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Comparison of outcomes following decompressive craniectomy in children with accidental and nonaccidental blunt cranial trauma

Clinical article

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Object. The goal of this study was to compare clinical outcomes following decompressive craniectomy performed for intracranial hypertension in children with nonaccidental, blunt cranial trauma with outcomes of decompressive craniectomy in children injured by other mechanisms.

Methods. All children in a prospectively acquired database of trauma admissions who underwent decompressive craniectomy over a 9-year span, beginning January 1, 2000, are the basis for this study. Clinical records and neuroimaging studies were systematically reviewed.

Results. Thirty-seven children met the inclusion criteria. Nonaccidental head trauma was the most common mechanism of injury (38%). The mortality rate in patients with abusive brain injury (35.7%) was significantly higher ($p < 0.05$) than in patients with other causes of traumatic brain injury (4.3%). Children with inflicted head injuries had a 12-fold increase in the odds of death and 3-fold increase in the odds of a poor outcome (King's Outcome Scale for Closed Head Injury score of 1, 2, or 3).

Conclusions. Children with nonaccidental blunt cranial trauma have significantly higher mortality following decompressive craniectomy than do children with other mechanisms of injury. This understanding can be interpreted to mean either that the threshold for decompression should be lower in children with nonaccidental closed head injury or that decompression is unlikely to alter the path to a fatal outcome. If decompressive craniectomy is to be effective in reducing mortality in the setting of nonaccidental blunt cranial trauma, it should be done quite early.
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Key Words • **decompressive craniectomy** • **nonaccidental trauma** • **children** • **outcome** • **mortality**

Traumatic brain injury resulting from nonaccidental trauma is a major cause of morbidity and mortality in children.[15,28] Intracranial hypertension is commonly a major component in the pathophysiology, and most treatment strategies quite reasonably target ICP and CPP. Therapy that targets these parameters has improved clinical outcomes.[4,5] When intracranial hypertension is refractory to noninvasive and minimally invasive measures, decompressive craniectomy must be considered, but there is no consensus on the timing of decompressive craniectomy.[24] Reports predominantly dealing with adult patients have described the results from decompressive craniectomy done late in the course of refractory intracranial hypertension, and some of these have suggested that early decompressive craniectomy can result in more favorable outcomes.[16,32] Other reports have addressed outcomes following decompressive craniectomy in children with TBI,[1,12,21,23,25,37] and a significant proportion of these patients were victims of abusive head trauma.[1,25] Importantly, little is known about the comparative benefits of decompressive craniectomy in children who have sustained abusive head trauma.

This investigation examines the outcome, following

Abbreviations used in this paper: CPP = cerebral perfusion pressure; GCS = Glasgow Coma Scale; ICP = intracranial pressure; ISS = Injury Severity Score; KOSCHI = King's Outcome Scale for Closed Head Injury; TBI = traumatic brain injury.

decompressive craniectomy, in children with TBI and compares cases involving abusive head trauma with those involving other mechanisms of injury.

Methods

All children who underwent decompressive craniectomy at The Children's Hospital, Denver/Aurora, Colorado between January 1, 2000, and December 30, 2008, were included in this study. Two patients who underwent emergency decompressive craniectomy in outside facilities and were immediately transferred to our institution were also included in this study. This investigation was done with approval by the Colorado Multiple Institutional Review Board. Data collected for analysis include patient age, sex, and mechanism of head injury, GCS score, ISS, presence of retinal hemorrhage at time of initial evaluation, and ICP measurements. Long-term functional outcomes in patients who survived were evaluated using the King's Outcome Scale for Closed Head Injury (KOSCHI).[7] This assessment tool was described by Crouchman et al.[7] and was proposed as a specific pediatric adaptation of the GOS. It is reported to have increased sensitivity at the milder end of the disability range compared with the GOS and is easy to use.[7] A KOSCHI score of 5 (good recovery) or 4 (independent) is regarded as a favorable outcome and a score of 3 (severe disability), 2 (vegetative state), or 1 (death) is regarded as a poor outcome. The KOSCHI is shown in Table 1. Also recorded for the present study were the presence of visual impairment, including cortical visual impairment, at the time of discharge, the long-term need for CSF shunts or gastrostomies, and the development of posttraumatic epilepsy. The outcomes following decompressive craniectomy in children with TBI resulting from abusive head trauma are compared with outcomes following TBI from other mechanisms of injury.

Hypodensity on all available preoperative CT scans was graded by 2 pediatric neuroradiologists (N.V.S. and L.V.F.), using a scale they devised especially for this study. Briefly, in this scale each lobe of each hemisphere is assigned a score of 0 (no hypodensity), 0.5 (less than half the lobe involved), or 1 (more than half involved). The lobes include the frontal, temporal, parietal, and occipital lobes and the insula. Basal ganglia hypodensity is arbitrarily scored as part of the insula. Scores from each lobe, then each hemisphere, are added to give the total score. Total scores for both hemispheres may range from 0 to 10. The 2 radiologists graded each scan by consensus. If more than 1 preoperative scan was available for review, the most recent prior to decompressive craniectomy was chosen.

All children were initially evaluated by the pediatric trauma team and a member of the pediatric neurosurgical

TABLE 1: KOSCHI category definitions*

Category		Definition
1	Death	
2	Vegetative	The child is breathing spontaneously and may have sleep/wake cycles. He may have non-purposeful or reflex movements of limbs or eyes. There is no evidence of ability to communicate verbally or non-verbally or to respond to commands.
3	Severe disability	(a) The child is at least intermittently able to move part of the body/eyes to command or make purposeful spontaneous movements; for example, confused child pulling at nasogastric tube, lashing out at carers, rolling over in bed. May be fully conscious and able to communicate but not yet able to carry out any self care activities such as feeding. (b) Implies a continuing high level of dependency, but the child can assist in daily activities; for example, can feed self or walk with assistance or help to place items of clothing. Such a child is fully conscious but may still have a degree of post-traumatic amnesia.
4	Moderate disability	(a) The child is mostly independent but needs a degree of supervision/actual help for physical or behavioural problems. Such a child has overt problems; for example, 12 year old with moderate hemiplegia and dyspraxia insecure on stairs or needing help with dressing. (b) The child is age appropriately independent but has residual problems with learning/behavior or neurological sequelae affecting function. He probably should have special needs assistance but his special needs may not have been recognized/met. Children with symptoms of post-traumatic stress are likely to fall into this category.
5	Good recovery	(a) This should only be assigned if the head injury has resulted in a new condition which does not interfere with the child's well being and/or functioning; for example: • Minor headaches not interfering with social or school functioning • Abnormalities on brain scan without any detectable new problem • Prophylactic anticonvulsants in the absence of clinical seizures • Unsightly scarring of face/head likely to need cosmetic surgery at some stage • Mild neurological asymmetry but no evidence of effect on function of limb (includes isolated change in hand dominance in young child). (b) Implies that the information available is that the child has made a complete recovery with no detectable sequelae from the head injury.

* Reproduced with permission from Crouchman et al.: **Arch Dis Child 84:**120–1124, 2001.

service. Following a primary survey, resuscitation, and stabilization, further management of the head injury was dependent upon clinical details and findings on neuroimaging. Intracranial mass lesions deemed surgical were treated with immediate operative evacuation. The cranial bone flap was left off if there was evidence of significant cerebral swelling. Decisions regarding significance were made by the attending pediatric neurosurgeon. In the absence of mass lesions, intraparenchymal ICP monitors were inserted in patients with abnormalities on brain imaging primarily if they had GCS scores of 8 or less or if the mental status could not be followed by serial neurological examinations because of sedation or other injuries. A decision for decompressive craniectomy was considered if there was a sustained ICP greater than 20 mm Hg that remained refractory to aggressive medical measures and CSF drainage.

A large unilateral hemicraniectomy was made on the side of a mass lesion or, in the absence of a mass lesion, on the side of the most prominent cerebral edema (Fig. 1). For bifrontal mass lesions, a large bifrontal craniectomy was made. The dura mater was always opened and a dural substitute laid over the dural defect. Bone flaps were sent to the hospital tissue bank for cryopreservation.

All diagnoses of nonaccidental trauma were confirmed by a prompt and very thorough investigation by the Child Advocacy and Protection Team of the Children's Hospital, and all were reported to appropriate authorities. In the cases of accidental trauma, there was either no reason for suspicion of nonaccidental trauma or investigation failed to support that diagnosis.

The 2-sided t-test, Student test, Mann-Whitney test, and Fisher exact test (2 tailed) were used to evaluate the data, using a confidence level of 95%. A probability value of 0.05 was adopted as the threshold for statistical significance.

Results

Thirty-seven children met the criteria established for inclusion. The causes of TBI in our patients are shown in Table 2. Twenty-one patients (57%) were male and 16 (43%) were female. Nonaccidental trauma accounted for

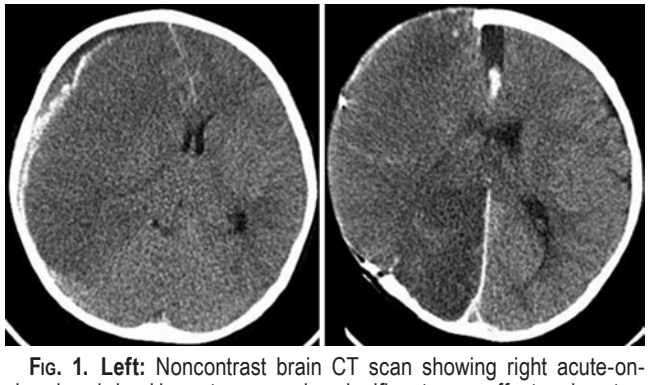

Fig. 1. Left: Noncontrast brain CT scan showing right acute-on-chronic subdural hematoma causing significant mass effect and contralateral midline shift. Note extensive right hemispheric ischemic changes. **Right:** Noncontrast brain CT scan obtained following right-sided decompressive craniectomy showing herniation of edematous brain through the cranial defect and resolution of previously noted midline shift.

TABLE 2: Mechanisms of head injury in 37 patients*

Mechanism	No. of Pts (%)
nonaccidental trauma	14 (38)
MVA	7 (19)
auto vs pedestrian	5 (14)
fall	4 (11)
sports injury	3 (8)
auto vs bicyclist	2 (5)
other†	2 (5)

* MVA = motor vehicle accident.

† One patient had been kicked in the head by a horse and a second patient was assaulted in a fight.

14 (38%) of the 37 cases of TBI; 23 (62%) of the children had other mechanisms of trauma. The overall mean (SD) age at craniectomy was 6.0 ± 1.5 years (range 9 weeks–15 years). The mean age at craniectomy for the children with abusive head trauma was 2.2 ± 1.0 years, whereas that for patients with accidental head trauma was 8.4 ± 1.8 years. This difference is significant ($p < 0.05$). The median time from arrival in the emergency department to arrival in the operating room was 93 minutes for abusive head trauma patients and 100.5 minutes for patients with TBI resulting from other mechanisms ($p = 0.76$, Mann-Whitney test). The mean GCS score for all patients was 5.5 ± 1.1. The mean GCS for abusive head trauma patients was 4.5 ± 0.9, and the mean GCS for patients with other mechanisms of TBI was 6.2 ± 1.6 ($p > 0.05$). Preoperative ICP measurements were recorded in 16 of the 37 patients; 8 of these patients had nonaccidental trauma. The mean ICP in these 8 abusive head trauma patients was 34 ± 32.7 mm Hg; the mean ICP in the 8 accidental head trauma patients in whom it was measured was 25 ± 14.2 mm Hg ($p > 0.05$). The mean ISS on admission for all patients was 28.1 ± 2.5. The mean ISS for abusive head trauma patients was 26.7 ± 1.9 and that for accidental head trauma patients was 28.9 ± 3.9 ($p > 0.05$). Preoperative CT scans obtained in 29 patients (13 with nonaccidental head trauma and 16 with accidental head trauma) were available for grading of hypodensity using the scale devised by the 2 pediatric neuroradiologists in this study. The mean grading score in the 13 patients with nonaccidental head trauma was 4.9 ± 1.4, whereas the mean score in the 16 with accidental head trauma was 2.9 ± 0.9. This difference was statistically significant ($p = 0.02$, Mann-Whitney test, 2 tailed). The values of the various predictors of outcome are summarized in Table 3. As shown in Table 3, the only predictors of outcome that differed significantly between the 2 groups were the age of the patients and the scores on the CT hypodensity grading scale. Since it is well recognized that children with nonaccidental head trauma are significantly younger than those with accidental head trauma, we sought to assess the impact of CT appearances on the outcome in our patients. There was, however, no correlation between the scores on the CT hypodensity grading scale and outcome of patients based on KOSCHI scores either in patients with nonaccidental trauma or those with other mechanisms of injury (Spearman correlation).

TABLE 3: Comparison of predictors of outcomes in 27 patients*

Variable	Nonaccidental Trauma (14 pts)	Other Mechanisms (23 pts)	Significant ($p < 0.05$)
mean age (yrs)	2.19 ± 1.02	8.38 ± 1.80	yes
mean ISS	26.71 ± 1.93	28.91 ± 3.89	no
GCS	4.5 ± 0.93	6.2 ± 1.61	no
median time to OR (mins)†	93	100.5	no
mean preop ICP (mm Hg)‡	34 ± 32.7	25 ± 14.2	no
score on CT hypodensity grading scale§	4.9 ± 1.4	2.9 ± 0.9)	yes

* OR = operating room; pts = patients.
† Measured from time of arrival in emergency department.
‡ Data based on 16 patients (8 in each group).
§ Data based on 29 patients (13 with nonaccidental trauma and 16 with TBI due to other mechanisms).

The findings on initial head CT scans are summarized in Table 4. Acute subdural hematomas were identified in 14 (100%) of the children with abusive head injury and in 13 (57%) of the children with other causes of TBI. Only 1 (7.1%) of the patients with abusive head trauma had a calvarial fracture, whereas 15 (65%) of patients with other causes of TBI had such fractures. Fifty percent (7) of the children who had abusive head trauma had only a single abnormality on neuroimaging, whereas 4 patients (17%) of those with accidental causes of TBI had only a single such abnormality.

All deaths occurred within 30 days of head injury. There were 5 deaths in the 14 abusive head trauma patients (mortality rate 35.7%). In 4 of these patients care was withdrawn due to a poor prognosis related to cardiorespiratory failure, extremely poor neurological status, and/or continued intractably elevated ICP (1 of these patients may have progressed to brain death, but had been receiving pentobarbital.) Only 1 death occurred in the 23 patients with accidental causes of TBI (mortality rate 4.3%). This death was from intraoperative cardiac failure after dural opening; and the patient could not be resuscitated. This difference in mortality rate is statistically significant ($p < 0.05$). The odds ratio of death in nonaccidental trauma patients compared with accidental trauma patients was 12.2 ($p = 0.02$), meaning that the odds of death were 12-fold greater in patients with nonaccidental head trauma.

TABLE 4: Radiographic findings present on initial brain CT scan*

Abnormality or No. of Abnormalities	Nonaccidental Trauma (14 pts)	Other Mechanisms (23 pts)
subdural hematoma	14 (100)	13 (57)
edema	11 (46)	12 (52)
skull fracture	1 (7)	15 (65)
diffuse axonal injury	2 (14)	1 (4)
contusion	2 (14)	6 (26)
epidural hematoma	0 (0)	4 (17)
SAH	2 (14)	1 (4)
infarction	1 (7)	1 (7)
IPH	1 (7)	3 (13)
cerebral contusion	0 (0)	4 (17)
1 abnormality	7 (50)	4 (17)
2 abnormality	4 (29)	15 (65)
3 or more abnormalities	3 (21)	4 (17)

* Values represent numbers of patients (%). Abbreviations: IPH = intraparenchymal hemorrhage; SAH = subarachnoid hemorrhage.

The single death in an accidental trauma patient was intraoperative. The patient developed pulseless electrical activity and could not be resuscitated. The 5 deaths in patients with nonaccidental trauma were all similar to each other. One patient was pronounced brain dead, and the rest were removed from the ventilator (2 at or near brain death but without official brain death examinations, 1 with extremely elevated ICP, and 1 with cardiorespiratory failure).

Four of the 31 surviving patients (13%) were discharged directly to their homes, and none of these patients had been injured by nonaccidental trauma. Twenty-seven patients (87%) were discharged to rehabilitation facilities. All of the 9 surviving abusive head trauma patients were discharged to rehabilitation facilities. At the time of discharge visual impairment was present in 66.7% of the children with abusive head trauma compared with 9.1% of those with accidental head trauma ($p < 0.05$). Ophthalmoscopic examinations were documented in 13 of the 14 patients with abusive head trauma, and in 12 of these patients retinal hemorrhages were seen. Of the 9 surviving patients with nonaccidental trauma, the presence or absence of cortical visual impairment could be determined from the medical records in 7; 4 (57%) had cortical visual impairment and 3 did not. Of the 2 surviving nonaccidental trauma patients for whom the cortical visual impairment status could not be determined, 1 was found not to have retinal hemorrhages. There was no significant difference between the 2 groups with respect to the need for CSF shunts or gastrostomies or the development of posttraumatic epilepsy (Table 5). The mean follow-up period was 23.9 months (range 1–94 months).

Overall, a poor outcome was present in 57% of the patients with nonaccidental head trauma and in 30% of the children with accidental head trauma. Although the difference was not statistically significant ($p = 0.17$, Fisher exact test), there was a 3-fold greater odds of poor outcome in patients with nonaccidental head trauma (OR 3.04). Among survivors, the mean KOSCHI for patients with nonaccidental trauma was 3.7, whereas that for patients with TBI from other causes was 4.0 ($p > 0.05$).

Discussion

Among 37 children with blunt cranial trauma sufficiently severe to require decompressive craniectomy, those who were victims of nonaccidental trauma had significantly worse survival than did those from other etiologies of trauma. The odds of death were 12-fold greater in children with inflicted trauma than in children who

TABLE 5: Summary of outcomes

Outcome	Nonaccidental Trauma	Other Mechanisms	Significance (p <0.05)
mortality	35.7	4.3	yes
poor outcome†	57.2	30.4	no‡
visual impairment	66.7	9.1	yes
shunt placement	33.3	27.3	no
gastrostomy tube	33.3	27.3	no
epilepsy	33.3	27.3	no

* All values represent percentages of patients. The mortality and poor outcome data are based on 37 patients (14 in the nonaccidental trauma group and 23 in the other mechanisms group). Data on visual impairment, shunt placement, gastrostomy tube use, and epilepsy were available for the surviving patients only. Thus, the percentage values for these variables are based on 9 patients in the nonaccidental trauma group and 22 in the other mechanisms group.

† KOSCHI score 1, 2, or 3.

‡ Odds ratio 3.04.

suffered similarly severe TBI from other causes. The aggregate mortality rate for this series was 16.2%, but there was a statistically significant difference in the distribution of deaths by etiology of trauma ($p < 0.05$). The mortality rate for the 14 children with nonaccidental trauma was 35.7%, but for the 23 patients with other causes of TBI, it was only 4.3%. The published aggregate mortality rates following decompressive craniectomy for pediatric TBI resulting from all mechanisms of injury have ranged from 25% to 31.4%.[20,21,25]

Following the primary trauma, a cascade of secondary events, such as hypoxia, edema, ischemia, increases in vascular permeability, and loss of cerebral autoregulation, commonly results in progressively rising ICP and further reduction in cerebral perfusion.[17,29,30] As ICP rises above normal values, cerebral arterial autoregulation initially compensates quite well to maintain stable or acceptable CPP, but when autoregulation fails due to cerebral injury and when ICP is sufficiently elevated to directly compromise CPP, and hence cerebral blood flow, ischemia and parenchymal infarction soon follow. This paradigm has its foundation in the Monro-Kellie doctrine.[26,31] Therapies targeting ICP and CPP are central components in all management strategies for severe closed head injury.[2,21] Empirical support comes from clinical studies that have demonstrated associations of clinical outcomes with ICP and CPP.[4,5] Various combinations of interventions, such as sedation, mild hyperventilation, neuromuscular paralysis, administration of osmotic agents, administration of diuretics, and CSF drainage, are commonly used as first-tier measures to reduce ICP. There is no drug or combination of drugs that can consistently and reliably halt the pathophysiological cascade of edema, inflammation, and impaired vascular permeability (and more edema) that accentuates or drives intracranial hypertension toward brain death.

When intracranial hypertension is refractory to noninvasive measures and CSF drainage, the more aggressive option of decompressive craniectomy becomes the remaining rational choice. Interestingly, Harvey Cushing[8] was the first to describe decompressive craniectomy for TBI. Recent studies suggest that decompressive craniectomy, when performed early in the course of management of TBI, can result in more favorable outcomes than does persistence with noninvasive steps.[12,23,37] The renewed interest in decompressive craniectomy in the management of TBI is evidenced by 2 clinical trials currently underway—the RESCUEicp (Randomized Evaluation of Surgery for Uncontrollable Elevation of Intracranial Pressure) trial[19] and the DECRA (DEcompressive CRAniectomy) trial.[34]

There have been several reports on decompressive craniectomy in children with TBI,[1,12,20,23,25,37] but few have specifically addressed its efficacy with nonaccidental or inflicted head trauma.[6] This is unfortunate because TBI resulting from nonaccidental trauma accounts for 28% of all head injuries in children under 2 years of age and 1.6% of head injuries in children of all ages.[9,27] In a prospective population-based study, Barlow and Minns[3] reported an annual incidence of 24.6 cases of inflicted head injury per 100,000 children younger than 1 year of age. The fact that the majority of children with inflicted head injuries have poor outcomes heightens the public health significance of nonaccidental trauma. Makaroff and Putnam,[28] in a review 19 articles on outcomes following inflicted head trauma in children, found an overall mortality rate of 25% and significant neurological impairment in approximately half of the cases. Interestingly, nonaccidental head trauma was the most common mechanism of head injury (38%) in our series of 37 children who underwent decompressive craniectomy, and the same has been true in similar series reported by Kan et al.[25] (23.5%) and Adamo et al.[1] (86%).

There is wide variation in surgical techniques reported in the literature for pediatric decompressive craniectomy.[1,18,20,23,25] The authors of this series made unilateral frontotemporoparietal craniectomies on the side of the mass lesion or, in the absence of a mass lesion, on the side with the most severe cerebral edema. Bifrontal mass lesions were treated with bifrontal craniectomies. Large durotomies were made in all patients in this series. Taylor et al.[37] performed bitemporal craniectomies without durotomies in their 13 patients, and Cho et al.[6] performed bifrontal craniectomies in 5 patients and unilateral frontotemporoparietal craniectomies in another 5 patients.

The KOSCHI was used to assess long-term functional outcome and independence. An alternative well-described scale, which has been used extensively by other researchers, is the GOS.[20,21,23] We used the KOSCHI, however, because it is a specific pediatric adaptation of the GOS and is reported to have more sensitivity at the milder end of the disability range.[7] The outcomes of patients with inflicted head injuries were compared with those with other mechanisms of closed head injury, after correcting for such well-recognized confounding factors as initial GCS score after injury, preoperative ICP, time to decompressive craniectomy, and the presence and degree of systemic injuries (none of which differed significantly between our 2 groups of patients).[13,21] The finding that GCS scores did not differ significantly between our patients with accidental trauma and those with nonac-

cidental trauma may simply reflect the reduced reliability of GSC scores in younger children. A poor outcome (KOSCHI category 1, 2, or 3) was found in 57% of our patients with nonaccidental trauma but only 30% of those with other mechanisms of injury, although this difference was not statistically significant ($p = 0.17$). Nevertheless, these data do demonstrate a 3-fold greater odds of poor outcome in the patients with nonaccidental trauma. Kan et al.[25] reported a mean KOSCHI score of 4.5 in 51 children at a mean of 18.6 months following decompressive craniectomy, and Adamo et al.[1] reported KOSCHI scores of 3 or 4 at 1 year following decompressive craniectomies (86% of the patients had suffered nonaccidental trauma). Using the GOS, Jagannathan et al.[20] reported a mean 2-year score of 4.2 in their report on decompressive craniectomy in pediatric patients with TBI.

The incidence of visual impairment in our nonaccidental trauma survivors was significantly greater than in our other group. This is likely a reflection of the incidence of retinal hemorrhages in our NAT patients—12/13, or 92%—and incidence of cortical visual impairment in our nonaccidental trauma survivors—4/7, or 57%. In the literature, there is a 75%–80% incidence of retinal hemorrhage in nonaccidental trauma patients with subdural hematoma.[33] We found no significant difference between the 2 groups in terms of posttraumatic hydrocephalus as evidenced by the need for shunt placement, need for gastrostomy, or the development of posttraumatic epilepsy.

It is important to note that whether decompressive craniectomy leads to reduced morbidity and mortality and/or better outcomes in victims of nonaccidental trauma than no decompressive craniectomy is not and cannot be answered by this series. We did not compare patients who underwent decompressive craniectomy with patients who did not.

However, we noted worse outcomes following decompressive craniectomy in patients with inflicted head trauma. The explanation for this is not clear, but several factors may be reasonably considered. We believe that the indications for decompressive craniectomy were uniform over the span of this study and, more specifically, were not skewed toward being more aggressive in children with more severe injuries or those with nonaccidental trauma. It is reasonable to believe that the reduction in ICP following decompressive craniectomy should be independent of mechanisms of injury and therefore comparably effective. Thus factors other than intracranial hypertension and its consequences must be sought to explain the differentially worse outcomes that occur in children with nonaccidental trauma. Patients who are victims of accidental trauma are, with rare exceptions, rushed to medical attention, and the medical history is usually forthcoming and reliable. On the other hand, victims of nonaccidental trauma may have significant delays between injury and medical attention, may have received multiple traumas over hours or even days, and may have sustained contributing injuries, such as suffocation, that are never recognized.

Hypoxic-ischemic encephalopathy is commonly present on postmortem microscopy in children with nonaccidental trauma, and these observations are likely to be related to the high incidence of apnea observed in infants who have sustained this type of injury, as reported by Johnson et al.[22] and Geddes et al.[14] Based on a retrospective study of 28 children with nonaccidental trauma, Johnson et al.[22] reported that 57% had a history of apnea prior to hospitalization and 82% had been intubated on arrival. This frequency of presenting complaints of respiratory abnormalities, including apnea, by caretakers is well known to clinicians who are involved in the care of nonaccidental trauma victims. The predilection for axonal injuries at the cervicomedullary junction in these patients may explain the high incidence of apnea,[14,36] and such injuries are likely associated with angular acceleration/deceleration of the head. Geddes et al.[14] noted foci of ischemic-type staining in the dorsal brainstem, particularly in the lower medulla, in autopsies of NAT victims. In other studies, autopsies have disclosed injuries in the cervicomedullary and high cervical spinal cord.[22,35] These injuries may be related to anatomical and biomechanical characteristics operant in this usually infant population. During shaking or impact, small children's disproportionately large heads are at risk for significant angular acceleration relative to their necks, which typically have, compared with older children or adults, underdeveloped musculature and cervical spines with relatively lax ligaments, more vertically oriented facet joints, and less completely ossified vertebral bodies. As a consequence, the cervicomedullary junction and upper cervical spinal cord may be particularly vulnerable to injury in younger children.[10,11] The high incidence of respiratory problems on presentation of patients with nonaccidental trauma is also consistent with the interpretation that medical attention was sought only after respiratory problems indicated to the caretaker that further delay in seeking medical attention after the inflicted injury could no longer be tolerated and death was becoming a consideration.

Hypoxia can be worsened by hypotension. In addition to causing apnea or respiratory abnormalities, high cervical spinal cord or cervicomedullary injuries in children may result in hypotension or cardiorespiratory arrest. A combined role of ischemia and hypoxia is supported by Johnson et al.,[22] who noted that the first recorded blood pressure in nonaccidental trauma patients was less than 80 mm Hg in 50% of their cases and that the arterial pH was less than 7.3 in 54%. They argued that because prehospital resuscitative measures had been initiated before these children were assessed, the recorded values may underestimate the true extent of initial hypotension and acidosis.

Conclusions

The data from this investigation strongly support the position that the etiology of trauma affects prognosis in children with blunt cranial trauma who undergo decompressive craniectomy. Nonaccidental trauma is associated with a much worse prognosis than are other seemingly comparable injuries due to other causes. This understanding can be interpreted to mean either that the threshold for decompression should be lower in children with nonaccidental head trauma or that the die is cast in many of these cases and decompression will probably not alter the path to a poor or fatal outcome. Whether decompressive craniectomy leads to reduced morbidity and mortality

and/or better outcomes in patients with nonaccidental trauma than no decompressive craniectomy cannot be answered by this series. We did not compare patients who underwent decompressive craniectomy with patients who did not. Until more data of high quality are available, we choose to recommend thoughtful case-by-case evaluation with a bias toward earlier decompressive craniectomy in children with nonaccidental trauma. If decompressive craniectomy is to be effective in reducing mortality in the setting of nonaccidental blunt cranial trauma, it should be done quite early.

Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author contributions to the study and manuscript preparation include the following. Conception and design: Wilkinson, Oluigbo, Handler. Acquisition of data: Oluigbo, Stence. Analysis and interpretation of data: Wilkinson, Oluigbo, Stence, Fenton. Drafting the article: Wilkinson, Oluigbo, Handler. Critically revising the article: all authors. Reviewed submitted version of manuscript: all authors. Statistical analysis: Oluigbo. Administrative/technical/material support: Wilkinson, Oluigbo, Fenton, Handler. Study supervision: Wilkinson, McNatt, Handler.

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