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Near-infrared spectroscopy “under pressure” as a post-cardiac arrest monitoring technique of cerebral autoregulation

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A conceptual cornerstone of neurocritical care, including post-cardiac arrest (PCA) patients, is to mitigate the progress of hypoxic-ischaemic brain injury and minimize organ dysfunction [1, 2]. As in many other forms of neurocritical illness, it would seem intuitive that rapidly identifying and correcting brain ischaemia must play a major role in patient management [3]. Invasive measurement of intracranial pressure (ICP) or tissue oxygenation which enables estimations of cerebral autoregulation has been adopted in an approach to determine optimal mean arterial pressure (MAP) [4, 5]. However, the use of such monitoring and concomitant treatment strategies have not yet been shown to improve patient-centred outcomes [6]. Many studies are ongoing and eagerly awaited [6]. Invasive ICP monitors have not been used to any great extent in cardiac arrest patients [7]. The reasons for this are manifold and include the use of anticoagulation and targeted temperature management (TTM), that may increase the risk of bleeding and serious complications [2, 8]. Therefore, a non-invasive method to identify cerebral ischaemia, measure cerebral autoregulation and estimate individualized optimal blood pressures would be very appealing. Near-infrared spectroscopy (NIRS) provides a non-invasive method to measure cerebral oxygenation and, indirectly, cerebral blood flow [9]. The absolute regional cerebral oxygen saturation value (rSO₂) in itself does not appear to be associated with outcome in PCA patients [10]. Instead, observational studies have shown that NIRS enables identification of the limits of cerebral autoregulation and identification of an optimal MAP for each patient [11-13] using a cerebral oximetry index (COx). This optimal MAP may be different from the "one size fits all" haemodynamic targets recommended by guidelines and may be much higher in many patients [11, 14, 15]. As MAP is an important driver of cerebral perfusion, a higher MAP could alleviate cerebral ischaemia, but, thus far, the evidence of targeting a higher MAP in PCA patients appears inconclusive [16, 17].

In the current issue of *Resuscitation*, Hoiland and colleagues challenge the accuracy of NIRS derived COx as a means to estimate the limits of autoregulation in cardiac arrest patients and thus its accuracy for determining optimal MAP [18]. This renowned research group has published a series of experimental and clinical studies focusing on brain oxygenation [19, 20]. In this small observational study, cerebral autoregulation was measured as the moving correlation coefficient between ICP and MAP to calculate the pressure reactivity index, PRx, and non-invasively using rSO₂ and MAP to calculate COx. Using Bland-Altman plots, they studied the agreement between the estimated optimal MAP and the upper and lower limits of cerebral autoregulation. They found fairly good agreement between the optimal MAP identified by PRx or COx, with a mean difference of only 1.4 mmHg but with variability in agreement ranging from -23 mmHg to 25 mmHg. The upper and lower limits of autoregulation had even more variation and very poor agreement. Additionally, applying the commonly used cut-off value for detecting impaired autoregulation of >0.3 for COx and PRx, the sensitivity of COx to detect impaired autoregulation assessed by PRx was only 3%.

The authors must be congratulated for conducting an interesting and thought-provoking study that challenges the accuracy of NIRS as a means to measure cerebral autoregulation against PRx as the reference standard. Some issues need to be considered when interpreting the results, which are appreciated by the authors as well. The study included only 10 patients with different cardiac arrest aetiology and multiple measurements over time. Importantly, in the study there were notable delays in initiating ICP measurement. The study may also not fully appreciate the influence of actual oxygen and carbon dioxide levels on the accuracy of NIRS algorithms to derive rSO₂ and thus autoregulation measurements [21, 22]. Furthermore, differences in algorithms between NIRS monitors to derive rSO₂ were not assessed leaving questions related to external validity unanswered.

How should these findings guide the field of PCA care in the future? A nihilistic interpretation could be to abandon the use of NIRS altogether. But is the use of invasive ICP measurement to measure PRx really a realistic alternative in PCA patients? It has been used in small observational studies of PCA patients only and never as part of an interventional protocol. The question is also whether ICP is really elevated in PCA patients early during intensive care, as suspected or anticipated intracranial hypertension forms the necessary clinical rationale to institute ICP monitoring. Two studies conducted in PCA patients undergoing TTM suggest that this is not the case [2, 8].

Any strategy aiming at early estimation of optimal MAP and thereby tailoring treatment in PCA patients would need to be implemented soon after hospital or intensive care unit admission. NIRS combined with a strategy of systematically increasing MAP with a vasopressor while keeping oxygen and carbon dioxide constant could be a feasible and expeditious approach to estimate optimal MAP. This MAP could then be targeted during the following 48–72 hours. Based on the findings of Hoiland and colleagues, would it may be wise to set the NIRS-derived optimal MAP target high rather than low? [18]. One may debate whether identifying an optimal MAP would be enough rather than aiming to characterize the limit of autoregulation. The findings of Hoiland and colleagues notwithstanding, the lack of other non-invasive and safe alternatives should prompt more studies of NIRS, COx and optimization of MAP before this technique is scrapped altogether. Such studies and protocols will benefit from taking onboard the important findings presented by Hoiland and colleagues.

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