Assessment of a noninvasive cerebral oxygenation monitor in patients with severe traumatic brain injury

Clinical article

GUY ROSENTHAL, M.D.,1 ALEX FURMANOV, R.N., B.S.N.,1 EYAL ITSHAYEK, M.D.,1 YIGAL SHOSHAN, M.D.,1 and VINEETA SINGH, M.D.2

1Department of Neurosurgery, Hadassah-Hebrew University Medical Center, Jerusalem, Israel; and 2Department of Neurology, University of California, San Francisco, California

Object. Development of a noninvasive monitor to assess cerebral oxygenation has long been a goal in neurocritical care. The authors evaluated the feasibility and utility of a noninvasive cerebral oxygenation monitor, the CerOx 3110, which uses near-infrared spectroscopy and ultrasound to measure regional cerebral tissue oxygenation in patients with severe traumatic brain injury (TBI), and compared measurements obtained using this device to those obtained using invasive cerebral monitoring.

Methods. Patients with severe TBI admitted to the intensive care unit at Hadassah-Hebrew University Hospital requiring intracranial pressure (ICP) monitoring and advanced neuromonitoring were included in this study. The authors assessed 18 patients with severe TBI using the CerOx monitor and invasive advanced cerebral monitors.

Results. The mean age of the patients was 45.3 ± 23.7 years and the median Glasgow Coma Scale score on admission was 5 (interquartile range 3–7). Eight patients underwent unilateral decompressive hemiepithromy and 1 patient underwent craniotomy. Sixteen patients underwent insertion of a jugular bulb venous catheter, and 18 patients underwent insertion of a Licox brain tissue oxygen monitor. The authors found a strong correlation (r = 0.60, p < 0.001) between the jugular bulb venous saturation from the venous blood gas and the CerOx measure of regional cerebral tissue saturation on the side ipsilateral to the catheter. A multivariate analysis revealed that among the physiological parameters of mean arterial blood pressure, ICP, brain tissue oxygen tension, and CerOx measurements on the ipsilateral and contralateral sides, only ipsilateral CerOx measurements were significantly correlated to jugular bulb venous saturation (p < 0.001).

Conclusions. Measuring regional cerebral tissue oxygenation with the CerOx monitor in a noninvasive manner is feasible in patients with severe TBI in the neurointensive care unit. The correlation between the CerOx measurements and the jugular bulb venous measurements of oxygen saturation indicate that the CerOx may be able to provide an estimation of cerebral oxygenation status in a noninvasive manner.

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KEY WORDS • brain oxygenation • non-invasive monitor • jugular bulb venous monitor • severe traumatic brain injury

Although monitoring of intracranial pressure (ICP) remains the foundation for the care of brain-injured patients in the neurointensive care unit, advanced cerebral monitoring has been used increasingly in recent years to monitor brain oxygenation and cerebral blood flow (CBF). The most recent update of the Guidelines for the Management of Severe Traumatic Brain Injury provides recommended thresholds of brain oxygenation in patients with severe traumatic brain injury (TBI).7 Most current monitoring technologies involve the placement of an invasive monitor in the brain parenchyma, with all the attendant risks. Development of a noninvasive method to monitor cerebral physiology in a continuous and clinically useful fashion has long been a goal in neurocritical care. The CerOx monitor (Ornim Medical Ltd.) uses near-infrared spectroscopy and ultrasound to measure cerebral oxygenation and blood flow in a noninvasive manner. The monitor employs a new technology, ultrasound-tagged near-infrared spectroscopy (UT-NIRS), leveraging the effect of ultrasound waves on light to tag it. This technology harnesses the ability of near-infrared light to measure regional oxygen saturation in combination with ultrasound that can achieve localization via the acousto-optic effect.25,27 We sought to assess the feasibility of using the CerOx monitor to measure regional cerebral oxygenation in the neurointensive care unit and to compare CerOx-based measurements with hemispheric measures of cerebral oxygenation in patients with severe TBI.

Abbreviations used in this paper: CBF = cerebral blood flow; CPP = cerebral perfusion pressure; FiO2 = fraction of inspired oxygen; GCS = Glasgow Coma Scale; ICP = intracranial pressure; MAP = mean arterial blood pressure; PbtO2 = partial pressure of brain tissue oxygen; rCsat = regional cerebral tissue oxygen saturation; SjvO2 = jugular bulb venous oxygen saturation; TBI = traumatic brain injury; UT-NIRS = ultrasound-tagged near-infrared spectroscopy.
Methods

Research Subjects

Institution review board approval for the study was received from the Hadassah Medical Organization.

Eligible subjects were TBI patients, admitted to the intensive care unit at Hadassah-Hebrew University Hospital and requiring ICP monitoring and advanced neuromonitoring. Inclusion criteria included a Glasgow Coma Scale (GCS) score of ≤ 8 on admission or multisystem injury requiring ICP monitoring. Intracranial pressure was monitored with an external ventricular drain or intraparenchymal sensor. Patients were treated in accordance with the Brain Trauma Foundation’s Guidelines for the Management of Severe Traumatic Brain Injury and with the following goals: PaO₂ > 100 mm Hg; PaCO₂ > 35 mm Hg and < 45 mm Hg; cerebral perfusion pressure (CPP) ≥ 50 mm Hg and ≤ 70 mm Hg; central venous pressure 5–7 mm Hg; ICP < 20 mm Hg; partial pressure of brain tissue oxygen (PtO₂) > 15 mm; hemoglobin > 10 g/dl; and Na > 135 mEq/L and < 145 mEq/L. Patients who had surgical mass lesions underwent surgical intervention in accordance with the Guidelines for the Surgical Management of Severe Traumatic Brain Injury.

Jugular Bulb Venous Saturation Monitoring

Jugular bulb venous saturation monitors were inserted under ultrasound guidance. Plain skull radiographs or axial CT scans confirmed the correct placement of the catheter tip.

Brain Tissue Oxygen Monitoring

Patients with severe TBI underwent insertion of a Licox intraparenchymal brain tissue oxygen monitor (Integra LifeSciences Corp.) as part of standard neurointensive care at our institution. Licox probes were inserted over Kocher’s point into the frontal lobe of the least-injured hemisphere. Probes were allowed to stabilize for at least 12 hours following insertion before any oxygen challenge was performed.

Noninvasive Cerebral Oxygen Saturation Monitoring

Regional cerebral tissue oxygen saturation (rCsat) was measured using the CerOx 3110 monitor, which utilizes UT-NIRS technology; UT-NIRS is a hybrid technology based on locally modulating coherent light with a localized low-power ultrasound wave via the acousto-optic effect. Like other NIRS-based devices, UT-NIRS illuminates tissue with near-infrared light. The UT-NIRS device then adds rapid, brief pulses of ultrasound into the tissue over the region of interest. The ultrasound waves induce a local, artificial modulation in the detected light intensity in a manner similar to the Doppler effect. The modulated light signal is then analyzed on the basis of the changes in light intensity induced by the ultrasound wave. In this manner, only light from a specific volume of brain tissue is selected for analysis, allowing measurement from a specific predetermined region of interest. The tissue volume measured with the device is approximately 1 cm³. The UT-NIRS device uses 3 light wavelengths of light (780–830 nm) that are modulated by ultrasound as described above. A spectral analysis is then performed to calculate the saturation of hemoglobin within the region of interest.

The measurement system consisted of 2 separate probes placed on the skin in the frontal region (after shaving the head, when needed) and secured with a special adhesive pad (Fig. 1). Each probe illuminates the tissue with laser light at 3 wavelengths and “collects” the scattered light back to the detector. The probe also incorporates a small ultrasound transducer that provides low-power waves for inducing the UT-NIRS signal. Probes were placed bilaterally over the frontal region of the patient’s head. Patients in whom a large subgaleal hematoma in the frontal region(s) obscured the UT-NIRS signal were excluded from the study.

Physiological Challenges

At our institution, challenge testing is performed as part of routine clinical care of severe TBI patients who undergo advanced cerebral monitoring to assess cerebral vascular autoregulation (mean arterial blood pressure [MABP] challenge), the response to hyperoxia to test proper functioning of brain tissue oxygen probes (oxygen challenge), and cerebrovascular CO₂ reactivity (hyperventilation challenge).

MABP Challenge

To assess cerebral vascular autoregulation, we increased MABP by initiating a noradrenaline drip to achieve a 10– to 15-mm Hg rise in MABP. In patients already treated with a noradrenaline drip, the rate of infusion was increased to achieve the desired rise of 10–15 mm Hg in MABP. This portion of the MABP challenge occurs over a 20- to 30-minute time period. Physiological variables, including systemic blood pressure, MABP, percutaneous systemic oxygen saturation, ICP, and PtO₂, were recorded.
Use of the CerOx monitor in severe TBI

during the MABP challenge and during all subsequent challenges. No CSF drainage occurred during any portion of the challenges, so as not to confound interpretation of the challenges. All patients are maintained on sedation during all challenges (either with propofol and remifentanil or midazolam and morphine). An arterial blood gas test and a venous blood gas test from the jugular venous catheter are performed when the goal MABP is reached. The purpose of increasing MABP is to observe the response of CBF, ICP, and PbtO₂ to an increase in pressure. In those patients in whom these parameters stay relatively constant during a rise in MABP, cerebral autoregulation is presumed to be intact. In those patients in whom a definite rise in seen in these parameters along with the increase in MABP, cerebral autoregulation is presumed to be impaired. The goals of CPP are then set as recommended in the Guidelines for the Management of Severe Traumatic Brain Injury, with a lower CPP goal (50–60 mm Hg) for those patients with impaired autoregulation and a higher CPP goal (60–70 mm Hg) for those patients with preserved autoregulation. Patients who are hemodynamically unstable or have baseline high blood pressure are deemed unsuitable for a MABP challenge. Also, patients with extremely labile ICP may be deemed unstable for MABP challenge as vaspressors may initiate a spike in ICP that may be difficult to control if autoregulation is impaired. Clinical judgment is crucial to determine which patients may safely undergo a MABP challenge to avoid harm.

At the conclusion of the MABP challenge, the phenylephrine drip was discontinued (in patients who had not been receiving pressor support to maintain CPP) or returned to the baseline infusion rate (in patients who had been receiving phenylephrine to maintain CPP), and the MABP was allowed to return to the prechallenge level and followed by a 30-minute observation period before initiation of the oxygen challenge.

**Oxygen Challenge**

During the oxygen challenge, we increased the fraction of inspired oxygen (FiO₂) from baseline to 1.0 for approximately 20 minutes. Arterial and venous blood gas samples were drawn from the radial artery and jugular bulb venous catheter prior to initiating the oxygen challenge (baseline) and at the end of the oxygen challenge. The FiO₂ was maintained at 1.0 during subsequent challenges (hyperventilation challenge) to reduce the risk of tissue hypoxia or desaturation during the hyperventilation challenge. Oxygen challenge was performed routinely with the purpose of assessing proper functioning of the Licox brain tissue oxygen monitor as previously described. In addition to testing probe functionality, an oxygen challenge helps to determine the maximal PbtO₂ that can be achieved at an FiO₂ of 1.0, which is often an indicator of lung function.

**Hyperventilation Challenge**

Immediately after completing the oxygen challenge, a hyperventilation challenge was initiated to assess cerebral CO₂ vasoreactivity, as previously described. The purpose of the hyperventilation challenge is to assess the degree to which ICP can be reduced with moderate hyperventilation without impairing brain tissue oxygenation so that an optimal point can be determined as a therapeutic goal in the event of a rise in ICP. Patients who have pulmonary complications with difficulty in oxygenation or ventilation do not undergo hyperventilation challenge, as the potential to worsen pulmonary status exists.

The respiratory rate was increased by 25% and then by increments of 2 breaths per minute with the goal of decreasing PaCO₂ by 10 mm Hg. This portion of the challenge also occurs over a 15- to 20-minute period. A hyperventilation challenge was considered successful if the PaCO₂ decreased by 5 mm Hg or more on the arterial blood gas test. As preset safety precautions, hyperventilation was terminated when PbtO₂ approached 15 mm Hg or if it decreased more than 50% of the value attained during an oxygen challenge to ensure patient safety. Arterial and jugular bulb venous blood gases were sampled at the conclusion of the challenge. The total time course for a 3-step challenge (MABP, oxygen, and hyperventilation) was approximately 120 minutes. For each individual patient a daily assessment was made to determine whether it was safe to undergo a 3-step challenge.

**Statistical Analysis**

Means and standard deviations were calculated for continuous physiological data. The values for jugular bulb venous oxygen saturation (SjvO₂) and arteriovenous oxygen difference were taken directly from the venous blood draw and are thus point measurements. Values of continuously measured physiological parameters including MABP, ICP, CPP, PbtO₂, and rCsat were averaged over a 1-minute period corresponding to the draw time of the venous blood gases. A 2-way ANOVA model corrected for repeated measures within the same patients was used to analyze differences at each of the challenge conditions. Linear correlation coefficients between CerOx monitor rCsat measurements and jugular bulb venous saturation were evaluated using a Bland-Altman correction for repeated measurements in the same patients. This correlation was tested in a multivariate analysis adjusted to other physiological parameters. The relationship between CerOx-measured rCsat and physiological parameters was evaluated using a general linear model, corrected for multiple measurements in the same patient. The level of statistical significance level was defined as p = 0.05. All analyses were carried out using SPSS version 20.01 software (IBM).

**Results**

We studied 18 patients (13 male, 5 female) with severe TBI using the CerOx 3110 monitor. Patient characteristics are detailed in Table 1. The mean age was 45.3 ± 23.7 years (range 20–80 years), and the median GCS on admission was 5 (interquartile range 3–7). The side of surgical intervention (if performed) and the location of invasive monitors are also detailed in Table 1. Eight patients underwent unilateral decompressive hemicraniectomy and 1 patient underwent craniotomy. Sixteen patients underwent insertion of a jugular bulb venous...
catheter, which in 5 patients was ipsilateral to the side of surgical intervention and in 3 patients contralateral to the side of surgical intervention. In those patients who did not undergo surgical intervention the jugular bulb venous catheter was placed on the right side in 6 patients and on the left side in 2 patients. Eighteen patients underwent insertion of a Licox brain tissue oxygen monitor, which was generally placed in the least-injured hemisphere per our clinical protocol. Mean values for physiological parameters at the time of the challenges are presented in Table 2. Importantly, no complications associated with use of the CerOx noninvasive monitor were observed in any patients. DAI = diffuse axonal injury; EVD = external ventricular drain; GOS-E = Glasgow Outcome Scale–Extended; IPM = intraparenchymal monitor; IVH = intraventricular hemorrhage; MCA = motorcycle accident; MVA = motor vehicle accident; SAH = subarachnoid hemorrhage; SDH = subdural hematoma.

We studied the relationship between rCsat and jugular bulb hemispheric measures of venous saturation. Good quality jugular bulb venous data were obtained in 14 (78%) of 18 patients. Eleven patients had both good-quality jugular bulb venous data and CerOx data that could be compared. We compared CerOx rCsat measurements to the jugular bulb venous saturation on both the ipsilateral and contralateral sides to the jugular bulb catheter. We found a strong correlation (r = 0.60, p < 0.001) between the jugular bulb venous saturation from the venous blood gas and the UT-NIRS measure of rCsat on the side ipsilateral to the catheter (Fig. 2) that remained significant when adjusted for multiple measurements in the same patient (p < 0.001). In contrast, the correlation between the jugular bulb venous saturation and the CerOx rCsat on the contralateral side was substantially weaker (r = 0.23, p = 0.004) and was not significant when adjusted for multiple measurements in the same patient (p = 0.31). We then assessed the relationship between jugular bulb venous saturation with CerOx rCsat on the ipsilateral and contralateral sides and physiological parameters that may influence cerebral perfusion including MABP, ICP, and brain tissue oxygen tension in a multivariate analysis (Table 3). Only CerOx rCsat on the side ipsilateral to the catheter was related to jugular bulb venous saturation in a statistically significant manner. The other parameters including MABP, ICP, and brain tissue oxygen tension did not reach statistical significance.

**TABLE 1: Patient characteristics**

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs), Sex</th>
<th>Mechanism of Injury</th>
<th>Initial GCS Score</th>
<th>Type of Intracranial Injury</th>
<th>ICP Monitor (type &amp; side)</th>
<th>Licox Monitor Side</th>
<th>Side of Op</th>
<th>Side of SjvO₂ Monitor</th>
<th>GOS-E Score at 6 mos</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>22, M fall</td>
<td>3</td>
<td>contusion, SAH</td>
<td>EVD, rt</td>
<td>rt</td>
<td>rt</td>
<td>none</td>
<td>rt</td>
<td>6</td>
</tr>
<tr>
<td>2</td>
<td>20, M bike accident</td>
<td>7</td>
<td>SAH, IVH, DAI</td>
<td>EVD, rt</td>
<td>rt</td>
<td>rt</td>
<td>none</td>
<td>rt</td>
<td>4</td>
</tr>
<tr>
<td>3</td>
<td>58, M fall</td>
<td>9</td>
<td>SDH, contusion, SAH</td>
<td>EVD, lt</td>
<td>lt</td>
<td>rt</td>
<td>lt</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>21, F MVA</td>
<td>5</td>
<td>contusion, SAH, IVH, DAI</td>
<td>EVD, rt</td>
<td>rt</td>
<td>none</td>
<td>lt</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>42, M MVA</td>
<td>3</td>
<td>contusion, IVH, SAH, SDH (tentorial), DAI</td>
<td>IPM, rt</td>
<td>rt</td>
<td>rt</td>
<td>none</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>20, M MCA</td>
<td>6</td>
<td>contusion, SAH, IVH, DAI</td>
<td>EVD, rt</td>
<td>rt</td>
<td>none</td>
<td>rt</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>79, F fall</td>
<td>6</td>
<td>SDH</td>
<td>EVD, rt</td>
<td>rt</td>
<td>lt</td>
<td>2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>59, M fall</td>
<td>3</td>
<td>SDH</td>
<td>EVD, rt</td>
<td>rt</td>
<td>lt</td>
<td>lt</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>23, M MVA</td>
<td>3</td>
<td>SDH, SAH</td>
<td>EVD, rt</td>
<td>rt</td>
<td>lt</td>
<td>lt</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>46, M MVA</td>
<td>3</td>
<td>contusion, SAH, SDH (tentorial)</td>
<td>IPM, rt</td>
<td>rt</td>
<td>none</td>
<td>rt</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>72, M fall</td>
<td>4</td>
<td>SDH, contusion, SAH</td>
<td>EVD, lt</td>
<td>lt</td>
<td>rt</td>
<td>rt</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>47, F struck by falling object</td>
<td>5</td>
<td>SDH, contusion, SAH</td>
<td>EVD, lt</td>
<td>lt</td>
<td>rt</td>
<td>none</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>80, F fall</td>
<td>3</td>
<td>SDH</td>
<td>EVD, rt</td>
<td>rt</td>
<td>lt</td>
<td>lt</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>78, M fall (attempted suicide)</td>
<td>4</td>
<td>contusion, SAH</td>
<td>EVD, rt</td>
<td>rt</td>
<td>none</td>
<td>none</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>23, M fall</td>
<td>7</td>
<td>contusion, SAH</td>
<td>EVD, rt</td>
<td>rt</td>
<td>none</td>
<td>rt</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>16</td>
<td>29, M MVA</td>
<td>9</td>
<td>contusion, SAH, SDH</td>
<td>EVD, rt</td>
<td>lt</td>
<td>none</td>
<td>lt</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>17</td>
<td>22, M MVA</td>
<td>7</td>
<td>contusion, SDH, SAH</td>
<td>EVD, rt</td>
<td>lt</td>
<td>rt</td>
<td>rt</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>18</td>
<td>74, F fall</td>
<td>7</td>
<td>SDH, contusion, SAH</td>
<td>IPM, lt</td>
<td>lt</td>
<td>lt</td>
<td>rt</td>
<td>1</td>
<td></td>
</tr>
</tbody>
</table>

* CerOx probes were placed over the frontal region bilaterally in all patients. DAI = diffuse axonal injury; EVD = external ventricular drain; GOS-E = Glasgow Outcome Scale–Extended; IPM = intraparenchymal monitor; IVH = intraventricular hemorrhage; MCA = motorcycle accident; MVA = motor vehicle accident; SAH = subarachnoid hemorrhage; SDH = subdural hematoma.
We also studied the relationship between UT-NIRS regional measures of cerebral oxygenation and physiological parameters of MABP, ICP, and brain tissue oxygen tension. Since patients were stable at the time of challenges without extremes of hypotension or intracranial hypertension, it is not surprising that MABP and ICP were not related to cerebral oxygenation as measured by CerOx (Table 4). Interestingly, brain tissue oxygenation measured by Licox was also not significantly related to CerOx measures of oxygenation, indicating that these 2 measures of brain oxygenation are not strongly related to each other.

Discussion

Our findings indicate that noninvasively monitoring brain oxygenation with the CerOx monitor in the neuro-intensive care unit in severe TBI patients is feasible. The correlations between the UT-NIRS measurements and the jugular venous bulb measurements of oxygen saturation indicate that the CerOx monitor may be able to provide a noninvasive estimation of cerebral oxygenation status in brain-injured patients. Interestingly, we found that only the ipsilateral measurements of rCsat were significantly correlated to jugular bulb venous saturation. This finding is compatible with the observations of Stocchetti et al. who found a difference in jugular bulb venous oxygen saturation and oxygen content of greater than 15% between the hemispheres in 15 of 32 patients with severe TBI, suggesting that substantial differences in cerebral oxygenation exist between the hemispheres in patients with severe TBI and that unilateral hemispheric measures may not adequately assess overall cerebral oxygenation status. Our results suggest that the CerOx monitor may be able to provide a noninvasive measure of regional cerebral oxygenation that reflects differences in oxygenation between the hemispheres. Whether the CerOx rCsat measurements will be useful in helping guide clinical care requires further study.

Not surprisingly, the UT-NIRS measurements of regional cerebral tissue oxygenation did not correlate with systemic and cerebral physiological parameters including MABP and ICP. This is likely due to the fact that at the time that data were acquired patients were stable in the intensive care unit without extremes of ICP, MABP, CPP, or systemic oxygenation. Even within the context of physiological challenges to assess cerebral autoregulation and

### Table 2: Mean values for physiological parameters at baseline and during challenges

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Baseline</th>
<th>MABP Challenge</th>
<th>Oxygen Challenge</th>
<th>Hyperventilation Challenge</th>
</tr>
</thead>
<tbody>
<tr>
<td>MABP (mm Hg)</td>
<td>80 ± 15</td>
<td>88 ± 17†</td>
<td>80 ± 14</td>
<td>76 ± 15‡</td>
</tr>
<tr>
<td>ICP (mm Hg)</td>
<td>10 ± 7</td>
<td>10 ± 5</td>
<td>12 ± 6†</td>
<td>9 ± 4†</td>
</tr>
<tr>
<td>CPP (mm Hg)</td>
<td>70 ± 17</td>
<td>78 ± 18†</td>
<td>68 ± 14</td>
<td>66 ± 15</td>
</tr>
<tr>
<td>PbtO2 (mm Hg)</td>
<td>23.9 ± 10.3</td>
<td>25.7 ± 8.3</td>
<td>31.0 ± 14†</td>
<td>32.6 ± 12.5</td>
</tr>
<tr>
<td>SjVO2 (%)</td>
<td>75 ± 9</td>
<td>78 ± 8</td>
<td>80 ± 8†</td>
<td>69 ± 10†</td>
</tr>
<tr>
<td>AVDO2 (ml O2/100 g)</td>
<td>2.9 ± 1.2</td>
<td>2.7 ± 1.2</td>
<td>2.4 ± 1.2</td>
<td>4.5 ± 1.4†</td>
</tr>
<tr>
<td>CerOx rCsat (%)§</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ipsilateral</td>
<td>56 ± 12</td>
<td>56 ± 11</td>
<td>55 ± 11</td>
<td>51 ± 12</td>
</tr>
<tr>
<td>contralateral</td>
<td>55 ± 11</td>
<td>55 ± 10</td>
<td>54 ± 12</td>
<td>56 ± 10</td>
</tr>
</tbody>
</table>

* Values are presented as the mean ± SD. AVDO2 = arteriovenous oxygen difference.
† p < 0.001
‡ p < 0.05.
§ Measured using CerOx; ipsilateral and contralateral refer to the side of the jugular bulb catheter.

![Graph demonstrating the relationship between jugular bulb venous saturation taken from venous blood gases and the ipsilateral CerOx rCsat (r = 0.60, p < 0.001).](image)
cerebral CO₂ vasoreactivity, the parameters of MABP, ICP, CBF, and cerebral oxygenation vary within a narrow range. This very narrow range of values likely precludes a strong correlation to MABP or ICP to cerebral oxygenation, which may be seen at the more extreme ranges of high and low values. Experimental studies have clearly demonstrated that the relationship between both ICP and CPP and brain oxygenation is weak unless extremes of either ICP or CPP are reached.¹⁵,¹⁶ The lack of a significant relationship between brain tissue oxygen tension and rCsat is also anticipated. Gupta and colleagues directly compared brain tissue oxygen tension with jugular bulb venous saturation during hyperventilation challenges in patients with severe TBI and found that changes in brain oxygen tension did not correlate with changes in SjvO₂ when patient data were pooled.¹⁷ Although both PbtO₂ and rCsat measure cerebral tissue oxygenation, brain tissue oxygen tension is primarily influenced by arterial oxygen tension and CBF.²⁰–²³,²⁶–²⁵ In contrast, rCsat is a measure of regional tissue saturation that seems to be influenced primarily by the degree of oxygen extraction determined by the balance between oxygen delivery and utilization. In this regard, the rCsat measure is similar to the SjvO₂ measurements that are most strongly influenced by the interaction between blood oxygen content and CBF, on the one hand, and cerebral oxygen utilization, on the other hand.¹⁴,¹⁶ The UT-NIRS measurements of regional tissue saturation have the potential benefit of providing a noninvasive correlate of hemispheric oxygenation status that may reflect the balance between oxygen delivery and utilization.

Limitations

The current study has several limitations. First, it is a small study that intended to assess the feasibility of using the CerOx monitor in patients with severe TBI in the neurointensive care unit. The small size of this study precludes any assessment of the relationship of rCsat measurements to outcome. Second, comparing a hemispheric measurement of cerebral oxygenation such as jugular bulb venous saturation to a regional measurement of brain tissue oxygenation is problematic. Unfortunately, current technology does not provide a definitive measurement of regional cerebral tissue oxygen saturation that can be compared with the CerOx noninvasive measure. Last, while the CerOx monitor allows for continuous measure of rCsat, we compared the measurements obtained to discrete values of hemispheric venous saturation from the jugular bulb catheter. The continuous measure of jugular bulb venous saturation entails technical difficulties including a relatively small amount of time for collecting good-quality data and the need for frequent recalibrations.¹⁹ While our approach substantially limits the number of comparisons, we felt that the venous gases drawn at defined physiological conditions allowed a more definitive assessment of oxygenation status.

Conclusions

Measuring regional cerebral tissue oxygenation with the CerOx monitor in a noninvasive manner is feasible in patients with severe TBI in the neurointensive care unit. The CerOx rCsat measures correlate to hemispheric measures of cerebral oxygenation in the ipsilateral jugular bulb but not to those on the contralateral side, suggesting that it may be able to noninvasively identify regional differences in cerebral oxygenation. Further studies are required to determine the clinical utility of the CerOx measures of regional cerebral tissue oxygenation.

Disclosure

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Address correspondence to: Guy Rosenthal, M.D., Department of Neurosurgery, Hadassah-Hebrew University Medical Center, P.O. Box 12000, Kiryat Hadassah, Jerusalem 91120. email: rosenthalg@hadassah.org.il.