Cerebral Oximetry

Monitoring the Brain as the Index Organ

Based on a prospective evaluation of 1,178 consecutive adult patients undergoing on-pump cardiac surgery, in this issue of Anesthesiology, Heringlake et al. present compelling evidence that baseline cerebral oxygen saturation (ScO2) is an independent risk factor for 30-day and 1-yr mortality. It is noteworthy that in this population, failure of oxygen supplementation to increase ScO2 beyond a cut-off value of approximately 50% (ScO2<50%) indicated the potential for significantly higher 30-day morbidity and mortality than in oxygen responders. In the highest risk group (i.e., additive EuroSCORE more than 10), it was found that ScO2<50% was a more accurate predictor of 30-day mortality than the EuroSCORE. In the overall study cohort, the accuracy of basal preoperative ScO2 for predicting 30-day and 1-yr mortality was equivalent in both N-terminal pro-B-type natriuretic peptide and high-sensitivity troponin-T, sensitive biomarkers that recently have been significantly associated with the incidence of long-term cardiovascular death and heart failure in patients with stable coronary artery disease.2

Of note is the somewhat less than intuitive fact that in this study, ScO2<50% was correlated with all-cause morbidity and mortality rather than reflecting primarily cerebrovascular risk. Paradoxically, this sensitivity of ScO2 to systemic morbidity encapsulates what is both a potential strength and a limitation to most current cerebral oximetry monitoring in that it reflects regional cerebral tissue oxygen saturation rather than measuring cerebral arterial oxygenation exclusively.

Because cerebral autoregulation reflects the coupling of cerebral oxygen delivery to cerebral metabolic rate and occurs primarily via modulation of cerebral blood flow in the presence of decreased cerebral arterial oxygen content, whether due to hypoxemia or moderate hemodilution (cerebral arterial oxygen content > 12 mg·100g−1·min−1), cerebral oxygen delivery is maintained by proportionate increases in cerebral blood flow.3 As ScO2 is weighted for approximately 70–75% venous blood, reflecting regional tissue saturation in an area of frontal cortex approximating 1 cm3 in the presence of stable cerebral arterial oxygen content, decreases in ScO2 reflect an increased oxygen extraction ratio and are indicative of a relative decrease in cerebral perfusion.

For example, in the absence of other compensations, a 15% decrease in cerebral oxygen delivery—whether resulting from decreased cerebral blood flow, uncompensated anemia, or some other such combination—would result in a greater than 20% relative decrease in ScO2. Accordingly, because there are physiologic mechanisms to preserve cerebral blood flow at the expense of relative systemic hypoperfusion, the presence of low ScO2 may thus reflect significant systemic circulatory compromise. This was illustrated graphically in a recent case report in which variable cardiac output during implantation of a left ventricular assist device was reported as being tracked more promptly and sensitively by changes in ScO2 than by continuous cardiac output monitoring.4 It was also the underlying concept of our previously reported randomized trial in which it was demonstrated that actively limiting intraoperative decreases in ScO2 in cardiac surgical patients resulted in a significant reduction in overall systemic morbidity and mortality.5 Similar to the results of Heringlake et al., in that study, it was also observed that patients experiencing major complications had a baseline ScO2 that was significantly decreased than those without, although the threshold of ScO2<50% was not specifically examined.

One challenge of cerebral oximetry is the fact that perturbations in ScO2, although highly sensitive, are conversely relatively nonspecific. It is for the clinician to determine whether a decrease in ScO2 reflects a derangement of systemic perfusion, regional cerebral hypoperfusion, relative hypoxemia, increased cerebral metabolic rate, or some other such combination of factors. This is the approach that was used in the algorithm developed by Denault et al., outlining a systematic and physiologically based approach to managing decreases in ScO2.6

It should also be of note that the correlation of low baseline ScO2 with systemic morbidity and mortality, as demonstrated here in cardiac surgical patients, is not completely unanticipated. In various other studies, the diagnostic and prognostic import of baseline ScO2 has been investigated in both surgical and nonsurgical cardiac patients.

In a preoperative study of 33 patients with valvular heart disease who were contrasted with healthy age-matched volunteers, it was demonstrated that a decrease in ScO2 during exercise corresponded with other markers of impaired cardiopulmonary function.7 That the decrease in ScO2 in this group occurred despite preservation of cutaneous earlobe saturation is further corroboration that ScO2 is primarily a measure of cerebral tissue rather than cutaneous scalp oxygenation. In a large longitudinal study of 344 patients with demonstrated coronary artery disease, a similar decrease in

Accepted for publication September 6, 2010. The authors are not supported by, nor maintain any financial interest in, any commercial activity that may be associated with the topic of this article. Dr. Murkin is a consultant and has received lecture/travel fees from Somanetics Corporation and Nonin Medical but has no stock equity or other such financial interests.

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ScO₂ during exercise was significantly correlated with long-term adverse outcomes, including worsening heart failure and sudden cardiac death, throughout the ensuing 3- to 4-yr period. The correlation among ScO₂, cerebral symptoms, and cardiac failure also was examined in a small study of nine patients with heart failure in whom it was demonstrated that decreased ScO₂ was associated with adverse cerebral symptoms, including dizziness and drowsiness, and in whom subsequent treatment of heart failure was associated with increased ScO₂ and improvement of cerebral symptomatology.

Although numerous previous investigations, including retrospective, case control, and prospective studies, have demonstrated a correlation between cumulatively decreased intraoperative ScO₂ and adverse outcomes, few have examined the prognostic import of basal ScO₂. In a study of 20 neonates without preexisting brain damage undergoing arterial switch operations, decreased preoperative ScO₂ was found to be associated with a longer interval until normalization of postoperative ScO₂ and decreased Developmental Quotient scores at 30–36 months. A study of baseline ScO₂ in 143 infants and children undergoing repair of congenital heart defects demonstrated that perioperative death was associated with baseline cerebral saturation less than 50% and that low baseline ScO₂ was predictive of perioperative mortality.

As such, the study by Heringlake et al. raises a number of intriguing questions. If the observations of this study can further be confirmed in other settings, could baseline ScO₂, particularly if it is refractory to oxygen therapy, be employed as a sensitive, yet quick and simple addition to the preoperative assessment of the patient undergoing noncardiac surgery analogous to its role demonstrated here in cardiac surgical patients?

In a study by Denault et al. of cardiac surgical patients, baseline ScO₂ was shown to have better sensitivity and specificity to predict abnormal cardiac function than hemodynamic variables determined by pulmonary artery catheterization. Could ScO₂<50% therefore be used as a threshold for the use of such invasive pulmonary artery catheterization monitoring in at-risk noncardiac surgery patients? Furthermore, could baseline ScO₂<50% act as a preemptive marker for patients requiring more intensive monitoring and expectant care in the postoperative period, thus enabling previous planning and mobilization of necessary resources? In addition, perhaps most noteworthy, as suggested by some reports, would early preoperative treatment of decreased ScO₂ enhance postoperative recovery and decrease perioperative morbidity? Although such speculations are currently beyond the evidence presented here, the report by Heringlake et al. is a provocative step toward that direction.

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References


