PROJECT TITLE: The impact of extracranial contamination on regional cerebral oxygen
saturation: a comparison of three cerebral oximetry technologies

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SUMMARY:

Cerebral oximetry is a non-invasive technology using near-infrared spectroscopy (NIRS) to
measure regional cerebral oxygen saturation (rSO_2). 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 rSO_2 measurements accurately account for oxygen
saturation contamination from extracranial tissue.

Twelve healthy volunteers had three (the FORE-SIGHT, INVOS, and EQUANOX) NIRS
devices 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 scalp extracranial
tissue beneath the NIRS cerebral oximeters. Comparisons among the three devices were made of
the NIRS measurements prior to and following hypoxia-ischemia produced in the scalp tissue
with inflation of the head cuff.

The induction of extracranial hypoxia-ischemia resulted in a significant reduction in rSO_2
measurements in all three NIRS devices studied. At 5 minutes post-inflation of the pneumatic
head cuff, the INVOS demonstrated a $16.6 \pm 9.6\%$ decrease 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 ($p = 0.0035$).

Therefore, extracranial contamination appears to significantly affect NIRS measurements of
cerebral oxygen saturation. Although the clinical implications of these apparent inaccuracies
require further study, it suggests that the oxygen saturation measurements provided by cerebral
oximetry do not solely reflect that of the brain alone.

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Sophie Davie

Introduction

       Cerebral oximetry using near-infrared spectroscopy (NIRS) is a non-invasive technique used to measure regional cerebral oxygen saturation (rSO$_2$), thus providing information pertaining to cerebral oxygen supply:demand and oxygen extraction ratios.[1] Several studies have demonstrated an increased incidence of adverse perioperative outcomes in patients who demonstrate significant cerebral oxygen desaturation during surgery. These negative outcomes include neuropsychological dysfunction,[2] increased length of hospital stay,[3] and major organ morbidity and mortality.[4] Indeed, even baseline measurements of rSO$_2$ have demonstrated a relationship to adverse outcomes.[4-6] Although cerebral oximetry is being increasingly 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, significant improvement in perioperative outcomes might be possible.[4]

       When placed on the forehead, near-infrared light emanating from a cerebral oximeter optode array passes through the extracranial tissues (such as the scalp and skull) to reach the underlying cortical frontal lobe of the brain.[9,10] It is in the near-infrared light range that the oxygen transporting chromophore, hemoglobin, absorbs light.[11] Due to these light absorbing properties, cerebral oxygen saturation changes can be determined by measuring the differential absorption of various wavelengths of light that are transmitted through the forehead and subsequently reflected.[10] The average photon path from a light source located on the forehead is curvilinear, passing through the skull and deeper tissues before resurfacing (Figure 1). Due to the nature of this pathway, resurfaced light is representative of the oxygen saturation of both the cerebral and extracranial tissue. To distinguish between cerebral oxygen saturation and extracranial contamination, commercial cerebral oximeters employ a process of spatial resolution. Spatial resolution is based upon the principle that the depth of tissue interrogated is proportional to the distance between the optode light emitter and detector.[12] 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 extracranial contamination.[13]

       There are an increasing number of NIRS devices available for clinical use. The various devices all have intrinsic spatial resolution capabilities allowing cerebral tissue oxygen saturation to be differentiated from extracranial contamination. However, they differ in numerous important aspects related to the acquisition of their cerebral oxygen saturation measurements. Variations include the light source technology, the wavelengths of light emitted, and the distance between the various light emitters and detectors. The INVOS 5100C-PB (Covidien; Boulder, CO) utilizes light-emitting diode (LED) technology and emits two wavelengths of light (730 nm and 810 nm). Detector distances are 30 mm and 40 mm from the light source.[1] The EQUANOX Classic 7600 (Nonin Medical Inc; Plymouth, MN) also utilizes LED technology, however it emits light of three different wavelengths (730 nm, 810 nm and 880 nm). It 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 co-located with the proximal detector for the other (Figure 1). Emitter to detector distances are 20 mm and 40 mm. The FORE-SIGHT (CAS Medical Systems Inc; Brandford, CT) utilizes a fiberoptically transmitted light amplification by stimulated emission of radiation (LASER) technology and emits four different wavelengths of light (690 nm, 780 nm, 804 nm, 850 nm). Detector distances are 15 mm and 50 mm from the light source.[1] The emitter-

detector spacing and depth of penetration of the various cerebral oximeters is demonstrated schematically in Figure 1. All of these inter-device technologic differences, in particular the light emitter to detector distances, suggest 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 superficial 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.

Sophie Davie

Materials and Methods

After institutional Research Ethics Board approval and written informed consent, volunteers aged 18 to 40 years 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 non-invasive blood pressure cuff (IntelliVue MP90, Philips Medizinsysteme; Boeblingen, Germany) was applied to the left arm, a peripheral pulse oximeter (Adult reusable SpO2 sensor M1191B, Philips Medizinsysteme; Boeblingen, Germany) was placed on the right index finger, and a scalp surface pulse oximeter (Maxfast[TM], 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 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 optode 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 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-minute 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 (Figure 2). Once extracranial hypoxia was established, the head cuff remained inflated for 5 minutes with rSO_2 and other physiologic measurements recorded at 2 and 5 minutes of inflation. Following this, the head cuff was deflated allowing the superficial tissues to be reperfused. Measurements were recorded again following one minute of reperfusion. This inflation-deflation series was repeated in triplicate with each cerebral oximetry device, for a total of 9 measurements per subject, with the data averaged at each time for each subject.

Statistical Analysis

All data are presented as mean ± standard deviation (SD). Comparisons of the rSO_2 and other physiologic parameters between baseline and the various inflation time points were performed for each device, as well as between different devices, using a paired Student's t-test (SAS version 9.2, SAS Institute Inc., Cary NC). Adjustment for multiple comparisons of the rSO_2 changes were performed using the Dunn-Šidák correction,[14] with a $p < 0.05$ (after correction) being considered significant.

Sophie Davie

Results

Study subjects (n = 12) were 24 ± 2.6 years of age; 4 (33%) were male and 8 (67%) were female. Throughout the cycle of head cuff inflations and deflations, heart rate, blood pressure and SaO_2 did not change significantly. 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 minutes post-inflation of the pneumatic head cuff. The relative change from baseline for the INVOS was 13.9 ± 8% at 2 minutes (p = 0.0001) and 16.6 ± 9.6% at 5 minutes (p = 0.0001). The FORE-SIGHT demonstrated a 10.3 ± 5.2% decrease from baseline at 2 minutes (p < 0.0001) and an 11.8 ± 5.3% decrease at 5 minutes (p < 0.0001). The EQUANOX demonstrated a 6.6 ± 4.6% (p = 0.0007) decrease from baseline at 2 minutes and a 6.8 ± 6.0% reduction from baseline (p = 0.0035) at 5 minutes post-inflation of the head cuff. Figure 3 illustrates the differences between rSO_2 measurements at baseline and 5 minutes post-inflation for the three devices.

Inter-device comparisons were also performed at both 2 and 5 minutes post-inflation of the head cuff to determine if there were significant differences between the devices with respect to the influence of extracranial contamination. Compared to one another, the INVOS and FORE-SIGHT did not show significantly different changes from their respective baselines at either time point (p = 0.1329 at 2 minutes; p = 0.0750 at 5 minutes). The INVOS and EQUANOX, however, demonstrated significantly different degrees of extracranial contamination at both 2 and 5 minutes (p = 0.0063, p = 0.0057, respectively). Similarly, the EQUANOX and FORE-SIGHT had a significant difference in their changes from baseline at 2 minutes (p = 0.0012), though not at 5 minutes (p = 0.1461) (Figure 4). However, based on the degree of change from baseline that occurred with the induction of the extracranial hypoxia-ischemia, the various inter-device 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, the INVOS, FORE-SIGHT and EQUANOX, 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 question as to the accuracy of these devices to measure intracerebral oxygen saturation.

NIRS devices can provide continuous information about rSO_2 in a non-invasive manner. These characteristics are unique to this technology and, as such, NIRS is being increasingly used in many clinical settings.[7] 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.[7,15] In these instances, NIRS devices may potentially detect important but clinically silent episodes of cerebral desaturation.[16] 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. Inter-device technologic differences may explain the discrepancy amongst the devices. For example, the INVOS and EQUANOX demonstrated a significant difference in their degrees of extracranial contamination. This may be partly due to the variations in their optode array design, including the emitter to detector distances and the number of light emitters present. The INVOS demonstrates a relatively small distance between its near and far field detectors compared to the EQUANOX, and this appears to be insufficient to appropriately distinguish extracranial from cerebral tissue. Additionally, 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.

Our results may partly explain the observation that the administration of vasoconstrictors such as phenylephrine have paradoxically been reported to result in cerebral desaturation.[17] This is counter-intuitive as increases in blood pressure should maintain cerebral perfusion if autoregulatory mechanisms are intact. However, in a study by Nissen et al, phenylephrine and ephedrine were compared to determine whether they had adverse effects on cerebral oxygen saturation when utilized to reverse anesthesia-induced hypotension.[17] Their results demonstrated that phenylephrine administration (which increases blood pressure by vasoconstriction) caused a decrease in rSO_2 determined by cerebral oximetry, where as ephedrine (which increases blood pressure principally by increasing heart rate and myocardial contractility) did not. Indeed, the authors noted a 14% decrease in rSO_2 subsequent to the administration of phenylephrine. The authors concluded that ephedrine should be used preferentially over phenylephrine to adjust MAP following anesthesia-induced hypotension.[17] The results of our present study, however, may explain these potentially misleading observations. It is possible that this vasoconstrictor is working principally on the extracranial tissue, and reductions in rSO_2 are simply a result of extracranial desaturation from the peripheral vasoconstriction and may not be a result of a true cerebral desaturation. Furthermore, the Nissen et al study utilized the INVOS cerebral oximeter

for their measurements of rSO_2, which demonstrated the lowest signal to noise ratio (i.e. lowest accuracy) in our study. This concept is further supported by the results of a study performed by Johnston et al who determined that CBF was not affected by phenylephrine in a dog model of CPB.[18]

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.[4] In their study, patients were randomized to either a control or interventional group, and were either conventionally managed without the use of NIRS or had a NIRS-directed algorithm to optimize cerebral saturation. Compared to the control group, the interventional group demonstrated a decreased incidence of major organ morbidity and mortality.[4] However, a similar study by Slater et al did not find that a NIRS-guided intervention improved patient outcome. Indeed, the main end point of their study, cognitive decline, had a similar incidence in both interventional and control groups.[3] The study by Slater did, however, re-confirm an overall association between decreases in rSO_2 and adverse neurologic outcome when both groups were combined as a single cohort.[3] Confounding these and other NIRS trials may be a suboptimal rSO_2 signal to noise ratio. That is, if one is attempting to use NIRS to guide therapy and improve outcome but there is an inherent contamination of the cerebral signal, it 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.[19] 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 rSO_2 only to interrogate the symmetry of cerebral blood flow (CBF) such as during aortic arch surgery,[20] some contamination is likely insignificant. That is, interruptions in carotid blood flow, for example, would be detected by both a reduction in intracranial or extracranial oxygen saturation.

Furthermore, the 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 rSO_2 measurement.[4] The presence of extracranial contamination is arguably less important in this context, especially if it is assumed that the proportion of extracranial blood flow (and extracranial oxygen saturation) compared to intracranial blood flow (and rSO_2) remains constant. However, during cardiopulmonary bypass (CPB), when blood is shunted away from non-essential 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 rSO_2. In this case, even interventions based on relative cerebral oximetry measurements could be erroneous.

The importance of optimizing NIRS accuracy is highlighted by a recent cardiac surgery study by Heringlake et al, that 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 they had used the cerebral oximeter we found to have the greatest amount of contamination. Therefore, the absolute rSO_2 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 question 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 rSO_2 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.[21] There is an emerging concept of targeting mean arterial pressure (MAP) specifically to a patient's CBF autoregulatory range, as opposed to a set MAP value for all patients, which may help protect from cerebral hypoperfusion.[22] Recent studies have suggested that NIRS measurements of rSO_2 reliably represent CBF autoregulation in patients undergoing CPB.[21] If, however, rSO_2 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 to measures of jugular venous oxygen saturation ($SjvO_2$).[10] We did not have $SjvO_2$ measurements to determine if cuff inflation caused jugular venous desaturation. However, due to 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 rSO_2. However, this is unlikely since our changes occurred almost immediately following head cuff inflation. If the decreases were due to an adverse physiologic response, reductions would have proceeded gradually. Furthermore, blood pressure, heart rate and SaO_2 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 rSO_2 measurements.[23] 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 rSO_2 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 co-morbidities. 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. Despite a small sample size of young and healthy subjects, the results of this study warrant additional scrutiny into the accuracy of NIRS devices.

Sophie Davie

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Table 1. Physiologic and cerebral oximetry results.

	HR (bpm)	p- value (HR)	MAP (mm Hg)	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 minutes of inflation										
FORE-SIGHT	71 ± 6	$0.4970^\dagger$	89 ± 9	$0.7997^\dagger$	99 ± 1	$0.9261^\dagger$	NR*	-	65 ± 4	$<0.0001^\dagger$
INVOS	73 ± 6	$0.9468^\dagger$	89 ± 8	$0.5620^\dagger$	99 ± 1	$0.1875^\dagger$	NR*	-	65 ± 7	$0.0001^\dagger$
EQUANOX	70 ± 7	$0.5641^\dagger$	88 ± 10	$0.9310^\dagger$	99 ± 1	$1.0000^\dagger$	NR*	-	70 ± 7	$0.0007^\dagger$
5 minutes of inflation										
FORE-SIGHT	73 ± 4	$0.8502^\S$	90 ± 10	$0.6246^\S$	99 ± 1	$1.0000^\S$	NR*	-	64 ± 5	$<0.0001^\S$
INVOS	73 ± 5	$0.9466^\S$	88 ± 8	$0.7543^\S$	99 ± 1	$0.4880^\S$	NR*	-	63 ± 8	$0.0001^\S$
EQUANOX	70 ± 6	$0.6605^\S$	91 ± 9	$0.3753^\S$	99 ± 1	$0.7600^\S$	NR*	-	69 ± 6	$0.0035^\S$
Reperfusion										
FORE-SIGHT	73 ± 5	$0.9048^\P$	89 ± 11	$0.7678^\P$	99 ± 1	$1.0000^\P$	97 ± 3	$0.2062^\P$	73 ± 4	$0.8478^\P$
INVOS	75 ± 6	$0.4688^\P$	88 ± 9	$0.8007^\P$	99 ± 1	$0.2829^\P$	98 ± 2	$0.5807^\P$	78 ± 10	$0.5582^\P$
EQUANOX	73 ± 8	$0.7942^\P$	90 ± 10	$0.5092^\P$	99 ± 1	$0.3083^\P$	98 ± 1	$0.5373^\P$	76 ± 11	$0.8292^\P$

Data is presented as mean $\pm$ SD where appropriate.

* NR = no reading; head cuff was inflated until scalp pulse oximeter signal was lost due to cessation of scalp blood flow

$\dagger$ comparative p values between baseline and 2 minutes following head cuff inflation of the rSO_2 and other physiologic measurements
$\S$ comparative p values between baseline and 5 minutes following head cuff inflation of the rSO_2 and other physiologic measurements
$\P$ comparative p values between baseline and reperfusion of the extracranial tissue of the rSO_2 and other physiologic measurements

HR = heart rate; bpm = beats per minute, MAP = mean arterial pressure; SaO_2 = finger arterial oxygen saturation; $SscO_2$ = scalp arterial oxygen saturation; rSO_2 = regional cerebral oxygen saturation
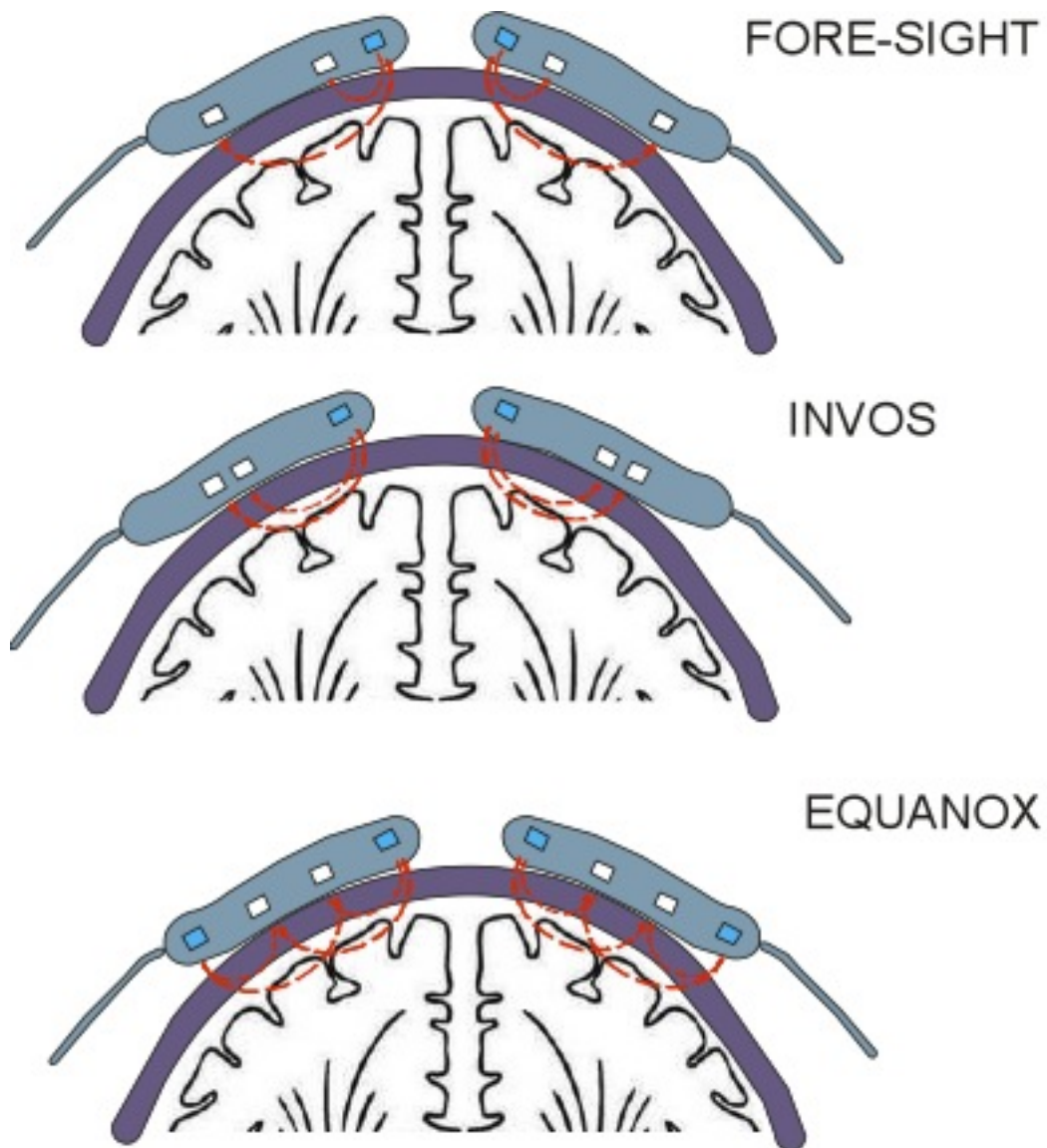
Figure 1. Schematic diagram demonstrating the curvilinear photon path through the superficial (i.e. extracranial) and cerebral tissues represented by the different cerebral oximeters used during the study. The cerebral oximetry optode array, placed on the subject forehead, contains light emitters (blue boxes) and light detectors (white boxes). Light (red arrows) from the 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.
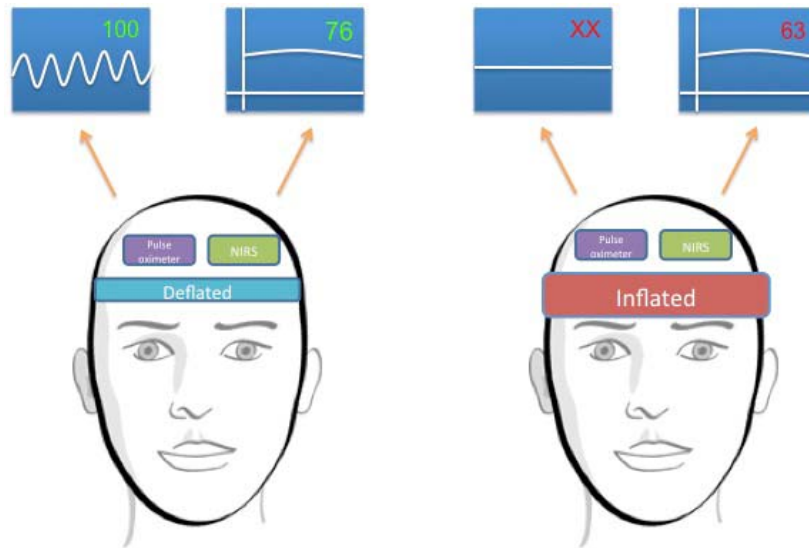Adapted from 'http://www.picturesof.net/pages/081012-211268-229048.html'

Figure 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 extracranial tissue hypoxia-ischemia.
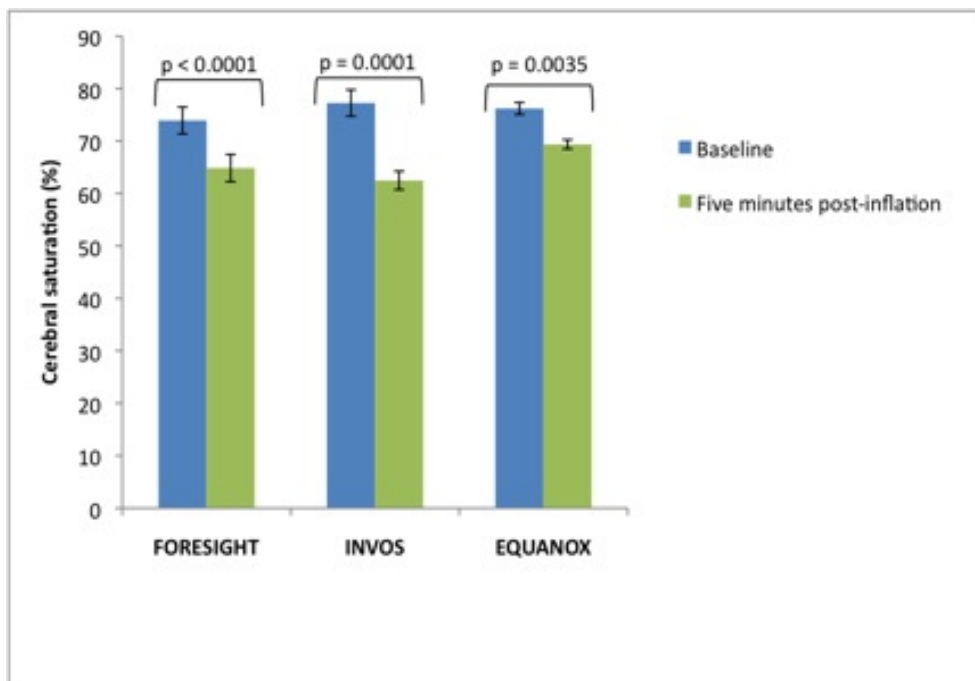Adapted from 'http://www.hairdirect.com/images/px_learn/faceshape-oval.gif'

Figure 3. Regional cerebral oxygen saturation measurements of the three NIRS devices studied at baseline and at five minutes post-inflation of a head cuff. All three of the cerebral oximeters demonstrated a statistically significant reduction in cerebral oxygen saturation at 5 minutes post-inflation. Error bars represent the standard deviations.
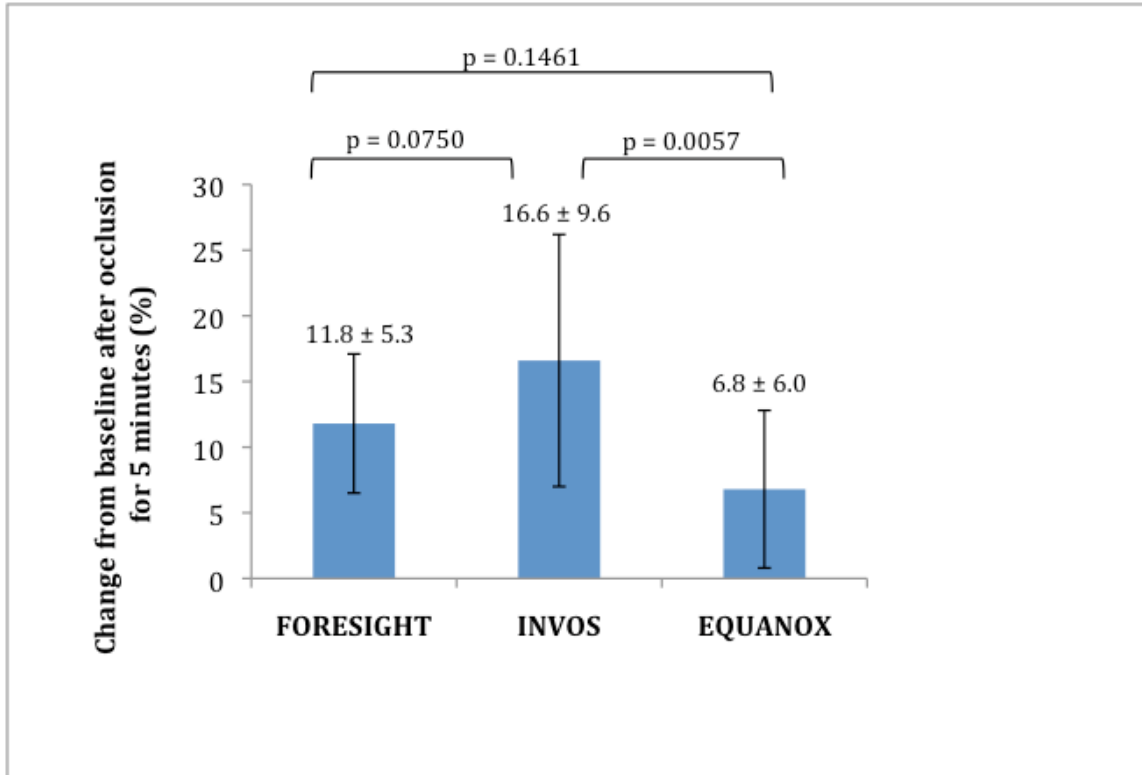
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Figure 4. Percent change from the baseline rSO$_2$ measurement of the three NIRS devices studied after five minutes of occlusion with the head cuff. P values of inter-device statistical analyses are shown. Error bars represent the standard deviations.

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