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Hyperdynamic pulsatile flow and ventricular dilation in experimental communicating hydrocephalus

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Background

In communicating hydrocephalus (CH), where either there is no obvious physical blockage within the subarachnoid space, or the obstruction can be variable in location, explanations for the symptoms and clear-cut effective treatments have been elusive. A few investigators have begun to stress the importance of pulsatile vascular and CSF dynamics. While it is known that pulsatile flow through the cerebral aqueduct is often significantly elevated in hydrocephalus, a clear link between abnormal pulsations and ventriculomegaly has been yet to be established. The purpose of this study was to characterize the temporal changes in intracranial pulsatility in a novel model of CH.

Materials and methods

Kaolin (25%) was injected into the basal cisterns (n = 8) after anterior exposure of the C1-clivus interval. On days 1, 2, 8, 15, and 31, animals were scanned on a 9.4 Tesla magnet. Ventricular volume was based on CSF-bright 3D-TrueFISP images and aqueductal flow was assessed using a gradient echo phase contrast sequence.

Results

Animals developed ventricular dilation and increased aqueductal pulsations according to two very distinct patterns. Group 1 animals (n = 4) developed severe ventriculomegaly, which progressed steadily for most of the time period investigated. Aqueductal pulsations were also significantly elevated and increased with time in correlation with ventricular volume ($R2 = 0.75$, $p < 0.0001$). Ventricular volumes and pulsations increased to approximately 10 and 30 times normal, respectively. Group 2 animals (n = 2) developed mild ventriculomegaly, which changed very little after Day 1. Aqueductal pulsations were increased on Day 1 and remained so until Day 15, whereupon they returned to control levels and remained low. Ventricular volumes in this group increased to approximately 3 times normal and pulsations increased to approximately 6 times normal (before decreasing on Day 15). In both groups, ventricular volume and aqueductal pulsations were found to be elevated even on Day 1 post CH-induction.

Conclusion

These preliminary results suggest that basal cistern-induced CH is associated with an immediate change in ventricular CSF pulsatility. While the severely hydrocephalic animals demonstrate a clear connection between ventricular dilation and ventricular pulsations, the mildly hydrocephalic animals seem to indicate that this relationship is not strictly held. In fact, the late drop in pulsatility

may be indicative of a compensatory mechanism, which may help to control progressive ventriculomegaly.