**NIRS: A trustworthy guide through deep hypothermic circulatory arrest?**

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During complex aortic arch repair, surgical access may require interruption of systemic perfusion for relatively protracted periods. While moderate (25°C – 30°C) and deep (<25°C) hypothermia remain a mainstay for cerebral and systemic protection, there is relatively little ability to monitor cerebral well-being during such times since electroencephalogram (EEG) becomes progressively attenuated below 25°C. Accordingly, cerebral near infrared spectroscopy (NIRS) has been advocated to understand some of the cerebral responses to deep hypothermia and as a means of monitoring and detecting onset of cerebral ischemia during deep hypothermic circulatory arrest (DHCA).[1,2] While some groups monitor jugular venous oxygen saturation (SjO2) using retrograde cannulation of the internal jugular vein as an index of cerebral metabolic suppression during cooling, correlation has not been demonstrated between SjO2 and cerebral NIRS during DHCA,[3] likely indicative of the fact that NIRS is a highly regional measure of cerebral cortical oxygen tissue saturation (ScO2), whereas SjO2 is a measure of cerebral mixed venous oxygen saturation and thus reflective of global changes in venous oxygenation and, as such, potentially less sensitive to regional perfusion inhomogeneities.

In addition to DHCA, some centers utilize retrograde cerebral perfusion (RCP) via the superior vena cava or, increasingly, selective anterograde cerebral perfusion (SACP) via the innominate or subclavian artery. There have been a variety of case reports of the ability of cerebral NIRS to detect onset of cerebral ischemia during aortic arch surgeries and there is growing interest in the role of cerebral NIRS as a measure of adequacy of perfusion in this setting.[4-8] There is evidence that RCP may not provide sufficient nutritive flow to sustain cerebral integrity for an extended interval,[9] as has been reflected in lower ScO2 values seen during NIRS monitoring in RCP versus SACP.[10,11] More recently, however, utilizing cerebral oximetry and augmented RCP pressure, normalization of rSO2 was observed after increasing RCP perfusion pressure to 44 mmHg suggesting that such NIRS-guided perfusion management may improve substrate delivery during RCP.[12]

In a review of the role of NIRS monitoring during SACP, a study was undertaken in 46 consecutive patients in whom SACP was established by separate concomitant perfusion of the innominate and the left carotid arteries or by
perfusion of the right subclavian artery (with or without left carotid artery perfusion) and during which bilateral regional cerebral tissue oxygen saturation index was monitored by INVOS 4100 NIRS and which used stroke as the primary clinical end point, along with indices of diagnostic performance of the NIRS device.[13] In this series six patients died in hospital, and 6 patients (13%) in whom regional cerebral tissue oxygen saturation values were significantly lower during SACP experienced a perioperative stroke. Regional cerebral tissue oxygen saturation decreasing to between 76% and 86% of baseline during selective antegrade cerebral perfusion, had a sensitivity of up to 83% and a specificity of up to 94% in identifying individuals with stroke. It was concluded that using NIRS monitoring of regional cerebral tissue oxygen saturation during SACP allows detection of clinically important cerebral desaturations and can help predict perioperative neurologic sequelae supporting its use as a noninvasive trend monitor of cerebral oxygenation.[13]

In adult patients cerebral malperfusion can occur either as a consequence of kinking or obstruction of perfusion cannula during cerebral perfusion for circulatory arrest procedures, or due to migration of aortic endoclamp cannula during minimal access cardiac surgery with potential compromise of cerebral perfusion.[14,15] There are also increasing reports that bilateral rSO2 monitoring can detect contralateral desaturation during unilateral selective cerebral perfusion. This can result from an incomplete circle of Willis which in some series has a prevalence of up to 50% and has been estimated to be a factor in cerebral malperfusion in approximately 15% of patients.[16,17] In a more recent case report, cerebral rSO2 monitoring was utilized during selective cerebral perfusion in the absence of systemic CPB during repair of traumatic aortic arch rupture and detected both episodes of cerebral malperfusion and, most critically, acute thrombosis of carotid artery graft leading to thrombectomy and restoration of flow.[18]

In a recent study of 30 patients undergoing aortic arch surgery, intraoperative regional oxygen saturation values were recorded and analyzed and postoperative complications were collected and compared with the integrals of regional oxygen saturation and time (area under the threshold) spent beneath predetermined absolute threshold limits.[18] There were 30 major and 29 minor complications identified, and logistic regression showed statistically significant associations between area under the threshold and severe adverse outcome incidence for regional oxygen saturation thresholds of 60% (P = 0.038) and 65% (P= 0.025). Patients who spent more than 30 minutes under the absolute threshold of 60% had an extended hospital stay of 4 days leading to additional costs.[19]

In summary, it appears that non-invasive, continuous, bilateral cerebral oximetry monitoring using NIRS provides a reliable index of adequacy of perfusion during DHCA. As discussed here, recent evidence indicates that cerebral oximetry can
detect inadequate perfusion in real-time identified as significant decreases in cerebral rSO2 values that can often be ameliorated by identification of catheter malposition, inadequate perfusion pressure or incomplete bihemispheric perfusion. Failure to restore rSO2 values or sustained decreases below 60% are associated with adverse outcomes and prolonged hospitalization.

References:


