Pediatric Anesthesia and Critical Care Journal 2016;4(1):18-23
doi:10.14587/paccj.2016.4

PACCJ

Additional applications of intraoperative cerebral oxygenation monitoring using near infrared spectroscopy

E. Dewhirst[1], M. Hakim[2], J. D. Tobias[2,3]

[1]Department of Anesthesiology & Pain Medicine, The Ohio State University College of Medicine, Columbus, Ohio, USA
[2]Department of Anesthesiology & Pain Medicine, Nationwide Children's Hospital, Columbus, Ohio, USA
[3]Department of Pediatrics, The Ohio State University College of Medicine, Columbus, Ohio, USA

Corresponding author: [1]E. Dewhirst, Department of Anesthesiology & Pain Medicine, The Ohio State University College of Medicine, Columbus, Ohio, USA. Email: Elisabeth.Dewhirst@osumc.edu

Keypoints

1. Cerebral oxygenation monitoring with near infrared spectroscopy (NIRS) may be useful in various clinical scenarios as an adjunct to non-invasive blood pressure and pulse oximetry.
2. NIRS may also be used in clinical scenarios where these monitors fail or cannot be used.
3. Cerebral NIRS provides a non-invasive means of ensuring adequate oxygen delivery to vital tissues and thereby serves as an additional monitor of cardiac output and oxygen delivery.

Abstract

One of the major goals of intraoperative care is the maintenance of adequate cardiac output and delivery of oxygen to the tissues. Standard intraoperative monitoring includes the non-invasive measurement of blood pressure (NIBP) and the use of pulse oximetry to monitor systemic oxygenation. Although generally effective, these monitors may fail. Monitors of tissue oxygenation such as cerebral oximetry using near infrared spectroscopy (NIRS) are being used more commonly in anesthesia practice. Clinical use in both adult and pediatric patients has demonstrated the potential utility of these devices in monitoring cerebral and tissue oxygenation. We report two pediatric patients in whom NIRS monitoring was used as the primary monitor of perfusion and cardiac output for a significant portion of the surgical procedure when other non-invasive monitors either failed or could not be used due to patient-related issues. The potential perioperative uses of NIRS monitoring are reviewed and these unique applications discussed.

Keywords: near infrared spectroscopy, cerebral oxygenation, intraoperative monitoring, tissue ischemia

Introduction

The key component of intraoperative care is the assurance of adequate cardiac output and delivery of oxygen to the tissues. While routine intraoperative monitoring includes the non-invasive monitoring of blood pressure (NIBP) and systemic oxygenation (pulse oximetry), there is significant interest in measuring end-organ tissue oxygenation. As such, monitors of tissue oxygenation such as cerebral oximetry using near infrared spectroscopy (NIRS) are being used more commonly in anesthesia practice.[1,2] The NIRS monitor features a skin sensor applied to the forehead with a laser light source

and two photodetectors. Using optical technology based on the relative absorption of infrared light by different hemoglobin species, the monitor generates a measurement of regional tissue oxygen saturation (rSO_2). Sampling is predominantly venous (70-75%) rather than arterial (25%) and does not require pulsatile flow. NIRS has been shown to correlate to invasive monitors of cerebral oxygenation, including mixed venous and jugular bulb oxygen saturation.[3,4] Data in both adult and pediatric patients have suggest the potential utility of monitoring cerebral oxygenation with NIRS in improving perioperative neurological outcomes.[5,6] We report two pediatric patients in whom NIRS monitoring was used as the primary monitor of perfusion and cardiac output for a significant portion of the surgical procedure. The first patient, undergoing bedside closure of sternotomy wound, had failure of non-invasive blood pressure (NIBP) and pulse oximetry (SpO_2) monitors during a period of acute hemorrhage and cerebral rSO_2 was used to direct resuscitative efforts. In the second case, due to associated osteogenesis imperfecta (OI), NIBP monitoring was not feasible due to the risk of fractures and cerebral rSO_2 was used to guide intraoperative anesthetic care.

Case reports

Institutional Review Board approval is not required at Nationwide Children's Hospital for the presentation of case reports involving fewer than 3 patients.

Patient 1.

A 5-month-old, 8.4 kg infant presented to the emergency department with increasing respiratory distress and impending respiratory failure. Admission chest radiograph demonstrated cardiomegaly and physical examination revealed signs of congestive heart failure including hepatomegaly and jugular venous distention. The infant was admitted to the Cardiothoracic ICU with a tentative diagnosis of congestive heart failure due to dilated cardiomyopathy. The infant's trachea was intubated and inotropic support started with milrinone and epinephrine. Echocardiography revealed a dilated cardiomyopathy with depressed function. The right coronary artery was noted to be arising from the aorta, but the left coronary was not visualized initially. With subsequent imaging, the left coronary artery was noted to be arising from the pulmonary artery thereby confirming the diagnosis of anomalous left coronary artery from the pulmonary artery (ALCAPA). The infant was subsequently taken to the operating room for surgical intervention. Following cardiopulmonary bypass, a left ventricular assist device (LVAD) was placed to provide added hemodynamic support during the postoperative period and the chest was left open. The infant's hemodynamic parameters stabilized over the ensuing 48 hours and the LVAD was removed although the chest was not closed. The following day, the decision was made to close the chest. Standard American Society of Anesthesiologists' monitors were placed including a pulse oximeter on the right foot and a NIBP on the left calf. Additionally, cerebral rSO_2 was measured bilaterally with NIRS probes on the forehead. Baseline cerebral rSO2 values were 50-55. Anesthesia was induced with fentanyl (total of 15 µg/kg) and midazolam (0.15 mg/kg) and neuromuscular blockade provided by vecuronium (0.2 mg/kg). During chest closure, the aortic suture where the LVAD cannula had been placed, was dislodged resulting in significant blood loss with an immediate decrease in the cerebral rSO_2 to 35-40. During this time, neither the pulse oximeter nor the NIBP were functioning. The heart rate increased to 180 beats/minute. Resuscitation was started with packed red blood cells (pRBCs) and titrated to achieve a cerebral rSO_2 of 50-60. As the cerebral rSO_2 returned to baseline, the HR decreased to 130-140 beats/minute. Over the next 30 minutes, a total of 100 mL/kg of pRBCs were transfused as the bleeding was controlled. As hemodynamic stability was re-established, both the pulse oximeter and the NIBP were able to record values. The chest was successfully closed and the patient's trachea was extubated the following day. The remainder of his hospital course

was unremarkable and he was eventually discharged home.

Patient 2.

The patient was a 15 month-old 5.74 kg boy who presented for a diagnostic upper gastrointestinal endoscopy, exchange of the gastrostomy tube, direct laryngoscopy, bronchoscopy and auditory brainstem evoked responses (ABER) evaluation. His past medical history included osteogenesis imperfecta, Chiari type I malformation, restrictive lung disease, hydrocephalus, and tracheostomy with ventilator dependence. His past surgical history included gastrostomy and circumcision. Current medications included polyethylene glycol (Miralax®) oral powder, ranitidine 7.5 mg via the G-tube once daily, cetirizine 2.5 mg via the G-tube once daily, and iron/vitamin supplements. Preoperative physical examination revealed a 15-month-old with multiple bony deformities and a tracheostomy in place. Vital signs revealed a HR of 161 beats/minute, a respiratory rate of 42 breaths/minute, and a room air oxygen saturation of 95%. NIBP was not evaluated due to the OI and concerns of fractures. On physical examination macrocephaly was noted and occasional rales were present on auscultation of the lung fields. Preoperative laboratory evaluation including electrolytes, renal function, coagulation function, blood glucose, and hepatic function were normal. The patient was held *nil per os* for 6 hours and transported to the operating room and routine American Society of Anesthesiologists' monitors were applied. Instead of NIBP, cerebral rSO_2 was assessed using NIRS throughout the procedure with the application of bilateral cerebral sensors. Baseline cerebral rSO_2 prior to the induction of general anesthesia varied from 50-65. Anesthesia was induced with the inhalation of sevoflurane via the patient's indwelling tracheostomy followed by the placement of an intravenous cannula. Fentanyl (1 µg/kg) and rocuronium (1 mg/kg) were administered intravenously. Mechanical ventilation was provided throughout the procedure and anesthesia maintained with sevoflurane (exhaled concentration 2-3%) in 50% air/oxygen. The patient was positioned supine and the general surgery team proceeded with upper GI endoscopy and feeding tube replacement. Direct laryngoscopy and bronchoscopy was then performed by the otorhinolaryngology service followed by ABER evaluation. The patient remained anesthetized for 145 minutes. HR varied from 112 to 179 beats/minutes, the end-tidal carbon dioxide from 30 to 36 mmHg, and the oxygen saturation from 99-100%. Cerebral rSO_2 values ranged from 75 to 85 bilaterally with only momentary decreases to the low 50s during apnea for the bronchoscopy. Following completion of the procedures, the patient was awakened and transferred to the Post Anesthesia Care Unit (PACU) for observation of hemodynamic and respiratory function. The remainder of his postoperative course was uncomplicated and he was discharged home the next day.

Discussion

During the provision of anesthetic care, standard monitoring includes the NIBP, pulse oximetry and continuous electrocardiography. These monitors ultimately serve to indirectly provide information regarding tissue perfusion and oxygen delivery to vital organs. Cerebral rSO2 monitoring using NIRS provides a direct measure of tissue oxygenation and thus may be a valuable addition to routine patient monitoring in various clinical scenarios. In rare circumstances, such as the two we report, cerebral rSO_2 monitoring may be used instead of conventional devices such as pulse oximetry and NIBP. Cerebral rSO2 does not require pulsatile flow and therefore functions during low flow states as was seen in our first patient when pulse oximetry and NIBP monitoring may be unreliable or ineffective.[2,7] The response time has been shown to be faster than that noted with pulse oximetry.[8] Aside from interference from ambient light, intravenous dyes, or rare circumstances of extreme elevations of bilirubin, cerebral rSO_2 monitoring using NIRS is generally free from artifact interference that prevents the monitor from functioning effectively.[9-11]

The efficacy of cerebral rSO_2 monitoring with NIRS during low flow states is demonstrated by studies using it to provide real time information on tissue oxygenation during cardiopulmonary resuscitation (CPR) in the adult population.[12-14] In general, these studies demonstrate improved survival with cerebral rSO2 values greater than 40 at the time of initiation of CPR as well during and immediately following resuscitation. It has been suggested that the higher rSO_2 values may reflect higher-quality CPR. Ito and colleagues report 92 patients in whom cerebral rSO2 was monitored during CPR after arrival to hospital following cardiac arrest.[12] Half of the patients with cerebral rSO_2 values greater than 40 had good neurological outcome versus 0 of the patients with cerebral rSO_2 values less than 25% (P<0.0001). Ibrahim and colleges monitored cerebral rSO_2 from the onset of resuscitation for up to 48 hours following the event in a cohort of 27 patients experiencing in hospital cardiac arrest.[13] When comparing resuscitation survivors to non-survivors, survivors had significantly higher cerebral rSO_2 values at the initiation of CPR (35 versus 17.5, P=0.03) and during resuscitation (36 versus 15, P=0.0008).

In the pediatric population, cerebral rSO2 has been used to guide fluid resuscitation in the ICU setting in children with hypovolemia from acute dehydration.[15] In a cohort of 17 moderately dehydrated children presenting to the emergency department, pulse oximetry and cerebral rSO_2 remained unchanged throughout rehydration. The somatic rSO_2 increased from 79 to 87 and the somatic to cerebral rSO_2 difference increased from 5 to 13 following intravascular fluid resuscitation. Higher volume resuscitation (33-40 mL/kg) resulted in a greater increase in somatic rSO_2 when compared to low-volume resuscitation (20 mL/kg). Similarly in our first patient, NIRS was used to guide resuscitation and blood replacement therapy during massive hemorrhage when both NIBP and pulse oximetry failed.

Cerebral rSO_2 has been used intraoperatively to identify the safe lower limits for arterial blood pressure in children.[16] In a cohort of 60 patients less than 3 months of age undergoing general anesthesia, if the systolic blood pressure was maintained within 20% of baseline, there was less than a 10% chance of experiencing cerebral desaturation, defined as a >20 decrease from baseline. Similarly, no change in cerebral rSO_2 was noted in a cohort of 19 pediatric patients when controlled hypotension (mean arterial pressure of 55-65 mmHg) was induced to limit intraoperative blood loss during spinal surgery.[17] There was no patient who had a greater than 20 decrease in cerebral rSO_2 when the MAP was $\geq$ 55 mmHg. The greatest decreases in cerebral rSO_2 were noted when there was a low mean arterial pressure in association with a low hemoglobin (less than 8 gm/dL) and hypocapnia demonstrating the importance of these parameters on cerebral oxygen delivery.

Although there may be significant inter-individual variation in cerebral rSO_2 values, the literature suggests that a decrease of more than 20 from baseline or an absolute value less than 40-50 results in an increased potential for cerebral hypoxia and neurologic dysfunction. Murkin and colleagues assigned 200 patients undergoing coronary artery bypass grafting to an intervention group, where action was taken if the rSO_2 fell below 75% of baseline and a non-intervention or control group.[18] Significantly more control patients had major organ morbidity or mortality (death, ventilation for more than 48 postoperative hours, stroke, myocardial infarction, return for re-exploration) versus the intervention group patients (P = 0.048).

In a prospective cohort study of 1178 adult patients undergoing cardiac surgery using cardiopulmonary bypass, Heringlake and colleagues measured preoperative cerebral rSO_2 and sought to explore its relationship with clinical outcome.[19] Preoperative cerebral rSO_2 values were found to correlate to other indicators of cardiopulmonary function including left ventricular ejection fraction, chemical markers of cardiac injury (troponin and B-type natriuretic peptide), and clinical illness scores. Cerebral rSO_2 values of <50 were independent risk factors for 30-

day and 1-year mortality. Within the highest risk patient group based on a EuroSCORE >10, 1-year mortality was twice as high in the group with cerebral rSO_2 values <50. The potential predictive values of a cerebral rSO_2 value less than 40-50 are supported by animal data showing increased lactate, minor and major EEG changes, and decreased ATP levels at cerebral rSO_2 values of 44, 42, 37, and 33 respectively.[20]

In summary, direct tissue monitoring with cerebral rSO_2 may be useful in various clinical scenarios as an adjunct to routine monitoring with NIBP and pulse oximetry or in clinical scenarios where these monitors fail or cannot be used. The literature in the adult population has suggested improvements in postoperative neurologic function when cerebral rSO_2 is monitored and maintained. As a direct measure of cerebral tissue oxygenation, cerebral rSO_2 provides a non-invasive means of ensuring adequate oxygen delivery to vital tissues and thereby serves as an additional monitor of cardiac output and oxygen delivery. As such, it may act as a valuable tool to guide intraoperative anesthetic care.

References

1. Sun JS, Elsey N, Tobias JD. Perioperative management of a patient with suspected cerebral vascular insufficiency: Utility of cerebral oxygenation monitoring using near infrared spectroscopy. Pediatr Anesth Crit Care J 2014;2:105-11.
2. Tobias JD. Cerebral oxygenation monitoring: near infrared spectroscopy. Expert Rev Med Devices 2006;3:235-43.
3. Tortoriello TA, Stayer SA, Mott AR, et al. A noninvasive estimation of mixed venous oxygen saturation using near-infrared spectroscopy by cerebral oximetry in pediatric cardiac surgery patients. Paediatr Anaesth 2005;15:495-503.
4. Ikeda K, MacLeod DB, Grocott HP, et al. The accuracy of a near-infrared spectroscopy cerebral oximetry device and its potential value for estimating jugular venous oxygen saturation. Anesth Analg 2014;119:1381-92.
5. 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;101:740-7.
6. Austin EH 3rd, Edmonds HL Jr, Auden SM, et al. Benefit of neurophysiologic monitoring for pediatric cardiac surgery. J Thorac Cardiovasc Surg 1997;114:707-15.
7. Madsen PL, Secher NH. Near-infrared oximetry of the brain. Prog Neurobiol 1999;58:541-60.
8. Tobias JD. Cerebral oximetry monitoring with near infrared spectroscopy detects alterations in oxygenation before pulse oximetry. J Intensive Care Med 2008;23:384-8.
9. Baek HY, Lee HJ, Kim JM, et al. Effects of intravenously administered indocyanine green on near-infrared cerebral oximetry and pulse oximetry readings. Korean J Anesthesiol 2015;68:122-7.
10. McDonagh DL, McDaniel MR, Monk T. The effect of intravenous indigo carmine on near-infrared cerebral oximetry. Anesth Analg 2007;105:704-6.
11. Madsen PL, Skak C, Rasmussen A, Secher NH. Interference of cerebral near-infrared oximetry in patients with icterus. Anesth Analg 2000;90:489-93.
12. Ito N, Nanto S, Nagao K, et al. Regional cerebral oxygen saturation on hospital arrival is a potential novel predictor of neurological outcomes at hospital discharge in patients with out-of-hospital cardiac arrest. Resuscitation 2012;83:46-50.
13. Ibrahim AW, Trammell AR, Austin H, et al. Cerebral oximetry as a real time monitoring tool to assess quality of in-hospital cardiopulmonary resuscitation and post-cardiac arrest care. J Am Heart Assoc 2015 (in press).
14. Genbrugge C, Dens J, Meex I, et al. Regional cerebral oximetry during cardiopulmonary resuscita-

tion: useful or useless? J Emerg Med 2016;50:198-207.

15. Hanson SJ, Berens RJ, Havens PL, Kim MK, Hoffman GM. Effect of volume resuscitation on regional perfusion in dehydrated pediatric patients as measured by two-site near-infrared spectroscopy. Pediatr Emerg Care 2009 ;25:150-3.
16. Michelet D, Arslan O, Hilly J, et al. Intraoperative changes in blood pressure associated with cerebral desaturation in infants. Paediatr Anaesth 2015;25:681-8.
17. Shear T, Tobias JD. Cerebral oxygenation monitoring using near infrared spectroscopy during controlled hypotension. Pediatr Anesth 2005;15:504-8.
18. Murkin JM, Adams SJ, Novick RJ, et al. Monitoring brain oxygen saturation during coronary bypass surgery: A randomized, prospective study. Anesth Analg 2007; 104:51-8.
19. Heringlake M, Garbers C, Käbler JH, et al. Preoperative cerebral oxygen saturation and clinical outcomes in cardiac surgery. Anesthesiology 2011;114:58-69.
20. Kurth CD, Levy WJ, McCann J. Near-infrared spectroscopy cerebral oxygen saturation thresholds for hypoxia-ischemia in piglets. J Cereb Blood Flow Metab 2002;22:335-41.