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A model of communicating hydrocephalus based on the spatial and spectral redistribution of intracranial pulsations

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Background

Hydrocephalus has traditionally been understood as a disorder of the absorption of CSF. Recent work by Johnston, Cserr and others has challenged the traditional understanding of CSF absorption, and flow MRI studies of Greitz, Bateman, and others suggest that pulsatility plays a central role in the pathogenesis of hydrocephalus. Based on our recent experimental insight into the dynamics of intracranial pulsatility in dogs, and a new model of intracranial dynamics, we discuss a model of the pathophysiology of hydrocephalus in which ventriculomegaly is driven by the redistribution of pulsations in the brain.

Materials and methods

We model the cranium as a frequency-sensitive notch filter that suppresses the arterial pulse in the brain. This redistributes the kinetic energy of pulsatility at the heart rate (i.e. cardiac frequency) to smooth, pulseless arterial flow (i.e. zero frequency), which is the cerebral blood flow. This represents the normal spectral distribution of the transfer function between the arterial pulse and the ICP pulse, and is a manifestation of the normal cerebral windkessel mechanism.

Results

Disturbance of the normal compliance and resistance in the subarachnoid space, such as occurs in communicating hydrocephalus, alters the normal distribution of pulsatility in the cranium. This redistribution can have two distinct manifestations: 1) spatial redistributions in which pulsations are redirected from one part of the cranial cavity to another, such as increased ventricular pulsation at the expense of subarachnoid pulsation, and 2) spectral redistributions in which pulsations are redistributed between flow components at the cardiac frequency and alternate frequency components, such as the enhanced cardiac-related pulsatile flow at the expense of zero-frequency smooth flow. The loss of cerebral blood flow and augmentation of pulsatility is the main manifestation of an impaired windkessel mechanism, and leads to venous stasis, venous hypertension, and reduction in cerebral blood flow.

Conclusion

We propose that the salient features of clinical and experimental hydrocephalus can be explained as consequences of impairment of the cerebral windkessel mechanism, the mechanism by which the cerebral vasculature renders vascular perfusion of the microvasculature nearly smooth. Asymmetrical obstruction of the CSF pathways causes spa-

tial and spectral redistribution of vascular and CSF pulsations in the cranium. In this model, communicating hydrocephalus is fundamentally an impairment of cerebral blood flow and of the cerebral windkessel mechanism. Ventriculomegaly and CSF malabsorption are the consequences of this impairment. Based on this model, we can suggest significant new and non-intuitive approaches to the treatment of hydrocephalus.