Global and regional techniques for monitoring cerebral oxidative metabolism after severe traumatic brain injury

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Disruption of normal mechanisms of oxygen delivery and metabolism is a hallmark of severe traumatic brain injury (TBI). In the past, investigations into the status of cerebral oxygen metabolism depended on changes in the differences in oxygen content between arterial and jugular venous blood. The development of jugular venous oximetry permitted continuous monitoring of jugular venous oxygen saturation, thereby overcoming earlier limitations caused by intermittent sampling. Neuromonitoring techniques that utilize only jugular vein sampling provide information only about global cerebral metabolism, but direct measurement of brain tissue oxygen tension via intraparenchymal probes makes possible the assessment of regional cerebral oxygen metabolism. Regional and global neuromonitoring techniques are not competitive or mutually exclusive. Rather, they are best regarded as complementary, with each providing valuable information that has a direct bearing on patient outcomes. The authors review the currently available techniques used in the monitoring of cerebral oxidative metabolism in patients who have sustained severe TBI.

KEY WORDS • arteriovenous oxygen difference • brain tissue oxygenation • jugular venous oxygen saturation • neuromonitoring • oxygen tension

GLOBAL CEREBRAL METABOLISM

Arteriovenous Difference of Oxygen

This technique for the monitoring of AVDO₂ is based on the difference in oxygen content between simultaneously obtained samples of blood entering and exiting the brain. Blood located anywhere within the arterial system (for example, the radial artery) can be sampled to determine the oxygen content of blood entering the brain. However, accurate sampling of blood exiting the brain requires cannulation of the jugular bulb, which contains only blood that has passed through the brain and has not yet mixed with that from the face, neck, and other extracerebral tissues.

The relationship between AVDO₂, CBF, and CMRO₂ (which is determined by the equation CMRO₂ = AVDO₂ × CBF) is illustrated in Fig. 1. If CBF is coupled with CMRO₂, then AVDO₂ remains constant as CBF changes. However, if CBF and CMRO₂ are uncoupled, then changes in CBF while CMRO₂ remains constant, are reflected as changes in AVDO₂.

The relationship between CBF and AVDO₂ becomes much less predictable when ischemia or infarct is present. These conditions can be detected by measuring the AVDL and using it to determine the lactate–oxygen index by the equation lactate–oxygen index = −AVDL/AVDO₂. A lactate–oxygen index greater than or equal to 0.08 indicates the presence of ischemia. Except during ischemia or other pathological events characterized by an extremely low CMRO₂, CBF has been shown to correlate well with AVDO₂. In severely head-injured patients with an AVDO₂ of less than 2.9 ml/dl, an average CBF value of 53 ± 18 ml/100 g/min has been demonstrated; in those with an AVDO₂ between 2.9 ml/dl and 6.8 ml/dl, an average CBF value of 42 ± 12 ml/100 g/min was shown; and in those with an AVDO₂ greater than 6.8 ml/dl an average CBF value of 23 ± 7 ml/100 g/min was found.

Despite the benefits of monitoring AVDO₂ and AVDL, these techniques have significant limitations. Sampling of cerebral oxidative metabolism in this manner can be performed only intermittently. The acquisition and analysis of these samples is labor intensive, a problem that grows in significance as the frequency of sampling increases.

Abbreviations used in this paper: AVDL = arteriovenous difference of lactate; AVDO₂ = arteriovenous difference of oxygen; CBF = cerebral blood flow; CMRO₂ = cerebral metabolic rate of oxygen; PbO₂ = brain tissue oxygen tension; SjVO₂ = jugular venous oxygen saturation; TBI = traumatic brain injury.

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Jugular Venous Oxygen Saturation

Several of the drawbacks of AVDO2 monitoring may be overcome by using continuous SjvO2 monitoring. This technique requires that a fiberoptic oxygen-sensing catheter be placed in the jugular bulb (Fig. 2). A drop in SjvO2 to below 50% for at least 10 minutes has been identified as a critical threshold. The development of such episodes correlates with a worsened outcome in patients who have sustained TBI, especially if they occur more than once during a patient's course of treatment. Episodes of jugular venous oxygen desaturation have been reported in 40% of severely head-injured patients. Importantly, most desaturation events last less than an hour, making it unlikely that they would have been detected by intermittent monitoring techniques such as AVDO2 measurement.

In comparison with monitoring of AVDO2, jugular venous oximetry offers the advantages of a continuous and real-time display of the parameter of interest. Both AVDO2 and SjvO2 monitoring require cannulation of the jugular bulb. In addition, SjvO2 monitoring is susceptible to artifacts caused by baseline drift of the catheter or by lodging of the sensor tip of the catheter against the wall of the vein. A drop in SjvO2 to below 50% requires that the possibility of such artifacts be eliminated by adjusting the position of the catheter and/or of the patient's head, as well as by checking the calibration of the catheter against a sample of jugular venous blood. Even if no episodes of jugular venous desaturation occur, SjvO2 catheters should be calibrated every 8 to 24 hours to minimize spurious readings caused by baseline drift.

REGIONAL CEREBRAL METABOLISM

Analysis of blood obtained from the jugular bulb provides a composite value of hemoglobin saturation for blood exiting the entire brain. However, in patients with severe TBI considerable regional heterogeneity of cerebral metabolism is often demonstrated. Global monitors such as jugular bulb catheters may not reveal the presence of regions of severely depressed metabolism if the abnormal metabolic profile generated by such areas is diluted and masked by the greater volume of blood flowing from relatively normal brain. Discrepancies in values obtained simultaneously from bilateral SjvO2 catheters have also been reported.

Brain Tissue Oxygen Tension

Fortunately, various techniques for monitoring regional cerebral metabolism have become available. Small catheters originally developed for direct and continuous monitoring of arterial blood PO2 soon became invaluable for monitoring PbtO2. These catheters are inserted directly into the brain parenchyma, in a fashion similar to that used for the insertion of fiberoptic or miniaturized strain gauge catheters for measuring intracranial pressure (Fig. 3). They may be inserted during a craniotomy or via a burr hole or twist-drill hole. One type of catheter, which has already been approved by the United States Food and Drug Administration, measures PO2, PCO2, pH, and temperature. Another type, which is used widely in Europe but has not yet been approved by the Food and Drug Administration, measures only PO2 and requires that a second small temperature probe be inserted to permit automatic temperature correction of the PO2 measurements.

Brain tissue oxygen tension values reflect the balance between oxygen delivery to the cerebral extracellular space and oxygen consumption by cerebral tissue. Reported "normal" and critical values vary depending on the type of catheter used. Analysis of data obtained from authors who used the European catheter suggests that "normal" PbtO2 may be approximately 35 mm Hg or higher and that a critical value for poor outcome or death may be approximately 6 to 10 mm Hg.
Cerebral metabolism monitoring after TBI

Although the major drawback of this technique is that the catheters must be inserted into the brain parenchyma, insertion-related hemorrhagic and infectious complications are quite rare. Moreover, the catheters seem to perform well, causing little baseline drift and further offsetting the potential risks associated with their insertion.\textsuperscript{1,12}

This stability of performance has led some investigators to suggest that PbtO\textsubscript{2} monitoring is superior to SjvO\textsubscript{2} monitoring for detecting ischemic events in head-injured patients.\textsuperscript{3,12} However, such investigations overlook the fact that SjvO\textsubscript{2} and PbtO\textsubscript{2} catheters differ fundamentally in the type of data each provides. The former measures global cerebral metabolism, as discussed above. On the other hand, PbtO\textsubscript{2} catheters sample only a very small volume of brain tissue, which makes them quite useful for obtaining regional measurements. If homogeneity of metabolism is an expected finding in the brain of a patient who has sustained TBI (for example, because the initial computerized tomography scan revealed no contusions or other sizeable lesions), then the PbtO\textsubscript{2} data can probably be interpreted as indicative of the status of global metabolism. However, if the probe lies near a contused area, then the data that it provides may reflect the metabolic status only of the area of abnormality, which may be very different from that found in most of the brain.

We have compared SjvO\textsubscript{2} and PbtO\textsubscript{2} as indicators of cerebral ischemia in 58 patients with severe TBI.\textsuperscript{3} Ischemic episodes were defined as all decreases of SjvO\textsubscript{2} to less than 50% and/or of PbtO\textsubscript{2} to less than 8 mm Hg. Brain tissue oxygen tension monitoring detected 64% of these episodes, and SjvO\textsubscript{2} monitoring detected 70%. Jugular venous oxygen saturation more consistently reflected a decrease in oxygenation from hyperventilation, whereas PbtO\textsubscript{2} was more sensitive to changes in arterial PO\textsubscript{2}. These data suggest that PbtO\textsubscript{2} and SjvO\textsubscript{2} monitoring are best considered as complementary, rather than competing, techniques. The regional nature of PbtO\textsubscript{2} monitoring is put to optimal use by placing PbtO\textsubscript{2} probes in tissue most vulnerable to ischemia but that may be salvageable with aggressive intervention. Several groups are currently investigating whether monitoring PbtO\textsubscript{2} combined with aggressive treatment of critically low values, especially during the first hours after injury, improves outcome after TBI.\textsuperscript{5}

CONCLUSIONS

Monitoring of cerebral oxidative metabolism has evolved from indirect and intermittent measurement of the amount of oxygen consumed by the whole brain to direct measurement of cerebral oxygen tension in specific areas of interest. Appreciation of the unique characteristics of the various currently available methods maximizes the usefulness of the information provided and may thereby help patients achieve the best possible outcome.

References


Fig. 3. Axial computerized tomography scan revealing placement of the PbtO\textsubscript{2} catheter (arrow) in the region of abnormal hypodensity.