

**HHS PUBLIC ACCESS**

Author manuscript

Pediatr Crit Care Med. Author manuscript; available in PMC 2017 November 01.

Published in final edited form as:

Pediatr Crit Care Med. 2016 November ; 17(11): 1064–1072. doi:10.1097/PCC.0000000000000937.

Intracranial Pressure Monitoring in Infants and Young Children with Traumatic Brain Injury

Rebecca R. Dixon, MD¹, Maryalice Nocera, RN, MSN², Adam J. Zolotor, MD, DrPH^{2,3}, and Heather T. Keenan, MDCM, PhD¹¹Pediatric Critical Care, University of Utah School of Medicine, Salt Lake City, UT²University of North Carolina Injury Prevention Research Center, University of North Carolina at Chapel Hill, Chapel Hill, NC³Department of Family Medicine, University of North Carolina School of Medicine, Chapel Hill, NC

Abstract

Objective—To examine the use of intracranial pressure monitors and treatment for elevated intracranial pressure in children less than or equal to 24 months with traumatic brain injury in North Carolina between April 2009 and March 2012, and compare this to a similar cohort recruited 2000 – 2001.

Design—Prospective, observational cohort study.

Setting—12 PICUs in North Carolina.

Patients—All children less than or equal to 24 months with traumatic brain injury, admitted to an included PICU

Interventions—None

Measurement and Main Results—Use of intracranial pressure monitors and treatments for elevated intracranial pressure were evaluated in 238 children with traumatic brain injury. Intracranial pressure monitoring (RR 3.7, 95% CI 1.5-9.3) and intracranial pressure therapies were more common in children with GCS less than or equal to 8 compared to GCS greater than 8. However, only 17% of children with GCS less than or equal to 8 received a monitoring device. Treatments for elevated intracranial pressure were more common in children with monitors; yet, some children without monitors received therapies traditionally used to lower intracranial pressure. Unadjusted predictors of monitoring were GCS less than or equal to 8, receipt of cardiopulmonary resuscitation, non-white race. Logistic-regression showed no strong predictors of intracranial pressure monitor use. Compared to the 2000 cohort, children in the 2010 cohort with GCS less than or equal to 8 were less likely to receive monitoring (RR 0.5, 95% CI 0.3-1.0), although the estimate was not precise, or intracranial pressure management therapies.

For Reprints: Rebecca R. Dixon, MD, Pediatric Critical Care, University of Utah School of Medicine, P.O. Box 581289, Salt Lake City, UT 84158-1289, Rebecca.Dixon@hsc.utah.edu, T 801.213.3723, F 801.581.8686.

This work was performed at The University of Utah School of Medicine, Salt Lake City, UT and The University of North Carolina School of Medicine, Chapel Hill, NC

Conclusion—Children in the 2010 cohort with a GCS less than or equal to 8 were less likely to receive an intracranial pressure monitor or hyperosmolar therapy than children in the 2000 cohort; however, about 10% of children without monitors received therapies to decrease intracranial pressure. This suggests treatment heterogeneity in children less than or equal to 24 months with traumatic brain injury and a need for better evidence to support treatment recommendations for this group of children.

Keywords

pediatrics, traumatic brain injury; intracranial pressure; intracranial hypertension; brain edema; inflicted head injury

Introduction

Pediatric traumatic brain injury (TBI) is an important cause of pediatric morbidity and mortality in the United States. The Centers for Disease Control and Prevention (CDC) estimates that over half a million children under 15 years of age suffer TBI annually; approximately 35,000 of these children are hospitalized and over 2,000 die from brain injury-related causes (1). Children who suffer TBI at a young age are particularly vulnerable to injury because of the nature of the developing brain (2-5). Very young children who acquire significant brain injury have poor long-term outcomes (6, 7). The unique susceptibility of very young children to poor long-term social, emotional, and cognitive sequelae after severe TBI, as well as the lifelong care and education-related costs, suggest a need to improve acute care to this specific group of individuals acutely after TBI (8, 9).

The Guidelines for the Acute Medical Management of Severe Traumatic Brain Injury in Infants, Children, and Adolescents (Guidelines) were first released in 2003 (10) and updated in January 2012 (11). These guidelines were developed by an expert panel sponsored by the Brain Trauma Foundation, and were endorsed internationally. The Guidelines support a Level III recommendation that ICP monitors be considered in infants and children with severe TBI; level III recommendations are based on prospective or retrospective data that are purely observational (12). Despite this Guideline recommendation, studies have shown wide variation in ICP monitor placement, especially in young children (13-18).

The purpose of this paper is to describe neurosurgical and critical care interventions in a prospective cohort of children 24 months of age who were admitted to 12 North Carolina Pediatric Intensive Care Units (PICU) with serious or fatal traumatic brain injuries during a 36 month study period. In order to evaluate changes in clinical practice since guidelines for management of pediatric TBI were introduced in 2003, we compared neurosurgical and critical care interventions in this cohort with the same interventions in a previously described North Carolina cohort (16).

Methods

This study analyzes clinical data from a larger prospective cohort study performed in North Carolina that aimed to test a statewide educational intervention to prevent abusive head trauma. As part of the larger study, all children under two years of age admitted to a

pediatric intensive care unit (PICU) or step down unit in the state of North Carolina with a TBI were identified for a 36-month period, April 2009 through March 2012 (2010 cohort). As part of the larger study, cases of children born in North Carolina who were hospitalized in other states and children who died pre-hospital were identified. However, clinical data were obtained only from cases identified at all twelve PICUs in North Carolina. These PICUs represent teaching hospitals, private hospitals and public non-teaching hospitals. The University of North Carolina IRB and the IRB of each participating hospital approved this study.

In order to qualify for the study, children had to be less than or equal to 24 months of age and have a TBI with radiologic, pathologic, or intra-operative evidence of intracranial injury. Families were consented for collection of clinical data at each participating institution; then data were abstracted from the clinical record including injury, demographic and clinical data. In the event that we were unable to make contact with family before child was discharged, clinical data, outcome, and survival status was recorded without identifying data (DOB, county of residence, birth hospital name, dates of hospitalization). Clinical data included the pediatric Glasgow Coma Score (GCS), pre-hospital events such as the need for CPR, specific medical treatments such as anticonvulsants for seizure prophylaxis, and surgical treatments that occurred in the PICU. Intracranial pressure management was recorded including placement of an ICP monitor or ventriculostomy, use of hyperosmolar therapies, high dose barbiturates, active cooling, hyperventilation, and steroid use. Some therapies reported in this study are not recommended in the Guidelines for ICP management (i.e. hyperventilation and steroids). They are reported here to describe variation in ICP management in this cohort, and to allow the evaluation of changes in clinical practice since guidelines for pediatric TBI were introduced. This study was observational; thus, all patient management decisions were made at the discretion of the surgical and intensive care teams at each hospital. Finally, the child's Pediatric Outcome Performance Category (POPC) at hospital discharge and disposition were recorded. These methods replicated a prior 24 month prospective cohort study of children in North Carolina under 2 years of age with TBI performed from 2000 to 2001 (2000 cohort) (16).

Definitions

Initial GCS score was defined as the first available score after emergency department (ED) resuscitation; in some cases this GCS was obtained in the ED, in other cases, this initial GCS was obtained on admission to the pediatric intensive care unit. Severe TBI was defined as GCS \leq 8. Injuries were classified as abusive or non-abusive by the treating clinicians at each hospital and reviewed by study investigators for consistency. Intracranial pressure monitors included both intraventricular monitors and fiberoptic monitors. If an intraventricular device was used to drain fluid only and no pressure was recorded, it was not counted as a pressure monitor because it was not used to direct ICP management. Hyperventilation was defined as a PCO₂ \leq 35 for at least six consecutive hours. Hyperosmolar therapy included the use of mannitol or 3% saline. Barbiturate coma was defined as barbiturates given to burst suppression as noted on electroencephalographic recordings or given for the control of ICP or intractable seizures. Steroid use was defined as steroid use for the treatment of TBI and not for the treatment of stridor or solely peri-

extubation. Academic centers were defined as centers with a campus co-located with a medical school.

Statistical Analysis

Simple frequencies and percentages were used to describe characteristics of the children, their injuries, and their outcomes. Risk ratios with 95% confidence intervals were calculated using chi squared analysis to evaluate associations between ICP management and GCS score category (GCS ≤ 8 vs. GCS >8), between ICP management and ICP monitoring, and between ICP monitor use and demographic and clinical covariates. Risk ratios with 95% confidence intervals were calculated using chi square analysis to compare the proportion of children who received ICP management in the 2010 cohort compared to those who received ICP management in the 2000 cohort.(16) A logistic regression model was used to calculate the adjusted odds of ICP monitoring and ICP management among children with GCS ≤ 8 in the 2010 cohort. Statistical analyses were performed using STATA, version 13.0 (StataCorp, College Station, TX).

Results

There were a total of 270 children younger than 2 years of age with TBI admitted to one of the twelve PICUs in the state of North Carolina during the 36-month study period. Of these 270 children, 22 died, for a fatality rate of 8.1%. Clinical data were available for 238 of the 270 (88%) children. Characteristics of the 238 children included in the cohort are shown in Table 1. Most children were ≤ 1 year of age (80.7%) and male (58.4%). More than half of the children were admitted with abusive head trauma (55.5%) (Supplemental Table 1). Mechanisms of injury in the remainder of the children included: unintentional drop or fall (62.7%), motor vehicle collision (31.3%), and other mechanisms (5.9%), such as unintentionally hit by a softball or “tornado struck home.” A quarter of the children had an initial Glasgow Coma Score ≤ 8 . Ninety-nine children (41.6%) were intubated during their hospital stay (median days of mechanical ventilation: 2, Interquartile Range 1-7). Thirty-six children (15.1%) received cardiopulmonary resuscitation, either at the scene or during hospitalization. Eighteen children died after admission to the PICU, all of whom had severe TBI (GCS ≤ 8) and six of whom had an ICP monitor. The overall in-hospital case fatality rate was 7.6% (18/238). Of the 132 children with abusive head trauma, 42 (32%) had severe TBI; and, of the children with severe TBI and abusive head trauma 11 (28%) died (Supplemental Table 1). In hospital fatality rates were similar between children with abusive and non-abusive injuries (8.3% vs. 6.9%, $p = 0.68$). Of the in-hospital fatalities, 13 (72.2%) had a PICU length of stay ≤ 2 days. Of the 220 children who survived to hospital discharge, 27 (12.3%) had a hospital discharge outcome of “Severe” as rated by the *Pediatric Overall Performance Category* (POPC) and 28 (12.7%) children were discharged to inpatient rehabilitation facilities. Twenty-six children had initial GCS missing. Of the 26, three had ICP monitors and a 24-hour GCS indicating a moderate or severe TBI. The remaining 23 did not have ICP monitors. Of these 23 children, 15 had mild injuries indicated by a documented 24-hour GCS of 15 ($n = 3$) or a PICU LOS ≤ 48 hours with no mechanical ventilation ($n = 12$). The remaining 8 children had no available GCS and were either intubated or had PICU

length of stay >48 hours. None of these 8 children received therapies typically used to control elevated ICP.

Medical therapies and neurosurgical interventions for the 212 children with documented initial GCS are shown in Table 2. The most frequently used treatment for intracranial pressure management was hyperosmolar therapy with hypertonic saline or mannitol (15.5%). When the likelihood of medical and surgical intervention was examined based on dichotomized admission GCS, children with GCS of ≤ 8 were more likely to have their ICP measured (risk ratio: 3.7; 95% confidence interval, 1.5-9.3) and were more likely to be intubated (risk ratio: 4.2; 95% confidence interval, 3.2-5.7). Additionally, children with GCS ≤ 8 were more likely to receive most therapies used to manage ICP, including active cooling, hyperventilation, intubation, and hyperosmolar therapy. The mean PICU length of stay for children with GCS ≤ 8 was 11.9 days (standard deviation 25.7), range 1-181 days. If the three children with the longest PICU length of stay (46 days, 77 days, and 181 days) are removed from this evaluation, the PICU length of stay for children with GCS ≤ 8 was 7.1 days (standard deviation 7.3), range 1-33 days. The median duration of mechanical ventilation was 2 (interquartile range 2,9). Although the likelihood of ICP monitoring was greater for children with GCS ≤ 8 , only 17% of children with severe TBI received a monitoring device. Of the 7 children with GCS >8 who received ICP monitors, 6 had moderate TBI (GCS 9-12) and 1 had mild TBI (GCS 13-15). Similarly, although children with GCS ≤ 8 were more likely to receive ICP management, children with GCS >8 did receive all such therapies.

When management strategies to reduce elevated ICP were examined based on whether an ICP monitor was present, all therapies were associated with monitor use (Table 3). However, some children received therapies typically used to reduce ICP with no monitor in place. In fact, 51.5% of children who received hyperosmolar therapy did not have an ICP monitor in place; similarly 42.9% of children who were put in a barbiturate coma, 28.6% of children who were cooled, and 25.0% of children who were hyperventilated did not have a monitor in place. When we examined therapies in aggregate, of the 40 children who received 1 or more therapies typically used to lower ICP (barbiturate coma, active cooling, hyperventilation, or hyperosmolar therapy), 22 (58%) did not have an ICP monitor in place.

Table 4 shows unadjusted predictors of ICP monitoring in this cohort. Unadjusted predictors included GCS ≤ 8 , whether the child had ever required cardiopulmonary resuscitation, being non-white (vs. white, non-Hispanic). Non-white children were slightly more likely to have a GCS ≤ 8 , though this difference did not reach the level of significance (35% vs 17% $p = 0.06$) (not shown in Table).

In a logistic-regression model controlling for covariates, there were no strong predictors of ICP monitor use, including age (OR 0.9, 95% CI 0.2-3.3), GCS less than 8 (OR 2.8, 95% CI 0.8-10.4), CPR (OR 2.6, 95% CI 0.8-9.3), race (OR 3.4, 95% CI 0.9-12.9), abusive head trauma versus unintentional TBI (OR 1.1, 95% CI 0.4-3.1), or being cared for at an academic center (OR 1.0, 95% CI 0.3-3.0).

Table 5 shows a comparison between the 2010 and 2000 cohorts with GCS ≥ 8 . (16) The two cohorts were comparable by age groups. The median age of the 2000 cohort was 6 months (IQR: 3, 13) similar to the 2010 cohort (median age 6 months, IQR: 4, 14). The proportion of children less than one year of age with GCS ≥ 8 was similar between the 2000 and 2010 cohorts: (74% versus 67.8%, respectively). Compared to the 2000 cohort, children in the 2010 cohort with a GCS ≥ 8 were less likely to have their ICP measured, although the estimate was not precise. Furthermore, children in this cohort were less likely to receive hyperosmolar therapy and less likely to be hyperventilated than children in the 2000 cohort. Children in the 2010 cohort were more likely to be intubated and to receive anticonvulsants.

Discussion

The most notable findings of this prospective observational cohort study are the overall decrease in the use of ICP monitors among children with severe TBI compared to one decade ago and the continued use of therapies typically used to control ICP without a monitor in place. These findings are particularly notable because The Guidelines for the Acute Medical Management of Severe Traumatic Brain Injury in Infants, Children, and Adolescents were released after conclusion of data collection for the 2000 cohort, and prior to the start of data collection for the current 2010 cohort. These practice guidelines support the use of ICP monitors in infants and young children with severe TBI and do not support the use of ICP related therapies in children without ICP monitors in place.

Observational studies have shown wide variability in the treatment of children with severe TBI (14). In the pediatric population, younger children receive ICP monitors less frequently than older children (15, 16, 19, 20), and in a study using National Trauma Data Bank data, infants were less likely to have an ICP monitor placed than children 1 – 5 years of age (18). Our results differ from previous reports, in that age > 1 year was not a predictor of ICP monitor placement in this cohort of children ≤ 24 months. However, the overall rate of ICP monitor placement for children with severe head injury in this cohort (17%) was similar to the rate of monitoring in infants < 1 year reported in previous studies (15-20%), and substantially less than the rate of monitoring in children ≥ 1 (30-59%) (15, 16, 20).

We suspect that the decrease in ICP monitoring in this cohort compared with the 2000 cohort reflects the uncertainty of applying recommendations meant for older children to infants, especially in light of the fact that 80.7% of this cohort was ≤ 1 year of age. The 2003 Guidelines, which were available for the entirety of care for the 2010 cohort, support a recommendation for ICP monitoring in infants and children with severe TBI (10). These recommendations are based on expert opinion; lack of controlled trials in pediatric TBI management likely influences practice variability, documented in both this study and others (15, 18, 19, 21). The treatment evidence for infants is even weaker than for older children. Infants differ from older children in pathophysiology following TBI and they may not respond to therapies similarly to them. There is extensive literature documenting infants' risk for increased ICP; diffuse cerebral edema, as opposed to focal injury; a higher incidence of seizures; and high risk for poor outcomes, when compared to older children and adults. This suggests that therapies specifically aimed at and trialed in infants are needed (3, 4, 22-30). However, we expected that the availability of published guidelines, despite the lack

of controlled trials, would have led to an increased use of ICP monitors since 2000. Our analyses suggest the opposite, and argue for considerable clinical equipoise in the management of severely brain-injured young children that may allow for clinical trials in this group.

The fact that 60 percent of children in the 2010 cohort who received one or more therapies traditionally used to decrease ICP (barbiturate coma, active cooling, hyperventilation, hyperosmolar therapy, or steroids) did not have an ICP monitor in place is striking; and, is consistent with previous reports in the pediatric literature (19, 31). The most recent Brain Trauma Foundation Guidelines support a Level II recommendation for use of hypertonic saline for treatment of severe pediatric TBI, but specify that its use should be determined by intracranial hypertension (32). In contrast with this recommendation, 51.5% of children in our cohort that received hyperosmolar therapy did so without an ICP monitor in place, suggesting that clinicians targeted a serum sodium or serum osmolality level without knowledge of intracranial pressure. Therefore, a therapy with potential adverse effects is administered without the ability to provide goal-directed ICP management. It is difficult to know whether this is a reasonable approach to severe TBI in infants or not. A recent randomized controlled trial in adults with severe TBI suggested that very close clinical management achieved similar results to ICP management using a monitor (33). This trial used a very clear protocol with careful clinical examination and frequent use of CT scanning. We do not know whether children who received ICP related therapies with no monitor in this cohort received the same level of clinical monitoring as was done in this adult study; however, CT scans in children are not as sensitive to intracranial pressure as adults (13). Use of hyperventilation for children with GCS ≤ 8 decreased over the past decade, a practice shift that is in keeping with the Guidelines, which recommend against sustained hyperventilation. Hyperosmolar therapy for children with GCS ≤ 8 also decreased over the past decade, despite the Guidelines' Level II recommendation for this therapy in the setting of intracranial hypertension. The decreased use of hyperosmolar therapy is coincident with decreased ICP monitoring since 2000. However, this shift in practice away from Guideline-recommended interventions indicates that young children with severe TBI are potentially being treated less aggressively from both surgical and medical standpoints.

This study has several limitations. The data were collected by hospital medical record abstraction; therefore, details of clinical decision-making are not available. We are unable to determine, for example, why some children received ICP monitors and others did not. Similarly, we are unable to determine why some children without ICP monitors received ICP-related medical therapy. Our dataset does not include dose frequency or duration of therapy; therefore, we are unable to determine whether children received a given therapy for several days or just one dose. This was a statewide study, with data collected at 12 different centers, but hospital identifying information is not included in the dataset; therefore, there may be hospital-level variation in care that we are unable to account for in our analysis. Although reflective of multiple hospitals (academic and non-academic), this study was conducted in a single large state; it is possible that medical practice in North Carolina is not generalizable to other areas of the United States.

The strengths of this study are that it includes a statewide cohort of all brain-injured children under 24 months of age in North Carolina during the 36-month study period; and, that it reflects practice at 12 different hospitals across the state, thus increasing its generalizability. Unlike studies that use large databases, this study was able to examine medical, surgical and pharmaceutical therapies as well as outcomes in this cohort.

In conclusion, infants and very young children appear to receive less aggressive therapy in North Carolina than they received 10 years ago. It is possible that this change in practice is due to the lack of evidence-based, infant-specific Guideline recommendations. This variability in practice may represent the clinical equipoise needed for trials of therapies, specifically in this young age group.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

Financial Disclosures: This study was funded by a grant to Dr. Zolotor from the Centers for Disease Control and Prevention, National Center for Injury Prevention and Control (5U49CE001275)

References

1. Coronado, VG., Faul, M., Wald, MM., et al. CDC Stacks | Traumatic brain injury in the United States; emergency department visits, hospitalizations, and deaths, 2002-2006 - 5571 | Library Collection. 2010.
2. Giza CC, Prins ML. Is being plastic fantastic? Mechanisms of altered plasticity after developmental traumatic brain injury. *Dev Neurosci*. 2006; 28(4-5):364–379. [PubMed: 16943660]
3. Anderson V, Spencer-Smith M, Leventer R, et al. Childhood brain insult: can age at insult help us predict outcome? *Brain*. 2009; 132(Pt 1):45–56. [PubMed: 19168454]
4. Anderson V, Catroppa C, Morse S, et al. Functional plasticity or vulnerability after early brain injury? *Pediatrics*. 2005; 116(6):1374–1382. [PubMed: 16322161]
5. Taylor HG, Alden J. Age-related differences in outcomes following childhood brain insults: an introduction and overview. *J Int Neuropsychol Soc*. 1997; 3(6):555–567. [PubMed: 9448369]
6. Ewing-Cobbs L, Prasad MR, Kramer L, et al. Late intellectual and academic outcomes following traumatic brain injury sustained during early childhood. *J Neurosurg*. 2006; 105(4 Suppl):287–296. [PubMed: 17328279]
7. Keenan HT, Hooper SR, Wetherington CE, et al. Neurodevelopmental consequences of early traumatic brain injury in 3-year-old children. *Pediatrics*. 2007; 119(3):e616–623. [PubMed: 17332181]
8. Shi J, Xiang H, Wheeler K, et al. Costs, mortality likelihood and outcomes of hospitalized US children with traumatic brain injuries. *Brain Inj*. 2009; 23(7):602–611. [PubMed: 19557562]
9. Zaloshnja E, Miller T, Langlois JA, et al. Prevalence of long-term disability from traumatic brain injury in the civilian population of the United States, 2005. *J Head Trauma Rehabil*. 2008; 23(6): 394–400. [PubMed: 19033832]
10. Adelson PD, Bratton SL, Carney NA, et al. Guidelines for the acute medical management of severe traumatic brain injury in infants, children, and adolescents. *Pediatr Crit Care Med*. 2003; 4:S1–S75. [PubMed: 12847336]
11. Kochanek PM, Carney N, Adelson PD, et al. Guidelines for the acute medical management of severe traumatic brain injury in infants, children, and adolescents-second edition. *Pediatr Crit Care Med*. 2012; 13(Suppl 1):S1–82. [PubMed: 22217782]

12. Kochanek PM, Carney N, Adelson PD. Guidelines for the acute medical management of severe traumatic brain injury in infants, children, and adolescents-second edition. Chapter 3: Indications for intracranial pressure monitoring. *Pediatr Crit Care Med.* 2012; 13(Suppl 1):S11–17.
13. Bailey BM, Liesemer K, Statler KD, et al. Monitoring and prediction of intracranial hypertension in pediatric traumatic brain injury: clinical factors and initial head computed tomography. *J Trauma Acute Care Surg.* 2012; 72(1):263–270. [PubMed: 21841509]
14. Bell MJ, Adelson PD, Hutchison JS, et al. Differences in Medical Therapy Goals for Children With Severe Traumatic Brain Injury—An International Study. *Pediatr Crit Care Med.* 2013; 14(8):811–818. [PubMed: 23863819]
15. Bennett TD, Riva-Cambrin J, Keenan HT, et al. Variation in intracranial pressure monitoring and outcomes in pediatric traumatic brain injury. *Arch Pediatr Adolesc Med.* 2012; 116(7):641–647.
16. Keenan HT, Nocera M, Bratton SL. Frequency of intracranial pressure monitoring in infants and young toddlers with traumatic brain injury. *Pediatr Crit Care Med.* 2005; 6(5):537–541. [PubMed: 16148812]
17. Tasker RC, Fleming TJ, Young AE, et al. Severe head injury in children: intensive care unit activity and mortality in England and Wales. *Br J Neurosurg.* 2011; 25(1):68–77. [PubMed: 21083365]
18. Van Cleve W, Cleve WV, Kernic MA, et al. National variability in intracranial pressure monitoring and craniotomy for children with moderate to severe traumatic brain injury. *Neurosurgery.* 2013; 73:746–52. [PubMed: 23863766]
19. Morris KP, Forsyth RJ, Parslow RC, et al. Intracranial pressure complicating severe traumatic brain injury in children: monitoring and management. *Intensive Care Med.* 2006; 32:1606–1612. [PubMed: 16874495]
20. Van Cleve W, Kernic MA, Ellenbogen RG, et al. National Variability in Intracranial Pressure Monitoring and Craniotomy for Children With Moderate to Severe Traumatic Brain Injury. *Neurosurgery.* 2013; 73(3):746–752. [PubMed: 23863766]
21. Alkhoury F, Kyriakides TC. Intracranial Pressure Monitoring in Children With Severe Traumatic Brain Injury : National Trauma Data Bank-Based Review of Outcomes. *JAMA Surg.* 2014; 149:544. [PubMed: 24789426]
22. Stocchetti N, Conte V, Ghisoni L, et al. Traumatic brain injury in pediatric patients. *Minerva Anesthesiol.* 2010; 76(12):1052–1059. [PubMed: 21178914]
23. Adelson PD. Pediatric trauma made simple. *Clin Neurosurg.* 2000; 47:319–335. [PubMed: 11197709]
24. Maxwell WL. Traumatic brain injury in the neonate, child and adolescent human: an overview of pathology. *Int J Dev Neurosci.* 2012; 30(3):167–183. [PubMed: 22212603]
25. Adelson PD, Kochanek PM. Head injury in children. *J Child Neurol.* 1998; 13(1):2–15. [PubMed: 9477242]
26. Kochanek PM, Clark RSB, Ruppel RA, et al. Biochemical, cellular, and molecular mechanisms in the evolution of secondary damage after severe traumatic brain injury in infants and children: Lessons learned from the bedside. *Pediatr Crit Care Med.* 2000; 1(1):4–19. [PubMed: 12813280]
27. Udomphorn Y, Armstead WM, Vavilala MS. Cerebral blood flow and autoregulation after pediatric traumatic brain injury. *Pediatr Neurol.* 2008; 38(4):225–234. [PubMed: 18358399]
28. Murphy S. Pediatric Neurocritical Care. *Neurotherapeutics.* 2012; 9(1):3–16. [PubMed: 22183817]
29. Chambers IR, Jones PA, Lo TYM, et al. Critical thresholds of intracranial pressure and cerebral perfusion pressure related to age in paediatric head injury. *J Neurol Neurosurg Psychiatry.* 2006; 77(2):234–240. [PubMed: 16103043]
30. Potts MB, Koh S-E, Whetstone WD, et al. Traumatic injury to the immature brain: inflammation, oxidative injury, and iron-mediated damage as potential therapeutic targets. *NeuroRx.* 2006; 3(2):143–153. [PubMed: 16554253]
31. Bennett TD, Statler KD, Korgenski EK. Osmolar therapy in pediatric traumatic brain injury. *Crit Care Med.* 2012; 40(1):208–215. [PubMed: 21926592]
32. Kochanek PM, Carney N, Adelson PD. Guidelines for the acute medical management of severe traumatic brain injury in infants, children, and adolescents-second edition. Chapter 8: Hyperosmolar therapy. *Pediatr Crit Care Med.* 2012; 13(Suppl 1):S36–41.

33. Chesnut RM, Temkin N, Carney N, et al. A trial of intracranial-pressure monitoring in traumatic brain injury. *N Engl J Med.* 2012; 367(26):2471–2481. [PubMed: 23234472]

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

Table 1

Demographic and clinical characteristics of 238 children < 2 years of age with traumatic brain injury (N = 238)

	n	%
Age		
1	192	80.7
1-2 years	46	19.3
Missing	0	0
Gender		
Male	139	58.4
Female	99	41.6
Missing	0	0
Race/Ethnicity		
White, non-Hispanic	108	45.4
Other	109	45.8
Missing	21	8.8
Initial Glasgow Coma Scale		
Mild (GCS 13)	129	54.2
Moderate (GCS 9-12)	24	10.1
Severe (GCS 8)	59	24.8
Missing	26*	10.9
Abusive Head Trauma		
Yes	132	55.5
No	102	42.9
Undetermined	4	1.7
Missing	0	0
Intubated during hospital stay		
Yes	99	41.6
No	138	58.0
Missing	1	0.42
Cardiopulmonary resuscitation at scene		
Yes	31	13.0
No	205	86.1
Missing	2	0.8
Cardiopulmonary resuscitation ever		
Yes	36	15.1
No	201	84.5
Missing	1	0.4
Initial unit admission		

	n	%
Critical care	219	92.0
Stepdown unit	12	5.0
Pediatric floor	7	2.9
Missing	0	0
Survived		
Yes	220	92.4
No	18	7.6
Missing	0	0
Outcome (Pediatric Overall Performance Category)		
Good	144	60.5
Mild disability	23	9.7
Moderate disability	22	9.2
Severe disability	27	11.3
Survived, unknown	4	1.7
Missing	0	0
Days of mechanical ventilation		
Median	2	
Interquartile range	1-7	
Disposition (n = 220 surviving children)		
Home	113	51.8
Relative	45	20.6
Foster care	31	14.2
Rehab	28	12.8
Friend of family	1	0.46
Missing	2	0.91

* Of the 26 children missing an initial GCS, 19 were never intubated and 1 was intubated briefly. Three were intubated and had a PICU length of stay of 4 days or longer; 3 had 24 hour GCS recorded suggestive of moderate or severe injury.

Table 2

Risk of neurosurgical intervention or intracranial pressure management on intensive care unit admission based on Glasgow Coma Score (GCS) ≥ 8 ; chi square analysis (N = 212) *

	GCS ≥ 8 n (%)	GCS < 8 n (%)	RR (95% CI) GCS ≥ 8 = referent
ICP measured			
Yes	10 (17.0)	7 (4.6)	3.7 (1.5-9.3)
No	49 (83.1)	146 (95.4)	
Missing	0	0	
Ventriculostomy			
Yes	7 (11.9)	11 (7.2)	1.6 (0.7-4.0)
No	52 (88.1)	141 (92.1)	
Missing	0	1 (0.65)	
Craniotomy			
Yes	6 (10.2)	10 (6.5)	1.5 (0.6-4.1)
No	53 (89.8)	142 (92.8)	
Missing	0	1 (0.65)	
Barbiturate coma			
Yes	4 (6.8)	3 (2.0)	3.4 (0.8-14.8)
No	55 (93.2)	148 (96.7)	
Missing	0	2 (1.31)	
Active Cooling			
Yes	5 (8.5)	2 (1.3)	6.4 (1.2-32.3)
No	54 (91.5)	150 (98.0)	
Missing	0	1 (0.65)	
Hyperventilation			
Yes	4 (6.8)	0	∞
No	55 (93.2)	152 (99.4)	
Missing	0	1 (0.65)	
Intubation			
Yes	58 (98.3)	35 (22.9)	4.2 (3.2-5.7)
No	1 (1.7)	117 (76.5)	
Missing	0	1 (0.65)	
Hyperosmolar therapy			
Yes	20 (33.9)	11 (7.2)	4.8 (2.4-9.3)
No	38 (64.4)	141 (92.2)	
Missing	1 (1.7)	1 (0.65)	
Steroids			
Yes	5 (8.5)	1 (0.65)	13.1 (1.6-109.8)
No	53 (89.8)	151 (98.7)	

	GCS = 8 n (%)	GCS > 8 n (%)	RR (95% CI) GCS > 8 = referent
Missing	1 (1.69)	1 (0.65)	

* Excludes children without an initial GCS recorded in the medical chart.

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

Table 3

Risk of management strategies to reduce elevated intracranial pressure based on whether an intracranial pressure (ICP) monitor was in place (N= 238)

	Intracranial Pressure Monitor		RR (95% CI) No ICP monitor = referent
	Yes No. (%)	No No. (%)	
Barbiturate coma			
Yes	4 (20.0)	3 (1.4)	14.4 (3.5-59.0)
No	16 (80.0)	213 (97.7)	
Missing	0	2 (0.92)	
Active Cooling			
Yes	5 (25.0)	2 (0.92)	27.1 (5.6-131.0)
No	15 (75.0)	215 (98.6)	
Missing	0	1 (0.46)	
Hyperventilation			
Yes	3 (15.0)	1 (0.46)	32.6 (3.5-298.6)
No	17 (7.3)	216 (92.7)	
Missing	0	1 (100.0)	
Hyperosmolar therapy			
Yes	16 (80.0)	17 (7.8)	10.2 (6.1-16.9)
No	4 (20.0)	199 (91.3)	
Missing	0	2 (0.92)	
Therapies in aggregate			7.3 (4.7-11.2)
1 or more therapies to reduce ICP	16 (80.0)	22 (10.1)	
Zero therapies to reduce ICP	4 (20.0)	196 (89.9)	

Table 4

Predictors of intracranial pressure (ICP) monitor use; chi square analysis (N = 238)

	Intracranial Pressure Measured		RR (95% CI) No ICP monitor = referent
	Yes No. (%)	No No. (%)	
Age			
1	15 (75.0)	177 (81.2)	0.9 (0.7-1.2)
> 1	5 (25.0)	41 (18.8)	
Missing	0	0	
Received CPR			
Yes	8 (40.0)	28 (12.8)	3.1 (1.6-5.9)
No	12 (60.0)	189 (86.7)	
Missing	0	1 (0.46)	
Race/Ethnicity			
White, non-Hispanic	5 (25.0)	103 (47.3)	1.5 (1.1-2.1)
Other	14 (70.0)	95 (43.6)	
Missing	1 (5.0)	20 (9.2)	
Injury type			
Abusive	13 (65.0)	119 (54.6)	1.2(0.8-1.6)
Non-abusive	7 (35.0)	95 (43.6)	
Undetermined	0	4 (1.8)	
GCS 8			
Yes	10 (50.0)	49 (22.5)	2.3 (1.5-3.7)
No	7 (35.0)	146 (67.0)	
Missing	3 (15.0)	23 (10.5)	
Hospital type			
Academic	13 (65.0)	126 (57.8)	1.1 (0.8-1.6)
Non-academic	7 (35.0)	92 (42.2)	
Missing	0	0	

Table 5

Likelihood of receiving an intracranial pressure directed therapy in 2010 cohort compared to 2000 cohort among children with GCS ≥ 8 , Referent = year 2000.

	2010 (n=60)	2000 (n=54)	RR (95% CI)
ICP measured			
Yes	10	18	0.5 (0.3-1.0)
No	49	36	
Ventriculostomy			
Yes	7	9	0.7 (0.3-1.8)
No	52	45	
Barbiturate Coma			
Yes	4	2	1.8 (0.3-9.6)
No	55	52	
Cooling			
Yes	5	8	0.6 (0.2-1.6)
No	54	46	
Hyperventilation *			
Yes	4	13	0.3 (0.1-0.8)
No	55	41	
Intubation			
Yes	58	48	1.1 (1.0-1.2)
No	1	6	
Hyperosmolar therapy			
Yes	20	28	0.7 (0.4-1.0)
No	38	25	
Steroids *			
Yes	5	4	1.2 (0.3-4.1)
No	53	50	

* Use of hyperventilation (except for episodes of herniation) and steroids are not recommended for controlling elevated intracranial pressure in the Guidelines.