Comparison of two commercially available near-infrared spectroscopy instruments for cerebral oximetry

Technical note

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Two near-infrared spectroscopy (NIRS) devices were compared with regard to their responses to changes in cerebral hemoglobin oxygenation induced by hypoxia and hypercapnia in five healthy volunteers.

Sensors belonging to each NIRS device were placed on opposite sides of the volunteer’s forehead. The INVOS3100A device, approved by the United States Food and Drug Administration, records the percentage of oxyhemoglobin (HbO2) saturation and the investigational NIRO500 device records absolute changes in HbO2, deoxyhemoglobin, and total hemoglobin in micromolar concentrations referenced to an arbitrary baseline. The volunteers breathed separate mixtures of 7% CO2 in O2 and 10% O2 for 5 minutes in random order. Arterial blood pressure, end-tidal CO2 (ETCO2), arterial O2 saturation, and electrocardiographic data were continuously monitored.

Hypercapnia increased (p < 0.01) ETCO2 from 42 ± 2 to 56 ± 3 mm Hg (mean ± standard deviation), resulting in a 7.3 ± 0.2% increase (p < 0.005) in cerebral HbO2 saturation detected by the INVOS3100A device and an 11.6 ± 3 μM increase (p < 0.0008) in HbO2 detected by the NIRO500. Hypoxia decreased (p < 0.01) arterial HbO2 saturation from 98 ± 1 to 87 ± 3%, causing a 5.1 ± 1.2% decrease (p < 0.01) in the percentage of HbO2 saturation detected by the INVOS3100A device and a 9.7 ± 6.3 μM decrease in HbO2 detected by the NIRO500.

The responses of the NIRO500 and the INVOS3100A instruments to changes in cerebral oxygenation resulting from hypercapnia and hypoxia were generally similar; however, responses tended to be greater when recorded by the NIRO500 device, perhaps because, unlike the INVOS3100A device, the NIRO500 does not correct for skin and bone contamination.

Key Words • oxyhemoglobin • cerebrovascular carbon dioxide response • hypercapnia • hypoxia • neuromonitoring

Clinical Material and Methods

Study Protocol

According to an institutional review board–approved protocol, which included informed consent, the medical history of each volunteer was obtained and an EKG study was performed immediately before the study commenced. Exclusion criteria included the presence of any respiratory ailment, a history of respiratory problems, cardiac dysrhythmias, long-term use of medication, and hypertension.

Five volunteers comprised the study population. The NIRO500 optodes and the light-emitting diode sensors of the INVOS3100A device were placed on the head (one on each side of the forehead), while the volunteer was seated and quietly breathing room air through a mouthpiece equipped with nose clips. End-tidal CO2, SaO2, obtained using pulse oximetry, arterial pressure (Finipress; Ohmeda Corp., Columbia, MD), and EKG data were continuously monitored and stored on a personal computer.
The experimental paradigm consisted of five steps. 1) For the first 5 minutes, the volunteer inhaled room air and stable baseline measurements were obtained. 2) At the end of this 5-minute period, unbeknownst to the volunteer, the inspired gas was switched to the first test gas (7% CO₂ in O₂ or 10% O₂/5% CO₂/85% N₂). The order in which the test gases were administered was randomized. While monitoring continued the volunteer breathed the gas for 5 minutes or until voluntarily disconnecting from the mouthpiece. 3) The inspired gas was switched back to room air. During 5 minutes of breathing the normal control gas mixture, stable recordings were again obtained. 4) The inspired gas was switched to the second test gas for 5 minutes. 5) Inhalation of room air resumed.

Acquisition of data from the NIRO500 was accomplished via a serial port into a 486 computer running appropriate software (Software Wedge program; Tal, Inc., Philadelphia, PA). The data from the INVOS3100A were collected on a 3.5-in computer diskette. Statistical analysis of the data was performed using analysis of variance for repeated measures and post hoc analysis was performed using Tukey’s multiple comparison test with statistical significance assigned to a maximum probability value of 0.05.

**In Vivo NIRS**

This technique exploits the fact that light in the near-infrared region (600–1000 nm) penetrates tissue to a depth of 8 to 10 cm. Because the adult head is thicker than 8 to 10 cm, NIRS is used in the reflectance mode, that is, light is passed into and received on the same side of the head. The NIRS technique relies on the fact that Hb is a strong absorber of near-infrared light and that the amount of absorption varies with the degree of Hb oxygenation. The light absorbed by a compound is proportional to the molar absorption coefficient times the log of the ratio of transmitted light to incident light (log₁₀ I/I₀) and the pathlength (Beer Lambert law). In NIRS, the problem for quantitation is the measurement of pathlength. At present, there is no commercially available instrument that continuously measures pathlength, thus allowing for measurement of absolute Hb concentration in micromolar concentrations.

The necessity of knowing the pathlength was resolved for the INVOS3100A instrument by taking the ratio of the HbO₂ absorption signal to absorption at the isobestic point, allowing calculation of the percentage of HbO₂. Normal adult rSO₂ values range from 65 to 75%. The INVOS3100A device uses two detectors positioned 30 and 40 mm from the light-emitting diode. The subtraction of the near from the far signal allows the subtraction of skin and bone, which is effective. An important aspect of this instrument is that the percentage of HbO₂ is an absolute value that correlates with cerebral venous blood.

The NIRO500 device uses four wavelengths with laser diodes to separate the signals of three chromophores: HbO₂, Hb, and cytochrome oxidase. Total Hb is the calculated sum of HbO₂ and dHb. Changes in these measurements are recorded in micromolar concentrations, but are referenced to an arbitrary baseline of zero.

**Results**

Figures 1 and 2 illustrate changes observed in one volunteer. Using the INVOS3100A device (Fig. 1), we observed that a 7% decrease in rSO₂ occurred during hypox-
Cerebral oximetry

### TABLE 2
**Physiological parameters during baseline (room air) normocapnic–normoxic breathing and at the height of hypercapnic–hypoxic breathing**

<table>
<thead>
<tr>
<th>Gas Mixture</th>
<th>MABP (mm Hg)</th>
<th>ETCO₂ (mm Hg)</th>
<th>SaO₂ (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>normocapnic</td>
<td>91 ± 9</td>
<td>42 ± 2</td>
<td>97 ± 1</td>
</tr>
<tr>
<td>hypercapnic</td>
<td>102 ± 9</td>
<td>56 ± 3*</td>
<td>99 ± 1</td>
</tr>
<tr>
<td>normoxic</td>
<td>90 ± 5</td>
<td>39 ± 1</td>
<td>98 ± 1</td>
</tr>
<tr>
<td>hypoxic</td>
<td>87 ± 8</td>
<td>39 ± 1</td>
<td>87 ± 3*</td>
</tr>
</tbody>
</table>

* p < 0.01 compared with corresponding normocapnic or normoxic value.

Changes we observed using the INVOS3100A and NIRO500 devices during hypercapnia were almost two-fold greater than those reported by Grubhofer, et al., who compared the NIRO500 and INVOS3100 instruments during hyperventilation. A four-point decrease in rSO₂ occurred with hyperventilation sufficient to decrease ETCO₂ by 15 mm Hg. The difference in response is probably explained by the well-known difference in cerebrovascular reactivity at CO₂ levels above and below normal. The increase in CBF is much greater when the CO₂ level is above normal than when it is below normal. Other investigators have also shown that NIRS reliably detects changes in cerebral oxygenation during both hypercapnia and hypocapnia and during hypoxia. This technology has shown its usefulness in a variety of clinical applications, including the detection of cerebral ischemia during carotid endarterectomy, regional low-flow perfusion during cardiopulmonary bypass in neonates, and cardiopulmonary bypass. Cerebral oximetry is valuable in the intensive care unit, where it is used to monitor for cerebral vasospasm after subarachnoid hemorrhage and to optimize arterial blood pressure, ETCO₂, or hematocrit (unpublished data).

It must be recognized that cerebral oximetry performed using NIRS does not measure CBF, although in the metabolizing brain, oxygenation and desaturation will occur when CBF increases and decreases, respectively. The fact that it does not measure CBF becomes apparent in the infarcted brain or in cases of brain death in which there is no tissue perfusion. It is not surprising, therefore, that early reports of findings of NIRS and, in particular, data obtained using the INVOS3100A device, did not correlate with the absence of cerebral perfusion. Based on this the authors implied that NIRS does not work. It is also unreasonable to expect that cerebral oximetry performed using NIRS would correlate with internal jugular bulb oximetry in head-injured patients. Head injury itself is very heterogeneous and therefore, jugular bulb O₂ saturation cannot be a standard for comparison with cerebral oximetry performed using NIRS. Although the concept of microvascular shunt placement and the notion that capillary PO₂ is lower than venous PO₂ in areas with shunts are not new, verification of the concept was provided by Ince and Sinaasappel, who used palladium phosphorescence to measure capillary PO₂ and indicated that the PO₂ gap was evidence of shunting in sepsis and shock.

### TABLE 3
**Cerebral HbO₂ responses to hyperoxic hypercapnia and normocapnic hypoxia in five volunteers**

<table>
<thead>
<tr>
<th>Study Group</th>
<th>HbO₂</th>
<th>dHb</th>
<th>tHb</th>
<th>HbO₂</th>
<th>dHb</th>
<th>tHb</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normocapnic</td>
<td>11.6</td>
<td>7.3</td>
<td>11.6</td>
<td>3.3</td>
<td>3.3</td>
<td>3.3</td>
</tr>
<tr>
<td>Hypercapnic</td>
<td>*7.3</td>
<td>4.4</td>
<td>*7.3</td>
<td>1.9</td>
<td>1.9</td>
<td>1.9</td>
</tr>
</tbody>
</table>

* Hyperoxic hypercapnia induced by 7% CO₂ in O₂ and normocapnic hypoxia induced by 10% O₂/5% CO₂. Abbreviations: ΔM = change in micromolar concentration; ∆SO₂ = change in percentage of HbO₂.

† p < 0.0008 compared with zero.
‡ p < 0.005 compared with zero.

### Discussion

The responses we observed using the NIRO500 instrument were generally similar to but somewhat greater than those observed using the INVOS3100A device. This may be due to the fact that, unlike the INVOS3100A, the NIRO500 makes no correction for skin and bone contamination. The subtraction technique used with the INVOS3100A and the more recent models, the 4100 and 5100, effectively removes contamination by skin and bone. These findings are consistent with our observations during carotid endarterectomy in an earlier study in which the INVOS3100A was four times less sensitive to extracranial contamination than the NIRO500.
Disclosure

Dr. Nemoto serves as a consultant to Somanetics Corp., the manufacturers of the INVOS cerebral oximeter. He receives unrestricted educational grants and an equipment loan from Somanetics Corp. to conduct clinical applications research.

References


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