ANESTHESIOLOGY

Effect of Extracerebral Contamination on Near-infrared Spectroscopy as Revealed during Organ Donation: A Prospective Observational Study in Brain-dead Organ Donors

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EDITOR'S PERSPECTIVE

What We Already Know about This Topic

- Near-infrared spectroscopy is commonly used as a clinical measure of cerebral perfusion and oxygenation
- It is still unclear how much, and in what circumstances, perfusion of extracerebral tissues influences the output of the monitor

What This Article Tells Us That Is New

- Brain-dead patients (who have absent cerebral circulation) show surprisingly high cerebral oxygen saturation, which then drops when the aorta is clamped and extracerebral circulation ceases
- These observations call into question the reliability of near-infrared spectroscopy in detecting isolated cerebral ischemia

ABSTRACT

Background: Near-infrared spectroscopy (NIRS) has been utilized widely in anesthesia and intensive care to monitor regional cerebral oxygen saturation (rS_cO_2). A normal oxygenation of extracerebral tissues may overlay and thereby mask cerebral desaturations, a phenomenon known as extracerebral contamination. The authors investigated the effect of a cessation of extracerebral tissue perfusion on rS_cO_2 in patients with anoxic brains.

Methods: In a single-center, prospective, observational study, brain-dead adults undergoing organ donation were investigated. rS_cO_2 was measured bifrontally using the INVOS 5100C/7100 as well as the ForeSight Elite system. To achieve an efficient conservation of organs and to prevent a redistribution of the perfusion fluid to other tissues, the aorta was clamped before organ perfusion. rS_cO_2 was monitored until at least 40 min after aortic clamping. The primary outcome was the amount of extracerebral contamination as quantified by the absolute decrease in rS_cO_2 after aortic clamping. Secondary outcomes were the absolute rS_cO_2 values obtained before and after clamping.

Results: Twelve organ donors were included. Aortic clamping resulted in a significantly ($P < 0.001$) greater absolute decrease in rS_cO_2 when comparing the INVOS (43.0 ± 9.5%) to the ForeSight (27.8 ± 7.1%) monitor. Before aortic clamping, near-normal rS_cO_2 values were obtained by the INVOS (63.8 ± 6.2%) and the ForeSight monitor (67.7 ± 6.5%). The rS_cO_2 significantly ($P < 0.001$) dropped to 20.8 ± 7.8% (INVOS) and 39.9 ± 8.1% (ForeSight) 30 min after clamping, *i.e.*, a condition of a desaturation of both extracerebral and cerebral tissues.

Conclusions: The abrupt end of extracerebral contamination, caused by aortic clamping, affected both NIRS monitors to a considerable extent. Both the INVOS and the ForeSight monitor were unable to detect severe cerebral hypoxia or anoxia under conditions of normal extracerebral oxygenation. While both NIRS monitors may guide measures to optimize arterial oxygen supply to the head, they should not be used with the intention to detect isolated cerebral desaturations.

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Near-infrared spectroscopy (NIRS) is an established technique to monitor cerebral oxygenation in anesthesia and intensive care medicine.[1–3] It is based on the phenomenon that oxygenated and deoxygenated hemoglobin

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differ in their absorption spectrum of near-infrared light. On the path from the light emitter to the light detector, the photons pass through the scalp, the cranial bone, and cerebral tissue, which differ in their vessel supply in such a way that only the last is supplied by intracranial vessels originating from the internal carotid artery (ICA) (fig. 1). In contrast, the first two are supplied by the external carotid artery (ECA) and are therefore not affected by intracranial pathologies. Although NIRS was developed with the aim of measuring intracranial oxygen saturation, extracranial oxygen saturation may overlay and affect the NIRS signal for the anatomical reasons mentioned above. This interfering influence is known as extracerebral contamination[4–8] and is thought to be minimized by spatial resolution when using two light receivers.[3] Since the photons then reach the receiver *via* two different light paths (fig. 1), which are influenced by the extracranial saturation to a different extent, the effect of extracerebral contamination can theoretically be eliminated or at least reduced.[9]

Brain death is characterized by cerebral perfusion arrest resulting in brain anoxia. Therefore, cerebral oxygen saturation in brain-dead patients should theoretically be at 0%. However, NIRS studies in these patients revealed either a regional cerebral oxygen saturation (rS_cO_2) of 74 ± 4%, similar to healthy volunteers (74 ± 6%),[10] or a wide range of rS_cO_2 values (51 ± 27%)[11] extending up into the normal range.

These unintuitive high rS_cO_2 values are presumably caused by extracerebral contamination.[4,6–8,12] This effect has been investigated under conditions of either cerebral normoxia and extracerebral ischemia[4,6,7] or combined cerebral and extracerebral hypoxia,[8,12–14] but not during normal extracerebral oxygenation. To serve as monitors of cerebral oxygenation, NIRS devices are expected to yield valid results, including the clinical situation of a normal extracerebral perfusion but a suspected cerebral desaturation.

Therefore, we performed a prospective study in brain-dead organ donors. The objective was to investigate extracerebral contamination by comparing the times with normal extracerebral perfusion and extracerebral perfusion arrest caused by organ donation.

Materials and Methods

With approval of the local ethics committee (Medical Faculty of the University Bonn, Bonn, Germany; reference No. 416/22-EP), we performed a single-center, prospective, observational study at the University Hospital Bonn (Bonn, Germany). Written informed consent was waived by the institutional review board according to §6 of the North Rhine-Westphalian Health Data Protection Act. Patients were recruited from June 2021 to June 2023. This manuscript satisfies the STrengthening the Reporting of OBservational studies in Epidemiology (STROBE) criteria for observational studies.

Brain-dead adults undergoing surgery for organ donation were included. Children and adolescents were excluded. In Germany, brain death is officially called "irreversible loss of brain function" and described as "the final, irreversible loss of all function of the cerebrum, cerebellum, and brainstem" in §3 of the German Transplantation Law.[15] Irreversible loss of brain function was diagnosed based on the clinical manifestations of coma, absent brainstem reflexes, and apnea by at least two experts with several years of experience in intensive care of brain-injured patients, one of whom had to be a neurologist or neurosurgeon. Irreversibility was demonstrated by supplementary testing (electroencephalogram, perfusion scintigraphy, or computed tomography [CT] angiogram) or by confirming the abovementioned clinical manifestations after a waiting period. Permission for organ donation was obtained under the "opt-in" system, requiring informed consent either by patients during their lifetimes or by the relatives at the time of death.

After organ allocation, patients were transferred to the operation theater for multiorgan donation. NIRS devices from two different manufacturers were applied. The first NIRS monitor was the ForeSight Elite Tissue Oximeter Module (CAS Medical Systems, Inc., USA), which was connected to a HemoSphere Advanced Monitor (Edwards Lifesciences Corp., USA). The ForeSight Elite employs five wavelengths of light (690, 730, 770, 810, and 870 nm) with two detectors 15 and 50 mm apart from the light source.[3] The second NIRS device was the INVOS 5100C or 7100 monitor (Medtronic Inc., USA), which differed in their design but not their algorithm to measure cerebral oxygenation.[16] Therefore, they were regarded as identical monitors. The INVOS device provided two wavelengths of light (730 and 810 nm), with a distance between the light source and the two detectors of 30 and 40 mm.[3] NIRS optodes were placed on the patient's forehead above the frontal cortex according to the manufacturer's recommendations. Ventilator settings and circulatory support were chosen to maintain cardiorespiratory parameters in the physiologic range. The fraction of inspired oxygen was set to maintain peripheral arterial oxygen saturation above 92%. Only in cases of lung donation, the fraction of inspired oxygen was set to 1.0 at certain times. Muscle relaxants and opioids were given before skin incision to blunt spinal reflexes. Open laparotomy and/or thoracotomy was performed depending on the organs donated

To allow for later replacement of the 37°C warm blood by cold perfusion liquid, the (abdominal and/or thoracic) aorta was cannulated for perfusion fluid inflow. Heparin was given intravenously (400 units/kg body weight), the inferior vena cava (and/or the right atrium of the heart) was opened for outflow of venous blood, and organ perfusion was started. To achieve a better organ preservation and to prevent distribution of the perfusion fluid to other body regions (*e.g.*, extremities or head), the aorta was clamped. For perfusion of abdominal organs, the aorta was clamped below the diaphragm, enabling a retrograde flow from the inflow cannula *via* the abdominal aorta to abdominal organs.

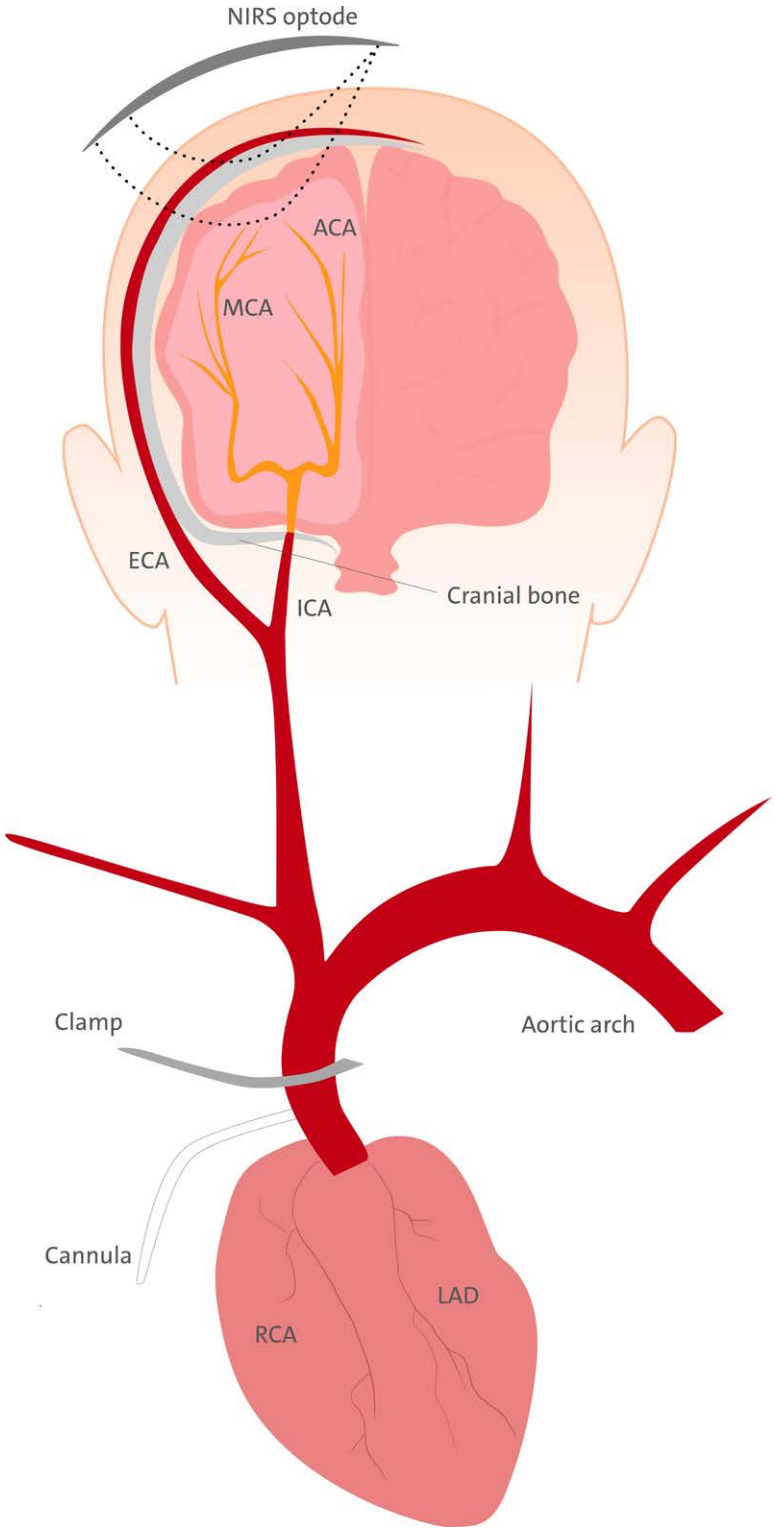

Fig. 1. Illustration of the arterial blood supply to the head, the brain, and the heart. The common carotid artery divides into the external carotid artery (ECA), which supplies the scalp as well as the cranial bone, and the internal carotid artery (ICA), which feeds the brain *via* its branches anterior (ACA) and middle cerebral arteries (MCA). During irreversible loss of brain function, neither the ACA nor the MCA (nor any other cerebral vessel) is perfused, as shown by the *yellow* color. According to the light path of the near-infrared spectroscopy (NIRS) optode (*upper*), NIRS is affected by perfusion of both ICA and ECA. The ascending aorta is clamped to facilitate perfusion of the coronary arteries with cold perfusion liquid. Clamping results in a sudden cessation of arterial blood supply to the ECA. LAD, left anterior descending artery; RCA, right coronary artery.

In contrast, the heart was perfused as known from coronary artery bypass grafting. The ascending aorta was clamped and coronary arteries were perfused from an inflow cannula positioned proximal to the clamp (fig. 1). Therefore, blood flow to the head and its extracerebral tissue ceased immediately after aortic clamping, whereas the blood flow to the brain had already stopped hours earlier when irreversible loss of brain function was diagnosed. Asystole occurred soon after aortic clamping, and any circulatory support by fluids or catecholamines was stopped. Respiration was terminated except for cases of lung donation (where it was maintained until the end of lung explantation).

rS_cO_2 was measured by NIRS from the beginning of surgery until at least 40 min after aortic clamping, or until skin closure was performed. NIRS data were stored on a universal serial bus stick for later analysis. NIRS data were imported, synchronized, and analyzed using in-house written software (MATLAB, MathWorks, USA).

The primary outcome was the absolute decline in rS_cO_2 after aortic clamping, which was determined by comparing the rS_cO_2 obtained during the minute before aortic clamping with the 1-min period 30 min after clamping. Hence, rS_cO_2 values were time-averaged during these 1-min periods. Secondary outcomes were the relative decrease in rS_cO_2 at the abovementioned time points, as well as the detailed time course of rS_cO_2 until 40 min after aortic clamping.

Statistical Analysis

A data analysis and statistical plan was written and filed with the institutional review board before data were accessed. The primary endpoint is a combination of both NIRS devices; we hypothesized that both devices showed a decrease in rS_cO_2 values. This hypothesis was tested by a paired *t* test (before *vs.* 30 min after clamping) separately for both devices. Only if both tests showed a significant decrease at a significance level of 0.01 was the null hypothesis (no decrease in either device) rejected. Data were tested for normal distribution using the Shapiro–Wilk test before the paired *t* test was applied. To account for multiple measurements within one participant (two devices and time points per participant), we used a linear mixed model to assess the differences between devices and time points. The participant identifier was included as random intercept in the regression model. We performed five statistical tests (two for the combined primary endpoint and three for the linear mixed model).

A Bonferroni correction was applied to correct for multiple (n = 5) statistical testing, and statistical significance was therefore assumed at a $P < 0.05/5 = 0.01$. A power analysis was performed assuming an expected difference in rS_cO_2 means of 8% and an expected SD of 6%. For a desired sample size of 12 and $P = 0.05/5 = 0.01$, a power of 0.91 was calculated. Statistical analysis was performed using SigmaPlot (Version 14.0, Systat, Inpixon, USA). Unless otherwise mentioned, values are shown as mean ± SD, and changes in rS_cO_2 are absolute saturation changes (expressed in percent saturation), and not relative changes in the sense of a change by × percent.

Results

A total of 15 organ donors were investigated, of whom 3 were excluded, in whom only one of the two NIRS monitors was available, leaving 12 patients for final analysis. They consisted of seven women and five men with a mean age of 52.6 ± 18.1 yr, ranging between 25 and 83 yr. Supplementary diagnostics were performed in 11 patients in addition to clinical testing: an isoelectric electroencephalogram was obtained in 9 patients, and cerebral perfusion arrest was shown by CT angiography in 2 patients.

Before aortic clamping, 9 of 12 donors showed normal rS_cO_2 in the range between 60% and 80%, according to both monitors. The lowest observed rS_cO_2 was 54% for the INVOS and 58% for the ForeSight monitor. Aortic clamping was associated with a significant ($P < 0.001$) drop in rS_cO_2 from 63.8 ± 6.2% to 20.8 ± 7.8% 30 min thereafter according to the INVOS monitor (fig. 2). The lower measurement limit of the INVOS monitor (rS_cO_2 = 15%) was reached in 7 of the 12 patients (fig. 3). The ForeSight monitor showed a significant ($P < 0.001$) drop in rS_cO_2 from 67.7 ± 6.5% before clamping to 39.9 ± 8.1% 30 min after clamping (fig. 2). The lowest observed rS_cO_2 value was 24%, and a lower measurement threshold was never reached with this monitor (fig. 3).

The linear mixed model showed no significant difference in rS_cO_2 between the devices during the minute before clamping (table 1). Aortic clamping was associated with a significant ($P < 0.0001$) drop in rS_cO_2 by 27.8% (absolute saturation) as assessed using the ForeSight device. For the INVOS device, this drop is 15.2% (absolute saturation) more pronounced than for the ForeSight device, *i.e.*, the rS_cO_2 drop is significantly ($P = 0.0003$) different between the two devices (table 1).

The detailed time course of rS_cO_2 is shown in figures 3 and 4 for individual patients and the entire study population, respectively. From the time point 2 min after clamping, the INVOS monitor showed a significantly ($P < 0.01$) lower rS_cO_2 than the ForeSight monitor.

Both the absolute and the relative decrease in rS_cO_2 were significantly more pronounced using the INVOS monitor (43.0 ± 9.5% and 67.2 ± 12.3%, respectively) as compared to the ForeSight device (27.8 ± 7.1%, $P < 0.001$, and 41.2 ± 10.4%, $P < 0.001$).

Discussion

During organ donation in brain-dead patients, we observed three phenomena that require consideration. First, near-normal rS_cO_2 values were measured at the

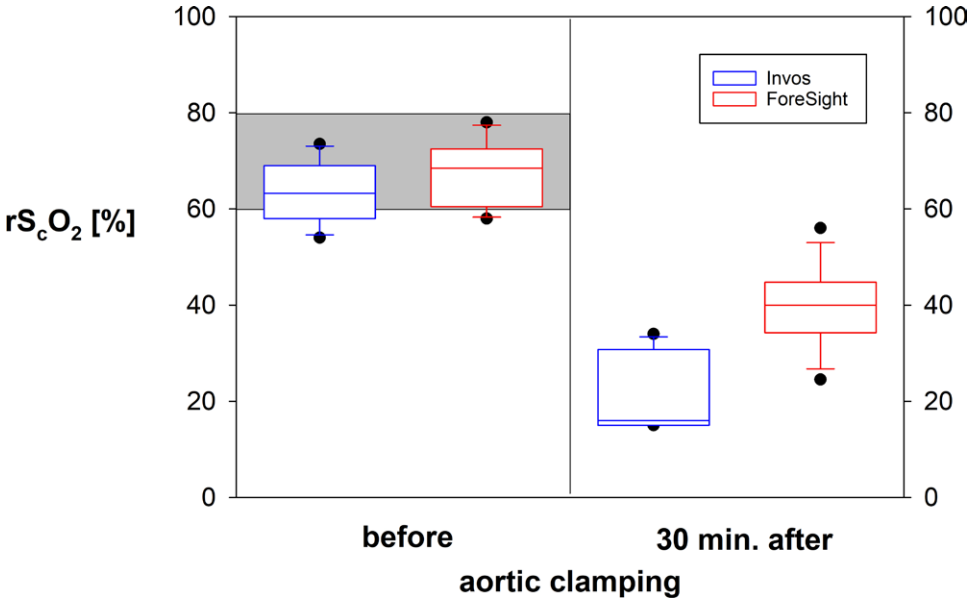

Fig. 2. The regional cerebral oxygen saturation (rS_cO_2) is shown comparing two NIRS devices (INVOS 5100/7100 [Medtronic Inc., USA] and ForeSight [CAS Medical Systems, Inc., USA]) and the two time periods before aortic clamping and 30 min thereafter. Clamping was associated with a significantly ($P < 0.001$) greater drop in absolute rS_cO_2 when comparing the INVOS (43.0 ± 9.5%) to the ForeSight (27.8 ± 7.1%) monitor. The *boxplots* indicate median and 25th and 75th percentiles, and the *circles* display 5th and 95th percentiles. The *gray area* denotes the normal range of rS_cO_2 (60 to 80%) as expected in healthy volunteers breathing room air.

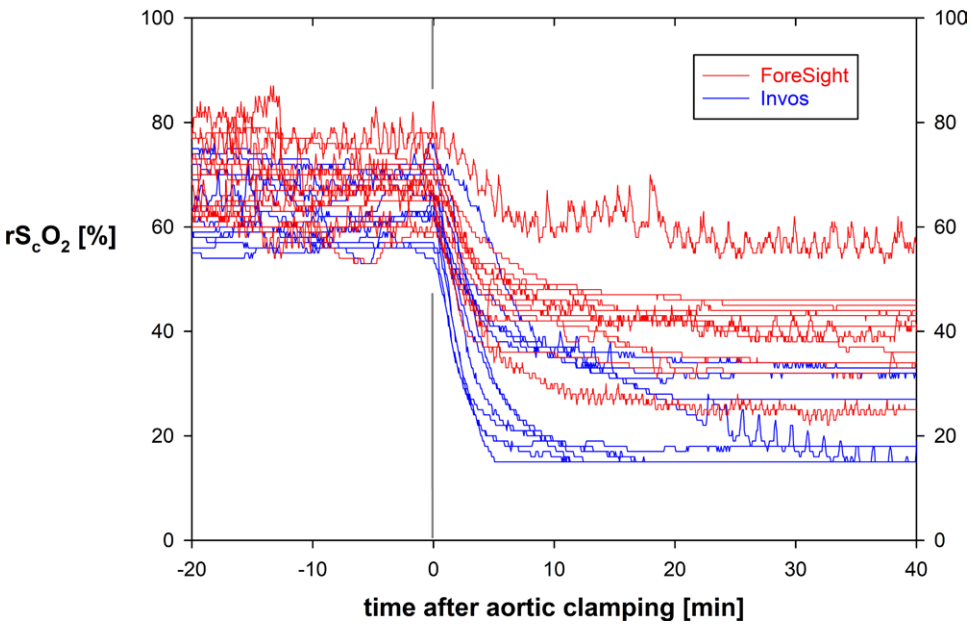

Fig. 3. Individual time course of regional cerebral oxygen saturation (rS_cO_2), as assessed using the ForeSight (CAS Medical Systems, Inc., USA; illustrated in *red*) and the INVOS (Medtronic Inc., USA; illustrated in *blue*) monitors from 20 min before clamping to 40 min thereafter.

beginning of the operation despite the presence of cerebral anoxia. Second, a rapid drop in rS_cO_2 was observed after aortic clamping, which was more pronounced with the INVOS than the ForeSight monitor. Third, rS_cO_2 remained well above 0 even at the end of surgery. The lowest observed rS_cO_2 values at the end of surgery were

Table 1. Comparison between the ForeSight and the INVOS Devices Applying a Linear Mixed Model

	Estimate	95% CI	*P* Value
Intercept	67.7	63.7 to 71.7	< 0.0001
Device (INVOS)	–3.8	–9.0 to 1.2	0.151
Period (post)	–27.8	–32.9 to –22.7	< 0.0001
Post: INVOS	–15.2	–22.4 to –8.0	0.0003

Estimates and CI are given as absolute saturation. Post: time period 30 min after clamping. ForeSight (CAS Medical Systems, Inc., USA) and INVOS (Medtronic Inc., USA).

15% for the INVOS monitor and 24% for the ForeSight monitor.

Cerebral oxygenation can be estimated based on the cerebral arterial (S_cao_2) and venous (S_cvo_2) oxygen saturation, as well as the ratio between arterial (Ra) and venous (Rv) blood volume in the brain:

$$rS_cO_2 = (S_cao_2 \times Ra + S_cvo_2 \times Rv)$$

The NIRS monitors investigated differ in their assumed Ra/Rv ratio. Whereas the INVOS device applies a 25% arterial to 75% venous ratio,[16] the ForeSight uses a 30% to 70% ratio.[17] Assuming an arterial and venous oxygen saturation in the brain of 97% and 60.7%, respectively, we would expect an rS_cO_2 of 69.8% (97% × 0.25 + 60.7% × 0.75) and of 71.6% (97% × 0.30 + 60.7% × 0.70) for the INVOS and ForeSight monitors, respectively, in healthy volunteers when breathing room air. This difference in Ra/Rv ratio can explain the slightly (and nonsignificantly) higher rS_cO_2 values seen using the ForeSight in comparison to the INVOS device at the beginning of surgery.

Assuming an rS_cO_2 SD of 5% and rounding the above given rS_cO_2 values to 70% gives normal rS_cO_2 values ranging from 60 to 80% (70% ± 1.96 × 5%). In contrast, an rS_cO_2 of 0% (0% × Ra + 0% × Rv) is expected during irreversible loss of brain function due to the loss of brain perfusion and oxygenation. In fact, a cessation of cerebral perfusion is an inherent feature of the brain death concept and was explicitly shown in two of the patients. However, neither the INVOS nor the ForeSight monitor depicted this condition of cerebral anoxia correctly. Instead, no monitor displayed rS_cO_2 values less than 54%, and three-fourths of donors exhibited rS_cO_2 values within the normal range (60 to 80%) of brain-healthy subjects. Using the normal range of 51.0 to 81.8% proposed by Chan *et al.*[18] for cardiac surgery patients, even all organ donors exhibited a normal cerebral oxygenation. These results confirm earlier findings of identical rS_cO_2 values obtained in healthy volunteers and brain-dead donors reported by Muellner *et al.*[10] Such incorrect measurements are presumably caused by extracerebral contamination,[4–8] *i.e.*, the normal oxygenation of the extracerebral tissue overlying a cerebral desaturation. The light path from the NIRS light emitter to the sensor proceeds for the most part through extracerebral tissues such as forehead skin, muscle, bone,

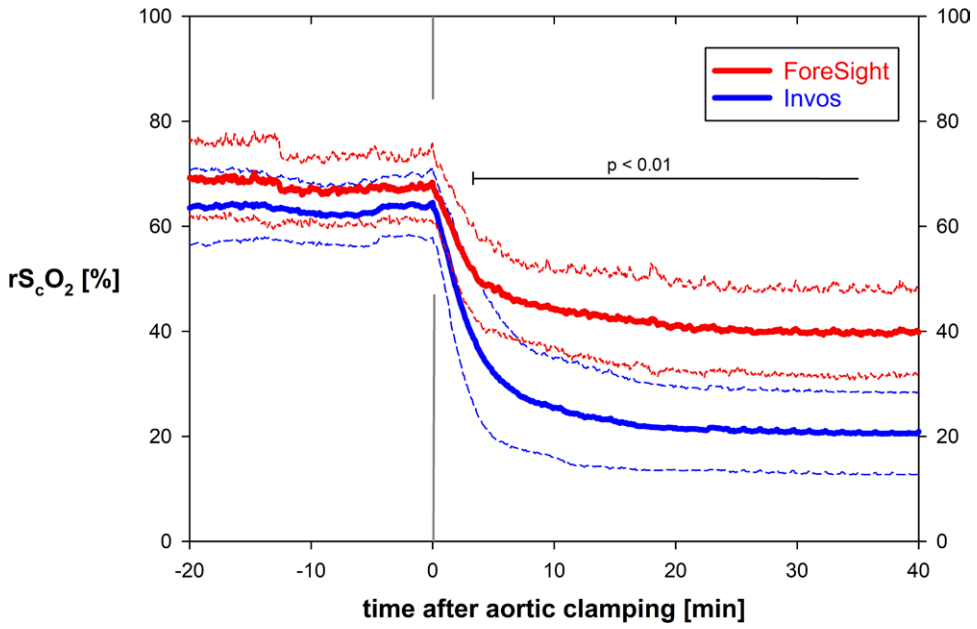

Fig. 4. Time course of regional cerebral oxygen saturation (rS_cO_2), as assessed using the ForeSight (CAS Medical Systems, Inc., USA; illustrated in *red*) and the INVOS (Medtronic Inc., USA; illustrated in *blue*) monitors from 20 min before clamping to 40 min thereafter. From 2 min after clamping on, the INVOS yielded a significantly ($P < 0.01$) lower rS_cO_2 than the ForeSight monitor. Data are mean ± SD.

and cerebrospinal fluid, which all absorb NIRS light to some extent. Strategies have been developed to reduce extracerebral contamination by a dedicated sensor design with different light pathways[3,9]; however, our results refute this strategy as insufficient.

According to the characteristics of brain death, brain perfusion was absent during the entire donation procedure and thus never changed during surgery. Aortic clamping resulted in a sudden cessation of blood flow to the head and its extracerebral tissues only. The measured rS_cO_2 dropped in line with these extracerebral perfusion changes, which supports the hypothesis that NIRS predominantly reflects extracerebral rather than cerebral oxygenation.[12,19,20] Davie and Grocott,[4] Greenberg *et al*.,[6] and Kato *et al*.[7] reported that the rS_cO_2 as assessed using the INVOS monitor is more affected than the ForeSight device when manipulating extracerebral perfusion under conditions of cerebral normoxia. Our results confirm that this holds true for conditions of cerebral anoxia as well. So far, the influence of extracerebral tissue on rS_cO_2 can be quantified exactly only in computational simulation studies.[21] However, this has not yet been possible in *in vivo* human studies[5] such as ours. Nevertheless, our results contradict the estimation that only 15% of cerebral oximetry measurements are derived from extracerebral tissue and 85% from the superficial cerebrum.[22]

At first sight, the INVOS seems to be more sensitive to extracerebral contamination than the ForeSight monitor. At the end of surgery, with its lack of both extracerebral and cerebral perfusion, we would have expected to monitor an rS_cO_2 of approximately 0%, but neither monitor did so. In more than half of patients, the INVOS monitor reached its lower measurement threshold of 15%, indicating that any value between 0% and 15% might have occurred. In contrast, the ForeSight device neither reached a lower limit nor fell less than 24%. Moreover, the final mean rS_cO_2 was 40%, which raises concerns regarding the validity of the ForeSight device to measure low cerebral oxygenations. To our knowledge, the monitor was validated against jugular bulb oximetry, but only for oxygen saturations above 52%.[17]

Murkin and Arango pointed out[22] that tissue oxygenation in the dying or dead brain could be high, low, or near normal because of remaining blood in capillaries and venous capacitance vessels.[23] Accordingly, a discordantly high rS_cO_2 may reflect the pathophysiology of nonmetabolizing yet nonperfused tissue.[11] Furthermore, the presence of light-absorbing pigments (chromophores), lack of blood flow pulsatility, algorithmic factors, and the already mentioned extracerebral contamination might explain the observations of normal rS_cO_2 before aortic clamping. If these factors can mask zero cerebral blood flow (as in brain death), the question arises whether these same factors disguise a reduced cerebral blood flow (as in focal cerebral ischemia) as well. Thus, it remains to be clarified what the quantitative and qualitative nature of cerebral ischemia must be to be detected by NIRS.

NIRS monitoring is commonly used in cardiovascular, noncardiovascular, and neonatal anesthesiology to monitor cerebral oxygenation.[1,2,18] In a systematic review on NIRS monitoring in awake carotid endarterectomy, Khan *et al*.[24] reported that the commonly applied threshold of a 20% rS_cO_2 decrease from baseline has a low sensitivity of 70.5% (95% CI, 54.1 to 82.9%) to detect intraoperative cerebral ischemia. Therefore, NIRS monitoring misses approximately 30% of patients with cerebral ischemia (false negative rate), which limits its usefulness as a monitor of cerebral ischemia. Overall, the NIRS results between carotid endarterectomy and brain death do not compare well because the perfusion ratio of ICA to ECA is opposite: while the ECA is well-perfused in brain death, it is clamped and hence nonsupplied in carotid endarterectomy. *Vice versa*, ICA is not perfused by definition in brain death, whereas it is supplied in carotid endarterectomy (at least in cases where a shunt is inserted). A poor association between a 20% decrease in rS_cO_2 and cerebral status was also reported in aortic arch surgery.[25]

Algorithms on how to treat desaturations observed using NIRS have been published and applied.[26] These include measures such as correcting venous return from the head by verifying head position, raising arterial blood pressure, and correcting anemia or arterial carbon dioxide and/or oxygen partial pressure.[26] Following our assumption that the investigated NIRS monitors measure predominantly extracerebral oxygenation, the abovementioned procedures remain effective in treating desaturations, as they affect both extracerebral and cerebral oxygenation. However, in critical brain events or diseases such as head injury, subarachnoid hemorrhage, ischemic insults, or intracranial hemorrhage, cerebral ischemia with consecutive cerebral deoxygenation occur without changes in extracerebral oxygenation. We assume that the investigated NIRS devices become unreliable under these circumstances and should not be used to monitor cerebral oxygenation in these indications. While low measured rS_cO_2 values indicate cerebral hypoxia, normal measured rS_cO_2 can by no means be used to infer normal cerebral oxygenation.

Our study has several limitations. First, the number of investigated organ donors is low. However, due to the great effect size of aortic clamping, we do not expect different results when including more patients. Second, cerebral perfusion arrest was explicitly shown in two patients only. Since the concept of brain death and irreversible loss of brain function is conclusive and generally accepted in medicine,[15] we have no doubt that any of the other patients would show anything other than a perfusion arrest, either. Third, we measured neither forehead skin oxygenation nor jugular bulb oxygenation. The first, although it is a proxy for extracerebral oxygenation, requires pulsatile blood flow, which would have been unavailable in our donors subsequent to aortic clamping. The second measures false high values under conditions of brain death, since the jugular bulb then receives influx from extracerebral instead of cerebral veins.[27]

Fourth, we applied the INVOS 5100C/7100 and ForeSight Elite monitors only. It remains unknown whether the observed results can be transferred to other NIRS monitors until equivalent investigations are performed.

We conclude that both the INVOS and the ForeSight monitor are affected by extracerebral contamination to an extent, that they are unable to detect severe cerebral hypoxia or anoxia under conditions of normal extracerebral oxygenation. While both NIRS monitors may guide measures to optimize arterial oxygen supply to the head, they should not be used with the intention to detect isolated cerebral desaturations.

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Competing Interests

Drs. Soehle and Thudium received honoraria for lectures from Medtronic Inc. (Minneapolis, Minnesota). The other authors declare no competing interests.

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