Cerebral Oximetry in Cardiac Surgery

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Stroke and neurocognitive dysfunction are common after cardiac surgery with rates of approximately 3-6% and 30-50%, respectively (1,2). Multiple etiologies have been proposed for these neurological insults. Microembolism, macroembolism, hyperperfusion, or some combination of these are the most likely culprits. In all of these scenarios the final pathway is tissue ischemia, resulting in neurodegeneration. The ability to insure adequate cerebral perfusion is further complicated by the fact that nearly half of patients presenting for cardiac surgery have either intracranial or extracranial atherosclerotic disease. Given this high level of risk, a monitor capable of detecting perioperative cerebral ischemia could significantly reduce adverse events in this patient population.

Cerebral oximetry was first described more than 25 years ago, however the interest in its use during cardiac surgery is a recent phenomenon. This technology is similar to pulse oximetry in that it uses differences in light absorption between oxygenated and deoxygenated hemoglobin to measure regional oxygen saturation. Cerebral oximeters emit sequentially pulsed near-infrared light that is capable of penetrating the skull, and detect photons reflected back through the skull to determine oxygen saturation. The device estimates that blood in the brain microvasculature is approximately 70% venous and 30% arterial. Thus, the measurement represents a balance between oxygen delivery and consumption of the brain.

There are currently two cerebral oximeters approved by the FDA for clinical use. The INVOS device (Somanetics Corporation, Troy, Michigan) was the first FDA approved cerebral oximeter. In one of the first clinical studies, Henson and colleagues demonstrated a correlation between changes in cerebral oxygen saturations and jugular bulb saturations using the INVOS device. They also noted a wide variation in baseline values among individuals (3). This suggests that the INVOS device is best used as a trend monitor since the absolute value may not accurately reflect actual cerebral oxygen saturation. In contrast, a newer device to the market, the Fore-Sight cerebral oximeter (CAS Medical Systems, Branford, CT), is designed to measure absolute brain oxygen saturation and does not require a baseline measurement. An absolute value of less than 55% is considered high risk for cerebral ischemia regardless of baseline value. In a study of 18 individuals, McLeod et al. showed a “strong correlation” between brain oxygen saturation calculated from arterial (30%) and jugular bulb (70%) blood samples and that measured by the Fore-Sight oximeter (4). To date, no studies comparing these two devices have been published.

Preliminary clinical studies in humans suggest that cerebral oximetry may be a useful intra-operative monitor. The majority of the published data uses the INVOS device. In a retrospective study of 2,279 patients, Goldman and colleagues demonstrated a decrease in the stroke rate in cardiac surgical patients after implementation of cerebral oximetry in their practice (5). In a smaller prospective, observational trial, Yao et al. observed an association between cerebral desaturation and neurocognitive dysfunction in 101 patients undergoing cardiac surgery. Yao found that patients with a cerebral oxygen saturation of less than 40% for longer than 10 minutes had an increased incidence of neurocognitive dysfunction (6). In a randomized, prospective study, Casati and associates demonstrated an association between reductions in cerebral oximetry and postoperative neurocognitive dysfunction. In this study of elderly patients undergoing major abdominal surgery, cerebral saturations in the treatment group were maintained at 75% of baseline values by following a specified treatment algorithm (7). A similar protocol was used in a recently published prospective, randomized trial of 200 cardiac surgical patients by Murkin and colleagues. They demonstrated a lower incidence of major organ morbidity and mortality when cerebral saturations were maintained above 75% of baseline. Major organ morbidity and mortality was defined as stroke, renal failure, prolonged ventilation, deep sternal infection, reoperation, or death (8). Unfortunately, this study did not test for neurocognitive dysfunction, nor was it powered to detect differences in stroke. Although this research supports the use of cerebral oximetry in cardiac surgery, further studies are needed to determine if this technology can be used to decrease the incidence of stroke and neurocognitive dysfunction in this patient population.

The findings of Murkin suggest that cerebral oximetry may be useful as an index for systemic oxygen delivery and consumption. The role of cerebral oximetry as an index for systemic oxygen balance is further supported by several studies that show changes in cerebral oxygen saturation correlate with changes in pulmonary artery oxygen saturation (9,10). Murkin and colleagues also demonstrated improved length of stay outcomes in diabetic patients when cerebral desaturations were treated based on a preset protocol (11). Similarly, Casati and colleagues demonstrated a shorter hospital and recovery room stay when cerebral saturations were maintained at 75% of baseline by following a preset algorithm (7). Thus, when cerebral saturations, using the INVOS device, are maintained above 75% of baseline using a preset algorithm (see Table), there may be a decrease in adverse outcomes and length of stay in cardiac surgical patients.

In summary, the development of a neurological monitor capable of detecting ischemic events during cardiac surgery is long overdue. Emerging evidence suggests that cerebral oximetry may be capable of detecting ischemic events, guiding therapeutic interventions, and possibly reducing the incidence of neurological and systemic insults during cardiac surgery. Although most of the research discussed used the INVOS product, data from other products may be forthcoming, and more definitive studies still need to be performed.

Table: Cerebral Desaturation Treatment Algorithm

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<tbody>
<tr>
<td>1.</td>
<td>Increase inspired oxygen to 100%.</td>
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<tr>
<td>2.</td>
<td>Check head and cannula position to ensure adequate venous drainage.</td>
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<td>3.</td>
<td>If PaCO2 &lt; 40 mmHg, increase PaCO2 to &gt; 40 mmHg.</td>
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<td>4.</td>
<td>If MAP &lt; 50, increase MAP to &gt; 60 mmHg.</td>
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<td>5.</td>
<td>If hematocrit &lt; 20%, transfuse PRBC’s.</td>
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<td>6.</td>
<td>If none of the above interventions improve cerebral saturation, decrease cerebral oxygen consumption by increasing anesthesia depth.</td>
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References can be found on www.scahq.com
References


