Cerebral Oximetry During Cardiac Surgery
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Introduction
Multiple etiologies are responsible for stroke and neurocognitive dysfunction following cardiac and non-cardiac surgeries, which are broadly divided into embolic- and perfusion-related insults. Epiaortic ultrasound, transcranial Doppler and screening carotid ultrasonography have all been used to reduce the incidence of perioperative neurologic injury associated with cardiac surgery. Larger arterial vessels (aorta, middle cerebral and carotid arteries) are targeted for embolic sources of cerebral injury using ultrasound technology. Cerebral oximetry monitoring with near-infrared spectroscopy complements these ultrasound techniques by targeting the cerebral microcirculation through cutaneous determination of regional oxygen saturation (rSO₂). Because cerebral tissue (with its limited oxygen reserve) is sensitive to changes in oxygen delivery, cerebral oximetry monitoring may also serve as an index of perfusion and decreased oxygen delivery to other major organs.
The INVOS™ cerebral/somatic oximeter provides clinicians with a trend of intraoperative regional cerebral oxygenation. The data obtained from rSO2 monitoring may be used to reverse decreasing cerebral perfusion and avert prolonged ischemia of the brain and other major organs. This article highlights an intraoperative case that demonstrates the sensitivity of this monitoring device when surgical and other factors impact upon cerebral perfusion.

Case
The accompanying figure displays the regional oximetry trends of an adult patient with a history of transient ischemic attack, who presented for coronary artery bypass grafting and closure of a patent foramen ovale. Baseline regional cerebral oximetry values were 69 and 70, which yielded a desaturation threshold of 55 (20% below the baseline). Oximetry values were maintained above this threshold throughout the prebypass period, despite a downward trend 20 minutes after induction of anesthesia that was reversed with appropriate hemodynamic management.

The desaturation threshold was breached after initiation of cardiopulmonary bypass (CPB) due to decreased pump flow associated with impaired venous drainage. A hole in the vena cava was discovered and repaired by the surgeon. Venous return to the CPB machine improved and rSO2 measurements increased (see graph). These changes were coincident with an increased PaCO2, which likely contributed to increased rSO2 because of cerebral vasodilation. In response to the PaCO2 value of 59 mm Hg, oxygen sweep (or CO2 removal via CPB) was increased and regional cerebral oximetry values subsequently declined slightly over the next 15 minutes. A PaCO2 of 31.7 prompted a decrease in oxygen sweep. Regional cerebral oximetry values remained constant throughout the remainder of CPB, but trended higher after CPB and were in the 70s at the end of the surgical procedure.

Discussion
This case illustrates the value of cerebral oximetry monitoring during cardiac surgery and the association between rSO2 values and physiologic and hemodynamic changes. This patient began with relatively normal rSO2 values and maintained symmetry in cerebral oximetry monitoring throughout the case. Lower flows during cardiopulmonary bypass and lower PaCO2 led to decreased rSO2 values bilaterally, while higher CPB flow and higher PaCO2 led to increased rSO2. Lower rSO2 values were associated with impaired venous drainage and reduced CPB flows, and guided therapeutic intervention towards improving hemodynamic perfusion and manipulation of PaCO2.

The patient had no intraoperative episodes of asymmetric rSO2 changes that would have suggested an embolic or mechanical effect. An asymmetric or unilateral change in regional oxygen saturation generally indicates reduced oxygen delivery to one cerebral hemisphere. Mechanical problems that cause unilateral changes in rSO2 include malposition of the aortic perfusion cannula used for cardiopulmonary bypass, cerebral embolism, aortic dissection and axial head rotation that compromises unilateral carotid blood flow in patients with carotid artery disease.

Recent evidence suggests that maintenance of rSO2 above 75% of baseline results in a lower incidence of major organ morbidity, defined as cerebral vascular accident, renal failure, deep sternal infection, prolonged mechanical ventilation, reoperation or perioperative death. Our approach is to initiate a systematic process that maintains or increases rSO2, values based on the most likely etiology. For example, decreases in rSO2 values that occur before cardiopulmonary bypass are assessed in terms of hemodynamic perfusion. Cardiac function is evaluated using transesophageal echocardiography and measuring thermodilution cardiac output. Therapy for improving tissue oxygenation is then directed at increasing systemic vascular resistance or cardiac contractility depending on the assessment of ventricular function. Decreased rSO2 that occurs during cardiopulmonary bypass is generally treated by increasing pump flow and/or systemic vascular resistance, as indicated.

If changes in hemodynamic perfusion do not improve rSO2 values, an arterial blood gas is analyzed to determine if an increased PaCO2 is warranted. Mechanical ventilation (pre/post bypass) or oxygen sweep (during bypass) is adjusted to maintain the PaCO2 greater than 40 mm Hg. A BIS™ monitor (Covidien) is used to assess anesthetic depth. Measurements approaching 60 are appropriately treated by increasing anesthetic depth or administering muscle relaxants. These maneuvers increase rSO2 by decreasing oxygen consumption.

Finally, hemoglobin concentration is measured and red blood cells are transfused if rSO2 values do not improve with manipulation of blood pressure, cardiac index, PaCO2, anesthetic depth and muscle relaxation. These aspects of patient management were all addressed during this case, and rSO2 values near baseline were achieved after cardiopulmonary bypass before leaving the operating room, as indicated in the accompanying figure. The patient had an uneventful recovery with normal neurocognitive function after surgery.