Risk of Intracranial Hypertension?

A number of small studies demonstrate a high incidence of intracranial hypertension in children with severe TBI (20, 21, 23, 24, 26, 28, 31, 33). Some of these studies identify in preliminary fashion other clinical factors that, in combination with severe TBI in a child, are indicative of a high incidence of intracranial hypertension. In these patients, "diffuse cerebral swelling" on CT scan is 75% specific for the presence of intracranial hypertension (26). In a study of 56 severely brain-injured patients (39 of whom had severe TBI), 32% of children had an initial ICP measurement >20 mm Hg but 50% had ICP maximum >20 mm Hg at some point during their intensive care

DOI: 10.1097/PCC.0b013e31823f440c

Table 1. Evidence table

Reference	Study Description	Data Class, Quality, and Reasons	Results and Conclusion
Studies from previous guidelines			
Alberico et al, 1987 (19)	Design: single-center, prospective, observational study N = 100 Age: 0–19 yrs Glasgow Coma Scale score: ≤7 Purpose: Assessment of relationship between ICP and outcome Outcome: GOS score at 3 months and 1 yr	Class III Poor quality: no control for confounders	Reducible intracranial hypertension was significantly associated with better outcome than nonreducible intracranial hypertension
Barzilay et al, 1988 (20)	Design: retrospective case series with analysis of minimum ICP N = 56 Age: mean 6.2 yrs Purpose: assessment of relationship between ICP and outcome in patients treated for high ICP with hyperventilation and medical management Outcome: survival at hospital discharge	Class III Poor quality: no control for confounders	For children with severe TBI, ICP maximum was 16.9 ± 3.1 in survivors (N = 32) and 53.7 ± 10.8 in nonsurvivors (N = 9); $p < 0.01$
Bruce et al, 1979 (21)	Design: single-center, observational study N = 85, 40 had ICP monitoring Age: 4 months to 18 yrs Purpose: assess relationship between ICP monitoring and medical management in a protocol emphasizing hyperventilation therapy to control intracranial hypertension, but also including barbiturates, mannitol, and/or surgery Outcome: dichotomized GOS at 6 months	Class III Poor quality: no control for confounders	Intracranial hypertension (ICP >20 mm Hg) was more prevalent in children without (80%) than with (20%) spontaneous motor function Of the total group (N = 85): 87.5% of children achieved good recovery or moderate disability; 3.5% persistent vegetative state, 9% died. Of those who had ICP monitoring (N = 40): Level of ICP related to outcome: ICP <20 (N = 9): 67% good recovery/moderate disability; 11% severe disability/persistent vegetative state; 22% died ICP >20 ≤40 (N = 17): 88% good recovery/moderate disability; 6% severe disability/persistent vegetative state; 6% died ICP >40 (N = 14): 57% good recovery/moderate disability; 7% severe disability/persistent vegetative state; 36% died
Chambers et al, 2001 (9)	Design: single-center, observational study N = 84 Age: 0–16 yrs Purpose: assessment of relationship between ICP and CPP and outcome Outcome: GOS at 6 months	Class III Poor quality: no control for confounders; unclear if patient selection was unbiased	ICP maximum predictive of poor outcome was >35 mm Hg in adults and children
Downard et al, 2000 (22)	Design: retrospective review N = 118 Age: <15 yrs Glasgow Coma Scale score: mean 6, 84% <8 Purpose: assess relationship among ICP, CPP, and outcome in children with severe TBI in two trauma centers Outcome: the final available GOS in the medical record	Class III Poor quality: as an intervention study; moderate quality as a prognosis study: logistic regression performed to determine factors associated with GOS, but no comparison of groups based on any intervention	In a stepwise logistic regression analysis, ICP >20 mm Hg was significantly associated with an increased risk of death

Table 1.—*Continued*

Reference	Study Description	Data Class, Quality, and Reasons	Results and Conclusion
Esparza et al, 1985 (23)	Design: single-center, observational study N = 56 Age: 3 months to 14 yrs Purpose: assessment of relationship between ICP monitoring and surgical and medical therapy and outcome after severe TBI in children Outcome: GOS dichotomized as good (mild disability) or poor (disability, persistent vegetative state, or death)	Class III Poor quality: no control for confounders; unclear if outcome assessment was unbiased	Outcomes were as follows: 93% good, 3% poor for patients with ICP maximum ≤20 mm Hg, 71% good, 29% poor for patients with ICP maximum 20–40 mm Hg; 0% good, 100% poor for patients with ICP maximum 40–60 mm Hg and 0% good, 100% poor for patients with ICP maximum >60 mm Hg (no significance test reported)
Kasoff et al, 1988 (24)	Design: single-center, retrospective, observational study N = 25 Age: 3 months to 17 yrs Purpose: assess relationship between ICP and outcome in children treated with mannitol and if refractory, mannitol plus barbiturates Outcome: mortality	Class III Poor quality: no control for confounders; unclear if patients selection was unbiased	Mortality rate was 20% Children with elevated ICP had a lower survival rate than children with normal ICP, although no statistical analysis is presented Mean highest ICP of those who died was 81 mm Hg (range, 55–120); for ICP only group 18.7 (range, 10–30), for mannitol group 42.11 (range, 10–70), for pentobarbital and mannitol group 72 (range, 30–120) Four children had normal ICP and did not require medical therapy; nine required mannitol therapy and eleven mannitol and then barbiturate therapy for sustained intracranial hypertension
Michaud et al, 1992 (25)	Design: single-center, observational study N = 51 Age: 3 months to 14 yrs Purpose: assessment of relationship between ICP and outcome Outcome: GOS at discharge	Class III Moderate quality: no power calculation; otherwise met all criteria	94% of children with ICP maximum <20 mm Hg vs. 59% with ICP maximum >20 mm Hg survived ($p = 0.02$) 48% of children with ICP elevation >1 hr survived compared to 89% of children with ICP elevated for <1 hr Outcome was also better in children with ICP elevation for <1 hr No statistically significant relationship was found between peak ICP and degree of disability
Shapiro and Marmarou, 1982 (26)	Design: retrospective case series N = 22 Age: 3 months to 15 yrs Purpose: study the use of pressure volume index assessment using external ventricular drains Outcome: GOS—time of assessment not indicated	Class III Poor quality (diagnostic study): narrow spectrum of patients enrolled; small sample size; unclear if reliability of test assessed	86% of children with severe TBI had ICPs exceeding 20 mm Hg "Diffuse cerebral swelling" on computed tomography scan was 75% specific for the presence of intracranial hypertension Intracranial hypertension could be controlled in 14 of the 16 children whose pressure volume index was measured, and in those patients, there were no deaths

Table 1.—*Continued*

Reference	Study Description	Data Class, Quality, and Reasons	Results and Conclusion
New studies			
Adelson et al, 2005 (27)	Design: randomized controlled trial of hypothermia treatment N = 75 Age: <17 yrs Purpose: ICP monitoring and randomized, controlled trial of moderate hypothermia vs. normothermia plus medical management of intracranial hypertension Outcome: GOS at 3 and 6 months	Class III Poor quality: no control for confounders. (class II for hypothermia trial)	High (no. not specified) incidence of intracranial hypertension ICP >20 was most sensitive and specific for poor outcome Low mean ICP, percent time ICP <20 and mean CPP were all significantly associated with good outcome Mean ICP was lower in patients who had a good outcome versus those with a poor outcome (good, 11.9 mm Hg; poor, 24.9 mm Hg; p = .036) The percent time less than 20 mm Hg differed between outcome groups (good, 90.8% ± 10.8%; poor, 68.6% ± 35.0%; p = .01)
Cruz et al, 2002 (28)	Design: single-center retrospective study N= 45 Age: 1–12 yrs Purpose: assessment of the effect of ICP monitoring and medical therapy on outcome; also examined relationship to oxygen metabolism through jugular bulb catheter Outcome: GOS at 6 months	Class III Poor quality: no control for confounders	82% had favorable outcome, 17.8% unfavorable; 4.4% died; 13.3% had severe disability Higher ICP ($p \leq .02$) for days 1–5 was significantly associated with decreased cerebral oxygen extraction and worse clinical outcome
Grinkeviciute et al, 2008 (29)	Design: single-center prospective observational study N= 48 Age: 2.4 months to 18 yrs Purpose: examination of relationship between ICP, CPP, and outcome in children including 13 treated with decompressive craniectomy for medically refractory intracranial hypertension Outcome: GOS at 6 months	Class III Poor quality: no control for confounders	Survival rate was 97.9% (1 death); favorable outcome in 89.6% There was no difference in ICP maximum in groups with good (22.2 mm Hg) vs. poor (24.6 mm Hg) outcomes
Jagannathan et al, 2008 (30)	Design: single-center observational study N = 96 Age: 3–18 yrs Purpose: assessment of relationship between ICP, treatment and outcome in patients treated with variable combination of evacuation of mass lesions, ventricular drainage, medical management and decompressive craniectomy Outcome: GOS at 2 yrs	Class III Moderate quality: unclear if analysis of ICP monitoring controlled for confounders	Death was associated with refractory raised ICP (p = .0001), but not with ICP maximum, irrespective of the surgical or medical methods(s) used for successful reduction of intracranial hypertension Outcome: quality of life was related to medical management of elevated ICP (p = .04) Long-term outcomes were not correlated with peak ICP
Pfenninger and Santi, 2002 (31)	Design: retrospective single-center observational study N = 51 Age: 1 month to 16 yrs Purpose: assess relationship between ICP, medical or surgical management or jugular venous monitoring and outcome Outcome: GOS at 6–12 months	Class III Poor quality: no control for confounders	Moderate to severe intracranial hypertension (mean sustained ICP ≥20 mm Hg) was associated with poor outcome ($p < .05$) 69% of monitored patients had sustained ICP >20 mm Hg

Table 1.—*Continued*

Reference	Study Description	Data Class, Quality, and Reasons	Results and Conclusion
Wahlstrom et al, 2005 (32)	Design: single-center observational study N = 41 Age: 3 months to 14.2 yrs Purpose: assess affect of ICP management using the Lund protocol on outcome Outcome: GOS assessed between 2.5 and 26 months	Class III Poor quality: no control for confounders	Survival rate was 93%; favorable outcome (GOS 4 and 5) in 80% ICP in 3 nonsurvivors was significantly higher than in 38 survivors (mean 43 ± 26 mm Hg vs. 13 ± 4 mm Hg) The relationship between ICP and outcome in survivors was not statistically analyzed
White et al, 2001 (33)	Design: retrospective observational study N = 136 admitted to pediatric intensive care unit; 37 with ICP monitoring Age: 0–17 yrs Purpose: assess relationship between ICP and survival Outcome: survival	Class III Poor quality: no control for confounders for ICP analysis	14% of survivors and 41% of nonsurvivors had ICP >20 mm Hg in the first 72 hrs Those with lower mean ICP were more likely to be survivors ($p <$ .005) ICP maximum and ICP measured 6, 12, and 24 hrs after admission were all significantly lower in survivors

ICP, intracranial pressure; GOS, Glasgow Outcome Scale score; CPP, cerebral perfusion pressure; TBI, traumatic brain injury.

course (20). Intracranial hypertension (ICP >20 mm Hg) may also be significantly more prevalent in children with severe TBI who do not demonstrate spontaneous motor function (80%) than those who do (20%) (21).

These studies suggest that children presenting with severe TBI are at notable risk of intracranial hypertension. No specific markers have been identified that reliably determine the presence or absence of intracranial hypertension without monitoring in this population.

Are ICP Data Useful in Managing Pediatric Severe TBI?

Fifteen studies involving 857 pediatric patients demonstrated an association between intracranial hypertension (generally >20 mm Hg) and poor neurologic outcome or death (9, 19–28, 30–33).

One small study of 48 patients failed to demonstrate a clear association between intracranial hypertension and poor outcome (29). Specifically, a study by Grinkeviciute et al reported similar mean ICP in children with good and poor outcome. In their study, however, children with higher peak ICP were immediately and successfully treated with decompressive craniectomy.

These studies suggest that ICP is an important prognostic variable. It also plays a strong role both independently and as a component of cerebral perfusion pressure in directing the management of pediatric patients with severe TBI.

Does ICP Monitoring and Treatment Improve Outcome?

Two studies of combined treatment strategies also suggest that improved clinical outcomes are associated with successful control of intracranial hypertension (19, 30). A prospective observational study of 100 children with severe TBI treated with varying combinations of hyperventilation, diuretics, cerebrospinal fluid drainage, sedation, pharmacologic paralysis, and barbiturates reported that children whose intracranial hypertension was successfully lowered had better 1-yr outcomes than children whose intracranial hypertension was uncontrollable (but worse than those without intracranial hypertension) (19). A retrospective review of a prospectively acquired TBI database showed that reduced survival and worsened outcome in children with severe TBI were associated with intracranial hypertension refractory to treatment rather than peak ICP *per se* (30). In this study, successful control of intracranial hypertension, irrespective of treatment modality (osmolar therapy, cerebrospinal fluid drainage, decompression, etc.), was deemed to be important.

Although they represent only class III evidence for long-term outcome related to ICP monitoring and are only correlative, these studies support the association of successful ICP monitor-based management of intracranial hypertension with improved survival and neurologic outcome.

VI. INFORMATION FROM OTHER SOURCES

A. Indications From the Adult Guidelines

The adult guidelines offer the following recommendation.

> Level II: ICP should be monitored in all salvageable patients with a severe TBI (Glasgow Coma Scale score of 3–8 after resuscitation) and an abnormal CT scan. An abnormal CT scan of the head is one that reveals hematomas, contusions, swelling, herniation, or compressed basal cisterns.
>
> Level III: ICP monitoring is indicated in patients with severe TBI with a normal CT scan if two or more of the following features are noted at admission: age >40 yrs, unilateral or bilateral motor posturing, or systolic blood pressure <90 mm Hg.

What Patients Are at High Risk of ICP Elevation? Patients with severe TBI (Glasgow Coma Scale ≤8) are at high risk for intracranial hypertension (8, 34). The combination of severe TBI and an abnormal head CT scan suggests a high likelihood (53% to 63%) of raised

ICP (34). However, even with a normal admission CT scan, intracranial hypertension may be present (35, 36). Data collected predominantly in adult patients suggest that detection and treatment of intracranial hypertension may protect cerebral perfusion pressure, avoid cerebral herniation, and improve neurologic outcome (8, 11, 34, 37–39).

In certain conscious patients with CT findings suggesting risk of neurologic deterioration (hematomas, contusions, swelling, herniation, or compressed basal cisterns), however, monitoring may be considered based on the opinion of the treating physician (35, 38). Inability to perform serial neurologic examinations, because of pharmacologic sedation or anesthesia, may also influence a clinician's decision to monitor ICP in an individual patient (40, 41).

How Does ICP Data Influence Patient Management? ICP data allow the management of severe TBI by objective criteria. This is particularly important because many, perhaps all, medical and surgical measures for the treatment of intracranial hypertension have significant potential adverse consequences (2, 7, 42). Thus, ICP monitoring allows the judicious use of interventions such as hyperosmolar therapy, sedatives, neuromuscular blockade, barbiturates, ventilator management, etc., with a defined end point that is correlated with clinical outcome. This may avoid potentially harmful, overly aggressive treatment.

Does ICP Monitoring Improve Outcome? In adults, intensive management protocols for severe TBI, including ICP monitoring, have been associated with lowered mortality rates as compared with historical controls or centers in other countries not using monitoring techniques (8, 43–45). A study by Eisenberg et al (46) reported that improved ICP control was associated with improved outcome in severely head-injured patients with medically intractable intracranial hypertension. Finally, in a small, single-institution study of patients triaged according to the attending neurosurgery call schedule, mortality was over four times higher in nonmonitored than in monitored patients with severe TBI (47).

B. Information Not Included as Evidence

Various class III studies have demonstrated improved outcomes, vs. historical controls, in the era of ICP monitor-directed intensive therapy of patients with severe TBI (11, 35, 43, 48, 49). Two specific ICP monitor-directed therapies effective in treating acute intracranial hypertension have been associated with improved survival and clinical outcomes after severe TBI in children. As indicated in the evidence table, a study by Bruce et al (1) reported that aggressive therapy with hyperventilation and/or barbiturates to treat intracranial hypertension in 85 children with severe TBI resulted in 87.5% good outcomes and only 9% mortality. Not included as evidence, Peterson et al (50) performed a retrospective study of severe TBI in 68 infants and children, which showed that effective treatment of refractory intracranial hypertension using continuous infusion of hypertonic (3%) saline resulted in a mortality rate (15%) lower than expected as a result of trauma severity score (40%). There were only three deaths in this study (4%) resulting from uncontrolled intracranial hypertension.

VII. SUMMARY

Four lines of evidence support the use of ICP monitoring in children with severe TBI: a frequently reported high incidence of intracranial hypertension in children with severe TBI, a widely reported association of intracranial hypertension and poor neurologic outcome, the concordance of protocol-based intracranial hypertension therapy and best-reported clinical outcomes, and improved outcomes associated with successful ICP-lowering therapies. Evidence reviewed in the adult guidelines mirrors that for pediatric patients, further suggesting that ICP monitoring is of clinical benefit in patients with severe TBI.

Intracranial hypertension is both difficult to diagnose and is associated with poor neurologic outcomes and death in infants and young children. Intracranial hypertension may be present in children with open fontanelles and sutures (18). ICP monitoring is of significant use in these patient populations.

The presence of intracranial hypertension can also be influenced by the type of pathology on CT such as diffuse injury or specific etiologies such as traumatic sinus thrombosis.

By contrast, ICP monitoring is not routinely indicated in children with mild or moderate TBI. Treating physicians may, however, in some circumstances, choose to use ICP monitoring in conscious children who are at relative risk for neurologic deterioration as a result of the presence of traumatic mass lesions or in whom serial neurologic examination is precluded by sedation, neuromuscular blockade, or anesthesia.

VIII. KEY ISSUES FOR FUTURE INVESTIGATION

- Studies of specific subpopulations of pediatric patients with TBI in whom ICP monitoring is indicated; in particular, in the categories of infants and young children with abusive head trauma and/or infants with open fontanelles and sutures.
- Studies of the incidence of intracranial hypertension based on clinical and radiologic parameters in children of different ages and injury mechanisms.
- Focused multivariate analyses of children with intracranial hypertension to predict those who respond better to specific ICP-lowering therapies.
- Careful monitoring of the impact of adoption of ICP monitoring-directed protocols by hospitals and health systems should be undertaken to provide further evaluation of the impact of these measures on outcome as well as system performance variables.
- Studies are also needed to determine whether the type of ICP monitor (e.g., ventricular, parenchyma) or approach to monitoring (e.g., continuous or intermittent with cerebrospinal fluid drainage) influences outcome.

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