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Detection of cerebral ischemia in neurovascular surgery using quantitative frequency-domain near-infrared spectroscopy

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Object. There is great value in monitoring for signs of ischemia during neurovascular procedures. Current intraoperative monitoring techniques provide real-time feedback with limited accuracy. Quantitative frequency-domain near-infrared spectroscopy (Q-NIRS) allows measurement of tissue oxyhemoglobin (HbO₂), deoxyhemoglobin (HHb), and total hemoglobin (tHb) concentrations and brain tissue oxygen saturation (SO₂), which could be useful when monitoring for evidence of intraoperative ischemia.

Methods. Using Q-NIRS, the authors monitored 25 neurovascular procedures including aneurysm clip placement, arteriovenous malformation resection, carotid endarterectomy, superficial temporal artery–middle cerebral artery (MCA) bypass surgery, external carotid artery–MCA bypass surgery, encephaloduromyosynangiosis, and balloon occlusion testing. The Q-NIRS technology provides measurable cerebral oxygenation values independent from those of the scalp tissue. Thus, alterations in the variables measured with Q-NIRS quantitatively reflect cerebral tissue perfusion. Bilateral monitoring was performed in all cases.

Five of the patients exhibited evidence of clinical ischemic events during the procedures. One patient suffered blood loss with systemic hypotension and developed diffuse brain edema intraoperatively, one patient suffered an ischemic event intraoperatively and developed an occipital stroke postoperatively, and one patient showed slowing on electroencephalography intraoperatively during carotid clamping; in two patients balloon occlusion testing failed. In all cases of ischemic events occurring during the procedure, Q-NIRS monitoring showed a decrease in HbO₂, tHb, and SO₂, and an increase in HHb.

Conclusions. Quantitative frequency-domain near-infrared spectroscopy provides quantifiable and continuous real-time information about brain oxygenation and hemodynamics in a noninvasive manner. This continuous intraoperative oxygenation monitoring is a promising method for detecting ischemic events during neurovascular procedures.

KEY WORDS • ischemia • near-infrared spectroscopy • brain tissue oxygen monitoring • hemoglobin • bypass surgery

DESPITE advances in surgical techniques, the risk of perioperative stroke during neurovascular procedures remains a significant potential source of morbidity and death.²⁰ Intraoperative cerebral ischemia can occur because of prolonged temporary occlusion of intracranial cerebral arteries during aneurysm clip placement, from inadvertent or planned permanent occlusion of a cere-

bral artery, from sustained elevation of intracranial pressure, or from a severe drop in blood pressure. Several procedures are used to reduce intraoperative cerebral ischemic insults, including deep hypothermic circulatory arrest and EEG burst suppression^{7,31,35} to reduce cerebral oxygen consumption.³⁰

Current intraoperative monitoring techniques such as electroencephalography, transcranial Doppler ultrasonography, SSEP, and CW-NIRS provide only indirect evidence of ischemic insult, and have other technique-specific limitations. Quantitative frequency-domain NIRS, however, is able to provide a continuous measurement of absolute cerebral oxygenation. For this reason, we investigated the use of Q-NIRS for the detection of ischemia during neurovascular procedures.

Abbreviations used in this paper: CEA = carotid endarterectomy; CSF = cerebrospinal fluid; CW-NIRS = continuous-wave near-infrared spectroscopy; EEG = electroencephalographic; HbO₂ = oxyhemoglobin; HHb = deoxyhemoglobin; ICA = internal carotid artery; Q-NIRS = quantitative frequency-domain NIRS; SO₂ = oxygen saturation; SSEP = somatosensory evoked potential; tHb = total hemoglobin.

Clinical Material and Methods

Patient Population

Patients with neurovascular procedures performed between May 2004 and February 2006 at the Department of Neurosurgery at the University of Illinois at Chicago were included in the study. Institutional review board approval for the study was obtained from the University of Illinois at Chicago and the University of Illinois at Urbana-Champaign. Patients signed informed consent forms and Health Insurance Portability and Accountability Act documents before surgery. No intraoperative decisions were made based on the results of Q-NIRS monitoring. We monitored 25 neurovascular procedures using the Q-NIRS tissue oximeter (Oxiplex TS, ISS Inc.).

Patient demographic data, diagnoses, treatments, and outcomes are summarized in Table 1. Cases were chosen based on the potential for ischemic insults. During temporary vessel occlusion at the time of surgery, EEG burst suppression was induced in all patients who underwent operations for aneurysms or bypass procedures using up to 2% isoflurane inhalational anesthesia. End-tidal carbon dioxide was monitored continuously and maintained at 30 mm Hg to avoid changes in cerebral blood volume. The clinical outcomes of the surgical interventions were recorded postoperatively (Table 1).

Quantitative Frequency-Domain NIRS Methods

The Q-NIRS tissue oximeter was brought to the operating room and was turned on 30 minutes before surgery to reach room temperature. Prior to the Q-NIRS measurements, the optical probes were calibrated using a silicone phantom with optical properties comparable to those of brain tissue. After patients were placed in a state of anesthesia, they were positioned on the operating table, their scalps were shaved, and monitoring probes were attached bilaterally (one each on the affected and the contralateral side). The NIRS and the frequency-domain multidistance method quantitatively separate absorption and scattering contributions.¹² By determining the light absorption coefficients at two wavelengths, this method uses the differences in the absorption spectra of HbO₂ and HHb in tissue to calculate the absolute levels of hemoglobin saturation and total blood volume ($\mu\text{mol/L}$). The four source-detector distances of the probe ranged from 1.98 to 3.50 cm with a mean distance of approximately 2.74 cm. A minimum depth of 0.3 cm of the superficial cortex covering a 1 cm² area was monitored. Using a two-layer model described elsewhere,⁵ the HbO₂ and HHb concentration and SO₂ of the adult brain can be calculated with good accuracy independent of the scalp and skull contributions. Absorption was recorded at two wavelengths (690 and 830 nm), corresponding to the maximum absorp-

TABLE 1
Summary of patient baseline and treatment outcome data*

Case No.	Age (yrs), Sex	Diagnosis	Treatment	Outcome
1	68, F	rt ICA ophthalmic aneurysm	aneurysm clipping	good
2	38, F	lt ICA ophthalmic aneurysm	aneurysm clipping	good
3	48, F	parietal AVM	resection	good
4	69, F	rt ICA stenosis	CEA	good
5	68, M	lt ICA stenosis	CEA	watershed infarcts in temporal-occipital region
6	68, M	lt ICA stenosis	CEA	good
7	76, M	rt ICA stenosis	CEA	good
8	52, F	rt MCA stenosis	rt STA-MCA bypass	good
9	85, F	bilateral VA stenosis	lt ECA-lt VA bypass	good
10	63, M	lt ICA occlusion	lt STA-MCA bypass	good
11	63, F	moyamoya disease	rt STA-MCA bypass	good
12	44, M	lt ICA occlusion	lt STA-MCA bypass	good
13	58, M	intracranial hypertension, IJV occlusion	rt transverse sinus-subclavian venous bypass	brain edema, death after 1 wk
14	73, F	lt ICA occlusion	lt STA-MCA bypass	good
15	52, F	rt ICA stenosis	rt STA-MCA bypass	good
16	59, F	rt ICA occlusion	rt ECA-ICA bypass	good
17	59, M	rt ICA occlusion	rt STA-MCA bypass	good
18	65, F	lt ICA occlusion	lt STA-MCA bypass	good
19	66, F	lt MCA aneurysm	lt ECA-ICA bypass	good
20	21, M	moyamoya disease	rt EDMS	good
21	52, F	rt ICA aneurysm	BOT	passed
22	57, F	rt sphenoid wing meningioma	BOT	passed
23	80, F	rt ICA cavernous aneurysm	BOT	hypoperfusion on SPECT
24	72, F	rt skull base tumor	BOT	passed
25	81, F	lt ICA cavernous aneurysm	BOT	failed clinically

* AVM = arteriovenous malformation; BOT = balloon occlusion testing; ECA = external carotid artery; EDMS = encephaloduromysynangiosis; IJV = internal jugular vein; MCA = middle cerebral artery; SPECT = single photon emission computed tomography; STA = superficial temporal artery; VA = vertebral artery.

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tion of HbO₂ and HHb, respectively. Continuous arterial blood pressure, heart rate, and peripheral SO₂ were also recorded at the same time. This approach allowed us to measure cerebral tissue SO₂, quantitative tissue hemoglobin (HbO₂ and HHb) concentrations, and tHb concentrations in both hemispheres of the brain. Intraoperative peripheral hemoglobin measurements were also obtained during the monitoring procedure.

For surgery involving the anterior cerebral artery, we placed the probes on the medial frontal region of the head. For other surgeries, the probes were placed on the parietal region of the head. The probes were firmly attached by means of a medical adhesive. The duration of brain oxygenation and hemodynamic changes were recorded during the entire procedure.

Results

Intraoperative Monitoring

Continuous recordings of quantitative HbO₂, HHb, tHb, and SO₂ were obtained from both cerebral hemispheres during the 25 procedures. The HbO₂ concentration level obtained before skin incision was used as the baseline point. Any acute event with a decrease in HbO₂ concentration below the baseline point was recorded and noted. These events were manifested by a decrease in HbO₂ and tHb with a concomitant rise in HHb, which represents a decrease in cerebral blood volume (indirectly measured by a decrease

in tHb) and an increase in oxygen consumption (indirectly measured by an increase in HHb).

Of all patients monitored, five showed evidence of clinical ischemic events during the procedures: one patient suffered blood loss with systemic hypotension and developed diffuse brain edema intraoperatively; one patient suffered an intraoperative ischemic event and developed an occipital stroke postoperatively; one patient showed a slowing of brain activity on electroencephalography intraoperatively during carotid clamping; and in two patients balloon occlusion testing failed. In all recorded clinical ischemic events, Q-NIRS monitoring showed a decrease in HbO₂, tHb, and SO₂, with an increase in HHb.

In the five patients with ischemic events, the HbO₂ decreases ranged from 3.3 to 11.5 $\mu\text{mol/L}$ (20.5–82.5% from baseline); tHb decreases ranges from 1 to 9.7 $\mu\text{mol/L}$ (1.8–35.6% from baseline); and SO₂ decreases ranged from 3 to 26.2% (4.4–90% from baseline). An increase in HHb from 2.8 to 4 $\mu\text{mol/L}$ (8–38.3% from baseline) was detected. For the 20 patients who did not suffer ischemic events, HbO₂ decreases ranged from 0.5 to 11 $\mu\text{mol/L}$ (2–24% from baseline); tHb decreases on the ipsilateral side ranged from 0 to 10 $\mu\text{mol/L}$ (0.7–25% from baseline); and SO₂ decreases ranged from 1 to 16% (1.5–36.3% from baseline). An increase in HHb from 2.2 to 9.5 $\mu\text{mol/L}$ (2.4–35.8% from baseline) was detected in these 20 patients. Based on these data, HbO₂ was the parameter that best discriminated between patients with and without ischemic events ($p = 0.03$, two-tailed t-test; Fig. 1).

FIG. 1. Graph showing a comparison of the average percentage increase or decrease for HbO₂, HHb, tHb, and SO₂ using Q-NIRS monitoring in patients who were ischemic or nonischemic.

In addition to vessels that are compromised intraoperatively, acute events can be precipitated by the following: an acute drop in blood pressure, especially in the setting of impaired autoregulation (in patients undergoing bypass surgery for cerebral hypoperfusion); an acute drop in systemic hematocrit (in patients with bleeding); or systemic hypoxemia (in patients with compromised respiratory systems). No patient experienced instances of respiratory compromise during surgery. An acute decrease in hematocrit was recorded in one patient (Case 13) with severe intraoperative bleeding.

Illustrative Cases

Case 4

This 69-year-old woman presented with recent onset of recurrent transient ischemic attacks over a 3-week period. She was evaluated using cerebral angiography, which showed a 70% stenosis in the right ICA. Quantitative frequency-domain NIRS detected lower oxygenation (HbO₂) at baseline on the affected side (3 $\mu\text{mol/L}$) compared with the nonaffected side (11 $\mu\text{mol/L}$). During a right CEA, the right ICA was clamped temporarily; electroencephalography showed slowing after 2 minutes, which necessitated arterial shunt placement. Quantitative frequency-domain NIRS monitoring revealed immediate cerebral ischemia after the ICA was clamped: HbO₂ decreased from 4 to 0.7 $\mu\text{mol/L}$ (82.5% from baseline); tHb dropped from 18.5 to 16.2 $\mu\text{mol/L}$ (12.4% from baseline); SO₂ dropped from 20 to 2% (90% from baseline); and HHb increased from 15 to 19 $\mu\text{mol/L}$ (21% from baseline). This ischemic state improved after an intraluminal shunt was placed; however, it did not reestablish oxygenation to the baseline level. A second ischemic event was noted when the ICA was clamped again briefly to extract the arterial shunt (Fig. 2). The changes detected by Q-NIRS monitoring preceded the intraoperative slowing of brain activity on electroencephalography. The patient had no neurological complications after surgery.

Case 13

This 58-year-old man presented with severe intracranial hypertension secondary to bilateral occlusion of both internal jugular veins after radical neck dissection and past radiation therapy. Right transverse sinus–subclavian vein bypass surgery was performed using a Gore-Tex venous graft. During anastomosis, severe bleeding from the sinus was encountered, along with hypotension and massive intraoperative brain swelling. Continuous Q-NIRS monitoring showed acute ischemia in both hemispheres, but more severely on the ipsilateral side: HbO₂ decreased from 18.2 to 6.7 $\mu\text{mol/L}$ (63.1% from baseline); tHb decreased from 25.5 to 16.4 $\mu\text{mol/L}$ (35.6% from baseline); SO₂ decreased from 81.5 to 55.5% (32% from baseline); and HHb increased from 4.5 to 7.3 $\mu\text{mol/L}$ (38.3% from baseline) (Fig. 3). These changes corresponded to a decrease in the mean systemic arterial pressure to 40 mm Hg, a drop in the hematocrit, brain edema, and an increase in intracranial pressure with brain herniation outside the calvarial defect.

FIG. 2. Case 4. Graphs showing Q-NIRS values of HbO₂, tHb, SO₂, and HHb during a right CEA. Oxygenation baseline was lower on the affected side. Arrows denote the following stages of the operation: 1, right ICA was clamped and EEG waves slowed, resulting in HbO₂, tHb, and SO₂ decreases and an increase in HHb on the ipsilateral side; 2, shunt placement, resulting in significant improvement of cerebral oxygenation but not to baseline level; 3, ICA reclamped; and 4, reestablishment of ICA flow with possible postischemic hyperemia.

Case 25

This 81-year-old woman presented with a 1-week history of worsening left eye palsy and ptosis. She was evaluated using cerebral angiography, which demonstrated a 2.5-cm left cavernous ICA aneurysm. The patient underwent endovascular balloon occlusion testing. The balloon was placed in the ICA; 3 minutes after inflation of the balloon, aphasia developed and the patient experienced a right facial droop and right upper- and lower-extremity weakness.

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These symptoms were associated with a concomitant and immediate decrease in cerebral oxygenation as demonstrated by Q-NIRS monitoring on the ipsilateral side: HbO₂ decreased from 15.5 to 11.5 $\mu\text{mol/L}$ (25.8% from baseline); tHb decreased from 30 to 27 $\mu\text{mol/L}$ (10% from baseline); SO₂ dropped from 50 to 44% (12% from baseline); and HHb increased from 13.5 to 15.2 $\mu\text{mol/L}$ (11.1% from baseline). The contralateral hemisphere showed a compensatory increase in brain oxygenation. The balloon was deflated with subsequent complete resolution of the symptoms and restoration of brain oxygenation as shown by Q-NIRS. The continuous EEG monitoring showed no changes, demonstrating greater sensitivity of Q-NIRS monitoring to the clinical ischemic event (Fig. 4).

Discussion

Neuromonitoring Methods

There is an inherent risk of perioperative stroke during neurovascular operations. The primary cause of perioperative stroke is hypoperfusion due to temporary or permanent occlusion of a vessel, intraoperative retraction, or systemic hypotension.^{1,46} Inadvertent permanent occlusion of a feeding or perforating artery occurs in 3.1% of patients undergoing aneurysm surgery and is a primary cause of intraoperative complications.¹⁸ In these patients, the prompt and accurate recognition of insufficient collateral circulation is crucial to a good neurological outcome. Transcranial Doppler ultrasonographic, EEG, and SSEP monitoring are established methods of neuromonitoring during neurovascular procedures.⁴ The role of intraoperative EEG monitoring can be limited during aneurysm surgery if burst suppression is used. Electrophysiological monitoring, particularly with SSEPs, is an accepted method of detecting procedure-related cerebral ischemia.^{16,24,25,33,34,36} There is a considerable time delay after potential ischemic events,^{3,24} however, with limited predictability of these events on an individual basis.^{14,36} Additionally, anesthesia can alter SSEPs through several other mechanisms.^{21,23}

In this study we examined the role of Q-NIRS monitoring of ischemic events during neurovascular procedures. Twenty-five patients were monitored throughout the neurosurgery. In all cases with evidence of ischemic events (brain edema, slowing of electroencephalography, postoperative stroke, and failure of balloon occlusion testing), there was a decrease in HbO₂ and tHb concentrations and brain tissue SO₂, with an increase in HHb concentrations.

Role of NIRS

Continuous-wave NIRS has been available since 1977 as a tool to measure changes in the hemoglobin oxygenation state in the human brain.¹⁷ Use of CW-NIRS allowed observation of dynamic changes in regional blood flow in real time by measuring concentration changes in hemoglobin, but failed to provide absolute values of hemoglobin concentrations. Thus, CW-NIRS measurements could not be compared between individuals, or between regions within the same individual.

The use of semiquantitative NIRS (INVOS 3100A, Somanetics Corp.)—a form of CW-NIRS—for neuromonitoring in carotid artery surgery has been assessed,²⁷ but studies in which semiquantitative NIRS was used, failed to demon-

FIG. 3. Case 13. Graphs showing Q-NIRS values of HbO₂, tHb, SO₂, and HHb during a right transverse sinus–subclavian vein bypass. Arrows denote the following stages of the operation: 1, acute bleeding with drop in mean arterial pressure and an associated decrease in HbO₂, tHb, and SO₂ and an increase in HHb in both hemispheres; 2, blood transfusion and subsequent slow improvement of mean arterial pressure; and 3, recurrence of bleeding and moderate hypotension.

strate clinical usefulness in this setting.^{19,42} Moreover, Beese et al.² used semiquantitative NIRS to study patients with restricted cerebral circulation, as indicated by the loss of cortical SSEPs; their results showed that severe cerebral ischemia, as indicated by cortical potential loss, occurred in some patients with minimal or even no change of the regional cerebral oxygenation displayed using semiquantitative NIRS. Data from previous studies demonstrated that absolute numbers of regional SO₂ are of little value in interpreting results obtained by semiquantitative NIRS.^{6,8,26,28,32,40,41} Recently, Q-NIRS measurements were introduced, which enabled accurate quantification of brain tissue oxygenation.^{5,9,15,39,43}

FIG. 4. Case 25. Graphs showing Q-NIRS values of HbO_2 , tHb , SO_2 , and HHb during a balloon occlusion test on the left side. Arrows denote the following stages of the operation: 1, inflation of the balloon within the left ICA; 2, patient became aphasic, experienced right facial droop and right upper- and lower- extremity weakness, corresponding with an immediate decrease in HbO_2 , tHb , and SO_2 , an increase in HHb in the left hemisphere, and an increase in brain oxygenation in the right hemisphere; 3, occlusion balloon was deflated, resulting in complete resolution of the symptoms and restoration of normal oxygenation in the left hemisphere.

Quantitative frequency-domain NIRS has two major advantages compared with CW-NIRS: it provides quantitative values of hemoglobin concentrations (HbO_2 , HHb , tHb), and it directly monitors brain tissue oxygenation independently from oxygenation of the skin and scalp.^{5,15} Unlike CW-NIRS, Q-NIRS readings of cerebral oxygen concentration are not affected by extracranial layers of the scalp, skull, and CSF.^{11,22,29} When brain tissue is measured by NIRS, photons travel across layers of the cranium display-

ing different optical properties as the light's path to the brain is intersected by the scalp, skull, and CSF. Unless the layered structure is accounted for, measurements could be inaccurate. There are results from many studies demonstrating the ability of Q-NIRS to isolate brain tissue oxygenation from skin and scalp oxygenation. Franceschini et al.¹⁰ demonstrated in multilayered silicone phantoms that Q-NIRS successfully recovered the optical properties of the lowest layer at approximately 1 cm from the surface. This phantom was next used to model the multilayer architecture of the head (skin, scalp, CSF, and brain). Furthermore, Choi et al.⁵ used Q-NIRS to demonstrate that the adult human head can be reasonably described by a two-layer model, and by using this model, the hemoglobin concentration and SO_2 of the brain can be calculated with good accuracy. Other studies have used concomitant Q-NIRS and functional magnetic resonance imaging of brain hemodynamics to generate functional maps during task-related brain activation. A high temporal correlation between the blood oxygenation level-dependent signal (using functional magnetic resonance imaging) and the HHb concentration (using Q-NIRS) was demonstrated.³⁹ This correlation can only be explained by the simultaneous detection of brain activation by both methods.

Detection of Ischemic Events

Due to the quantitative nature of Q-NIRS measurements, recordings can be compared over time as well as with recordings obtained in other patients. Factors that could affect absolute cerebral tissue oxygenation intraoperatively include the baseline status of the cerebral tissue perfusion, presence and duration of vessel compromise, increase in intracranial pressure, extent of brain oxygen consumption (including the effects of EEG burst suppression), systemic changes in blood pressure, and systemic oxygen carrying capacity (as reflected by hematocrit). A change in any one of these variables can result in a decrease in brain tissue oxygen content, which can precipitate brain ischemia. In this study, changes in tissue oxygenation during surgery occurred in patients both with and without ischemic events, although the absolute decrease in HbO_2 appeared to be the best discriminating factor between the groups (Fig. 1). A tissue oxygenation threshold that patients have to reach before developing ischemia may not solely depend on the degree of oxygenation decrease. Given that the ischemic insult to the brain depends on both the degree and duration of decreased oxygenation, a future paradigm that combines these two factors may be the most appropriate for predicting ischemic insults, although such a predictive model is beyond the scope of this study.

Our findings support a useful role for Q-NIRS in intraoperative monitoring, although the small number of documented procedure-related ischemic events limits the ability to generalize our results. Based on further data collection, we hope that in the future we will further validate a threshold value that indicates severe cerebral ischemia.

Limitations of Q-NIRS

The main limitation of the Q-NIRS technology is its restriction to cortical brain tissues and the area beneath the probe. Although cortical tissues are primarily at risk of infarction after prolonged arterial occlusion, deeper tissues

supplied by perforators can be selectively at risk as well, and cannot currently be monitored using Q-NIRS. Furthermore, only relatively small regions of any particular vascular territory can be monitored because the average monitored area is approximately 1 cm². The use of multiple probes may reduce this particular limitation, but does not currently address the lack of monitoring for deep tissues. Although the accuracy of Q-NIRS recordings is independent of the skull and scalp,^{5,13,37–39,43–45} increased skull thickness reduces the breadth and depth by which the near-infrared light travels into the brain tissues, thus reducing the area of tissue measured by this method. In the future, advances in Q-NIRS technology that allow the ability to monitor deeper tissues over a larger area would be optimal.

Currently, given the limitations of the technology, Q-NIRS may not be optimal for detection of focal territory or ischemia in perforating vessels. For example, during aneurysm or distal bypass surgery, the Q-NIRS probes may not predictably monitor the specific cortical, or deep, region of tissue at risk. The primary clinical applications of Q-NIRS to cerebrovascular surgery would be procedures in which there is potential for large territory ischemia, such as balloon occlusion testing and CEA. In such cases, the hemispheric reduction in blood flow would be easily amenable to Q-NIRS monitoring. Additionally, procedures involving the risk of severe intraoperative bleeding with the potential for global hypoperfusion would be amenable to monitoring with Q-NIRS.

Conclusions

Quantitative frequency-domain NIRS provides quantitative, noninvasive, and continuous real-time information about brain oxygenation and hemodynamics directly related to vascular events. It appears that the use of this technique is feasible and can provide accurate information in the operating room during certain neurovascular procedures. Continuous intraoperative oxygenation monitoring with Q-NIRS to provide real-time feedback on absolute tissue oxygenation may improve the outcome of neurovascular procedures by minimizing ischemia.

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Disclaimer

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References

- Andrews RJ, Bringas JR: A review of brain retraction and recommendations for minimizing intraoperative brain injury. **Neurosurgery** 33:1052–1064, 1993
- Beese U, Langer H, Lang W, Dinkel M: Comparison of near-infrared spectroscopy and somatosensory evoked potentials for the detection of cerebral ischemia during carotid endarterectomy. **Stroke** 29:2032–2037, 1998
- Buchthal A, Belopavlovic M, Mooij JJ: Evoked potential monitoring and temporary clipping in cerebral aneurysm surgery. **Acta Neurochir (Wien)** 93:28–36, 1988

- Cheng MA, Theard MA, Tempelhoff R: Anesthesia for carotid endarterectomy: a survey. **J Neurosurg Anesthesiol** 9:211–216, 1997
- Choi J, Wolf M, Toronov V, Wolf U, Polzonetti C, Hueber D, et al: Noninvasive determination of the optical properties of adult brain: near-infrared spectroscopy approach. **J Biomed Opt** 9:221–229, 2004
- Colier WN, van Haaren NJ, Oeseburg B: A comparative study of two near infrared spectrophotometers for the assessment of cerebral haemodynamics. **Acta Anaesthesiol Scand Suppl** 107:101–105, 1995
- Doyle PW, Matta BF: Burst suppression or isoelectric encephalogram for cerebral protection: evidence from metabolic suppression studies. **Br J Anaesth** 83:580–584, 1999
- Duncan LA, Ruckley CV, Wildsmith JA: Cerebral oximetry: a useful monitor during carotid artery surgery. **Anaesthesia** 50:1041–1045, 1995
- Fantini S, Hueber D, Franceschini MA, Gratton E, Rosenfeld W, Stubblefield PG, et al: Non-invasive optical monitoring of the newborn piglet brain using continuous-wave and frequency-domain spectroscopy. **Phys Med Biol** 44:1543–1563, 1999
- Franceschini MA, Paunescu LA, Fantini S, Pratesi S, Maier JS, Donzelli GP, et al: Frequency-domain optical measurements in vitro on two- and three-layered tissue-like phantoms, and in vivo on infant heads. **OSA TOPS** 21:232–236, 1998
- Gomersall CD, Joynt GM, Gin T, Freebairn RC, Stewart IE: Failure of the INVOS 3100 cerebral oximeter to detect complete absence of cerebral blood flow. **Crit Care Med** 25:1252–1254, 1997
- Gratton E, Fantini S, Franceschini MA, Gratton G, Fabiani M: Measurements of scattering and absorption changes in muscle and brain. **Philos Trans R Soc Lond B Biol Sci** 352:727–735, 1997
- Gratton E, Toronov V, Wolf U, Wolf M, Webb A: Measurement of brain activity by near-infrared light. **J Biomed Opt** 10:11008, 2005
- Holland NR: Subcortical strokes from intracranial aneurysm surgery: implications for intraoperative neuromonitoring. **J Clin Neurophysiol** 15:439–446, 1998
- Hueber DM, Franceschini MA, Ma HY, Zhang Q, Ballesteros JR, Fantini S, et al: Non-invasive and quantitative near-infrared haemoglobin spectrometry in the piglet brain during hypoxic stress, using a frequency-domain multidistance instrument. **Phys Med Biol** 46:41–62, 2001
- Jabre A, Symon L: Temporary vascular occlusion during aneurysm surgery. **Surg Neurol** 27:47–63, 1987
- Jobsis FF: Noninvasive, infrared monitoring of cerebral and myocardial oxygen sufficiency and circulatory parameters. **Science** 198:1264–1267, 1977
- Kassell NF, Torner JC, Haley EC Jr, Jane JA, Adams HP, Kongable GL: The International Cooperative Study on the Timing of Aneurysm Surgery. Part I: overall management results. **J Neurosurg** 73:18–36, 1990
- Kuroda S, Houkin K, Abe H, Hoshi Y, Tamura M: Near-infrared monitoring of cerebral oxygenation state during carotid endarterectomy. **Surg Neurol** 45:450–458, 1996
- Lavine SD, Masri LS, Levy ML, Giannotta SL: Temporary occlusion of the middle cerebral artery in intracranial aneurysm surgery: time limitation and advantage of brain protection. **J Neurosurg** 87:817–824, 1997
- Lopez JR, Chang SD, Steinberg GK: The use of electrophysiological monitoring in the intraoperative management of intracranial aneurysms. **J Neurol Neurosurg Psychiatry** 66:189–196, 1999
- McKeating EG, Monjardino JR, Signorini DF, Souter MJ, Andrews PJ: A comparison of the Invos 3100 and the Critikon 2020 near-infrared spectrophotometers as monitors of cerebral oxygenation. **Anaesthesia** 52:136–140, 1997
- McPherson RW, Sell B, Traystman RJ: Effects of thiopental, fentanyl, and etomidate on upper extremity somatosensory evoked potentials in humans. **Anesthesiology** 65:584–589, 1986

24. Mizoi K, Yoshimoto T: Permissible temporary occlusion time in aneurysm surgery as evaluated by evoked potential monitoring. **Neurosurgery** 33:434–440, 1993
25. Mooij JJ, Buchthal A, Belopavlovic M: Somatosensory evoked potential monitoring of temporary middle cerebral artery occlusion during aneurysm operation. **Neurosurgery** 21:492–496, 1987
26. Olsen KS, Svendsen LB, Larsen FS: Validation of transcranial near-infrared spectroscopy for evaluation of cerebral blood flow autoregulation. **J Neurosurg Anesthesiol** 8:280–285, 1996
27. Pollard V, Prough DS: Cerebral near-infrared spectroscopy: a plea for modest expectations. **Anesth Analg** 83:673–674, 1996
28. Pollard V, Prough DS, DeMelo AE, Deyo DJ, Uchida T, Stoddart HF: Validation in volunteers of a near-infrared spectroscope for monitoring brain oxygenation in vivo. **Anesth Analg** 82:269–277, 1996
29. Pollard V, Prough DS, DeMelo AE, Deyo DJ, Uchida T, Widman R: The influence of carbon dioxide and body position on near-infrared spectroscopic assessment of cerebral hemoglobin oxygen saturation. **Anesth Analg** 82:278–287, 1996
30. Reinsfelt B, Westerlind A, Houltz E, Ederberg S, Elam M, Ricksten SE: The effects of isoflurane-induced electroencephalographic burst suppression on cerebral blood flow velocity and cerebral oxygen extraction during cardiopulmonary bypass. **Anesth Analg** 97:1246–1250, 2003
31. Roach GW, Newman MF, Murkin JM, Martzke J, Ruskin A, Li J, et al: Ineffectiveness of burst suppression therapy in mitigating perioperative cerebrovascular dysfunction. Multicenter Study of Perioperative Ischemia (McSPI) Research Group. **Anesthesiology** 90:1255–1264, 1999
32. Samra SK, Dorje P, Zelenock GB, Stanley JC: Cerebral oximetry in patients undergoing carotid endarterectomy under regional anesthesia. **Stroke** 27:49–55, 1996
33. Schramm J, Koht A, Schmidt G, Pechstein U, Taniguchi M, Fahlbusch R: Surgical and electrophysiological observations during clipping of 134 aneurysms with evoked potential monitoring. **Neurosurgery** 26:61–70, 1990
34. Schramm J, Zentner J, Pechstein U: Intraoperative SEP monitoring in aneurysm surgery. **Neurol Res** 16:20–22, 1994
35. Stone JG, Young WL, Marans ZS, Solomon RA, Smith CR, Jamdar SC, et al: Consequences of electroencephalographic-suppressive doses of propofol in conjunction with deep hypothermic circulatory arrest. **Anesthesiology** 85:497–501, 1996
36. Taylor CL, Selman WR, Kiefer SP, Ratcheson RA: Temporary vessel occlusion during intracranial aneurysm repair. **Neurosurgery** 39:893–906, 1996
37. Toronov V, Franceschini MA, Filiaci M, Fantini S, Wolf M, Michalos A, et al: Near-infrared study of fluctuations in cerebral hemodynamics during rest and motor stimulation: temporal analysis and spatial mapping. **Med Phys** 27:801–815, 2000
38. Toronov V, Walker S, Gupta R, Choi JH, Gratton E, Hueber D, et al: The roles of changes in deoxyhemoglobin concentration and regional cerebral blood volume in the fMRI BOLD signal. **Neuroimage** 19:1521–1531, 2003
39. Toronov V, Webb A, Choi JH, Wolf M, Michalos A, Gratton E, et al: Investigation of human brain hemodynamics by simultaneous near-infrared spectroscopy and functional magnetic resonance imaging. **Med Phys** 28:521–527, 2001
40. Williams IM, Mortimer AJ, McCollum CN: Recent developments in cerebral monitoring-near-infrared light spectroscopy. An overview. **Eur J Vasc Endovasc Surg** 12:263–271, 1996
41. Williams IM, Picton A, Farrell A, Mead GE, Mortimer AJ, McCollum CN: Light-reflective cerebral oximetry and jugular bulb venous oxygen saturation during carotid endarterectomy. **Br J Surg** 81:1291–1295, 1994
42. Williams IM, Picton AJ, Hardy SC, Mortimer AJ, McCollum CN: Cerebral hypoxia detected by near infrared spectroscopy. **Anaesthesia** 49:762–766, 1994
43. Wolf M, Franceschini MA, Paunescu LA, Toronov V, Michalos A, Wolf U, et al: Absolute frequency-domain pulse oximetry of the brain: methodology and measurements. **Adv Exp Med Biol** 530:61–73, 2003
44. Wolf M, Wolf U, Choi JH, Gupta R, Safonova LP, Paunescu LA, et al: Functional frequency-domain near-infrared spectroscopy detects fast neuronal signal in the motor cortex. **Neuroimage** 17:1868–1875, 2002
45. Wolf M, Wolf U, Choi JH, Toronov V, Paunescu LA, Michalos A, et al: Fast cerebral functional signal in the 100-ms range detected in the visual cortex by frequency-domain near-infrared spectrophotometry. **Psychophysiology** 40:521–528, 2003
46. Zhong J, Dujovny M, Perlin AR, Perez-Arjona E, Park HK, Diaz FG: Brain retraction injury. **Neurol Res** 25:831–838, 2003

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