Editorial

Patient-specific intracranial pressure

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The accompanying article by Lazaridis et al. describes in detail a study challenging the conventional wisdom of traditional intracranial pressure (ICP) monitoring in the setting of traumatic brain injury (TBI). A number of recent studies have failed to show that controlling or reducing ICP below an absolute threshold results in improvement in neurological outcome or mortality rate. Prior reports have challenged the dogma of ICP monitoring as well. Still, most centers continue to treat posttraumatic brain swelling with control of the absolute ICP to values below 20 or 25 mm Hg, a practice that dates back more than 2 decades, with no supporting Level I evidence. In an age of increased capability for sophisticated monitoring of physiological parameters and neurochemical changes, traditional ICP monitoring continues to play a key role in the management of TBI, despite the mounting evidence questioning its value.

Changes in the way we practice require good evidence for an alternative strategy. It is refreshing, therefore, to see a study such as this one in our literature. Rather than proposing the use of additional, expensive, and untested measures, Dr. Lazaridis’ group has proposed an analysis of currently collected physiological parameters that instead poses the use of additional, expensive, and untested measures. Their approach is rooted in the observation that patients seem to fare better when ICP changes happen slowly and cerebrovascular reactivity remains normal. It has long been observed that patients with chronic increases in ICP (for example, due to slow-growing tumors, pseudotumor, and hydrocephalus) are often minimally symptomatic. The difference between such patients and those with more acute changes in ICP (such as those due to acute epidural hematomas) probably has to do with the lack of cerebrovascular compensation in the latter group. This study attempts to quantify this cerebrovascular reactivity in the setting of TBI, and to examine whether this index has a prognostic significance.

This is an elegant study, and appears to have prognostic value, as these authors show. The more difficult question to ask is whether correcting abnormalities in the derived measures would make a difference in outcome. It is possible that measures such as “ICP dose” and pressure reactivity index (PRx) are mere markers of a bad outcome and cannot be changed with intervention. Only further work looking at how these calculated ICP parameters correlate with other biomarkers and therapeutic interventions will resolve this important issue.

The calculated ICP parameters could also help us address why certain patients deteriorate when their abnormally high ICP is reduced too rapidly. It is well known that certain patients with long-standing intracranial hypertension actually get worse when their ICP is normalized. Once again, cerebrovascular reactivity changes are suspected in these situations. Physiological parameters such as ICP dose may help guide therapy in these situations, and prevent neurological sequelae.

The authors of this study are helping usher in a new age of physiological monitoring in the neurocritical care unit—one that goes well beyond traditional cardiopulmonary measures and absolute ICPs. Additional studies will further outline the utility, implications, and limitations of these cerebrovascular measures. My only regret is that the software that they have created is available only as a commercial product. Scientific progress and iterative improvements are always more robust when tools such as these are shared with other academic units.

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Disclosure

The author reports no conflict of interest.

References

Response

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We would like to thank Dr. Sagher for his favorable remarks on our article. Monitoring and management of ICP is a key feature of medical and surgical management in patients with severe TBI. In conjunction with optimization of cerebral perfusion pressure, the control of raised ICP has formed the cornerstone of brain trauma guidelines, and together these have led to a reduction in mortality from TBI. Nevertheless, the specifics of ICP monitoring and management have recently come under significant scrutiny in works that are referenced in both our paper and in Dr. Sagher’s editorial.

What then are the main criticisms against the traditional approach to intracranial hypertension? 1) The ICP threshold set at 20–25 mm Hg has resulted from observational studies and noncontrolled series comprising an overall low level of evidence for the validity of the chosen value. Furthermore, the methods used do not allow for differentiation between a potentially modifiable therapeutic target and a mere surrogate of injury severity. 2) Absolute thresholds ignore the variability of brain injury types and host characteristics and responses. They also mandate the same unvarying goal throughout a patient’s course. Clinical experience suggests that different patients tolerate different levels of intracranial hypertension depending on the clinical scenario; we believe that the state of cerebral blood flow (CBF) regulation is a critical qualifier of how “tolerable” a given ICP may be. 3) Secondary brain injury and insults are treated as unidimensional excursions over a certain number, whereas degree and time range of excursion are not considered. 4) A range of potentially important pathophysiological variables that describe the relationships among CBF, perfusion pressure, and oxygen delivery and utilization, as well as cellular metabolism and mitochondrial state remain unaccounted. 5) The therapeutic interventions that are used carry a price that potentially outweighs the benefits of achieving a fixed ICP goal.

Although this list may not be exhaustive, we have attempted to address some of the issues by quantifying secondary brain injury using the concept of a “dose” of intracranial hypertension. The use of multidimensional measures makes intuitive sense in quantifying injury burden, and offers the opportunity to investigate the contribution of individual components. Furthermore, we have used the PRx to individualize and “qualify” ICP insults; we found this index to improve mortality prediction over insults informed by the generic thresholds of 20 and 25 mm Hg. The value of PRx in individualizing the ICP threshold needs prospective verification. This approach could be further expanded with the incorporation of measures such as brain tissue oxygenation, microdialysis parameters, and/or direct CBF monitoring. Ultimately, the additional consideration of patient demographic information and brain imaging data can lead to the creation of patient-specific trajectories and physiological latent states, offering a more comprehensive picture in which ICP is a part and not the whole.

Finally, ICM+ software (www.neurosurg.cam.ac.uk/icmplus) is available under a very modest license fee from the University of Cambridge; this was triggered by the necessity for legal protection of the authors rather than for financial profit.

References


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