Cerebral Oximetry – Any Value?

Hilary P. Grocott, MD, FRCPC, FASE
Professor, Departments of Anesthesia and Surgery
University of Manitoba
Winnipeg, Manitoba, Canada

Cerebral oximetry monitoring, used with the intention of optimizing perioperative outcomes, is increasingly being used in the setting of both cardiac and non-cardiac surgery. Cerebral oximetry had its early beginnings in the 1980s with the work of Jöbsis and colleagues.1 By using multi-wavelength light sources in the near infrared range, these early investigations demonstrated the potential utility of exploiting the ability of the differential absorption of light by oxygenated and de-oxygenated hemoglobin in brain (and possibly other) tissue. Cerebral oximetry integrates venous and arterial signals to give a mixed (in an approximately 3:1 ratio of venous to arterial blood) overall tissue oximetric signal. By integrating this oximetric data and comparing it to validated direct measurements of jugular venous saturation, these devices (with at least 5 commercial devices now on the market worldwide and likely more to be approved soon), produce a continuous output of tissue (i.e. brain) oxygen saturation. This saturation information can then be integrated with our understanding of oxygenation delivery and utilization conditions to allow modifications to be made in perioperative physiologic conditions with the aim of optimizing overall tissue oxygenation, and ideally, corresponding end-organ function and outcome.

Cerebral oximetry was initially used to monitor the brain in an attempt to mitigate brain injury after cardiac surgery. As there had been a well described pattern of brain injury known since the advent of cardiac surgery, to have a monitor to potentially determine when these injurious events were occurring was clearly seen as advantageous. What followed in the literature was a logical time course and pattern of observational and anecdotal case reports outlining the use of these devices. These reports were largely in two categories: those describing catastrophe avoidance and those describing various clinical decision making opportunities.

Despite cerebral oximetry being available for almost two decades, the accumulation of supportive data has been slow. It has only been relatively recently that randomized controlled data specifically defining the utility of cerebral oximetry have been published. Murkin et al published a trial of 200 patients in which a physiologically-based interventional strategy was utilized to maintain the cerebral saturation signals within 75% of their baseline reading.2 This interventional strategy was based upon optimizing both oxygen supply and utilization in the brain. For example, following establishment of the baseline reading (an important initial step in utilizing cerebral oximetry), the investigators instituted an interventional algorithm if the patient’s saturation dropped 20% from their baseline. This intervention included ruling out mechanical causes such as cannula malplacement or jugular venous impingement due to head position, and followed with techniques to optimize oxygen supply to the brain. For example, if patients were hypocapnic, PaCO₂ was returned to a normal level. In addition, the mean arterial pressure (MAP) was increased modestly, and as well, increases in FiO₂ were made. If these parameters failed to return the saturation to normal, and if there was significant anemia, the patients were transfused to improve oxygen carrying capacity. If these efforts to improve
oxygen delivery failed, additional methods to suppress cerebral oxygen metabolism were used including administration of additional propofol and modest cooling. This management strategy has been further elucidated by Denault and colleagues. Although that particular study was not powered to adequately examine neurological outcome, the results did demonstrate a trend toward the stroke reduction in the interventional group. However, a unique aspect to the study was that not only was there a trend toward an improvement in neurologic outcome, but that there was an improvement in an overall outcome as identified by reduction in major organ morbidity.

Indeed, this study described that the use of these technologies may have come full circle from only examining brain perfusion (as a means to improve neurologic outcome) to the point of monitoring brain perfusion as an index of overall organ function. That is, the brain may simply represent an index organ for overall tissue perfusion. Part of this stems from the brain being the only major organ that is within reach of the light sources that these devices utilize. Ironically however, in some respects its protective mechanisms (i.e. autoregulation) make it the last organ to be compromised in a situation of impaired blood flow and oxygenation. That is, based on the hierarchy of blood flow that occurs in the various organs (i.e. the brain is the most preserved and the last to desaturate), it is likely that when the brain does desaturate, the other organs have long since desaturated. As a result, those patients that have significant cerebral desaturations likely have other organ desaturation and this may be responsible for the other adverse perioperative outcomes described. Arguably, the brain is exactly the opposite of the “canary in the coal mine” in that its oxygenation status is maintained long after other organs (such as those perfused by the splanchnic vasculature) have been compromised. Thus, although it is probably important to maintain its saturation, covert tissue compromise is likely occurring frequently despite our confidence that we are doing all the right things. This probably accounts for the lack of robust correlation to overall outcomes in several other cerebral oximetry trials.

Although the vast majority of investigations into the potential utility for cerebral oximetry have come from studies involving cardiac surgery patients, there have also been some notable studies focusing on its potential utility in the non-cardiac surgery patient. For example, Casati et al studied cerebral oximetry in an elderly non-cardiac surgical population, again demonstrating that improvements in neurologic outcome (i.e. postoperative cognitive dysfunction) could be reduced if this type of monitor was used with a similar type interventional strategy. Although this study was in high-risk patients undergoing major abdominal surgery, patients requiring thoracic surgery represent a similarly higher risk group.

In the thoracic surgery population, there are several recent studies that have focused on the potential utility of cerebral oximetry specific to this setting. The onset of one lung ventilation (OLV), an unquestionable necessity in the thoracic surgery population, increases the potential for perioperative hypoxemia. In addition, there is significant potential for changes in the hemodynamic profile, which may also have some impact on brain perfusion, but other organs as well. In addition, the patient undergoing thoracic surgery is often elderly and has numerous comorbidities. This increases the potential for perioperative complications. Compared to investigations in the cardiac population, which arguably is still to emerge from its monitoring infancy, these thoracic surgery studies are even more rudimentary; despite this, several insights can be gained from these reports.
Hemmerling et al were the first to report decreases in cerebral oxygen saturation in a small study of 20 patients undergoing OLV for thoracic surgery. They highlighted the potential for hypoxia inherent with OLV that, in part, is variably related to significant physiologic disturbances, including hypoxic pulmonary vasoconstriction, pulmonary arterial venous shunting, reductions in oxygen partial pressure concomitant with OLV, and hemodynamic changes that may result in increases in pulmonary pressures and reductions in cardiac output. As a result, OLV may both decrease oxygen content as well as cardiac output-dependent oxygen delivery. These are important variables that may have an impact on oxygenation status in numerous organ systems, including the brain. However, although these authors were able to demonstrate significant decreases in oxygen saturation during OLV, the definition of desaturation was based on a reduction from an artificially elevated baseline cerebral saturation (obtained while on 100% oxygen with bilateral ventilation). This definition could excessively increase the incidence of desaturation, which they defined as an absolute decrease of 15% from their baseline, falsely drawing attention to the importance of desaturation. For example, the majority of their patients, despite having significant saturation reductions, still had absolute oxygen saturations well above what would be considered a significant or dangerous level of desaturation. For example, if the baseline saturation was 80% and this decreased to 63% during OLV, this was considered significant desaturation, but 64% per se would still be well above what would be considered as dangerous.

Indeed, one of the inconsistencies with many of these studies relates to the incidence and severity of desaturation. In particular, the definition of desaturation varies considerably. Although clearly reductions in cerebral saturation can occur in these settings, the significance of these reductions must be tightly linked to postoperative outcomes. Thus far, there is insufficient investigation to truly understand the potential utility for the thoracic surgery population, however, there are important signals that have been described. Furthermore, the relatively small size of these studies also reduces the confidence in the conclusions.

In an important subsequent follow up study, the same investigative group attempted to correlate the degree of cerebral saturation with postoperative complications. Once again, a large number of patients had decreases in saturation of >15% from the baseline. They demonstrated that the minimum absolute value during OLV correlated with an increased incidence of postoperative complications. These complications were defined by the Clavien and SOFA scores, which are two different scores that evaluate the severity of postoperative complications. In a similar fashion to the cardiac surgery trial by Murkin et al, most of these complications were non-neurologic related, and were principally related to respiratory, renal or atrial fibrillation complications.

Cerebral complications after thoracic surgery have received little focused investigation. Tang et al recently reported a trial (n=76) where cognitive function was assessed before operation, then 3 and 24 hours postoperatively using the Mini-Mental Status Exam (MMSE). 29% of the patients had a decrease in their MMSE of >2 points at 3 hours after surgery, with 10% having a decrease persisting at 24 hours. Postoperative cognitive dysfunction was variably correlated with absolute saturations of <65, 60 and 55% respectively, in a “dose” dependent nature. Indeed, the odds ratio having developing early cognitive dysfunction with even a brief (<5 minute exposure to a saturation of <65% was 2.03 [95% CI, 0.74 to 5.59]). So, in addition to
previous studies having shown a correlation of desaturation with non-neurologic complications, their study suggests that there may be some relationship to the neurologic “injury” manifest as cognitive dysfunction.

The trials reported thus far are important additions to our understanding of perioperative complications. There is clearly sufficient information available to expand the use of this device and integrate it into one’s practice, but additional information will be necessary to fully understand the capabilities of this equipment and the interventional protocols that can be used to optimize patient outcomes. The current data looks very promising, however, it is only with rigorous further randomized blinded data that we will truly understand the capability that this technology offers both cardiac and non-cardiac patients.

References