Cerebral oxygenation following decompressive hemicraniectomy for the treatment of refractory intracranial hypertension

MICHAEL F. STIEFEL, M.D., PH.D., GREGORY G. HEUER, M.D., PH.D., MICHELLE J. SMITH, B.S., STEPHANIE BLOOM, M.S.N, EILEEN MALONEY-WILENSKY, M.S.N, VINCENTE H. GRACIAS, M.D., M. SEAN GRADY, M.D., AND PETER D. LEROUX, M.D.

Department of Neurosurgery and Division of Trauma Surgery and Critical Care, University of Pennsylvania School of Medicine, Philadelphia, Pennsylvania

Object. Medically intractable intracranial hypertension is a major cause of morbidity and mortality after severe brain injury. One potential treatment for intracranial hypertension is decompressive hemicraniectomy (DCH). Whether and when to use DCH, however, remain unclear. The authors therefore studied the effects of DCH on cerebral O\textsubscript{2} to develop a better understanding of the effects of this treatment on the recovery from injury and disease.

Methods. The study focused on seven patients (mean age 30.6 ± 9.7 years) admitted to the hospital after traumatic brain injury (five patients) or subarachnoid hemorrhage (two patients) as part of a prospective observational database at a Level I trauma center. At admission the Glasgow Coma Scale (GCS) score was 6 or less in all patients. Patients received continuous monitoring of intracranial pressure (ICP), cerebral perfusion pressure (CPP), blood pressure, and arterial O\textsubscript{2} saturation. Cerebral oxygenation was measured using the commercially available Licox Brain Tissue Oxygen Monitoring System manufactured by Integra NeuroSciences. A DCH was performed when the patient’s ICP remained elevated despite maximal medical management.

Conclusions. All patients tolerated DCH without complications. Before the operation, the mean ICP was elevated in all patients (26 ± 4 mm Hg), despite maximal medical management. After surgery, there was an immediate and sustained decrease in ICP (19 ± 11 mm Hg) and an increase in CPP (81 ± 17 mm Hg). Following DCH, cerebral oxygenation improved from a mean of 21.2 ± 13.8 mm Hg to 45.5 ± 25.4 mm Hg, a 114.8% increase. The change in brain tissue O\textsubscript{2} and the change in ICP after DCH demonstrated only a modest relationship (r\textsuperscript{2} = 0.3). These results indicate that the use of DCH in the treatment of severe brain injury is associated with a significant improvement in brain O\textsubscript{2}.

KEY WORDS • INTRACRANIAL PRESSURE • CEREBRAL OXYGENATION • HEMICRANIECTOMY • DECOMPRESSION SURGERY

D ESPITE advances in the understanding and treatment of severe brain swelling and intracranial hypertension, outcomes for patients with these conditions remain poor. In particular, among patients with persistent ICP elevation after TBI or SAH, death or a poor outcome is an almost universal occurrence.\textsuperscript{24,41,47,50} Experimental studies demonstrate that brain swelling and increased ICP may be associated with reduced CBF at a time when demand may be increased.\textsuperscript{26,46,51,69} Clinical studies, however, demonstrate a relationship between poor outcome and increased, decreased, or normal CBF.\textsuperscript{34,35,72} Other studies demonstrate that cerebral infarction can occur despite normal ICP and CPP and that not all episodes of cerebral ischemia are associated with increased ICP.\textsuperscript{36,70,72} It can be inferred from these various studies that an understanding of ICP alone may be inadequate in the treatment of some patients and that additional measures of cerebral metabolism, CBF, or the adequacy of CBF may be necessary for effective patient care.

Recently, continuous direct measurement of O\textsubscript{2} in brain tissue has become possible in the US with the development of a commercially available brain tissue O\textsubscript{2} monitor (Licox Brain Tissue Monitoring System: Integra NeuroSciences, Plainsboro, NJ). Several lines of evidence suggest this may be a useful monitor to supplement ICP monitors in patients requiring neurocritical care after TBI or SAH.\textsuperscript{3,4,15,51,61,72,75–78} The Licox monitor is particularly useful because it provides a measure of local tissue O\textsubscript{2} delivery. This brain tissue measurement indicates the relative adequacy of CBF to metabolic demand, an amount that is indirectly extrapolated from, but not directly provided by, conventional ICP and CPP monitoring.

Strategies to reduce elevated ICP are central to the care of patients with severe brain injury. Nevertheless, limitations and potential deleterious effects are associated with many medical therapies, including cerebrospinal fluid drainage, hyperventilation, and administration of barbiturates or mannitol for elevated ICP.\textsuperscript{31,11,18,23,29,40,48} Consequently, despite questions about whether and when to perform DCH, sever-

Abbreviations used in this paper: CBF = cerebral blood flow; CPP = cerebral perfusion pressure; CT = computerized tomography; EEG = electroencephalography; ICP = intracranial pressure; SAH = subarachnoid hemorrhage; SjvO\textsubscript{2} = jugular venous O\textsubscript{2} saturation; SD = standard deviation; TCD = transcranial Doppler.
al authors have recommended this surgery in selected patients with persistent cerebral swelling or intractable intracranial hypertension following TBI or SAH.1,10,21,42,52,53,66,79 It is hypothesized that the vicious circle of extensive edema caused by elevated ICP, which results in ischemia of neighboring brain tissue and further infarction, may be interrupted by DCH. Experimental studies indicate that DCH may increase CPP and optimize perfusion, thus allowing the functionally compromised but viable brain to survive.16 Little is known about the metabolic changes that occur in the brain after DCH; therefore, the authors examined how DCH affected cerebral oxygenation when the procedure was performed to treat intractable intracranial hypertension.

**CLINICAL MATERIAL AND METHODS**

**Patient Population**

As part of a prospective observational database at a Level I trauma center, seven consecutive patients who underwent direct brain O2 monitoring before and after DCH for medically intractable ICP following severe TBI or SAH were studied over a 6-month period. To be included the patients had to have had severe TBI (GCS68 score < 8) or poor-grade aneurysmal SAH (Hunt and Hess Grade IV or V and GCS score < 8). Patients with surgical mass lesions were excluded from this study. The research was approved by the institutional review board of the Hospital of the University of Pennsylvania.

**Multimodality Monitoring**

Patients were treated in the neurosurgical intensive care unit. A triple-lumen introducer for monitoring brain tissue O2, ICP, and brain temperature (Integra NeuroSciences, Plainsboro, NJ) was inserted through a burr hole created in the frontal region of each patient.30 The monitor was placed on the side of maximal injury or swelling based on findings on the admission CT scan. Heart rate, blood pressure through an arterial line, arterial O2 saturation, and SjvO2 also were recorded. All physiological variables were continuously monitored before and after DCH by using a bedside monitor (Component Monitoring System M1046-9090C; Hewlett Packard, Andover, MA).

**Management of ICP**

Medical management for intracranial hypertension followed a stair-step approach in accordance with published recommendations.3,7,37,42,43,54,57 Each patient was fully resuscitated, intubated, and, with the head of the bed initially elevated 30°C, provided with mechanical ventilation. Ventilation was adjusted to maintain PCO2 at approximately 35 mm Hg and PaO2 at greater than 100 mm Hg. Intravenous morphine or fentanyl was administered and patients received continuous propofol infusion for sedation. An intravenous mannitol bolus was administered when ICP was greater than 20 mm Hg for more than 5 minutes. Neosynephrine was titrated when CPP was less than 65 mm Hg for more than 15 minutes. If no response to mannitol was observed, an external ventricular drainage system was placed and paralysis was induced by administering pancuronium. Additional mannitol was administered if, despite CSF drainage, the ICP remained greater than 20 mm Hg; this was continued until a maximum serum osmolarity of 320 mOsm was measured. Optimized hyperventilation was initiated as a second-tier therapy when sedation, paralysis, osmotherapy, and external ventricular drainage did not reduce the ICP or prevented pressure waves. Hyperventilation was increased to a PaCO2 of 25 mm Hg while maintaining the SjvO2 at greater than 50% and brain tissue oxygenation at greater than 25 mm Hg. If hyperventilation was required for longer than 24 hours or failed to reduce the ICP, barbiturates (a bolus followed by continuous infusion) were administered to induce EEG-monitored burst suppression. In each of these patients a Swan–Ganz catheter was inserted and vasopressors were administered to maintain CPP at greater than 65 mm Hg if barbiturate-associated hypotension was present.

**Decompressive Hemiancietomy**

Decompressive hemicraniectomy was performed when

---

### TABLE 1

**Summary of patient demographics**

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs, Sex)</th>
<th>Pathological State</th>
<th>Ad-DCH Time (days)*</th>
<th>Admission GCS Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>20, M</td>
<td>TBI</td>
<td>16.0</td>
<td>3</td>
</tr>
<tr>
<td>2</td>
<td>25, F</td>
<td>SAH</td>
<td>3.5</td>
<td>3</td>
</tr>
<tr>
<td>3</td>
<td>26, M</td>
<td>TBI</td>
<td>0.4</td>
<td>6</td>
</tr>
<tr>
<td>4</td>
<td>43, M</td>
<td>TBI</td>
<td>0.2</td>
<td>3</td>
</tr>
<tr>
<td>5</td>
<td>45, F</td>
<td>SAH</td>
<td>1.6</td>
<td>3</td>
</tr>
<tr>
<td>6</td>
<td>24, M</td>
<td>TBI</td>
<td>7.0</td>
<td>3</td>
</tr>
<tr>
<td>7</td>
<td>31, M</td>
<td>TBI</td>
<td>0.4</td>
<td>3</td>
</tr>
</tbody>
</table>

* Time from hospital admission (Ad) to DCH.

---

### TABLE 2

**Comparison of ICP and CPP before and after DCH**

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Pre-DCH (mm Hg)</th>
<th>Post-DCH (mm Hg)</th>
<th>% Change</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ICP</td>
<td>CPP</td>
<td>ICP</td>
<td>CPP</td>
</tr>
<tr>
<td>1</td>
<td>28 ± 15</td>
<td>64 ± 16</td>
<td>15 ± 6</td>
<td>65 ± 13</td>
</tr>
<tr>
<td>2</td>
<td>22 ± 5</td>
<td>88 ± 12</td>
<td>16 ± 4</td>
<td>102 ± 12</td>
</tr>
<tr>
<td>3</td>
<td>26 ± 8</td>
<td>75 ± 14</td>
<td>42 ± 11</td>
<td>62 ± 13</td>
</tr>
<tr>
<td>4</td>
<td>21 ± 7</td>
<td>66 ± 8</td>
<td>7 ± 4</td>
<td>103 ± 10</td>
</tr>
<tr>
<td>5</td>
<td>27 ± 9</td>
<td>68 ± 21</td>
<td>19 ± 4</td>
<td>68 ± 16</td>
</tr>
<tr>
<td>6</td>
<td>32 ± 12</td>
<td>67 ± 13</td>
<td>18 ± 3</td>
<td>82 ± 12</td>
</tr>
<tr>
<td>7</td>
<td>29 ± 6</td>
<td>72 ± 9</td>
<td>16 ± 4</td>
<td>85 ± 18</td>
</tr>
</tbody>
</table>

* Values are expressed as means ± SDs of physiological values obtained 1 hour before DCH and 3 hours after the procedure.
Cerebral oxygenation after decompressive hemicraniectomy

FIG. 1. Upper: Bar graph showing measurements (means ± SDs) of ICP and brain tissue oxygenation (BtO2) pre- and post-DCH for all seven patients. B: Bar graph illustrating the mean changes in ICP and brain tissue oxygenation from pre-DCH values following DCH. Standard deviation bars have been omitted for clarity.

ICP remained greater than 20 mm Hg, particularly if plateau waves were observed, and there was a progressive decline in cerebral oxygenation, based on brain tissue or SjvO2 monitoring, or the brain tissue oxygenation was at least 20 mm Hg for more than 15 minutes despite maximal medical management including second-tier therapies (optimized hyperventilation and barbiturates). Transcranial Doppler studies were obtained and patients underwent follow-up CT scanning of the head while they were en route to the operating room. At surgery, a large bone flap with a diameter of at least 12 cm (including the frontal, parietal, and temporal bone, and parts of the occipital squama) was removed so that the floor of the middle cerebral fossa could be exposed. The dura mater was attached to the craniotomy edge to prevent epidural bleeding. The dura was then opened in a cruciate fashion and left widely opened. DuraGen (Integra NeuroSciences) was applied over the dural defect and the bone was not replaced. The temporal muscle was loosely reapproximated and the skin was closed. After the DCH was completed the ICP, brain temperature, and brain tissue monitors were replaced on the same side as the surgery when feasible or else in the opposite frontal region.

Statistical Analysis

For statistical analysis, physiological variables were separated into pre- and post-DCH categories. Data are expressed for the 1-hour interval immediately preceding DCH and for the first 3 hours following DCH as means ± SDs or as the median if the data were not normally distributed. Variables were compared by using the Kruskal–Wallis nonparametric analysis of variance or the chi-square test. Statistical analyses were performed using a commercially available statistical program (InStat for the Macintosh, version 2.03; GraphPad Software, Inc., San Diego, CA). Statistical significance was defined as a probability value lower than 0.05.

Results

Characteristics of Patients

Seven patients, all with an admission GCS score of 6 or lower following TBI (five patients) or SAH (two patients), were studied (Table 1). There were two women and five men with a mean age of 30.6 ± 9.7 years. Each patient underwent DCH for persistently elevated ICP that was refractory to medical management. All seven patients had begun to receive optimized hyperventilation as a second-tier therapy for ICP control; four were also receiving barbiturate-induced EEG burst suppression (Cases 1, 2, 5, and 6). Computerized tomography scans of the head obtained before DCH revealed severe cerebral swelling in all patients but no surgical mass lesions. In no patient was there evidence of vasospasm based on findings of a TCD examination. Monitoring of ICP and brain tissue O2 was performed on the same side as the maximal cerebral swelling observed on the head CT scan and the subsequent DCH in all seven patients.

Relationship Between ICP and DCH

The mean ICP was significantly elevated (26 ± 4 mm Hg) during the hour before DCH despite maximal medical therapy (Table 2). In addition, each patient demonstrated progressively more frequent spikes in ICP that were less responsive to treatment before DCH. Fluid resuscitation or vasopressors were required to maintain the CPP (preoperative mean 71 ± 8 mm Hg) (Table 2) before DCH. Following DCH, ICP was reduced in six (86%) of seven patients to 15 ± 4 mm Hg (Table 2 and Fig. 1) and CPP was maintained at 84 ± 16 mm Hg without the use of vasopressor therapy. In addition, DCH allowed medical treatment in-

### Table 3

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Pre-DCH* (mm Hg)</th>
<th>Post-DCH* (mm Hg)</th>
<th>% Change</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>39.30 ± 19.40</td>
<td>98.90 ± 22.70</td>
<td>152</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>2</td>
<td>34.00 ± 15.70</td>
<td>39.50 ± 11.30</td>
<td>16</td>
<td>0.5</td>
</tr>
<tr>
<td>3</td>
<td>30.40 ± 6.20</td>
<td>51.60 ± 33.00</td>
<td>70</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>4</td>
<td>0.63 ± 0.06</td>
<td>30.40 ± 7.40</td>
<td>4725</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>5</td>
<td>12.60 ± 6.40</td>
<td>41.00 ± 17.40</td>
<td>225</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>6</td>
<td>13.30 ± 4.30</td>
<td>21.10 ± 3.80</td>
<td>58</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>7</td>
<td>17.90 ± 10.30</td>
<td>36.00 ± 5.00</td>
<td>100</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

* Values are expressed as means ± SDs. Measurements were obtained 1 hour before and 3 hours after the procedure.
including optimized hyperventilation, barbiturates, and osmo-
therapy to be withdrawn. The data in Table 2, except that for
Case 3, represent ICP and CPP values measured without the
use of barbiturates, osmotherapy, and hyperventilation. The
patient in Case 3, however, continued to have refractory in-
creased ICP because of devastating pulmonary and cardio-
ac injuries. He also had severe coagulopathy, which con-
tributed to a contralateral intracranial hematoma. When that
intracerebral hematoma was evacuated, the ICP was more
easily controlled.

Relationship Between Cerebral Oxygenation and DCH

Brain tissue O₂ was monitored in all patients before and
after DCH to determine the effect of DCH on cerebral oxy-
genation (Table 3 and Fig. 1). Before DCH, the mean brain
tissue oxygenation was 21.2 ± 13.8 mm Hg (Table 3 and
Fig. 1). Three patients (Cases 1–3) did not have cerebral
hypoxia, but during the hours before DCH they experienced
a progressive reduction in brain tissue O₂. It was in part this
reduction in brain tissue O₂ that prompted us to perform
DCH. Three patients (Cases 4–6) demonstrated moderate or
severe cerebral hypoxia (brain tissue oxygenation < 15 mm
Hg for > 15 minutes) and one patient (Case 7) had mild
cerebral hypoxia (brain tissue oxygenation 15–20 mm Hg)
before DCH. Decompressive hemicraniectomy resulted in
a significant improvement in brain tissue oxygenation, in-
creasing in all of the patients to a mean of 45.5 ± 25.4
mm Hg (Fig. 1 and Table 3). This improvement in brain tis-
ue O₂ was also observed in the patient who experienced a
contralateral intracerebral hematoma and continued to have
high ICP until the hematoma was evacuated (Case 3).
Improved cerebral oxygenation was observed immediately
after surgery and was sustained for the duration of monitor-
ing. Furthermore, as each patient was weaned from treat-
ment, brain tissue oxygenation remained greater than 25
mm Hg. Monitoring of ICP and brain tissue O₂ was contin-
ued until both the ICP and brain tissue O₂ were normal for
longer than 24 hours without treatment. In two patients
(Cases 1 and 2) SjvO₂ was measured, but no clear trend asso-
ciated with DCH was shown. In both patients SjvO₂ im-
proved less than 10%. Those undergoing DCH early or late
in their hospital course showed similarly improved brain tis-
ue O₂; the time between admission and surgical interven-
tion did not appear to have an effect on improving the level
of brain tissue O₂. Moreover, whether patients received only
hyperventilation or, alternatively, hyperventilation plus bar-
biturates as a second-tier therapy did not change the effect
of DCH on brain tissue O₂. The relationship between chang-
es in ICP and brain tissue oxygenation is illustrated in Fig.
1. The change in brain tissue O₂ and the change in ICP fol-
lowing DCH demonstrated only a modest relationship (r² =
0.3). No relationship was observed between the change in
CPP and the change in brain tissue O₂ in large part because
the vasopressors that were used increased CPP before DCH
was performed.

**Discussion**

The surgical management of intracranial hypertension is
directed toward improving cerebral perfusion and prevent-
ing ischemic damage and mechanical compression of the
brain. In this prospective study of seven patients, we ob-
served that DCH can have significant effects on the injured
brain, including a reduction in ICP, an elevation in CPP, and
a sustained improvement in cerebral oxygenation.

**Methodological Considerations**

In this study we obtained information about brain tissue
O₂ in consecutive patients in whom brain O₂ was monitored
before and after DCH and who were treated in a standard
fashion. Despite our methods, there are limitations to the
study. First, the sample size is small and thus we cannot
comment on patient outcome in general. Nevertheless, the
level of brain tissue O₂ may be a useful “surrogate biomark-
er” to help predict outcome because in several studies an
association between the intensity, number, and duration of
episodes of cerebral hypoxia and patient outcome has been
observed. Consistent with this relationship between
brain tissue O₂ and patient outcome, we observed that
patients in our study were more likely to have poor out-
comes if severe brain hypoxia was present before DCH,
whereas patients experienced favorable outcomes if DCH
was performed before severe cerebral hypoxia developed.
A much larger study will be needed to determine whether
DCH has a favorable impact on outcome or whether brain
tissue O₂ monitoring can be helpful in selecting those pa-
ients with intracranial hypertension who will benefit most
from DCH. Second, the study sample includes patients with
TBI and SAH, both of which are complex pathophysio-
logical entities. Although there are pathophysiological dif-
ferences between these conditions, there are also many
similarities, particularly increased ICP, cerebral edema,
alterations in CBF, and impaired autoregulation. Because
all patients in our study had medically intractable intracra-
nial hypertension, cerebral edema but no cerebral infarction
or other structural lesion on the head CT scan, and no evi-
dence of vasospasm apparent on TCD studies, the patho-
physiological differences between TBI and SAH do not in
our view detract from the primary observation of this paper:
that DCH significantly improves the level of brain tissue O₂.

**Brain O₂ Monitoring**

Cerebral infarction is common among patients who die
after TBI or SAH and can be prevented by performing DCH
to control ICP. At present, there is no ideal system of moni-
toring, including ICP monitoring and an estimate of CPP,
that can reliably detect cerebral ischemia after TBI or SAH.
Furthermore, investigators in clinical studies have demon-
strated that cerebral ischemia and infarction can occur in the
face of normal ICP and adequate CPP. Because they in-
dicate the relative adequacy of CBF to metabolic demand,
jugular bulb and direct brain tissue O₂ monitoring can be
used instead of quantitative CBF measurement. There is
substantial experience in the use of continuous SjvO₂ mon-
itoring to estimate CBF and also to understand O₂ use. There
are many limitations to this method; in particular, it is
not a good measure of local or regional abnormalities,
and unreliable readings occur in as many as 50% of record-
ings.

Recently it has been possible to measure local brain tis-
ue O₂ continuously. Several converging lines of evidence
show that this is a reliable and safe addition to monitoring
strategies in the treatment of patients with brain injury.
In animal studies it had been shown that continuous measurement of brain tissue O$_2$ is associated with changes in blood oxygenation and ventilation and variations in ICP and CPP. Clinical experience with brain tissue O$_2$ monitoring has been predominantly in patients with TBI, but there is now increasing experience in patients with SAH. These various studies demonstrate that cerebral hypoxia is common after severe brain injury and that these episodes of hypoxia sometimes can occur when CPP is normal. In addition, a significant association between the number and intensity of episodes of cerebral hypoxia and a poor outcome is well described. The localized nature of the brain tissue O$_2$ monitors is regarded as both a virtue and a potential limitation. The local use is important because the greatest metabolic changes are often observed regionally or around contusion sites. In our patients both ICP and brain tissue O$_2$ monitors were placed on the side of the maximal injury observed on a CT scan of the head; this is also the side on which the DCH was performed. It is conceivable that changes in brain tissue O$_2$ may occur in other regions of the brain that are not monitored; this is a phenomenon that has been well described for ICP monitoring. It is worth noting, however, that ICP monitors detect hydraulic differences that may be influenced more by variations in the local anatomy, such as in the falx or tentorium, than by the changes in O$_2$ delivery detected by brain tissue O$_2$ monitors. When brain tissue O$_2$ monitoring is performed in undamaged brain areas (that is, the penumbra), the values obtained can also be reliably extrapolated to evaluate global oxygenation even though the probe measures O$_2$ tension in a small volume of tissue.

In our practice we use multiple cerebral (ICP, SjvO$_2$, and brain tissue O$_2$) and hemodynamic measures in concert to understand cerebral pathophysiology. These continuous measures can be supplemented by point-in-time blood flow studies such as TCD, Xe-CT, or single-photon emission computerized tomography scanning to assist in the further elucidation of the cause of increased ICP or metabolic alterations in these patients. In this study we observed that continuous monitoring of brain O$_2$ is safe and may be a useful method to monitor changes in O$_2$ delivery during the treatment of intracranial hypertension. In particular, we observed that brain tissue O$_2$ immediately and consistently increased in all patients who underwent DCH for intracranial hypertension. We also observed that monitoring may, in some patients, help guide us to know when DCH is appropriate. For example, although DCH was performed primarily for intractable intracranial hypertension, in three patients it was also performed for newly developed episodes of cerebral hypoxia or a progressive decline in brain O$_2$. The remaining four patients all had cerebral hypoxia that had appeared early in their treatment. Whether DCH should be performed in the presence of cerebral hypoxia or when there is a progressive decline in brain tissue O$_2$ before cerebral hypoxia develops, however, is uncertain. Nevertheless, the resolution of elevated ICP in patients with adequate brain O$_2$ before DCH may result in a subset of patients who could experience a reasonable recovery and in whom continued aggressive treatment is justified.

Management of ICP and DCH

Increased ICP is a major predictor of mortality and morbidity in patients with TBI or SAH. Therefore, significant effort in neurocritical care is directed at preventing and treating intracranial hypertension. Brain edema and increased ICP can be treated in some patients by using, alone or in combination, medical therapies such as sedation, optimized hyperventilation, osmotherapy, or barbiturate administration. Unfortunately, these treatments may not be effective in some patients and each can produce deleterious effects. For example, the safety and value of hyperventilation in the treatment of intracranial hypertension is still a subject of debate because it can produce excessive arterial vasocostriction, which may result in cerebral ischemia. Nevertheless, knowledge about cerebral oxygenation or blood flow may limit the potential deleterious consequences or assist us to select a subset of patients who will benefit from hyperventilation therapy.

Decompressive surgery to reduce ICP has been advocated for more than 50 years, particularly for cerebellar infarction or massive middle cerebral artery infarction. Considerable debate, however, continues about the usefulness of DCH for intractable intracranial hypertension after TBI or SAH. Whereas DCH can effectively reduce elevated ICP, authors of numerous studies differ on whether the procedure improves overall patient outcome. Furthermore, it is uncertain whether DCH should be reserved for cases in which refractory intracranial hypertension is treated using the traditional stair-step approach to ICP or whether it should be performed in a prophylactic manner before intractable intracranial hypertension develops. One reason for this dilemma is that there is no single reliable system to monitor brain function in sedated or comatose patients who have elevated ICP. Furthermore, the optimal time to perform DCH has not yet been determined. At present only ICP monitoring is used in the majority of patients with brain injury; however, ICP monitoring and a subsequent estimate of CPP may not reflect the cerebral metabolic need of the individual patient.

Monitoring of brain tissue O$_2$ may become an ideal tool to help resolve this dilemma, because O$_2$ and glucose delivery, among other variables, are known to be important metabolic determinants in the recovery of injured cerebral tissue. Nevertheless, the impact of DCH on many parameters of cerebral metabolism, including brain O$_2$, is only beginning to be elucidated. In this study we examined changes in cerebral O$_2$ associated with DCH. Our results show that DCH is associated with a significant improvement in cerebral oxygenation. Our results are preliminary but provocative and we suggest that additional studies are required to determine whether brain O$_2$ monitoring can assist the physician in deciding when or in whom DCH should be performed.

Financial Disclosure

Licox Brain Tissue Oxygen Monitoring Systems were provided by Integra NeuroSciences, Plainsboro, New Jersey. The authors and their institution have no financial interest in any of the materials or devices used in the patients described in this article.

Acknowledgment

We acknowledge the hard work performed by the nurses in the Neurosurgical Intensive Care Unit at the Hospital of the University of Pennsylvania who cared for the patients in this study and assisted us in data acquisition.
References


Cerebral oxygenation after traumatic brain injury.

Cerebral oxygenation after decompressive hemicraniectomy


Manuscript received June 3, 2003. Accepted in final form April 14, 2004. This work was supported by a grant from the Sharpe Foundation for Neurosurgical Research.