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Cerebral Oximetry Monitoring in Anesthesiology

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Cerebral oximetry is a neurological monitoring modality that was developed in the 1970s[1] for adult[2] and pediatric[3] cardiac surgery; today, this type of monitoring is also applied to noncardiac surgery,[4,5] cardiology,[6] resuscitation,[7] trauma,[8,9] neurology,[10] and neurosurgery.[11] The technology is similar to that of pulse oximetry, but it is applied to the brain and indicates a saturation number that reflects mainly venous blood. The objectives for this issue of *Anesthesiology Rounds* are: to highlight the significance of cognitive dysfunction in cardiac surgery; to describe the functioning of cerebral oximetry; to define the role of oximetry in the prevention of cognitive dysfunction; and to introduce the current protocol used at the Montreal Heart Institute.

SIGNIFICANCE OF COGNITIVE DYSFUNCTION IN CARDIAC SURGERY

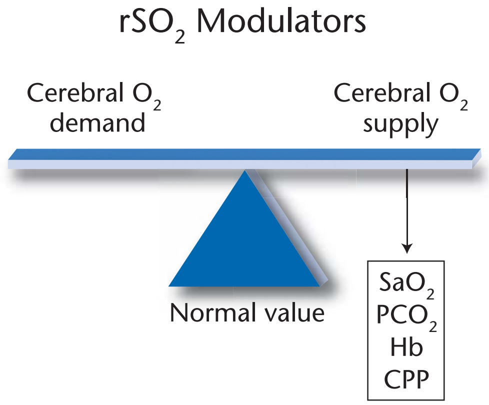
Neurological and neurocognitive dysfunction in cardiac surgery has been a major incentive for developing a monitor of neurological function. In 2001, Newman et al[12] demonstrated that more than half (56%) of the patients undergoing cardiac revascularization surgery presented with cognitive dysfunction at discharge. Furthermore, another study noted neurological anomalies in 6% of patients undergoing surgery for cardiac congenital anomalies.[13] Several factors can cause these frequent cognitive dysfunctions; for example, embolism, inflammation, hypoperfusion, systemic desaturation, and intraoperative anemia could constitute plausible mechanisms. However, it is possible that the common denominator of all these complications is an imbalance between the oxygen supply to the brain and the demand, ie, global microcirculatory tissue hypoxia. This concept is the basis for cerebral oximetry monitoring, which measures regional cerebral saturation (rSO_2; Figure 1). An imbalance between oxygen supply and demand will be detected by the presence of desaturation (Figure 2). In animal models of cardiopulmonary bypass (CPB), this desaturation is associated with cell injury.[14]

In adult cardiac surgery, Yao et al[15] used multivariate logistic regression in 101 patients to demonstrate that the severity of cerebral desaturation was the only factor predicting deterioration among the cognitive tests used. In a study examining 143 children undergoing cardiac surgery, rSO_2 baseline values < 50% or a decrease below that threshold predicted increased mortality.[16] In a study of the same population, Dent et al[17] established a correlation between cerebral anomalies observed with magnetic resonance and duration of rSO_2 at ≤45%. In elderly patients (72 ± 5 years) undergoing noncardiac surgery, Casati et al[18] noted that 26% of patients suffered cerebral desaturation during the procedure. In hepatic transplantation, a correlation was noted between rSO_2 decreases and an increase in neuron-specific enolase, a marker of cerebral injury.[19] However, the clinical outcomes of these rSO_2 decreases were not associated with cerebrovascular accidents.

PRINCIPLE OF OPERATION FOR CEREBRAL OXIMETRY

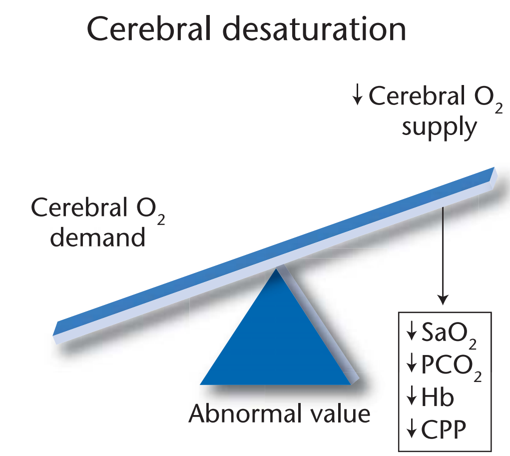
Cerebral oximetry is based on the Beer-Lambert law, which stipulates that the concentration of a substance can be measured by the magnitude of light absorption.

FIGURE 1: **Regional cerebral oximetry value (rSO_2) depends on the balance between cerebral oxygen supply and demand**



This is influenced by oxygen saturation of arterial blood (SaO_2), carbon dioxide partial pressure (PCO_2), hemoglobin (Hb) and cerebral perfusion pressure (CPP).

FIGURE 2: **Cerebral desaturation implies an imbalance between cerebral oxygen supply and demand**



SaO_2: oxygen saturation of arterial blood
PCO_2: carbon dioxide partial pressure
Hb: hemoglobin; CPP: cerebral perfusion pressure.

There are a few cerebral oximetry systems, such as the NIRO 500/1000 (Hamamatsu Photonics, UK) and the near infrared spectroscopy, (NIRS; Somanetics Corp, Troy, MI) that function differently. The NIRS is approved as a trend monitor by the Food and Drug Administration in the United States, and by Health Canada;[20] nevertheless, there is controversy as to the real significance of the absolute rSO_2 value obtained by cerebral oximetry (see following sections). On the other hand, changes in the oximetry signal with time compared with baseline and the correction of rSO_2 decreases are the basis for common applications of this type of monitoring.

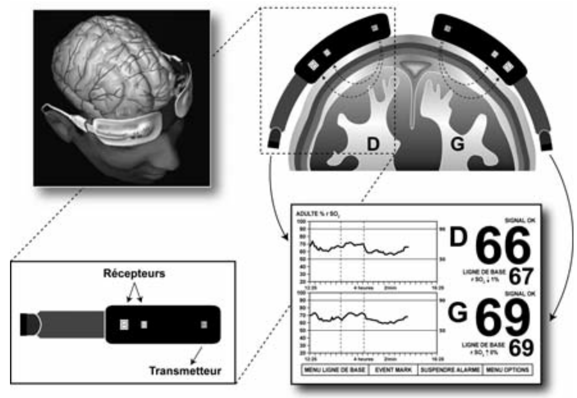
Since 2002, we have used the NIRS INVOS 4100 model, which has an infrared transmitting diode that sends photons of two different wavelengths (730 and 810 nm) through the frontal region. These photons are absorbed by the chromophore component of oxygenated and deoxygenated hemoglobin in blood vessels >1 mm. These vessels under investigation have a content with venous predominance (proportions, venous: arterial: capillary = ~70%: 25%:5%). Photon penetration takes the shape of an arch going through the scalp, the bone, and the cerebral tissue. The length of the path of the arch is equal to a third of the distance between transmitter and receiver. Photons go through these regions and a nonabsorbed fraction is picked up again by two silicon photodiodes. These two receivers are located 30 mm (proximal) and 40 mm (distal) away from the transmitter, respectively (Figure 3). Photon penetration is about 1.5 cm and investigated volume is about 1.5 cm^3. The signal obtained by the proximal receiver comes from a more superficial source, whereas the signal obtained by the distal receiver includes this superficial zone as well as a deeper component. The proximal signal is then subtracted from the distal signal to obtain the value of a distal signal without the superficial or extracranial components. The result thus reflects cerebral tissue saturation. The term, rSO_2, is taken as a measure of regional cerebral oxygenation (Figure 3).

In this manner, it is understood that any anatomical interference can modify the final signal result. For instance, scalp, frontal bone (bone cyst), or subdural structures (frontal sinus) anomalies can constitute confounding variables that can be associated with abnormal oximetry baseline values. The standard cerebral oximetry value is approximately 67% ± 10% and measurement accuracy is about 3%-6%.[22,23] The standard rSO_2 value is usually lower in female[24] and elderly[25] patients. A decrease in rSO_2 with time indicates a reduction in cerebral blood flow. It is generally accepted that: the ischemic threshold of cerebral oximetry is approximately 47%; lactate production increases at 45%; electroencephalographic activity decreases between 35%-40%; and the 30%-35% threshold is associated with cellular dysfunction. This latter threshold is also associated with increased operative mortality (Figure 4).[26] A relative change of 20% compared with baseline or an absolute value <50% are usually considered triggers for intervention.[2,4,15,27-29]

ADVANTAGES IN CORRECTING DESATURATIONS MEASURED BY CEREBRAL OXIMETRY

Two recent randomized trials in noncardiac[30] and cardiac[31] surgery support the hypothesis that correcting desaturation is associated with a shorter hospital stay and fewer postoperative complications. A study by Casati et al[30] assessed 122 patients from 5 Italian university hospital centres. These patients were

FIGURE 3: Principle of operation in cerebral oximetry (see text).



The resultant of each signal (right and left) is sent on a screen that shows the change of the two signals with time (adapted from Taillefer and Denault[2]).

undergoing major abdominal surgery with a duration of >2 hours. In total, 66 patients were randomized to cerebral oximetry monitoring without intervention and 56 to the intervention group. Twelve patients in the control group and six in the intervention group developed postoperative complications. However, this difference was not statistically significant (P=0.20). Cerebral desaturation was observed in 23% of patients in the control group and 20% in the intervention group. Time spent in the recovery room was shorter in the intervention group (25 min vs 47 min in the control group), as was hospital stay (intervention group: 10 days vs 24 days in the control group). These differences were statistically significant. Neurological examination (Mini Mental State Examination; MMSE) conducted before the procedure and 7 days after surgery remained unchanged when comparing all patients in the control group with those of the intervention group. However, if the examination was limited only to those patients who experienced at least one desaturation event, MMSE results were lower in the control group 7 days after surgery, compared with the intervention group (P=0.02). Further, a correlation was observed between the extent of desaturation and performance decline with the MMSE; these results are similar to those of the Yao study in cardiac surgery.[15] Finally, the authors noted a correlation between cerebral desaturation and hospital length of stay.[30]

Murkin et al[31] conducted a study in 200 patients at the London Health Science Centre in Ontario. These patients were scheduled for coronary revascularization surgery. The randomization process was similar to that of the previous study, and a series of interventions were identified *a priori* for the intervention group. The two groups were similar except for the use of aprotinin, which was more frequent in the intervention group (83% vs 69%). Cerebrovascular accidents were noted in 4 patients from the control group and in 1 patient from the intervention group. The postoperative morbidity and mortality index, based on the Society of Thoracic Surgeons score, was 11 in the control group vs 3 in the intervention group (P=0.048). These two studies support the hypothesis that correcting cerebral desaturation episodes has an impact on the prevention of postoperative complications.

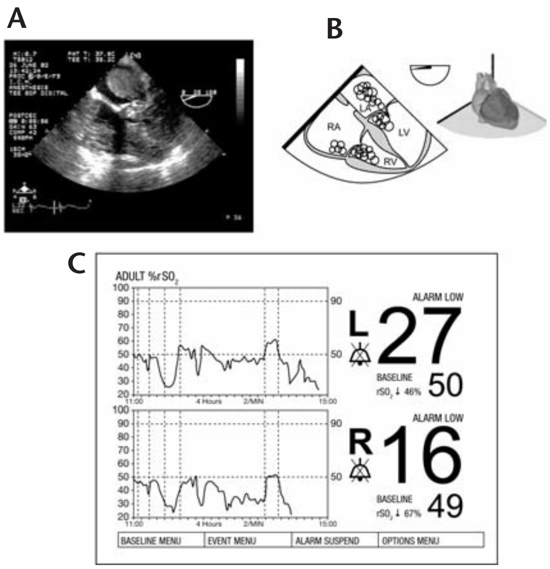
In addition, retrospective studies noted a reduction in neurological complications, hospital stay, morbidity, and mortality, after implementation of cerebral oximetry monitoring in the operating room.[4,28,32-35] Dozens of case reports have revealed the usefulness of this monitoring system that allows, among other things, the detection and prevention of catastrophes such as accidental occlusion of brain vessels and air embolism.[36-39]

LIMITATIONS

There are numerous questions and controversies pertaining to cerebral oximetry, and these have recently been debated.[35,40-42] One of the arguments against cerebral oximetry is based on the interpretation of the signal's absolute value. For example, when used on cadavers, the mean oximetry values are 51%![43] Moreover, it is important to realize that oximetry signals give information only on a cortical frontal region of 1.5 cm^3 and, as mentioned earlier, anatomic factors can distort this value. Cerebral ischemia in an area adjacent to, but different from, the area under study could escape detection. The proposed ratio (venous: arterial: capillary = ~ 70%:25%:5%) could be different in certain types of intracranial conditions associated with cerebral edema. On the other hand, it must be recognized that cerebral oximetry is a trend monitoring technique, and it is the change over time that has prognostic value and indicates possible and desirable interventions. Retrospective studies demonstrating a connection between oximetry measurements and postoperative complications suffer from methodological problems (this issue was reviewed in 2005[2]). At that time, no randomized trial had been published; since then, the two published randomized trials did not have the required power to demonstrate a statistically significant reduction of cerebral pathologies and cognitive disorders, even though a tendency was observed. A study examining a larger population is warranted and would probably demonstrate this association.

Futhermore, would the prevention of cerebral desaturation be an indirect means to prevent tissue hypoperfusion? Does the early correction of cerebral desaturation allow the prevention of systemic injury, as observed in the studies of Casati and Murkin? The hypothesis that regional oxygenation measurements

FIGURE 4: First experience with cerebral oximetry at the Montreal Heart Institute



A 73-year-old man was re-operated for an aortic pseudo-aneurysm and developed a hypercoagulability syndrome postoperatively. (A-B) Four-chamber view by midesophageal section. We observed clots in all cardiac chambers. (C) Numerous cerebral desaturations were observed from the beginning of the procedure (Adapted from Denault et al.[48] Chapter 23:518).

play a significant role in resuscitation is supported by the Rivers et al[44] study on intensive care patients. In that study, continuous measurements of the central venous saturation were used and the study revealed that a protocol of central desaturation correction based on a resuscitation algorithm was associated with lower mortality rates. In summary, despite the described limitations, the studies of Casati and Murkin definitely arouse interest in the application of this technique and in developing research in this field.

Finally, the clinical impact of cerebral oximetry depends on several factors. In cardiac surgery, its impact depends not only on the expertise of the anesthesiologist concerning the physiological basis of this technique, but also on a deep understanding of surgical procedures. At the same time, it is equally important for the surgeon and the perfusionist to be familiar with its use, since they may be invited to act appropriately to correct cerebral desaturation. Finally, as with all complex processes, communication between the various players is key to success.

THE MONTREAL HEART INSTITUTE ALGORITHM

Cerebral oximetry has been used since June 2002 at the Montreal Heart Institute in both clinical (operating room, electrophysiology, and cardiac catheterization suites) and research settings.[2,45] We have been able to discern a correlation between desaturation and clinical outcome (Figure 4) and to observe several situations of isolated cerebral desaturation that have been corrected. The recent developments of minimally invasive cardiac surgery and the prevention of risks for accidental cerebrovascular occlusion have been critical factors encouraging the implementation of cerebral oximetry in operating rooms. Over time, we have developed a systematic approach based on identification and treatment of an abnormal rSO_2, which was recently described[46] and is summarized in Figure 5. This approach is based on our experience with the intervention procedures to correct abnormal rSO_2 values described in the studies by Casati[30] and Murkin.[31]

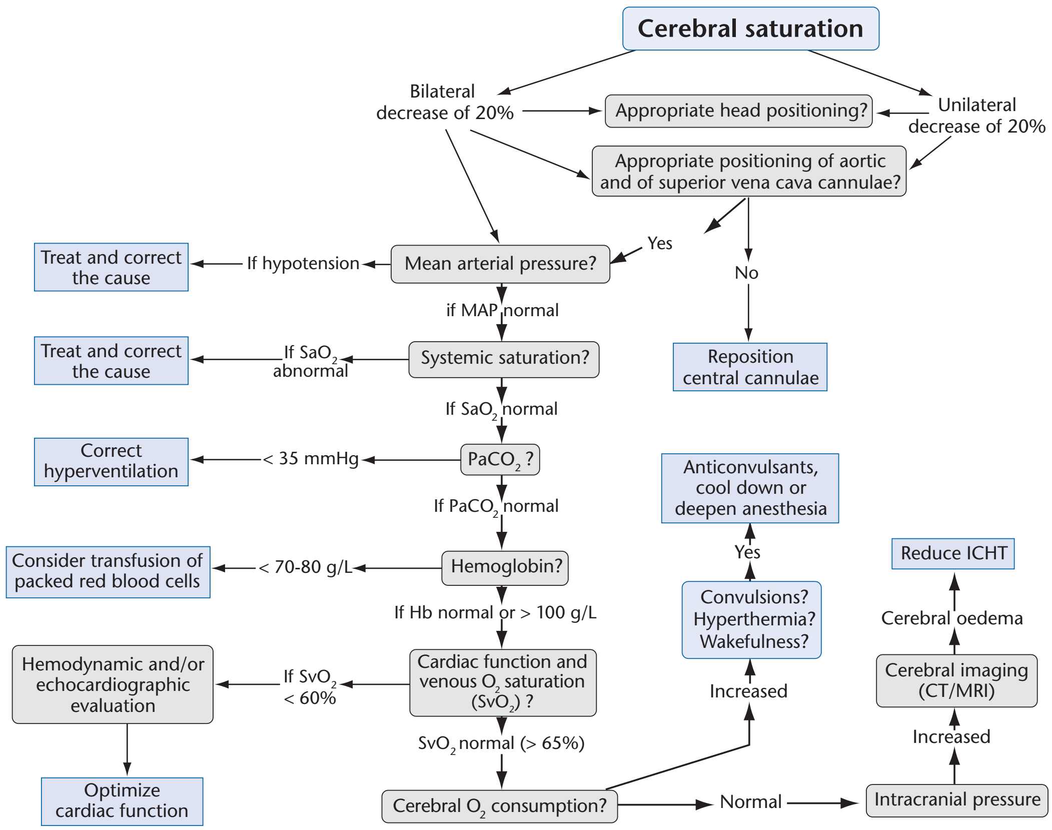
CONCLUSION

Is it possible that in the future, we will compare the advent of cerebral oximetry in our operating rooms to that of pulse oximetry and capnography? The answer to this question will be determined in the next few years. Both the pulse oximeter and the capnograph have had a significant impact on the practice of anesthesia and, interestingly, both are still subject to debate.[47] A new era has begun, and the understanding of mechanisms that lie behind cognitive neurological lesions will need to be refined and combined with the means to prevent their occurrence in cardiac and noncardiac surgery. Cerebral oximetry has the potential to become a key player in this development.

KEY POINTS TO RETAIN:

- Cerebral oximetry is based on the Beer-Lambert law, which stipulates that the concentration of a substance can be measured by the magnitude of light absorption.
- The normal cerebral oximetry value is approximately 67% ± 10% (3%-6% accuracy). The normal rSO_2 value is generally lower in female and elderly patients.
- Cerebral desaturation represents an imbalance between oxygen supply to the brain and oxygen demand.
- Decreased intraoperative cerebral saturation is associated with increased mortality and morbidity.
- Two randomized studies in cardiac and noncardiac surgery suggest that correction of cerebral desaturation reduces postoperative morbidity and length of hospital stay.

FIGURE 5: Algorithm for the use of cerebral oximetry



Hb: hemoglobin; ICHT: intracranial hypertension; MAP: mean arterial pressure; $PaCO_2$: carbon dioxide partial pressure; MRI: magnetic resonance imaging; SaO_2: oxygen saturation of arterial blood; SvO_2: oxygen saturation of venous blood; CT: Computed tomography. (Adapted from Denault et al[46]).

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References

1. Jobsis FF. Noninvasive, infrared monitoring of cerebral and myocardial oxygen sufficiency and circulatory parameters. *Science* 1977;198:1264-1267.
2. Taillefer MC, Denault AY. Cerebral near-infrared spectroscopy in adult heart surgery: systematic review of its clinical efficacy. *Can J Anaesth.* 2005;52:79-87.
3. Kurth CD, Steven JL, Montenegro LM, et al. Cerebral oxygen saturation before congenital heart surgery. *Ann Thorac Surg.* 2001;72:187-192.
4. Samra SK, Dy EA, Welch K, Dorje P, Zelenock GB, Stanley JC. Evaluation of a cerebral oximeter as a monitor of cerebral ischemia during carotid endarterectomy. *Anesthesiology.* 2000;93:964-970.
5. Vernieri F, Tibuzzi F, Pasqualetti P, et al. Transcranial Doppler and near-infrared spectroscopy can evaluate the hemodynamic effect of carotid artery occlusion. *Stroke.* 2004;35:64-70.
6. Madsen PL, Nielsen HB, Christiansen P. Well-being and cerebral oxygen saturation during acute heart failure in humans. *Clin Physiol.* 2000;20:58-64.
7. Nemoto EM, Yonas H, Kassam A. Clinical experience with cerebral oximetry in stroke and cardiac arrest. *Crit Care Med.* 2000;28:1052-1054.
8. Gracias VH, Guillamondegui OD, Stiefel MF, et al. Cerebral cortical oxygenation: a pilot study. *J Trauma.* 2004;56:469-472.
9. Dunham CM, Ransom KJ, Flowers LL, Siegal JD, Kohli CM. Cerebral hypoxia in severely brain-injured patients is associated with admission Glasgow Coma Scale score, computed tomographic severity, cerebral perfusion pressure, and survival. *J Trauma.* 2004;56:482-489.
10. Sokol DK, Markand ON, Daly EC, Luerssen TG, Malkoff MD. Near infrared spectroscopy (NIRS) distinguishes seizure types. *Seizure.* 2000;9:323-327.
11. Shojima M, Watanabe E, Mayanagi Y. Cerebral blood oxygenation after cerebrospinal fluid removal in hydrocephalus measured by near infrared spectroscopy. *Surg Neurol.* 2004;62:312-318.
12. Newman MF, Kirchner JL, Phillips-Bute B, et coll. Longitudinal assessment of neurocognitive function after coronary-artery bypass surgery. *N Engl J Med.* 2001;344:395-402.
13. McKenzie ED, Andropoulos DB, DiBardino D, Fraser CD Jr. Congenital heart surgery 2005: the brain: it's the heart of the matter. *Am J Surg.* 2005;190:289-294.
14. Hagino I, Anttila V, Zurakowski D, Duebener LF, Lidov HG, Jonas RA. Tissue oxygenation index is a useful monitor of histologic and neurologic outcome after cardiopulmonary bypass in piglets. *J Thorac Cardiovasc Surg.* 2005;130:384-392.
15. Yao FS, Tseng CC, Ho CY, Levin SK, Illner P. Cerebral oxygen desaturation is associated with early postoperative neuropsychological dysfunction in patients undergoing cardiac surgery. *J Cardiothorac Vasc Anesth.* 2004;18:552-558.
16. Fenton KN, Freeman K, Glogowski K, Fogg S, Duncan KF. The significance of baseline cerebral oxygen saturation in children undergoing congenital heart surgery. *Am J Surg.* 2005;190:260-263.

17. Dent CL, Spaeth JP, Jones BV, et al. Brain magnetic resonance imaging abnormalities after the Norwood procedure using regional cerebral perfusion. *J Thorac Cardiovasc Surg*. 2006;131:190-197.
18. Casati A, Fanelli G, Pietropaoli P, et al. Monitoring cerebral oxygen saturation in elderly patients undergoing general abdominal surgery: a prospective cohort study. *Eur J Anaesthesiol*. 2007;24:59-65.
19. Plachky J, Hofer S, Volkmann M, Martin E, Bardenheuer HJ, Weigand MA. Regional cerebral oxygen saturation is a sensitive marker of cerebral hypoperfusion during orthotopic liver transplantation. *Anesth Analg*. 2004;99:344-349.
20. Kim MB, Ward DS, Cartwright CR, Kolano J, Chlebowski S, Henson LC. Estimation of jugular venous O2 saturation from cerebral oximetry or arterial O_2 saturation during isocapnic hypoxia. *J Clin Monit Comput*. 2000;16:191-199.
21. Sehic A, Thomas MH. Cerebral oximetry during carotid endarterectomy: signal failure resulting from large frontal sinus defect. *J Cardiothorac Vasc Anesth*. 2000;14(4):444-446.
22. McCormick PW, Stewart M, Goetting MG, Dujovny M, Lewis G, Ausman JI. Noninvasive cerebral optical spectroscopy for monitoring cerebral oxygen delivery and hemodynamics. *Crit Care Med*. 1991;19:89-97.
23. Kurth CD, Thayer WS. A multiwavelength frequency-domain near-infrared cerebral oximeter. *Phys Med Biol*. 1999;44:727-740.
24. Yao FS, Yao D, Jin J. Age and gender differences in cerebral oxygen saturations. Paper presented at: the Annual Meeting of the American Society of Anesthesiologists; October 23-27, 2004; Las Vegas, Nevada. *Anesthesiology*. 2004;101:A193.
25. Kishi K, Kawaguchi M, Yoshitani K, Nagahata T, Furuya H. Influence of patient variables and sensor location on regional cerebral oxygen saturation measured by INVOS 4100 near-infrared spectrophotometers. *J Neurosurg Anesthesiol*. 2003;15:302-306.
26. Ausman JI, McCormick PW, Stewart M, et al. Cerebral oxygen metabolism during hypothermic circulatory arrest in humans. *J Neurosurg*. 1993;79:810-815.
27. Edmonds HL Jr. Advances in neuromonitoring for cardiothoracic and vascular surgery. *J Cardiothorac Vasc Anesth*. 2001;15:241-250.
28. Edmonds HL Jr, Cao L, Yu QJ. In the elderly, coronary artery bypass grafting associated with more brain O_2 desaturation and cognitive dysfunction. *Ann Thorac Surg*. 2002;73:S375 (Abstract).
29. Cho H, Nemoto EM, Yonas H, Balzer J, Sclabassi RJ. Cerebral monitoring by means of oximetry and somatosensory evoked potentials during carotid endarterectomy. *J Neurosurg*. 1998;89:533-538.
30. Casati A, Fanelli G, Pietropaoli P, et al. Continuous monitoring of cerebral oxygen saturation in elderly patients undergoing major abdominal surgery minimizes brain exposure to potential hypoxia. *Anesth Analg*. 2005;101:740-747.
31. Murkin JM, Adams SJ, Novick RJ, et coll. Monitoring brain oxygen saturation during coronary bypass surgery: a randomized, prospective study. *Anesth Analg*. 2007;104:51-58.
32. Austin EH III, Edmonds HL Jr, Auden SM, et al. Benefit of neurophysiologic monitoring for pediatric cardiac surgery. *J Thorac Cardiovasc Surg*. 1997;114(5):707-715, 717.
33. Higami T, Kozawa S, Asada T, et al. Retrograde cerebral perfusion versus selective cerebral perfusion as evaluated by cerebral oxygen saturation during aortic arch reconstruction. *Ann Thorac Surg*. 1999;67:1091-1096.
34. Goldman S, Sutter F, Ferdinand F, Trace C. Optimizing intraoperative cerebral oxygen delivery using noninvasive cerebral oximetry decreases the incidence of stroke for cardiac surgical patients. *Heart Surg Forum*. 2004;7(5):E376-E381.
35. Edmonds HL Jr. Pro: all cardiac surgical patients should have intraoperative cerebral oxygenation monitoring. *J Cardiothorac Vasc Anesth*. 2006;20:445-449.
36. Janelle GM, Mnookin S, Gravenstein N, Martin TD, Urdaneta F. Unilateral cerebral oxygen desaturation during emergent repair of a DeBakey type 1 aortic dissection: potential aversion of a major catastrophe. *Anesthesiology*. 2002;96:1263-1265.
37. Yeh T Jr, Austin EH III, Sehic A, Edmonds HL Jr. Rapid recognition and treatment of cerebral air embolism: the role of neuromonitoring. *J Thorac Cardiovasc Surg*. 2003;126:589-591.
38. Fukada J, Morishita K, Kawaharada N, et al. Isolated cerebral perfusion for intraoperative cerebral malperfusion in type A aortic dissection. *Ann Thorac Surg*. 2003;75:266-268.
39. Sakaguchi G, Komiya T, Tamura N, et al. Cerebral malperfusion in acute type A dissection: direct innominate artery cannulation. *J Thorac Cardiovasc Surg*. 2005;129:1190-1191.
40. Hoffman GM. Pro: near-infrared spectroscopy should be used for all cardiopulmonary bypass. *J Cardiothorac Vasc Anesth*. 2006;20:606-612.
41. Muehlschlegel S, Lobato EB. Con: all cardiac surgical patients should not have intraoperative cerebral oxygenation monitoring. *J Cardiothorac Vasc Anesth*. 2006;20:613-615.
42. Davies LK, Janelle GM. Con: all cardiac surgical patients should not have intraoperative cerebral oxygenation monitoring. *J Cardiothorac Vasc Anesth*. 2006;20:450-455.
43. Schwarz G, Litscher G, Kleinert R, Jobstmann R. Cerebral oximetry in dead subjects. *J Neurosurg Anesthesiol*. 1996;8:189-193.
44. Rivers E, Nguyen B, Havstad S, et al. Early goal-directed therapy in the treatment of severe sepsis and septic shock. *N Engl J Med*. 2001;345:1368-1377.
45. Piquette D, Deschamps A, Belisle S, et al. Effect of intravenous nitroglycerin on cerebral saturation in high-risk cardiac surgery: [L'effet de la nitroglycerine intraveineuse sur la saturation cerebrale dans les chirurgies cardiaques a haut risque]. *Can J Anaesth*. 2007;54:718-727.
46. Denault A, Deschamps A, Murkin JM. A proposed algorithm for the intraoperative use of cerebral near-infrared spectroscopy. *Semin Cardiothorac Vasc Anesth*. 2007;11:274-281.
47. Pedersen T, Dyrlund PB, Moller AM. Pulse oximetry for perioperative monitoring. *Cochrane Database Syst Rev*. 2003;CD002013
48. Denault AY, Couture P, Tardif JC, Buithieu J. *Transesophageal Echocardio-graphy Multimedia Manual: A Perioperative Transdisciplinary Approach*. New York, NY: Marcel Dekker; 2005.

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