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INVITED EDITORIAL COMMENTARY

Brain Venous Blood Outflow

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Venous outflow has been examined as a potential descriptor of the etiology of various diseases such as migraines, tinnitus, multiple sclerosis, and Alzheimer's disease [1, 2]. However, results were often discouraging, with the exception of idiopathic intracranial hypertension, where a model of functional coupling between intracranial pressure (ICP) and sagittal sinus pressure (SSP) has been produced [3]. In this model, because of collapsible walls of transverse sinus, rise in ICP may compress sinus, rising sinus pressure which leads to rise in SSP, rise in ICP, further compression of sinus, etc. working as a positive feedback loop. Despite unequivocal evidence, cerebral sinus and venous stenting are readily used in clinical practice [4].

Neurocritical care management of jugular venous outflow in acute diseases (traumatic brain injury, subarachnoid hemorrhage, meningitis, etc.) is uncommon. Unnerbäck et al. [5] retrospectively compared the patterns of pulsatile blood flow in the interior jugular veins (IJVs) with mean ICP recorded in a group of patients, observing an inverse relationship between venous outflow pulsatility and ICP. However, the pulsatility of venous flow within the internal jugular veins was studied extracranially. Is this decrease in pulsatility transmitted from the intracranial compartment (secondary to a decrease in sagittal sinus flow pulsatility), or the heart (due to the decreased pulsatility of right atrial pressure [RAP])?

The association between high ICP and low IJV flow pulsatility does not include causality analysis. It may be that both intracranial hypertension and dampened IJV flow pulsatility stem from a factor which was not identified in this paper (i.e., the relationship between ICP and RAP can be a prime link). To examine causality, JIV ultrasonography can be employed when studying elevated ICP (i.e., during lumbar infusion studies in hydrocephalic patients [6]). Increases in ICP during such procedures influence a slight increase in arterial blood flow pulsatility [6], but the transmission pathway to the sagittal sinuses (and further distally, to the IJV) is unclear. Heightened ICP prompts a surge in mean arterial blood pressure and heart rate variability [6], but its effect on RAP has never been studied. Phase-contrast magnetic resonance imaging (PCMRI) [7] suggests that IJV flow pulsatility is largely dependent on RAP pulsatility and eclipses sagittal sinus flow pulsatility.

I believe that this work [5] is a fine introduction to combined MRI blood flow studies and ICP monitoring. In the presented material, both measurements are close in time, but not performed simultaneously. However, former work of the same authors [8] demonstrates this possibility with current technology. Bolstered by this landmark paper, the simultaneous study of pressure and flow can broaden the horizons in brain fluid dynamics. It is complex and intellectually challenging, but with an advent of 4D PCMRI [9] it may increase understanding of many neurological diseases.

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