

NIH Public Access

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Pediatr Crit Care Med. Author manuscript; available in PMC 2008 May 2.

Published in final edited form as: *Pediatr Crit Care Med.* 2005 September ; 6(5): 537–541.

Frequency of intracranial pressure monitoring in infants and young toddlers with traumatic brain injury^{*}

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Abstract

Objective—To examine the use of intracranial pressure monitors and treatments for elevated intracranial pressure in brain-injured children of <2 yrs of age and compare them with the recently published management guidelines.

Design—Prospective, population-based study.

Setting—All pediatric intensive care units in the state of North Carolina.

Patients—All patients of <24 months of age admitted to a pediatric intensive care unit with a traumatic brain injury between January 2000 and December 2001.

Interventions-None.

Measurements and Main Results—Use of intracranial pressure monitoring devices and treatments for elevated intracranial pressure were measured. There were 136 children admitted to a pediatric intensive care unit with brain injury. A total of 54 (39.7%) had an admission Glasgow Coma Score of \leq 8, and 80% were infants. Thirty-three percent of children with a Glasgow Coma Score of \leq 8 received monitoring. Hyperosmolar therapy was the most frequently used treatment (57.1%). Treatment for elevated intracranial pressure was more common in, but not limited to, children with monitors. Logistic-regression modeling showed that children of \leq 12 months of age had an odds ratio of 0.2 (95% confidence interval, 0.1–0.6) of receiving a monitor compared with children aged 12–24 months.

Conclusions—Brain injury in young children may lead to many years of lost quality of life. The utility of monitoring intracranial pressure in infants has not been well established, which may be a reason for its low use. As most infants with traumatic brain injury survive, high-quality studies with neurodevelopmental measures as the primary outcome are urgently needed to document best practice in this subpopulation.

Keywords

traumatic brain injury; intracranial pressure; hyperosmolar therapy; seizures; inflicted head injury

LEARNING OBJECTIVES

On completion of this article, the reader should be able to:

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*See also p. 611.

- 1. Recall that children with severe traumatic brain injury who are younger than 1 year of age are less likely to undergo intracranial pressure monitoring than older children.
- **2.** Identify reasons for the considerable variability between institutions in the management of severe traumatic brain injury in pediatric patients.
- **3.** Recall that the use of corticosteroids was given a negative recommendation in the most recent Guidelines for the Management of Severe Traumatic Brain Injury in Infants, Children, and Adolescents.

All of the authors have disclosed that they have no relationships or interests in any commercial companies pertaining to this educational activity.

Wolters Kluwer Health has identified and resolved any faculty conflicts of interest regarding this educational activity.

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Recent evidence-based guidelines have been published on the management of elevated intracranial pressure (ICP) in the pediatric population (1). The guidelines were based on an exhaustive review of the pediatric literature and used implications from the adult literature when no pediatric literature existed. One of the most impressive findings of the review was the lack of class 1 evidence for ICP monitoring or management in this population. Many authors specifically commented on the lack of data for infants and, especially, on the lack of data for children with inflicted traumatic brain injury (TBI). Infants and children with inflicted injuries are often excluded from studies of management and outcome; however, in the <2-yr-old age group, approximately half of the cases of TBI admitted to the pediatric intensive care unit (PICU) are abusive injuries (1,2).

Infants are at higher risk of death from head injury than older children (3). Surviving children in earlier developmental stages may have poorer outcomes than those in later developmental stages. It was once thought that the plasticity of the infant brain would allow for improved outcomes, but there is now evidence that developing neurologic pathways may be at more risk than those that have already been established (4). Therefore, providing these very young patients with optimal management is of critical importance.

The primary goal of this study is to describe actual neurosurgical and intensive care interventions in all children ≤ 2 yrs of age who presented to any of the nine PICUs in North Carolina during a 2-yr period and compare that with the now published guidelines.

MATERIALS AND METHODS

The methods of this study have been previously described (2). Institutional review board approval was obtained at all institutions. All children of <24 months of age who experienced a TBI of sufficient severity to be admitted to a PICU or step-down unit in North Carolina between January 2000 and December 2001 were identified. There were nine PICUs in North Carolina that treated children with TBI during that time period. These units represented public teaching hospitals, public nonteaching hospitals, and private hospitals. Each of the nine units was called three times weekly to identify qualifying patients. Retrospective hospital admissions data were checked at each institution every 6 months during the study period to ensure that no child had been missed. To qualify for the study, children had to have evidence of intracranial pathology on radiographic studies, pathology, or operatively. All radiographs were reviewed by an independent pediatric radiologist to confirm the presence of a brain injury. Charts were

abstracted for demographic data and mechanism of injury. In addition, clinical data were abstracted from the medical record, including Glasgow Coma Scale (GCS) at admission and specific management modalities of elevated ICP in the PICU within 48 hrs of admission. Management items abstracted included the use of ICP monitors (intraventricular monitors and fiberoptic pressure monitors), use of hyperventilation, hyperosmolar therapy, high-dose barbiturates, active cooling, steroids, and craniectomy. The use of phenytoin for seizure prophylaxis was also recorded. The placement of ICP monitors was at surgical discretion. Results of ICP monitoring and maintenance of cerebral perfusion pressure were not recorded.

Definitions

Infants were defined as children ≤ 12 months of age. Hyperventilation was defined as a PCO₂ of ≤ 35 torr maintained for at least three sequential arterial blood gases. The medical records were reviewed to ensure that the therapies noted were intended for the management of ICP and were not the result of spontaneous hyperventilation by the patients. Mechanism of injury was divided into inflicted and noninflicted TBI. The determination of whether the injury was inflicted or noninflicted was made by the child abuse team at each participating hospital. In cases in which a determination had not been made, the authors reviewed the cases and made a determination using the method of Stier et al. (5) as previously described. Children were divided by GCS scores grouped into >8 and ≤ 8 to make comparisons with recommendations from the evidenced-based review. Barbiturate coma is defined as barbiturates given to burst suppression as noted on electroencephalographic monitoring for the control of intracranial hypertension or intractable seizures.

Analysis

Simple frequencies and percentages were used to display characteristics of the children and the types of therapies that they received. Risk ratios with 95% confidence intervals were generated to explore associations between types of ICP measurement and GCS score category. Additional risk ratios were generated to explore whether ICP management measures were related to the use of ICP monitoring. Finally, a logistic-regression model was used to examine predictors of ICP monitors. Covariates were excluded if they did not change the estimate by $\geq 10\%$.

RESULTS

There were a total of 138 children with TBI admitted to one of the nine PICUs or step-down units identified in the state of North Carolina during the study period. Of these 138 children, two did not have a GCS recorded and were excluded from this analysis. The characteristics of these children are shown in Table 1. Of the 136 children identified, 26 died after admission to the PICU, for an in-hospital case fatality rate of 19.1%. Fatality rates were similar between children with inflicted and noninflicted injuries (16.7% vs. 21.9%, p > .05). There were 109 infants (80.1%). Of the in-hospital fatalities, 16 (61.5%) had a PICU length of stay of ≤ 1 day. Approximately half of the children had inflicted TBI. Institutions ranged in number of TBI admissions from two cases (1.5%) to 31 cases (23%). Those institutions who admitted $\geq 10\%$ of cases (five of nine institutions) monitored between 4% and 44% of children admitted with TBI. The primary admitting service was most frequently the critical care service. If the diagnosis was inflicted TBI, critical care was the admitting service in 72% of cases, as those children generally did not get admitted via the trauma service. Children with inflicted TBI were more likely to have admitting diagnoses of apnea, seizures, or respiratory distress with no history of trauma than children with noninflicted TBI (6). When a surgical service was not the admitting service, there was documentation of a neurosurgical consultation in 84% of cases. Of the 11 cases in which there was no documented neurosurgical consultation, ten were

inflicted TBI cases, five had an admission GCS of 3, and the remainder had an admission GCS between 5 and 13.

A total of 25 children (18.4%) had their ICP measured using either an intraventricular catheter (n = 15) or a fiber-optic catheter (n = 18). Seven children had both types of monitors. Of the children with a GCS of \leq 8, 33.3% received a monitoring device, compared with 8.5% of children with a GCS of >8. The most frequent treatments after TBI were hyperosmolar therapy with mannitol (23.5%), hyperventilation for presumed or actual elevated ICP (10.3%), and active cooling (7.4%). Barbiturate coma was used in four children (2.9%), and steroids were used in six children (4.4%). The most infrequently used treatment was craniectomy for the control of ICP: no child had a craniectomy specifically for ICP control. There were 17 children with craniotomy for removal of subdural hematomas. The use of phenytoin for seizure prophylaxis was used in 41.9% of children.

When use of an ICP monitor and specific interventions were examined by grouped admission GCS, children with a GCS of ≤ 8 were more likely to have their ICP measured (risk ratio, 2.2; 95% confidence interval, 1.5–3.2) and were more likely to receive most therapies, including hyperosmolar therapy with mannitol, hyperventilation, and cooling (Table 2). The exceptions were barbiturate coma and phenytoin. Barbiturate coma was uncommonly used in both groups, whereas phenytoin was used frequently in both groups. All children placed into barbiturate coma had continuous electroencephalographic monitoring.

Seizures were frequent in this cohort of children with TBI and were diagnosed either clinically or by electroencephalographic monitoring. A total of 50 of the 136 children (36.8%) had seizures during their PICU stay. Children with inflicted injuries were more likely (risk ratio, 2.5; 95% confidence interval, 1.5–4.3) to have seizures than children with noninflicted TBI. Of those children who had seizures, 84.0% had seizures within the first 48 hrs after admission, and the remainder had their seizures after 48 hrs. There were 27 electroencephalographic reports in the medical records (ten showed diffuse slowing, seven showed seizures). Barbiturate coma was instituted for seizure control in one patient.

When therapies to reduce ICP were examined by whether the child had an ICP monitor in place, therapies were associated with the use of a monitor (Table 3). However, some children received specific therapies to reduce ICP with no monitor. Predictors of intracranial monitoring included age of >1 yr, GCS of \leq 8, and whether the child had ever required cardiopulmonary resuscitation (Table 4). In a logistic-regression model controlling for GCS, whether the child had ever received cardiopulmonary resuscitation, race (white or other), and injury type (inflicted vs. noninflicted), the odds for children \leq 1 yr of age of receiving an ICP monitor was 0.2 (95% confidence interval, 0.1–0.6) compared with children >1 yr. Intentionality of injury was not a strong predictor of ICP monitor use.

DISCUSSION

The guidelines for management of elevated ICP in children appeared in July 2003 (1), after the collection of these data. They discuss multiple treatment modalities and the class of evidence used to support or refute those treatments. Class 1 evidence represents randomized controlled trials. Class 2 evidence included prospective and retrospective cohort studies and case control studies based on reliable data. Class 3 evidence drew from clinical series, case reviews, and expert opinion. This population-based study from the state of North Carolina represents care given to young children for whom these guidelines would have applied and reflect actual treatment of young children across multiple settings.

The most striking finding from this study was the infrequent use of ICP monitors in children <1 yr of age compared with children >1 yr of age. In this cohort of children, ICP monitoring

occurred in only 33% of cases in which the GCS was ≤ 8 . If a child was ≤ 1 yr old, ICP was less frequently monitored than in children between 1 and 2 yrs of age. This lack of ICP monitor use was not secondary to the large proportion of children with inflicted TBI in the younger age group, in which there may have been concerns about delayed recognition of the injury or anoxic brain injury, as the decreased risk of having an ICP monitor placed remained after adjustment for injury type and receipt of cardiopulmonary resuscitation. Speculative reasons for lack of monitoring may have been that the children did not get admitted by the trauma service; therefore, neurosurgery was unaware of these children. However, 84% of the children had formal neurosurgical consultations documented in the medical record, making this less likely. It is possible that neurosurgeons thought that the fontanelle in infants serves as an adequate "pop-off" for elevated ICP in infants; however, infants do experience elevated ICP (7), and the reliability of predicting ICP by how tight the fontanelle feels to the surgeon has not been assessed. Computerized tomographs are also an inadequate judge of elevated ICP (8); therefore, the only reliable method of measuring ICP in this age group is with a monitoring device. Some children may not have received monitoring because their prognosis was thought to be very poor. Indeed, most children who died did so in the first 24 hrs after admission.

Recommendations on ICP monitoring state that there is insufficient evidence to support a treatment standard or guideline on ICP monitoring in infants and children. However, ICP monitoring is supported as an option (1). Evidence was strong enough to support a treatment guideline in adults (9). Infants are the most difficult patients from which to infer data from adult studies; however, it is difficult to understand why infants would be expected to tolerate elevated ICP better than children in the 1- to 2-yr-old age range.

Airway management typically included intubation in this group of patients: 94.1% of patients with a GCS of ≤ 8 were intubated at the scene or in the emergency department. According to the guidelines, very little literature exists on the relationship of hypoxia to outcome, although hypoxia has been related to poor outcome in several retrospective studies (9).

Recommendations on specific therapies for ICP management in infants and children exist largely at the level of recommendations. Hyperosmolar therapy with mannitol was the next most frequent treatment modality used in treatment of TBI. Approximately 57% of children with a GCS of \leq 8 received mannitol. It was used more frequently in children with an ICP monitor in place, but was also used in 13% of unmonitored children. Other treatment modalities used were hyperventilation, barbiturate coma, cooling, and steroids.

Of the ICP treatments used, steroid use is the only modality that has a negative recommendation in TBI (1). However, hyperventilation to a CO_2 of <35 torr is not recommended based on concerns of reduced cerebral blood flow and secondary brain injury (10,11). Although treatments for ICP were used more frequently in children with monitors, it is of some concern that ICP was treated in some children, exposing them to potential toxicity without any method of monitoring the outcome.

Phenytoin was the most frequently used medication in children after TBI. There was a minimal difference in its use between children with a presenting GCS of ≤ 8 vs. >8. It was unclear from the medical record whether the intent of the phenytoin administration was to prevent late seizures or early seizures. No hospital was consistent in its use of phenytoin (i.e., gave phenytoin to all children with a GCS of <8); therefore, it seems that its use was at the discretion of the treating practitioner. The finding that seizures are frequent, especially in children with inflicted injuries, suggests that adequate monitoring for seizures and seizure control should be a priority. This is especially of concern in children who are receiving neuromuscular blocking agents and children with a low motor GCS.

The limitations of this study are several. The data were collected from the hospital medical records; therefore, at times it was not clearly detailed whether an ICP monitor had been considered and why a specific patient received monitoring and another patient did not. Many items, such as achievement of cerebral perfusion pressure and use of neuromuscular blockading agents and sedatives, were not collected and cannot be compared with the guidelines. It was also unclear why some children received treatment for elevated ICP without a monitor. Because this was not a randomized study, and placement of monitors may have been related to the perceived outcome of the children, no statement can be made from this study about the utility of monitoring and treating elevated ICP in young children.

The strength of this study is that it reflects practice across a state for all severely injured children during a 2-yr time period. It also reflects the practice in all types of hospitals (academic and nonacademic), which increases its generalizability.

CONCLUSIONS

Brain injury in infants and young children may lead to many years of lost quality of life. This study shows that there was no consensus on the measurement or treatment of elevated ICP in infants after a TBI. Variations in monitoring and treatment occurred both between and within hospitals. Variability in treatment of elevated ICP in children has been noted previously in the United States and the UK (3,12). This variability of treatment can be partially explained by the lack of clear evidence for most modalities of treatment. Although it is fairly easy to use adult data when treating a 16-yr-old patient, it is much harder to bridge treatment guidelines from adults to infants. As the majority of infants who reach the PICU survive, it is important to give them the best quality of survival possible. Although clinical equipoise seems to exist in practice, with some clinicians pursuing aggressive monitoring and others not, it seems unlikely that a randomized controlled trial of placement of ICP monitors with goal-directed therapy in infants would be clinically acceptable because all institutions used ICP monitoring in some cases. Indeed, one retrospective case series of children, which included children from birth to 2 yrs of age, found that no child with a mean cerebral perfusion pressure of <40 mm Hg survived, and a mean ICP of >20 was a significant predictor of death (13). Although this study had some methodologic challenges, these data would suggest that a randomized controlled trial of ICP monitoring in infants is not clinically acceptable. However, a well-designed, multiple-center study of ICP management directed toward finding optimal cerebral perfusion pressure in infants with neurodevelopmental status as the primary outcome would be ethically feasible and have important practice implications.

Acknowledgements

Supported, in part, by grant R49/CCR402444 to the University of North Carolina Injury Prevention Research Center from the National Center for Injury Prevention and Control; H. T. Keenan is supported by a grant from the National Institute for Child Health and Human Development (K23 HD041040–01A2).

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Clinical characteristics of 136 children <2 years of age with traumatic brain injury

	n	%
Age		
≤1 yr	109	80.1
1–2 yrs	27	18.9
Race/ethnicity		
White	66	48.5
Other	70	51.5
Glasgow Coma Scale		
≦8	54	39.7
>8	82	60.3
Inflicted injury		
Yes	72	52.9
No	64	47.1
Intubated at scene or in emergency department		
Yes	67	49.3
No	66	48.5
Missing	3	2.2
Received cardiopulmonary resuscitation		
Yes	41	30.1
No	94	69.1
Unknown	1	0.7
Primary admitting service		
Critical care	70	51.5
Surgical service	63	46.3
Missing	3	2.2
Survive		
Yes	110	80.8
No	26	19.2
Days of mechanical ventilation $(n = 66)$		
Mean, 4.4 ± 5.1		
Median, 1.0		
Interquartile range, 1–7		
· · ·		

Risk of neurosurgical intervention or intracranial pressure management on intensive care unit admission based on Glasgow Coma Score (GCS) of ≤ 8

	GCS ≤8 n (%)	GCS >8 n (%)	RR (95% CI)
ICP measured			
Yes	18 (33.3)	7 (8.5)	2.2 (1.5-3.2)
No	36 (66.7)	75 (91.5)	
Ventriculostomy			
Yes	9 (16.7)	6 (7.4)	1.6 (1.0-2.6)
No	45 (83.3)	75 (92.6)	× /
Barbiturate coma			
Yes	2 (3.7)	2 (2.5)	1.3(0.5-3.5)
No	52 (96.3)	80 (97.5)	
Cooling			
Yes	8 (14.8)	12 (2.5)	2.2(1.5-3.2)
No	46 (85.2)	80 (97.5)	
Hyperventilation	()		
Yes	13 (24.1)	1 (1.2)	2.8(2.1-3.7)
No	41 (75 9)	81 (98.8)	
Intubation	11 (700)	01 (3010)	
Yes	48 (88.9)	19 (23.2)	8.3 (3.8-18.0)
No	6(111)	63 (76.8)	0.0 (0.0 - 0.0)
Mannitol	0 (1111)	00 (7010)	
Yes	28 (52.8)	4 (4 9)	36(25-52)
No	25(472)	78 (95.1)	010 (210 012)
Phenytoin	25 (17.2)	70 (33.1)	
Yes	26 (48 1)	31 (37.8)	13(09-20)
No	28 (51.9)	51 (62 2)	1.5 (0.9 2.0)
Steroids	28 (51.7)	51 (02.2)	
Vec	A(7A)	2 (2 5)	1.7(1.0-3.2)
No	50 (92.6)	80 (97 5)	1.7 (1.0-5.2)

RR, relative risk; CI, confidence interval; ICP, intracranial pressure.

Risk of management strategies designed to reduce elevated intracranial pressure based on whether an intracranial pressure monitor was in place (n = 25)

	Intracranial Pressure Monitor		
	Yes No. (%)	No No. (%)	RR (95% CI)
Barbiturate coma			
Yes	3 (12.0)	1 (0.9)	4.5 (2.3-8.9)
No	22 (88.0)	110 (99.1)	
Cooling			
Yes	9 (36.0)	1 (0.9)	7.1 (4.3–11.7)
No	16 (64.0)	110 (99.1)	
Hyperventilation			
Ŷes	9 (36.0)	5 (4.5)	4.9 (2.7-8.9)
No	16 (64.0)	106 (95.5)	
Mannitol			
Yes	18 (72.0)	14 (12.6)	7.8 (3.8–18.2)
No	7 (28.0)	97 (87.4)	
Phenytoin			
Yes	18 (72.0)	39 (35.1)	3.6 (1.6-8.0)
No	7 (28.0)	72 (64.9)	· · · · · ·
Steroids		. ,	
Yes	2 (8.0)	4 (3.6)	1.9 (0.6–6.2)
No	23 (92.0)	107 (96.4)	, (, ,

RR, relative risk; CI, confidence interval.

Predictors of intracranial pressure monitor use

	Intracranial Pressure Measured			
	Yes No. (%)	No No. (%)	RR (95% CI)	
Age, yrs				
≤1	15 (60.0)	94 (84.7)	0.4 (0.2–0.7)	
>1	10 (40.0)	17 (15.3)		
Received cardiopulmonary resuscitation				
Yes	13 (48.0)	28 (25.2)	2.5 (1.2-5.0)	
No	12 (52.0)	82 (73.9)		
Missing	0	1 (0.9)		
Race				
White	12 (52.0)	54 (48.6)	0.9(0.4-2.0)	
Other	13 (48.0)	57 (51.4)		
Injury type				
Inflicted	11 (44.0)	53 (47.7)	0.9(0.4-1.8)	
Noninflicted	14 (56.0)	58 (52.3)	· · · · ·	
Glasgow Coma Score of <8				
Yes	18 (72.0)	36 (32.4)	2.2(1.5-3.2)	
No	7 (28.0)	75 (67.6)		

RR, relative risk; CI, confidence interval.

Odds of receiving an intracranial pressure monitor if ≤ 1 yr compared with 1–2 yrs of age, controlling for if ever received cardiopulmonary resuscitation, race, and Glasgow Coma Score of ≥ 8 , is 0.2 (95% CI, 0.1–0.6). Adding inflicted injury into the model does not change the estimate by $\geq 10\%$.