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Patient Safety Tip of the Week

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Normal Pressure Hydrocephalus: Unrealistic Expectations?

Sixty years ago, our mentors Raymond Adams and C. Miller Fisher published a paper in the New England Journal of Medicine identifying normal pressure hydrocephalus as a potentially treatable condition (Adams1965). After that publication there was a flurry of activity to identify patients who might benefit from CSF shunting. But the number of patients who actually improved after shunting was disappointing and some shunted patients developed complications like subdural hematomas. This neurologist personally can only count on one hand the number of my own patients who improved significantly after shunting.

The clinical "triad" of normal pressure hydrocephalus (NPH) is dementia, incontinence, and gait disturbance. The syndrome is characterized by enlarged ventricles without obstruction to CSF flow and "normal" CSF pressure on lumbar puncture. The concept is a good one. The Law of Laplace states that wall tension in a spherical object or space is proportional to the pressure and the radius of the sphere. So, even though the CSF pressure measured by lumbar puncture is "normal", the tension exerted on the ventricular walls is elevated in the enlarged ventricles. Motor fibers from the leg area of the cortex run most closely to the walls of the lateral ventricles, providing a possible explanation for the gait disturbance. The gait disturbance, though, is probably not due to leg weakness but rather is a gait "apraxia" and may instead by related to pressure on frontal lobe structures. Cortical projections related to the bladder also descend near the walls of the lateral ventricles and pressure on them may explain the uninhibited neurogenic bladder seen in NPH.

In clinical experience, those patients in whom the gait disturbance is more prominent than the cognitive decline fare best with shunting. In selecting patients for potential shunting, we use the response to CSF drainage following lumbar puncture as an indicator of possible benefit.

Finally, a randomized controlled trial of ventriculoperitoneal shunt surgery for NPH has been performed. Luciano et al. (Luciano 2025) conducted a double-blind, randomized, placebo-controlled trial involving participants selected for shunt surgery on the basis of gait-velocity improvement with cerebrospinal fluid (CSF) drainage. Participants were randomly assigned to an open-shunt valve setting or a placebo valve of a noninvasively adjustable shunt. The primary outcome was the change in gait velocity 3 months after surgery. Secondary outcomes were the change at 3 months in the Tinetti scale total score (a measure of gait and balance, with lower scores indicating worse gait and balance), Montreal Cognitive Assessment (MoCA) score (lower scores indicate worse cognition), and Overactive Bladder Questionnaire score (higher scores indicate worse urinary incontinence).

99 participants underwent randomization and received the assigned intervention. At 3 months, gait velocity had increased in the open-shunt group and was unchanged in the placebo group (treatment difference of 0.21 m per second was statistically significant). 80% of the participants in the open-shunt group had a change in gait velocity that surpassed the substantial meaningful change threshold of 0.10 m per second in older adults. A significantly greater improvement in the open-shunt group than the placebo group was seen for the Tinetti scale score but not the MoCA score or the Overactive Bladder Questionnaire score.

Regarding adverse events, the most serious adverse events in the trial were two parenchymal hemorrhages at the time of surgery, one of which ultimately resulted in death (that was in the placebo group), and three subdural hematomas that resulted in treatment. More participants in the open-shunt group had subdural bleeding (12% vs. 2%) and positional headaches (59% vs. 28%). However, three subdural hematomas and three hygromas were treated with noninvasive adjustment of the shunt valve setting. Similarly, low-pressure headaches were treated by adjustment of the shunt valve setting. An equal percentage had cerebral bleeding (2% in both groups). More participants in the placebo group reported falls (46% vs. 24%), but the number of participants with clinically significant falls was low and did not differ substantially between groups (2 in the open-shunt group and 3 in the placebo group).

The authors concluded that ventriculoperitoneal shunting resulted in significant improvements at 3 months in gait velocity and a measure of gait and balance but not in measures of cognition or incontinence.

Perhaps the biggest limitation of the trial is that the follow up to date has only been 3 months. The authors plan to continue evaluating patients for at least 12 months. That should provide an answer to the sustainability of the gait improvement and perhaps indicate whether there is any further change in cognitive performance of bladder symptoms.

With any medical intervention we weigh the potential benefits vs the potential risks. While this clinical trial demonstrates ventriculoperitoneal shunting resulted in significant improvements in gait, there were risks as well. And the question of sustainability remains

unanswered. Perhaps most importantly, it tells us that we must be circumspect in what we tell our patients about expectations for cognitive function. Improvement in gait is definitely an important goal, but many patients and their families agree to the surgery in hopes that there will be a cognitive benefit as well.

The accompanying editorial by our colleague and good friend Allan Ropper (Ropper 2025) notes that the results of the trial affirm the existence of idiopathic normal-pressure hydrocephalus as a clinical entity and show that CSF drainage is usually effective. He points out that the results are a signature advance given that no truly equivalent trials have been performed. But he also cautions that the durability of the effect of shunting on gait and any effect on cognition need to be evaluated at 12 months.

We applaud the investigators for undertaking this much needed clinical trial and anxiously await their 12-month follow up.

While we traditionally include NPH as a "treatable" cause of cognitive decline, it is important that we and our patients and their families have realistic expectations when we consider ventriculoperitoneal shunting

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