Correspondence

References


(Accepted for publication January 25, 2000.)

Jugular Bulb Hemoglobin Oxygen Saturation Is Not a Standard for Comparison with Cerebral Oximetry in Head Injury

To the Editor—Minassian et al.1 studied the correlation between cerebral cortical oxyhemoglobin saturation and jugular bulb oxyhemoglobin saturation in patients with head injury, and concluded that, "ScO2 assessed by NIRS does not adequately reflect changes in SvO2 in patients with severe head injury." However, jugular bulb oximetry may not be an appropriate standard of cerebral oxygenation measurement in patients with head injury. The transverse sinuses are fed by both hemispheres. Jugular bulb oxyhemoglobin saturation may tell us little except if this value is low, in which case it would indicate cerebral ischemia or hypoxia. However, if the value is normal or high, one cannot be sure that a large part of the brain is not hypoxic or ischemic. The situation in global phenomena such as global cerebral ischemia, encephalopathies, and cardiopulmonary bypass may be different.

If one samples blood from the right jugular bulb and perfusion to the right hemisphere is completely arrested, which may occur with the use of a carotid clamp during carotid endarterectomy, the blood must come from the opposite hemisphere. If there is no perfusion to the brain, the blood will likely enter the catheter in a retrograde fashion. In addition, because the near-infrared spectroscopy (NIRS) is a regional measurement, what can the expectations be in head injury? There is also the problem of microvascular shunting, which is discussed in an excellent review by Ince and Sinadsappel2 who used a combination of methods to demonstrate microvascular shunting in sepsis and shock.

We evaluated NIRS monitoring in the intensive care unit in children with head injury.3 Fluctuations in intracranial pressure that were in phase with cerebral cortical oxygenation suggest a vasodilatory process. If these fluctuations are out of phase with cerebral cortical oxygenation, the development of edema and vascular compression is indicated. Cerebral oximetry, whether in the operating room or in the intensive care unit, necessitates thoughtful consideration of all physiologic factors. Where possible, one should try to understand the injury by viewing the computed tomography scan and noting where contusions and accumulations of fluid or blood and regions of infarction are located to avoid placement of the sensors over those areas. The sensor may be placed over the at-risk area of the brain and the progression or subsidence of the disease with therapy may be followed. The sensor may then be used to optimize temperature, arterial blood pressure, and other variables. Finally, spontaneous fluctuations in cerebral oxygen availability with fluctuations in arterial pressure can provide insight into the quality of cerebrovascular autoregulation in the brain region that is being examined.

Edwin M. Nemoto, Ph.D.
Professor
Department of Neurological Surgery
University of Pittsburgh School of Medicine
Pittsburgh, Pennsylvania
nemoto@neuronet.pitt.edu

References


(Accepted for publication February 10, 2000.)

In Reply:—We thank Dr. Nemoto for the interest that he brought to our study of the correlation between cerebral cortical oxyhemoglobin saturation and jugular bulb saturation in patients with head injury. We do not agree that jugular bulb saturation measurement is not a standard of cerebral oxygenation assessment in patients with head injury. Because of the pioneering work of Robertson et al.,1 assessment of the global adequacy of cerebral blood flow to metabolism by jugular oximetry has shown the ability of this measurement to help clinicians avoid global cerebral hypoperfusion. This is especially true when therapeutic maneuvers, such as hyperventilation, are used to control refractory intracranial hypertension.2,3 We are aware that this measurement is a global one; therefore, a normal jugular oxygen saturation

Anesthesiology, V 92, No 6, Jun 2000
value cannot rule out the possibility that some area in the brain may be hypoxic or ischemic. We agree that this is a major limitation of the method. However, the same criticism could be brought against the measurement of cerebral oxygenation by methods that monitor a very limited cerebral area, such as near-infrared spectroscopy (NIRS) or direct cerebral oxygen measurement with microdialysis probes. In practice, clinicians would benefit from simultaneous measurements (i.e., global and local) in patients with head injury.

The aim of our study was to highlight the difficulties inherent in the interpretation of NIRS data in patients with head injury. We purposely studied conditions in which cerebral blood flow could reasonably be expected to vary in the same direction in the territories that were simultaneously monitored using three methods: NIRS, transcranial Doppler ultrasonography, and jugular oxygen saturation. Among the multiple reasons that may explain the discrepancies that were observed and discussed in our study, the clinical setting is an important reason to consider. Indeed, measurement of light transmission by NIRS may be more difficult to perform in adults than in children or in head trauma with edema than during carotid clamping. A partition between intracranial and extracranial blood and also between arteriolar and venous compartments seems to be dependent on the different therapies that are used. It may explain why clinicians who work in different domains obtain NIRS data with different levels of accuracy and clinical relevance. Furthermore, improvement in NIRS technology and in the modeling of the light pathway through an adult skull should allow for the identification of the mechanisms that underlie the discrepancies that we observed between different monitoring techniques, and may find solutions that will correct for these discrepancies.

Aram Ter Minassian, M.D.
Staff Anesthesiologist
Laurent Beydon, M.D.
Associate Professor
Department d’Anesthésie
Centre Hospitalier Universitaire Larrey
Angers Cedex, France
lbeydon.angers@in vivo.edu

References

(Correspondence accepted for publication February 10, 2000.)

Cognitive Impairment: Yes or No?

To the Editor—The article by Williams-Russo et al.1 and the editorial by Raja and Haythornthwaite2 imply that hypotensive anesthesia for total hip replacement (THR) is safe in terms of long-term cognitive impairment. The editorial states that, "the lack of any short or long-term cognitive impairment in the study by Williams-Russo et al. with the use of hypotensive epidural anesthesia is encouraging," and that, "no declines in cognitive function were found in an elderly normotensive population." In the Discussion, Williams-Russo et al. state that, "Complication rates after elective THR observed in the study of high-risk patients are similar to or lower than those of previous studies of elective hip and knee replacement in unselected patients receiving normotensive anesthesia." Unfortunately, this statement does not translate to the lack of any short-term or long-term cognitive impairment. Williams-Russo et al. cite their own previous work with bilateral total knee replacement that used the same battery of neuropsychological tests.3 The 1995 study showed that 5% of patients had ‘clinically significant cognitive impairment’ at 6 months. In the Results, the authors included not only baseline, 1-week, and 6-months scores for each test, but also include the percentage of patients with a decline in score from the baseline that is worse than the minimally clinical important difference for that test, a difference that they had defined. This was most helpful when interpreting the data because the standard deviations of the means for the tests were very large. This clinical important difference data were not included in their most-recent study in which, once again, the standard deviations are large in relation to the means, which make comparison extremely difficult. I would be interested in seeing this data (if available) and to know what percentage of patients in the hypotensive groups had clinically important cognitive impairment according to their previous definition, to compare this number with the 1995 incidence of 5%. Is this impairment similar or is it lower? Do the authors think that these patients did better than the patients in their previous study? If so, could they please comment on why this should be so? The conclusion of Williams-Russo et al. that there was no difference in early and late-term cognitive, cardiac, and renal complications in elderly patients between the two hypotensive groups for THR seems appropriate, given the data that were presented. As for the safety of total joint replacement in terms of long-term cognitive impairment, as implied by Raja and Haythornthwaite’s editorial, a 5% incidence of this complication is hardly encouraging, regardless of whether the anesthetic is normotensive or hypotensive. If this incidence is lower with hypotensive anesthesia, this observation certainly deserves an explanation.

Gerald Edelist, M.D., F.R.C.P.(C)
Professor
Department of Anaesthesia

Anesthesiology, V 92, No 6, Jun 2000