Constricted venous outflow: Does it alter cerebrospinal fluid dynamics?

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Abstract
Few topics in medicine have in recent years produced such controversy as chronic cerebrospinal venous insufficiency (CCSVI). Since this vascular syndrome was first identified in 2009, it has divided opinion. On one extreme, are those who advocate that CCSVI causes multiple sclerosis (MS), while at the other end of the spectrum are those who deny that the syndrome exists at all. Consequently, the debate on CCSVI has become polarized – a situation, which all too easily, can result in a loss of objectivity.

So what is one to make of CCSVI? Are there any grounds for believing that it might be associated with neurological disease, or is the syndrome a complete irrelevance? Perhaps some insights can be gained from the world of fluid dynamics and biomechanics. Because there are no obvious moving parts, it is all too easy to ignore the biomechanics of the brain. Yet the intracranial space is a finely tuned dynamic mechanism, involving three fluids (blood, cerebrospinal fluid (CSF) and interstitial fluid), all of which interact with each other in a precise and orderly manner. Traditionally, the cerebral venous system has been viewed simply as a series of collecting vessels channelling blood back to the heart. However, recent research is revealing that the cerebral venous system plays an important role in regulating the intracranial fluid system and the dynamics of the CSF system.

Recently a new cervical plethysmography technique has been developed, which revealed the hydraulic resistance of the cerebral venous drainage system to be on average 63.5% greater in MS patients diagnosed with CCSVI compared with CCSVI negative healthy controls. This suggests that CCSVI is associated with mild venous hypertension (<5 mmHg) in the dural sinuses; something that would tend to inhibit the bulk flow of CSF, as some have observed in MS patients. Venous hypertension of this magnitude would also tend to reduce intracranial compliance, thus altering the dynamics of the CSF in the aqueduct of Sylvius (AoS). Indeed, a number of studies have revealed increased aqueductal CSF pulsatility in MS patients and CCSVI positive healthy controls; something which suggests that CCSVI is associated with altered CSF dynamics, irrespective of whether or not MS is present.

While there is evidence linking mild venous hypertension with changes in CSF dynamics, the clinical implications of these changes are unclear. It may be that increased CSF pulsatility in the AoS induces ventricular reflux and edema formation in the periventricular white matter, as has been observed in patients with normal pressure hydrocephalus. Given, that ventricular reflux has also been implicated with periventricular lesion formation in MS, there is need for further research to assess whether or not CCSVI is associated with CSF reflux from the lateral ventricles into the brain parenchyma.

References