Commentary

Cerebral perfusion pressure and brain ischaemia: can one size fit all?

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See related research by Marin-Caballas *et al.* in this issue [http://ccforum.com/content/9/6/R670]

Abstract

Current recommendations regarding the management of patients after traumatic brain injury include reduction in brain tissue pressure (i.e. intracranial pressure) and maintenance of an adequate arterial pressure; these measures combined should result in cerebral perfusion pressure sufficient to achieve adequate oxygen delivery. After almost 20 years of observational studies comparing cerebral perfusion pressure and indices of cerebral oxygenation, it is apparent that there is no single value for cerebral perfusion pressure that, if achieved, will provide adequate cerebral oxygen delivery in all patients. Traumatic brain injury remains a common problem, and this should encourage researchers and clinicians to design better and adequately powered trials of monitors and associated interventions.

In this issue of *Critical Care*, Marin-Caballas and coworkers [1] investigate the relationship between cerebral perfusion pressure (CPP) and brain tissue oxygen tension ($PtiO_2$). Identifying the optimal CPP following traumatic brain injury (TBI) is as crucial to best care for this vulnerable patient population as determining what is the best parameter (with acceptable sensitivity and specificity) for detecting impending brain ischaemia.

Marin-Caballas and coworkers investigated the relationship between CPP and $PtiO_2$ using an observational study design. The patients enrolled had suffered varying degrees of diffuse axonal injury, and six of the 22 patients underwent surgical evacuation of a mass lesion. Observation is always a good place to start exploring a research question when little is known about the subject. However, there are many data already available on $PtiO_2$ monitoring after TBI, and the relationship between $PtiO_2$ and physiological variables has been extensively investigated [2,3]. A randomized controlled trial could have been performed, because there is equipoise among clinicians and guideline authors as to what is the optimal CPP, and this might have allowed standardization of other patient parameters. It is not possible to address the relationship between CPP and $PtiO_2$ in only 22 patients, in whom spontaneous fluctuations in CPP (but mostly intracranial pressure [ICP]) were related to $PtiO_2$ measurements. An important confounding factor in this observational study is the patient management protocol, which mandates that when a low $PtiO_2$ is detected, all causes of this possible impending ischaemia should be corrected and CPP increased. Thus, low $PtiO_2$ is likely to be associated with lower CPPs, and higher $PtiO_2$ will be associated with higher CPPs solely because of the management protocol.

Although an impressive 1672 data points were recorded, these should have been analyzed per patient and summary data subsequently analyzed for all 22 patients combined or with all data presented for each individual participant. Many factors can influence $PtiO_2$, and many of them are documented in Table 1 of the report by Marin-Caballas and coworkers. There appears to have been little physiological stability in the patient cohort, making any interpretation of the relationship between CPP and $PtiO_2$ impossible; for instance, the core temperature ranged between 31°C and 39°C [4,5], ICP between 0 mmHg and 69 mmHg [6,7], and haemoglobin between 6.7 g/dl and 14 g/dl. With large fluctuations in ICP documented and relative arterial pressure stability described, the authors might have considered looking at the relationship between ICP and $PtiO_2$ rather than that between CPP and $PtiO_2$.

This commentary may appear somewhat critical, but it is worth emphasizing the potential for successful intervention to prevent critical reductions in $PtiO_2$, and Marin-Caballas and coworkers describe a protocol with the potential to achieve this aim. Mixenberger and colleagues [8] showed that, in patients who had suffered TBI, low brain tissue oxygenation

CPP = cerebral perfusion pressure; ICP = intracerebral pressure; $PtiO_2$ = brain tissue oxygen tension; TBI = raumatic brain injury.

was associated with a worse outcome on neuropsychological testing, especially with respect to executive function and memory. These patients also exhibited reduced ability to work compared with their preinjury level. These data suggest that there is possible predictive value of brain tissue oxygen for global functional recovery after head injury.

No clinical index will improve outcome on its own. The data generated by clinical indices must be incorporated into treatment protocols, which require development adhering to evidence-based medicine guidelines and then tested in a rigorous way. To date there has been no adequately powered intervention study (powered for outcome [9]) in TBI with which to modulate a monitored physiological variable that is closely associated with outcome, with outcome assessed as the primary end-point.

Current recommendations regarding the management of patients after traumatic brain injury include reduction in brain tissue pressure (i.e. ICP) and maintenance of an adequate arterial pressure; these measures combined should result in CPP sufficient to achieve adequate oxygen delivery. After almost 20 years of observational studies comparing CPP and indices of cerebral oxygenation, it is apparent that there is no single value for CPP that, if achieved, will provide adequate cerebral oxygen delivery in all patients. Thus, in order to minimize exposure to the risks associated with CPP interventions and to maximize benefit, clinicians must measure or assess cerebral oxygen delivery. $PtiO_2$ can provide this information and allows titration of ICP and mean arterial pressure interventions to a directly measured end-point and not a surrogate (i.e. ICP/CPP). TBI remains a common problem, and this should encourage us to design better and adequately powered trials of monitors and their associated interventions.

Competing interests

The author(s) declare that they have no competing interests.

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