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"The Role of Non-Invasive Brain Oximetry in Adult Critically ill Patients without Primary Non-Anoxic Brain Injury"

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NIRS, a gadget or a must for critically ill patients?

Minerva Anestesiologica

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If we were able to define a common base for medical practice among neurointensivists, maintaining cerebral oxygenation would certainly be a major goal in neuro-intensive care patients. Several monitoring techniques have been developed to optimize cerebral oxygenation, from the less invasive (transcranial Doppler as a surrogate of cerebral blood flow) to the more invasive (intracranial pressure for monitoring cerebral perfusion pressure and brain tissue oxygenation probes). Each of these methods has its advantages and inconveniences, and the objective is probably to collect arguments to take the best possible treatment options. Among all these techniques, Near-infrared spectroscopy (NIRS) monitoring is particularly attractive because this monitoring technique is non-invasive, easy to use and gives continuous values that can be challenged against therapeutic changes. However, it is important to situate its place in the monitoring arsenal.

In year 2013, a systematic review suggested with a low-level evidence, that regional oxygen saturation index (rSO2) could be used to predict postoperative neurologic complications after cardiac surgery but data were insufficient to conclude that interventions that could improve rSO2 desaturation would be able to prevent stroke or postoperative cognitive dysfunction.[1] Eight years later what is the evidence on the use of NIRS to monitor the brain in the ICU ? The review by Badenes et al.[2] in this issue of the journal gives precise answers and perspectives on this technique.

First, all monitoring techniques are prone to errors and it is especially important to fully understand the limitations of NIRS monitoring to extract relevant information for clinical practice. A major problem is that different monitors give different values that cannot be compared. Several studies showed highly significant differences in local cerebral tissue oxygenation levels from one device to the other, making interpretation of studies with different devices difficult and hazardous.[3] In addition, extracranial contamination of the signal (despite unpublished algorithms used to minimize it) has been shown from the beginning of the monitoring technique and has long been reported as a limitation.[4] Also, significant bias and large variation in reading errors were found in healthy subjects submitted to hypoxia. This has been related to a fixed ratio of arterial and venous cerebral blood in the algorithms assumed by the manufacturers which is probably not true in human physiology during hypoxia.[5] Likewise, more than twenty years ago, the use of vasopressors as norepinephrine in traumatic brain injury patients was associated with a spurious decrease in rSO2 despite an increase in cerebral perfusion pressure and did not adequately reflect changes in jugular venous oxygen saturation.[6]

Some studies have been done in the ICU to determine if there was a correlation between rSO2 and brain tissue oxygen pressure (PbtiO2) for neuro-intensive care patients.[7] Even if several statistical approaches demonstrated direct correlation between rSO2 and

PbtiO2, the diagnostic accuracy of rSO2 to detect cerebral hypoxia was limited showing that NIRS is not a substitute of direct PbtiO2 monitoring. Moreover, a work using ^{15}oxygen positron emission tomography data showed that ischemia in TBI patient was common but not systematically associated with intracranial hypertension and inconsistently detected by jugular or brain tissue oximetry.[8] The high between- and within-patient cerebral circulation heterogeneity possibly reflecting abnormalities in brain flow-metabolism coupling makes clinical reasoning challenging in routine clinical practice.

Although there is no doubt that adequate cerebral oxygenation is important for preventing ischemia, the pathophysiology of brain ischemia is much more complex than oxygenation alone. Multiple other mechanisms leading to brain ischemia, not directly related to oxygenation, have been described. For example, cortical spreading depolarization, inflammation and apoptosis, microvascular injury, reperfusion injury and oxidative stress, blood-brain barrier disruption can all contribute to brain damage without involving alterations in rSO2. Finally, the heterogeneity of cerebral circulation changes and the multiple metabolic pathways leading to brain ischemia in most brain-injured patients explain that the interpretation of any brain monitoring technique, including NIRS, is not straightforward. NIRS is probably easier to interpret in cardiac surgery patients who have a non-injured brain before surgery.

All these limitations surely explain why majority of studies showing strong clinical application are in cardiac surgery, where the pathophysiological model of cerebral hypoxia is more pure and less complex.

However, a practical attitude can be extracted from all this literature. As no clear absolute threshold for ischemia is identified, NIRS monitoring should be used as a trend monitor with the assumption that there is no “baseline” value in critically ill patients. In any case, it is essential to keep in mind that it is needed to look at changes over time. A good example has been demonstrated in a recent study in post cardiac-surgical patients which showed a progressive postoperative decline in rSO2 from the day of surgery until day 7 with 97% of patients below baseline value at discharge.[9] A given value at one time point was not associated with clinical deterioration. Hence, it seems appropriate to set an alarm value on NIRS to look at the determinants of brain oxygenation in a dynamic manner. However, acute changes in rSO2 may give important information at the bedside. For instance, two studies showed the interest of using NIRS to detect intracranial hematoma in head trauma making it possible to manage this complication before cerebral herniation occurs.[10,11] Another example well explained by Badenes et al. is the use of NIRS to show dysfunctional cerebral autoregulation associated with delirium and outcomes in ICU patients.[12] For this purpose, NIRS seems easier to use than methods based on transcranial Doppler monitoring.

One of the lessons we can learn from the literature is that we certainly expect too much of our monitoring techniques. For example, a Cochrane database review did not find any clinical benefit of pulse oximetry in 22 992 patients enrolled in several studies with a good methodology. This does not mean we should not use pulse oximetry, but rather that it is difficult nowadays to demonstrate the benefit of a monitoring technique.[13] In this regard, the review from Badenes et al. gives updated information on how to use NIRS in critically ill patients, not as one more gimmick but as an added information to raise our attention on the patient's brain before it is too late.

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