NTRACRANIAL hypertension that does not respond to maximal medical management occurs in 10 to 15% of patients with severe head injury and contributes to increased morbidity and mortality rates.\cite{34,45,46,73,76,77,79-83,88}\footnote{Abbreviations used in this paper: CI = confidence interval; CPP = cerebral perfusion pressure; CT = computerized tomography; GCS = Glasgow Coma Scale; GOS = Glasgow Outcome Scale; ICP = intracranial pressure; TBI = traumatic brain injury.} Miller, et al.,\cite{81} reported a 100% mortality rate when ICP above 20 mm Hg was uncontrollable by medical means. Among patients with ICPs greater than 40 mm Hg, Marshall and colleagues\cite{79} recorded a 42% mortality rate. A report published in 1982 by Saul and Ducker\cite{101} indicated an 84% death rate when ICP was greater than or equal to 25 mm Hg. In 1991 Marmarou, et al.,\cite{73} reported a significant relationship between outcome and the proportion of hourly ICP measurements greater than 20 mm Hg (p < 0.0001). Additionally, a post hoc analysis by Juul, et al.,\cite{56} of data from the international Selfotel trial revealed a mortality rate of 56.4% when intracranial hypertension, defined as an ICP of 20 mm Hg or greater, was noticed before and during neurological deterioration. In a stepwise regression analysis of multicenter hypothermia study data, Clifton, et al.,\cite{26} showed a significant relationship between an ICP greater than 25 mm Hg and a poor outcome.

Among patients with unilateral hemispheric or global brain swelling following TBI, intractable intracranial hypertension is an ominous sign. Despite maximal medical management, including pentobarbital coma in this group of patients, there is a high risk of morbidity and death.\cite{38,79} In a

Outcome following decompressive craniectomy for malignant swelling due to severe head injury

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*Department of Neurosurgery and R Adams Cowley Shock Trauma Center, University of Maryland School of Medicine, Baltimore, Maryland; and Mailman School of Public Health, Columbia University, New York, New York*

Object. The aim of this study was to assess outcome following decompressive craniectomy for malignant brain swelling due to closed traumatic brain injury (TBI).

Methods. During a 48-month period (March 2000–March 2004), 50 of 967 consecutive patients with closed TBI experienced diffuse brain swelling and underwent decompressive craniectomy, without removal of clots or contusion, to control intracranial pressure (ICP) or to reverse dangerous brain shifts. Diffuse injury was demonstrated in 44 patients, an evacuated mass lesion in four in whom decompressive craniectomy had been performed as a separate procedure, and a nonevacuated mass lesion in two. Decompressive craniectomy was performed urgently in 10 patients before ICP monitoring; in 40 patients the procedure was performed after ICP had become unresponsive to conventional medical management as outlined in the American Association of Neurological Surgeons guidelines. Survivors were followed up for at least 3 months posttreatment to determine their Glasgow Outcome Scale (GOS) score.

Decompressive craniectomy lowered ICP to less than 20 mm Hg in 85% of patients. In the 40 patients who underwent ICP monitoring before decompression, ICP decreased from a mean of 23.9 to 14.4 mm Hg (p < 0.001). Fourteen of 50 patients died, and 16 either remained in a vegetative state (seven patients) or were severely disabled (nine patients). Twenty patients had a good outcome (GOS Score 4–5). Among 30-day survivors, good outcome occurred in 17, 67, and 67% of patients with postresuscitation Glasgow Coma Scale scores of 3 to 5, 6 to 8, and 9 to 15, respectively (p < 0.05). Outcome was unaffected by abnormal pupillary response to light, timing of decompressive craniectomy, brain shift as demonstrated on computerized tomography scanning, and patient age, possibly because of the small number of patients in each of the subsets. Complications included hydrocephalus (five patients), hemorrhagic swelling ipsilateral to the craniectomy site (eight patients), and subdural hygroma (25 patients).

Conclusions. Decompressive craniectomy was associated with a better-than-expected functional outcome in patients with medically uncontrollable ICP and/or brain herniation, compared with outcomes in other control cohorts reported on in the literature.

**KEY WORDS** • severe head injury • traumatic brain injury • decompressive craniectomy • intracranial pressure • cerebral perfusion pressure • outcome

In 1991 Marmarou, et al.,\cite{79} reported a significant relationship between outcome and the proportion of hourly ICP measurements greater than 20 mm Hg (p < 0.0001). Additionally, a post hoc analysis by Juul, et al.,\cite{56} of data from the international Selfotel trial revealed a mortality rate of 56.4% when intracranial hypertension, defined as an ICP of 20 mm Hg or greater, was noticed before and during neurological deterioration. In a stepwise regression analysis of multicenter hypothermia study data, Clifton, et al.,\cite{26} showed a significant relationship between an ICP greater than 25 mm Hg and a poor outcome.

Among patients with unilateral hemispheric or global brain swelling following TBI, intractable intracranial hypertension is an ominous sign. Despite maximal medical management, including pentobarbital coma in this group of patients, there is a high risk of morbidity and death.\cite{38,79} In a
multicenter trial of high-dose pentobarbital for the control of intractable ICP, 86% of patients whose condition did not respond to pentobarbital died after other conventional medical therapy failed.\textsuperscript{34}

Given the near-total failure of researchers to define a novel drug or therapeutic maneuver to improve outcome\textsuperscript{11,16,33,36,67,68} as well as the problems with the safety and efficacy of major sedative and hypnotic agents (such as sodium pentobarbital and propofol) to medically manage malignant ICP\textsuperscript{20,21,34,105,120} decompressive craniectomy seems a reasonable alternative for treating intracranial hypertension with the hope of improving outcome following severe TBI complicated by diffuse swelling. Decompressive craniectomy reduces ICP in patients with TBI, but the long-term functional outcome following this procedure for the treatment of intractable hypertension (ICP > 20 mm Hg) remains unknown. Nonetheless, the literature on decompressive craniectomy indicates that during the past four decades, the incidence of a major disability or vegetative state has remained relatively constant, although there has been a gradual decrease in the mortality rate and an increase in the proportion of patients with a good outcome\textsuperscript{2,3,28,39,42,45,47,59,93,94,102,107,110,117,121,123}. Improvements in outcome may reflect the introduction of CT scanning, better prehospital care, earlier and more aggressive surgical and intensive care of patients with TBI, and application of the Guidelines for the Management of Severe Head Injury.\textsuperscript{5,18,22,46,55,56,78} New clinical research has provided evidence that decompressive craniectomy may improve O\textsubscript{2} delivery to brain cells when the incidence of cerebral ischemia is at its peak.\textsuperscript{54,109} Data from clinical and animal studies are inconclusive regarding whether normobaric hyperoxia is beneficial after TBI.\textsuperscript{1,113}

In this retrospective cohort study, we evaluated decompressive craniectomy for the treatment of 50 patients with severe closed TBI and brain swelling, and we report on the outcome following this surgical procedure.

**Clinical Material and Methods**

**Patient Population**

From March 2000 to March 2004, 967 patients with severe closed head injury (GCS Score 3–8) arrived at the University of Maryland Shock Trauma Center after a mean scene time of 59 minutes. One hundred four (10.7%) of these patients underwent decompressive craniectomy; 50 of these patients with malignant brain swelling are included in this outcome study. Among the 54 patients excluded from our analysis were 45 patients who had undergone prophylactic decompressive craniectomy during evacuation of a mass lesion and nine patients who had undergone resection in addition to planned decompressive craniectomy for swelling. Included in the study cohort were four patients who had undergone secondary decompressive craniectomy for intractable intracranial hypertension 3 to 6 days following evacuation of a mass lesion (two epidural hematomas, one subdural hematoma, and one contusion/intracerebral

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**Fig. 1.** Schematic showing the algorithm used in classifying 104 patients who had undergone decompressive craniectomy during a 4-year period. Among the patients included in this report were four with secondary decompression following a primary surgery for evacuation of an intracranial hematoma. Nine of 53 patients with diffuse swelling were excluded from our analysis because they had undergone lobectomy or contusionectomy at the time of decompressive craniectomy.
hematoma) and two patients with a nonevacuated hemorrhagic contusion (>25 ml) in an eloquent location (internal capsule and Wernicke area). The other patients in the cohort had no surgically treatable lesion, and the intractable ICP was primarily due to brain swelling. Patient selection was performed according to the design outlined in Fig. 1.

Prehospital Management

Prehospital management was completed according to the standards of the Maryland Institute for Emergency Medical Services Systems, which is compatible with the Brain Trauma Foundation Guideline for Prehospital Management of Traumatic Brain Injury.

On admission to the Trauma Resuscitation Unit the patients were examined and further treated according to the Advanced Trauma Life Support guidelines and the American Association of Neurological Surgeons/Congress of Neurological Surgeons Guidelines for the Management of Severe Head Injury.18

Demographic Data

The postresuscitation GCS score was 3 in one patient, 4 to 8 in 37, 9 to 12 in 10, and 13 to 15 in two patients. Among the 50 patients in the cohort who had undergone decompressive craniectomy, one half were 22 to 46 years old, and the majority were males (66%) who had incurred a TBI in a motor vehicle accident (80%). Only one patient was hypertensive, with a systolic blood pressure of 81 mm Hg initially and a corresponding O2 saturation of 100%. Three patients had low O2 saturation rates (83, 85, and 87%), but they did not have systolic hypotension (systolic blood pressure 140, 170, and 112 mm Hg; Table 1).

<table>
<thead>
<tr>
<th>TABLE 1</th>
<th>Summary of characteristics in 50 patients with severe closed head injury who underwent decompressive craniectomy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Variable</td>
<td>No. (%)</td>
</tr>
<tr>
<td>total no. of patients</td>
<td>50</td>
</tr>
<tr>
<td>mean age (yrs)</td>
<td>25.3</td>
</tr>
<tr>
<td>motor vehicle accident</td>
<td>40 (80)</td>
</tr>
<tr>
<td>male sex</td>
<td>33 (66)</td>
</tr>
<tr>
<td>median scene time (minutes)</td>
<td>59</td>
</tr>
<tr>
<td>admission GCS motor score (postresuscitation)</td>
<td>1–4: 29 (58): 5–6: 21 (42)</td>
</tr>
<tr>
<td>abnormal pupillary response to light</td>
<td>11 (22)</td>
</tr>
<tr>
<td>multiple trauma</td>
<td>40 (80)</td>
</tr>
<tr>
<td>mean head abbreviated injury score</td>
<td>mean 4.5</td>
</tr>
<tr>
<td>mean injury severity score</td>
<td>32.3 ± 10.4</td>
</tr>
<tr>
<td>&gt;5-mm shift on CT</td>
<td>13 (26)</td>
</tr>
<tr>
<td>compressed or obliterated basal cisterns</td>
<td>35 (70)</td>
</tr>
<tr>
<td>mean ICP before decompressive craniectomy (mm Hg)</td>
<td>23.9 ± 5.8</td>
</tr>
<tr>
<td>barbiturate coma</td>
<td>28 (56)</td>
</tr>
<tr>
<td>timing of decompressive craniectomy</td>
<td>w/in 48 hrs: 17 (34) 3–11 days: 33 (66)</td>
</tr>
<tr>
<td>follow up (mos)</td>
<td>mean 10.5 ± 7.8</td>
</tr>
<tr>
<td>range</td>
<td>3–43</td>
</tr>
</tbody>
</table>

Imaging Studies

Soon after resuscitation each patient underwent head CT scanning and his or her injury profile was classified according to National Institutes of Health Traumatic Coma Data Bank (Table 2).8

Initial Medical Management

During their initial medical treatment, 10 patients showed evidence of neurological worsening (a decrease of ≥ two points in the motor score of the GCS or an abnormal pupillary response to light) and underwent decompressive craniectomy within the first 24 hours of injury (nine went directly to the operating room on completion of the head CT study) without ICP monitoring.

Maximal Medical Management Prior to Decompression

Maximal medical management prior to decompressive craniectomy was provided according to the Guidelines for the Management of Severe Head Injury.18 A pulmonary artery or central venous catheter was inserted in each patient for central and pulmonary wedge pressure monitoring and fluid management (desired central venous pressure 12–15 cm H2O). Prior to decompressive craniectomy, an intraventricular cannula was inserted in 39 patients (a parenchymal device preceded the cannula in 25) and a parenchymal device alone was introduced in one patient (Camino; Integra Neurosciences, Plainsboro, NJ). Of the 10 patients who had undergone decompression within the first 24 hours of injury, nine went to the operating room directly and one went within 24 hours postinjury because of neurological worsening (change in pupillary response to light or a decrease of two points in the GCS motor score). Two of the nine patients transported to the operating room directly from the Trauma Resuscitation Unit underwent only a single ICP reading immediately before exploration (11 and 34 mm Hg). These data were excluded from our analysis. Postoperatively, 49 of 50 patients received an intraventricular cannula and one patient received a Camino device.

Medical management included intubation, ventilation, oxygenation (fraction of inspired O2 40–100%), head elevation (30–45°), fluid resuscitation, and sedation with either sedatives (short-acting benzodiazepines such as midazolam...

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[1–2 mg/hour], or propofol [5–10 µg/kg/min] or narcotics (morphine sulphate [1–2 mg/hour] or fentanyl [25–100 µg/hour]). Short-acting muscle relaxants, vecuronium (0.1 mg/kg/30 min) or pancuronium (0.04–1 mg/kg/hr), were used for brief periods only. External ventricular drainage, mild hyperventilation (PCO₂ 30–33 mm Hg), and osmotherapy (0.25 g/kg mannitol every 4–6 hours) or hypertonic saline (3% 250- to 500-ml boluses) up to a serum osmolality of 320 mOsm/kg H₂O were the next steps. Blood pressure was supported if necessary with dopamine (2–5 µg/kg/min and titrate), dobutamine (2.5–10 µg/kg/min and titrate), and Neosynephrine (100–180 µg/minute and titrate). Pento-barbital coma (10 mg/kg/hr for 4 hours, then 2–3 mg/kg/hr until burst suppression on electroencephalography) was induced in 28 patients, and in another 14 patients maximal sedation was induced using either sedatives (propofol [mean dosage 66.7 ± 16.7 µg/kg/min] or midazolam [1–2 mg intravenously every 15 minutes]) or narcotics (morphine [1–2 mg/hour] or fentanyl [25–100 µg/hour]). Hypothermia was not used therapeutically.

Technique and Timing of Decompressive Craniectomy

Decompressive craniectomy was performed in the frontoparietotemporal region in 49 patients and in the bifrontal region in one patient. The mean anteroposterior diameter of the bone flap was 15 cm, with extension into the temporal base. In each case, expansive duraplasty was performed using an allograft. Decompressive craniectomy was undertaken within 24 hours postinjury in 13 patients and during the 11 days postinjury in the others (Fig. 2). Bone flaps were stored in the tissue bank (< 70°C) from 3 to 6 months before reimplantation.

Patients were followed up from 3 to 43 months posttreatment (mean 10.4 months, median 8 months) and their GOS score was determined.

Statistical Analysis

Data were analyzed using statistical software (SAS Institute, Inc., Cary, NC). We used the Student t-test to compare continuous variables and the chi-square statistic to compare categorical variables.

Kaplan–Meier survival analysis was used to calculate the cumulative incidence of 30-day survival. Patients were followed up until death within the first 30 days after craniectomy or until 30 days after craniectomy. Cox proportional hazards regression was used to determine the effect of a variety of potential predictors on the risk of death in the first 30 days after decompressive craniectomy. Among 30-day survivors, we analyzed the effect of possible predictors of good outcome by using logistic regression. Good outcome was defined as a GOS score of 4 or 5.

Data collection was approved by the institutional review board at the University of Maryland, and data analysis was approved by the institutional review board at Columbia University.

Results

Intracranial Pressure and CPP

Among the 40 patients whose ICP was measured before decompressive craniectomy, the mean proportion of hour-
**Outcome after decompressive craniectomy for severe head injury**

**Table 3**

Predictors of death in the first 30 days after decompressive craniectomy in patients with diffuse injury due to TBI

<table>
<thead>
<tr>
<th>Variable</th>
<th>No. of Patients (% dead)</th>
<th>Crude Rate Ratio for Death</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>timing of DC</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>early</td>
<td>17 (23.5)</td>
<td>1.0 referent</td>
<td></td>
</tr>
<tr>
<td>late</td>
<td>33 (21.2)</td>
<td>0.9</td>
<td>0.3–3.0</td>
</tr>
<tr>
<td>brain shift before DC</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt; 5-mm shift</td>
<td>13 (7.7)</td>
<td>0.32</td>
<td>0.04–2.29</td>
</tr>
<tr>
<td>no significant shift</td>
<td>37 (27.0)</td>
<td>1.0 referent</td>
<td></td>
</tr>
<tr>
<td>admission GCS score</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3–5</td>
<td>15 (20.0)</td>
<td>1.0 referent</td>
<td></td>
</tr>
<tr>
<td>6–8</td>
<td>23 (21.7)</td>
<td>1.1</td>
<td>0.3–4.69</td>
</tr>
<tr>
<td>9–15</td>
<td>12 (25.0)</td>
<td>1.2</td>
<td>0.2–6.0</td>
</tr>
<tr>
<td>admission GCS motor score</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1–4</td>
<td>29 (24.1)</td>
<td>1.3</td>
<td>0.4–4.6</td>
</tr>
<tr>
<td>5–6</td>
<td>21 (19.0)</td>
<td>1.0 referent</td>
<td></td>
</tr>
<tr>
<td>age (yrs)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 20</td>
<td>17 (23.5)</td>
<td>1.2</td>
<td>0.3–4.0</td>
</tr>
<tr>
<td>20–49</td>
<td>33 (21.2)</td>
<td>1.0 referent</td>
<td></td>
</tr>
<tr>
<td>&gt; 50</td>
<td>0 (0.0)</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>abnormal pupillary response†</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>no</td>
<td>38 (26.7)</td>
<td>1.0 referent</td>
<td></td>
</tr>
<tr>
<td>yes</td>
<td>11 (18.2)</td>
<td>0.8</td>
<td>0.2–3.8</td>
</tr>
</tbody>
</table>

* DC = decompressive craniectomy; NA = not applicable.
† Information on pupillary response was missing in one patient.

**Table 4**

Predictors of good outcome in 30-day survivors of decompressive craniectomy in patients with diffuse injury due to TBI

<table>
<thead>
<tr>
<th>Variable</th>
<th>No. w/ Good Outcome (%)</th>
<th>Crude Odds Ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>timing of DC</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>early</td>
<td>13 (38.5)</td>
<td>1.0 referent</td>
<td></td>
</tr>
<tr>
<td>late</td>
<td>26 (57.7)</td>
<td>2.2</td>
<td>0.6–8.5</td>
</tr>
<tr>
<td>brain shift before DC</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt; 5-mm shift</td>
<td>12 (50.0)</td>
<td>0.9</td>
<td>0.2–3.6</td>
</tr>
<tr>
<td>no significant shift</td>
<td>27 (51.8)</td>
<td>1.0 referent</td>
<td></td>
</tr>
<tr>
<td>admission GCS score</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3–5</td>
<td>12 (16.7)</td>
<td>1.0 referent</td>
<td></td>
</tr>
<tr>
<td>6–8</td>
<td>18 (66.7)</td>
<td>10.0</td>
<td>1.6–60.9</td>
</tr>
<tr>
<td>9–15</td>
<td>9 (66.7)</td>
<td>10.0</td>
<td>1.2–78.1</td>
</tr>
<tr>
<td>admission GCS motor score</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1–4</td>
<td>22 (36.4)</td>
<td>4.2</td>
<td>1.1–16.3</td>
</tr>
<tr>
<td>5–6</td>
<td>17 (70.6)</td>
<td>1.0 referent</td>
<td></td>
</tr>
<tr>
<td>age (yrs)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 20</td>
<td>13 (61.5)</td>
<td>1.9</td>
<td>0.5–7.2</td>
</tr>
<tr>
<td>20–49</td>
<td>26 (46.1)</td>
<td>1.0 referent</td>
<td></td>
</tr>
<tr>
<td>≥ 50</td>
<td>0 (0.0)</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>abnormal pupillary response*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>no</td>
<td>29 (58.6)</td>
<td>1.0 referent</td>
<td></td>
</tr>
<tr>
<td>yes</td>
<td>9 (33.3)</td>
<td>0.3</td>
<td>0.1–1.7</td>
</tr>
</tbody>
</table>

* Information on pupillary response was missing in one patient.

The overall mortality rate was 28%; 11 patients died during the first 30 days after decompressive craniectomy and three died later (34 days after decompression, and 2 and 7 months after discharge from the hospital). The cumulative probability of survival in the first 30 days after decompression was 77.6% (Fig. 3).

We analyzed six established potential predictors of death during the first 30 days after decompressive craniectomy including patient age, admission GCS score, GCS motor score, abnormal pupillary response to light, shift in the midline structures, and timing of decompressive craniectomy; one of these variables was statistically significantly associated with death, possibly because of the small number of patients in each subset (Table 3).

**Glasgow Outcome Scale Score in 30-Day Survivors**

Among the 39 patients who survived the first 30 days after decompression and were followed up for at least 3 months posttreatment, 20 (51.3%) had a good outcome: 13 (65%) with GOS Score 4 and seven (35%) with GOS Score 5.

Several known predictors of good outcome were evaluated (Table 4). Patients with admission GCS scores of 6 to 15 were 10-fold more likely to have a good outcome compared with patients having admission GCS scores of 3 to 5. Similarly, patients having an admission GCS motor score of 5 or 6 were 4.2-fold more likely to have a good outcome compared with patients having a GCS motor score of 1 to 4 (95% CI 1.1–16.3). Abnormal pupillary response to light, timing of decompressive craniectomy, midline shift demonstrated on CT scanning, and patient age were not statistically significantly associated with outcome in this cohort.

**Complications Associated With Decompressive Craniectomy**

Subgaleal or subdural hygroma developed in 25 patients after a mean of 7.7 days following decompression and was usually located ipsilateral to the craniectomy site and rarely interhemispheric. The volume of the subdural hygroma fluid measured 10 to 120 ml (mean 51 ml) and was resorbed weeks to months later (Fig. 4).

One patient experienced delayed healing of the incision, and three had bone flap infections requiring removal and later synthetic material cranioplasty. Computerized tomography imaging demonstrated evidence of increased swelling.
Evidence for Decompressive Craniectomy

Pathophysiology of Posttraumatic Brain Edema. One of the consequences of TBI is an acute and precipitous increase in total brain water. Mechanoporation, ischemia, and energy failure at the level of mitochondria are some of the causes of cytotoxic and vasogenic brain edema and intracranial hypertension resistant to maximal medical management. Furthermore, increased carotid artery blood flow, indicating reduced cerebral vascular resistance. In the present study, there was no evidence of worsening cortical edema following cranial decompression. In the experimental studies of Burkert and Plaumann, there was enhanced brain tissue PO$_2$ following decompressive craniectomy.

Effect of Decompressive Craniectomy on ICP Dynamics, Cerebral Blood Flow, and Brain Tissue PO$_2$. The effectiveness of decompressive craniectomy in reducing ICP has been proven in all recently reported clinical investigations. Hase, et al.,$^{29,31,32,39,45,47,49,87,94,110,117,121}$ evaluated brain...
elasticity in 33 patients who had and in 14 patients who had not undergone decompressive craniectomy following severe head injury. In the present study, patients who had undergone decompressive craniectomy experienced improved compliance against increases in intracranial volume compared with patients with an intact skull. In Whitfield and colleagues' clinical study of 26 patients treated using decompressive craniectomy for brain swelling after severe TBI, the amplitude of ICP waves was significantly reduced (p < 0.02) and compensatory reserve was increased (p < 0.05). In five patients with severe head injury treated using decompressive craniectomy, Yamakami and Yamaura noted increased cerebral blood flow immediately underneath the area of decompression and related it to improved outcome. Other researchers have proven that surgical decompression of the brain significantly increases brain tissue PO₂, as measured using the Licox Device (Integra Neurosciences).

Outcome Following Decompressive Craniectomy

Once ICP is controlled by decompressive craniectomy, outcome improves compared with that in cases in which ICP was not controlled. In a series of 35 patients with diffuse injury treated with bifrontal decompressive craniectomy, Polin, et al., reported a mortality rate of 23% and a favorable outcome rate in 37% of patients; this group was compared with a control group (92 patients) formed of patients whose data were accrued in the Traumatic Coma Data Bank and who had a 30.8% mortality rate and a favorable outcome rate of 15.4%. In a randomized trial of bifrontal decompressive craniectomy in 27 pediatric patients with closed head injury and diffuse swelling, three of 13 patients in the decompression arm died and seven had a good outcome. In the control group, six of 14 patients died and only two patients made a good recovery. In a series of 115 patients with severe closed head injury, 27 did not respond to medical treatment; decompressive craniectomy was performed in three patients and a pentobarbital coma was induced in 24. Seventeen of 24 patients in the pentobarbital coma did not respond to this treatment and 82.4% of these patients died. In 10 patients in whom maximal medical treatment had failed (seven in a pentobarbital coma) and in whom subtemporal decompressive craniectomy had been performed, the mortality rate was 40%. According to reports published since 1988 (including the University of Maryland series), 323 patients have undergone decompressive craniectomy for posttraumatic brain swelling and intractable intracranial hypertension. The collective mortality rate in this group of patients was 22.3%, whereas the proportion of patients with a good outcome was 48.3%; 29.4% of patients remained severely disabled or in a vegetative state (Table 5).

Table 5: Outcome following decompressive craniectomy for posttraumatic brain swelling

<table>
<thead>
<tr>
<th>Authors &amp; Year</th>
<th>No. of Patients</th>
<th>GOS Score 4–5</th>
<th>GOS Score 2–3</th>
<th>GOS Score 1</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gower, et al., 1988</td>
<td>10</td>
<td>4</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>Gaab, et al., 1990</td>
<td>37</td>
<td>29</td>
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<td>Polin, et al., 1997</td>
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<td>Guerra, et al., 1999</td>
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<td>De Luca, et al., 2000</td>
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<td>Taylor, et al., 2001</td>
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<td>Whitfield, et al., 2001</td>
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<td>Schneider, et al., 2002</td>
<td>62</td>
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<td>30</td>
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<td>Albanese, et al., 2003</td>
<td>13</td>
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<tr>
<td>present study</td>
<td>50</td>
<td>20</td>
<td>16</td>
<td>14</td>
</tr>
<tr>
<td>total (%)</td>
<td>323 (100)</td>
<td>156 (48.3)</td>
<td>95 (29.4)</td>
<td>72 (22.3)</td>
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These results compare favorably with those reported by Miller, et al., Marshall, et al., Saul and Ducker, Juul, et al., Marmarou, et al., and Clifton, et al., who reported mortality rates of 42 to 100% when an ICP greater than 20 mm Hg was not controlled despite maximal medical management.
Complications of Decompressive Craniectomy

Once intraoperative prophylactic antibiotic agents are administered, the decompressive craniectomy procedure itself should not enhance susceptibility to superficial or deep wound infections more than the 3 to 5% incidence reported in the neurosurgical literature.9,41,63,84,116 Although expansive duraplasty with a dural substitute has reportedly been associated with an increased chance of infection,19 we noted no such increase in infection. Cranioplasty with frozen bone implant also has an associated risk of infection.20,97,112 In two of 36 patients who had survived and undergone cranioplasty a mean of 4 months after decompression, bone flap infection occurred and the infected autologous bone required removal.

Although a disturbing finding on CT studies, the pathogenesis and significance of subdural or subgaleal hygroma following decompressive craniectomy has not been well researched. Subdural or subgaleal hygromas, which have been found as early as within days of decompression and may resorb several months thereafter, seem to have the same course of appearance and resolution as those seen in patients with severe head injury who have not undergone decompressive craniectomy.3,10,37,55,62,68 Two of the 25 patients with hygroma in our study experienced spontaneous hemorrhage inside the hygroma cavity followed by subsequent resolution.66 None of the patients needed surgical evacuation or insertion of a shunt device, and only two of 25 suffered delayed hydrocephalus (Fig. 4). Although traumatic rupture of the arachnoid trabeculae and transient dynamic changes of cerebrospinal fluid circulation seem to be a logical explanation, some authors have related hygromas to increased CPP.62

Enhancement of brain edema and at times fresh hemorrhages were noted ipsilateral to the site of decompression in eight of the patients in our study. In 1979 Cooper, et al.,27,28 noted enhanced cerebral edema on cranial CT scans obtained following decompressive craniectomy for severe head injury. These investigators studied cold-induced brain edema in response to decompressive craniectomy in dogs and found a sevenfold accentuation in edema formation as indicated by the extravasations of Evans blue in the interstitial space of the brain.27 The authors proposed that an imbalance in intravascular and interstitial pressures produced by bone removal may be one reason for intensified edema. In Rinaldi and colleagues’38 experimental studies, decompressive craniectomy following cold-induced brain lesions in rabbits did not enhance brain edema, according to Evans blue. Gaab and associates’38 study of decompressive craniectomy in cats indicated interruption of experimental cold-induced brain edema following resection of the necrotic brain. In our study, three of eight patients with increased brain edema following decompressive craniectomy died; one was left with severe disability and four had a good recovery (Fig. 5).

Revascularization and remodeling of a frozen bone flap following autologous cranioplasty may not be predictable due to bone resorption. Depending on the duration of follow-up examinations and the frequency of CT studies, bone resorption may be noticed in up to 50% of patients.5,46,52,91 In our series, three of six cases of bone flap resorption required repeated surgery because of disfigurement and unusual cracking sounds (Fig. 6). New therapies may modulate osteoclast-mediated bone resorption and decrease the chance of multiple surgical interventions in these patients.114

Conclusions

Studies of patients with TBI indicate increased morbidity and mortality rates when intracranial hypertension is not medically manageable. Although the primary effects of severe head injury are not easily mitigated by currently available pharmaceutical agents, increased ICP can be interrupted by decompressive craniectomy, therefore alleviating some of the events leading to secondary cerebral insults. Past experience with preventive decompressive craniectomy has been disappointing. Nonetheless, data from the last seven series on decompressive craniectomy for intractable intracranial hypertension as a consequence of brain swelling have indicated an improved outcome compared with outcome following conservative management. In the present study, a reduced mortality rate is parallel with the increased proportion of patients with a good outcome. Furthermore, in patients with severe head injury and brain swelling, those with an admission GCS score greater than 6 are especially good candidates for decompressive craniectomy.

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