A noninvasive estimation of mixed venous oxygen saturation using near-infrared spectroscopy by cerebral oximetry in pediatric cardiac surgery patients

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Summary

Background: Near-infrared spectroscopy (NIRS) is a noninvasive optical monitor of regional cerebral oxygen saturation (rSO2). The aim of this study was to validate the use of NIRS by cerebral oximetry in estimating invasively measured mixed venous oxygen saturation (SvO2) in pediatric postoperative cardiac surgery patients.

Methods: Twenty patients were enrolled following cardiac surgery with intraoperative placement of a pulmonary artery (PA) or superior vena cava (SVC) catheter. Five patients underwent complete biventricular repair – complete atrioventricular canal (n = 3) and other (n = 2). Fifteen patients with functional single ventricle underwent palliative procedures – bidirectional Glenn (n = 11) and Fontan (n = 4). Cerebral rSO2 was monitored via NIRS (INVOS 5100) during cardiac surgery and 6 h postoperatively. SvO2 was measured from blood samples obtained via an indwelling PA or SVC catheter and simultaneously correlated with rSO2 by NIRS at five time periods: in the operating room after weaning from cardiopulmonary bypass, after sternal closure, and in the CICU at 2, 4, and 6 h after admission.

Results: Each patient had five measurements (total = 100 comparisons). SvO2 obtained via an indwelling PA or SVC catheter for all patients correlated with rSO2 obtained via NIRS: Pearson’s correlation coefficient of 0.67 (P < 0.0001) and linear regression of r² = 0.45 (P < 0.0001). Separate linear regression of the complete biventricular repairs demonstrated an r = 0.71, r² = 0.50 (P < 0.0001). Bland–Altman analysis showed a bias of +3.3% with a precision of 16.6% for rSO2 as a predictor of SvO2 for all patients. Cerebral rSO2 was a more accurate predictor of SvO2 in the biventricular repair patients (bias –0.3, precision 11.8%), compared with the bidirectional Glenn and Fontan patients.

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Conclusions: Regional cerebral oximetry via NIRS correlates with SvO$_2$ obtained via invasive monitoring. However, the wide limits of agreement suggest that it may not be possible to predict absolute values of SvO$_2$ for any given patient based solely on the noninvasive measurement of rSO$_2$. Near-infrared spectroscopy, using the INVOS 5100 cerebral oximeter, could potentially be used to indicate trends in SVO$_2$, but more studies needs to be performed under varying clinical conditions.

Keywords: mixed venous oxygen saturation; near-infrared spectroscopy; cardiothoracic surgery; cerebral oximetry; perioperative care

Introduction

Near-infrared spectroscopy (NIRS) is a noninvasive optical technique to assess microcirculatory oxygenation (1,2). NIRS relies on the relative transparency of biological tissues to near-infrared light (700–900 nm) where oxygenated and deoxygenated hemoglobin have distinct absorption spectra. By measuring the attenuation of light at several wavelengths and distances between emitter and detector, it is possible to determine a value for cerebral oxygen saturation (rSO$_2$) (3).

Cerebral oximetry differs from pulse oximetry in several respects. Although both use near-infrared light signals, pulse oximetry monitors the pulsatile signal component reflecting arterial circulation, whereas cerebral oximetry monitors the nonpulsatile signal component reflecting tissue circulation (arterioles, capillaries, and venules). Because the cerebral microcirculation contains arterial, venous, and capillary components, cerebral saturation represents a ‘weighted average’ of the tissue circulation, with approximately 75–85% of the signal originating from venules (4,5). Because NIRS monitoring is noninvasive and portable, it can provide real-time measurements of these changes at the bedside (5–7).

In postoperative cardiac surgery patients, maintaining an adequate cardiac output is a critical determinant of outcome. When the cardiac output begins to decline there is a decrease in the mixed venous oxygen saturation (less than the normal 65–70%) because of reduced tissue blood flow and greater oxygen extraction (increased A – V oxygen difference) (8). Thus, measurement of the mixed venous oxygen saturation (SvO$_2$) is an indicator of cardiac output. Catheters placed in the pulmonary artery in these postoperative patients allow the direct measurement of SvO$_2$. In patients with intracardiac shunting or cavopulmonary anastomosis, such as those undergoing bidirectional Glenn (BDG) or Fontan completion, oxygen saturation in the superior vena cava (SVC) can be used as a substitute for SvO$_2$. Complications from an indwelling intracardiac catheter, however, include bleeding at the time of removal, catheter breakage and entrapment at the time of removal, infection, thrombus formation and embolization (9–11). Invasively measured SvO$_2$ from a central venous catheter does not provide continuous monitoring, and acute deteriorations of SvO$_2$ may be missed. In addition, repeated blood sampling leads to increased blood loss, central venous catheter colonization, contamination, and increased risk of infection. It is prudent, therefore, to develop noninvasive measures to monitor SvO$_2$.

The purpose of this study was to compare cerebral rSO$_2$ with oximetrically measured SvO$_2$ from an indwelling pulmonary artery or SVC catheter in infants and children after cardiac surgery. We hypothesize that the two methods would correlate closely, and that the noninvasive cerebral rSO$_2$ would be adequate for monitoring SvO$_2$.

Methods

Patient selection and data collection

After approval from our Institutional Review Board for human subject research and informed parental consent, patients were enrolled in the study protocol. Inclusion criteria included consecutive patients...
who underwent a reparative or palliative cardiac surgical procedure in which an intracardiac pulmonary artery catheter or percutaneous SVC catheter in patients with cavopulmonary anastomosis was used.

The INVOS 5100 Cerebral Oximeter (Somanetics Corp., Troy, MI, USA), is a two-channel (R + L) cerebral oximeter, with an adult (>40 kg) and a pediatric (≤40 kg) Somasensor. The single-use sensors differ by shape and dimension, and are placed on the right, left, or bilateral forehead by a self-adhesive layer. The INVOS 5100 sensor has two detectors to measure the ratio of oxyhemoglobin to total hemoglobin, with the resulting percentage equal to the value for regional cerebral oxygen saturation (rSO2). The proximal detector receives a signal from the peripheral tissue and the distal detector receives a signal from the extra- and intracranial tissues; by subtracting the proximal from the distal value, rSO2 is obtained. Depending on which sensor is connected, the INVOS 5100 uses sensor-dependent algorithms to calculate rSO2, with the pediatric algorithm being adjusted for the stronger signal reflected because of the thinner skull of a child allowing more ambient light to enter the head.

Currently all patients who undergo cardiac surgery at our institution have continuous cerebral oximetry monitoring in the operating room and those patients entered into the study had cerebral oximetry monitoring continued in the cardiac intensive care unit (CICU) for 6 h after their arrival. Pediatric Somasensors were placed on the right or left forehead for all patients by a single investigator (TAT). Care was taken to ensure proper adhesion of the sensor to the forehead and securing of cables from the sensor to the monitor. Oxymetric measurements of SvO2 (Radiometer ABL 700; Diamond Diagnostics, Holliston, MA, USA) were made at five time periods and simultaneous rSO2 was correlated. The time periods were: in the operating room after weaning from cardiopulmonary bypass, in the operating room after sternal closure, and at 2, 4, and 6 h after arrival to the CICU. Simultaneous arterial blood gases were also measured at these time periods. Six hours after arrival to the CICU, the cerebral oximeter probe was removed and the study protocol was terminated.

**Statistical analysis**

Results are presented as mean ± SD or as mean ± SEM when indicated. Measured SvO2 was correlated with rSO2 measurements using Pearson’s correlation coefficient. Analysis of agreement between the two measurements was assessed using the method of Bland and Altman (12,13). Statistics within subjects was performed in accordance with a theory previously suggested by the same authors (14). Bias was calculated as the mean difference between rSO2 and the SvO2 for each patient; a positive bias (mean difference) indicates that the rSO2 measure was higher on average. Precision of bias estimate was defined as 2 SD of the mean difference. Analysis of covariance (ANOVA) was used to determine intra-subject variation. The relationship between rSO2 and SvO2 was also determined by linear regression. Data were analyzed using SPSS for Windows (version 11.0; SPSS, Inc., Chicago, IL, USA). Statistical significance was accepted at P < 0.05.

**Results**

Twenty patients were enrolled, 13 females and seven males, with a mean age of 1.7 years (range 5 months to 8 years) from July 2002 through January 2003. The mean weight was 8.9 kg (range 5.1–24.4). Their diagnoses were diverse and included acyanotic and cyanotic forms of congenital heart disease (Table 1). There were five patients who underwent complete biventricular repairs (repair of complete atrioventricular canal, n = 3; other, n = 2), and 15 patients with functional single ventricle who underwent palliative procedures (BDG, n = 11; Fontan, n = 4). No functional single ventricle patients were noted to have venovenous collaterals and no biventricular repair patients had postoperative intracardiac shunting by transesophageal echocardiogram. No complications occurred during cerebral oximetry monitoring in the operating room or in the CICU.

Measurements were made for the 20 patients at five time periods after weaning from cardiopulmonary bypass, giving a total number of 100 observations. Table 2 delineates the physiological variables at each of the five time periods after weaning from cardiopulmonary bypass. The median rSO2 measured by NIRS was 67.5 ± 9.8%, with a range of 43–90%. Corresponding measurements of SvO2

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were 63.0 ± 10.0%, with a range of 32–83%. The mean rSO2 for the entire patient population was slightly higher than the mean SvO2 at each of the five time periods, with the trend in mean rSO2 following the trend in the mean SvO2 at each of the five time periods (Figure 1). Results from individual subjects are presented in Figure 2; with two representative subjects from the biventricular repair group, BDG group, and Fontan group respectively. Correlation and agreement between NIRS measurement of rSO2 and cooximetry measurement of SVO2 is presented in Table 3. Figure 3 demonstrates the linear regression plot and Figure 4 the Bland–Altman plot of rSO2 and SvO2 for all patients.

When comparing the differences between mean rSO2 values measured by NIRS and mean SvO2 values from the pulmonary artery or SVC catheter, considerable intersubject variation was found. However, the mean intrasubject standard deviation of the difference between rSO2 by NIRS and SvO2 was low (4.0 ± 2.1%). This indicated a consistent bias within subjects. Therefore, all rSO2 values by NIRS were corrected for bias by the intrasubject mean difference between rSO2 by NIRS and SvO2 values measured.
from the pulmonary artery or SVC catheter. The correlation coefficient found by analysis of covariance between intrasubject bias-adjusted rSO\(_2\) values and SvO\(_2\) was 0.63 (\(P < 0.0001\)). A Bland–Altman plot of the bias-adjusted NIRS rSO\(_2\) values and SvO\(_2\) values revealed limits of agreement of −10.1 and 13.4, still within a wide range of variability.

### Discussion

Mixed venous oxygen saturation is commonly used as an indicator of the adequacy of whole-body oxygenation, as it reflects the balance between tissue oxygen delivery and oxygen consumption. The utility of serial measurements of SvO\(_2\) in the care of critically ill patients has been documented by many investigators (15–19), and is facilitated by the availability of pulmonary artery catheters that provide continuous measurement of SvO\(_2\) (20,21). In adult cardiac surgery patients, Jamieson et al. (22) demonstrated a more than 10% fall in SvO\(_2\) before mean arterial blood pressure, heart rate or pulmonary capillary wedge pressure were noted to change, aiding in the identification of early manifestations of inadequate tissue perfusion.

Measurement of SvO\(_2\) is a reliable and valuable indicator of cardiopulmonary function in the immediate postoperative period, even in infants with complicated repair of cardiac malformations (23–26).

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**Table 2**

<table>
<thead>
<tr>
<th>Time 1 (n = 20)</th>
<th>Time 2 (n = 20)</th>
<th>Time 3 (n = 20)</th>
<th>Time 4 (n = 20)</th>
<th>Time 5 (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean time from CPB (min)</td>
<td>23.4 ± 17.2</td>
<td>52.8 ± 20.5</td>
<td>209.7 ± 33.4</td>
<td>320.0 ± 76.8</td>
</tr>
<tr>
<td>rSO(_2) (%)</td>
<td>64.0 ± 10.6</td>
<td>65.6 ± 10.6</td>
<td>68.1 ± 12.1</td>
<td>67.5 ± 13.4</td>
</tr>
<tr>
<td>SvO(_2) (%)</td>
<td>61.1 ± 9.8</td>
<td>61.6 ± 8.3</td>
<td>64.2 ± 8.8</td>
<td>63.8 ± 9.9</td>
</tr>
<tr>
<td>HR (b/min)</td>
<td>132.0 ± 22.6</td>
<td>131.6 ± 23.3</td>
<td>134.1 ± 22.3</td>
<td>130.8 ± 22.5</td>
</tr>
<tr>
<td>MAP (mmHg)</td>
<td>58.3 ± 8.2</td>
<td>58.7 ± 8.8</td>
<td>65.5 ± 11.7</td>
<td>65.1 ± 8.5</td>
</tr>
<tr>
<td>LAP mean (mmHg)</td>
<td>5.2 ± 1.5</td>
<td>6.0 ± 1.5</td>
<td>6.4 ± 3.0</td>
<td>5.8 ± 2.2</td>
</tr>
<tr>
<td>CVP mean (mmHg)</td>
<td>7.6 ± 3.2</td>
<td>7.9 ± 3.5</td>
<td>6.5 ± 3.2</td>
<td>6.4 ± 2.8</td>
</tr>
<tr>
<td>PAP mean (mmHg)</td>
<td>16.9 ± 7.2</td>
<td>16.8 ± 7.2</td>
<td>16.2 ± 8.3</td>
<td>16.3 ± 9.4</td>
</tr>
<tr>
<td>SaO(_2) (%)</td>
<td>94.3 ± 5.9</td>
<td>94.4 ± 5.3</td>
<td>92.5 ± 6.4</td>
<td>91.1 ± 7.3</td>
</tr>
<tr>
<td>P(_e)CO(_2) (mmHg)</td>
<td>29.4 ± 4.3</td>
<td>32.0 ± 5.4</td>
<td>37.1 ± 6.2</td>
<td>38.8 ± 17.0</td>
</tr>
<tr>
<td>pH(_a)</td>
<td>7.39 ± 0.06</td>
<td>7.41 ± 0.07</td>
<td>7.38 ± 0.05</td>
<td>7.39 ± 0.04</td>
</tr>
<tr>
<td>pHa</td>
<td>7.35 ± 0.07</td>
<td>7.33 ± 0.10</td>
<td>7.34 ± 0.04</td>
<td>7.36 ± 0.05</td>
</tr>
<tr>
<td>PaO(_2) (mmHg)</td>
<td>79.2 ± 56.3</td>
<td>88.3 ± 63.3</td>
<td>110.2 ± 82.4</td>
<td>103.3 ± 76.2</td>
</tr>
<tr>
<td>P(_v)O(_2) (mmHg)</td>
<td>30.8 ± 5.3</td>
<td>31.3 ± 4.9</td>
<td>32.9 ± 4.6</td>
<td>33.1 ± 5.4</td>
</tr>
<tr>
<td>PaCO(_2) (mmHg)</td>
<td>40.2 ± 9.4</td>
<td>38.9 ± 6.7</td>
<td>39.7 ± 5.4</td>
<td>39.5 ± 4.6</td>
</tr>
<tr>
<td>P(_v)CO(_2) (mmHg)</td>
<td>45.0 ± 6.2</td>
<td>46.2 ± 7.0</td>
<td>46.4 ± 6.5</td>
<td>47.1 ± 9.2</td>
</tr>
<tr>
<td>Lactate</td>
<td>1.6 ± 0.8</td>
<td>1.5 ± 0.7</td>
<td>1.5 ± 0.8</td>
<td>1.7 ± 0.9</td>
</tr>
<tr>
<td>Rectal temperature (°C)</td>
<td>36.1 ± 0.5</td>
<td>36.4 ± 0.6</td>
<td>37.2 ± 0.8</td>
<td>37.6 ± 0.8</td>
</tr>
</tbody>
</table>

Data are shown as mean ± SD.

CPB, cardiopulmonary bypass time; CVP, central venous pressure; P\(_e\)CO\(_2\), end tidal carbon dioxide; HR, heart rate; LAP, left atrial pressure; MAP, mean arterial blood pressure; PaCO\(_2\), arterial carbon dioxide tension; PaO\(_2\), arterial oxygen tension; PAP, pulmonary arterial pressure; pHa, arterial pH; pH\(_v\), systemic venous pH; P\(_e\)CO\(_2\), systemic venous oxygen tension; P\(_v\)O\(_2\), systemic venous oxygen tension; rSO\(_2\), cerebral oxygen saturation; SaO\(_2\), arterial oxygen saturation; SvO\(_2\), mixed venous oxygen saturation.

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**Figure 1**

Mean cerebral oxygen saturation (rSO\(_2\)) and mixed venous oxygen saturation (SvO\(_2\)) ± SD for the entire patient population plotted at each of the five time periods. 1 = after weaning from cardiopulmonary bypass, 2 = after sternal closure, 3 = 2 h after arrival to the cardiac ICU, 4 = 4 h after arrival to the cardiac ICU, and 5 = 6 h after arrival in the cardiac ICU. There were 20 measurements (one per patient) at each time-point.
Monitoring of SvO₂ improves survival following the Norwood procedure for stage I palliation of hypoplastic left heart syndrome by providing a more precise estimation of the pulmonary to systemic flow ratio ($Q_p/Q_s$) and earlier identification of decreased cardiac output (24).

Figure 2
Cerebral oxygen saturation (rSO₂ ->), corresponding mixed venous oxygen saturation (SvO₂ --) and arterial oxygen saturation (SaO₂ -o-) at the five time periods: in the operating room after weaning from cardiopulmonary bypass, in the operating room after chest closure, and at 2, 4, and 6 h after arrival in the CICU. The top panel represents two bidirectional Glenn patients (patients 3 and 10), the middle panel two Fontan patients (patients 12 and 15), and the bottom panel two biventricular repair patients (patients 18 and 19).
In this preliminary study with a small number of patients we have shown a correlation between rSO₂ and SvO₂ ($r = 0.67$). The strength of this relationship was similar among the three surgical groups (biventricular repair, BDG, and Fontan): strongest in the biventricular repair patients ($r = 0.71$) and weakest in the Fontan patients ($r = 0.45$). This difference may be related to cerebral venous congestion that patients with single ventricle physiology develop after cavopulmonary anastomosis. Like other tissues, the cerebral vasculature has a greater volume of venous blood than arterial blood. Various sources estimate between 70 (4) and 90% (27) venous blood by volume. Although these volumes cannot be measured in vivo, INVOS cerebral oximeter values correlate well when a 75% venous volume is assumed during volume changes which occur as a result of changes in PaCO₂ (28). The compartment ratio of 75% venous and 25% arterial is likely not constant, but changes continuously in response to changes in cerebral vascular resistance, pulmonary vascular resistance, and tissue oxygen demands. When the compartment ratios change without a change in blood volume, accuracy of the cerebral oximeter does not change. However, comparison between the rSO₂ and SvO₂ does change. We would, thus, anticipate a greater discrepancy of rSO₂ and SvO₂ among the BDG and Fontan patients.

The differences in venous congestion and changes in the ratio between the arterial and venous compartments may have contributed to the intra- and intersubject differences between rSO₂ and SvO₂ that we observed. In addition to the obvious reasons for intersubject variability differences between rSO₂ and SvO₂, which include type of cardiac surgery, type of congenital heart disease, and compartment ratio, there are a number of other possibilities which could play a role. Among these is the position of the sensor around the curved head of children, ambient light, differences in transfusion of blood components, and soft tissue differences that include swelling with impact on absorption. The cerebral rSO₂ values measured in our study revealed a large range of cerebral oxygenation values similar to those found in previous studies which were performed in children with structurally normal hearts (3, 29–31). Prior studies have attributed the large range of cerebral oxygenation values to differences in positioning the sensors on the forehead, as well as individual and

### Table 3

<table>
<thead>
<tr>
<th></th>
<th>r-value</th>
<th>P-value</th>
<th>Bias</th>
<th>Precision</th>
</tr>
</thead>
<tbody>
<tr>
<td>All patients</td>
<td>0.67</td>
<td>&lt;0.0001</td>
<td>+3.3%</td>
<td>16.6%</td>
</tr>
<tr>
<td>Biventricular repair</td>
<td>0.71</td>
<td>&lt;0.0001</td>
<td>-0.3%</td>
<td>11.8%</td>
</tr>
<tr>
<td>BDG patients</td>
<td>0.60</td>
<td>&lt;0.0001</td>
<td>+3.6%</td>
<td>18.4%</td>
</tr>
<tr>
<td>Fontan patients</td>
<td>0.45</td>
<td>0.04</td>
<td>+7.1%</td>
<td>14.0%</td>
</tr>
</tbody>
</table>

BDG, bidirectional Glenn; NIRS, near-infrared spectroscopy; r, correlation coefficient; rSO₂, regional cerebral oxygen saturation; SvO₂, mixed venous oxygen saturation.
age-dependent anatomical and physiological differ-
ences (3). In addition, because the venous proportion
strongly determines near-infrared spectroscopic
measurement of cerebral oxygenation state, the
value is influenced by cerebral blood flow and
cerebral arteriovenous oxygen extraction, which
may differ among subjects and was not measured
in our study. Variations in extracranial blood flow
also affect rSO2 (32–34), although at least 85% of
rSO2 is exclusively from the brain.

The scatter of the Bland–Altman plot demonstra-
tes that the difference between rSO2 and SvO2
increases as the average decreases. Thus, the agree-
ment between the two measurements falls outside
–2 SD when the average of rSO2 and SvO2 is
approximately <50%. This occurred at all three
measurements in the same patient, when rSO2 was
32, 35, and 41%. The cerebral oximeter probe was
inadvertently removed from this patient in the
operating room, and a new probe had to be placed
upon arrival to the CICU. Incorrect placement of the
probe on the forehead or not acquiring a firm seal
with the skin may have created false readings for
rSO2 in this patient. Again, stressing the importance
of securing the sensor position and sensor-skin
coupling by firm taping. Precision in the Bland–
Altman analysis was 16.6% for rSO2 as a predictor of
SvO2 for all patients, which in a clinical setting is a
wide range of variability. The wide limits of agree-
ment between the two methods suggest that it is not
to possible to accurately predict absolute SvO2 for any
given patient based solely on the noninvasive
measurement of cerebral rSO2. Near-infrared spec-
tral imaging, using the INVOS 5100 cerebral oximeter,
is most useful for indicating trends in SvO2 rather
than absolute numbers. Further studies are required
to determine whether this noninvasive method can
provide a reliable trend monitor of SvO2 in a more
diverse group of patients under a wider range of
hemodynamic conditions.

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system and the pediatric Somasensors that were
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