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INVITED COMMENTARY

Transcranial Doppler: The Bedside Window to Explore Cerebral Hemodynamics

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Cerebral perfusion pressure (CPP), the driving force for cerebral blood flow, is the difference between mean arterial pressure (MAP) and intracranial pressure (ICP). Under normal conditions, cerebral blood flow remains constant across a wide range of CPP values. This is called cerebral autoregulation. However, in severe central nervous system (CNS) injury, this protective mechanism may become impaired, leading to a pressure-passive state in the CNS vasculature, where cerebral perfusion becomes directly dependent on MAP. Thus, targeting CPP is a logical approach to mitigate secondary insults in cases of severe brain injury. Several guidelines recommend a specific CPP target of at least 40 mm Hg during severe pediatric traumatic brain injury [1].

To calculate CPP we need two variables: MAP and ICP. While the gold standard for direct ICP measurement involves invasive methods, these often require neurosurgical expertise and can impose a significant financial burden. Although these procedures are associated with low complication rates, potential risks include infection, hemorrhage, and brain tissue injury. Thus, non-invasive methods for assessing ICP have been explored. Various techniques like optic nerve sheath diameter measurement, transcranial doppler (TCD) and quantitative pupillometry are widely studied in the literature [2].

A standard ultrasound machine can be used to perform TCD. It is important to note that TCD measures the blood flow velocity, not the *blood volume*. However, several factors beyond blood flow can influence velocity measurements. Variables such as age, sex, fever, hematocrit level, and pCO2 significantly impact blood flow velocity. For example, conditions like anemia, especially sickle cell anemia, can lead to increase in the cerebral blood flow velocity. Similarly, elevated pCO2 level causes cerebral vasodilation, which also contributes to increased velocity. Cerebral blood flow velocity also varies with age. These factors must be carefully considered, when interpreting TCD data. Despite its advantages, TCD has some limitations. Its accuracy depends on the skill of the operator, and it is limited to measuring blood flow in major arteries. Therefore, it is unable to detect regional variations in blood flow.

In this issue, Kapat et al. [3] studied the CPP and cerebral hemodynamics in children with status epilepticus (SE) using TCD. The authors enrolled 42 children with SE who were not on any anti-epileptic drugs and TCD was performed within 30 min of seizure control. Patients who required more than two doses of benzodiazepines for initial seizure control were excluded from the study. Several TCD parameters, including mean flow velocity, diastolic flow velocity, and peak systolic velocity were compared between patients with SE and age and sex-matched controls.

During the seizure episode, there is continuous electrical discharge of the neurons leading to increased metabolic demands in the affected area. Experimental and human data documented a rise in cerebral blood flow during the seizure episode [4]. This cerebral hyperemia along with the edema due to blood–brain barrier dysfunction causes a rise in ICP during the seizure episode. But most of the time, the increase in ICP is transient and ICP returns to normal after the termination of seizures. Prolonged seizures and conditions such as pre-existing cerebral edema can impair the return of ICP following seizures to normal, resulting in a potential increase in ICP.

In this study, the peak systolic, diastolic and mean flow velocities, measured by TCD were high in the study group as compared to the control group. The mean (SD) ICP in the study and control groups were 9.81 (3.05) and 8.63 (1.88) mmHg respectively. Although statistically significant, the increase in ICP in the study group did not reach the threshold for any intervention. The authors excluded sickle cell

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anemia patients as it alters the CBF. The baseline pCO2 levels were also comparable between the two groups. The controls were appropriately selected to account for the variability of CBF across different age groups.

The authors estimated CPP non-invasively using the formula proposed by Czosynka et al., which is MAP × (diastolic flow (Vd)/mean flow (Vm)) + 14 [5]. Originally developed for adult patients with severe head injury, this formula has demonstrated a fair correlation with invasively measured CPP. However, the 95% confidence limit for CPP estimation is ± 21 mm Hg [5]. While this level of accuracy may be sufficient for tracking trends in CPP, it is not reliable enough to replace invasive ICP monitoring. Pediatric studies further highlight the limitations, showing a wider confidence interval of − 15 to + 38 mmHg, indicating that the true CPP could vary significantly from the estimated value [6, 7].

This study has certain limitations. As it is a single-centre study, the generalization of the results to other populations is limited. Most of the pediatric studies, regrading non-invasive CPP measurement were done in severe traumatic brain injury patients, where monitoring of ICP and CPP is critical for guiding interventions [6, 7]. In this study, the authors included children with convulsive SE and excluded those requiring more than two doses of benzodiazepines. The elevation of ICP during these electrical events is often transient and ICP returns to normal after seizure control, thus not warranting any specific therapy. Nevertheless, this study highlights the emerging role of TCD in the non-invasive measurement of CPP in the pediatric population and offers valuable insights into the cerebral hemodynamics associated with SE.

In conclusion, the authors should be commended for undertaking this study, given the significant challenges of performing TCD within 30 min of a seizure episode and the steep learning curve associated with this technique. Most of the alterations in cerebral hemodynamics during seizures are transient and typically do not warrant intervention unless the seizures are prolonged, or refractory, or accompanied by pre-existing cerebral edema. While non-invasive measurement of CPP via TCD shows promise, it cannot replace invasive ICP monitoring until further studies validate its accuracy. The formula used to calculate TCD is not well-established in children and may require a correction factor, as it is primarily based on data from adults. Nevertheless, non-invasive CPP measurement through TCD might be useful for monitoring in situations, where invasive ICP monitoring is either unavailable or unfeasible, without the risks associated with invasive procedures.

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