Surgical decompression for traumatic brain swelling: indications and results

Waltraud Kleist-Welch Guerra, M.D., Michael R. Gaab, M.D., Ph.D., Hermann Dietz, M.D., Ph.D., Jan-Uwe Mueller, M.D., Jürgen Piek, M.D., Ph.D., and Michael J. Fritsch, M.D.

Department of Neurosurgery, Ernst Moritz Arndt University, Greifswald; and Department of Neurosurgery, Medizinische Hochschule, Hannover, Germany

Object. Decompressive craniectomy has been performed since 1977 in 57 patients with traumatic brain injury. The authors assess the efficacy of this treatment and the indications for its use.

Methods. The clinical status of the patients, their computerized tomography (CT) scans, and intracranial pressure (ICP) levels were documented prospectively in a standard protocol. At the beginning of the study, all patients older than 30 years were excluded. As of 1989 patients older than 40 years were excluded until 1991; since that time patients older than 50 years have been excluded. Primary brain or brainstem injury with fully developed bulbar brain syndrome, loss of auditory evoked potentials (AEPs), and/or oscillation flow in a transcranial Doppler ultrasound examination were contraindications to decompressive craniectomy. A positive indication for decompression was given in the case of progressive therapy-resistant intracranial hypertension in correlation with clinical (Glasgow Coma Scale [GCS] score, decerebrate posturing, dilating of pupils) and electrophysiological (electroencephalography, somatosensory evoked potentials, and AEPs) parameters and with findings on CT scans. Unilateral decompressive craniectomy was performed in 31 patients and bilateral craniectomy in 26 patients. In all cases, a wide frontotemporoparietal craniectomy was followed by a dura enlargement covered with temporal muscle fascia.

The outcomes of the treatment were surprisingly good. Only 11 patients (19%) died, three of whom died of acute respiratory disease syndrome. Five patients (9%) survived, but remained in a persistent vegetative state; six patients (11%) survived with a severe permanent neurological deficit, and 33 patients (58%) attained social rehabilitation. Two patients (3.5%) did not have a follow-up examination. The GCS score on the 1st day posttrauma and the mean ICP turned out to be the best predictors for a good prognosis. The results demonstrate the importance of decompressive craniectomy in the treatment of traumatic brain swelling.

Conclusions. Surgical decompression should be routinely performed when indicated before irreversible ischemic brain damage.

Key Words * brain trauma * brain edema * decompressive craniectomy * decompression surgery * intracranial pressure * B waves
Posttraumatic brain edema leading to refractory intracranial hypertension is the main prognostic factor in brain-injured patients,[51,57,62,64,67,69] despite progress in the diagnosis and therapy of brain injury.[3,46,64] Our own preliminary clinical results[21] were encouraging and recommended the controversial decompressive craniectomy, promoted for the first time by Kocher[39] in 1901. However, this rarely performed surgical procedure lacks a clearly defined indication and is considered to be one of several "second-tier therapies" in refractory intracranial hypertension by the American Association of Neurological Surgeons (AANS). A review of the literature published to date does not reveal a definitive answer to the question regarding the effectiveness of decompressive craniectomy.[13,25,36,49,59,66] To assess this, we began a prospective study with preliminary criteria (exclusion of patients older than 30 years of age and patients with devastating primary brain damage [no initial or early bulbar brain symptoms]) and a standard protocol.[21] In this article, we provide our analysis of the clinical data and we present the indications for decompressive craniectomy in patients with posttraumatic brain edema.

**CLINICAL MATERIAL AND METHODS**

**Patient Population**

From 1977 to 1997, 57 patients underwent decompressive craniectomy. Computerized tomography (CT) scans obtained in these patients revealed diffuse brain swelling that was either unilateral (31 patients) or bilateral (26 patients). As shown in Table 1, two patient subgroups were formed. The first group included 39 patients without focal lesions, in whom massive posttraumatic brain edema (uni- or bilateral) and therapy-resistant intracranial hypertension with clinical deterioration required decompressive craniectomy. The second group included 18 patients who had a space-occupying hematoma (contusion hemorrhage, epidural hematoma, or subdural hematoma) that had been removed in an initial surgical procedure. Massive reactive brain edema developed afterward in these patients and decompressive craniectomy was the second surgical procedure.

**Clinical Standard Management**

Forty patients received continuous epidural or intraventricular monitoring of intracranial pressure (ICP). The duration of ICP monitoring was 151.3 hours on average (range 21-557 hours, mean 6.3 days). Seventeen patients did not undergo placement of an ICP monitor. The patients in the latter group

```
<table>
<thead>
<tr>
<th></th>
<th>Unfavorable Outcome (GOS Score 1-3)</th>
<th>Favorable Outcome (GOS Score 4-5)</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1</td>
<td>16 (42%)</td>
<td>22 (58%)</td>
<td>38</td>
</tr>
<tr>
<td>Group 2</td>
<td>6 (35%)</td>
<td>11 (65%)</td>
<td>17</td>
</tr>
<tr>
<td>Total</td>
<td>22</td>
<td>33</td>
<td>55</td>
</tr>
</tbody>
</table>
```

* Group 1 was composed of patients who underwent primary decompression for swelling edema. Group 2 was composed of patients who underwent secondary decompression for diffuse or unilateral edema swelling or refractory ICP after already having undergone previous surgery. Two patients (one from each group) had no follow-up examination and are excluded from the table. There was no difference between groups according to the chi-square test ($\chi^2 = 0.227 < 3.84$ (for $\alpha = 0.05$)).
demonstrated rapid clinical deterioration immediately after admission (including patients who had been transported from other hospitals) and the CT scans obtained in these patients revealed massive brain edema and initial herniation (loss of subarachnoid space in the basal cisterns and a supratentorial, midline shift) that required urgent surgical intervention. From the inception of the study, conservative treatment followed a standardized protocol and included the following measures: hemodynamic stabilization; elevation of the head to a maximum of 30°; sedation with analgesic medication and, if indicated, muscle relaxation; artificial ventilation with controlled hyperventilation (PaCO₂ 28-32 mm Hg); osmotherapy with mannitol (0.25-1 g/kg administered intravenously 3-6 times a day); administration of tromethamine (Tris-buffer) in response to an acute rise in ICP (18 g/2 hours, depending on base excess); a course of barbiturate medications (thiopental or pentobarbital) until burst suppression electroencephalography (EEG).

Additional Tests

Since 1985, monitoring of somatosensory evoked potentials (SEPs) and auditory evoked potentials (AEPs) has been used for the evaluation of primary damage and a secondary rise in ICP. A reduction in SEPs to less than 50%, an increase in central conduction time, and differences in latency between neck potentials and the cortical primary complex measuring more than 9 msec in the presence of brainstem potentials (AEPs) indicated the need for immediate surgical decompression (see Fig. 5).

Starting in 1987, transcranial Doppler (TCD) ultrasonography for the evaluation of middle cerebral artery flow velocities has become an additional diagnostic measure (an increase in ICP is associated with an increase of the pulsatility index in TCD ultrasonography).[21]

Patients and Indications

Although the age limits of patients eligible to be enrolled in this study increased, the indications for decompressive craniectomy did not change during the whole study and included: the appearance of diffuse unilateral or bilateral brain swelling on the CT scan with correlating clinical deterioration; worsening of Glasgow Coma Scale (GCS)[63] score and/or dilation of pupils unresponsive to light; therapy-resistant increase in ICP to more than 30 mm Hg and/or a reduction in cerebral perfusion pressure (CPP) to less than 45 mm Hg; initial GCS score of 4 or higher and a GCS score of at least 4 on the 1st posttraumatic day. Patients with primary fatal brainstem damage, that is, an initial and persisting GCS score of 3 and/or bilaterally fixed and dilated pupils, did not undergo surgical decompression.

The differential indication between hemicraniectomy or bilateral decompression (frontoparietotemporal surgery) was given as follows: unilateral edema/swelling was treated by hemicraniectomy over the swollen hemisphere and bilateral diffuse edema/swelling was treated by bilateral decompression.

From 1977 to 1988, patients older than 30 years of age were excluded from the study. Because of the encouraging results, as of 1989 we changed the exclusion age to over 40 years, and as of 1991 we again changed the age to over 50 years. Two patients, aged 55 and 66 years old, were treated with decompressive craniectomy as an exception.

Surgical Technique

The procedure begins with either a large bifrontal skin incision or a unilateral/bilateral curvilinear incision in the frontotemporoparietal region. Subsequently, preparation of a myocutaneous flap and craniectomy with elevation of a free frontotemporoparietal bone flap is performed (Fig. 1). In bilateral
craniectomy, only a rim of bone remains on top of the superior sagittal sinus to avoid ligating the falx and sinus, which may contribute to complications. Thereafter, the dura is opened and, starting at the temporal base of the dura opening, the dura is enlarged with the temporal fascia in a "dovetail" manner, followed by watertight closure of the dura and a fascial graft. At the beginning of our study the bone flap was preserved by implantation into abdominal fat; since 1995, the bone has been stored under sterile conditions at -80°C. Reimplantation occurs 6 weeks to 3 months postsurgery.

Fig. 1. Computerized tomography scan demonstrating the extent of the craniectomy.

Data Collection

The following patient data were prospectively collected in a standard protocol: age of the patient, date of the accident, and mechanism of the injury; neurological status (GCS score and pupillary response) on the day of injury, on the 1st day posttrauma, and prior to decompression; ICP and CPP (these were initially recorded on paper; since 1986 data have been collected on-line and the maximum and mean values have been analyzed either off- or on-line[22]); data on conservative ICP therapy; the surgical procedure, complications, and date of bone flap reimplantation; duration of postoperative coma; and outcome (this was scored by using the Glasgow Outcome Scale [GOS];[33] the minimum follow-up duration was 12 months. A GOS score of good recovery [5] or moderate disability [4] was considered to be a "favorable" outcome). Only two patients were lost to follow-up review.

Statistical Analysis

The independent contribution of predictive factors to outcome was studied using logistic regression analysis (SPSS, version 6). Patient outcome was used as the dichotomous (GOS score 1-3 or 4-5) dependent variable. The analyses were made by a backward stepwise selection, which first fitted the model to the total group of variables and then eliminated the variables one by one on the basis of likelihood statistics, leaving the most important independent prognostic factors (a value of p = 0.1 was used for variable exclusion).

The two groups (1, only posttraumatic edema and 2, hemorrhage with secondary edema) did not show
significant differences with regard to outcome after severe head injury (chi-square test). Therefore, for overall statistical analysis, all patients were regarded as one homogeneous group. Student's t-test was used for analysis of patients with B-wave loss in ICP measurements. A probability level of less than 0.05 was deemed significant.

RESULTS

General Results

Following the clear criteria mentioned earlier, a surgical decompression was only indicated in 57 patients during the past 20 years or an average of three patients per year. With an estimated incidence of at least 100 patients per year treated at our institution for severe traumatic brain injury, the incidence of decompressive craniectomy is less than 3%. The patients' ages, genders (Fig. 2), and mechanisms of injury were distributed as expected.

![Fig. 2. Bar graph showing the distribution of patients' ages and gender. See Patients and Indications for details on changing age limits.](image)

Neurological Recovery and Prognostic Factors

Patient Age. The patients' clinical status at the most recent physical examination and the mean patient age in each category of outcome are shown in Table 2. Logistic regression analysis did not indicate age as a predictive factor to outcome. However, two patients older than 50 years of age had a poor outcome. The group of patients older than 40 years of age was small.

<table>
<thead>
<tr>
<th>GOS Score</th>
<th>No. of Cases (%)</th>
<th>Mean Age (yrs)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 (dead)</td>
<td>11 (19)</td>
<td>27.9</td>
</tr>
<tr>
<td>2 (vegetative)</td>
<td>5 (9)</td>
<td>24.8</td>
</tr>
<tr>
<td>3 (severely disabled)</td>
<td>6 (11)</td>
<td>24.7</td>
</tr>
<tr>
<td>4 (moderate disability &amp; social integration)</td>
<td>12 (21)</td>
<td>24.8</td>
</tr>
<tr>
<td>5 (no disability &amp; professional reintegration)</td>
<td>21 (37)</td>
<td>18.7</td>
</tr>
</tbody>
</table>

* There were 57 patients in this study, two (3.5%) of whom survived but did not undergo follow-up examination.
Clinical Status. Both initial GCS score and predecompressive GCS score were not independent predictors of outcome. Seven patients had an initial GCS score of 3 but no pupillary dysfunction. According to the inclusion criteria for the study, all patients had to improve to a GCS score of at least 4 before surgical decompression was performed. One patient improved to a GCS score of 6 on the 1st day, deteriorated to a GCS score of 4, but recovered completely after decompression at this stage. The other six patients with an initial GCS score of 3 improved to a GCS score of 4 preoperatively, but had a poor outcome (one patient showed severe neurological deficits, three patients remained in a vegetative state, and two patients died).

The final model derived from the logistic regression analysis explains 75% of the whole outcome. Only two variables, GCS score on the 1st day posttrauma (p = 0.0276) and the mean ICP (p = 0.0614) are predictive of outcome, although for the mean ICP this was not statistically significant. Table 3 shows the relationship between GCS score on the 1st day posttrauma and the corresponding mean ICP. From correlation between the two values, the prognosis can be estimated mathematically. For example if the GCS score on the 1st day posttrauma was 5 and the mean ICP was higher than 57 mm Hg, surgical decompression was less promising, because the likelihood for a good outcome was approximately 10%.

<table>
<thead>
<tr>
<th>GCS Score on 1st Day Posttrauma</th>
<th>Critical Mean ICP (mm Hg)</th>
<th>90% Confidence</th>
<th>50% Confidence</th>
<th>10% Confidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>4</td>
<td>8</td>
<td>29</td>
<td>50</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>14</td>
<td>36</td>
<td>57</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>21</td>
<td>42</td>
<td>63</td>
<td></td>
</tr>
</tbody>
</table>

The time span between the accident and surgical decompression was 12 hours to 18 days. According to this time interval, the dynamics of clinical deterioration varied (difference between GCS score on the 1st day and preoperatively). The average time of surgery was 4 days postaccident. Ten patients underwent surgery within 48 hours (four of these patients within 24 hours). The majority of patients with slow deterioration (> 2 days) had a favorable outcome. Within the group of patients who had been in a stable but poor clinical condition and demonstrated deterioration in ICP values, there was no difference in outcome. Patients who deteriorated in less than 48 hours did not show a difference regarding outcome (Fig. 3).
Fig. 3. Bar graph displaying the patients' neurological recovery with correlation to elapsed time until clinical deterioration. There was better outcome with slow deterioration.

**Intracranial Pressure.** The relationship between GCS score and mean ICP on the 1st day posttrauma in terms of outcome has already been described. An increase in ICP lasting 5 to 30 minutes or lasting 30 minutes to 2 hours was not found to be a significant prognostic factor. The phenomenon of the loss of the B waves, however, was of obvious importance to the prognosis. Patients with a good outcome (GOS Score 4 or 5) did not demonstrate a loss of B waves. Although the study includes only 12 patients who lost B-wave activity, there is a significant difference between patients with a good outcome and patients with a GOS score of 3 or less. Moreover, the loss of plateau waves seems to correlate with a poor prognosis. Because of the small number of patients, further differentiation was not possible (Table 4 and Fig. 4).

<table>
<thead>
<tr>
<th>TABLE 4</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Correlation of GOS score with plateau waves and B waves</strong></td>
</tr>
<tr>
<td>GOS Score</td>
</tr>
<tr>
<td>----------</td>
</tr>
<tr>
<td>B waves present</td>
</tr>
<tr>
<td>plateau waves registered</td>
</tr>
</tbody>
</table>

* According to Student's t-test.
Fig. 4. Tracings. Left: Typical example of preserved B-wave activity before surgical decompression in a patient who later had a GOS score of 5. Right: Minimal B-wave activity in a patient with a GCS Score of 3 who later died. The upper line represents mean ICP and the lower line B-wave activity.

**Postoperative Complications.** The complication rate after decompressive craniectomy was low and had no influence on the patient's prognosis. One patient (2%) had a wound infection after reimplantation of the bone flap. Four patients (7%) had postoperative seizures, which were related to the trauma rather than to surgery. Three (5%) of the patients developed persistent seizure activity that required medical treatment. Hygromas were the most frequent complication (15 patients [26%]). Puncture was found to be sufficient for the treatment of the hygroma in 10 (67%) of these patients. Five patients (33%) required placement of a ventriculo- or lumboperitoneal shunt. Eight patients (14%) developed hydrocephalus. There was no death or permanent deficits related to the surgical decompression itself.

**ILLUSTRATIVE CASES**

**Case 1**

This 21-year-old woman was involved in a motorcycle accident in Nigeria and sustained severe closed head injury. Three days after the initial medical treatment, the patient was transferred to Germany. On arrival at our department, the patient was unconscious and showed bilateral flexion responses to pain stimuli. A CT scan obtained in the patient demonstrated moderate generalized brain swelling with a small contusion in the left frontal lobe. Implantation of an epidural pressure monitor was performed. On the 5th day posttrauma, the patient's ICP started to rise and the short-term maximum values were measured at 70 mm Hg. The patient received mannitol, tromethamine, and barbiturate medications. Three days later her ICP rose out of control. The patient demonstrated intermittent mydriasis and decorticate posturing. Brainstem evoked potentials (BAEPs I-V) and SEPs were present, but demonstrated an increase in latency and a decrease in amplitude (Fig. 5). The CT scan obtained at this time showed an unchanged left frontal contusion but diffuse brain swelling and compression of the ventricles. A bilateral frontotemporoparietal decompressive craniectomy with a dura patch was performed. Postoperatively, the patient's ICP decreased to 10 to 20 mm Hg without any further critical increase. The patient recovered. After 1 year, the woman demonstrated hyperreflexia in the left upper extremity and discrete slowing in speech. Two years later, the patient was reintegrated into her professional career.
Case 1

This 7-year-old boy suffered a severe closed head brain injury and presented with dilated pupils and decorticate posturing. The following day, the child demonstrated decerebrate posturing. A CT scan obtained in the child revealed a left temporal fracture with an underlying rim of subdural hematoma and diffuse generalized brain edema, more pronounced on the left than on the right side. An epidural pressure...
transducer was implanted. The patient's BAEPs were diminished, but TCD ultrasonography
demonstrated sufficient blood flow. Despite maximum conservative therapy, the boy's ICP increased to
60 mm Hg. After 24 hours, a bilateral frontotemporoparietal decompressive craniectomy with a dura
patch was performed. Postoperatively, the patient's pupils remained dilated and fixed. After an initial
decrease in ICP to 25 mm Hg, a secondary rise up to 52 mm Hg was temporally seen on the 1st day after
decompression. The patient remained in a persistent vegetative state (GOS Score 2).

DISCUSSION

Posttraumatic brain swelling represents a pathophysiological entity that is not yet fully understood.
Different components, such as vasogenic and cytotoxic edema and, quite possibly, cerebral
vasocongestion, contribute to this phenomenon.[3] If conventional therapy fails in patients suffering from
decompensated intracranial hypertension, there is only a small number of second tier options left:
barbiturate coma, hypothermia, hyperventilation, or decompressive craniectomy. If the patient continues
to deteriorate under this treatment, the prognosis is fatal and further therapeutic efforts are in vain. This is
in agreement with European (European Brain Injury Consortium, 1995) and American (AANS, 1996)
guidelines for treatment of intracranial hypertension. The guidelines established by the AANS name
decompressive craniectomy as the last of the second-tier therapeutic options. However, among
second-tier measures, decompressive craniectomy leads to the fastest relief by immediate reduction of
intracranial hypertension and has the lowest rate of complications. Moreover, the procedure is simple and
safe. In our opinion, decompressive craniectomy should therefore be the first among second-tier
therapeutic options rather than the last one.

Comparison of Decompression and Barbiturate Coma

Barbiturate coma leads to a reduction in ICP under certain circumstances.[55] Most authors favor
barbiturate coma over decompressive craniectomy. The application of high doses of barbiturates,
however, is associated with several side effects. Reduction of mean arterial blood pressure may lead to a
reduction in CPP. Brainstem reflexes and EEG findings are diminished or completely suppressed,
especially in children, and it may take days before these effects wear off.[37] Additional side effects
include: respiratory complications, myocardial depression with reduction in heart volume per minute,
increased infection rate, disturbances in electrolyte balance, impairment of liver function, and leukocyte
depression.[56,69] The mortality rate in patients treated with highly doses of barbiturate medications for
raised ICP is extremely high: Gower, et al.,[25] published a rate of 82% and Piek[50] a rate of 58%.
Compared with these, decompressive craniectomy, as a second tier therapy, seems to result in a much
better outcome; the mortality rate in our group was only 19%. Surgical decompression should be
performed before inducing deep barbiturate coma in these patients; a surgical decompression performed
after failure of ultrahigh barbiturates is too late and would at least include the complication rate of this
medical treatment.

Comparison of Decompression and Hyperventilation

Forced hyperventilation as a therapy for intracranial hypertension is obsolete. Patients with increased ICP
and preexisting ischemia are at risk from hyperventilation, because hypocapnic vasoconstriction worsens
ischemic lesions.[67] We do not induce hyperventilation to a PaCO₂ of less than 28 mm Hg.

Comparison of Decompression and Hypothermia
Besides experimental studies, there are only initial clinical reports on the application of mild hypothermia in patients with severe brain injury.[8,44,60] Side effects of this therapy are "after-drop," irregular pharmacokinetics, disturbances in the monitoring of vital parameters, impairment of coagulation, and immunosuppression.[5,8,45,60] There are remaining questions concerning this therapy. What is the optimum brain temperature for maximum neuroprotection and minimum side effect? How long should this therapy last? When is the right time to start rewarming the patient, and how long should it take?[62] The influence of hypothermia-related neuroprotection during decompressive craniectomy was discussed by Doerfler and associates.[15] Further studies are necessary on this topic. Because surgical decompression produces no side effects, we recommend performing this procedure first and including hypothermia during or after surgical intervention, if at all required.

**Intracranial Pressure After Decompressive Craniectomy: Clinical and Experimental Studies**

Intracranial mass effect can be compensated without an increase in ICP by resorption of cerebrospinal fluid (CSF) and by shifting CSF into the spinal canal. When the reserve spaces become completely exhausted, mass effect leads to an exponential increase in ICP. The equation is expressed by the pressure-volume diagram. Results from Hase and associates,[26] as well as our own data,[19] provide evidence that decompression leads to a shift to the right of the pressure-volume curve and, therefore, to a massive increase in compliance. Polin, et al.,[52] also demonstrated a statistically significant reduction in postoperative ICP values after decompression. Dam Hieu and colleagues[13] showed that craniectomy performed in two children to treat uncontrollable intracranial hypertension led to a reduction in ICP by 30% and 45%. Opening of the dura and, in one case, a partial temporal lobectomy caused further reduction in the ICP. Jourdan, et al.,[34] reported on a series of nine patients with intracranial hypertension in whom craniectomy resulted in an ICP reduction of 15%, but opening of the dura achieved a reduction of 70%. Gower, et al.,[25] were able to reduce ICP by 34% in seven patients by performing a subtemporal decompressive craniectomy. Experimental studies confirm the results of the clinical reports. Our own experimental studies proved decompressive craniectomy to be the most effective measure in terms of reduction of ICP, survival rate, and normalization of EEG after freezing lesion injuries.[20] Moody and colleagues[47] used an epidural balloon in a rabbit model and achieved a 100% mortality rate. Using decompressive craniectomy, that mortality rate was reduced to 70%. After induction of experimental ultrasound trauma in anesthetized dogs, Burkert and Plaumann[6] performed decompressive craniectomy and dura opening, and found a rapid decrease in ICP after surgery. Rinaldi, et al.,[54] investigated the hemodynamic effects of decompression with dura opening in rabbits with cold injuries and brain edema. They were able to demonstrate a dramatic decrease in ICP without any alteration in the blood-brain barrier and normalization of cerebral blood flow velocities after decompression. Cooper, et al.,[9] reported a reduction in ICP in experimental cold injury but also observed a clear increase in brain edema after decompression. Hatashita and Hoff[27] showed that decompressive craniectomy associated with arterial hypertension may lead to significant brain edema in cats after experimental brain injury. Studies by the same authors in nontraumatized cats with craniectomies showed a significant reduction in ICP with an increase in compliance in comparison with normal control animals.[28] The increase in brain edema after decompressive craniectomy led to a discussion in the neurosurgical literature and to questioning the usefulness of the procedure when treating severely brain-injured patients. According to our clinical experience, brain edema only increases if the brain is already irreversibly severely damaged. Such patients have a poor prognosis, which is no argument against decompressive craniectomy but against the erroneous indication (decompression applied to late, fatal, primary brain injury).
Yamakami and Yamaura[70] investigated the effect of decompressive craniectomy on regional blood flow in contusions using single-photon emission CT (SPECT) in five patients with severe brain injury. The patients underwent craniectomy when ICP could not be controlled by medical therapy. Within 24 hours of decompression, the SPECT analysis showed a perfusion deficit in all patients that correlated with the contusions. In the region of brain decompression, an area of mild hyperperfusion was seen at the same time. One week after decompression, the region of hyperperfusion had increased in size and intensity. A CT scan obtained at 1 week revealed massive brain swelling in the decompression area. One month after decompression, no hyperperfusion remained and a CT scan obtained at that time did not show any brain swelling. Patients showed significant clinical improvement within this period of time. Even within the 1st postoperative week, while brain swelling was at its maximum, there was no negative effect on the neurological status of the patients. The authors discuss that an increase in cerebral blood flow after decompression causes congestion in the decompressed region and does not, as hypothesized by Cooper, et al.[9] parallel the development of acute brain edema. The focal increase in blood flow in the decompression area may protect the brain from secondary ischemic cell damage (lactate and potassium clearance). In our opinion, it is important to perform a sufficiently wide decompression to prevent herniation, even in the case of transitory brain swelling (bridging veins). Burkert and Plaumann[6] demonstrated not only ICP reduction after decompression, but also a decrease in intracerebral PaO2 after brain swelling occurred. The change in PaO2 was even more sensitive to brain swelling than the rise in ICP. After decompression, the PaO2 increased, but only in patients in whom craniectomy was performed early and extensively. When craniectomy was performed in the late phase of ICP elevation and decompression was too limited, the PaO2 rose but did not reach normal values. We agree that the craniectomy should be performed early, before severe impairment of brain perfusion occurs, and should yield a wide decompression.

**Surgical Technique**

According to Seydel,[58] even Hippocrates gave indications for trephination in head injury. The controversial decompressive craniectomy for treatment of intracranial hypertension has played an important role in neurosurgery since its very beginning.[12,31,32,61] Kocher[39] wrote the following in his book on brain concussion, ICP, and surgical treatment of diseases of the brain, published in 1901: "If there is no CSF pressure, but brain pressure does exist, pressure relief must be achieved by opening the skull. Relief of pressure by trephination is clearly indicated in all cases of brain pressure." Even the timing of decompression was discussed: "There are two problems regarding the effectiveness of the measure: in the early stage, it is not easy to diagnose increased brain pressure; in the late stage, the performance of the procedure alone will be of no further use."[39] Since that time, the literature has provided descriptions of a variety of decompressive craniectomies. Spiller and Franzier[61] have given an historical overview on cerebral decompression since 1891. The circular decompression inaugurated by Bauer[4] and Clark and coworkers[7] demonstrated the least effectiveness, because it only created a small opening in the skull. Subtemporal decompression craniectomy was performed by Cushing.[12] Good results with this technique were reported by Heppner and associates,[30] Alexander, et al.,[1] and Gower, et al.[25] even though this form of trephination faces the risk of temporal lobe herniation and necrosis.[36] Bifrontal craniectomy was performed with good results by Pereira, et al.,[49] and Polin, et al.[52] Venes and Collins,[66] Goncalves da Silva and associates,[24] Kjellberg and Prieto[38] had less convincing results. They reported a decrease in the incidence of mortality at the price of an increase in
that of morbidity after the procedure. Of 13 patients treated with bifrontal craniectomy, only one had a very good outcome.[66] The technique we use, that is, wide unilateral or bilateral frontotemporoparietal craniectomy with dura opening and dura patch plasty, has striking advantages over the technique described by Kerr.[36] It provides maximum supratentorial bilateral decompression and the possibility of lateral expansion of the brain. Tentorial herniation is, therefore, virtually nonexistent. The technique was first performed by Doyen as described by Marcotte[43] in 1896. A number of authors used it with different results.[10,14,18,23,34,36,42,53,59,68] The cause of the poor results despite the large use of decompression reported in the literature is probably erroneous indication or timing of the procedure. Often, the decompression was performed when the patient developed neurological pons signs with bilaterally dilated pupils or when fatal pontive damage was already primarily present.[10,23,29,30,35,38,59] In this instance, irreversible ischemic damage to the brain exists with no chance of recovery. Table 5 gives an overview of the results of several authors with analysis of mortality rate, survival rate, and patient outcome after decompressive craniectomy in patients with therapy-resistant traumatic brain edema, including the results of our own patient group. Because of the partially insufficient documentation, a complete overview cannot be given. However, Table 5 shows that this surgical form of therapy has a clear indication in the treatment of refractory intracranial hypertension. In patients with surgical decompression, there is less risk of shifting from a fatal outcome to a vegetative state, but the chance of complete recovery of a patient who otherwise would not have survived or would be in a vegetative coma does exist.[23] Discouraging outcomes in patients do not invalidate the method;[23] good results, as in our series, confirm its usefulness.

**TABLE 5**

**Review of the literature on decompressive craniectomy in severe closed head injury**

<table>
<thead>
<tr>
<th>Authors &amp; Year</th>
<th>Total</th>
<th>Alive</th>
<th>Dead</th>
<th>Outcome Determined</th>
<th>GOS Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kerr, 1968</td>
<td>2</td>
<td>-</td>
<td>2</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Kjellberg &amp; Prieto, 1971</td>
<td>50</td>
<td>11</td>
<td>39</td>
<td>11</td>
<td>3</td>
</tr>
<tr>
<td>Ransohoff, et al., 1971</td>
<td>35</td>
<td>14</td>
<td>21</td>
<td>14</td>
<td>4</td>
</tr>
<tr>
<td>Venes &amp; Collins, 1975</td>
<td>13</td>
<td>0.9</td>
<td>4</td>
<td>9</td>
<td>2</td>
</tr>
<tr>
<td>Cooper, et al., 1976</td>
<td>50</td>
<td>5</td>
<td>45</td>
<td>45</td>
<td>3</td>
</tr>
<tr>
<td>Pereira, et al., 1977</td>
<td>12</td>
<td>6</td>
<td>6</td>
<td>6</td>
<td>1</td>
</tr>
<tr>
<td>Shigemori, et al., 1979</td>
<td>15</td>
<td>5</td>
<td>10</td>
<td>5</td>
<td>1</td>
</tr>
<tr>
<td>Yamaura, et al., 1979</td>
<td>154*</td>
<td>109</td>
<td>45</td>
<td>80</td>
<td>10</td>
</tr>
<tr>
<td>Gerl &amp; Taven, 1980</td>
<td>30</td>
<td>7</td>
<td>23</td>
<td>7</td>
<td>2</td>
</tr>
<tr>
<td>Crone &amp; Kelly, 1985</td>
<td>7</td>
<td>6</td>
<td>1</td>
<td>6</td>
<td>1</td>
</tr>
<tr>
<td>Alexander, et al., 1987</td>
<td>15</td>
<td>13</td>
<td>2</td>
<td>13</td>
<td>6</td>
</tr>
<tr>
<td>Karlen &amp; Stula, 1987</td>
<td>7</td>
<td>2</td>
<td>5</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Gower, et al., 1988</td>
<td>10</td>
<td>6</td>
<td>4</td>
<td>6</td>
<td>2</td>
</tr>
<tr>
<td>Hatashita, et al., 1993</td>
<td>3</td>
<td>-</td>
<td>3</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Whitfield &amp; Guazzo, 1995</td>
<td>1</td>
<td>1</td>
<td>-</td>
<td>1</td>
<td>-</td>
</tr>
<tr>
<td>Dam Hieu, et al., 1996</td>
<td>2</td>
<td>2</td>
<td>-</td>
<td>2</td>
<td>-</td>
</tr>
<tr>
<td>Polin, et al., 1997</td>
<td>35</td>
<td>27</td>
<td>8</td>
<td>27</td>
<td>14</td>
</tr>
<tr>
<td>total</td>
<td>441</td>
<td>223</td>
<td>218</td>
<td>194</td>
<td>56</td>
</tr>
<tr>
<td>present study</td>
<td>57</td>
<td>46</td>
<td>11</td>
<td>44</td>
<td>11</td>
</tr>
</tbody>
</table>

*Includes Group 1 patients only; Group 2 were not evaluated.
Indications and Prognostic Values

Our indication guidelines are summarized in Table 6. There is no indication for decompression craniectomy in patients with irreversible brainstem damage or in patients in whom irreversible herniation has occurred. Under these conditions, any therapeutic effort is in vain. The right time for surgical intervention is determined by clinical follow up, repeated CT scans, and continuous ICP and CPP monitoring. Also helpful are EEG, TCD ultrasonography, and monitoring of SEPs and BAEPs.

TABLE 6

<table>
<thead>
<tr>
<th>Indications for Decompressive Craniectomy</th>
</tr>
</thead>
<tbody>
<tr>
<td>age &lt; 50 years</td>
</tr>
<tr>
<td>brain swelling on CT scan</td>
</tr>
<tr>
<td>no fatal primary brain injury w/ irreversible brainstem signs or herniation w/ neurological pons signs</td>
</tr>
<tr>
<td>therapy-resistant increase in ICP (refractory intracranial hypertension)</td>
</tr>
<tr>
<td>intracranial hypertension w/ deterioration in clinical status (GCS score, decerebrate posturing, mydriasis), electrophysiological parameters (EEG findings, SEPs), &amp; TCD values (increase in pulsatility index, decrease in diastolic flow)</td>
</tr>
<tr>
<td>surgical intervention before irreversible brainstem damage or generalized ischemic brain damage (monitoring of ICP, and B wave, AEPs, &amp; SEPs)</td>
</tr>
</tbody>
</table>

Based on our results, a good outcome (GOS Score 4 or 5) can be expected in those patients not exceeding a calculated critical ICP (given in Table 3) prior to decompression. Whether posttraumatic brain swelling represents just a transitory disregulation or, rather, a complete disturbance of cerebral autoregulation remains unclear. Our results in monitoring the B wave support the latter hypothesis. We assume the presence of a central pacemaker whose dysfunction leads to the loss of the B wave.[2,41] Further factors point to a relationship among the presence of B-wave activity, periodic changes in flow velocity on TCD ultrasonography,[16,17,48] and the rhythmic modulations of background activity on EEG described by Machleidt[40] in 1980. If further evidence is found for the assumed relationships, the B wave will be a new parameter to control brainstem function. Thus, deterioration in neurological status while the patient is sedated could be detected before pupillary dilatation. Even though we could not determine a specific patient age for achieving a better outcome, we could confirm the well-known fact that younger patients generally will have a better outcome than older patients. This is also confirmed by the recent publication by Polin, et al.[52] A slowly developing deterioration seems to carry a better prognosis than a rapid one. Gerl and Tavan[23] reported that the prognosis was better in patients whose midbrain symptoms developed over days (3 days between trauma and decompression) instead of hours. It is important to perform a sufficiently wide craniectomy with dura opening and dura patch plasty to achieve sufficient decompression without herniation before irreversible ischemic brain damage occurs. Using the methodology described by Polin, et al., to involve a control population taken from the Traumatic Coma Data Bank is clearly an improvement over prior studies. These researchers also demonstrated a statistically significant increased rate of favorable outcomes in patients who underwent surgery (37%) compared with matched control patients (15%). Compared with other second-tier therapies, our results and those of others (see Table 5) are convincing and should pass the test of a prospective randomized clinical trial addressing this subject.

References


12. Cushing H: The establishment of cerebral hernia as a decompressive measure for inaccessible brain tumors; with the description of intramuscular methods of making the bone defect in temporal and occipital regions. Surg Gynecol Obstet 1:297-314, 1905


16. Droste DW, Krauss JK, Berger W, et al: Rhythmic oscillations with a wavelength of 0.5-2 min in


51. Piek J: Neue neuroprotektive Konzepte bei erhöhtem intrakraniellem Druck. Anästhesiol Intensivmed


58. Seydel K: *Antiseptik und Trepanation*. Munich: H Müller, 1886


Manuscript received May 11, 1998.

Accepted in final form September 8, 1998.

Address reprint requests to: Waltraud Kleist-Welch Guerra, M.D., Klinik und Poliklinik für Neurochirurgie, Ernst Moritz Arndt Universität Greifswald, Sauerbruchstrasse 8, 17487 Greifswald, Germany. email: neurosur@rz.uni-greifswald.de.