Pediatric Anesthesia and Critical Care Journal 2014;2(2):105-111
doi:10.14587/paccj.2014.22

PACCJ

Perioperative management of a patient with suspected cerebral vascular insufficiency: utility of cerebral oxygenation monitoring using near infrared spectroscopy

J. S. Sun[1], N. M. Elsey[2], J. D. Tobias[2,3]

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

Corresponding author: [2,3]J.D. Tobias, Department of Anesthesiology & Pain Medicine, Nationwide Children's Hospital, Columbus, Ohio, USA. Email: Joseph.Tobias@Nationwidechildrens.org

Key points

1. Various co-morbid conditions (cerebral vascular disease), changes in physiologic parameters (hypotension, hypothermia), and anesthetic agents can blunt the normal autoregulatory response of cerebral blood flow to changes in mean arterial pressure and the partial pressure of carbon dioxide in the blood.
2. During periods of nil per os, ongoing hydration with intravenous maintenance fluids is suggested to prevent hypovolemia and its potential impact on cerebral blood flow and oxygen delivery.
3. Intraoperative hypocarbia, hypercarbia, hypotension, hypovolemia, hyperthermia, and hypothermia have all been identified as potential risk factors for ischemic complications in patients with cerebral vascular insufficiency.
4. There is no evidence-based medicine comparing the effects and outcomes of patients receiving volatile versus intravenous anesthetic agents. Regardless of the agents chosen, maintenance of physiologic parameters at baseline is recommended.

Abstract

We present a child with clinical signs of cerebral vascular insufficiency who required anesthetic care during cerebral angiography. The impact of physiologic parameters including mean arterial pressure, partial pressure of carbon dioxide, and body temperature on the balance of cerebral oxygen demand and delivered are reviewed. The impact of anesthetic agents and co-morbid pathologic states on cerebral autoregulation are presented. Options for anesthetic care and the potential utility of cerebral oxygenation monitoring using near infrared spectroscopy (NIRS) are discussed.

Keywords: cerebral vascular insufficiency, near infrared spectroscopy, moyamoya

Introduction

Adequate cerebral oxygen delivery is dependent on the oxygen content of the blood and cardiac output. While the major etiologies of hypoxic-ischemic injury to the brain in infants and children are cardiac arrest or profound hypoxemia, rare congenital or acquired anomalies of the cerebra; vasculature may interfere with oxygen delivery to the central nervous system.[1] When clinical signs and symptoms suggest hypoxia or ischemia of the central nervous system, interventional radiologic procedures such as angiography may be indicated to delineate

the etiology.[2] Given the invasive nature of such procedures and the need for a motionless patient, these procedures require general anesthesia in the pediatric-aged patient.[3] We present a patient who required anesthesia care during cerebral angiography in order to evaluate signs and symptoms suggestive of cerebral vascular insufficiency with ischemia. The perioperative implications of such patients are reviewed, options for anesthetic care discussed, and the potential utility of cerebral oxygenation monitoring using near infrared spectroscopy (NIRS) presented.

Case report

Institutional Review Board approval is not required for single case reports at Nationwide Children's Hospital (Columbus, Ohio). The patient was a 16-month-old, 13 kilogram, boy who presented to Nationwide Children's with new-onset ataxia and left-sided facial droop. The patient had been evaluated at an outside hospital 4 days prior to admission, diagnosed with Bell's palsy, and started on oral prednisolone. His past history was significant for eczema, a recent upper respiratory infection, and acute otitis media. After admission to our institution, magnetic resonance imaging (MRI) revealed multiple areas of acute and chronic ischemic infarctions, as well as severe narrowing of the right ICA and MCA, and an area of focal stenosis of the left PCA with collateralization. Based on the MRI findings, a differential diagnosis of Moyamoya disease versus vasculitis was developed. The patient was subsequently scheduled for a cerebral angiogram under general anesthesia to distinguish between these two diagnoses. Prior to the procedure, a lengthy discussion was held between the pediatric anesthesiologist and neurologist caring for the patient, due to significant concerns of cerebrovascular compromise during general anesthesia. The pediatric neurology service specifically suggested tight blood pressure control throughout the procedure with a targeted systolic blood pressure (sBP) between 120-140 mmHg due to documented worsening of clinical signs and symptoms when the patient's sBP was less than 120 mmHg.

On the day of the procedure, the patient was held nil per os for 6 hours. During this time, ongoing hydration was maintained with maintenance intravenous fluids administered via a peripheral intravenous cannula. He was transported to the invasive radiology suite. Vital signs for the 24 hours prior to the procedure included a BP ranging from 119-140/52-89 mmHg with a heart rate of 88-156 beats/minute. Prior to the induction of anesthesia, standard American Society of Anesthesiologists' monitors were placed, with an initial non-invasive blood pressure of 114/49 mmHg (MAP 66 mmHg) and heart rate of 113 beats/minute. Additionally, bilateral cerebral oxygenation was non-invasively monitored using NIRS.[4] General anesthesia was induced through an in situ 22 gauge peripheral intravenous cannula in the left saphenous vein with glycopyrrolate 0.1 mg (7 µg/kg) and propofol 30 mg (2.3 mg/kg). To avoid any decrease in the BP related to propofol, phenylephrine 5 µg (0.4 µg/kg) was administered with the propofol. Tracheal intubation was facilitated with rocuronium (0.8 mg/kg). The patient's trachea was intubated with a 3.5 mm cuffed endotracheal tube (ETT) as there was no audible air leak around a 4.0 mm cuffed ETT at 30 cmH_2O. The cuff was inflated with less than 0.5 cc of air to seal the airway at 20 cmH_2O. Following anesthetic induction, a 22 gauge radial arterial catheter and two additional peripheral intravenous cannulae were placed. Anesthesia was maintained with 50-60% oxygen in air and sevoflurane (end-tidal concentration of 1-3%). During cannulae placement, the patient's BP decreased to 116/40 mmHg (MAP 59 mmHg), which was subsequently treated with phenylephrine (5 µg). This resulted in an increase of the BP to 127/69 mmHg. Due to the lability of the patient's BP during general anesthesia, an infusion of phenylephrine was initiated at 0.5 µg/kg/min to maintain the sBP at 120-140 mmHg. The initial NIRS readings for the left and right side cerebral oxygenation at the time of anesthesia induction were 84 and 76, respectively. No

significant decline was noted with the momentary decrease in the sBP to less than 120 mmHg. During the course of the procedure, it was noted that the cerebral oxygenation decreased to the 60-65 level following the injection of contrast into the left carotid artery for angiography. More concerning was the sharp decline into the 30's of the right-sided cerebral oxygenation during contrast injection of the right cerebral vasculature. Due to concerns of poor right-sided cerebral perfusion, during the remainder of the right vasculature evaluation, the phenylephrine infusion was increased to 0.7 µg/kg/min with a subsequent increase in sBP to 140-165 mmHg. A corresponding increase in the right-sided cerebral oxygenation from 30 to 60 was noted with the increase in the sBP. During the conclusion of the procedure and placement of the vascular hemostatic device, the phenylephrine infusion was tapered to off and the sBP was allowed to return back to its baseline values of 120-140 mmHg. No change in the NIRS was noted during the return of the sBP to baseline. Residual neuromuscular blockade was reversed with glycopyrrolate and neostigmine. Prior to emergence, dexmedetomidine 4 µg was administered and once the patient had resumed spontaneous ventilation and met extubation criteria, the ETT was removed. During the time surrounding tracheal extubation, the patient's sBP increased to 161 mmHg and fentanyl was administered in 5 µg increments to a total of 20 µg to provide analgesia and anxiolysis. Prior to disconnecting the NIRS monitor, the left and right cerebral oxygenation had returned to baseline values of 85 and 74, respectively. The patient was transported to the post-anesthesia care unit (PACU) with the arterial line in place for close perioperative monitoring. While in the PACU, it was noted that the patient was moving all four extremities and had returned to his baseline neurologic status. The cerebral angiogram revealed a narrowed left internal carotid artery and extensively narrowed right internal carotid artery with occlusions of the M1 and A1 segments. There was also extensive collateral formation on the right side. The angiography was diagnostic for Moyamoya. The day following the procedure, the patient was discharged home, but was subsequently readmitted for progression of his neurologic symptoms. He subsequently underwent an attempt at cerebral revascularization (pial synangiosis). Although he tolerated that procedure well, he has subsequently had progression of the disease process with additional episodes of cerebral infarction and progressive decline in his neurologic function. A follow-up with the patient four months after his surgery has shown that the patient has developmental delays in gross motor skills, fine motor skills, and speech. He takes some food by mouth, but requires a gastrostomy tube. A follow-up MRI scan of his brain three months after his surgery showed widespread encephaloclastic changes throughout his brain, affecting most of the right cerebral hemisphere and large portions of the left MCA territories. No further interventions are being considered at this point.

Discussion

In the healthy state, cerebral blood flow and oxygen delivery are tightly regulated to match the demands of the central nervous system. During alterations in BP or oxygen delivery, changes in the cerebral vasculature may compensate to maintain cerebral blood flow (CBF). In pathologic states, cerebral autoregulation may be non-functional or insufficient, such that alterations in MAP may result in cerebral ischemia provided there is no associated change in the cerebral metabolic rate for oxygen ($CMRO_2$). Furthermore, alterations in the extracranial or intracranial vasculature may mandate a higher than normal MAP to ensure adequate cerebral perfusion.

In our patient, general anesthesia was required to provide immobility during cerebral angiography. Although hemodynamic depression with a decrease in MAP may occur throughout anesthetic care, it is most common during anesthetic induction. Propofol was chosen for anesthetic induction given its beneficial effects on cerebral hemodynamics including a decrease of $CMRO_2$ and in-

tracranial pressure.[5,6] However, given the potential for hypotension following propofol which may impact cerebral perfusion pressure (CPP), MAP was maintained by the concomitant administration of phenylephrine.[7,8] In patients with co-morbid cardiovascular diseases, etomidate may be an effective alternative agent for anesthetic induction. While etomidate's effects on $CMRO_2$ and ICP are similar to those of propofol, it generally has minimal effects on cardiac function and MAP thereby maintaining or increasing CPP.[9,10]

In addition to an adequate depth of anesthesia, a neuromuscular blocking agent (NMBA) may be administered to facilitate endotracheal intubation and avoid movement during direct laryngoscopy. Movement and coughing during endotracheal intubation may have negative effects on CBF and ICP. Furthermore, there may be direct effects related to the NMBA. Succinylcholine may increase ICP and cause histamine release with deleterious effects on MAP and CPP.[11,12] We choose to use rocuronium given its negligible effects on hemodynamic status and limited histamine release. Both rocuronium and vecuronium have been shown to have minimal effects on ICP, MAP and CPP.[13] Given the need for prompt postoperative neurologic evaluation, the anesthetic should be planned to allow for early tracheal extubation including the use of train-of-four monitoring. Likewise, the agents for maintenance of anesthesia should allow for easy titration, have limited effects on MAP, ICP and CPP while providing rapid recovery. For maintenance anesthesia, we chose to continue with sevoflurane given the rapid dissipation of its effects and the ability to closely titrate the depth of anesthesia. However, sevoflurane can have effects on both ICP/CBF and MAP. Although sevoflurane has been shown to increase CBF and decreases CVR in a dose-dependent manner, CO_2-reactivity is preserved during 1.5% and 2.5% sevoflurane.[14] All of the volatile anesthetic agents may increase CBF; however, the effects are less with sevoflurane compared to isoflurane and desflurane and if needed, can be blunted by modest hyperventilation ($PaCO_2$ 30-35 mmHg).[15,16]Additionally, cerebral pressure autoregulation has been shown to better preserved with sevoflurane than the other volatile anesthetic agents.[17] A theoretical concern with the volatile anesthetic agents is that cerebral vasodilation may result in intracerebral steal especially in patients with cerebral vascular disorders such as Moyamoya.[18] It is postulated that vasodilatation occurs only in the normal vasculature resulting in shunting of blood away from the ischemic area.[18] Although we chose to use sevoflurane in a concentration ≤ 1 MAC, others have suggested the use of a total intravenous anesthetic technique.[19]

During emergence from anesthesia, both dexmedetomidine and fentanyl were administered to treat pain and prevent emergence delirium. Both agents have been shown to have limited effects on CBF and ICP provided that the MAP is kept constant.[20-23] Most importantly with dexmedetomidine, any decrease in CBF has been shown to match a concomitant decrease in cerebral metabolic rate for oxygen resulting in an increase in cerebral tissue oxygenation (PaO_2).[21,22]

Other factors known to affect CBF during the normal and pathologic states include changes in the partial pressure of oxygen and carbon dioxide in the blood (PaO_2 and $PaCO_2$) and body temperature.[24] In the healthy state, the relationship between $PaCO_2$ and CBF is linear so that CBF is twice the baseline state at a $PaCO_2$ 80 mmHg. No further increase in flow is possible beyond this point as the arterioles are maximally dilated. Conversely at a $PaCO_2$ of 20 mmHg, CBF is approximately half the baseline value and cannot decrease further as vasoconstriction is maximal. The baseline arteriolar tone and hence the MAP influences the CBF response to $PaCO_2$. Moderate hypotension impairs the response to changes in $PaCO_2$ while severe hypotension abolishes it altogether. However, in patients with cerebral vascular disease including Moyamoya, the response to changes in $PaCO^2$ may be particularly detrimental mandating close monitoring of ventilation and maintenance of normocarbia during anesthetic care.[25-27] Although hy-

perventilation has been a time-honored therapy for neurosurgical anesthesia as it can be helpful in decreasing ICP, the concomitant decrease in CBF has led to changes in practice both intraoperatively and in the ICU setting.[28-30] Intraoperatively, end-tidal CO_2 may be used to guide ventilation; however, for prolonged cases, intra-arterial access should be considered to allow for intermittent monitoring of $PaCO_2$. These issues may also occur postoperatively as hyperventilation with crying or agitation, can also lower $PaCO_2$ and cause cerebral vasoconstriction potentially precipitating an ischemic event. Techniques to reduce postoperative pain and agitation should be implemented to prevent such issues. However, the benefits of such techniques should be weighed against the risks of respiratory depression. In our patient, we chose to carefully titrate fentanyl along with dexmedetomidine to control postoperative agitation while limiting the effects on respiratory function.

Body temperature may also result in changes in CBF. Although hypothermia reduces $CMRO_2$ and may impart some degree of neuroprotection, significant hypothermia may result in cerebral vasospasm in patients with cerebral vascular disease thereby decreasing CBF resulting in ischemia.[31-33] Conversely, an increase in body temperature may increase $CMRO_2$ and likewise upset the balance of cerebral oxygen delivery and demand resulting in ischemia.[34] Given these concerns, continuous monitoring of core body temperature is suggested with techniques such as forced air warming to maintain normothermia.

While clinical signs and symptoms may alert the clinician to changes in cerebral blood flow during the awake state, these signs are unavailable during general anesthesia. As such, we chose to maintain the sBP at a level known to provide adequate CBF during the awake state. Additionally, we monitored cerebral oxygenation using NIRS during anesthetic care.[4] Recent evidence has demonstrated the potential applications of non-invasive monitoring of cerebral oxygenation especially when there may be altered autoregulation.[35,36] The potential utility of such monitoring was demonstrated in our patient as alterations in cerebral oxygenation during injections into the carotid artery alerted us to the need to titrate the phenylephrine infusion to increase MAP to improve CBF.

In summary, we describe the anesthetic management of a patient with presumed cerebral vascular insufficiency presenting for anesthetic care. Angiographic changes were suggestive of Moyamoya. During the perioperative care of such patients, changes in various physiologic parameters including MAP, $PaCO_2$, and body temperature can have significant impact on CBF and cerebral oxygenation. Given the impact of MAP on CBF and the potential changes that can occur intraoperatively, we would suggest consideration of invasive arterial BP monitoring. Additionally, this would also allow intermittent monitoring of $PaCO_2$. The co-morbid patient condition and pathologic involvement of the cerebral vasculature as well as the anesthetic agents may affect cerebral autoregulation. Given the interplay of these factors, we find cerebral oxygenation monitoring using NIRS helpful in the control of perioperative variables which affect CBF.

References

1. Gieron-Korthals M1, Colón J. Hypoxic-ischemic encephalopathy in infants: new challenges. Fetal Pediatr Pathol 2005;24:105-20.
2. Kaufman T, Kallmes D. Diagnostic cerebral angiography: archaic and complication-prone or here to stay for another 80 years? Am J Roentgenol 2008;190:1435-7.
3. Landrigan-Ossar M1, McClain CD. Anesthesia for interventional radiology. Paediatr Anaesth 2014;24:698-702.
4. Tobias JD. Cerebral oxygenation monitoring: near infrared spectroscopy. Expert Rev Med Devices 2006;3:235-43.

5. Spitzfaden AC, Jimenez D, Tobias JD. Propofol for sedation and control of intracranial pressure in children. J Pediatr Neurosurg 1999;31:194-200.
6. Tobias JD. Propofol: Effects on the central nervous system. J Intensive Care Med 2000; 15:237-246.
7. Pinaud M, Lelausque J, Chetanneau A, et al. Effects of propofol on cerebral hemodynamics and metabolism in patients with brain trauma. Anesthesiology 1990;73:404-9.
8. Tritapepe L, Voci P, Marino P, et al. Calcium chloride minimizes the hemodynamic effects of propofol in patients undergoing coronary artery bypass grafting. J Cardiothor Vasc Anes 1999;13:150-3.
9. Tobias JD. Etomidate: Applications in pediatric critical care and pediatric anesthesiology. Pediatr Crit Care Med 2000;2:100-6.
10. Moss E, Powell D, Gibson RM, McDowall DG. Effect of etomidate on intracranial pressure and cerebral perfusion pressure. Br J Anaesth 1979;51:347-52.
11. Minton MD, Grosslight K, Stirt JA, Bedford RF. Increases in intracranial pressure from succinylcholine: prevention by prior nondepolarizing blockade. Anesthesiology 1986;65:165-9.
12. Ertama PM. Histamine liberation in surgical patients following administration of neuromuscular blocking drugs. Ann Clin Res 1982;14:15-26.
13. Schramm WM, Strasser K, Bartunek A, Gilly H, Spiss CK. Effects of rocuronium and vecuronium on intracranial pressure, mean arterial pressure and heart rate in neurosurgical patients. B J Anaesth 1996;77:607-11.
14. Bundgaard H, von Oettingen G, Larsen KM, et al. Effects of sevoflurane on intracranial pressure, cerebral blood flow and cerebral metabolism. A dose-response study in patients subjected to craniotomy for cerebral tumours. Acta Anaesthesiol Scand 1998;42:621-7.
15. Holmström A, Akeson J. Desflurane increases intracranial pressure more and sevoflurane less than isoflurane in pigs subjected to intracranial hypertension. J Neurosurg Anesthesiol 2004;16:136-43.
16. Holmström A, Akeson J. Cerebral blood flow at 0.5 and 1.0 minimal alveolar concentrations of desflurane or sevoflurane compared with isoflurane in normoventilated pigs. J Neurosurg Anesth 2003;15:90-7.
17. Summons AC, Gupta AK, Matta BF. Dynamic cerebral autoregulation during sevoflurane anesthesia: a comparison with isoflurane. Anesth Analg 1999;88:341-5.
18. Yamada K, Miyamoto S, Nagata I, et al. Reverse steal phenomenon in Moyamoya disease. Jpn J Neurosurg 1995;4:145-50.
19. Sato K, Shirane R, Kato M, et al. Effect of anesthesia on cerebral circulation in Moyamoya disease. J Neurosurg Anesth 1999;11:25-30.
20. Aryan HE, Box KW, Ibrahim D, Desiraju U, Ames CP. Safety and efficacy of dexmedetomidine in neurosurgical patients. Brain Inj 2006;20:791-8.
21. Drummond JC, Dao AV, Roth DM, et al. Effect of dexmedetomidine on cerebral blood flow velocity, cerebral metabolic rate, and carbon dioxide response in normal humans. Anesthesiology 2008;108:225-32.
22. Drummond JC1, Sturaitis MK. Brain tissue oxygenation during dexmedetomidine administration in surgical patients with neurovascular injuries. J Neurosurg Anesth 2010;22:336-41.
23. Sperry RJ, Bailey PL, Reichman MV, et al. Fentanyl and sufentanil increase intracranial pressure in head trauma patients. Anesthesiology 1992;77:416-20.
24. Battisti-Charbonney A, Fisher J, and Duffin J. The cerebrovascular response to carbon dioxide in humans. J Physiol 2011;589:3039-48.
25. Ogawa A, Nakamura N, Yoshimoto T, Suzuki J. Cerebral blood flow in moyamoya disease. Part 2: Autoregulation and CO2 response. Acta Neurochirurgica 1990;105:107-11.

26. Kuwabara Y, Ichiya Y, Fukui M, et al. Response to hypercapnia in moyamoya disease. Cerebrovascular response to hypercapnia in pediatric and adult patients with moyamoya disease. Stroke 1997;28:701-7.
27. Digiusto M1, Bhalla T, Grondin R, Tobias JD. Perioperative care of the pediatric patient for pial synangiosis surgery. Int J Clin Exp Med 2013;6:231-8.
28. Muizelaar JP, Marmarou A, Ward JD, et al. Adverse effects of prolonged hyperventilation in patients with severe head injury: A randomized clinical trial. J Neurosurg 1991;75:731-9.
29. Wax DB, Lin HM, Hossain S, Porter SB. Intraoperative carbon dioxide management and outcomes. Eur J Anaesthesiol 2010; 27: 819-23.
30. Rozmiarek A, Taghon T, Tobias JD. Inadvertent hyperventilation during intraoperative anesthetic care in the pediatric population. ICU Director 2012;4:172-5.
31. Busto R, Dietrich WD, Globus MY, et al. Small differences in intraischemic brain temperature critically determine the extent of ischemic neuronal injury. J Cereb Blood Flow Metab 1987;7:729-38.
32. Malley RA, Frost EA. Moyamoya disease: pathophysiology and anesthetic management. J Neurosurg Anesth 1989;2:110-4.
33. Kurokawa T, Chem YJ, Tomita S, Kishikawa T, Kitamura K. Cerebrovascular occlusive disease with and without the moyamoya vascular network in children. Neuropediatrics 1985;6:29-32.
34. Sato K, Shirane R, Yoshimoto T. Perioperative factors related to the development of ischemic complications in patients with Moyamoya disease. Child Nerv Syst 1997;13:68-72.
35. Ono M, Joshi B, Brady K, et al. Risks for impaired cerebral autoregulation during cardiopulmonary bypass and postoperative stroke. Br J Anaesth 2012;109:391-8.
36. Joshi B, Ono M, Brown C, et al. Predicting the limits of cerebral autoregulation during cardiopulmonary bypass. Anesth Analg 2012;114:503-10.