

Published in final edited form as:

Best Pract Res Clin Anaesthesiol. 2014 December ; 28(4): 429–439. doi:10.1016/j.bpa.2014.09.002.

Cerebral and Tissue Oximetry

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Abstract

The use of near-infrared spectroscopy (NIRS) has been increasingly adopted in cardiac surgery to measure regional cerebral oxygen saturation. This method takes advantage of the fact that light in the near-infrared spectrum penetrates tissue, including bone and muscle. Sensors are placed at fixed distances from a light emitter, and algorithms subtract superficial light absorption from deep absorption to provide an index of tissue oxygenation. Although the popularity of NIRS monitoring is growing, definitive data that prove outcome benefits with its use remain sparse. Therefore, widespread, routine use of NIRS as a standard-of-care monitor cannot be recommended at present. Recent investigations have focused on the use of NIRS in subgroups that may benefit from NIRS monitoring, such as pediatric patients. Furthermore, a novel application of processed NIRS information for monitoring cerebral autoregulation and tissue oxygenation (e.g., kidneys and the gut) is promising.

Keywords

Near-infrared spectroscopy; NIRS; cerebral autoregulation; cardiac surgery; anesthesia; monitor

Background

The principal discoveries that underlie the monitoring of regional cerebral oximetry (rScO₂) include those by Jöbsis in the late 1970s and the theoretical framework by Norris before [1, 2]. He demonstrated that light in the near-infrared spectrum penetrates tissue, including bone and muscle, and that certain chromophores (e.g., cytochrome oxidase C) absorb light in this spectrum. In 1985, Ferrari and colleagues demonstrated that light emitted at the wavelength specific for the peak absorption of the chromophores oxyhemoglobin (920 nm) and total hemoglobin (760 nm) can be used to measure brain oxygenation in humans (Figure 1A) [3]. Cerebral oximetry has since gained widespread popularity, especially in adult and pediatric

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Conflict of interest

Jochen Steppan: none

cardiac surgery [4, 5]. Today, physicians can choose from a wide variety of devices for monitoring rScO₂, including INVOS™ (Somanetics/Covidien, Inc., Boulder, CO), FORESIGHT™ (CAS Medical Systems, Branford, CN), EQUANOX™ (Nonin Medical Inc., Plymouth, MN), CerOx™ (Ornim Medical, Lod, Israel), NIRO™ (Hamamatsu Photonics, Hamamatsu City, Japan), and TOS-96™ (Tostec, Tokyo, Japan). Now, these methods are also being used to monitor oxygenation in other tissue beds, such as extremities distal to the site of arterial cannulation during intra-aortic balloon counter-pulsation or cardiopulmonary bypass (CPB). Newer applications of cerebral oximetry include its use as a surrogate for cerebral blood flow (CBF) for bedside monitoring of autoregulation [6] and regional tissue perfusion [7]. Monitoring rScO₂ and tissue oxygenation may provide important clinical data on the balance between tissue oxygen supply and demand in near real-time, allowing for therapeutic interventions to prevent tissue ischemic injury [8]. However, despite a multitude of anecdotal reports, recent reviews have tempered the enthusiasm for routine use of rScO₂ by questioning whether it leads to improved patient outcomes [8, 9]. This review will describe the basic principle of rScO₂ and tissue oximetry; the general considerations and limitations for its use; its clinical applications in cardiac, carotid artery, and pediatric surgery; and emerging applications.

Basic Principle

An understanding of the physical principles of cerebral and tissue oximetry is important for the clinician to interpret oximetry results and appreciate its limitations. Several comprehensive reviews of this technology are available [8, 10, 11]. Oximetry is based on the transmission of light in the near-infrared spectrum across tissue and its absorption by chromophores (Figure 1A). For cerebral oximetry, self-adhesive emitter / sensor pads applied to the skin of the forehead measure light attenuation noninvasively at a set distance from a NIRS-emitting light source. The absorption of the emitted light is directly proportional to chromophore concentration, the absorption coefficient of the chromophore, and the distance that the light travels between the emitting source and the detector, as stated in the Beer-Lambert law [12]. A modification of the Beer-Lambert law can be expressed as follows:

$$[X] = \frac{\Delta A}{L \times \epsilon}$$

where [X] is the chromophore concentration, ΔA is light attenuation, L is the length that the light traveled, and ϵ is the chromophore extinction coefficient. Chromophore concentration can thereby be calculated by measuring light attenuation between the detector and the source distance and using a previously determined extinction coefficient [13]. An approach to determine rScO₂ utilizes the distinct absorption spectra of oxyhemoglobin and deoxyhemoglobin, which are maximally separated at 700 and 850 nm (Figure 1B). At a minimum, two wavelengths of light are needed to measure the relative concentration of two chromophores. The concentration of oxyhemoglobin can be measured as the ratio of light absorbed for oxyhemoglobin compared with light absorbed for total hemoglobin. However, the distance between the emitting light source and the detector cannot be measured directly, given the refraction, reflection, and scattering of the photons in biological tissue. Therefore,

only relative changes in oxygen saturation can be measured [14]. To overcome this limitation, device manufacturers use 1) computer modeling and simulation to estimate the distance traveled by the emitted light; 2) multi-distance spectroscopy [15]; 3) time-resolved spectroscopy with a laser light [16]; or 4) multiple near-infrared wavelengths with frequency domain analysis to determine a tissue absorption coefficient [17]. The latter two techniques obviate the need to know the actual distance traveled by light [18]. Given the many clinical devices available, manufacturers use different proprietary algorithms that are optimized to the specific device, as previously reviewed [19]. Importantly, one should keep in mind that because of the number and variability of these algorithms, oxygen saturation values can differ when measured by devices from different manufacturers [20]. These devices also use different algorithms to subtract NIRS absorption in superficial tissue (1 cm) from that in deeper tissue (2 cm) to yield oxygen saturation in the superficial frontal cortex [21].

General Considerations and Limitations

Unlike pulse oximetry, NIRS-based cerebral and tissue oximeters do not include plethysmography and, thus, do not differentiate between arterial and venous blood. Therefore cerebral and tissue oximeters do not provide an indicator of oxygen delivery but rather provide information on balance between regional oxygen supply and demand [22]. The absence of plethysmography has advantages insofar as measurements are provided even in the absence of a pulse, such as during CPB. However, a limitation with this technology is the possibility that one could erroneously measure the oxygenation state of tissue unrelated to the targeted organ perfusion (e.g., extra-cerebral tissue when measuring rScO₂). This potential error can be overcome by using the spatial resolution of the system (photon penetration is about one third the distance between receiver and transmitter). By adding detectors at multiple distances from the emitted light source, specific algorithms can subtract the absorption signal of superficial tissue (detected with sensors closest to the light transmitter) from the absorption signal received from deeper tissues (the more distant receivers). Moreover, because of variability in the exact composition of arteries, veins, capillaries, and nonvascular tissue penetrated [23], baseline values vary between subjects by approximately 10% [20], making it more appropriate to use cerebral and tissue oximetry as a trend monitor rather than as an absolute index of tissue oxygenation. These devices usually assume a relative and fixed amount of arterial versus venous blood (e.g. 30:70) to calculate oxygen saturation. However the relative concentrations of arterial versus venous blood in the area measured can vary significantly between and within subjects, especially with changes in partial pressure of carbon dioxide [24]. Acute changes in the distribution of hemoglobin between the arterial and venous system (e.g., opening of arterio-venous shunts, hematoma formation, and hemodilution) or changes in the distance between the sensor and light source (e.g., tissue edema) all influence oximetry readings without necessarily affecting tissue oxygenation [25–27]. Other external sources of error or signal attenuation include motion artifact [28], non-hemoglobin chromophores, like melanin pigmentation in hair [29], and bilirubin in patients with jaundice [30]. Skin color and melanin content do not appear to negatively affect cerebral and tissue oximetry, as they are confined to the superficial part of the skin.

Clinical Applications

Mortality after major cardiac surgery remains low despite the rising number of aged and high-risk patients. Nevertheless, the frequency of neurologic complications remains relatively unchanged over the past decade [31]. Clinicians have long sought a reliable way to monitor the adequacy of brain perfusion during CPB, particularly considering the importance of neurologic complications, such as stroke, delirium, and cognitive impairment, for patient outcome. Proposed monitors for this purpose include quantitative electroencephalography (EEG), transcranial Doppler, and carotid Doppler, none of which have been adopted widely, mostly owing to associated technical limitations [32]. Despite its limitations, there has been wide enthusiasm for rScO₂ monitoring during cardiac surgery. It could offer a potentially large clinical benefit by alerting clinicians to a decrease in cerebral oxygen supply early enough to implement corrective interventions. Although some studies have shown that low rScO₂ values (either absolute or relative) are associated with poorer patient outcome (early postoperative cognitive decline) or longer length of hospitalization [33], available data are insufficient to convincingly establish that preventing or promptly correcting low rScO₂ values improves outcome [34, 35]. Given the relatively high cost of the monitoring sensors (~ \$200 per patient) and the contradictory data on clear patient benefit, most authors agree that the routine use of cerebral or tissue oximetry as standard of care for *all* patients cannot be recommended at this time [8, 10, 36]. However, one often hears the counterargument that similar objections could be made for other standard-of-care monitors. For example, a recent Cochrane database review “found no evidence that pulse oximetry affects the outcome of anesthesia for patients” [37].

Cardiac Surgery

Many case reports and case series suggest that monitoring rScO₂ may provide early warning of CPB cannula malposition or rare, but catastrophic, oxygen delivery failure from the bypass circuit [38–40]. Several prospective observational studies found a link between decrements in rScO₂ from baseline and subtle neurologic changes, including postoperative delirium and cognitive dysfunction [22, 41–53]. These data, though, are not consistent, and the studies have had limitations, including small patient sample size, the use of a limited psychometric battery, and testing only early after surgery when many confounding factors can affect cognition. Several observational studies have found links between decrements in rScO₂ from baseline and postoperative stroke [22, 41, 54–56]. Goldman et al. [22] compared the rates of stroke in more than 1,000 patients who had rScO₂ monitoring during coronary artery bypass graft / valve surgery with stroke rates in a historical group. They found that the stroke rate was significantly higher in the group without cerebral monitoring (2.5% vs. 0.97%). However, the study used no defined protocol for managing patients with rScO₂ desaturation and made no statistical adjustment for potential confounding factors, such as with multivariate logistic regression analysis. This limitation is important, as the stroke rate in the monitored patients was lower than that in most series, raising the question of whether the historical group and the rScO₂-monitored group were at similar risk for stroke.

A larger question than any link between rScO₂ desaturation and patient outcome is whether corrective interventions can effectively reverse the desaturation and whether these

interventions lead to improved patient outcome. Murkin et al. [57] randomized 200 patients undergoing cardiac surgery to an intervention group with a specific protocol for managing rScO₂ desaturation (<75% of baseline value) and a control group who had rScO₂ blindly monitored. The algorithm for treating rScO₂ decrements included measures such as raising blood pressure, ensuring adequate CPB flow, normalizing PaCO₂, deepening anesthesia depth, or even instituting pulsatile CPB flow. These investigators found that control patients had longer desaturation times than patients in the intervention group ($p = 0.014$) and a longer duration of hospitalization in the intensive care unit ($p = 0.029$). Although major complications did not differ between the groups, the control patients were more likely to suffer major morbidity and mortality (death, ventilation >48 h, stroke, myocardial infarction, return for re-exploration) than were patients in the intervention group ($p = 0.048$). This study was not powered to compare the rates of stroke between the intervention and the control groups.

Based on these studies, even in this high-risk population, only low-level evidence links rScO₂ decrements from baseline to poor neurologic outcomes. Moreover, the available data are insufficient to conclude that interventions for correcting rScO₂ decrements lead to a lower risk for stroke, delirium, or postoperative cognitive dysfunction [9].

Pediatric Cardiac Surgery

Many groups have advocated for monitoring rScO₂ in pediatric patients undergoing cardiac surgery for congenital heart defects [68–71]. A retrospective analysis of 50 patients with hypoplastic left heart syndrome undergoing the Norwood procedure revealed that a mean rScO₂ < 56% for the first 48 postoperative hours has a sensitivity of 75% and a specificity of 79% for predicting adverse outcomes, such as death, prolonged intensive care unit stay, and need for extracorporeal membrane oxygenation [68]. Another small study in 25 pediatric surgery patients showed a positive correlation between rScO₂ and mixed venous oxygen saturation [70], whereas Bhutta and colleagues reported a correlation between cerebral oximetry and superior vena cava oxygen saturation in pediatric patients after heart transplant [71]. Moreover, intraoperative rScO₂ has been shown to be a sensitive indicator for the adequacy of cerebral perfusion in pediatric surgical patients who weigh less than 10 kg, as mixed venous oxygen saturation in those patients is more representative of lower body oxygenation [72]. Finally, one of the largest studies in pediatric cardiac surgery patients (n=250) evaluated different modalities of cerebral monitoring. Postoperative neurologic events occurred in 26% of patients who had a monitoring event that was not corrected intraoperatively but in only 6% of patients who received intraoperative treatment [73]. A recent systematic review of the role of rScO₂ in pediatric patients undergoing surgery for congenital heart disease identified 54 manuscripts (47 case series, 4 randomized trials, and 3 retrospective studies) and 13 review articles [74]. Taken together, the evidence failed to show that rScO₂ monitoring leads to a clinical improvement in short-term neurologic outcome in this patient population [74].

Carotid Artery Surgery

Carotid endarterectomy (CEA) surgery reduces the risk of stroke for patients with symptomatic carotid stenosis and subgroups of patients with asymptomatic stenosis [58–60]. These benefits are only realized if the rate of perioperative neurologic complications, including stroke, can be minimized. The risk for stroke after CEA depends on many factors, including the acuity and severity of symptoms and the individual center experience [61–63]. Ensuring cerebral perfusion to vascular territories ipsilateral to the side of carotid artery cross-clamping during CEA is paramount to reduce the risk of perioperative ischemic neurologic complications.

Clinicians use various strategies to monitor the adequacy of cerebral perfusion during carotid clamping, including stump pressure measurement (e.g., the blood pressure in the carotid artery distal to the cross clamp), transcranial Doppler, somatosensory evoked potentials (SSEPs), EEG, or use of regional anesthesia to allow assessment of the patient's mental status during carotid cross-clamping [64, 65]. Several investigations have examined the utility of monitoring rScO₂ during CEA surgery. One advantage of cerebral oximetry is that it is simple to use, particularly compared with multi-channel EEG and transcranial Doppler monitoring [64]. It has been reported that a 5% to 15% decrement in rScO₂ from baseline on the side ipsilateral to carotid cross-clamping has a 44% to 100% sensitivity and 44% to 100% specificity for predicting changes in CBF velocity as measured by transcranial Doppler, EEG slowing, or changes in SSEPs. [51–55] These studies have limitations such as small sample size. One of the largest studies to date, which involved almost 600 patients who underwent CEA under general anesthesia, found that a 11.7% decrease from baseline in rScO₂ had a sensitivity of 75% and a specificity of 77% for detecting neurological complications [66]. Others have reported that decrements in rScO₂ 20% from baseline were associated with neurologic symptoms during carotid artery cross-clamping in patients undergoing CEA with regional anesthesia. [57] The sensitivity of changes in rScO₂ compared with neurologic symptoms in awake patients was 80%, with a specificity of 82%.

Clinicians must consider that lower specificity, or false positive results, for detecting cerebral ischemia with rScO₂ monitoring may lead to unnecessary treatment—including shunt placement, which by itself carries a risk of embolic stroke [67]. False negative results may also have considerable consequences. In one study, rScO₂ was unchanged in 7 of 323 patients during carotid artery cross-clamping for CEA, despite evidence of cerebral ischemia based on EEG or SSEP monitoring. [56] In that study, the authors observed a discrepancy between rScO₂ findings and EEG and SSEP results in 24 (7.4%) patients. Thus, the current evidence suggests that rScO₂ may not have high clinical reliability as a sole monitor of the adequacy of cerebral perfusion during CEA surgery.

Emerging Applications

Cerebral Autoregulation

The main determinants of rScO₂ include oxygen content of the blood, CBF, tissue diffusivity of oxygen, and cerebral metabolic rate for oxygen. Because factors that affect blood oxygen content and cerebral metabolic rate are relatively stable over short periods of

time, members of our group have proposed that rScO₂ might serve as a clinically useful surrogate for CBF during bedside monitoring of cerebral autoregulation. Using rScO₂ signals from a standard NIRS monitor, specialized computer software calculates the continuous correlation between low-frequency (20 sec to 3 min) changes in rScO₂ and mean arterial blood pressure to render the variable cerebral oximetry index (COx) [6, 75, 76]. This approach is based on the rationale that CBF in the autoregulated range is constant regardless of blood pressure. Thus, when autoregulation is functional, the metrics of CBF and blood pressure do not correlate, and COx approaches zero or is negative. Conversely, when blood pressure is below or above the limits of autoregulation, the metrics of CBF and blood pressure do correlate, and COx approaches 1. An example of COx monitoring in a patient undergoing cardiac surgery with cardiopulmonary bypass is shown in Figure 2. Our approach was validated in a laboratory piglet model by comparing COx monitoring to laser Doppler flux measurement of CBF [77]. We have further validated COx monitoring by comparing it to transcranial Doppler monitoring in adult patients undergoing CPB [78]. Monitoring of COx has many advantages over other methods of autoregulation monitoring, as it is noninvasive, provides a continuous measurement that is not subject to electrical or motion artifact as transcranial Doppler monitoring is, and requires little caregiver intervention. We have validated a “plug-and-play” custom NIRS monitor that holds promise for providing clinicians with a feasible method of COx monitoring [79].

In our research thus far, we have made several clinically important observations, primarily in patients undergoing cardiac surgery. First, we have found that the mean arterial pressure at the lower limit of autoregulation varies widely between individuals, ranging from 40 to 90 mmHg [6, 80–82]. Consequently, when standard empiric methods are used to set blood pressure limits during cardiopulmonary bypass (or to define the tolerance to low blood pressure), some patients will have periods during surgery when their blood pressure is below the limits of autoregulation. These excursions of blood pressure below the lower limit of autoregulation were found to be associated with the risk for acute kidney injury after cardiac surgery and major organ morbidity and mortality [83, 84]. Monitoring COx might provide a means to individualize blood pressure targets during surgery and modify risk for adverse patient outcomes.

Tissue Perfusion

Although most clinical and research applications of NIRS monitoring focus on rScO₂ monitoring, the principle of light penetration into tissues and the absorption by chromophores is applicable to multiple regions of the body [85]. The muscle underlying the thenar eminence has been identified as a relatively homogeneous tissue compartment. Because it has a reasonably small overlying fat layer, only minor inter-individual variations, little tendency to develop edema, and little pigmentation, it can be easily accessed for NIRS [85]. Studies have shown that the distal extremities exhibit an early vasoconstrictor response in models of hypovolemia [86, 87]. Therefore, monitoring of thenar tissue oxygenation may be beneficial for detecting early signs of hypovolemia in patients who are still hemodynamically compensated and provide a guide to therapy before changes occur in traditional markers of volume status [88]. A minimum tissue oxygen value, as measured by NIRS, precedes peak lactate levels by more than 90 min in patients with hypovolemic shock,

indicating that regional tissue perfusion might be an earlier indicator of perfusion deficits [89]. Other potential sites that NIRS monitoring might be applied to (especially in the pediatric population) include relatively superficial organs like the kidney or gut [90–92]. In a study of 23 pediatric cardiac surgery patients, cerebral, splanchnic, renal, and muscle oximetry correlated with lactate levels [92]. Emerging data also support the possibility of using tissue oximetry to measure renovascular reactivity during hemorrhagic shock [93, 94]. Lastly, tissue oximetry could be used as an early marker to detect intraoperative tissue hypoxia during abdominal surgery that involves the intestines, pancreas, or kidneys [95, 96]. The major drawback to evaluating oxygenation of superficial organs with NIRS methodology is the large inter-individual variability in distance between the body surface and the organ of interest, secondary to overlying fat and muscle. The latter contains myoglobin, a chromophore that makes it difficult to distinguish light absorption arising from hemoglobin. Nevertheless some clinical data (mainly limited to small studies and case reports) suggest that tissue oximetry has value, especially in the pediatric population [97].

Acknowledgments

Funding Source:

Supported in part by a grant to Dr. Hogue from the National Institutes of Health (R01 HL092259-1).

Charles W Hogue, Jr: Research funding from Covidien, Inc (Boulder, CO), Advisory Board membership for Ornim (Israel), Data and Safety Monitoring Board, CSL Behring (King of Prussia, PA).

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Practice Points

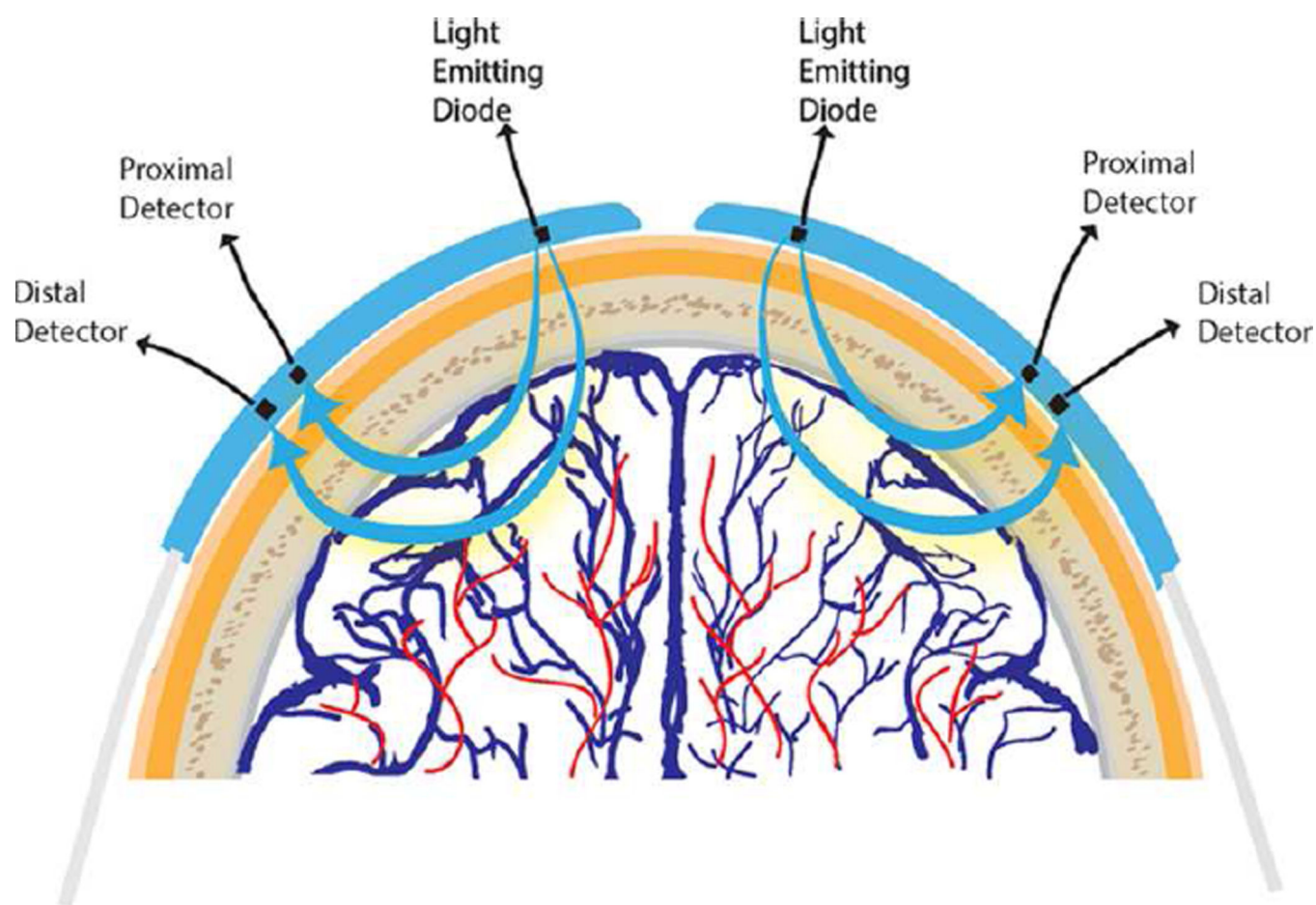
- Cerebral and tissue oximetry are simple and attractive ways to measure regional oxygenation
- Data are insufficient to recommend cerebral and tissue oximetry as standard-of-care monitoring for *all* patients undergoing anesthesia
- Patients undergoing cardiac surgery and carotid endarterectomy are more likely than other patients to benefit from cerebral oximetry
- Emerging data suggest that tissue oximetry provides complementary information for patients in hemorrhagic shock and for pediatric patients with impaired intestinal or renal perfusion
- By serving as a surrogate for CBF, cerebral oximetry may offer a clinically feasible method to monitor cerebral autoregulation

Research Agenda

- Define patient populations that will most likely benefit from cerebral and tissue oximetry
- Evaluate the risks and benefits of interventions to maintain blood pressure within an individual's autoregulation range during surgery and in the intensive care unit
- Validate tissue and organ NIRS monitoring

Summary

Cerebral or tissue oximetry is a simple and attractive way to measure regional oxygenation. This method takes advantage of the fact that light in the near-infrared spectrum penetrates tissue, including bone and muscle. When sensors are placed at fixed distances from a light emitter, computer algorithms can subtract superficial light absorption from deep absorption to provide an index of tissue oxygenation. Despite the growing popularity of this monitoring technique, data regarding outcome benefits remain sparse and contradictory. Even in high-risk patients undergoing cardiac surgery, only low-level evidence has shown a correlation between detection of low cerebral oxygen saturations with NIRS and poor neurologic outcomes. Available data are insufficient to conclude that interventions guided by cerebral oximetry improve neurologic outcome by reducing stroke rate and postoperative cognitive dysfunction. Therefore most authors agree that the routine use of cerebral or tissue oximetry as standard of care for *all* patients cannot be recommended at this time. Novel areas of application for NIRS include cerebral autoregulation and tissue oxygenation (e.g., kidneys and the gut). Additional studies are needed to evaluate the risks and benefits of using interventions to maintain blood pressure within an individual's autoregulation range and to maintain peripheral tissue and end organ oxygenation.



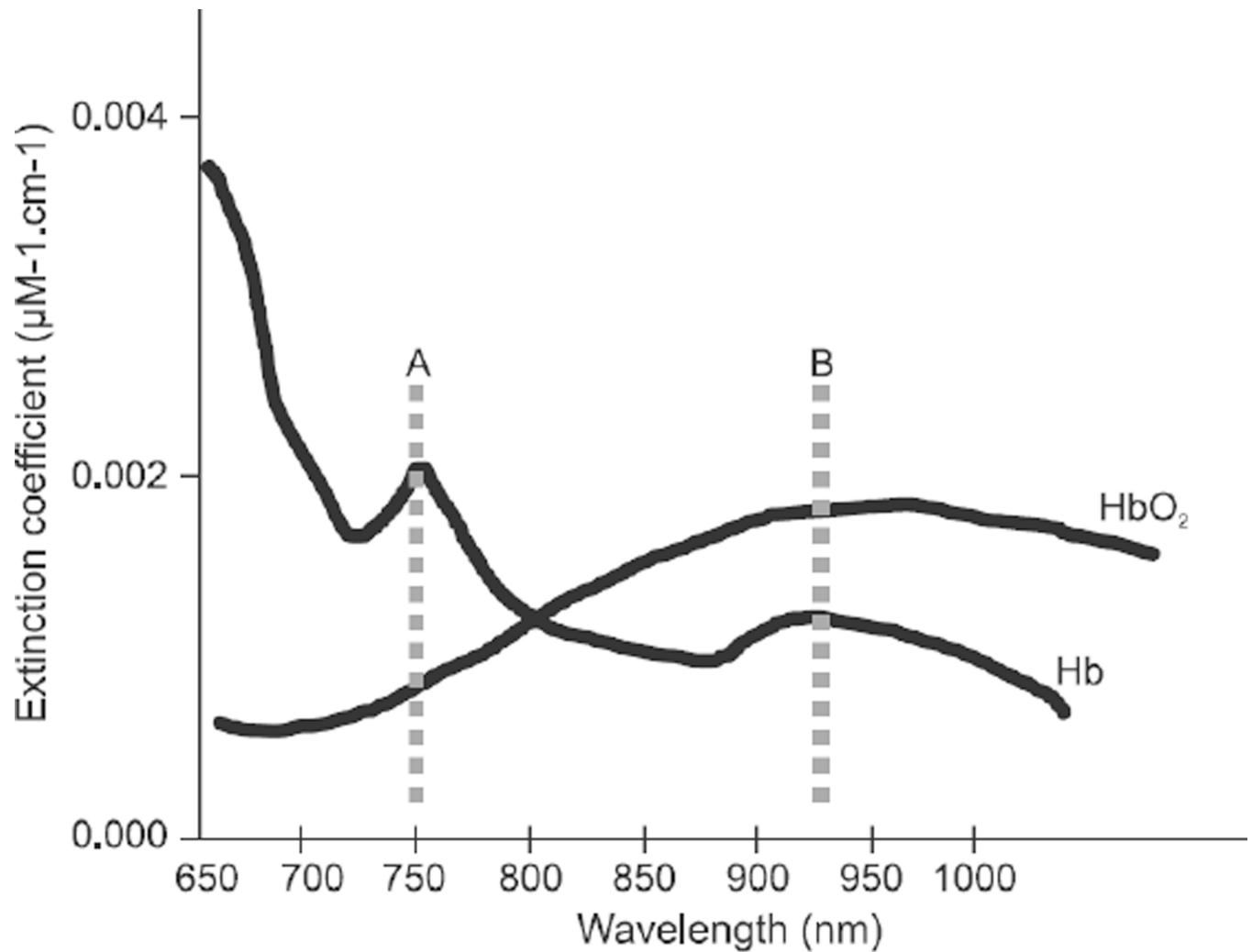


Fig. 1.

Cerebral oximetry. A: Schematic illustration of cerebral oximetry. Used with permission from *Anesth Analg*. 2013 Mar;116(3):663-76 B: Absorption spectrum of hemoglobin and oxyhemoglobin with the peak absorption wavelength of oxyhemoglobin (B: 920 nm) and total hemoglobin (A: 760 nm). Hb, deoxyhemoglobin; HbO₂, oxyhemoglobin. Used with permission from Lima et al., *Rev Bras Ter Intensiva*. 2011 August 23(3):341–351.

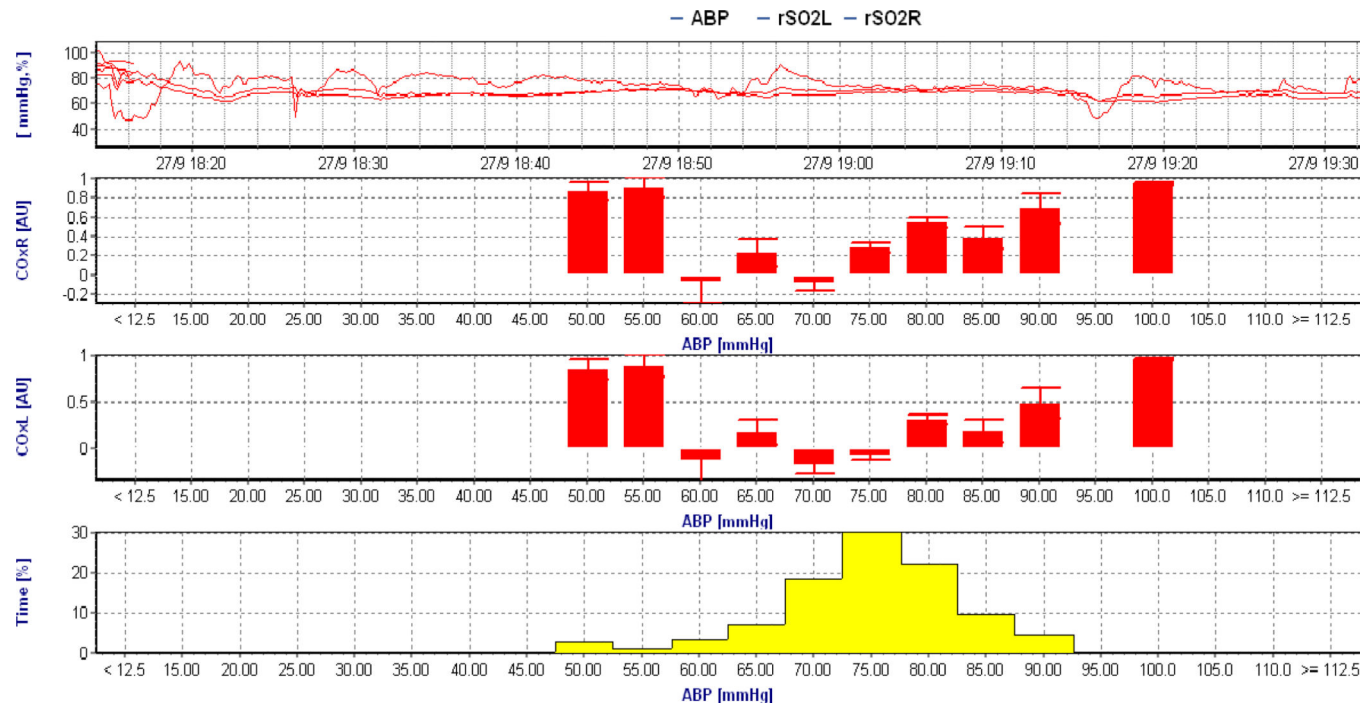


Fig. 2.

Cerebral oximetry index (COx) recording from a patient undergoing cardiopulmonary bypass. The top graph is the time series for mean arterial pressure (ABP) and the raw left and right regional cerebral oxygen saturation (rSO₂). The bottom graph is the percent of time the patient spent with a blood pressure at each 5 mmHg bin. The right and left COx data in the middle two graphs represent the correlation between regional cerebral oxygen saturation and mean arterial pressure. When mean blood pressure is in the autoregulation range, there is no correlation between regional cerebral oxygen saturation and blood pressure, represented by COx near zero. When blood pressure is outside the limits of autoregulation, regional cerebral oxygen saturation and blood pressure are correlated or cerebral blood flow is pressure passive. In this example, a mean blood pressure > 60 mmHg would ensure that blood pressure is above the lower limit of autoregulation. An upper limit of autoregulation is also demonstrated at a mean blood pressure of approximately 90 mmHg.