Surgical treatment for 'brain compartment syndrome' in children with severe head injury

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Objectives. Traumatic brain injury accounts for a high percentage of deaths in children. Raised intracranial pressure (ICP) due to brain swelling within the closed compartment of the skull leads to death or severe neurological disability if not effectively treated. We report our experience with 12 children who presented with cerebral herniation due to traumatic brain swelling in whom decompressive craniectomy was used as an emergency.

Design. Prospective, observational.

Setting. Red Cross Children's Hospital.

Subjects. Children with severe traumatic brain injury and cerebral swelling.

Outcome measures. Computed tomography (CT) scanning, ICP control, clinical outcome.

Results. Despite the very poor clinical condition of these children preoperatively, aggressive management of the raised pressure resulted in unexpectedly good outcomes.

Conclusion. Aggressive surgical measures to decrease ICP in the emergency situation can be of considerable benefit; the key concepts are selection of appropriate patients and early intervention.

S Afr Med J 2006; **96:** 969-975.

Compartment syndrome of the limbs or the abdomen is a well-known entity in general surgical and orthopaedic practice, characterised by an increase of pressure within a musculofascial compartment leading to progressive neurovascular dysfunction. Although it has not been described as such, raised pressure within the intracranial compartment is arguably the most dramatic example of the same pathological principle, given the rigidity of the cranium and the importance of its contents. When increasing intracranial volume exceeds the compensatory capacity of the brain, the ensuing rise of intracranial pressure (ICP) leads to brain herniation and cerebral ischaemia. If no intervention is forthcoming, severe neurological injury or death is inevitable as a consequence of what is, in essence, a 'brain compartment syndrome'.

In South Africa, traumatic brain injury (TBI) is common and probably represents an under-appreciated reality in our society. Traumatic injury accounts for most deaths in children in the 5 - 15-year-old age group.[1] Motor vehicle accidents, falls and assaults are common causes of injury in children and it is usually the severity of the head injury sustained that determines the eventual outcome. Of those who survive severe TBI, many are left neurologically disabled, placing a significant burden on the immediate family and State resources. Although there is little one can do once the primary injury has been sustained at the moment of impact, it is now recognised that avoidance or early treatment of secondary insults can make a dramatic difference to outcome in individual patients.[2] Of these secondary insults, raised ICP is the most widely recognised.

Space-occupying traumatic haematomas causing raised ICP are readily removed via emergency craniotomy, thereby relieving pressure on the brain. However, when the aetiology of raised ICP is swelling of the brain itself, the management becomes much more challenging. Medical treatment remains suboptimal and despite the promise of pharmacological research to interrupt the biochemical cascades that occur in severe brain trauma, nothing has been developed as yet that prevents the development of traumatic brain swelling in humans.

Decompressive craniectomy has been used in various forms, and for various indications, to manage the problem of increased pressure within the intracranial compartment. The rationale for the operation is intuitive – the removal of a section of the cranium enlarges the available space for brain swelling and reduces the contained pressure therein, thereby preventing cerebral herniation and allowing blood flow to return to the brain. Yet despite the apparent simplicity of the principle, it has remained controversial primarily because of concern regarding its effectiveness. However, careful analysis of the reported data reveals likely reasons for the lack of demonstrable benefit in the earlier literature on the subject.

In our experience with severe traumatic brain injury (TBI) in children, the use of decompressive craniectomy in carefully selected patients with brain swelling has produced dramatic ICP control in patients thought to be herniating. Given the severity of the clinical problem, the outcome in this series is surprisingly favourable. In this study we update our early

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results[3] with a larger number of patients and long-term follow-up. Based on our experience and increasing numbers of favourable reports from other centres, we believe that the key to the optimal use of the procedure is the identification of suitable patients, early intervention and the adequate technical performance of the operation.

Methods and materials

Patients were identified from a prospectively collected database of decompressive craniectomy operations performed for traumatic brain swelling (without haematomas) from September 1999 to September 2005. Collected data included (among others): mechanism of injury, initial clinical assessment, radiological findings, ICP readings, neurological deterioration, extracranial injury, timing of the operation, technical details of the operation, change in postoperative ICP readings and radiological findings, and clinical outcome. Outcome was reported based on clinical assessment at the last neurosurgical clinic visit and school reports. Craniectomy usually entailed removing a large part of the hemicranium ipsilateral to the side of most severe swelling, although in three cases bifrontal craniectomy was done where both hemispheres were equally swollen. Expansion of the dura was achieved by the insertion of a dural graft.

Results

Twelve patients had decompressive craniectomy performed for post-traumatic brain swelling. All had radiological evidence of brain swelling with obliteration of the basal cisterns on preoperative computed tomography (CT) imaging. In 11 patients there had been a documented secondary deterioration in neurological status or refractory rise in ICP that prompted the decision to operate (Table I). The combination of the radiological appearance of obliterated perimesencephalic cisternal spaces, the clinical signs and acute secondary deterioration was consistent with cerebral herniation. One patient had previously had an extradural haematoma taken out via craniotomy, after which he recovered but went on to develop secondary deterioration with brain swelling. All of these patients underwent surgery within 12 hours of this deterioration. In the one exception, there was no secondary deterioration – the decision to operate was made on the grounds of the poor clinical condition at the outset, swollen brain on CT, and elevated ICP. This was the only patient who failed to improve despite surgery. Although ICP control was achieved, he died on day 3 post trauma. Control of ICP and clinical improvement was seen in all of the other patients. Postoperative CT imaging confirmed the reappearance of the previously obliterated basal cisterns (Fig. 1). Increased brain oedema beneath the bone flap was not seen on postoperative imaging in any of the cases. The mean sustained ICP reduction achieved by craniectomy was 53.5% (range 39 - 61%) in patients who had both pre- and postoperative ICP monitoring. This was calculated from the difference between the mean preoperative ICP readings averaged over 5 hours and the mean postoperative ICP over 10 hours. The first 6 hours of readings in the postoperative period were excluded from analysis to avoid the potential confounding effect of general anaesthesia in lowering ICP. A longer period for analysis of postoperative readings was chosen to take into account possible later rises in ICP.

Outcome

Follow-up for survivors was conducted prospectively at the neurosurgical clinic with a minimum follow-up period of 1 year; the median duration of follow-up was 35 months (range 1 - 6 years). All survivors had a favourable outcome: 6 patients had a Glasgow Outcome Score (1 to 5) of 4, and 5 patients had a score of 5. One patient developed bone flap sepsis after the bone had been replaced, which required removal of the flap. Another demonstrated slight subsidence of the flap at follow-up. Asymptomatic subdural hygromas were observed in 2 patients. Long-term behavioural disturbances and mild cognitive deficits were relatively common (Table I).

Discussion

The use of decompressive craniectomy for the relief of intracranial hypertension was originally described by Kocher in 1905. Since then, its use in trauma has been surrounded by controversy. The debate is largely centred on the issue of whether it is of clinical benefit in TBI, and therefore, whether its use justifies the additional effort and risk that the operation may entail. There are also concerns about potential adverse consequences of cranial decompression, such as the possibility of increasing cerebral oedema beneath the bone flap, which may further exacerbate the problem of raised ICP and compromise tissue perfusion.

Exacerbation of cerebral oedema

In theory, brain tissue that is compressed may develop increased oedema when the transmural hydrostatic pressure gradient is reduced by craniectomy, thereby favouring enhanced vasogenic oedema.[4] Of the experimental data reported, the results from Hatashita *et al.*[5] are of particular interest. These investigators studied 4 groups of cats – 1 control group and 3 treatment groups which were subjected to: craniectomy alone, induced hypertension alone, and craniectomy plus induced hypertension. In their experiments, cerebral oedema after craniectomy occurred only when the mean arterial pressure was elevated. Enhanced oedema was not seen in the animals that underwent craniectomy without induced hypertension. In another experiment, evaluation of brain water content in 16 cats that underwent craniectomy compared with 16 controls also showed no increase

Table I. Patient data

No.	Age	Initial presentation	Initial imaging	Deterioration	Repeat CT scan	Outcome
1	6	Fall from height. Initial GCS 5T/15	Cisterns compressed, midline shift 1 cm	2T/15, unilateral unreactive pupil, Cushing's reflex, hemiparesis	Nil	Good outcome. Behavioural problems. Slightly sunken flap
2	6	MVA pedestrian, GCS 8/15	Small temporal contusion, cisterns open	GCS 5/ 15, unilateral fixed pupil, hemiparesis	Cisterns obliterated	Bone flap sepsis, mild cognitive deficits, special schooling
3	11	Blunt assault, GCS 14/15	Parietal contusion, cisterns open	GCS 8/15	Increased swelling, compressed obliterated	Good outcome
4	12	Fall from train, GCS 10/15	Right frontal contusion, cisterns open	GCS 8/15, bradycardic	Increased swelling, cisterns obliterated	Good outcome, behavioural change
5	5	Crush injury (gate), GCS 5T/15	Temporal contusion, cisterns compressed	GCS 4T/15, unilateral fixed pupil	Nil	Mild hemiparesis but independent, CN III palsy
6	9	MVA pedestrian, GCS 5T/15	Swelling, cisterns effaced	ICP uncontrolled	Iincreased swelling, cisterns obliterated	Good outcome. Mild cognitive deficits
7	11	Fall from height, GCS 8/15, left pupil fixed	EDH	GCS 5T/15	Cisterns obliterated, PCA infarct (from herniation)	Good outcome. Mild hemiparesis and VF defect but independent
8	7	MVA pedestrian, shocked, hypoxic, GCS 6T/15	Cisterns open	GCS 3T/15, pupils fixed bilaterally, ICP uncontrolled (50 mmHg), bradycardic	1 cm midline shift, cisterns obliterated	Good outcome. CN VI palsy unchanged
9	11	Gunshot wound, GCS 13/15	Gunshot tract high parietal, cisterns compressed	GCS 8/15, pupils bilaterally unreactive, hemiplegia	Nil	Good outcome, no motor deficits
10	6.5	MVA pedestrian (bus), GCS 3T/15, pupils bilaterally fixed, haemopneumothorax, extensive facial injuries, ICP elevated	Grossly swollen brain, DAI, depressed skull fracture, loss of grey-white interface	Nil	Nil	Died day 3 post-injury
11	6	MVA pedestrian, GCS 6T/15, unilateral pupil fixed, hypotensive	Contusions, cisterns effaced	GCS 5T/15, brain herniation decompressing through basal skull fracture (nose and ears), ICP uncontrolled	Nil	Good outcome but behavioural disinhibition
12	7	MVA, polytrauma, GCS 10T/15, left hemiparesis	Cisterns effaced	GCS 2T/15, pupil unreactive	Cisterns obliterated	Good outcome. Mild cognitive deficits

MVA = motor vehicle accident; GCS = Glasgow Coma Score; CN = cranial nerve; EDH = extradural haematoma; PCA = posterior cerebral artery; VF = visual field; DAI = diffuse axonal injury.

after craniectomy.[6] Neither was there any increased fluid extravasation detected by Evan's blue histological staining in 4 rabbits after craniectomy compared with 3 controls in an experiment by Rinaldi *et al.*[7]

In this current clinical series increased oedema was not visible radiologically in any of the patients in the postoperative period. There was also no late increase in ICP that would have been consistent with increased oedema.

Brain entrapment

Another concern about the operation is the potential for brain entrapment, which may occur when the oedematous brain is compressed against the sharp edges of the durotomy or bone defect, causing venous congestion by impaired venous drainage. To address this concern, Yoo and colleagues[8] demonstrated no increase in epidural pressure after craniectomy using a novel air-pouch system, Whitfield[9] performed angiography that demonstrated no venous obstruction, and Jaeger *et al.*[10] and Stiefel *et al.*[11] showed *improved* oxygenation of the involved brain segment after craniectomy. However, we still prefer to employ a large craniectomy, primarily to maximise the ICP reduction achieved, but also to dissipate the pressure over a larger surface area so as to diminish any possibility of brain entrapment. Not only

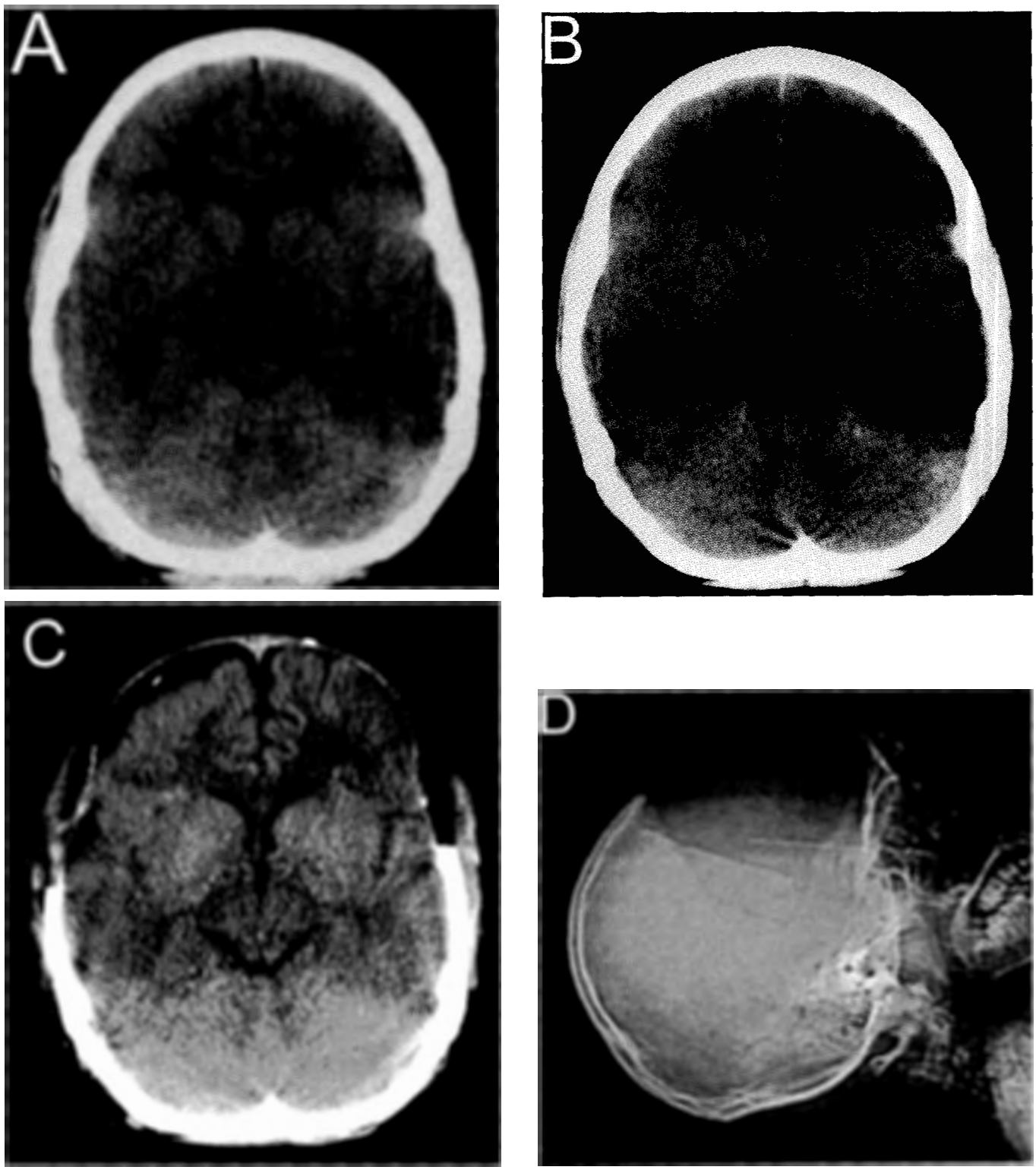

Fig. 1. A: Initial axial CT scan of patient 6 demonstrating a patent quadrageminal cistern and no significant brain swelling. B: CT scan of the same patient after clinical deterioration showing worsening brain swelling with cisternal obliteration; ICP baseline was 40 mmHg with spikes to 90 mmHg despite medical therapy. C: Axial CT scan after bifrontal craniectomy showing re-appearance of the basal cisterns. D: Lateral CT surview demonstrating the extent of the craniectomy.

are small craniectomies ineffective in ICP reduction, they may well lead to a focus of high pressure against a small defect in the bone and dura, promoting herniation through that defect if the pressure remains uncontrolled. In our experience with adults and children, this is not seen when the decompression is of adequate size, unless there is unsalvageable refractory brain swelling.

ICP reduction

The mechanical benefit of decompressive craniectomy is well documented in the experimental and clinical literature. Because the cranium represents a non-compliant system and cerebral perfusion pressure is adversely affected by rising ICP, the removal of a large bone flap expands the available volume for brain swelling, increases the compliance of the system and compensatory capacity of the cerebrospinal fluid axis, and improves cerebral perfusion.[6,12,13] It is also worth noting the additional benefit obtained by expanding the rigid dura mater. Yoo *et al.*[8] demonstrated this graphically with an 85% immediate reduction in ICP after craniectomy: 50% occurred when the bone flap was removed and a further 35% when the dura mater was opened and expanded. Other clinical reports have also confirmed the 2-step reduction in ICP, almost doubling the benefit with the dural augmentation.[10]

The mechanical benefits of craniectomy may be even greater in children, as it has been demonstrated that children have a lower pressure-volume index than adults, i.e. ICP is more sensitive to changes of intracranial volume.[14]

Medical therapy

Although medical therapy is often effective, particularly in patients who have a less aggressive course of elevated ICP, the treating physician must be aware of the limitations of the available options. External ventricular drainage is effective in reducing ICP by releasing CSF from the ventricular system. However, in patients with swollen brains the ventricles are usually effaced and accurate placement of a catheter is often impossible, the amount of CSF that can be drained is limited, and the risk of infection with prolonged drainage must be considered. Mannitol may also be effective in ICP reduction, but usually for a limited period of time only. Repeated doses may be associated with extravasation into the brain with reversal of its effect[15] and renal injury has been documented even without elevated serum osmolarity.[16] Hypertonic saline may be safer than mannitol but fewer data are available on its use. Barbiturates reduce ICP by decreasing metabolism, but prolonged weaning from ventilation after discontinuation and hypotension are common with its use, particularly with the high doses required to achieve burst suppression on the EEG.[17] Ward *et al.*[18] reported hypotensive episodes in more than 50% of subjects on barbiturates compared with 7% of controls. This is of particular concern given the strong association of hypotension with poor outcome.[2]

Clinical benefit of decompressive craniectomy

Early studies reported poor results from decompressive craniectomy in trauma, leading to the initial pessimism surrounding the operation. However, it was originally performed only as a salvage operation – most of these studies were limited by the poor clinical grade of patients and the prolonged period of refractory hypertension that patients were exposed to prior to surgery. Of 2 patients treated by Clark *et al.*,[19] one had decerebrate posturing and the other had no response to painful stimulus; both had bilaterally fixed pupils. Kerr and colleagues[20] also reported 2 patients, both of whom were decerebrate, one with a unilaterally fixed pupil and the other with bilaterally fixed pupils. Eighty-nine per cent of the 73 adults reported by Kjellberg and Prieto[21] displayed decerebrate posturing, with unilateral or bilaterally fixed pupils, while 11 of 13 patients reported by Venes and Collins[22] had evidence of brainstem dysfunction at the time of surgery. All of the 50 head-injured adults reported by Cooper *et al.*[23] had acute subdural haematomas and all but 2 were in a premorbid state, demonstrating decerebrate posturing and/or pupillary abnormalities – in fact, 23 had bilaterally fixed pupils and 27 had absent corneal reflexes. It is probably true that most of the patients in these series suffered severe primary brain injury, for which any radical therapy would probably fail to show benefit. By contrast, favourable outcome has been reported in more recent clinical reports.[24-26]

Complications and outcome

The complication rate in this series is low. One case of bone flap sepsis required removal of the flap. Another showed slight subsidence of the flap at follow-up. Behavioural disturbances were common at follow-up and preoperative signs such as hemiparesis and oculomotor palsy were not completely reversed by surgery in all cases. However, given the overall outcome and considering that avoidance of death and severe neurological morbidity was probably a benefit of this aggressive approach, these are considered relatively minor problems.

Clinical outcome

Although TBI is a heterogenous condition, a number of factors can be used to predict clinical outcome. The Glasgow Coma Score is perhaps the most common of these. Marshall *et al.* [27] analysed the outcome of 746 patients with severe TBI from the Traumatic Coma Data Bank (TCDB), including all patients with an initial GCS of 8 or less, or who had deteriorated to this level within 24 hours. Overall mortality was 36%. They

further analysed outcome according to a novel classification of diffuse injury by CT criteria (Table II). For patients with grade III swelling the mortality was 34%, while 23% remained vegetative and 27% had a severe disability. Only 16% had a favourable outcome. Of those with grade IV injury, 56% of the patients died, 18% were vegetative, 18% were left with a severe disability, and only 6% had a favourable outcome.

Table II. Classification of diffuse injury from the Traumatic Coma Data Bank (TCDB)[27]

Grade	CT features
I	No visible intracranial pathology
II	Cisterns present with midline shift of 0 - 5 mm and/or no high- or mixed-density lesion greater than 25 cc
III	Cisterns compressed or absent with midline shift of 0 - 5 mm
IV	Midline shift greater than 5 mm
Evacuated mass lesion	Any lesion surgically evacuated
Non-evacuated mass lesion	High- or mixed-density lesion greater than 25 cc, not surgically evacuated

Loss of pupillary reactivity is also a strong indicator of outcome. From the TCDB data, 47% of patients who had a unilateral unreactive pupil and 82% of those with bilaterally unreactive pupils died or were in a vegetative state at discharge. Pupillary abnormalities also appear to be associated with compression of the basal cisterns, which is unsurprising given the significance of the perimesencephalic cisterns as a feature of transtentorial herniation.[28] Complete obliteration of the cisterns has been shown to be associated with unilateral or bilateral pupillary abnormalities in 75% of cases.[29] These authors also found that outcome in 96% of their cases could be predicted by a combination of CT findings (brain parenchymal lesions and state of mesencephalic cisterns) and clinical findings (motor score and pupil abnormalities).

Finally, the association of raised ICP and poor outcome has been well documented. Outcome is worse in patients with persistently raised ICP as well as those who suffer secondary neurological deterioration with raised ICP.

Taking these various factors into consideration, the favourable outcome in this cohort of patients is unexpected. All patients fulfilled the TCDB criteria for analysis. All met the criteria for grade III or grade IV swelling. Six patients had one unreactive pupil, 3 had bilaterally unreactive pupils, and 5 patients had a GCS of 4 or less prior to surgery. With the exception of one patient, each demonstrated a secondary deterioration in clinical condition or ICP control that prompted the decision to operate. The mean sustained ICP reduction achieved by craniectomy was 53.5% in patients who had both pre- and postoperative monitoring. There were no late rises in ICP or CT evidence of increasing oedema after surgery. Postoperative CT scans of all the patients demonstrated patency of the previously obliterated basal cisterns. The absence of severely impaired survivors argues against the potential criticism that aggressive therapy leads to poor quality survival.

Outcome in children compared with adults

Although it is often thought that outcome after TBI in children is better than in adults, reported data are not consistent on this issue. Important potential confounding factors are the inclusion of elderly patients in the adult group (a cohort of patients in which the mortality is known to be significantly higher) mechanism of injury (e.g. cerebral gunshot wounds in adults), and evaluation of outcome. The data from the largest published series in children reflects a surprisingly high mortality figure for children. Johnson and Krishnamurthy[30] reported the results of a survey of 5 level 1 trauma centres in the US and compared outcome after severe TBI of 4 041 children with that of 14 789 adults. The overall mortality rate for children in the study was 36.5%; no hospital reported a mortality rate less than 30% and for patients who were referred indirectly, i.e. via a local hospital, the mortality was as high as 50%. The authors contend that mortality rates between adults and children are similar if selection bias and confounding factors are excluded. In their comparison of childhood and adult mortality after TBI sustained in motor vehicle accidents, there was no significant difference.

Another feature of childhood head injury often quoted is the predominance of cerebral hyperaemia as a cause of brain swelling. On this basis it has been claimed that raised ICP in children responds better to medical therapy. Although earlier reports seem to suggest this, more recent data have not upheld this contention.[31] Finally, although brain plasticity is often considered a mechanism by which children may recover better than adults, this is potentially countered by the early vulnerability of the developing brain. The extent to which either of these factors is more significant in a particular individual may be impossible to determine.[32]

Interpretation of results

Although the outcome was favourable in this series of patients, these results cannot necessarily be extrapolated across the spectrum of head injury in children. With the exception of the one patient who died, all patients in this series were selected because the primary injury was thought to be salvageable, but had been compounded by the secondary contribution of raised ICP. This is key to understanding the effectiveness of the operation despite the poor preoperative condition. In these patients, uncontrolled raised ICP rather than the primary injury was considered to be the prime determinant of mortality. This situation should be clearly distinguished from patients who present with a devastating primary brain injury that is unlikely to be consistent with meaningful outcome. These patients often present with posturing and pupillary abnormalities at

the outset, CT signs of severe brain injury and initial elevated ICP. Depending on the circumstances it may be inappropriate to pursue aggressive strategies in this group. In the one patient who died, the severity of the primary injury was probably not consistent with meaningful survival, given the fact that the Glasgow Coma Score was 3T/15, both pupils were fixed and the brain was grossly swollen at the outset. In retrospect perhaps this patient should not have been treated aggressively. Equally, there is a population of head-injured patients with a less aggressive course of raised ICP in whom medical therapy is effective and who therefore do not require surgery. The decision regarding optimal treatment should take all of these factors into account.

There are a few limitations to this study that should be considered. First, because surgical treatment of raised ICP was not specifically compared with continued medical treatment it is impossible to categorically claim that decompressive craniectomy is superior to medical treatment, even in these highly selected patients. Unfortunately, randomisation of patients in this context is extremely difficult, where the difference in outcome may well be survival or death. Only one randomised trial of decompressive craniectomy has been done.[26] The authors of this study made a valiant attempt to randomise patients between surgical and medical therapy, but the study was limited by slow patient recruitment, small numbers and a very conservative operative strategy. Still, the authors reported reduced ICP in the surgically treated group and improved outcome. There is now strong support, even in the Brain Trauma Foundation Guidelines, for the feasibility of performing trials of decompressive operations in the first instance, as opposed to second- or third-tier therapy for post-traumatic intracranial hypertension.[33]

Another limitation of this study is the lack of preoperative ICP monitoring in all of the patients undergoing craniectomy. Although it can be questioned whether one can confidently diagnose raised ICP from clinical status and CT imaging, the diagnosis is upheld in this setting where all patients who had not been monitored had neurological deterioration with cisternal obliteration on CT prior to surgery which improved after craniectomy. The decision to operate on these patients as an emergency was made on the basis of the clinical and CT findings of cerebral herniation with the rationale that delay in effective ICP reduction would lead to death or severe neurological impairment.

In conclusion, we believe that aggressive treatment of secondary insults after brain trauma can have a dramatic impact on outcome. The control of ICP is critical and treatment must be individualised according to the clinical circumstances. In the appropriate setting, decompressive craniectomy can achieve dramatic control of ICP and result in surprisingly good outcomes, sometimes even in dire circumstances. A multi-centred randomised trial of decompressive craniectomy in adults is now under way, initiated by Cambridge University (**www.rescueICP.com**). Given the favourable pressure-volume index and the relatively higher incidence of diffuse injury in severe TBI, we would recommend a similar trial in children.

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Accepted 4 July 2006.