CORRESPONDENCE

References


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 assessment 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.1 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 sensors may then 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.

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References


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

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