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Editorial Comment

Cerebral Oximetry in General Anaesthesia

Anaesthesia is about trust

Human brain is the most wonderful thing in the world. Our brain makes us who we are; it makes humans unique, ensuring our personality, consciousness and abstract thinking. Patients undergoing general anaesthesia entrust this miracle to us anaesthesiologists. We have to serve this trust and give back this unique structure without any damage or malfunction as much as possible.

During general anaesthesia, anaesthesiologists have several tools for monitoring the status of brain. One of these options is cerebral oximetry, which estimates regional tissue oxygenation by transcutaneous measurement of the frontal cortex (1). Although pulse oximetry has been a standard monitor for decades and cerebral oximetry has evolved for over three decades and has been commercially available for over two decades, cerebral oximetry is not a routine monitor during general anaesthesia. Cerebral oximeters, like pulse oximeters, use the method of light transmission and absorption to measure the ratio of oxygenated to deoxygenated haemoglobin in the cerebral tissue (2). Nevertheless, there is a difference between pulse oximetry and cerebral oximetry. The technology of pulse oximeters allows simultaneous measurement of peripheral oxygen saturation by photoabsorption and pulse rate by plethysmography. The latter helps to differentiate between arterial and venous blood, and pulse oximetry reflects information about only the oxygen supply because it monitors only arterial blood. By contrast, cerebral oximeters use only near-infrared spectroscopy (NIRS) without plethysmography; therefore, cerebral oximetry does not indicate oxygen delivery, rather balance between regional oxygen supply and demand. Absence of plethysmography is a disadvantage and an advantage at the same time: measurements are independent of pulsatile flow; therefore, NIRS is useful during cardiopulmonary bypass also (3). As mentioned above, NIRS is helpful to monitor cerebral oxygen utilisation. Why is it important? Cerebral blood flow and cerebral blood volume are mainly influenced by the cerebral perfusion pressure and the cerebral vascular resistance. The most potent regulatory stimulus of the cerebral arteriolar tone (i.e. the determinant of cerebrovascular resistance) is carbon dioxide in the arterial blood. In cases of hypercapnia, a dilation of the cerebral arterioles occurs, leading to increases in both cerebral blood flow and blood volume. By contrast, if hypocapnia is present, a vasoconstriction of the cerebral arterioles occurs, resulting in decreased blood flow and blood volume. Hypocapnia could thus lead to circumstances where cerebral tissue hypoxia may develop due to the cerebral arteriolar vasoconstriction despite an appropriate arterial oxygen saturation measured by pulse oximetry (4). We have to note that hypertension and diabetes mellitus are proven to influence arteriolar reactivity. These altered reactions may play a modifying role in the accurate diagnosis of cerebral desaturation. However, aside from arterial carbon dioxide, several factors have an influence on cerebral oxygenation (5, 6). Cerebral oximeters measure tissue oxygen saturation, which reflects haemoglobin saturation in arterial, venous and capillary blood. In the cerebral cortex, average tissue haemoglobin is distributed in a proportion of 70% in venous and 30% in arterial blood. Change in distribution of haemoglobin in arterial and venous blood-such as during hematoma formation, hemodilution caused by bleeding or fluid therapy, or opening of arterio-venous shunts-has an influence on regional oxygen saturation (rSO2). Cardiac output, arterial blood pressure, arterial oxygen content, haemoglobin concentration, motion artefacts, neural excitation, anaesthesia depth, phenylephrine administration, non-haemoglobin chromophores (i.e. melatonin in the hair), and bilirubin in patients with jaundice also influence cerebral regional oxygen saturation. Nevertheless, skin colour and melatonin does not affect rSO_2 values (7-11). As previously Pollard et al. (12) and recently Saracoglu et al. (13) described the effect of head position on cerebral oxygen saturation and cerebral blood flow during general anaesthesia. As mentioned above, NIRS is not able to distinguish between arterial and venous haemoglobin saturation; changes in cerebral arterial-venous blood volume ratio, which may result from changes in blood flow or venous distending pressure, may have influence on measurements. Saracoglu et al. (13) reported that extension of head and neck during thyroidectomy negatively and gradually affects carotid

Address for Correspondence: Dr. Tamás Végh E-mail: veghdr@gmail.com

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blood flow and cerebral oxygenation, becoming pronounced especially at the end of the surgery.

This study indicates a new field of use of cerebral oximetry, which is widely used in neonatology; paediatrics; thoracic, vascular, cardiac, and neuroanaesthesia; and neurology (14, 15). The use of cerebral oximetry in carotid endarterectomy to diagnose cerebral hypoperfusion and determine which patients received selective shunting has been compared with electroencephalograph monitoring and transcranial Doppler. However, it remains unclear whether cerebral oximetry serves as a reliable clinical monitor in carotid endarterectomy (16). Cerebral oximetry has been also used in traumatic head injury patients. It has a good sensitivity for detecting intracranial hematomas correlating with CT scan (17). Several studies explored the use of NIRS in cardiac surgery. It has been shown that cardiac anaesthesia guided by cerebral oximetry, especially during cardiopulmonary bypass, significantly reduced mortality and morbidity and was associated with shorter length of stay in the intensive care unit. Active treatment of decreased rSO2 values has prevented prolonged cerebral desaturation and decreased incidence of postoperative cognitive dysfunction. However, other studies did not find correlation between outcome and cerebral oxygen saturation (18, 19). In the field of thoracic anaesthesia, several manuscripts described increased incidence of postoperative cognitive dysfunction if cerebral oxygen saturation decreased more than 20% compared with baseline value, measured before anaesthesia induction during one-lung ventilation (20, 21). However, if normocapnia was maintained during one-lung ventilation, avoiding both hyper- and hypoventilation, cerebral oxygen saturation was maintained above the baseline value, which is a ventilatory strategy that could prevent postoperative cognitive dysfunction (4). Routine cerebral oximetry monitoring during general anaesthesia might be useful in high-risk patients, although evidence that early detection of cerebral desaturation and targeted intervention could improve neurological outcome has thus far proved elusive. Patients undergoing beach surgery in the beach chair position, which can result in hypotension, might benefit from cerebral blood and oxygen saturation monitoring (22). Additionally, NIRS monitoring might be useful in elderly patients undergoing prostatectomy, where haemodilution and hypotension may occur, worsening cerebral oxygenation. However, use of phenylephrine to maintain mean arterial pressure also reduces rSO2 and is augmented by hypocapnia caused by hyperventilation (23). Previous papers reported that during gynaecological laparoscopic procedures in the Trendelenburg position, cerebral oxygen saturation decreases. Spinal anaesthesia also reduces cerebral oxygen saturation due to the hypotension that may occur (24). Laparoscopic cholecystectomy in head-up position also can lead to cerebral desaturation despite maintenance of mean arterial pressure above 80 mmHg (25).

Near-infrared spectroscopy technology and cerebral oximetry have limitations. First, there is wide intra- and interindividual baseline variability in regional tissue oxygen saturation. Normal range lies between 60% and 75%, with a coefficient of variation for absolute baseline values of approximately 10% (26). This indicates that cerebral oximetry is best used as a trend monitor, and claims of absolute thresholds for cerebral ischemia hypoxia should be treated with caution (27). Second, current commercially available NIRS devices are usually designed to be placed on the forehead and they are not able to detect changes in areas located deeply from the monitored site, although global cerebral oxygen sufficiency can be evaluated (28).

In summary, cerebral oximetry is a promising technology because it monitors essential and important parameters of the human brain. For responsible use, it is important to know how various physiological processes affect cerebral NIRS measurement. We have to note that success rate of intervention protocols, which have been proposed to correct cerebral desaturation, is poorly reported. Only a few randomised controlled trials have been conducted to test if cerebral oximetry-guided intraoperative intervention improves neurologic or composite outcomes. Even though the preliminary results seem promising, well-designed, large-scale, randomised controlled trials are needed to assess beneficial effects of cerebral oximetry on short- and long-term outcome (29). Despite lack of evidence, anaesthesiologists have to take care about patients' cerebral condition, and, according to their best knowledge, serve the patients' trust. Monitoring cerebral oxygen saturation might be a suitable tool for it (30).

Tamás Végh[1,2]

[1]*Department of Anesthesiology and Intensive Care, University of Debrecen, Debrecen, Hungary*

[2]*Outcomes Research Consortium, Cleveland, USA*

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