J Neurosurg (5 Suppl Pediatrics) 105:337–342, 2006

Outcomes after decompressive craniectomy for severe traumatic brain injury in children

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Object. Severe traumatic brain injury (TBI) is often accompanied by early death due to transtentorial herniation. Decompressive craniectomy, performed alone or in conjunction with evacuation of the mass lesion, can reduce the incidence of raised intracranial pressure (ICP). In this paper the authors evaluate mortality and morbidity and long-term outcomes in children who underwent decompressive craniectomy for severe TBI at a single institution.

Methods. Children with severe TBI who underwent decompressive craniectomy at the Primary Children's Medical Center between 1996 and 2005 were identified retrospectively. Descriptive statistics were used to report postoperative mortality and morbidity rates. Long-term recovery in patients who survived was reported using the King's Outcome Scale for Closed Head Injury (KOSCHI).

Fifty-one children with a mean follow-up period of 18.6 months were identified. Nonaccidental trauma accounted for 23.5% of cases. The mean preoperative Glasgow Coma Scale (GCS) score was 4.6. Six patients underwent decompressive craniectomy for elevated ICP only; all other patients underwent decompressive craniectomy in conjunction with removal of the mass lesion. The mean postoperative GCS score was 9.7, and 69.4% of patients had normal ICP levels immediately after surgery. Sixteen children (31.4%) died, including five of six children who underwent decompressive craniectomy for raised ICP alone. Among surviving patients, 2.9% required a tracheostomy, 11.4% required a gastrostomy, 40% experienced posttraumatic shunt-dependent hydrocephalus, and 20% suffered posttraumatic epilepsy requiring antiepileptic agents. The mean KOSCHI score at the last follow-up examination was 4.5 and the mean time to cranioplasty was 2.3 months.

Conclusions. Posttraumatic hydrocephalus and epilepsy were common complications encountered by children with severe TBI who underwent decompressive craniectomy. In patients who underwent decompressive surgery for raised ICP only, the mortality rate was exceedingly high.

Key Words • traumatic brain injury • outcome • decompressive craniectomy • pediatric neurosurgery

Head injury is the leading cause of accidental death in children.[17] Despite advances in neurotrauma and neurocritical care, the morbidity and mortality rates in patients who suffer severe TBI remain high.[3,6] Early death is often a result of transtentorial herniation caused by mass lesions or cerebral swelling. When a significant mass lesion is present, the role of emergency surgical evacuation and decompression is obvious. However, the management of cases in which severe TBI is coupled with refractory ICP in the absence of a mass lesion is more controversial. In the past decade, there has been renewed interest in the use of decompressive craniectomy for such patients. Although authors of a few small reports and pilot studies have addressed the indications and timing of decompressive craniectomy for severe TBI,[13,19,28,32] surgical outcomes in children remain largely unknown. The purpose of this study was to describe cases of children who underwent decompressive craniectomy after severe TBI at our institution during a 10-year period.

Abbreviations used in this paper: CSF = cerebrospinal fluid; CT = computed tomography; GCS = Glasgow Coma Scale; ICP = intracranial pressure; KOSCHI = King's Outcome Scale for Closed Head Injury; NAT = nonaccidental trauma; SDH = subdural hematoma; TBI = traumatic brain injury.

Clinical Material and Methods

Patient Population

We retrospectively reviewed the Trauma Registry at the Primary Children's Medical Center to identify the cohort of children with severe TBI who underwent decompressive craniectomy between 1996 and 2005. Severe TBI was identified by a GCS score[33] of 8 or less after initial resuscitation. All patients underwent intubation either en route to the hospital or in the trauma bay. Decompressive craniectomy was performed either to reduce diffuse cerebral swelling or in conjunction with evacuation of the mass lesion.

Descriptive statistics were used to characterize the study

population and to report occurrences of death and morbid conditions after surgery. Variables that characterized the study population included patient age and sex, mechanism of injury, GCS score on arrival at the hospital, CT findings, concurrent injury, time to surgery, and preoperative ICP and treatment measures. Outcome variables consisted of perioperative GCS scores and ICP levels, death, cause of death, need for tracheostomy or gastrostomy, hydrocephalus, epilepsy requiring antiepileptic agents, infection, and time to cranioplasty. Functional long-term recovery in surviving patients was determined retrospectively by reviewing the medical records and using the KOSCHI[9] (Table 1), a validated outcome measure for children with severe head injury. A KOSCHI score of 5 (good recovery) or 4 (independent) was considered a favorable outcome.

Preliminary Examination and Conservative Management

All children with severe TBI (those with a GCS score ≤ 8) were examined by a trauma team that included a trauma surgeon and a neurosurgeon. After initial airway and hemodynamic management, a head CT scan was obtained. Patients in whom evidence of a significant mass lesion was found underwent emergency surgery. The remaining patients with severe diffuse TBI but no focal surgical lesions were admitted to the pediatric intensive care unit, where a Camino ICP monitor (Integra NeuroSciences, Plainsboro, NJ) was placed. If the ventricles were accessible, an external ventricular drain was also placed to remove excess CSF. A standard ICP protocol, which consisted of head elevation, sedation, paralysis, normothermia, normoventilation (PCO_2 range 35–40 mm Hg), and administration of inotropes, was followed to maintain an ICP of less than 20 mm Hg and a cerebral perfusion pressure of at least 60 mm Hg. The protocol was consistent throughout the 10-year period. In patients with sustained elevated ICP, intravenous administration of mannitol and barbiturate boluses was done, and a repeated CT scan was obtained to rule out the presence of new lesions.

TABLE 1
*The KOSCHI scores**

Score	Description	Definition
1	death	death
2	vegetative state	breathing spontaneously; no ability to communicate verbally or nonverbally or to respond to commands
3	severe disability	a) some purposeful movement or ability to follow commands; may be conscious & able to communicate; unable to care for self
		b) exhibits high level of dependency but can assist w/ own care; fully conscious but w/ posttraumatic amnesia
4	moderate disability	a) mostly independent but requires supervision or help; has overt problems
		b) age appropriately independent but w/ residual learning/behavior problems or neurological sequelae
5	good recovery	a) head injury resulted in new condition that does not affect well being or functioning
		b) complete recovery w/ no detectable sequelae

* Modified from Crouchman, et al.

Criteria for Early and Late Decompressive Hemicraniectomy

The criteria for early decompression (leaving the bone flap out at the time of removal of a mass lesion) included intraoperative brain swelling, poor preoperative patient condition, and high-impact mechanism of injury such as a motor vehicle accident or NAT. The criteria for late decompression (decompressive craniectomy without the presence of a mass lesion) included sustained ICP higher than 25 mm Hg that was refractory to an adequate trial of maximal medical therapy as outlined earlier (usually achieved within 5 hours of arrival at the hospital).

Surgical Technique

In cases in which surgery was performed to treat diffuse cerebral edema without a mass lesion, a wide unilateral hemicraniectomy was performed, including removal of bone in the frontal, temporal, and parietal regions ipsilateral to the hemisphere in which the greatest swelling was observed on CT scans. Although the amount of the cranium removed was not measured, we would estimate it to be between 60 and 80%. The craniectomy was performed from the temporal bone to the floor of the middle fossa to maximize the extent of decompression at the level of the perimesencephalic cisterns. The dura mater was opened widely in a stellate fashion to the extent of bone decompression, and a dural graft was placed to increase the available volume before closure (Figs. 1 and 2). The bone flap was most commonly stored in sterile conditions with cryopreservation or, occasionally, was placed in a subcutaneous abdominal pocket until subsequent cranioplasty.

When surgery was performed to evacuate a mass lesion, a similar wide craniectomy incorporating the mass lesion was performed. In each of these cases, the bone flap was not replaced because of intraoperative brain swelling, and duraplasty was performed after the mass lesion had been removed.

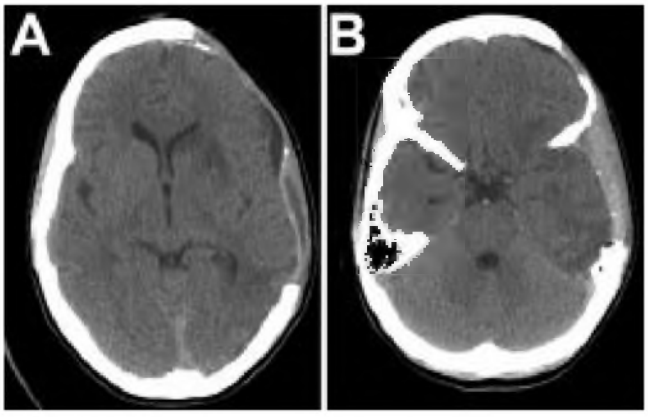

FIG. 1. Computed tomography scans. A: A wide left hemicraniectomy was performed, with removal of bone from the frontal, temporal, and parietal areas. B: The temporal bone was removed to the floor of the middle fossa to maximize the extent of decompression at the level of the perimesencephalic cisterns.

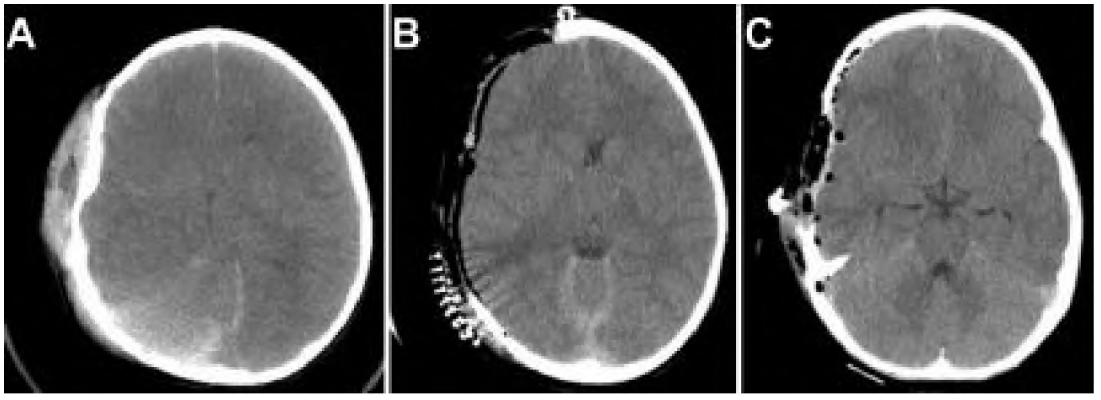

FIG. 2. Computed tomography scans. A: Image obtained in a 5-year-old girl who suffered a two-story fall revealing diffuse cerebral edema, cisternal effacement, and a tentorial SDH. B: A right hemicraniectomy was performed, again with generous removal of frontal and parietal bone. C: The temporal bone was again removed to the floor of the temporal fossa to ensure the adequate decompression of the perimesencephalic cisterns.

Results

Fifty-one children (33 boys and 18 girls; mean age 6.6 years) with severe TBI, who underwent decompressive craniectomy between November 1996 and September 2005, were identified by a review of the Trauma Registry. During the study period, 9214 patients were examined by the trauma service, 4074 of whom suffered some degree of head injury and 277 of whom required a craniotomy. Of the patients in whom a craniotomy was performed for trauma, 45 patients underwent a decompressive craniectomy in conjunction with removal of a mass lesion and six patients underwent a decompressive craniectomy for intractable ICP alone. In our cohort, NAT was the most common mechanism of injury and represented 23.5% of all cases (12 patients). The remaining patients suffered accidental trauma of various forms, including motor vehicle accidents, automobile–pedestrian accidents, bicycle accidents, falls, assaults, and all-terrain vehicle injuries. The concurrent injuries included two liver lacerations, four splenic lacerations, four pulmonary contusions, seven orthopedic injuries, three pneumothoraces, and one arterial injury. The mean preoperative GCS score was 4.6.

Nine patients underwent preoperative ICP monitoring and all of them had persistently elevated ICP (defined as $>$ 25 mm Hg; mean ICP in these patients was 34.2 mm Hg). The ICP was monitored before surgery in five of the six patients without a mass lesion. The sixth patient presented 15 hours after injury with a small amount of parafalcine and tentorial blood, diffuse edema, and a dilated right pupil. He underwent right-sided decompressive craniectomy on arrival without ICP monitoring.

Four of 45 patients with mass lesions did not undergo immediate craniectomy and were initially treated medically; ICP monitoring was performed in these patients. All four had intraparenchymal hematomas. Among the 41 other patients who underwent decompressive craniectomy in conjunction with mass lesion removal, there were five epidural hematomas (each associated with an underlying SDH or cerebral contusion), 18 SDHs, and 18 open or depressed skull fractures with an underlying epidural hematoma or contusion. The average time from arrival at the hospital to surgery was 2.2 hours for the 45 patients who required a craniotomy for hematoma removal (including the four patients who were initially treated medically). Among the six patients who underwent late decompression, the average time to surgery was 5.9 hours after arrival at the hospital and 9 hours after the accident.

In 40 children an ICP monitor was placed during the craniectomy; 69.4% of these children had an ICP of less than 20 mm Hg immediately postoperatively, and the mean postoperative GCS score was 9.7. During the follow-up period (mean 18.6 months), 16 children (31.4%) died, including five of the six children who underwent decompressive surgery for cerebral edema in the absence of a mass lesion (Table 2). The cause of death was TBI with intractable ICP in 15 patients and respiratory failure in one. Among the children who presented with NAT, the mortality rate was 25%. An ICP less than 20 mm Hg was noted immediately postoperatively in 77.1% of the patients who survived and in 50% of those who died.

Of the 35 survivors, one patient (2.9%) required a tracheostomy and four (11.4%) required a gastrostomy. Posttraumatic shunt-dependent hydrocephalus developed in 40% of the surviving children and required the placement of one lumboperitoneal, one subdural, and 12 ventriculoperitoneal shunts. Posttraumatic epilepsy requiring antiepileptic agents (phenobarbital, valproic acid, or phenytoin) developed in 20% of the children. Several surgery-related complications occurred. A postoperative wound infection developed in three children (8.6% of the survivors), two of whom suffered deep infections: a subgaleal fungal abscess in one patient and a subdural empyema in the other. Both cases of infection required multiple surgical drainage procedures and delayed cranioplasty. In both cases, the scalp was difficult to close at the time of decompressive craniectomy because of massive brain edema.

In all 35 survivors, the patient's own bone was replaced once the craniectomy site was soft and the patient's long-term survival was apparent. The mean time to replacement of the bone was 2.3 months. One patient experienced aseptic resorption of the bone flap and required an additional cranioplasty 5 months later. Another patient suffered an infection at the cranioplasty site requiring subsequent bone flap removal and delayed cranioplasty. Of the 35 survivors, 19 had KOSCHI Score 5, 11 Score 4, three Score 3, and two

TABLE 2

*Baseline characteristics and outcomes in patients who underwent decompressive craniectomy for control of ICP only**

Case No.	Age (yrs), Sex	Cause of Injury	Preop GCS Score & Clinical Status	Initial CT Findings	Time to Op From Scene (hrs)	Type of Op	ICP (mm Hg)		Outcome	Cause of Death
							Preop	Postop		
1	5, F	fall	3, pupils fixed & dilated bilat; CPR at scene	cerebral edema	4.2	rt-sided DC	60	20–30	dead	TBI
2	12, M	auto–bike	8, lt pupil dilated in trauma bay	lt temporal contusion & swelling	5	lt-sided DC	50	5	alive	NA
3	14, M	auto–bike	3, period of profound hypotension at scene	cerebral edema	20	bilat subtemporal decompressions	40	15	dead (WD)	TBI
4	8, M	ATV	3, CPR at scene; pupils fixed & dilated bilat	cerebral edema	4	rt-sided DC & exploratory laparotomy for hemodynamic instability	50	NA	dead (WD)	TBI
5	9, F	auto–bike	4, pupils fixed & dilated preop	rt temporal contusion, frontal pretectal hemorrhage, cerebral edema	5.7	rt-sided DC	40†	20	dead	TBI
6	0.3, M	NAT	5, dilated rt pupil on arrival	parafalcine/tentorial SDH, SAH, cerebral edema	15	rt-sided DC	NA	NA	dead	TBI

* ATV = all-terrain vehicle accident; auto–bike = automobile–bicycle accident; CPR = cardiopulmonary resuscitation; DC = decompressive craniectomy; NA = not applicable; SAH = subarachnoid hemorrhage; scene = scene of injury; WD = support withdrawn.

† Immediately preoperatively, ICP was 70 to 80 mm Hg.

Score 2 (mean KOSCHI Score 4.5) at the last follow-up examination.

Discussion

Head injury is the leading cause of accidental death in children,[17] and severe TBI occurs in 60% of children who suffer significant polytrauma of accidental or nonaccidental origin.[7] Despite advances in neurotrauma and neurocritical care, the mortality rate for patients with severe TBI remains high (range 34–60%).[4,26] After exclusion or evacuation of significant traumatic hematomas, the primary goal in the management of severe TBI is to prevent secondary insults by maintaining cerebral perfusion pressure and reducing ICP.

Standardized protocols exist for ICP management in children who have suffered severe TBI.[8] Induction of sedation, paralysis, and normothermia; mild hyperventilation; intravenous administration of mannitol; CSF drainage; and administration of inotropes are generally accepted baseline therapies to treat these children.[27] However, for children with diffuse cerebral edema and high ICP that is refractory to standard medical therapy, there is a lack of agreement among neurosurgeons as to which treatment option is best. Berger, et al.,[7] reported that 30% of survivors of severe TBI in the pediatric population experienced "refractory" ICP during their hospital course. In such circumstances, second-tier therapies have been proposed as treatment options and include cerebral blood flow–guided hyperventilation,[11] administration of barbiturates[10] or hypertonic saline,[20,30] lumbar drainage,[22] and decompressive craniectomy.[13,19,28,32] Although it is believed that these approaches may reduce ICP, they all have potential side effects and none has been shown conclusively to improve outcome.

Recent interest in decompressive craniectomy in the literature prompted us to review our experience with this procedure in children. Based on the results of this study we found the following to be true: 1) The mortality rate is high for patients who undergo late decompressive craniectomy for the treatment of intractable ICP (without a mass lesion); 2) Patients in whom decompressive craniectomy is performed at the time of surgery for clot removal have a high incidence of secondary complications such as hydrocephalus, epilepsy, and wound problems.

The high overall mortality rate demonstrated in our group (31.4%), despite the fact that decompressive procedures were performed, reflected the severity of the underlying injuries and was similar to that observed in the adult population.[2–4,24,25]

When separated into subgroups according to the indication for surgery, the mortality rate (83.3%; five of six patients) was much higher in children in our study who underwent a decompressive craniectomy for diffuse cerebral edema alone than in other cohorts reported previously. The initial GCS scores were 3 in three children and 4 and 5 in one each (mean score 4.3). Cardiopulmonary resuscitation was administered to two patients at the accident scene. All had a preoperative ICP of at least 40 mm Hg, the time from injury to operation was longer than 4 hours (range 4–20 hours) in all, and five of them had fixed pupils. These poor prognostic characteristics differ from the baseline parameters reported in other articles in the literature.

Taylor, et al.,[32] reported three deaths (23.1%) among 13 children treated with decompressive craniectomy (bitemporal craniectomy) for diffuse cerebral edema alone. The median preoperative GCS score was 6, the pupils were fixed in one child, and the mean preoperative ICP was 26.4 mm Hg. Of six children treated with decompressive craniectomy for diffuse cerebral edema, Josan and Sgouros[21] reported that all children survived and four eventually achieved a good recovery. The initial GCS score reported by these authors was higher than ours (mean score 6.8), and only two of their six children had dilated pupils before surgery. Aarabi, et al.,[1] reported a mortality rate of 28% in a series of 50 patients. In

that study the postresuscitation GCS score ranged from 6 to 8 in 23% of the patients and from 9 to 15 in 12%. Only 11% of the patients had pupillary dilation. Differences in patient conditions before treatment would appear to account for the significant differences in outcome between these studies.

The surgical techniques used within and between cohorts were also quite heterogeneous. For example, Ruf and colleagues[28] performed a bifrontal craniectomy in three patients, unilateral craniectomy in two, and occipital craniectomy in one. In contrast, Taylor, et al.,[32] performed a bitemporal craniectomy in all cases, but not duraplasty. Still others have used a craniotomy technique in which the bone flap was loosely attached.[31] The technique and extent of craniectomy may also affect ICP differently. The reported ICP reduction rate varies from 34% in cases in which subtemporal decompression was performed to 85% in cases involving more complex procedures,[16,34] and dural opening has been reported to contribute significantly to ICP reduction when compared with bone elevation alone.[29,34] As elevated ICP after severe TBI predicts poor outcome, the difference in surgical technique may be, in part, responsible for outcome differences. The timing of surgery, which ranged from 33 minutes to 29 hours in the reported studies,[19,32] was another important factor that may confound the true effect of decompressive craniectomy. In our study the mean time to surgery from arrival at the hospital was 5.9 hours in the six patients who underwent decompressive craniectomy for intractable ICP, and the time to surgery exceeded 10 hours in two of our patients. Perhaps earlier decompression should be considered, as there is evidence that early decompressive craniectomy leads to a better outcome.[32] In their report on seven children, Hejazi, et al.,[19] described complete recovery in all children 5 weeks after decompressive craniectomy; the time to surgery after trauma in their study ranged from 35 to 68 minutes.

Other complications were common among the survivors. Forty percent of children in our cohort suffered posttraumatic shunt-dependent hydrocephalus. This rate appears to be considerably higher than the reported incidence of posttraumatic hydrocephalus in patients with severe TBI. Recently, based on CSF dynamics and imaging studies, Marmarou and associates[23] reported that in 20% of patients with severe TBI (GCS score $\leq$ 8) hydrocephalus developed posttrauma. A high incidence of hydrocephalus in patients who undergo decompressive craniectomy may simply reflect the severity and pattern of injury (for example, the amount of traumatic subarachnoid or intraventricular hemorrhage). It is possible, however, that a decompressive craniectomy, particularly one with dural opening and use of a large dural patch, could alter CSF dynamics adversely and/or increase subarachnoid scarring resulting in hydrocephalus.

The incidence of epilepsy after TBI depends on the severity of injury.[5,14] In a well-conducted population study, the 5-year cumulative risk in adults was 10% after severe TBI.[5] In another study the risk in adults was reported to be approximately 15%.[12] Our finding of 20% is a matter of concern but may simply reflect the increased seizure tendency in the pediatric population.

Postoperative infection at the site of supratentorial craniotomy is generally reported to occur in less than 1% of cases.[15,18] Interestingly, all three patients in our study in whom a wound infection developed had elevated ICP postoperatively and presented with wound breakdown and necrosis. This association may suggest that pressure underneath the scalp flap with tension on the suture line is a significant contributor to postoperative wound infection after decompressive craniectomy for severe TBI.

Of the survivors in our study, 2.9% required a tracheostomy and 11.4% required a gastrostomy. The average KOSCHI score at the last follow-up visit (mean 18.6 months) among survivors was 4.5, suggesting that most survivors had regained functional independence. This result is encouraging, considering the high mortality rate associated with this group of patients. Similar results were found among the children who presented after an NAT. They had a mean KOSCHI score of 3.9, again implying that most of them had nondisabling deficits at the last follow-up examination.

Most of our patients presented with a mass lesion that required surgical evacuation. Leaving the bone flap out at that time appeared to help control ICP but was associated with an increased incidence of posttraumatic hydrocephalus and wound complications. Like other cohorts, the number of patients with refractory ICP from cerebral edema in the absence of a mass lesion was small. Nevertheless, the mortality rate in children who presented with a low initial GCS score and cerebral edema in the absence of a mass lesion was exceedingly high despite decompressive craniectomy and maximum medical therapy.

Conclusions

Based on our experience with decompressive craniectomy, we are not able to recommend late craniectomy for intractable ICP in the absence of a mass lesion in patients who experience severe neurological dysfunction. The favorable results noted when decompressive craniectomy is performed early in less severely injured patients seem more encouraging,[32] but further confirmation in a larger study is needed.

Acknowledgment

We thank Kristin Kraus for her editorial assistance in preparing the manuscript for publication.

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Manuscript received November 22, 2005.
Accepted in final form July 27, 2006.
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