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Magnetic Resonance Imaging of Normal Pressure Hydrocephalus

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Normal pressure hydrocephalus (NPH) is a syndrome found in the elderly, which is characterized by ventriculomegaly and deep white matter ischemia (DWMI) on magnetic resonance imaging (MRI) and the clinical triad of gait disturbance, dementia, and urinary incontinence. NPH has been estimated to account for up to 10% of cases of dementia and is significant because it is treatable by ventriculoperitoneal shunting. Patients with a known cause of chronic communicating hydrocephalus, that is, meningitis or hemorrhage, tend to respond better than patients with the so-called "idiopathic" form, most likely because of poor selection criteria in the past. Good response to shunting has been associated with hyperdynamic cerebrospinal fluid (CSF) flow through the aqueduct. In the early days of MRI, patients with a large CSF flow void extending from the foramen of Monro through the aqueduct to the fourth ventricle had an excellent chance of responding to ventriculoperitoneal shunting ($P < 0.003$). Today, we use phase-contrast MRI to measure the volume of CSF flowing through the aqueduct in either direction over a cardiac cycle. When this aqueductal CSF stroke volume is sufficiently elevated, there is an excellent chance of shunt responsiveness (100% positive predictive value in 1 study). Idiopathic NPH appears to be a "two-hit" disease—benign external hydrocephalus (BEH) in infancy followed by DWMI in late adulthood. As BEH occurs when the sutures are still open, these infants present with large heads, a finding also noted in patients with NPH. Although BEH has been attributed to immature arachnoidal granulations with decreased CSF resorptive capacity, this now appears to be permanent and may lead to a parallel pathway for CSF resorption via the extracellular space of the brain. With DWMI, the myelin lipid is lost, exposing the polar water molecules to myelin protein, increasing resistance to CSF outflow and leading to backing up of CSF and hydrocephalus.
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Normal Macroscopic Cerebrospinal Fluid Flow

The classic teaching is that cerebrospinal fluid (CSF) is formed primarily in the choroid plexus within the ventricles at a rate of 500 cc/d. It flows out of the fourth ventricle via the foramina of Lushka and Magendie into the subarachnoid space (SAS). Once in the SAS, the CSF either flows down around the spinal cord or flows up over the cerebral convexities, eventually to be primarily absorbed by the arachnoid granulations (macroscopic) and arachnoidal villi (microscopic) on either side of the superior sagittal sinus. This CSF resorption pattern was based on tracer studies performed many years ago using large molecules.

Superimposed on the slow egress of CSF from the ventricles to the SAS is a more prominent pulsatile motion due to the beating of the heart. During systole, blood flows into the brain causing it to expand inwards, compressing the ventricles, and outwards, compressing the cortical veins and SAS. The inward expansion leads to pulsatile outflow of CSF through the aqueduct and the rest of the ventricular system. This results in a normal CSF flow void on magnetic resonance imaging (MRI) studies through the aqueduct.[1] The systolic expansion forces CSF and venous blood out of the fixed volume of the skull by the Monro-Kellie hypothesis.[2] This results in the systolic outflow of CSF at the foramen magnum and from there down the SAS of the spinal canal or up over the convexities. During diastole, the volume of the brain decreases

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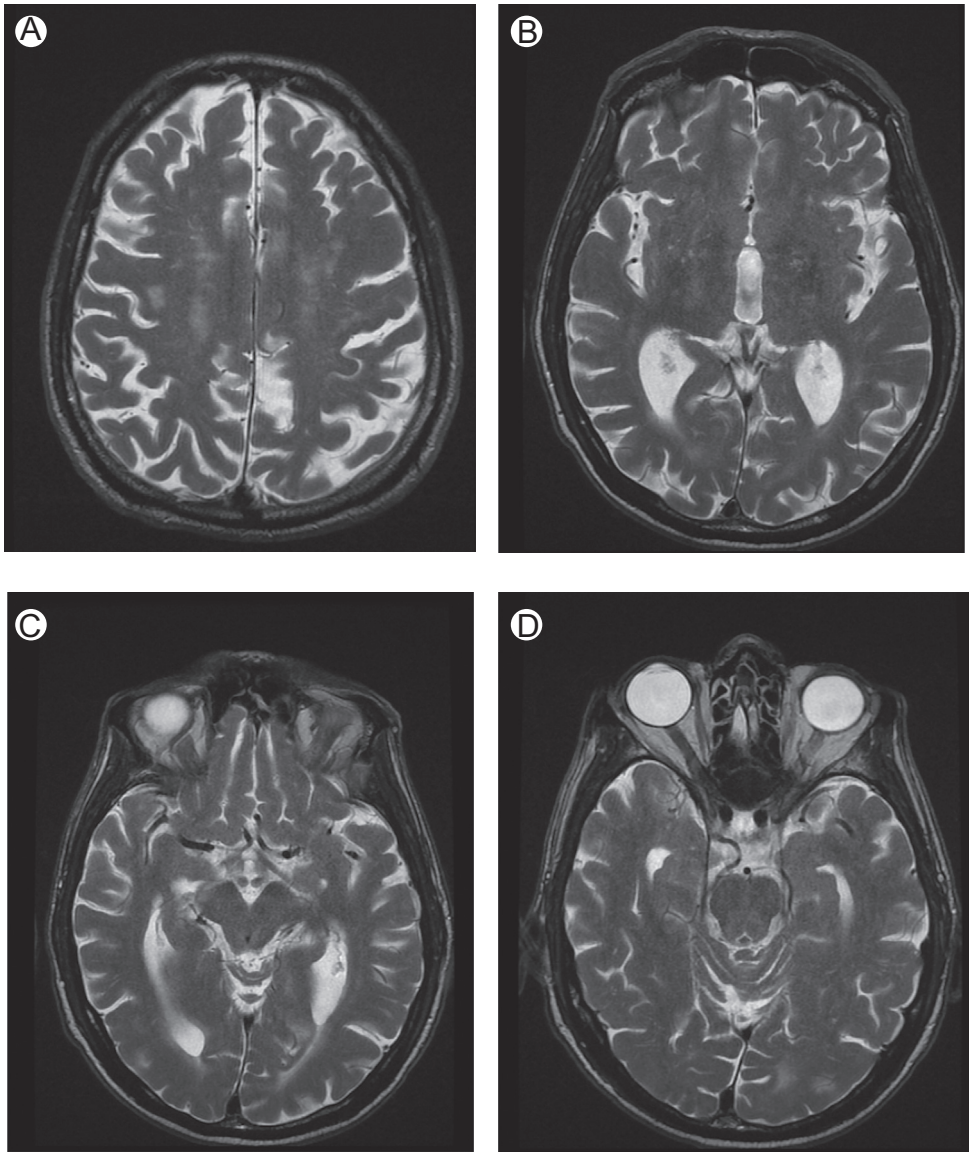

Figure 1 NPH on a T2-weighted fast spin echo, axial images. (A) Section through centrum semiovale demonstrates deep white matter ischemia. (B) Section through third ventricle shows loss of the waist do to slight enlargement with minimal CSF flow void. (C and D) Sections through aqueduct and upper fourth ventricle show CSF flow void. Although less conspicuous than in the past using conventional spin echo, the CSF flow void sign is now more specific for hyperdynamic flow, albeit less sensitive.

and CSF flows in a reverse direction through the foramen magnum and the aqueduct.

Although most CSF is produced by the choroid plexus, recent evidence suggests that a portion of the CSF is made by the capillaries in the brain parenchyma. Similarly, it has been estimated that up to 20% of the CSF uptake occurs in the brain parenchyma, via the lymphatics near the cribiform plate, or covering the cranial nerves in the basal cisterns. Regardless of the exact percentage of CSF produced by the brain vs the choroid plexus, the bulk flow of CSF is out of the lateral ventricles via the foramen of Monro through the third ventricle and aqueduct and then through the fourth ventricle.

Hydrocephalus was subdivided based on the point of obstruction of CSF flow by Dandy over 100 years ago. Obstruction proximal to the outlet foramina of the fourth ventricle was termed "obstructive hydrocephalus," whereas obstruction distal to the foramina of Lushka and Magendia was termed "communicating hydrocephalus." Most causes of obstructive hydrocephalus in adults are due to tumors obstructing the outflow of CSF upstream to the outlet foramina of the fourth ventricle. Most causes of communicating hydrocephalus are due to subarachnoid hemorrhage or meningitis, the former obstructing the arachnoidal villi and the latter often obstructing more proximally at the level of basal cisterns, particularly with viscous fungal, tubercular, or other granulomatous meningitities.

A subset of communicating hydrocephalus seen in the elderly is termed "normal pressure hydrocephalus (NPH)" (Fig. 1) and is defined by the clinical triad of gait disturbance, dementia, and incontinence in conjunction with a low opening pressure on lumbar puncture. A subset of communicating hydrocephalus seen in infants aged 6-12 months is termed