

Toward a Better Understanding of Normal Pressure Hydrocephalus

I read with interest the commentary and extensive literature review published by Bradley (1), but wish to reply to some of the comments made regarding my article on normal pressure hydrocephalus (NPH) (2). I will discuss the comments made regarding control selection, cardiac gating, and the selective measurement of the superior sagittal and straight sinus flow as well as those regarding the cause of the venous compression and the physiology of CSF aqueduct flow.

The selection of a non-biased control group is important. The problem with investigating patients in their seventh and eighth decades centers on knowing what is normal. The complete lack of any cerebral pathologic abnormality is aberrant for an 80-year-old individual. Is the cerebral pathologic abnormality normally found in the average person of this age allowable in a control group? The second control group I used contained patients with atrophy, ischemia, or both. I believe that, although not physically "normal", these people are a better group for comparison.

It is true that prospective cardiac gating does not measure the final 100 milliseconds of the cardiac cycle, but systole was the main focus. The effect of prospective gating on the results was to slightly overestimate the mean blood flow because the end diastolic flow, which is missed, is lower than the mean. This has the effect of slightly overestimating the pulsations in all patients, but as a reduction in pulsations was found in the test patients, any attempt to allow for this effect would only have increased the findings, not diminished them.

The question of measuring whole-brain blood flow was addressed on page 1581 of the article (2). In addition, as the deep and superficial vascular territories seem to be affected in opposite ways by NPH, adding them together would blur the effect.

Bradley asks the question, "What causes the previously normal venous resistance to become elevated in elderly patients?" and goes on to mention the Monroe-Kellie doctrine (1). The Monroe-Kellie doctrine is the very reason that the venous resistance must rise if spinal canal compliance is reduced. If the intracranial volume is fixed and brain and arterial expansion occur, then either CSF must be expelled in equal volume or the venous compartment compromised. As the amount of CSF shifted from the cranial cavity in NPH patients is about half of that of similar patients with atrophy, the veins must be compressed by a similar amount. The flow through a vessel is dependent on the forth power of the radius; even a small reduction in cal-

iber can cause significantly raised resistance. Perhaps a better question would be: "Why is the spinal compliance altered"?

The physiology of the aqueduct flow involves a combination of cerebral expansion and the difference between superficial versus deep brain compliance. The total CSF leaving the incisura of the tentorium is approximately 330 μL (3) compared with the aqueduct flow of 30 μL (4). Both are occurring because of brain expansion. Given that the brain is essentially a hollow sphere and that curved surfaces resist compressive forces, it is understandable that when the brain parenchyma expands in systole, there is 10 times more outward expansion than inward compression. Expanding inward would compress the parenchyma adjacent to the ependyma. If in NPH the superficial parenchyma is not compliant, then a greater percentage of the pulsation must be directed toward the ventricles. Bradley surmises in late NPH that irreversible small vessel (probably arteriolar) occlusions occur (1). Because occluded vessels do not pulsate, the aqueduct flow would be expected to drop with irreversible vessel loss, irrespective of the state of spinal compliance. The problem is the associated atrophy. My article supports the view that in NPH there is underlying atrophy, which is masked by the increased interstitial fluid. The atrophic control patients without hydrocephalus all showed increased pulsations throughout their brain regions and in their aqueduct flows. Thus, small-vessel occlusion (ischemia) and loss of parenchymal volume with preserved vessel patency (atrophy) may have opposite effects on brain pulsations. As atrophy is masked by increased interstitial fluid in late NPH, the developing irreversible ischemia reduces aqueduct flow. If a patient with pure atrophy and no significant small-vessel occlusion is mistaken for a patient with NPH (as may occur in early Alzheimer disease), then the increased aqueduct flow may be misleading.

I agree with Bradley's final point: larger studies are required to verify my hypothesis that NPH is caused by venous compromise.

GA Bateman, MD

*Department of Medical Imaging
John Hunter Hospital
Australia*

References

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