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Cerebral oxymetry: Validation and impact on outcomes

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In a manner analogous to pulse oximetry, near-infrared spectroscopic (NIRS) light can be used to measure cerebral tissue oxygen saturation. All clinical NIRS devices detect changes in the concentrations of oxygenated and deoxygenated hemoglobin but are unable to distinguish between arterial and venous concentrations.

CORTICAL NEAR INFRARED SPECTROSCOPY

This technique employs principles of optical spectrophotometry which exploits the fact that biological material including skull, is relatively transparent in the near infrared range. Light transmission depends on a combination of reflectance, scattering and absorption effects. Reflectance is primarily a function of the angle of the light beam to the tissue surface, while scattering decreases with increasing wavelength, favouring transmission of shorter near-infrared (NIR) light (650-1,100 nm). Absorption occurs at specific wavelengths, determined by the molecular properties of the materials in the light path. Above 1,300 nm water absorbs all photons over a pathlength of a few millimeters, while below 700 nm, increasing light scattering and intense absorption bands of hemoglobin (Hb) prevent transmission. In the 700-1,300 nm range, however, NIR light penetrates tissue several centimeters[1]. The absorption spectra of oxyhemoglobin (HbO_2) ranges from 800-850 nm, deoxyhemoglobin ranges from 650-800 nm, and Caa3 has a broad peak at 820-840 nm[2].

In order to compensate for extracerebral tissue, most common techniques employ either spatial resolution or temporal resolution. Spatial resolution commonly involves differentially spaced receiving optodes with the signal from the closer receiver measuring more superficial tissue and distal optode measuring both superficial and deeper tissues and cortical oxygenation being derived from a subtraction algorithm. Temporal resolution involves the principle that photon path length is proportional to tissue transmission time such that by using a pulsed NIR signal, deeper (cortical) tissue will be reflective of receiver-detected photons arriving «later» rather than «earlier» in a pulsed sequence.

NIRS DEVICES

NIRS devices employ sequentially pulsed light-emitting diodes or direct laser light to emit NIR light transcutaneously and detect returning photons either by photodiodes or fibre optic transmission to a photo-multiplier and can be used to determine the oxygen saturation status of cerebral tissue There are currently two FDA-cleared cerebral oximeters INVOS 5100 (Somanetics Corporation, Troy, MI) and Foresight (CAS Medical Systems, Branford, CT). There appears to be some difference in approach between these devices. INVOS is a dual-channel continuous wave spatially resolved spectrometer which has been designed to measure change in regional oxygen saturation (rSO_2). Using a proprietary subtraction algorithm, this device uses light emitting diode (LED) at 730 nm and 810 nm and differentially spaced receiving optodes to assess bifrontal cortical oxygenation.

Alternatively, Foresight is designed to measure absolute brain tissue oxygen saturation. This oximeter utilizes continuous wavelengths at 690 nm, 780 nm, 805 nm and 850 nm to derive SO_2. To date there have been no direct comparisons between these two technologies. A third device, NIRO-300 (Hamamatsu Photonics KH, Hamamatsu City, Japan), which is currently for investigational use only, employs spatially resolved spectroscopy to measure light attenuation as a function of source-detector separation which is theoretically not influenced by photon path length and can thus potentially give a measure of absolute tissue oxygen saturation[4].

CLINICAL STUDIES

Previous studies have indicated a positive predictive value between low rSO_2 and adverse CNS outcomes[9]. The use of cerebral oximetry identifies a number of otherwise unrecognized causes of cerebral hypoperfusion both during conventional CPB[10], and during beating heart surgery[11]. Various causes of cerebral hypoperfusion including inadvertent positioning of the head turned to extreme left side, cannula-obstructed venous outflow from brain, hypocapnia, low perfusion pressure, inadequate hemoglobin concentration, have all been detected and successfully treated by applied rSO_2 oximetry[12,13]. During beating heart procedures compromised cerebral perfusion can occur relatively frequently with an incidence nearly twice that occurring during CPB, as demonstrated using jugular oximetry in a randomized clinical study of 187 patients[14]. In a series of 550 beating heart patients combined EEG and cerebral oximetry identified episodes of cerebral ischemia in 15% of patients, which were treated by a combination of pharmacologically-improved cardiac output, increased perfusion pressure and cardiac repositioning[15].

The use of rSO_2 has demonstrated correlation between CAB patients having low rSO_2 values and cognitive dysfunction[16], prolonged hospital stay[17], and most recently, perioperative cerebrovascular accident (CVA)[18]. Dunham et al. showed that rSO_2 values correlated with cerebral perfusion pressure, Glasgow Outcome Score and mortality in patients with traumatic brain injuries[19], and several other groups have demonstrated the ability of rSO_2 to provide an early warning of cerebral ischemia[20-22].

In a large non-randomized series of 1,034 cardiac surgical patients reported by Goldman and colleagues, a significant reduction in perioperative stroke rate, from 2.01% to 0.97%, was observed in patients in whom rSO_2 cerebral oximetry was used to optimize and maintain intraoperative cerebral oxygenation in comparison to an untreated comparator group of 1,245 similar patients operated on in the immediately preceding 18 month interval[18].

In a prospective, randomized blinded study by Murkin et al in 200 patients undergoing coronary artery grafting, it was demonstrated that treatment of declining rSO_2 prevented prolonged desaturations and was associated with a shorter ICU stay and a significantly reduced incidence of major organ morbidity or mortality. The intervention protocol undertaken to return rSO_2 to baseline resulted in a rapid improvement in rSO_2 in most cases and did not add undue risk to the patient[23]. There were also numerically fewer clinical CVA in monitored patients directionally consistent with previous studies[18].

REFERENCES

1. McCormick PW, Stewart M, Goetting MG, et al. Noninvasive cerebral optical spectroscopy for monitoring cerebral oxygen delivery and hemodynamics. Crit Care Med 1991;19:89 97.
2. Josbis FF. Noninvasive, infrared monitoring of cerebral and myocardial oxygen sufficiency and circulatory parameters. Science 1977;198:1264 7.
3. Kurth CD, Thayer WS. A multiwavelength frequency-domain near-infrared cerebral oximeter. Phys Med Biol 1999;44:727-40.
4. Yoshitani K, Kawaguchi M, Miura N, Okuno T, Kanoda T, Ohnishi Y, Kuro M. Effects of hemoglobin concentration, skull thickness, and the area of the cerebrospinal fluid layer on near-infrared spectroscopy measurements. Anesthesiology 2007;106:458-462.
5. Gagnon RE, Macnab AJ, Gagnon FA, Blackstock D, LeBlanc JG. Comparison of two spatially resolved NIRS oxygenation indices. J Clin Monit Comput 2002;17:385-91.
6. Dunham C, Sosnowski C, Porter J. Correlation of noninvasive cerebral oximetry with cerebral perfusion in the severe head injured patients: a pilot study. J Trauma 2002;52:40-46.
7. Schwartz G, Litscher G, Kleinert R. Cerebral oximetry in dead subjects. J Neurosurg Anesthesiol 1996;8:189-193.
8. Maeda H, Fukita K, Oritani S. Evaluation of *post-mortem* oximetry with references to the causes of death. Forensic Sci Internat 1997;87:201-210.
9. Alexander HC, Kronenefeld MA, Dance GR. Reduced postoperative length of stay may result from using cerebral oximetry monitoring to guide treatment. (abstract) Ann Thorac Surg 2002;73:373-C.
10. Madsen PL, Nielsen HB, Christiansen P. Well-being and cerebral oxygen saturation during acute heart failure in humans. Clin Physiol 2000;20:158-64.
11. Austin EH 3rd, Edmonds HL Jr, Auden SM, Seremet V, Niznik G, Sehic A, Sowell MK, Cheppo CD, Corlett KM. Benefit of neurophysiologic monitoring for pediatric cardiac surgery. J Thorac Cardiovasc Surg 1997;114:707-15.
12. Nielsen H, Boushel R, Madsen P, Secher H. Cerebral desaturations during exercise reversed by O_2 supplementation. Am J Physiol 277 (Heart Circ. Physiol. 46): H1045-H1052, 1999.
13. Amosu O, Bhavani-Shankar K. Cerebral oxygenation during cesarean section. (abstract) Anesthesiology 2000;92:A85.
14. Diephuis JC, Moons KG, Nierich AN, Bruens M, van Dijk D, Kalkman CJ. Jugular bulb desaturation during coronary artery surgery: a comparison of off-pump and on-pump procedures. Br J Anaesth 2005;94:715-20.
15. Berlac PA, Rasmussen YH. Per-operative cerebral near-infrared spectroscopy (NIRS) predicts maternal hypotension during elective caesarean delivery in spinal anaesthesia. Int J Obstet Anesth 2005;14:26-31.
16. Yao FF, Tseng CA, Ho CA, Levin SK, Illner P. Cerebral oxygen desaturations is associated with early postoperative neuropsychological dysfunction in patients undergoing cardiac surgery. J Cardiothorac Vasc Anesth 2004;18:552-58.
17. Edmonds HL Jr. Multi-modality neurophysiologic monitoring for cardiac surgery. Heart Surg Forum 2002;5:225-28.
18. 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:E376-81.
19. Dunham CM, Ransom KJ, Flowers LL, Siegal JD, Kohli CM. Cerebral hypoxia in severely brain-injured patients is associated with admission Glasgow Coma Score, computed tomographic

severity, cerebral perfusion pressure, and survival. J Trauma 2004;56:482-91.
20. Papadimos TJ, Marco AP. Cerebral oximetry and an unanticipated circulatory arrest (letter to the editor). Anaesthesia 2004;59:309-10.
21. Fukada J, Morishita K, Kawaharada N, Yamauchi A, Hasegawa T, Satsu T, Abe T. Isolated cerebral perfusion for interoperative cerebral malperfusion in type A aortic dissection. Ann Thor Surg 2003;75:266-68.
22. Janelle GM, Mnookin S, Gravenstein N, Martin TD, Urdaneta F. Unilateral cerebral oxygen desaturations during emergent repair of DeBakey type 1 aortic dissection: potential aversion of a major catastrophe. Anesthesiology 2002;96:1263-5.
23. Murkin JM. Adams SJ, Novick RJ, Iglesias I, Bainbridge D, Schaefer B, Irwin B, Fox S. Monitoring brain oxygen saturation during coronary bypass surgery: A randomized, prospective study. Anesth Analg 2007;104:51-8.