Cerebral oxygenation, vascular reactivity, and neurochemistry following decompressive craniectomy for severe traumatic brain injury

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Object. This study addresses the changes in brain oxygenation, cerebrovascular reactivity, and cerebral neurochemistry in patients following decompressive craniectomy for the control of elevated intracranial pressure (ICP) after severe traumatic brain injury (TBI).

Methods. Sixteen consecutive patients with isolated TBI and elevated ICP, who were refractory to maximal medical therapy, underwent decompressive craniectomy over a 1-year period. Their mean age was 38 years and the median Glasgow Coma Score on admission was 5.

Results. Six months following TBI, 11 patients had a poor outcome (Group 1, Glasgow Outcome Scale [GOS] Score 1–3), whereas the remaining 5 patients had a favorable outcome (Group 2, GOS Score 4 or 5). Decompressive craniectomy resulted in a significant reduction (p < 0.001) in the mean ICP and cerebrovascular pressure reactivity index to autoregulatory values (< 0.3) in both groups of patients. There was a significant improvement in brain tissue oxygenation (PbtO₂) in Group 2 patients from 3 to 17 mm Hg and an 85% reduction in episodes of cerebral ischemia. In addition, the durations of abnormal PbtO₂ and biochemical indices were significantly reduced in Group 2 patients after decompressive craniectomy, but there was no improvement in the biochemical indices in Group 1 patients despite surgery.

Conclusions. Decompressive craniectomy, when used appropriately in protocol-driven intensive care regimens for the treatment of refractory elevated ICP, is associated with a return of abnormal metabolic parameters to normal values in patients with eventually favorable outcomes. (DOI: 10.3171/JNS/2008/108/5/0943)

KEY WORDS • cerebral microdialysis • cerebral oxygenation • decompressive craniectomy • intracranial pressure • vascular reactivity

Central to the management of severe TBI is the prevention of secondary brain injury through optimization of physiological parameters as treatment targets. Whereas control of ICP and CPP remains the cornerstone of severe TBI management, the measurement of PbtO₂ is increasingly being used to quantify cerebral ischemia and hypoxia. Cerebral microdialysis has been used as a “surrogate marker” for cerebral ischemia, and this technique can also be used to ascertain substrate delivery and metabolism.

Today, such multimodal monitoring techniques are increasingly used in the modern NICU and have been incorporated into protocol-driven treatment regimens. In addition to physiological monitoring, derived indices provide clinicians with useful information to guide targeted therapy in the neurointensive care setting. The PRx is one such index that takes advantage of the relationship between MABP and ICP by quantifying cerebrovascular reactivity and providing an approximation to the cerebrovascular autoregulatory reserve. Whereas targeted protocol therapies have been shown to improve outcome, the implementation of decompressive craniectomy in the treatment of elevated ICP that is refractory to medical therapy remains controversial. The exact role, timing, and efficacy of decompressive craniectomy remains the subject of ongoing clinical trials. It has been hypothesized that the damaging cycle of extensive edema caused by elevated ICP, which results in ischemia of neighboring brain tissue and further infarction, may be interrupted by surgical decompression. Yet little is known about the metabolic and autoregulatory changes that occur in the brain after surgery. The aim of this study is to investigate the effect of decompressive craniectomy on cerebral oxygenation, cerebrovascular reactivity, and neurochemistry in patients with severe TBI.

Abbreviations used in this paper: CPP = cerebral perfusion pressure; CT = computed tomography; GCS = Glasgow Coma Scale; GOS = Glasgow Outcome Scale; ICP = intracranial pressure; IQR = interquartile range; MABP = mean arterial blood pressure; NICU = neurointensive care unit; PbtO₂ = brain tissue oxygen tension; PRx = pressure reactivity index; TBI = traumatic brain injury.
Clinical Materials and Methods

Patient Selection

From January 2005 to January 2006, 16 consecutive patients with both severe TBI and elevated ICP refractory to medical therapy were enrolled in this prospective observational study. Institutional ethics committee approval had been obtained prior to commencement of the study. Patients with a devastating injury (who were not expected to survive more than 24 hours), fixed and dilated pupils, or bleeding diathesis were excluded from the study. Clinical illness management was in accordance with established neurocritical care guidelines. All patients underwent a CT scan of the brain on admission to the NICU. Patients received artificial ventilation if their GCS score was < 8 or if there was respiratory compromise. Three types of intraparenchymal probes were then placed on the side of the maximal injury or swelling based on these findings. The ICP was continuously monitored using a fiberoptic intraparenchymal gauge device (Codman and Shurtleff). Microdialysis catheters (CMA-70, CMA Microdialysis) were inserted into the perilesional brain tissue. Licox polargraphic Clark-type microcatheters (Integra Neuroscience) were also inserted using the same bolt system to measure PbtO₂ and brain temperature. A postoperative CT scan was then performed to ascertain adequate catheter positioning; in patients with contusions, the catheter tip was located within 1.5 cm of the edge of the contused brain tissue.

Treatment Protocol

Control of ICP followed an incremental regimen. A 30° elevation of the head of the bed was performed and patients were sedated adequately with propofol (2–10 mg/kg/hour) and received adequate analgesia (intravenous morphine 2–5 mg/hour). Boluses of 20% mannitol (2 ml/kg up to a plasma osmolality of 320 mOsm/L) were administered if sudden increases in ICP > 20 mm Hg were present. An external ventricular drain was inserted if there was evidence of hydrocephalus and cerebrospinal fluid drainage was affected. If these measures failed to keep ICP below 20 mm Hg, then body cooling to 36°C, paralysis, and a barbiturate coma were induced. The coma was achieved with intravenous thiopentone, administered as 250-mg boluses over 10–20 minutes (up to a total loading dose of 500–1000 mg) with a maintenance dose of 125–500 mg/hour titrated to ICP control or to the maintenance of burst suppression electroencephalography. The primary treatment targets were an ICP < 20 mm Hg and a CPP > 70 mm Hg. A PbtO₂ level > 20 mm Hg was the secondary treatment target. Cerebral microdialysis values were not used as treatment targets.

A surgical decompression was offered as the final stage of therapy if the medical therapy instituted in such a stepwise fashion failed to keep ICP below 20 mm Hg (for more than 1 hour), especially if ICP plateau waves were seen. Hyperventilation was not administered routinely for patients with TBI. As a means of ICP control, however, hyperventilation to a pCO₂ of 30–35 mm Hg (4–4.7 kPa) was administered for a period of 30 minutes before surgery to patients who were scheduled for decompressive craniectomy.

Surgical Technique

Two techniques for decompressive craniectomy were used in our study. In the first technique, a standard bifrontal decompressive craniectomy was used for generalized cerebral edema. A bicoronal scalp incision was made, and the temporalis muscles were reflected inferiorly. Bilateral large subtemporal decompressions were performed, with a large bifrontal craniectomy, extending posteriorly into the parietal bones approximately 3 cm posterior to the coronal sutures. The final bone cut was made anteriorly over the sagittal sinus after stripping the dura from the underlying sinus. A wide dural incision was made, extending medially to the sagittal sinus. The sagittal sinus was ligated and divided anteriorly between two 2-0 silk sutures, thus allowing the brain to “expand forward.” Any associated mass lesions were evacuated.

In the second technique, a wide unilateral decompressive frontotemporoparietal craniectomy was used to evacuate unilateral mass lesions. The craniectomy had a medial margin that reached 1 cm lateral to the midline, its anterosuperior diameter was at least 12 cm, and a subtemporal craniectomy reaching the middle cranial fossa was included. In both decompression techniques, expansive duraplasty was performed using an allograft. Bone flaps were maintained in wet gauze at −4°C and stored from 3 to 6 months before reimplantation.

Multimodal Monitoring

All patients underwent multimodal monitoring with clinical data continuously recorded on a Hewlett-Packard CareVue System. Mean arterial pressure was measured directly from the radial artery using a standard pressure monitoring kit (Baxter Healthcare). The PbtO₂ monitoring was begun 3 hours after the insertion of the Licox microcatheters, thus giving the latter adequate time to stabilize. The cerebral microdialysis catheters had a membrane length of 10 mm, a diameter of 0.52 mm, and a molecular mass cut-off of 20,000 D. The microdialysis catheters were perfused by a microinjection pump (CMA-106, CMA Microdialysis) with artificial cerebrospinal fluid at a rate of 0.3 μL/minute. The dialysates (9–18 mL), collected every hour, were analyzed for glucose, lactate, pyruvate, and glutamate through a microdialysis analyzer (CMA-600, CMA Microdialysis) using an enzyme reagent and colorimetric measurements. The mean hourly values for MABP, ICP, and CPP were matched with PbtO₂ and cerebral microdialysis readings collected during the same hour. The different variables were continuously recorded, displayed, and stored using an in-house software program for multimodal data acquisition. The PRx at a particular time point was calculated as a moving correlation coefficient between the last 30 consecutive samples of values for ICP and arterial blood pressure averaged for a period of 10 seconds.

Statistical Analysis

The demographic and clinical data collected that were continuous variables were reported as means ± standard deviations or as the median with IQR if it was nonnormally distributed. For statistical analysis, physiological and cerebral microdialysis variables were separated into pre- and postdecompressive craniectomy categories in 2 patient outcome groups. The two-sample paired t-test was performed
Metabolic parameters after decompressive craniectomy for TBI

if the normality and equality of variances assumption was satisfied; otherwise, the Mann–Whitney U-test was used. Multivariate linear regressions of the differences between these variables in the 2 groups was performed to examine the influence of these parameters on the values observed. A probability value < 0.05 was considered statistically significant for all comparisons. Statistical analyses were performed using a commercially available statistical program (SPSS version 12.0, SPSS, Inc.).

Results

Patient Characteristics

Sixteen patients with severe TBI and elevated ICP refractory to medical therapy were included in the study over a 1-year period. Patient ages ranged from 20 to 72 years with a mean age of 38.0 ± 15.7 years. There were 13 male (81%) and 3 female (19%) patients, and the median admission GCS score was 5 (IQR 3–7). The CT scans of the head obtained in the patients at admission revealed various cerebral pathologies underlying the severe TBI (Table 1). No other major systemic injuries were detected in all our study patients.

All patients eventually underwent a surgical decompressive craniectomy for persistently elevated ICP that was refractory to medical management. There were an equal number of patients who underwent bifrontal craniectomy and unilateral craniectomy (8 patients each; Table 1). The monitoring of ICP, PbtO₂, and PRx, together with cerebral microdialysis, was performed pre- and postoperatively ipsilateral to the side of maximal cerebral swelling. A total of 2938 hours of such data was collected. The mean duration of neuromonitoring in the NICU was 6.5 days (IQR 3–12 days). Although the average duration from the patients’ admission to decompressive surgery was 68.5 hours (IQR 24–288 hours), 2 patients were monitored closely in the NICU for < 200 hours before surgery.

Clinical Outcome

At six months after injury, 6 patients (37.5%) had died and 10 (62.5%) had survived; among the survivors, 3 (19%) were classified as vegetative and 2 (13%) were severely disabled. In the remaining survivors, 4 (25%) had a moderate disability and only 1 (6%) had a good recovery. The patients were divided into 2 groups according to the GOS assessment at 6 months. Group 1, consisting of patients with poor outcome (GOS Score 1–3), had 11 patients (69%), while Group 2, consisting of patients with favorable outcome (GOS Score 4 or 5), had 5 patients (31%; Table 1). There were no significant differences between the 2 groups of patients with respect to age, sex, GCS score on admission, interval between admission and surgery, pathology of head injury, or type of craniectomy. The differences between the 2 groups with respect to MABP (p = 0.02), ICP (p < 0.001), CPP (p < 0.001), PbtO₂ (p < 0.001), and GOS score (p = 0.003) were significant.

Intracranial Pressure and CPP

The difference in mean ICP between Group 1 (25 ± 4 mm Hg) and Group 2 (18 ± 4 mm Hg) patients was significant (p < 0.001) during the period of maximal medical management before surgery (Table 2). Fluid resuscitation and vasopressors were required to maintain the preoperative CPP of 62 ± 6 mm Hg in Group 1 and 79 ± 6 mm Hg in Group 2. Despite maximal medical management, all of our patients demonstrated progressively more frequent sudden increases in ICP. The difference in mean ICP 2 hours before surgery between Group 1 (27 ± 8 mm Hg) and Group 2 (23 ± 4 mm Hg) was also significant (p < 0.001). Following decompressive craniectomy, the mean ICP had reduced significantly (p < 0.001) to 18 ± 2 mm Hg and 13 ± 2 mm Hg in Groups 1 and 2, respectively. Nevertheless, the mean CPP levels were maintained above traditional autoregulatory thresholds, at 82 ± 3 mm Hg and 78 ± 4 mm Hg in Groups 1 and 2, respectively (Table 2).

### Table 1

<table>
<thead>
<tr>
<th>Group No.</th>
<th>Case No.</th>
<th>Age (yrs), Sex</th>
<th>Pathological Condition</th>
<th>Admission GCS Score</th>
<th>Interval from Admission to DC (hrs)</th>
<th>Type of DC</th>
<th>GOS Score</th>
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<tr>
<td>1</td>
<td>1</td>
<td>32, F</td>
<td>TSAH, DAI</td>
<td>8</td>
<td>24</td>
<td>BC</td>
<td>2</td>
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</table>

* ASDH = acute subdural hemorrhage; BC = bifrontal craniectomy; BF = bifrontal; BL = bilateral; BP = biparietal; DAI = diffuse axonal injury; DC = decompressive craniectomy; EDH = epidural hematoma; LC = unilateral craniectomy; LF = left frontal; LT = left temporal; RF = right frontal; TSAH = traumatic subarachnoid hemorrhage.
Pressure Reactivity Index

The mean PRx in both groups was impaired (> 0.3) before surgery (Table 2). After surgical decompression for intractable ICP, the mean PRx returned to autoregulatory values in all patients. The reduction in the mean PRx in Group 2 patients from 0.31 ± 0.2 to 0.19 ± 0.1 mm Hg, was greater (p < 0.001) than that in Group 1 (0.34 ± 0.2 to 0.25 ± 0.2 mm Hg; Table 2).

Cerebral Oxygenation

Decompressive craniectomy resulted in a significant (p < 0.001) improvement in brain tissue oxygenation in Group 2 patients from a suboptimum PbtO2 level of 3 ± 2 mm Hg to a normal value of 17 ± 4 mm Hg (Table 2). A significant reduction (p < 0.001) of 85% in episodes of cerebral hypoxia was observed in Group 2 patients after surgery compared with a moderate reduction (32%; p = 0.03) in similar episodes in Group 1 (Table 2).

Cerebral Microdialysis

Cerebral microdialysis was begun within 6 hours after head injury. A significant (p < 0.001) improvement in all cerebral microdialysate levels (glycerol, lactate, glucose, and glutamate) and in the lactate:pyruvate ratio, leading to improved cerebral metabolism after decompressive surgery, was observed in the Group 2 patients (Table 2). In contrast, Group 1 patients demonstrated no significant changes in glutamate and glucose levels, and marginal improvements (p = 0.04) in the lactate:pyruvate ratio and the lactate level after surgery. Only the glycerol level showed a significant (p < 0.001) reduction to normal values in Group 1 patients (Tables 2 and 3).

Decompressive Craniectomy and Secondary Insults

Before decompressive craniectomy, secondary insults with prolonged periods of intracranial hypertension (ICP > 20 mm Hg) and suboptimal CPP (< 70 mm Hg) were significantly greater (p < 0.001) in Group 1 patients than in Group 2 patients. Interestingly, secondary (hypoxic and metabolic) insults as quantified by suboptimum PbtO2 and microdialysis levels were greater in Group 2 than in Group 1 patients before surgery (Table 3). After decompressive craniectomy, however, these secondary insults were significantly reduced in Group 2 patients. Nevertheless, in both groups, a significant reduction in periods of impaired PRx (> 0.3) was observed after surgery (Table 3).

Discussion

Severe head injury results in a complex cascade of pathophysiological processes. In addition to cerebral edema and raised ICP causing brain herniation and the physical distortion of structures, hemodynamic alterations can lead to hypoxia and ischemic damage. At the cellular level, rearrangement of brain metabolism together with release of inflammatory mediators causes further cellular injury. Diffuse axonal injury that is typically present can be difficult to quantify or treat. Over time, such secondary insults can be worsened by other new insults such as infections, stress responses, hypo- or hyperglycemia, and electrolytic and metabolic abnormalities. Focusing on a single treatment target in the management of severe TBI would therefore be inadequate.

Studies have demonstrated that cerebral infarction can occur despite normal ICP and CPP and that not all episodes of cerebral ischemia are associated with elevated ICP. In our study, decompressive craniectomy significantly reduced ICP in all patients regardless of outcome. Furthermore, the mean postoperative ICP of patients was significantly reduced in Group 2 patients. Nevertheless, in both groups, a significant reduction in periods of impaired PRx (> 0.3) was observed after surgery (Table 3).
and blood flow may be necessary for effective patient treatment in severe TBI. Monitoring ICP alone and a subsequent estimate of CPP may not reflect the cerebral metabolic needs of the individual patient\textsuperscript{2,11,13,14} and provides little indication of the cerebrovascular reserve.

**Cerebrovascular Reactivity**

The PRx is a simple continuous dynamic index that quantifies cerebrovascular reactivity and approximates global cerebral autoregulatory reserve. The PRx uses the natural variation of slow spontaneous fluctuations in MAP and ICP in ventilated patients, and makes use of existing physiological parameters (MAP and ICP) that are routinely monitored in most NICUs. In our study, PRx was calculated as a Pearson correlation coefficient of 30 consecutive samples of arterial blood pressure and ICP obtained at 10-second intervals.\textsuperscript{3,4} Expressed between a range of 1 and −1, a PRx value of 0.3 has been shown in previous studies to be the critical value above which pressure reactivity is impaired.\textsuperscript{5,17,33}

With preserved pressure reactivity, a rise in MAP leads to a diminution of arteriolar caliber following vasodilatation, and therefore to decreased cerebral blood volume and ICP.\textsuperscript{3,4,17} With the pathological loss of vasoreactivity, an increase in MAP leads to passive changes in cerebral blood volume and hence increased ICP. A negative PRx is therefore indicative of intact cerebrovascular reactivity, whereas a positive PRx is found in a pressure-passive vascular bed in which cerebral autoregulation is abnormal.\textsuperscript{3,4,17} The PRx has also been demonstrated to have a significant correlation with the outcome of head injury, and this relationship is possibly stronger than the association between admission GCS and GOS scores.\textsuperscript{7,33} Return to the autoregulatory state after decompressive surgery as demonstrated by a decrease in PRx over time has previously been shown.\textsuperscript{33} The decrease in mean PRx in both groups in our study complement these findings. Decompressive craniectomy, often used within the framework of a multitier treatment regimen, resulted in an improvement in cerebral autoregulation. Interpreting isolated mean PRx values, however, would not provide sufficient information regarding the patient’s clinical progress and eventual outcome.

It is noteworthy that although the mean PRx in the poor outcome group (Group 1) dropped below the critical threshold of 0.3, its percentage change was not as great as that of the favorable outcome group (Table 3). Furthermore, there was more than twice the percentage of subnormal episodes of impaired reactivity in the poor outcome group compared with the favorable outcome group after surgery.

**Cerebral Oxygenation**

Cerebral hypoxia is a major cause of poor outcome in patients with severe TBI.\textsuperscript{25,26} As part of a multimodal monitoring strategy, PbtO\(_2\) probes were placed in the pericontusional penumbra area of the brain, so that regional cerebral oxygenation could be reliably monitored and correlated with cerebral perfusion.\textsuperscript{16} A PbtO\(_2\) level of <10 mm Hg indicates episodes of cerebral hypoxia, whereas levels of 10–15 mm Hg indicate impending hypoxia. Ironically, the mean PbtO\(_2\) level in the poor outcome group increased (p < 0.001) from 17 ± 4 mm Hg before surgery to 20 ± 3 mm Hg after surgical decompression. This increase may suggest that injury in the pericontusional penumbra around the probe led to uncoupling of metabolism within neuronal cells, such that they were unable to metabolize O\(_2\) despite an adequate blood supply to that area. In contrast, decompressive craniectomy in the group of patients with a favorable outcome resulted in significantly (p < 0.001) improved PbtO\(_2\) level, from a hypoxic level of 3 ± 2 mm Hg to a normal range value of 17 ± 4 mm Hg. This improvement in brain tissue oxygenation corresponded to a significant improvement in brain neurochemistry as measured by microdialysis; however, interpreting absolute PbtO\(_2\) values without regard to other parameters would be insufficient and is a potential risk. A seemingly “normal” reading of a single parameter is insufficient to explain a patient’s condition and it would be prudent to interpret these readings in reference to other parameters that are monitored simultaneously. In our study, we found a significant reduction (p < 0.001) in the periods of hypoxic insults (PbtO\(_2\) < 10 mmHg) from 100 to 15% in the favorable outcome group after successful surgical decompression and a correlation between prolonged periods of suboptimum PbtO\(_2\) and poor outcome (Table 3).

**Cerebral Microdialysis**

Measurement of brain neurochemistry is based on the

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**TABLE 3**

Percentages of abnormal levels of physiological and cerebral microdialysis values before and after decompressive craniectomy

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group 1 (Poor Outcome)</th>
<th>Group 2 (Favorable Outcome)</th>
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</thead>
<tbody>
<tr>
<td>% Before DC</td>
<td>% After DC</td>
<td>% Change</td>
</tr>
<tr>
<td>PRx (&gt;0.3)</td>
<td>57</td>
<td>42</td>
</tr>
<tr>
<td>ICP (&gt;20 mm Hg)</td>
<td>67</td>
<td>42</td>
</tr>
<tr>
<td>MAP (&lt;70 mm Hg)</td>
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<tr>
<td>CPP (&lt;70 mm Hg)</td>
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<td>5</td>
</tr>
<tr>
<td>PbtO(_2) (&lt;10 mm Hg)</td>
<td>22</td>
<td>15</td>
</tr>
<tr>
<td>glycerol (&gt;126 μmol/L)</td>
<td>28</td>
<td>1</td>
</tr>
<tr>
<td>lactate (&gt;3.8 mmol/L)</td>
<td>68</td>
<td>66</td>
</tr>
<tr>
<td>l/p ratio (&gt;27)</td>
<td>89</td>
<td>80</td>
</tr>
<tr>
<td>glutamate (&gt;10 mmol/L)</td>
<td>40</td>
<td>48</td>
</tr>
<tr>
<td>glucose (&lt;0.5 mmol/L)</td>
<td>21</td>
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</table>
understanding that secondary insults such as hypoxia result in alterations in brain metabolism. Cerebral microdialysis can be used as a measure of the degree of severity of secondary insult to the brain. These secondary insults have been shown to correlate with metabolic changes in brain energy metabolism. Extracellular glutamate—an excitotoxic amino acid that is released into the extracellular fluid—has been described as an early marker of cerebral ischemia. Cell membrane damage leads to a liberation of glycerol and an increase in glycerol levels. Low glucose concentration is a consequence of hyperglycolysis in an environment with extreme metabolic demands. Increased glycolysis also leads to elevated lactate levels and lactate:pyruvate ratios in the extracellular fluid.

In our study, the value of cerebral microdialysis in determining the recovery of injured cerebral tissue is well illustrated in the favorable outcome group, in which there were significant reductions in mean glycerol levels from 139 ± 55 to 22 ± 10 μmol/L, in lactate levels from 7 ± 4 to 3 ± 2 mmol/L, and in glutamate levels from 36 ± 54 to 3 ± 1 mmol/L (p < 0.001; Table 2). Before surgery, the patients in the poor outcome group demonstrated a greater percentage of secondary insults in the physiological parameters of mean ICP (> 20 mm Hg) and CPP (< 70 mm Hg), although their PbtO₂ and neurochemistry levels were seemingly better than those in the favorable outcome group. Of note is the finding that there were greater reductions in the hypoxic and metabolic insults after decompressive craniectomy among patients in the favorable outcome group compared with those in the poor outcome group (Table 3).

During the initial monitoring period, the microdialysate concentrations and PbtO₂ level of Group 2 (favorable outcome) were far more abnormal than those in Group 1 (poor outcome). These abnormal parameters served as warning signs. When carefully instituted medical management failed to treat intracranial hypertension, surgical decompression was advocated as the final stage of the treatment protocol, and this resulted in favorable patient outcomes.

Importance of Multimodal Monitoring and a Rational Protocol

There is much evidence to suggest that neuromonitoring techniques using the PRx, PbtO₂ monitoring, and cerebral microdialysis are reliable and safe strategies in the treatment of patients with acute TBI. A surgical decompressive craniectomy, often used within the framework of a multistage treatment regimen, has been shown to reduce ICP and improve CPP, thus allowing the functionally compromised but viable brain to survive. Although its effect on outcome is the subject of a randomized multicenter trial, decompressive craniectomy in our study showed that it remains an important tool in the range of treatment for severe TBI. The small size of this study is a limiting factor in its implications, yet it demonstrates the efficacy and utility of monitoring numerous modalities that can help guide treatment. Moreover, additional studies are required to determine if PRx, cerebral microdialysis, and PbtO₂ monitoring can assist the physician in deciding when or in whom decompressive craniectomy should be performed.

Conclusions

This study demonstrates the utility of a rational approach in monitoring different pathophysiological treatment targets in the management of severe TBI. In a complex pathophysiological process, it is prudent to monitor various parameters. Despite a return of cerebral autoregulation over time and improvement in cerebral oxygenation after surgical decompression, clinical outcomes may still be uncertain. Decompressive craniectomy, when used appropriately in protocol-driven intensive care regimens for the treatment of recalcitrant elevated ICP, is associated with a return of abnormal metabolic parameters to normal values in patients with eventual favorable outcomes.

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