

Impact of Extracranial Contamination on Regional Cerebral Oxygen Saturation

A Comparison of Three Cerebral Oximetry Technologies

Sophie N. Davie, B.Sc.,* Hilary P. Grocott, M.D., F.R.C.P.C.†

ABSTRACT

Background: Cerebral oximetry is a noninvasive technology using near-infrared spectroscopy (NIRS) to estimate regional cerebral oxygen saturation. Although NIRS cerebral oximetry is being increasingly used in many clinical settings, inter-device technologic differences suggest potential variation in the ability to accurately acquire brain oxygenation signals. The primary objective of this study was to determine if NIRS-derived regional cerebral oxygen saturation measurements accurately account for oxygen saturation contamination from extracranial tissue.

Methods: Twelve healthy volunteers had each of three NIRS devices (FORE-SIGHT [CAS Medical Systems Inc; Bradford, CT], INVOS 5100C-PB [Covidien; Boulder, CO], and EQUANOX Classic 7600 [Nonin Medical Inc; Plymouth, MN]) randomly applied to the forehead. After this, a circumferential pneumatic head cuff was positioned such that when inflated, hypoxia-ischemia would be produced in the extracranial scalp tissue beneath the NIRS cerebral oximeters. Comparisons among the three devices were made of the NIRS measurements before and following hypoxia-ischemia produced in the scalp tissue with inflation of the head cuff.

Results: The induction of extracranial hypoxia-ischemia resulted in a significant reduction in regional cerebral oxygen saturation measurements in all three NIRS devices studied. At 5 min postinflation of the pneumatic head cuff, the INVOS demonstrated a $16.6 \pm 9.6\%$ (mean \pm SD) decrease

What We Already Know about This Topic

- Cerebral oximetry employing near-infrared spectroscopy (NIRS) is used clinically to monitor cerebral oxygen saturation
- The impact of contamination by extracranial tissues on the accuracy of this technology is unknown

What This Article Tells Us That Is New

- The effect of scalp tissue hypoxia-ischemia on the accuracy of three commercial NIRS cerebral oximeters was compared in healthy volunteers
- The three cerebral oximeters had variable sensitivity to extracranial tissue contamination, as evident in significant reductions in apparent cerebral oxygen saturation with scalp ischemia

from its baseline ($P = 0.0001$), the FORE-SIGHT an $11.8 \pm 5.3\%$ decrease from its baseline ($P < 0.0001$), and the EQUANOX a $6.8 \pm 6.0\%$ reduction from baseline ($P = 0.0025$).

Conclusions: Extracranial contamination appears to significantly affect NIRS measurements of cerebral oxygen saturation. Although the clinical implications of these apparent inaccuracies require further study, they suggest that the oxygen saturation measurements provided by cerebral oximetry do not solely reflect that of the brain alone.

CEREBRAL oximetry using near-infrared spectroscopy (NIRS) is a noninvasive technique used to estimate regional cerebral oxygen saturation (rSO₂).¹ Several studies have demonstrated an increased incidence of adverse perioperative outcomes in patients who demonstrate substantial cerebral oxygen desaturation during surgery. These negative outcomes include neuropsychological dysfunction,² prolonged hospital length of stay,³ major organ morbidity, and mortality.⁴ Indeed, even low baseline measurements of rSO₂ have demonstrated a relationship to adverse outcomes.⁴⁻⁶ Although cerebral oximetry is increasingly being used in many clinical settings,^{7,8} it has yet to be adopted as a clinical standard of care. However, if effective NIRS-guided interventional strategies could be instituted, improvement in perioperative outcomes might be possible.⁵

When placed on the forehead, near-infrared light emanating from a cerebral oximeter sensor array passes through the

* Medical Student, † Professor, Department of Anesthesia & Perioperative Medicine, University of Manitoba, Winnipeg, Manitoba, Canada.

Received from the Department of Anesthesia & Perioperative Medicine, University of Manitoba, Winnipeg, Manitoba, Canada. Submitted for publication July 22, 2011. Accepted for publication January 6, 2012. Support was provided solely from institutional and/or departmental sources. Presented in part at the annual meeting of Society of Cardiovascular Anesthesiologists, April 30–May 4, 2011, Savannah, Georgia, and the annual meeting of the Canadian Anesthesiologists Society, June 24–28, 2011, Toronto, Ontario, Canada.

Address correspondence to Dr. Grocott: St. Boniface Hospital, CR3008 – 369 Tache Avenue, Winnipeg, Manitoba, Canada R2H 2A6. hgrocott@sbgh.mb.ca. Information on purchasing reprints may be found at www.anesthesiology.org or on the masthead page at the beginning of this issue. ANESTHESIOLOGY's articles are made freely accessible to all readers, for personal use only, 6 months from the cover date of the issue.

Copyright © 2012, the American Society of Anesthesiologists, Inc. Lippincott Williams & Wilkins. Anesthesiology 2012; 116:834–40

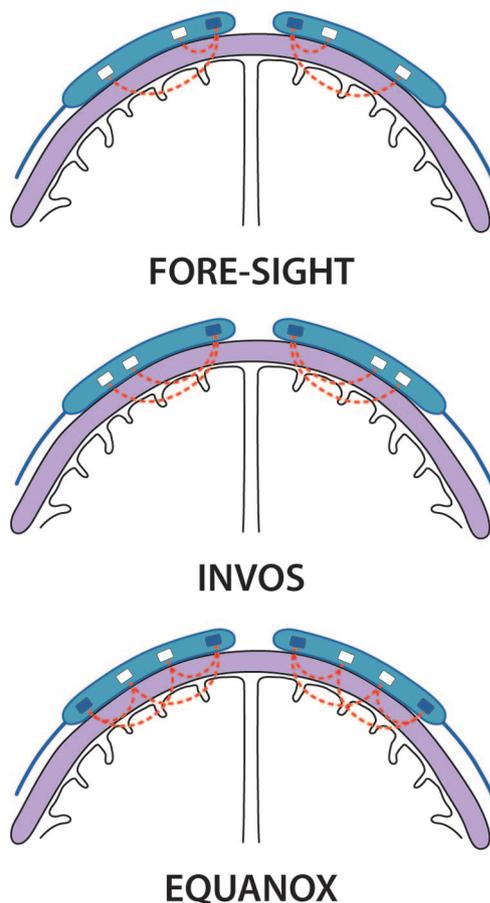


Fig. 1. Schematic diagram demonstrating the curvilinear photon path through the superficial (*i.e.*, extracranial) and cerebral tissues represented by the different cerebral oximeters used during the study. The cerebral oximetry optode array, placed on the subject forehead, contains light emitters (blue boxes) and light detectors (white boxes). Light (red arrows) from the light emitter follows a curvilinear path through both the extracranial tissue and cerebral tissue before resurfacing. The depth of tissue interrogated is proportional to the distance between the optode light emitter and detector. Automated algorithmic subtraction results in the cerebral oximeter displaying the cerebral tissue oxygen saturation.

extracranial tissues (such as the scalp and skull) to reach the underlying cerebral tissue.^{9,10} Hemoglobin is a light-absorbing chromophore and differing wavelengths of near-infrared light are absorbed when it is either oxidized or reduced.^{11,12} Hemoglobin oxygen saturation in cerebral tissue can be determined by measuring this differential absorption of various wavelengths of light as they follow a curvilinear path through the extracranial and cerebral tissue before being reflected (fig. 1).¹⁰ To distinguish between cerebral oxygen saturation and extracranial contamination, commercial cerebral oximeters employ a process of spatial resolution. Spatial resolution is, in part, based upon the principle that the depth of tissue interrogated is proportional to the distance between the optode light emitter and detector.¹³ Therefore, by using two detectors at different locations (*i.e.*, both near and distant from the light source), along with automated algorithmic subtraction

of this data, a final value representing the underlying cerebral tissue oxygen saturation results, theoretically free from any extracranial contamination.¹⁴

There are an increasing number of NIRS devices available for clinical use. The various devices all have intrinsic spatial resolution capabilities, but they differ in numerous important aspects related to the acquisition of their cerebral oxygen saturation measurements. Variations include the type of light source, the wavelengths of light emitted, and the distance between the various light emitters and detectors. Detector distances are 30 mm and 40 mm from the light source for the INVOS 5100C-PB (Covidien; Boulder, CO) and 15 mm and 50 mm for the FORE-SIGHT (CAS Medical Systems Inc; Branford, CT).¹ The EQUANOX Classic 7600 (Nonin Medical Inc; Plymouth, MN) contains two sets of spatially separate light sources as well as two light detectors arranged so that the distal light detector for one light source is colocated with the proximal detector for the other (fig. 1). For the EQUANOX, emitter to detector distances are 20 mm and 40 mm. All of these interdevice technologic differences suggest a potential for variation in the ability to acquire and spatially resolve rSO_2 signals.

The purpose of this study was to determine if NIRS-guided rSO_2 measurements from these cerebral oximeters are able to accurately account for extracranial contamination. We hypothesized that the cerebral oximeter with the shortest distance between the respective near and far field light detectors may have difficulty distinguishing extracranial from cerebral tissue, and thus may be more susceptible to extracranial contamination.

Materials and Methods

After approval from the institutional Research Ethics Board (University of Manitoba, Winnipeg, Canada) and written informed consent, volunteers aged 18–40 yr were enrolled in this study. Study subjects were healthy, with no known history of hypertension, diabetes, or other neurologic or cardiac disease.

With the study subject seated comfortably, a noninvasive blood pressure cuff (IntelliVue MP90; Philips Medizinsysteme, Boeblingen, Germany) was applied to the left arm, a peripheral pulse oximeter (Adult Reusable SpO_2 Sensor M1191B, Philips Medizinsysteme) was placed on the right index finger, and a scalp surface pulse oximeter (MaxfastTM; Covidien, Mansfield, MA) was placed on the left side of the forehead. A randomization envelope was then opened to determine the order in which the individual cerebral oximetric devices being studied would be applied. Three different NIRS cerebral oximeters were used in this study: the INVOS 5100C-PB, the FORE-SIGHT, and the EQUANOX Classic 7600. After cleaning the skin with an alcohol swab and ensuring that all hair was displaced from beneath the sensor, the first NIRS device sensor array was applied to the right side of the forehead. A custom-made pneumatic head cuff was then placed circumferentially around the head at the

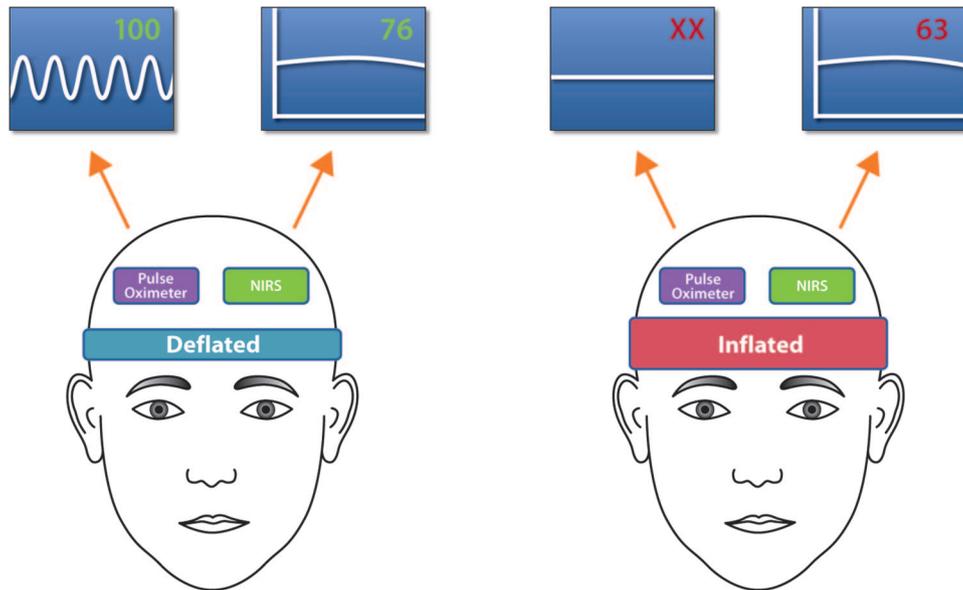


Fig. 2. Schematic diagram of placement of the circumferential pneumatic head cuff, surface scalp pulse oximeter, and cerebral oximetry optode array. With inflation of the head cuff, a loss of blood flow to the scalp results in a loss of signal from the surface scalp pulse oximeter and tissue hypoxia-ischemia. NIRS = near-infrared spectroscopy.

level of the forehead. The cuff was positioned below the occipital protuberance and above the supraorbital prominence to prevent it from moving during inflation. Proper placement of the cuff ensured that the cerebral and pulse oximeter sensors were above (*i.e.*, distal to) the cuff such that with its inflation, hypoxia-ischemia would be produced in the superficial tissues beneath the oximeters. After the placement of these devices, a 2-min stabilization period was allowed before recording baseline measurements of blood pressure, heart rate, rSO_2 , as well as scalp ($SscO_2$) and finger (SaO_2) oxygen saturations.

Once these baseline measurements were recorded, extracranial hypoxia-ischemia was induced by inflation of the head cuff to a pressure that exceeded the subject's systolic blood pressure. Cessation of blood flow to the superficial extracranial tissue, and therefore induction of local scalp tissue hypoxia-ischemia, was confirmed by a loss of the $SscO_2$ signal (fig. 2). Once extracranial hypoxia was established, the head cuff remained inflated for 5 min with rSO_2 and other physiologic measurements recorded at 2 and 5 min of inflation. Following this, the head cuff was deflated, allowing the superficial tissues to be reperfused. Measurements were recorded again after 1 min of the 5 min of reperfusion between the successive inflation-deflation series. This inflation-deflation series was repeated in triplicate with each cerebral oximetry device studied separately (fig. 3), for a total of nine measurements per subject, with the data averaged at each time-point for each subject.

Statistical Analysis

All data are presented as mean \pm standard deviation (SD). Comparisons of the rSO_2 and other physiologic parameters between baseline and the various inflation time points were per-

formed for each device, as well as between different devices, using a paired two-tailed Student *t* test (SAS version 9.2; SAS Institute Inc., Cary, NC). Twelve pair-wise comparisons of rSO_2 changes were made to analyze the differences among devices. Adjustment for these multiple comparisons was performed using the Dunn-Šidák correction,¹⁵ with a $P < 0.0043$ being considered statistically significant. The upper and lower limits of the 95% CIs were also calculated.

Results

Study subjects ($n = 12$) were 24 ± 2.6 yr of age; four (33%) were male and eight (67%) were female. Throughout the cycle of head cuff inflations and deflations, heart rate, blood pressure, and SaO_2 changes did not show statistical significance. However, following each head cuff inflation, $SscO_2$ signals were lost, coincident with the ensuing scalp hypoxia-ischemia, and NIRS-derived rSO_2 measurements decreased. With deflation of the head cuff and scalp reperfusion, $SscO_2$ and rSO_2 measurements returned to baseline. Table 1 outlines the detailed data recorded at the various time-points of the study.

The induction of extracranial hypoxia-ischemia demonstrated a significant decrease in rSO_2 measurements in all three devices studied at both 2 and 5 min postinflation of the pneumatic head cuff. The relative change from baseline for the INVOS was $13.9 \pm 8.0\%$ at 2 min ($P = 0.0001$, 95% CI, 8.9–19.0%) and $16.6 \pm 9.6\%$ at 5 min ($P = 0.0001$, 95% CI, 10.5–22.7%). The FORE-SIGHT demonstrated a $10.3 \pm 5.2\%$ decrease from baseline at 2 min ($P < 0.0001$, 95% CI, 7.0–13.6%) and an $11.8 \pm 5.3\%$ decrease at 5 min ($P < 0.0001$, 95% CI, 4.4–15.1%). The EQUANOX demonstrated a $6.6 \pm 4.6\%$ ($P = 0.0004$, 95% CI, 3.7–9.6%)

Serial head cuff inflations/deflations

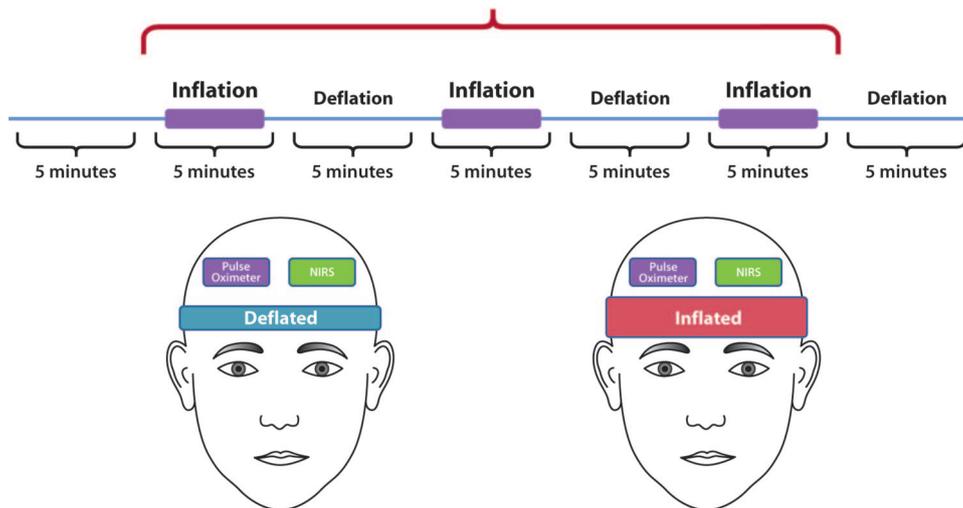


Fig. 3. Flow diagram of the experimental time course representing the series of head cuff inflations and deflations. NIRS = near-infrared spectroscopy.

decrease from baseline at 2 min and a $6.8 \pm 6.0\%$ reduction from baseline ($P = 0.0025$, 95% CI, 2.9–10.6%) at 5 min postinflation of the head cuff. Figure 4 illustrates the differences between rSO_2 measurements at baseline and 5 min postinflation for the three devices.

Interdevice comparisons were also performed at both 2 and 5 min postinflation of the head cuff to determine if there were significant differences between the devices with respect to the influence of extracranial contamination. Compared with one another, the INVOS and FORE-SIGHT did not

show significant different changes from their respective baselines at either time point ($P = 0.0443$ at 2 min; $P = 0.0250$ at 5 min). The INVOS and EQUANOX, however, demonstrated significantly different degrees of extracranial contamination at both 2 and 5 min ($P = 0.0021$, $P = 0.0019$, respectively). Similarly, the EQUANOX and FORE-SIGHT had a significant difference in their changes from baseline at 2 min ($P = 0.0040$), though not at 5 min ($P = 0.0487$) (fig. 5). However, based on the degree of change from baseline that occurred with the induction of the extracranial hypoxia-

Table 1. Physiologic and Cerebral Oximetry Results

	HR (bpm)	P Value (HR)	MAP (mmHg)	P Value (MAP)	Sao ₂ (%)	P Value (Sao ₂)	SscO ₂ (%)	P Value (SscO ₂)	rSO ₂ (%)	P Value (rSO ₂)
Baseline										
FORE-SIGHT	73 ± 9	—	88 ± 10	—	99 ± 1	—	99 ± 1	—	73 ± 4	—
INVOS	73 ± 7	—	87 ± 9	—	99 ± 1	—	98 ± 1	—	76 ± 10	—
EQUANOX	72 ± 8	—	88 ± 8	—	99 ± 1	—	99 ± 1	—	75 ± 9	—
2 min of inflation										
FORE-SIGHT	71 ± 6	0.4970*	89 ± 9	0.7997*	99 ± 1	0.9261*	NR	—	65 ± 4	<0.0001*
INVOS	73 ± 6	0.9468*	89 ± 8	0.5620*	99 ± 1	0.1875*	NR	—	65 ± 7	0.0001*
EQUANOX	70 ± 7	0.5641*	88 ± 10	0.9310*	99 ± 1	1.0000*	NR	—	70 ± 7	0.0007*
5 min of inflation										
FORE-SIGHT	73 ± 4	0.8502†	90 ± 10	0.6246†	99 ± 1	1.0000†	NR	—	64 ± 5	<0.0001†
INVOS	73 ± 5	0.9466†	88 ± 8	0.7543†	99 ± 1	0.4880†	NR	—	63 ± 8	0.0001†
EQUANOX	70 ± 6	0.6605†	91 ± 9	0.3753†	99 ± 1	0.7600†	NR	—	69 ± 6	0.0035†
Reperfusion										
FORE-SIGHT	73 ± 5	0.9048‡	89 ± 11	0.7678‡	99 ± 1	1.0000‡	97 ± 3	0.2062‡	73 ± 4	0.8478‡
INVOS	75 ± 6	0.4688‡	88 ± 9	0.8007‡	99 ± 1	0.2829‡	98 ± 2	0.5807‡	78 ± 10	0.5582‡
EQUANOX	73 ± 8	0.7942‡	90 ± 10	0.5092‡	99 ± 1	0.3083‡	98 ± 1	0.5373‡	76 ± 11	0.8292‡

Data are presented as mean ± SD where appropriate.

* Comparative *P* values between baseline and 2 min following head cuff inflation of the regional cerebral oxygen saturation and other physiologic measurements. † Comparative *P* values between baseline and 5 min following head cuff inflation of the regional cerebral oxygen saturation and other physiologic measurements. ‡ Comparative *P* values between baseline and reperfusion of the extracranial tissue of the rSO_2 and other physiologic measurements.

bpm = beats/min; HR = heart rate; MAP = mean arterial pressure; NR = no reading, because head cuff was inflated until scalp pulse oximeter signal was lost because of cessation of scalp blood flow; rSO_2 = regional cerebral oxygen saturation; Sao_2 = finger arterial oxygen saturation; $SscO_2$ = scalp arterial oxygen saturation.

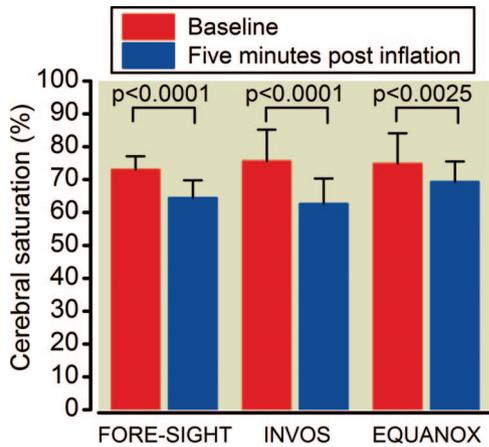


Fig. 4. Regional cerebral oxygen saturation measurements of FORE-SIGHT, INVOS 5100C-PB, and EQUANOX Classic 7600, the three near-infrared spectroscopy devices studied at baseline and at 5 min postinflation of a head cuff. All three of the cerebral oximeters demonstrated a statistically significant reduction in cerebral oxygen saturation at 5 min postinflation. Error bars represent the standard deviations.

ischemia, the various interdevice differences indicate a rank order of extracranial contamination, with the EQUANOX, FORE-SIGHT, and INVOS having increasing amounts of contamination in their respective rSO_2 measurements.

Discussion

These results demonstrate that NIRS measurements of rSO_2 are affected by extracranial tissue oxygen saturation. All three cerebral oximeters studied demonstrated a significant reduction in rSO_2 measurements following inflation of an occlusive head cuff positioned circumferentially around the forehead to induce extracranial tissue desaturation. This raises a question as to the accuracy of these devices to measure intracerebral oxygen saturation.

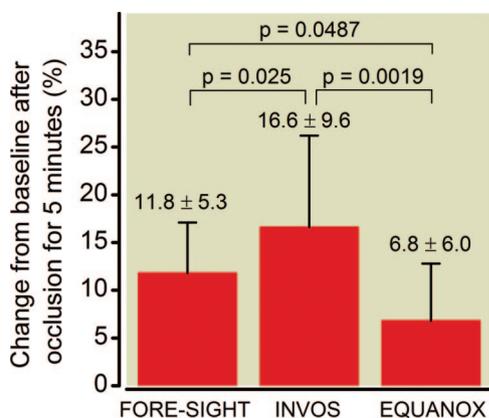


Fig. 5. Percent change from the baseline regional cerebral oxygen saturation measurement of FORE-SIGHT, INVOS 5100C-PB, and EQUANOX Classic 7600, the three near-infrared spectroscopy devices studied after occlusion of the head cuff for 5 min. *P* values of interdevice statistical analyses are also shown. Error bars represent the standard deviations.

NIRS devices can provide continuous information about rSO_2 in a noninvasive manner. These characteristics are unique to this technology and, as such, NIRS is being increasingly used in many clinical settings.⁸ For example, during cardiac surgery, situations may arise where cerebral hypoperfusion occurs despite otherwise normal physiologic parameters, such as blood pressure, heart rate, and SaO_2 .^{8,16} In these instances, NIRS devices may potentially detect important but clinically silent episodes of cerebral desaturation.¹⁷ If rSO_2 measurements are intended to guide clinical interventions, it is crucial that NIRS provides accurate information that appropriately reflects the cerebral environment, free from extracranial contamination.

Our results indicate that the spatial resolution of these devices is not sufficiently accurate to fully account for extracranial contamination. Indeed, if this technology were to appropriately focus on cerebral tissue alone, changes such as the induction of hypoxia in the extracranial environment should not have an impact on rSO_2 measurements. Interdevice technologic differences may explain the discrepancy among the devices. For example, the INVOS and EQUANOX demonstrated a significant difference in their degrees of extracranial contamination. This may be because of the variations in their sensor array design, including the emitter-to-detector distances and the number of light emitters present. The INVOS demonstrates a relatively short distance between its near and far field detectors compared with the EQUANOX, and consistent with our hypothesis, this appears to be insufficient to appropriately distinguish extracranial from cerebral tissue. In addition, the EQUANOX contains two light emitters, which may provide further accuracy by allowing this device to account for extracranial tissue variation and contamination throughout the entire curvilinear light path. It should be noted that the potential for NIRS technology (specifically, the INVOS device) to be affected by extracranial contamination was first described in 1994 by Germon *et al.*¹⁸ In similar fashion, they demonstrated an 18% reduction (compared with our 14%) in cerebral saturation with scalp ischemia. However, this early study was limited by its smaller sample size ($n = 8$) and lack of independent confirmation that scalp ischemia was reliably being produced.

These present results may partly explain the observation that the administration of vasoconstrictors such as phenylephrine have paradoxically been reported to result in cerebral desaturation.¹⁹ This is counterintuitive, because increases in blood pressure should maintain cerebral perfusion if autoregulatory mechanisms are intact. However, if the vasoconstrictor is working principally on the extracranial tissue, then reductions in rSO_2 could simply be a result of extracranial desaturation from the peripheral vasoconstriction and may not be a result of true cerebral desaturation. This concept is further supported by the results of a study performed by Johnston *et al.*, who determined that cerebral blood flow

(CBF) was not affected by phenylephrine in a dog model of cardiopulmonary bypass (CPB).²⁰

Despite an increasing use of cerebral oximetry, there are a limited number of prospective randomized controlled trials analyzing NIRS-guided therapy. Moreover, the studies that have been published to date provide inconsistent results. For example, a study of cardiac surgery patients performed by Murkin *et al.* demonstrated beneficial results for NIRS-guided interventions.⁵ In their study, patients were either conventionally managed without the use of NIRS or had a NIRS-directed algorithm to optimize cerebral saturation. Compared with the control group, the interventional group demonstrated a decreased incidence of major organ morbidity and mortality.⁵ However, a similar study by Slater *et al.* did not find that a NIRS-guided intervention improved patient outcome. Indeed, the main endpoint of their study, cognitive decline, had a similar incidence in both interventional and control groups.³ The study by Slater did, however, reconfirm an overall association between decreases in rSO₂ and adverse neurologic outcome when both groups were combined as a single cohort.³ Confounding these and other NIRS trials may be a suboptimal rSO₂ signal-to-noise ratio. That is, if one is attempting to use NIRS to guide therapy and improve outcome, but there is inherent contamination of the cerebral signal, this may adversely affect the success of the guided therapy.

The clinical significance of this apparent contamination is not certain. For example, it has recently been argued that the brain may serve as an index organ for overall tissue perfusion and related oxygen supply and demand ratios.¹ This proposed ability of cerebral oximetry measurements to represent other tissue perfusion may actually be a consequence of the extracranial contribution, making this contamination beneficial. Also, if one is using rSO₂ only to interrogate the symmetry of CBF, such as during aortic arch surgery,²¹ some contamination is likely clinically insignificant. That is, interruptions in carotid blood flow, for example, would be detected by both a reduction in intracranial and extracranial oxygen saturation.

However, the clinical significance of the extracranial contamination present is dependent on the protocol used in NIRS-guided interventions. For instance, the trial reported by Murkin *et al.* set a threshold value for initiating interventions as a relative change from baseline as opposed to an absolute rSO₂ measurement.⁵ The presence of extracranial contamination is arguably less important in this context, especially if it is assumed that the proportion of extracranial blood flow compared with intracranial blood flow remains constant. However, during CPB, when blood is shunted away from nonessential organs such as the extracranial tissue, the ratio of cerebral to extracranial blood flow is less constant, altering the signal-to-noise ratio in NIRS measurements of rSO₂. In this case, even interventions based on relative cerebral oximetry measurements could be erroneous.

The importance of optimizing NIRS accuracy was again highlighted by a recent cardiac surgery study by Heringlake

et al., which demonstrated a relationship between low preoperative baseline cerebral saturation and postoperative morbidity and mortality.⁶ One of the potentially troublesome issues with this study, however, was that the investigators used the cerebral oximeter we found to have the greatest amount of contamination. Therefore, the absolute rSO₂ thresholds that they used in their risk models may have been inaccurate. It is possible that the associations they described may have been more robust had either of the other NIRS technologies less prone to contamination been used. At the very least, our data raises questions as to whether these technologies can be considered similar in terms of the measurements they display.

Another example of the potential importance of inaccuracies in rSO₂ is related to work that recently described using NIRS technology for CBF autoregulatory measurement during CPB. Large portions of patients undergoing CPB have underlying cerebral vascular disease, and therefore may have abnormal limits of CBF autoregulation.²² There is an emerging concept of targeting mean arterial pressure specifically to a patient's CBF autoregulatory range, as opposed to a set mean arterial pressure value for all patients, which may help protect from cerebral hypoperfusion.²³ Recent studies have suggested that NIRS measurements of rSO₂ reliably represent CBF autoregulation in patients undergoing CPB.²² If, however, rSO₂ measurements contain contamination from extracranial tissue, the specificity of these measurements to determine cerebral autoregulation becomes questionable.

There were several limitations in our study. In the past, NIRS cerebral oximeters have been calibrated and compared with measures of jugular venous oxygen saturation.¹⁰ We did not have jugular venous oxygen saturation measurements to determine if cuff inflation caused jugular venous desaturation. However, because of the anatomic separation of intracranial and extracranial blood flow, this seems unlikely. Another limitation relates to the pain induced by extracranial tissue hypoxia. This painful stimulus could trigger physiologic increases in blood pressure and heart rate, potentially altering rSO₂. However, this is unlikely since our changes occurred almost immediately following head cuff inflation. If the decreases were because of an adverse physiologic response, reductions would have proceeded gradually. Furthermore, blood pressure, heart rate, and SaO₂ were monitored continuously and no significant changes in these parameters were observed (table 1). It could also be questioned whether occluding the extracranial tissue could cause scalp tissue edema, which may alter optical path lengths and explain changes in rSO₂ measurements.²⁴ However, this is also unlikely because any edema would take a substantial amount of time to develop, and does not explain the abrupt decrease in rSO₂ measurements. Lastly, the healthy study population chosen may limit the clinical applicability of our data. Cerebral oximetry is most commonly used in clinical settings such as cardiac surgery, where patients are older and have several

comorbidities. Therefore, further corroboration of these results in an elderly surgical population is required.

In summary, this study analyzed whether extracranial contamination in the form of hypoxia would alter rSO_2 measurements of the three currently available NIRS devices. The INVOS, FORE-SIGHT, and EQUANOX all demonstrated significant amounts of contamination. These findings demonstrate that NIRS devices are not measuring oxygen saturation from the brain alone.

The authors are very grateful for the assistance received from Ingrid Heinrichs (Administrative Assistant, University of Manitoba, Winnipeg, Manitoba, Canada) during manuscript preparation as well as from Chris Eselgroth (Graphic Artist, Asheville, North Carolina) for assistance with the preparation of the figures.

References

- Murkin JM: Cerebral oximetry: Monitoring the brain as the index organ. *ANESTHESIOLOGY* 2011; 114:12-3
- Yao FS, Tseng CC, Ho CY, Levin SK, Illner P: Cerebral oxygen desaturation is associated with early postoperative neuropsychological dysfunction in patients undergoing cardiac surgery. *J Cardiothorac Vasc Anesth* 2004; 18:552-8
- Slater JP, Guarino T, Stack J, Vinod K, Bustami RT, Brown JM 3rd, Rodriguez AL, Magovern CJ, Zaubler T, Freundlich K, Parr GV: Cerebral oxygen desaturation predicts cognitive decline and longer hospital stay after cardiac surgery. *Ann Thorac Surg* 2009; 87:36-44
- Fischer GW, Lin HM, Krol M, Galati MF, Di Luozzo G, Griep RB, Reich DL: Noninvasive cerebral oxygenation may predict outcome in patients undergoing aortic arch surgery. *J Thorac Cardiovasc Surg* 2011; 141:815-21
- Murkin JM, Adams SJ, Novick RJ, Quantz M, Bainbridge D, Iglesias I, Cleland A, Schaefer B, Irwin B, Fox S: Monitoring brain oxygen saturation during coronary bypass surgery: A randomized, prospective study. *Anesth Analg* 2007; 104:51-8
- Heringlake M, Garbers C, Käbler JH, Anderson I, Heinze H, Schön J, Berger KU, Dibbelt L, Sievers HH, Hanke T: Preoperative cerebral oxygen saturation and clinical outcomes in cardiac surgery. *ANESTHESIOLOGY* 2011; 114:58-69
- Grocott HP, Davie S, Fedorow C: Monitoring of brain function in anesthesia and intensive care. *Curr Opin Anaesthesiol* 2010; 23:759-64
- Denault A, Deschamps A, Murkin JM: A proposed algorithm for the intraoperative use of cerebral near-infrared spectroscopy. *Semin Cardiothorac Vasc Anesth* 2007; 11:274-81
- Jöbsis FF: Noninvasive, infrared monitoring of cerebral and myocardial oxygen sufficiency and circulatory parameters. *Science* 1977; 198:1264-7
- Kim MB, Ward DS, Cartwright CR, Kolano J, Chlebowski S, Henson LC: Estimation of jugular venous O₂ saturation from cerebral oximetry or arterial O₂ saturation during isocapnic hypoxia. *J Clin Monit Comput* 2000; 16:191-9
- Chance B: Spectrophotometry of intracellular respiratory pigments. *Science* 1954; 120:767-75
- Watzman HM, Kurth CD, Montenegro LM, Rome J, Steven JM, Nicolson SC: Arterial and venous contributions to near-infrared cerebral oximetry. *ANESTHESIOLOGY* 2000; 93:947-53
- Germon TJ, Evans PD, Barnett NJ, Wall P, Manara AR, Nelson RJ: Cerebral near infrared spectroscopy: Emitter-detector separation must be increased. *Br J Anaesth* 1999; 82:831-7
- Edmonds HL Jr: Pro: All cardiac surgical patients should have intraoperative cerebral oxygenation monitoring. *J Cardiothorac Vasc Anesth* 2006; 20:445-9
- Dunn O: Multiple comparisons among means. *J Am Stat Assoc* 1961; 56:52-64
- Taillefer MC, Denault AY: Cerebral near-infrared spectroscopy in adult heart surgery: Systematic review of its clinical efficacy. *Can J Anaesth* 2005; 52:79-87
- Wahr JA, Tremper KK, Samra S, Delpy DT: Near-infrared spectroscopy: Theory and applications. *J Cardiothorac Vasc Anesth* 1996; 10:406-18
- Germon TJ, Kane NM, Manara AR, Nelson RJ: Near-infrared spectroscopy in adults: Effects of extracranial ischaemia and intracranial hypoxia on estimation of cerebral oxygenation. *Br J Anaesth* 1994; 73:503-6
- Nissen P, Brassard P, Jørgensen TB, Secher NH: Phenylephrine but not ephedrine reduces frontal lobe oxygenation following anesthesia-induced hypotension. *Neurocrit Care* 2010; 12:17-23
- Johnston WE, DeWitt DS, Vinten-Johansen J, Stump DA, Prough DS: Phenylephrine does not reduce cerebral perfusion during canine cardiopulmonary bypass. *Anesth Analg* 1994; 79:14-8
- Cheng HW, Chang HH, Chen YJ, Chang WK, Chan KH, Chen PT: Clinical value of application of cerebral oximetry in total replacement of the aortic arch and concomitant vessels. *Acta Anaesthesiol Taiwan* 2008; 46:178-83
- Brady K, Joshi B, Zweifel C, Smielewski P, Czosnyka M, Easley RB, Hogue CW Jr: Real-time continuous monitoring of cerebral blood flow autoregulation using near-infrared spectroscopy in patients undergoing cardiopulmonary bypass. *Stroke* 2010; 41:1951-6
- Grocott HP: Avoid hypotension and hypoxia: An old anesthetic adage with renewed relevance from cerebral oximetry monitoring. *Can J Anaesth* 2011; 58:697-702
- Yoshitani K, Kawaguchi M, Miura N, Okuno T, Kanoda T, Ohnishi Y, Kuro M: Effects of hemoglobin concentration, skull thickness, and the area of the cerebrospinal fluid layer on near-infrared spectroscopy measurements. *ANESTHESIOLOGY* 2007; 106:458-62