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

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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. Given the invasive nature of such procedures and the need for a motionless patient, these procedures require general anesthesia in the pediatric-aged patient. 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. 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 cmH2O. The cuff was inflated with less than 0.5 cc of air to seal the airway at 20 cmH2O. 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 (CMRO₂). 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 CMRO₂ and in-

Sun et al. Anesthesia and cerebral vascular insufficiency
tracranial pressure. However, given the potential for hypotension following propofol which may impact cerebral perfusion pressure (CPP), MAP was maintained by the concomitant administration of phenylephrine. In patients with co-morbid cardiovascular diseases, etomidate may be an effective alternative agent for anesthetic induction. While etomidate’s effects on CMRO₂ and ICP are similar to those of propofol, it generally has minimal effects on cardiac function and MAP thereby maintaining or increasing CPP.

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. 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. 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, CO₂-reactivity is preserved during 1.5% and 2.5% sevoflurane. 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 (PaCO₂ 30-35 mmHg). Additionally, cerebral pressure autoregulation has been shown to better preserved with sevoflurane than the other volatile anesthetic agents. 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. It is postulated that vasodilation occurs only in the normal vasculature resulting in shunting of blood away from the ischemic area. Although we chose to use sevoflurane in a concentration ≤ 1 MAC, others have suggested the use of a total intravenous anesthetic technique. 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. 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 (PaO₂).

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 (PaO₂ and PaCO₂) and body temperature. In the healthy state, the relationship between PaCO₂ and CBF is linear so that CBF is twice the baseline state at a PaCO₂ 80 mmHg. No further increase in flow is possible beyond this point as the arterioles are maximally dilated. Conversely at a PaCO₂ 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 PaCO₂. Moderate hypotension impairs the response to changes in PaCO₂ while severe hypotension abolishes it altogether. However, in patients with cerebral vascular disease including Moyamoya, the response to changes in PaCO₂ may be particularly detrimental mandating close monitoring of ventilation and maintenance of normocarbia during anesthetic care. 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.\textsuperscript{28-30} Intraoperatively, end-tidal CO\textsubscript{2} may be used to guide ventilation; however, for prolonged cases, intra-arterial access should be considered to allow for intermittent monitoring of PaCO\textsubscript{2}. These issues may also occur postoperatively as hyperventilation with crying or agitation, can also lower PaCO\textsubscript{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\textsubscript{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.\textsuperscript{31-33} Conversely, an increase in body temperature may increase CMRO\textsubscript{2} and likewise upset the balance of cerebral oxygen delivery and demand resulting in ischemia.\textsuperscript{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.\textsuperscript{4} Recent evidence has demonstrated the potential applications of non-invasive monitoring of cerebral oxygenation especially when there may be altered autoregulation.\textsuperscript{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\textsubscript{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\textsubscript{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


