Editorial

Influence of body temperature in autoregulation measured by cerebral oximetry index need more research

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Cerebral autoregulation (CA) is the mechanism responsible for maintaining a relatively constant cerebral blood flow (CBF) over a wide range of arterial blood pressure. CA depends on several mechanisms to maintain a suitable CBF based on cerebral metabolic demands. These mechanisms protect the brain from oligemia or hyperemia. Under specific conditions, the range of CA is severely compromised, increasing the risk of cerebral swelling (1).

Many authors have tried to determine the relation between CA and intracranial pressure (ICP) (1). Few researchers assessed the influence of fever and CA. This is the second study of this authors addressing the relationship between body temperature and CA using monitored using near-infrared spectroscopy (NIRS)-derived cerebral oximetry index (Cox) as monitorization (2).

Monitoring CA in order to optimize cerebral perfusion pressure (CPP) and mean arterial pressure (MAP) bedside is promising. It is still a matter of debate since CA depends on multiple factors such as the patient pathology, the use of vasopressors, sedation, levels of hemoglobin, cardiac output, temperature probe location, history of hypertension (shift to right) among other variables.

Hyperthermia is commonly seen in critically ill patients with different neurological injuries. Increased body temperature is detrimental on CA. The metabolic rate of oxygen consumption increases and therefore autoregulation is altered since CBF change. Monitoring fever and CA could be useful for making CPP and ICP decisions based on each particular patient moment (3).

Adatia *et al.* assessed the relationship between body temperature and CA in comatose patients from different etiologies. They hypothesized that CBF gets worse with elevated temperature (2). CA was monitored using NIRS-based Cox. This method has been validated in patients with acute neurological injury. The NIRS-based Cox and the mean velocity index from transcranial Doppler demonstrated moderate correlation and agreement (3).

It was a retrospective analysis the authors collected data from 85 patients. Patients in coma, Glasgow Coma Scale (GCS) ≤8 during a 4 years period of time, 2013–2017. The etiologies were intracerebral hemorrhage, subarachnoid hemorrhage, acute ischemic stroke, intraventricular hemorrhage, status epilepticus, meningitis, encephalitis, traumatic brain injury and post cardiac arrest. The monitoring was initiated between 12 and 48 hours after the event and continued during 72 hours. NIRS bifrontal and MAP were used. A Cox index close to 0 was considered improvement in CA and worsened if it exceeded 0.1. Patients were grouped according a temperature pattern, 11 without changes, 9 that increased, 9 that decreased and 56 with a fluctuating pattern. A significant difference between the monitoring times of each group is reported. Increased temperature impaired CA expressed by Cox. The effect could be seen both for the total group and for the multivariate analysis adjusted for PCO_2, hemoglobin, MAP, vasopressors, and use of sedation.

Efforts are still needed to evaluate the effect of temperature on CA in the different subpopulations of critical neurological patients. Besides, the hours of measurement might underestimate the impact of hyperthermia and hypothermia on CA. On the other hand, extensive ranges of temperature changes are proposed (4,5).

Temperature was measured in different locations by different methods. It would be advisable to use the same method in all patients since it is known that there are differences between invasive and non-invasive measurements, and even among the different locations.

Questions arise about whether changes in CA depend on the type of brain injury, if there is homogeneity in coma state or if it depends on etiology as cited. The cerebral metabolic rate is assumed to be constant (6). On the other hand, it is not possible to define how important is the role of the heterogeneity in the different neurological illness.

Different treatments modalities, i.e., sedatives, hypothermia, were used in these patients. Sedatives and hypothermia could affect in different ways. Midazolam and propofol sedation have different effects on CA despite causing equivalent decreases in CBF velocity. Only midazolam is probably to improve CA (7). These drugs could act as confounders. NIRS-based Cox, as part of multimodality monitoring, might therefore usefully be used to address influence confounding factors such as carbon dioxide tension, oxygenation, and cerebral metabolic rate (8,9). These factors affect cerebral physiology over time (10).

The measured area is regional, is it representative of the entire brain? We don't know the right answer, probably not. The influence of the probably opposite effects of the vasopressors on the brain and skin is still unknown.

In addition, the authors stated that this study wasn't designed design to assess clinical outcome, even though they found no differences between groups on mortality rates.

This study demonstrates the relationship between high temperature and worsening CA. A relationship between CA and prognosis cannot be established.

Probably, the response of CA to fever represents an epiphenomenon for primary brain injury.

No single method is regarded as the gold-standard technique for monitoring CA. It is interesting to include NIRS in multimodal monitoring. We still need to know if this manipulation influences the daily management in order to get a better result.

More research is required, prospective studies and larger samples to address each brain injury in particular and the response to temperature changes by monitoring CA.

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Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

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