FOLLOWING severe head injury, patients run a high risk of developing secondary cerebral hypoxic and ischemic damage due to reduced cerebral perfusion pressure (CPP), either because of intracranial hypertension or arterial hypotension. This may lead to a compromise in cerebral blood flow (CBF) and, thus, in oxygen supply, which in turn may negatively affect final outcome.

To minimize the risk of secondary cerebral hypoxia and ischemia following trauma, care should be taken to evacuate mass lesions as early as possible. As a follow-up therapy, treatment of posttraumatic raised intracranial pressure (ICP) has long been a "state of the art." Recently, more emphasis has been directed toward CPP as the driving force of CBF. Monitoring of cerebral oxygenation may now yield more information on the causes of secondary cerebral hypoxia and ischemia.

In addition to jugular bulb oximetry (measurement of $\text{O}_2$ saturation in the jugular vein (SjvO$_2$)), an established method for monitoring global cerebral oxygenation, measurement of local brain tissue $\text{PO}_2$ (PtiO$_2$) in cerebral white matter has recently been introduced.
For this purpose, a polarographic Clark-type microcatheter is used. First clinical reports have offered descriptions of the time course and effects of ventilation (for example, hyperventilation, and a fraction of inspired \( \text{O}_2 \) of 1.0) or reduced CPP on Pti\( \text{O}_2 \) in severely head injured patients. To date, however, Pti\( \text{O}_2 \) monitoring has not been validated against other established methods such as Sjv\( \text{O}_2 \) monitoring. Moreover, although a 50% \( \text{O}_2 \) saturation in the jugular bulb is known to be indicative of impending ischemia, such a critical threshold has not been determined for brain Pti\( \text{O}_2 \). To define a critical threshold in brain Pti\( \text{O}_2 \), simultaneous measurements of Sjv\( \text{O}_2 \) and brain Pti\( \text{O}_2 \) were performed. Analyses focused particularly on accidental episodes of arterial hypotension, that is, reduced CPP. In addition, safety of the monitoring technique as well as the data quality of Sjv\( \text{O}_2 \) and Pti\( \text{O}_2 \) were compared and evaluated.

**Clinical Material and Methods**

**Patient Characteristics and Early Posttraumatic Management**

In this study 15 patients with severe head injury were investigated. Permission to measure Pti\( \text{O}_2 \) was granted by the local ethics committee. The median Glasgow Coma Scale score in this group on admission was 5 (range 4–8). All patients had been intubated and ventilated on the scene. After resuscitation, cranial computerized tomography (CT) was performed. Space-occupying lesions that were greater than 25 ml were evacuated immediately. Patients were classified according to group as “evacuated mass lesion” (six cases), “diffuse injury III” (two cases), and “diffuse injury II” (seven cases) (Table 1), according to the definitions of the Traumatic Coma Data Bank. In all patients coma lasted longer than 5 days posttrauma. There were 13 male and two female patients in the group with a median age of 29 years (range 15–66 years). The median time of treatment in the intensive care unit (ICU) was 23 days. Monitoring of brain Pti\( \text{O}_2 \) was performed up to 12 days posttrauma (median 9 days), whereas jugular bulb catheters usually had to be removed after a median of 4 days (Table 1).

**Intensive Care Management and Organization of Study**

In addition to the oxygenation parameters, the patients’ electrocardiogram, arterial blood pressure, ICP, body temperature, arterial \( \text{O}_2 \) saturation (Sa\( \text{O}_2 \)), and end-tidal \( \text{CO}_2 \) were monitored in all cases. Measurement of ICP was performed using a fiberoptic intraparenchymal device (Ca-mio Laboratories, San Diego, CA; Fig. 1). For combined analgesia and sedation a continuous infusion of fentanyl (0.05–0.5 mg/hour) and midazolam (2–15 mg/hour) was used. The Sjv\( \text{O}_2 \) monitoring was initiated 6 to 24 hours postinjury. A No. 7.5 French introducer was placed percutaneously into the internal jugular vein, on the right in 10 cases and on the left side in five cases, followed by a No. 5.5 French fiberoptic catheter (Swan–Ganz catheter, Pulmonary Opticath P575EH, Oximetrix-3 System; Abbott Laboratories, N. Chicago, IL). Prior to insertion, a calibration using the colorimetric method provided by the manufacturer was performed. A bedside x-ray film confirmed the catheter’s tip position at the C-2 vertebral body. Routinely, saturation readings of the catheter were corrected every 12 hours if the in \textit{vitro} determined oxygen saturation (CO-Oximeter IL-482; Instrumentation Laboratories, Watertown, MA) and the catheter’s measurements differed by more than 5%. To prevent blood clotting at the tip of the catheter, a heparinized saline solution was continuously infused (3 ml/hour).

**TABLE 1**

Demographics and monitoring characteristics in 15 severely head injured patients*

<table>
<thead>
<tr>
<th>Patients’ age (yrs)</th>
<th>median</th>
<th>29</th>
</tr>
</thead>
<tbody>
<tr>
<td>Range</td>
<td>15–66</td>
<td></td>
</tr>
<tr>
<td>ICU treatment (days)</td>
<td>median</td>
<td>23</td>
</tr>
<tr>
<td>Range</td>
<td>8–46</td>
<td></td>
</tr>
<tr>
<td>Diagnosis†</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diffuse injury II</td>
<td>7</td>
<td></td>
</tr>
<tr>
<td>Diffuse injury III</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Evacuated mass lesion</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>(subdural hematoma)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Brain tissue ( \text{PO}_2 ) monitoring (days)</td>
<td>median</td>
<td>9</td>
</tr>
<tr>
<td>Range</td>
<td>5–12</td>
<td></td>
</tr>
<tr>
<td>Jugular bulb oximetry monitoring (days)</td>
<td>median</td>
<td>4</td>
</tr>
<tr>
<td>Range</td>
<td>3–7</td>
<td></td>
</tr>
</tbody>
</table>

* ICU = intensive care unit.
Brain tissue PO$_2$ monitoring

![Graph](image)

**Fig. 2.** Original chart obtained in a 15-year-old boy with multiple contusions. A spontaneous decline in mean arterial blood pressure (MABP) of 20 mm Hg for 10 minutes and a subsequent reduction in cerebral perfusion pressure (CPP) led to a simultaneous decrease in jugular vein O$_2$ saturation (SjvO$_2$) and brain tissue PO$_2$ (PtO$_2$). Intracranial pressure (ICP) slightly increased, indicating cerebral vasodilation, and end-tidal CO$_2$ (ETCO$_2$) decreased. Data for correlation analysis were taken from the CPP baseline to its minimum value.

Within 12 hours postinjury a flexible polarographic Clark-type microcatheter (LICOX System; GMS mbH, Kiel, Germany) to be used for continuous brain PtO$_2$ monitoring was inserted into the nonlesioned frontal white matter (Fig. 1). The catheter placement was guided by a specific introducer that was tightly fixed on a special skull screw. After insertion, the catheter was firmly Luer-locked onto the introducer. The insertion depth of the probe was 34 mm (from the level of the dura to the catheter tip). Proper location of the catheter was ascertained using a CT scan. The measured tissue surface area was approximately 17 mm$^2$. Technical characteristics of this catheter include a stirring artifact of less than 4%, a response time $T_{90/10}$ of 70 seconds, and a sensitivity drift of $\pm 1\%$ per day. Brain PtO$_2$ values were continuously averaged over a 500-μsec period and adjusted to actual body core temperature by means of a computer (LICOX System; GMS mbH). Immediately after their removal from the brain, the PtO$_2$ catheters were checked for correct PO$_2$ measurements.

To maintain an arterial PO$_2$ above 100 mm Hg, an appropriate fraction of inspired oxygen measuring between 0.35 and 0.55 was chosen. Hemoglobin concentration was maintained above 10 g/dl throughout the monitoring period.$^6$

Intracranial hypertension, defined as an ICP measuring 20 mm Hg or higher that lasted for more than 10 minutes, was treated by head elevation, moderate hyperventilation (PaCO$_2$ 30–35 mm Hg), and intermittent boluses of mannitol up to a maximum of 3.5 g/kg body weight per 24 hours. Barbiturate-induced coma was used in cases of otherwise uncontrollable intracranial hypertension and guided by a burst-suppression electroencephalogram pattern (seven cases). If this treatment failed, a decompressive craniotomy was performed (two cases).

Usually, CPP was maintained above 70 mm Hg by treating the elevated ICP and by pharmacologically raising the mean arterial blood pressure (MABP) by administration of dopamine and noradrenaline.

**Data Collection and Analysis**

Analog signals of MABP (recorded from a radial arterial catheter leveled to the skull base), ICP, end tidal CO$_2$ (Capnometer model 14360; Hewlett Packard, Waltham, MA), SaO$_2$ by pulse oximetry (model 1020A; Hewlett Packard), brain PtO$_2$, and SjvO$_2$ were digitized at 0.1 Hz, then displayed, stored, and analyzed on a multimodal computer system with appropriate software (LabVIEW; National Instruments, Austin, TX) developed by one of the authors (T.F.B.). The CPP was calculated on line as the difference between MABP and ICP.

“Time of good data quality” was assessed off line by means of a stepwise procedure. Artifact detection was performed using a software routine (LabVIEW) that automatically limited data to a preset range that also allowed detection of obvious technical errors such as those caused by cable disconnection. Episodes of catheter malfunction (for example, dislocation or low light intensity of the jugular bulb catheter) were marked and saved in separate files. Additionally, all data were reviewed by a physician using a high-speed replay module of the monitoring program. All automatically marked episodes were reviewed in detail and excluded when clear evidence for their reliability could not be found. The total time of these episodes was determined separately for brain PtO$_2$ and SjvO$_2$.

**TABLE 2**

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Time of MABP reduction*</th>
<th>MABP (mm Hg)</th>
<th>ICP (mm Hg)</th>
<th>CPP (mm Hg)</th>
<th>Brain PtO$_2$ (mm Hg)</th>
<th>SjvO$_2$ (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>beginning of analyzed episodes</td>
<td>89.3 ± 27.4</td>
<td>25.2 ± 13.2</td>
<td>65.5 ± 23.3</td>
<td>16.8 ± 5.2</td>
<td>65.1 ± 7.8</td>
<td></td>
</tr>
<tr>
<td>end of analyzed episodes</td>
<td>53.6 ± 8.3</td>
<td>15.9 ± 8.3</td>
<td>36.5 ± 15.8</td>
<td>10.8 ± 5.4</td>
<td>55.2 ± 12.1</td>
<td></td>
</tr>
</tbody>
</table>

* Values are expressed as the mean ± standard deviation. Abbreviations: CPP = cerebral perfusion pressure; ICP = intracranial pressure; MABP = mean arterial blood pressure; PtO$_2$ = tissue PO$_2$; SjvO$_2$ = jugular vein O$_2$ saturation.

For correlation analysis, 18 events of a marked MABP decrease (3099 datasets, average duration 12 minutes (range 6–24 minutes)), with a consecutive drop in CPP were taken (Fig. 2 and Table 2). Values were only used for
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Analysis of SjvO2 and PtiO2

A synopsis of all 18 episodes of reduced MABP used for correlation analysis is given in Table 2. The average MABP reduction was 35.7 mm Hg. The response of ICP on MABP reduction was inconsistent. In 12 episodes ICP dropped simultaneously with the MABP reduction, whereas ICP increased in six events (Figs. 2 and 3). On average, ICP displayed a reduction of 9.3 mm Hg and CPP a reduction of 29.0 mm Hg (Table 2). Cerebral oxygenation deteriorated by an average of 6.0 mm Hg in PtiO2 and 9.9% in SjvO2 (Table 2). The correlation analysis of brain PtiO2 versus SjvO2 revealed a significant correlation ($r = 0.71$) of the second order (Fig. 4 left). In the range of 30% to 75% SjvO2, brain PtiO2 dropped by approximately 0.5 mm Hg per 1% venous O2 saturation decrease. At an SjvO2 of 50%, brain PtiO2 ranged from 3 to 12 mm Hg, the regression curve’s best fit value being 8.5 mm Hg (Fig. 4 left). An SjvO2 of 30% was associated with a brain PtiO2 close to 0 mm Hg and an SjvO2 of 70% corresponded to a brain PtiO2 of 20 mm Hg.

The analyses of CPP versus SjvO2 and CPP versus PtiO2 (Fig. 4 center and right) revealed a third-order regression curve and a close relationship of cerebral oxygenation and CPP below the breakpoint of 60 mm Hg. Above 60 mm Hg CPP cerebral oxygenation was grossly unaffected by CPP.

Results

Safety and Data Quality of SjvO2 and Brain PtiO2

After removal of the PtiO2 catheter from the brain the difference between the brain PtiO2 and the calculated PO2 in room air was low ($5.5 \pm 1.1$ mm Hg). These control measurements indicate that brain PtiO2 catheters produce reliable data up to 12 days after insertion. Insertion of PtiO2 catheters into brain tissue was not associated with infections or intracranial bleedings.

The difficulties encountered during SjvO2 monitoring included poor light intensity and catheter dislocation. Repetitive calibrations, both routine and additional, had to be performed. Ninety-three of a total number of 170 calibrations (55%) revealed a difference between SjvO2-monitored saturation and CO2 oximeter readings of more than 5%. Total SjvO2 monitoring time was also short due to increasing periods of poor light intensity, which appeared more frequently the longer the monitoring lasted (Table 1). Hence, no SjvO2 measurements were obtainable beyond posttrauma Day 7.

The total monitoring time of PtiO2 was twice as long as that of SjvO2 (Table 1). The “time of good data quality” differed markedly (95% (2491 hours) in PtiO2; 43% (607 hours) in SjvO2).

Statistical Analysis

Statistical analysis was performed using a nonlinear regression computer program (Sigmaplot; Jandel Scientific, Corte Madera, CA) that ran the Marquardt–Levenberg algorithm (minimizing the sum of the squared differences of given parameters). Values are given as the mean ± standard error of the mean unless otherwise indicated. A probability value of less than 0.01 was considered significant. Dotted lines appearing in the graphs in Fig. 4 indicate the 99% prediction interval.

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Fig. 3. Original chart obtained in a 21-year-old man with an acute subdural hematoma as the primary diagnosis. Therapy was terminated because of a devastating intracranial situation (extensive left-sided contusions in the frontotemporoparietal areas). A withdrawal of dopamine induced a massive decline in mean arterial blood pressure (MABP), cerebral perfusion pressure (CPP), intracranial pressure (ICP), brain tissue PO2 (PtiO2), and jugular vein O2 saturation (SjvO2). The PtiO2 probe was located in the non-injured right frontal lobe. ETCO2 = end-tidal CO2.

Analysis if the last calibration of SjvO2 had been made less than 4 hours earlier and there was no “low light” alarm on the SjvO2 monitor. Furthermore, episodes of marked cerebral hyperemia, defined as a SjvO2 greater than 75%, were also excluded. The underlying mechanism of MABP reduction in one case was a cardiac arrest—successfully treated by cardiopulmonary resuscitation—and in a second case was withdrawal of a catecholamine infusion following an “end-of-life decision” because of a devastating intracranial situation (Fig. 3). In five events technical difficulties with the catecholamine infusion caused arterial hypotension. In the remaining 11 episodes no obvious reasons for MABP reduction could be detected. Throughout these episodes, the SaO2 was unchanged, whereas there was a reduction in end-tidal CO2 caused by reduced pulmonary perfusion and thus reduced pulmonary CO2 exchange (Figs. 2 and 3).
Brain tissue PO\textsubscript{2} monitoring

![Graphs displaying results of correlation analysis.](image)

**Fig. 4.** Graphs displaying results of correlation analysis. **Left:** Correlation analysis of brain tissue PO\textsubscript{2} (PtiO\textsubscript{2}) versus jugular vein O\textsubscript{2} saturation (SjvO\textsubscript{2}). The regression curve follows a function of the second order. Taking the hypoxic threshold of 50% SjvO\textsubscript{2} for comparison, brain PtiO\textsubscript{2} ranged between 3 and 12 mm Hg with a regression curve best fit value of 8.5 mm Hg (horizontal lines). **Center:** Correlation analysis of cerebral perfusion pressure (CPP) and SjvO\textsubscript{2}. The regression curve follows a function of the third order. A decline in SjvO\textsubscript{2} is observed below a CPP of 60 mm Hg, suggesting an intact cerebral autoregulation. **Right:** Correlation analysis of CPP and brain PtiO\textsubscript{2}. The regression curve follows a function of the third order. The curve demonstrates a marked decline in PtiO\textsubscript{2} below a CPP of 60 mm Hg.

**Discussion**

**General Aspects of Monitoring of Cerebral Oxygenation**

To detect impending cerebral ischemia in patients with severe head injuries, monitoring of MABP, ICP, and CPP as basic parameters of cerebral hemodynamics is imperative. There are situations, however, in which the ICP is normal and the CPP appears to be sufficient, but cerebral hypoxia may be present. Such episodes can be attributed to therapeutically induced hypocapnia or impaired arterial oxygen supply.\textsuperscript{5,25} Monitoring of cerebral oxygenation is capable of detecting harmful events, leading to hypoxic episodes that have been shown to exert negative effects on clinical outcome.\textsuperscript{30}

The first method used to monitor cerebral oxygenation in clinical examinations was jugular bulb oximetry. As an invasive technique it allows continuous assessment of SjvO\textsubscript{2} as an indicator of global cerebral oxygenation as well as calculation of arteriojugular venous O\textsubscript{2} content difference.\textsuperscript{5,25}

Tissue O\textsubscript{2} tension measurement represents an invasive method to monitor local cerebral oxygenation. Thus far, there are only a few reports on its clinical use.\textsuperscript{14,15,34}

Finally, near-infrared spectroscopy (NIRS) has been introduced for noninvasive monitoring of cerebral oxygenation.\textsuperscript{13,18} This procedure has been shown to detect cerebral hypoxic events intraoperatively and in the neurointensive care setting.\textsuperscript{11,12} However, numerous problems exist. Measurements tend to be unstable and do not reliably reflect episodes of cerebral maloxygenation.\textsuperscript{33} Moreover, the tissue volume monitored by this technique is undetermined. Although the optodes are positioned above the frontal region, the whole brain probably contributes to signal changes. However, the percentage distribution of venous, capillary, and arterial blood representing the NIRS signal is undefined. Nonetheless, NIRS is regarded as having an intermediate status between global (SjvO\textsubscript{2}) and local (PtiO\textsubscript{2}) methods to assess cerebral oxygenation.\textsuperscript{18} Clearly, technical refinements have to be made before this method can be introduced into clinical routine for long-term monitoring. As a noninvasive and simple technique, it bears the most interesting potential for the future to monitor cerebral oxygenation.

**Practicality and Reliability of Brain PtiO\textsubscript{2} and SjvO\textsubscript{2} Monitoring**

Monitoring of SjvO\textsubscript{2} is prone to many technical problems and vigilance is required to obtain reliable data. Even with frequent recalibration and special attention, problems such as poor light intensity, head movements, spontaneous waves, or sudden changes remain the underlying causes of a rather low “time of good data quality,” as has been shown by this and other studies.\textsuperscript{5,10,30} For correlation analysis, great attention was paid to eliminate such artifacts (see Clinical Material and Methods). In discussing the reliability of SjvO\textsubscript{2} data, the catheter’s location (left or right) has to be considered. Stocchetti, et al.,\textsuperscript{32} found relevant discrepancies in bilateral SjvO\textsubscript{2} measurements. Robertson and associates,\textsuperscript{30} on the other hand, moderated this problem and advise the physician to place the catheter in the jugular bulb that receives the highest flow, as does Dearden.\textsuperscript{5} We followed these guidelines. In conclusion, the absolute value of a single SjvO\textsubscript{2} data point is of lesser diagnostic relevance than its trend over time. Also, the 50% threshold in SjvO\textsubscript{2} should be used cautiously, because there are no reports on increased arteriojugular venous lactate content difference below this threshold.

In contrast to the frequent recalibration required by jugular bulb oximetry, the brain PtiO\textsubscript{2} catheter only needs calibration prior to its insertion and no recalibration is necessary. Control measurements after removal demonstrate a good stability. During the monitoring time, there were nearly no artifacts. Based on these characteristics, brain PtiO\textsubscript{2} monitoring is more suitable for continuous and routine use than jugular bulb oximetry. Clearly, PtiO\textsubscript{2} monitoring is a local technique, as the measured tissue
surface area of approximately 17 mm² demonstrates. It remains to be studied whether there are remarkable tissue O₂ tension differences between various regions or differences between "normal" and contused or infarcted tissue. In this study it was verified by CT scanning that the probes were located within the frontal white matter without obvious pathology, suggesting that this area is largely unaffected by trauma. This ensures reliable PtiO₂ data acquisition because PtiO₂ in the cerebral white matter is regarded to be much more homogeneously distributed than in the cerebral gray matter.\textsuperscript{31} The argument that the PtiO₂ probe could induce microvascular compression, thus underestimating the actual PtiO₂ cannot be rejected. This may be a reason why Van Santbrink, et al.,\textsuperscript{34} found low PtiO₂ values early after insertion of the probe.

**Critical Threshold in Monitoring Cerebral Oxygenation**

In animal experiments (using cats and dogs) the PtiO₂ in cerebral white matter was found to be 25 to 30 mm Hg in noninjured tissue.\textsuperscript{14,15} There are no data on PtiO₂ in the cerebral white matter of healthy humans. In severely head injured patients without intracranial hypertension or CPP decrease, PtiO₂ was also found to be in the range of 25 to 30 mm Hg.\textsuperscript{14,15} Until the present, no hypoxic threshold in PtiO₂ monitoring has been defined. In one report on a series of 22 patients with severe head injuries, five of six patients with PtiO₂ values of 5 mm Hg or below died or stayed vegetative.\textsuperscript{34} For the routine use of PtiO₂ monitoring in clinical practice, however, such a threshold should be identified as a guide for specific therapy. The 50% SjvO₂ threshold certainly does not represent a clearly delineated border because of side-to-side and interindividual differences as well as technical difficulties, as mentioned above. This may partially explain the variance in PtiO₂ (3–12 mm Hg) at the given 50% SjvO₂. The scattering of brain PtiO₂ at a SjvO₂ of 50% (Fig. 4 left) may also be related to local or regional differences in PtiO₂ measurements. Based on the data presented we propose that a brain PtiO₂ of 10 mm Hg and lower should be considered "hypoxic" and thus should be treated vigorously, for example, by an induced elevation of CPP. Nevertheless, the significance of this PtiO₂ threshold on clinical outcome, as shown by Robertson\textsuperscript{24} and Sheinberg and associates\textsuperscript{20} for SjvO₂ monitoring, needs to be confirmed by further investigations.

**Cerebral Oxygenation and Cerebral Perfusion Pressure**

In the present study we also address the effect on cerebral oxygenation of a reduced CPP caused by a decline in systemic arterial blood pressure. Such events appear rather frequently in the treatment of severely head injured patients and arterial hypotension is accepted as a major mechanism of secondary brain damage.\textsuperscript{31} This has been confirmed by studies that involved continuous monitoring of SjvO₂ in which sudden drops in CPP were identified as causing cerebral O₂ desaturation and worsening outcome.\textsuperscript{28,30} In animals, the level of PtiO₂ was found to decrease continuously with a falling CPP. At a CPP of 40 mm Hg, known to be associated with a significantly decreased CBF, PtiO₂ was approximately 15 mm Hg and at a CPP of 20 mm Hg, PtiO₂ was found to be 10 mm Hg.\textsuperscript{14,15} To our knowledge, there are no comparable human data on PtiO₂ versus CPP available.

The results of correlation analyses of CPP versus SjvO₂ and CPP versus brain PtiO₂ strongly suggest a preserved cerebral autoregulation in these patients, although true tests for pressure autoregulation, for example, CBF measurements, have not been performed. Oxygenation parameters were stable if CPP was above 60 mm Hg. This finding of a presumably preserved cerebral autoregulation following severe head injury is in accordance with the observations of others.\textsuperscript{2,7,28,29} It represents the basis for the concept of a CPP-guided therapy for successful treatment of intracranial hypertension by raising systemic blood pressure, which would result in cerebral vasoconstriction and a compensatory ICP reduction.\textsuperscript{28,29} It is still under debate in the literature whether and how often autoregulation is disturbed after trauma and how this should be diagnosed.\textsuperscript{23,27,29} In the present study there were only three events in which an impaired autoregulation might be postulated, because ICP and MABP decreased continuously while CPP remained in the normal autoregulatory range.

There is ongoing discussion about the CPP level necessary to guarantee sufficient CBF in severe head injury. Some authors assume that the cerebral autoregulation curve is depressed and shifted to the right after trauma. Hence, head-injured patients would need a CPP higher than 60 mm Hg to maintain CBF.\textsuperscript{2,19,20,27-29} The data we have presented on SjvO₂ and brain PtiO₂ suggest that a CPP of 60 mm Hg is always sufficient to maintain a "normal" cerebral oxygenation and that no improvement in SjvO₂ or PtiO₂ can be expected by raising the CPP above 60 mm Hg. The latter finding confirms recent results of Cruz, et al.,\textsuperscript{7} in which no changes in CBF and arterio-jugular venous O₂ content difference were observed when CPP was in the range of 60 to 130 mm Hg. Below this level of 60 mm Hg cerebral oxygenation may decrease. This is true at least for the subacute posttraumatic period during which our measurements took place. However, it could well be that a higher CPP is necessary during the acute stage (within 24 hours posttrauma) if CBF were to be markedly reduced.\textsuperscript{1} Chan, et al.,\textsuperscript{2} have advocated a CPP of 70 mm Hg to maintain a sufficient blood flow. This is based on the observation that below 70 mm Hg the pulsatility index measured by transcranial Doppler sonography increases and SjvO₂ starts to decline. These findings are close to our observations. Some studies focusing on outcome support even higher CPP levels. For example, McGraw\textsuperscript{19} and Mendelow, et al.,\textsuperscript{20} reported better outcome if CPP is maintained above 80 mm Hg. Only larger trials could clarify whether these CPP levels are in fact beneficial. Nevertheless, the present study once again demonstrates the importance of strictly maintaining CPP above at least 60 mm Hg.

**Conclusions**

In analyzed episodes of monitoring patients with severe head injury, SjvO₂ and brain PtiO₂ were found to run parallel. A hypoxic threshold of 8.5 mm Hg PtiO₂ (range 3–12 mm Hg) correlating with a SjvO₂ of 50% was established. For clinical use, a threshold of 10 mm Hg is proposed.

Marked declines in MABP and reduced CPP below 60 mm Hg have a substantially negative effect on cerebral...
Brain tissue PO$_2$ monitoring

oxygenation in severely head injured patients. At a CPP of 60 mm Hg and above no improvement in cerebral oxygenation was observed.

Brain PiO$_2$ monitoring is a useful addition to existing neuromonitoring methods. Hopefully, it will help to improve the management and final outcome of comatose patients.

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