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Evidence for CSF-vascular compliance coupling in normal pressure hydrocephalus

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Background

The underlying pathophysiology in Normal Pressure Hydrocephalus (NPH) most likely involves changes in brain parenchyma, vascular resistance, and CSF circulation resulting in decreased intracranial compliance and diminished cerebral blood flow (CBF). Changes in blood vessel reactivity may play an important role in the pathophysiology and diagnosis of NPH. As a result, it has been suggested that Transcranial Doppler (TCD), a non-invasive assessment of blood flow velocity in major intracranial arteries, may be useful in distinguishing specific blood vessel segments and reactivity patterns unique to NPH. The present study is a prospective, clinical trial of 30 patients undergoing extended CSF trial lumbar drainage for the diagnosis of NPH using standard TCD monitoring and newly developed Hemodynamic Vascular Analysis (HVA, New Health Sciences, Inc., and K.C.).

Materials and methods

In thirty patients between 53–90 y.o. (mean 72.2) who presented with ventriculomegaly and one or more of the triad symptoms (gait impairment, cognitive impairment, and urinary incontinence), TCD measures were obtained immediately before and after 36-hour, lumbar CSF drainage procedure for the diagnosis of NPH. TCD parameters (MFV, PSV, SA, and PI) as well as data converted to HVA through algorithmic calculations and fast Fourier transform were obtained from 23 vessel segments and/or ensembles of segments in 16 CSF drainage "responders" and 14 "non-responders." HVA parameters of flow dynamics included Hemodynamic Flow Index (MFV/PI), Hemodynamic Pressure Index (lnSA/PI), and Hemodynamic Compliance Index (lnSA/MFV). TCD-HVA values at presentation and percent change with CSF trial drainage for responders and non-responders were reported.

Results

Overall, we found significant differences between responders and non-responders for MFV and PSV at presentation ($p \leq 0.01$) and after CSF removal ($p \leq 0.05$). We also found that extended CSF drainage resulted in a change in pulsatility (PI) with functional response. These findings were further confirmed by HVA which showed a unique pattern of specific vessel sites (4 parameters) that was significantly different between responders and non-responders in combination and individually ($p < 0.017$); specifically decreased flow velocities in middle cerebral and vertebral arteries, and diminished compliance in the anterior cerebral artery were reported. This discriminate function was applied to the specific cohort and was able to classify treatment responders from non-responders with 81.8% sensitivity and 75% specificity.

Conclusion

Our findings showing a change in pulsatility (PI) associated with CSF drainage indicate the influence of the CSF

space on vessel reactivity. Further, the association between pulsatility and CNS function suggests that this influence may be clinically significant. The differences in mean and peak velocity at presentation and after CSF removal may reflect underlying deficits and recovery of blood flow. Although the initial identification of specific vessels and patterns associated with CSF trial response using TCD-HVA may be unique to NPH, these preliminary findings are not physiologically intuitive and must be validated in a prospective study in a new patient set.

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