

# The Incidence and Risk Factors for Hypotension During Emergent Decompressive Craniotomy in Children with Traumatic Brain Injury

Patrick Miller, MD\*

Christopher D. Mack, MS||

Marla Sammer, MD‡

Irene Rozet, MD\*

Lorri A. Lee, MD\*

Saipin Muangman, MD\*

Marjorie Wang, MD, MPH§

Will Hollingworth, PhD‡

Arthur M. Lam, MD, FRCPC\*§

Monica S. Vavilala, MD\*†§||

We conducted a retrospective cohort study in children <13 yr with traumatic brain injury (TBI) at a Level 1 pediatric trauma center to describe risk factors for intraoperative hypotension (IH) during emergent decompressive craniotomy. Between 1994 and 2004, 108 children underwent emergent decompressive craniotomy for TBI. Overall, 56 (52%) patients had IH. Independent risk factors for IH were each 10 mL estimated blood loss/kg (ARR 1.15 95% CI 1.08–1.22), each mm of computed tomography (CT) midline shift (ARR 1.04 95%CI 1.01–1.07), each 10 mL of CT lesion volume (ARR 1.03 95%CI 1.01–1.05), and emergency department (ED) hypotension (5/5 patients with ED hypotension had IH). CT midline shift  $\geq 4$  mm predicted IH (ARR 1.67 95% CI 1.06–2.63), independent of blood loss. IH occurred frequently during emergent decompressive craniotomy in children with TBI. ED hypotension, blood loss, CT lesion volume, and CT midline shift predicted IH. Anesthesiologists can expect children with preoperative CT midline shift  $\geq 4$  mm to have IH during this procedure.

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**H**ypotension after initial traumatic brain injury (TBI) may lead to secondary brain injury and worsen outcome (1–7). Patients with TBI, who undergo decompressive craniotomy, may be at particular risk of intraoperative hypotension (IH) due to blood loss, inadequate intravascular volume resuscitation, changes in sympathetic tone and/or general anesthesia. However, the incidence and risk factors for IH during this procedure in children with TBI are not well described.

There is only one study describing arterial blood pressure changes during decompressive craniotomy in children. In 1982, Uchida et al. (8) reported hypotension in children who underwent emergent evacuation of intracranial mass lesions between 1964 and 1976. Of the 36 children, aged 2 mo to 9 yr, 41% had

hypotension during emergent evacuation of subdural hematoma (SDH), subdural hygroma, or subdural effusion. Five patients had at least one episode of unmeasurable arterial blood pressure, and one child developed cardiac arrest. Of the risk factors considered, hypotension was associated with young age—infancy, in particular—and hematoma size larger than 8% of intravascular volume on head computed tomographs (CT). However, there were very few (5/36) children older than 5 yr of age, and non-SDH intracranial mass lesions were not considered. Additionally, hypotension attributable to blood loss was not considered, and intravascular volume calculation was based on theoretical age-related normative values rather than an estimate of intravascular volume at the time of surgery. Since publication of their study in *Critical Care Medicine* more than 20 yr ago, there has been no further characterization of the impact of decompressive craniotomy on arterial blood pressure in children. To provide more information on IH in children with traumatic intracranial mass lesions requiring neurosurgical intervention, we aimed to describe the incidence and the risk factors for IH during emergent pediatric decompressive craniotomy.

## METHODS

After IRB approval, a list of children <13 yr who underwent emergent decompressive craniotomy over

From the Departments of \*Anesthesiology, †Pediatrics, ‡Radiology, and §Neurological Surgery, University of Washington, Seattle, Washington; and ||Harborview Injury Prevention and Research Center, Seattle, Washington.

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Address correspondence and reprint requests to Monica S. Vavilala, MD, Department of Anesthesiology, Harborview Medical Center, 325 Ninth Avenue, Box 359724, Seattle, WA 98104. Address e-mail to vavilala@u.washington.edu.

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a 10-yr period between 1994 and 2004 at Harborview Medical Center (HMC; Level 1 pediatric trauma center covering the Pacific Northwest) was generated from the HMC anesthesia database. Anesthetic records with CPT codes reflecting evacuation of either, SDH, epidural hematoma (EDH), intraparenchymal hemorrhage (IPH), decompressive craniotomy or craniotomy were retrieved. Nonemergent cases were excluded. Medical records were reviewed for demographics, mechanism of injury, and for other potential contributors of IH.

### Anesthetic Data

Anesthetic records were reviewed for documentation of: 1) systolic blood pressure (SBP) and heart rate (HR), 2) anesthesia preparation time (anesthesia ready time minus anesthesia start time), 3) surgical time (surgery end time minus surgery start time), 4) estimated blood loss (EBL), 5) intraoperative volume (crystalloid, colloid, and blood product) administration, and 6) relevant surgical data such as surgical complications and opening of dura. Hypotension on arrival to the operating room, ASA status, intraoperative deaths, and all cause mortality, before hospital discharge, were also noted.

### Radiographic Data

The head CT corresponding to the time immediately preceding surgery was reviewed by the study radiologist (MS), who was blinded to all other patient information. The head CT source images were viewed on a General Electric Picture Archiving and Communication System (GE PACS). For patients imaged before this date, the radiology file room hard copy films electronic archiving system was searched for head CTs, and hard copy records were digitally scanned into PACS. Total blood volume on the CT examinations was determined by viewing the examination on the workstations in "blood brain windows." For each slice, the area of blood was measured using the "region of interest" tool. Then, the area of hemorrhage on each slice was summed, and blood volume was calculated by multiplying each area by slice thickness. This method uses volume calculation using parallel cross sections (a midpoint Riemann sum) (9). Finally, for each examination, the type of lesion was recorded as either EDH, SDH, subarachnoid, IPH, or mixed. We used the standard definition of midline shift (subfalcine herniation) on head CT as the largest perpendicular distance between an imaginary line between the frontal crest and to protuberantia occipitalis. Midline shift was measured on workstations, and measurements were made on the three consecutive slices that visually had the most herniation. The modal value of the three slices was recorded as amount of midline shift (mm).

### Definition of Hypotension

Hypotension, in the emergency department (ED) or operating room was defined as any SBP <5th percentile for age (Brain Trauma Foundation Guidelines Definition = 70 mm Hg + 2 (age in yrs); (10,11). IH was

defined as any hypotensive episode from first recorded SBP to last recorded SBP. Mean arterial blood pressure (MAP) was calculated as diastolic blood pressure + 1/3 pulse pressure.

### Outcomes

The main outcome measure was IH. Secondary outcomes included intraoperative IV vasopressor use (calcium, ephedrine, phenylephrine, and epinephrine), cardiopulmonary resuscitation and intraoperative mortality. We also examined all cause mortality before discharge from the hospital.

### Potential Predictors of Outcome

Relevant potential predictors included: 1) clinical characteristics (age, weight, gender, pupil asymmetry, admission motor Glasgow coma scale [GCS motor] score, presence of extracranial injuries, and diagnosis of inflicted TBI [iTBI]); 2) radiographic features (lesion type on CT of the head, CT lesion volume, and CT midline shift); and 3) ED and intraoperative factors: (a) IV volume (blood products, crystalloid or hypertonic solutions) administered, (b) ED SBP, (c) SBP, MAP, and HR on arrival to the operating room, (d) ED length of stay, (e) anesthesia preparation time, and (f) surgical time.

### Statistical Analysis

Descriptive statistics were used to examine demographics, mechanism of injury, potential predictors, and outcomes. Student's *t* tests and logistic regression were used to examine differences in continuous potential predictors between patients with and without IH.  $\chi^2$  tests and logistic regression were used to test for associations between potential predictors and the main outcome (IH) among binomial variables. Significant potential risk factors of IH were then entered into a bivariate modified Poisson regression model (12). Potential confounders or predictors were subsequently entered into a final multivariate model to determine the relative risk and 95% CI of IH, adjusted for gender (ARR 95% CI). CT lesion volume was analyzed in units of 10 mL of volume, EBL was analyzed in units of 10 mL of EBL/kg, and CT midline shift was analyzed per 1-mm shift. The amount of crystalloid was examined per 10 mL/kg. ED length of stay, anesthesia preparation time, and surgery time were examined as continuous variables. The cutoff point in determining the amount of CT midline shift that best predicted IH as a binomial variable was determined by examining the area under the curve (AUC) of a Receiver-Operating-Characteristic (ROC) analysis.

## RESULTS

### Demographics

One hundred and eight children ( $5.3 \pm 3.7$  yr), including 14 (13%) infants underwent emergent decompressive craniotomy (Table 1). All 108 anesthetic and medical records were available for review. The

**Table 1.** Preoperative Characteristics of 108 Patients who Underwent Emergent Decompressive Craniotomy Between 1994 and 2004

Age (yr)	5.3 ± 3.7
Age <3 yr (n %)	40 (37%)
Gender	65 M: 43 F
GCS motor (range)	3.6 ± 2.4 (1–6)
Pupil asymmetry (n %)	19/106 (18%)
ED hypotension (n %)	5 (5%)
ED LOS (min)	112 ± 83
Other injuries (n %)	12 (11%)
All cause mortality <sup>a</sup> (n %)	16 (15%)
MAP/HR on arrival to OR	0.76 ± 0.23 (0.28–1.55)
Hypotension on arrival to OR (n %)	0 (0%)

Data as mean ± sd (range).

GCS = glasgow coma scale score; ED = emergency department; LOS = length of stay; OR = operating room; MAP = mean arterial blood pressure; HR = heart rate.

<sup>a</sup> Mortality prior to hospital discharge.

**Table 2.** Intraoperative Characteristics of 108 Patients

IH (n %)	56 (52%)
Anesthesia preparation time (min)	27 ± 17 (0–100)
Surgery time (min)	122 ± 48 (23–240)
EBL/kg	12 ± 15 (0–85)
Crystalloid administered (mL/kg)	61 ± 47 (5–270)
pRBCs administered (mL/kg)	10 ± 20 (0–141)
Vasopressor use (n %)	32 (29%)
Intraoperative CPR (n %)	4 (4%)
Intraoperative mortality (n %)	1 (<1%)

Data are presented as mean ± sd (range).

IH = intraoperative hypotension; EBL/kg = estimated blood loss per kg; pRBCs = packed red blood cells; CPR = cardiopulmonary resuscitation.

mechanism of injury was motor vehicle crash (75%), falls (9%), iTBI (9%), and sports-related (7%; Table 1). The most common operative diagnoses were EDH (53%), SDH (27%), IPH (11%), Other (7%), and combined SDH/EDH (2%).

### Preoperative Clinical Characteristics

Five (5%) children had at least one episode of documented hypotension in the ED; all 5 had IH. Forty (37%) received IV mannitol in the ED. None of the 108 children were hypotensive on arrival to the operating room. The average GCS motor score was 4 ± 2 (range 1–6). Nineteen (18%) children had pupil asymmetry in the ED (Table 1). Eleven percent had other injuries; 88% were orthopedic.

**Table 3.** Clinical Characteristics of Children who Received Intraoperative Cardiopulmonary Resuscitation

Age (yr)/ Gender	Cause	OR mortality	All cause mortality <sup>a</sup>	CT lesion	CT midline shift (mm)	CT lesion volume (mL/kg)	ED Hypotension
0.6/M	PEA	no	no	SDH	7	9.5	no
2/F	Asystole	no	no	EDH SDH	n/a	n/a	no
2/M	PEA	no	no	SAH SDH	0	11.5	yes
2/M	Asystole	yes	yes	IPH	5	3.3	yes

n/a = missing data; OR = operating room; CT = computed tomography; ED = emergency department; IPH = intraparenchymal hemorrhage; SDH = subdural hematoma; EDH = epidural hematoma; SAH = subarachnoid hemorrhage; PEA = pulseless electrical activity.

<sup>a</sup> Mortality prior to discharge.

### Preoperative Radiographic Data

We were able to retrieve 98 (90%) the CT radiographs, either as hard copies or as digital images. The 10 missing films were from the earlier study years (1995–1996) when the CTs were produced as hard copies that could not be located. Fifty-four of the 98 available examinations were digitally scanned into PACS from hard copies (55%); the remaining 44 (45%) were found archived in PACS in their originally digitally acquired format. Midline shift ranged from 0 to 28 mm (4.97 ± 4.80 mm), and CT lesion volume ranged from 0.38 to 389.5 mL (46.0 ± 51.5 mL). Radiographic diagnoses matched operative diagnoses in all cases.

### Risk Factors for IH

Fifty six (52%) children had at least one episode of IH during decompressive craniotomy (Tables 2 and 3). Differences in clinical characteristics of patients with and without IH are detailed in Table 4. Seven bivariate factors were associated with IH (Table 5). Of the significant risk factors entered in the final model (CT midline shift, SDH, and blood loss), only CT midline shift and blood loss predicted IH. CT midline shift ≥4 mm predicted IH independent of blood loss (Table 6). ED hypotension was not entered in the final model because all 5 children with ED hypotension had IH.

### Anesthetic Factors and IH

Overall, children received three times as much crystalloid and an equal amount of packed red blood cells as the amount of blood lost during surgery. All children received <1 minimum alveolar concentration (MAC) of either isoflurane (56/108) or sevoflurane (52/108) with (80/108) or without (28/108) of IV fentanyl (1 mcg/kg boluses) and muscle relaxants (100/108). IH was not associated with choice of volatile anesthetic. All patients received 1 mg/kg IV mannitol before decompression in the operating room. Anesthesia preparation time was considered as an indicator of delayed vascular access, delay in fluid resuscitation and/or time to surgical decompression.

### Age and IH

There was no association between IH and age (RR 0.96; 95% CI 0.92–1.02) or IH and weight (RR 0.99; 95% CI 0.98–1.01; Fig. 1). Infancy did not predict IH.

**Table 4.** Clinical Characteristics of Patients With and Without Intraoperative Hypotension (IH)

	IH (n = 56)	No IH (n = 52)	P value
Extracranial injuries (%)	5/55 (9%)	7/51 (14%)	0.45
Glasgow coma scale score—motor	3.4 ± 2.4	3.8 ± 2.5	0.46
Pupil asymmetry (n %)	11/55 (20%)	8/51 (16%)	0.56
ED length of stay (min)	99 ± 81	123 ± 85	0.17
ED hypotension (%)	5 (0.1%)	0.0	<b>0.04</b>
Last ED hematocrit (%)	33.4 ± 5.5	35.3 ± 3.9	<b>0.04</b>
ED mannitol	17/51 (33%)	24/55 (44%)	0.28
Preoperative MAP/HR ratio (mm Hg/beats per minute)	0.76 ± 0.27	0.78 ± 0.20	0.74
CT lesion volume (mL)	59 ± 64	32 ± 27	<b>0.009</b>
CT midline shift (mm)	6.1 ± 5.3	3.8 ± 3.9	<b>0.015</b>
Lowest intraoperative SBP (mm Hg)	112 ± 17	121 ± 16	<b>0.009</b>
Subdural hematoma (n; %)	23/55 (42%)	11/51 (22%)	<b>0.03</b>
Anesthesia preparation time (min)	30 ± 17	25 ± 16	0.14
Surgery time (min)	106 ± 91	127 ± 92	0.25
EBL (mL/kg)	16 ± 19	7.8 ± 6.3	<b>0.004</b>
Crystalloid administered (mL/kg)	74 ± 58	45 ± 27	<b>0.002</b>
pRBCs administered (mL/kg)	15 ± 26	3 ± 06	<b>0.001</b>
Vasopressor use (n %)	36/55 (65%)	2/52 (4%)	<b>&lt;0.001</b>
Intraoperative CPR (n %)	4/54 (7%)	0	<b>0.06</b>
Intraoperative mortality (n %)	1/56 (2%)	0	0.52
All cause mortality <sup>a</sup> (n %)	11/55 (20%)	4/51 (8%)	0.07

Data are presented as mean ± sd.

CT = computed tomography; SBP = systolic blood pressure; EBL = estimated blood loss; Kg = kilogram; CPR = cardiopulmonary resuscitation; ED = emergency department; MAP/HR = mean arterial blood pressure to heart rate; pRBC = packed red blood cells.

<sup>a</sup> Mortality prior to discharge.

Bolded values indicate statistical significance.

However, 50% of the 32 children who received vasopressors and all 4 children who received intraoperative cardiopulmonary resuscitation were <3 yr of age; one 2-yr-old male (0.09%) died in the operating room (Table 3).

### iTBI and IH

Ten children (3 boys, 7 girls; 3 mo to 10 yr; mean 3.3 ± 3.2 yr) had iTBI. Four (40%) patients had pupil asymmetry in the ED. Six (60%) had midline shift on

CT and seven (70%) received IV mannitol in the ED. All 10 children had GCS <8 (6.9 ± 5.2; GCS motor 3 ± 2). None had extracranial injuries. The most common CT lesion was intracranial hemorrhage (50%), followed by SDH (40%), and EDH (10%). Sixty percent (6/10) had IH but none of these patients received intraoperative cardiopulmonary resuscitation. However, 3 (30%) children died before discharge from the hospital.

### DISCUSSION

The main findings of this study are that during emergent decompressive craniotomy for TBI: 1) IH occurred in more than 50% of children, 2) CT lesion volume, CT midline shift, blood loss and ED hypotension were independent risk factors for IH, and 3) CT midline shift ≥4 mm predicted IH independent of blood loss. We also found that 10% of children who presented for surgery were victims of iTBI. These data provide new information regarding the incidence and

**Table 5.** Bivariate Risk Factors for the Development of Intraoperative Hypotension (IH)

	ARR (95% CI)
pRBC administered (10 mL/kg)	<b>1.11 (1.05–1.17)</b>
CT midline shift (mm)	<b>1.04 (1.01–1.07)</b>
CT lesion volume (per 10 mL)	<b>1.03 (1.01–1.05)</b>
Subdural hematoma	<b>1.6 (1.1–2.3)</b>
ED hypotension	<b>Not calculable<sup>a</sup></b>
EBL (10 cc/kg)	<b>1.15 (1.09–1.21)</b>
Crystalloid administered (10 cc/kg)	1.05 (1.03–1.07)
Pupil asymmetry	1.2 (0.8–1.8)
Glasgow coma scale—motor	1.0 (0.9–1.0)
ED length of stay	1.0 (1.0–1.0)
Anesthesia preparation time	1.0 (1.0–1.0)
Surgery time	1.0 (1.0–1.0)
Extracranial injuries	0.8 (0.4–1.6)
Last ED hematocrit	0.8 (0.4–1.6)
Preoperative MAP/HR ratio (mm Hg/beats per min)	0.9 (0.4–2.0)

Rate Ratios 95% CI.

CT = computed tomography; ED = emergency department; pRBC = packed red blood cells; EBL = estimated blood loss; MAP = mean arterial blood pressure; HR = heart rate.

<sup>a</sup> All five children with hypotension in the Emergency Department (ED) had IH.

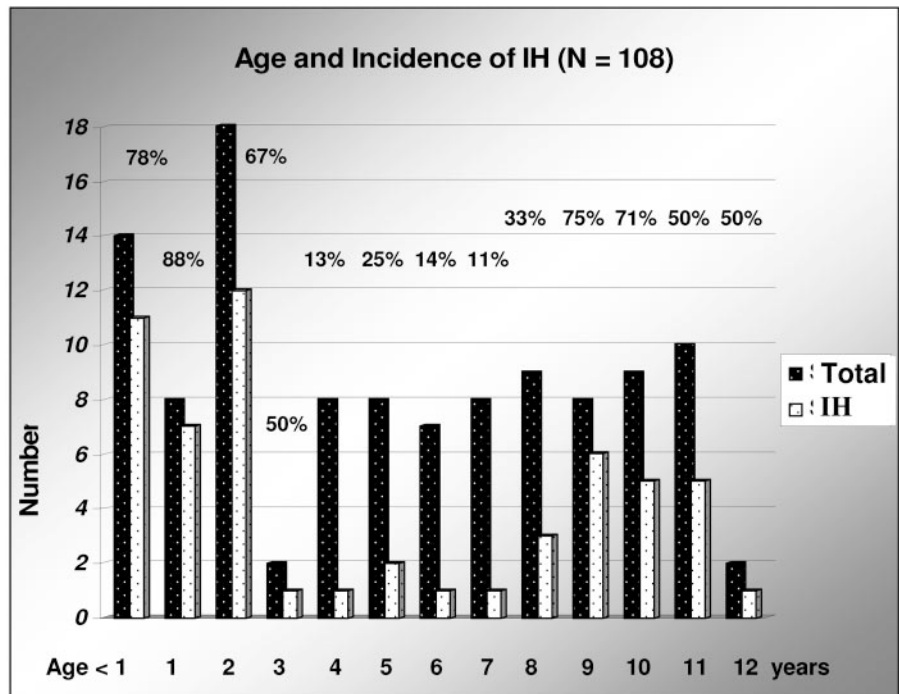
Bolded values indicate statistical significance.

**Table 6.** Risk Factors for Intraoperative Hypotension (IH) During Emergent Pediatric Decompressive Craniotomy: Final Multivariate Poisson Regression Adjusted for Gender

	ARR (95% CI)
CT midline shift ≥4 mm	1.67 (1.06–2.63)
Estimated blood loss in operating room (10 mL/kg)	1.15 (1.08–1.22)

Hypotension in the Emergency Department (ED) was not entered in the final model because all five ED patients with hypotension had IH.

Crude RR for IH and computed tomography (CT) midline shift ≥4 mm = 1.04 (95% CI 1.01–1.07).



**Figure 1.** Age distribution and incidence of intraoperative hypotension (IH) in 108 patients who underwent emergent pediatric decompressive craniotomy between 1994 and 2004. Percentages refer to percentage of IH by age.

risk factors associated with IH during emergent decompressive craniotomy in children with TBI.

Traumatic intracranial hematomas may be associated with a poor neurological outcome, and adult data suggest that cerebral ischemia occurring during evacuation of these lesions may be attributed to hypotension (13–15). Efforts to prevent hypotension in these patients are, therefore, of paramount importance. However, these data show that the incidence of IH in children is high (52%) during emergent decompressive craniotomy. Some possible reasons include: 1) risk factors for IH during this procedure have not been sufficiently examined, 2) there is a lack of awareness and anticipation of the high incidence of IH in children during this procedure, 3) difficult vascular access may hinder timely intravascular volume resuscitation, 4) clinicians may simply tolerate lower blood pressure in children compared to adults, 5) children with IH may be refractory to treatment and/or 6) IH with dural opening may be difficult to reliably prevent despite adequate intravascular fluid administration because of vasoparalysis that may accompany decompression. Additionally, this procedure is uncommon, even at Level 1 pediatric trauma centers (10.8 cases/yr at HMC). In fact, the incidence of IH during decompressive craniotomy may be more frequent than what we report because unlike Uchida et al. (8) who defined hypotension as SBP 20% below normal for age, we defined hypotension more narrowly, as SBP <5th percentile for age, according to the Brain Trauma Foundation Guidelines published in 2003 (10). This means that severe IH occurs in more than half of the children undergoing this procedure.

Consistent with some earlier findings (8,16), we found that ED hypotension, blood loss (15% per 10 mL

EBL/kg), CT lesion volume (3% per mL) and CT midline shift (4% per mm) were independent risk factors for IH. ED hypotension appears to be a predictor of IH, but the small number of patients and the findings in this group precluded obtaining formal estimates. One possible reason for the association between these factors and IH includes hypovolemia, where blood in the cranial vault reflects depletion of the same amount of blood from the systemic circulation. Crystalloid and packed red blood cells administration were associated with IH, probably because children who developed IH received more intravascular volume resuscitation. However, hypovolemia may not be the only causative factor of IH for two reasons: 1) 51/56 patients who developed IH did not have ED hypotension, and 2) our clinical observation is supported by findings that CT midline shift predicted IH independent of blood loss.

An alternate explanation for the development of IH during this procedure may be a sudden decrease in sympathetic tone (cessation of Cushing's response) when intracranial pressure (ICP) is relieved during dural opening. Four studies suggest that this reason, rather than hypovolemia, might explain IH during this particular procedure (16–18)<sup>1</sup>. In 1996, Kawaguchi et al. (16) reported that patients with acute SDH and at 17 mm CT midline shift had a decrease in MAP >20% from baseline during decompression. Our data show that even less of a midline shift ( $\geq 4$  mm) may be sufficient to cause IH in children. In 2004, Kinoshita et al. (17) reported preoperative hypertension (MAP

<sup>1</sup>Lee LA, Vavilala MS, Visco E, et al. Evaluation of a Head Injury Decompression Score (HIDS) to predict intraoperative hypotension during decompressive craniotomy. *Anesthesiology* 2002;A-260.

>131 mm Hg) in adults with IH. Meyer et al. (18) reported increased preoperative pulsatility and resistance indices by transcranial Doppler ultrasonography (suggestive of increased ICP) in 26 children with acute SDH. Finally, in 2002, Lee et al.<sup>1</sup> reported a scoring system to predict IH based on CT midline shift, papillary asymmetry, SDH, and MAP/HR ratio of either <0.8 (hypovolemia) or >2.5 (increased ICP). However, deriving MAP/HR ratios to differentiate hypovolemia from increased ICP in children is problematic because arterial blood pressure and HR change in opposite directions with age. Therefore, similar to adults, CT midline shift ( $\geq 4$  mm) probably reflects the presence of underlying high ICP and high preoperative sympathetic tone and is a clinically useful preoperative predictor for IH during this procedure.

In our experience, IH occurs coincident with dural opening. However, in this study, the timing of dural opening was not recorded. Thus it is not possible to comment on the relative contribution of hypovolemia versus changes in sympathetic tone on the development of hypotension in each patient. Regardless of the etiology of IH (sympathetic tone versus hypovolemia), however, preventing IH ultimately lies in correcting intravascular volume deficits, before, and in support of sympathetic tone, during, neurosurgical decompression (19).

We decided to select either CT midline shift or CT lesion volume in the final regression model because they are correlated variables and were equally good predictors of IH. We selected CT midline shift primarily because it is easier for anesthesiologists to measure CT midline shift than it is to calculate CT lesion volume. Additionally, midline shift information is frequently available in the medical record, whereas CT lesion volume size calculations are not routinely performed by radiologists in real time. Moreover, CT lesion volume may not include measurement of surrounding cerebral edema, making CT midline shift a more desirable marker of IH. Finally, we selected CT midline shift  $\geq 4$  mm as the cutoff because it had the largest AUC of all the cutoffs examined.

We examined IH by age groups based on our clinical impression that young children are at particular risk of IH; but, unexpectedly, the present data do not support our hypothesis. In this study, while children <3 yr of age definitely had the highest risk of IH, IH also occurred in 58% of older children who were matched for a variety of relevant clinical factors (8). On the other hand, all four cases of cardiopulmonary resuscitation occurred in children <3 yr of age, suggesting that perhaps life-threatening hypotension occurs more often in younger children, perhaps due to a less well developed sympathetic nervous system. Importantly, the present data show that anesthesiologists can expect IH in all children with TBI undergoing this procedure; even in older children typically considered to pose less of a challenge with regard to vascular

access and the ability to resuscitate. Despite its association with poor outcome (20,21), iTBI was not associated with IH, suggesting that the other proximate predictors examined in this study were more important in the development of IH than the preexisting factors associated with iTBI.

The major limitation of this study is our reliance on a medical record not designed for data collection. However, given the relatively low frequency of these procedures, ours was the most efficient study design. We searched for, but did not find, interaction between CT lesion volume and midline shift when an interaction term was entered in the model, either because of the small sample size or, alternatively, because midline shift can occur due to factors independent of CT lesion volume. Although all patients had indwelling arterial cannulation for these procedures, few had central venous lines placed; we did not have recorded continuous arterial blood pressure or central venous pressure data in a sufficient number of subjects for analysis. We cannot comment on difficulty with vascular access and its effect on resuscitation quality. We also cannot guarantee that all cases of IH were captured, or that we considered all potential confounding factors. We could not determine the relative contribution of hypovolemia versus increased sympathetic tone on the development of IH in each patient. Finally, we could only examine short-term surrogate outcomes such as IH. Despite these limitations, our data provide anesthesiologists with clinically useful information regarding risk factors for IH.

This is the largest systematic characterization of hypotension during emergent decompressive craniotomy in children with TBI. Results of this study indicate that children of all ages are at risk of IH and that the presence of preoperative CT midline shift  $\geq 4$  mm predicts IH during this procedure. The relationship between CT midline shift and IH, independent of blood loss, advocates for the role of altered sympathetic tone in the development of IH. Finally, our data propose a potential role for IH as a contributor for secondary injury after TBI.

## REFERENCES

1. Fearnside MR, Cook RJ, McDougall P, McNeil RJ. The Westmead Head Injury Project outcome in severe head injury. A comparative analysis of pre-hospital, clinical and CT variables. *Br J Neurosurg* 1993;7:267-79.
2. Chesnut RM, Marshall LF, Klauber MR. The role of secondary brain injury in determining outcome from severe head injury. *J Trauma* 1993;34:216-22.
3. Chesnut RM. Avoidance of hypotension: *conditio sine qua non* of successful severe head injury management. *J Trauma* 1997;42: S4-S9.
4. Kokoska ER, Smith GS, Pittman T, et al. Early hypotension worsens neurological outcome in pediatric patients with moderately severe head trauma. *J Pediatr Surg* 1998;33:333-8.
5. Pigula FA, Wald SL, Shackford SR, Vane DW. The effect of hypotension and hypoxia on children with severe head injuries. *J Pediatr Surg* 1993;28:310-14.
6. Vavilala MS, Bowen A, Lam AM, et al. Blood pressure and outcome after severe pediatric traumatic brain injury. *J Trauma* 2003;55:1039-44.

7. Coates BM, Vavilala MS, Mack CD, et al. Influence of definition and location of hypotension on outcome following severe pediatric traumatic brain injury. *Crit Care Med* 2005;33:2711-2712.
8. Uchida M, Yamaoka H, Imanishi Y. Hypotension during surgery for subdural hematoma and effusion in infants. *Crit Care Med* 1982;10:5-9.
9. Ostebee A, Zorn P. *Calculus: from graphical, numerical, and symbolic points of view, Vol. 2.* San Diego: Saunders College Publishing, 1995.
10. Horan MJ. Task force on blood pressure control in children, report of the second task force on blood pressure in children. *Pediatrics* 1987;79:1.
11. Adelson PD, Bratton SL, Carney NA, et al. Guidelines for the acute medical management of severe traumatic brain injury in infants, children, and adolescents. Chapter 4. Resuscitation of blood pressure and oxygenation and pre-hospital brain-specific therapies for the severe pediatric traumatic brain injury patient. *Pediatr Crit Care Med* 2003;4:S12-S18.
12. Zou G. A modified Poisson regression approach to prospective studies with binary data. *Am J Epidemiol* 2004;159:702-6.
13. Gopinath SP, Cormio M, Ziegler J, et al. Intraoperative jugular desaturation during surgery for traumatic intracranial hematomas. *Anesth Analg* 1996;83:1014-21.
14. Chesnut RM, Marshall SB, Piek J, et al. Early and late systemic hypotension as a frequent and fundamental source of cerebral ischemia following severe brain injury in the Traumatic Coma Data Bank. *Acta Neurochir Suppl (Wien)* 1993;59:121-5.
15. Kochanek PM, Clark RS, 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;1:4-19.
16. Kawaguchi M, Sakamoto T, Ohnishi H, et al. Preoperative predictors of reduction in arterial blood pressure following dural opening during surgical evacuation of acute subdural hematoma. *J Neurosurg Anesthesiol* 1996;8:117-22.
17. Kinoshita K, Kushi H, Sakurai A, et al. Risk factors for intraoperative hypotension in traumatic intracranial hematoma. *Resuscitation* 2004;60:151-5.
18. Meyer PG, Ducrocq S, Rackelbom T, et al. Surgical evacuation of acute subdural hematoma improves cerebral hemodynamics in children: a transcranial Doppler evaluation. *Childs Nerv Syst* 2005;21:133-7.
19. Haglund MM, Grady MS, Kanev PM, et al. Rapid infusion system for neurosurgical treatment of massive intraoperative hemorrhage. *Anesth Analg* 1996;83:1014-21.
20. Ransom GH, Mann FA, Vavilala MS, et al. Cerebral infarct in head injury: relationship to child abuse. *Child Abuse Negl* 2003;27:381-92.
21. Barlow KM, Thomson E, Johnson D, Minns RA. Late neurologic and cognitive sequelae of inflicted traumatic brain injury in infancy. *Pediatrics* 2005;116:e174-85.

## ERRATUM

In the December 2005 issue of *Anesthesia & Analgesia*, in the article by Anghelescu et al., "The Safety of Patient-Controlled Analgesia by Proxy in Pediatric Oncology Patients" (2005;101:1623-7), the authors have identified an error and wish to publish a correction.

In the abstract, the sentence that reads "In the PCA by proxy group two respiratory complications and two neurological complications were observed." should read "In the PCA by proxy group two respiratory complications, two neurological complications and one mixed respiratory and neurological complication were observed."

On page 1624, in the Results section, the sentences that read "PCA was self-managed during 26 of the 28 periods in which respiratory status was altered and during 33 of the 35 periods in which neurological changes were reported. Therefore, serious side effects occurred in only 4 of 576 (0.87%) of periods of PCA by proxy." should read "PCA was self-managed during 26 of the 28 periods in which respiratory status was altered, during 33 of the 35 periods in which neurological changes were reported and 1 of the 7 periods in which both neurological and respiratory complications were observed. Therefore, serious side effects occurred in only 5 of 576 (0.87%) of periods of PCA by proxy."

The authors regret not detecting this inconsistency between the table and the text prior to publication.