REVIEW

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When do common symptoms indicate normal pressure hydrocephalus?

ABSTRACT

The Adams triad (gait impairment, dementia, and urinary incontinence) of normal pressure hydrocephalus (NPH) is well known, but other illnesses present with similar symptoms. Accurate diagnosis of this rare disorder depends on careful evaluation. A ventriculoperitoneal shunt is the only effective therapy, and deciding whether the patient will benefit is the final challenge in the evaluation process.

KEY POINTS

An abnormal gait is typically the first and major symptom in NPH: absence of this symptom or the relative prominence of dementia or urinary complaints makes another diagnosis more likely.

In NPH, ventriculomegaly is evident by computed tomography or magnetic resonance imaging.

Ancillary testing can help determine if shunt therapy is likely to succeed.

Removing cerebrospinal fluid (CSF) and placing a shunt typically results in faster gait and fewer urinary symptoms. If CSF drainage remains adequate and unobstructed, benefits can be maintained indefinitely.

AN 86-YEAR-OLD MAN reports that his gait has been progressively slowing over the last 3 to 4 years and he has been falling down. He also reports having urinary urgency and increasing difficulty with word-finding, organization, and memory over the past 3 years, and urinary incontinence over the past year. He has benign prostatic hypertrophy and also had a stroke some time in the past that left him with mild residual left-visual-field loss.

On physical examination, he has multiple gait abnormalities. He has a hard time standing and starting to walk, and he walks slowly, taking short steps. He keeps his feet far apart and does not raise them very high off the ground, pausing between steps with both feet planted ("increased double stance time"). He also takes a long time to turn around.

His score on the Mini-Mental State Examination is 18/30, indicating moderate cognitive impairment.

A RARE DISEASE WITH COMMON SYMPTOMS

Although this patient has the classic Adams triad of symptoms of normal pressure hydrocephalus (NPH)—abnormal gait, cognitive impairment, and urinary dysfunction—these are not unusual in elderly patients. How to distinguish this rare condition from other diseases can be challenging.

This article illustrates the presentation of NPH, the differential diagnosis, and how to determine if a patient is likely to benefit from treatment with ventriculoperitoneal shunt.

NPH is uncommon

NPH usually begins in the sixth or seventh

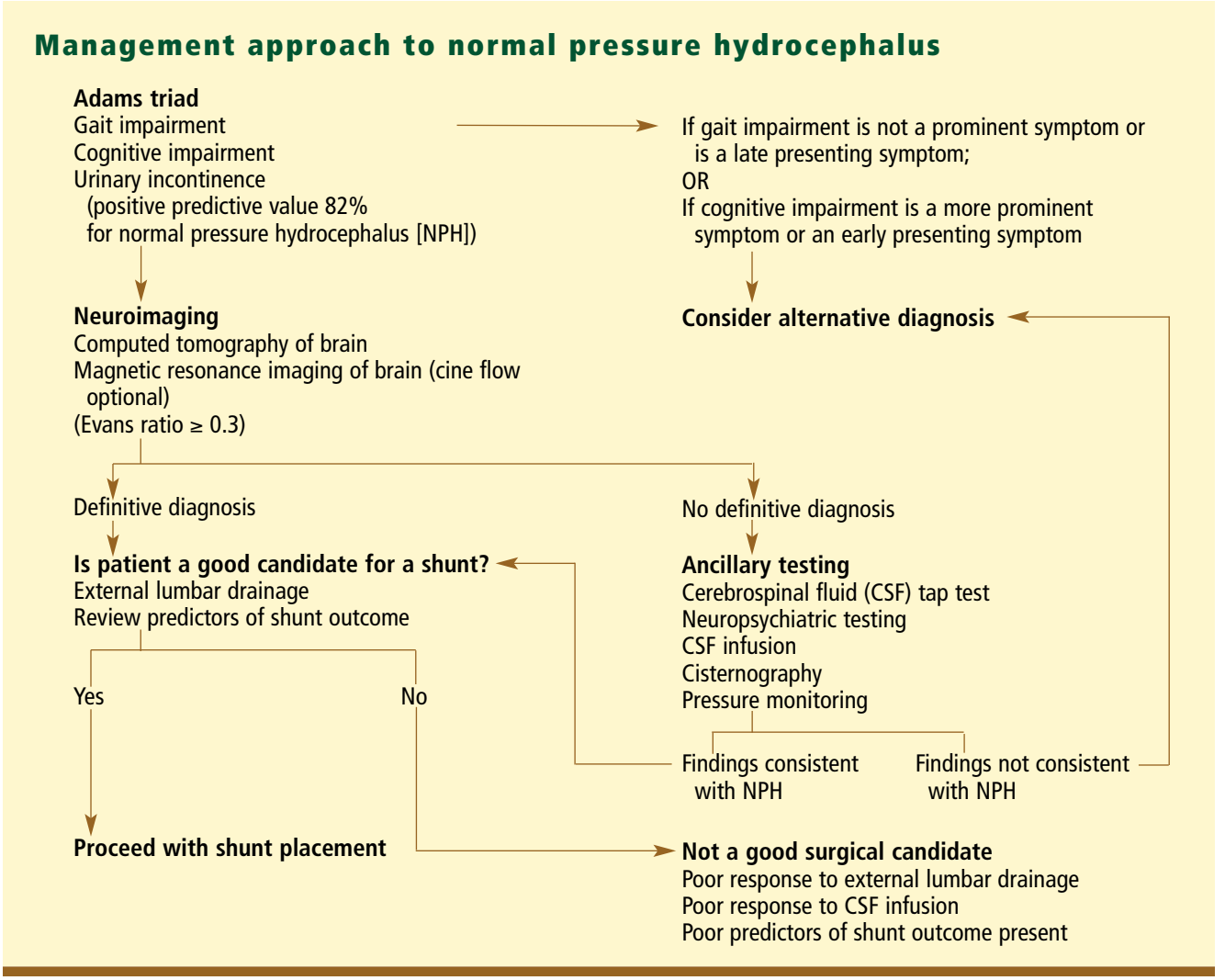

FIGURE 1

decade of life.[1,2] The incidence rate is controversial: estimates range from as rare as 1.3 per million to as frequent as 4 per 1,000, a disparity attributed to different diagnostic criteria and populations sampled.[3–5]

Why do cerebral ventricles enlarge?

The underlying pathophysiology of NPH remains unclear. A likely hypothesis is that ventriculomegaly results from excess accumulation of cerebrospinal fluid (CSF). The natural balance between CSF production and resorption is believed to be disturbed, although exactly where it is disturbed is unknown and likely varies individually.[6]

An alternative theory is that an imbalance exists between expansive ventricular pressures and opposing forces within the brain parenchyma. Normal aging reduces the elasticity of the neuropil (the dense fibrous network of glia and neurons in the gray matter), extracellular matrix, and parenchyma, which give way to the expansive force of the ventricles. Patients with NPH have normal CSF pressure overall, but abnormal pressure spikes called B waves may have a "water hammer" effect, slowly increasing ventricular size by exerting intermittent high pressures on the brain parenchyma.[7–9]

RULING OUT OTHER CAUSES OF SYMPTOMS

FIGURE 1 shows an approach for evaluating patients with suspected NPH.

The entire Adams triad may not be evi-

dent when a patient initially presents but is likely to develop as the disease progresses.[10] Because many diseases of elderly people cause signs and symptoms similar to those of NPH, it is important to do a thorough evaluation before making the diagnosis.

Impaired gait is prominent

Impaired gait, usually the first symptom to appear, occurs in 89% of people diagnosed with idiopathic NPH.[11] The abnormal gait is typically wide-based with reduced step height, stride length, and velocity. In time, it becomes difficult to initiate gait. This so-called "magnetic gait" may be caused by the expanding ventricles breaking the connection between the basal ganglia and the frontal cortex. Antigravity muscle reflexes become uninhibited, producing simultaneous contraction of opposing muscles while walking.[12,13]

At first, a patient with NPH may not have the classic abnormal gait: severity increases as the disease progresses. Important findings are slow walking (perhaps based only on reports of the patient or family), imbalance or unsteadiness, and difficulty with starting to walk.

Other conditions that cause gait abnormalities must be considered by themselves or as contributing factors (TABLE 1).

- Lumbar canal stenosis is common in elderly people. Patients typically feel pain in the lower back or leg, which worsens with spinal extension and improves while walking down steps or pushing a shopping cart. Magnetic resonance imaging (MRI) of the lumbosacral spine aids in the diagnosis.
- Peripheral neuropathy can contribute to gait impairment and can be identified with a detailed neurologic examination.
- Parkinsonism is characterized by a resting "pill rolling" tremor of the hands, bradykinesia, rigidity, and freezing. Parkinson-like features can occur in late NPH and typically do not respond to levodopa.[14]

Urinary incontinence is 'urge'-type

Urinary frequency and urgency typically appear early in NPH, perhaps caused by stretching of periventricular nerve fibers, leading to partial loss of inhibition of bladder contractions.[8] Gait impairment may also contribute to incontinence if a patient cannot get to a bathroom on time. An estimated 45% to 90% of patients with NPH experience urinary incontinence.[11]

Like gait impairment, urinary incontinence has many possible causes. It is helpful to distinguish urge incontinence (which is more likely to be due to NPH) from stress incontinence (which is less likely to be due to NPH). Bladder outlet obstruction from benign prostatic hypertrophy is a common cause of urinary retention and stress incontinence in men. Incontinence can also be caused by diseases affecting autonomic regulation of the bladder, such as diabetic neuropathy or multiple system atrophy. Other frequent culprits are diuretics and medications with anticholinergic effects.

Urodynamic testing does not provide any findings that are specific to NPH. However, it can help to identify the physiologic basis for urinary incontinence, especially for patients with a mixed picture with multiple suspected causes and if it is feared that empiric therapy would worsen symptoms.

Cognitive impairment is subcortical

Up to 77% of patients with NPH develop dementia.[11] The cognitive changes are typically subcortical, manifesting as memory

TABLE 1

Diseases that cause gait problems resembling those of normal pressure hydrocephalus

- **Alzheimer disease**
- **Cerebrovascular disease**
  - Binswanger disease
  - Lobar stroke
  - Lacunar stroke
- **Lumbar canal stenosis**
- **Parkinsonism**
  - Corticobasal ganglionic degeneration
  - Lewy body disease
  - Parkinson disease
  - Progressive supranuclear palsy
- **Peripheral neuropathy**
- **Traumatic brain injury**
- **Tumors**

Impaired gait is usually the first symptom of NPH

impairment as well as decreased attention, alertness, and the speed of mental processing.[8,15] The cortical deficits of aphasia, apraxia, and agnosia are usually not present.

Other causes of cognitive impairment

- Depression can be difficult to distinguish from NPH-associated dementia: both often involve apathy and bradyphrenia, and the neuropsychiatric profiles of the two conditions are similar.[8]
- Alzheimer disease should be strongly considered if cognitive impairment is the prominent feature. It classically presents as a slow and progressive decline in memory and function, with prominent cortical deficits of aphasia and apraxia, with or without agnosia. In cases of coexisting NPH and Alzheimer disease, hippocampal atrophy may be seen on MRI.
- Dementia with Lewy bodies and dementia resulting from long-standing Parkinson disease, like the dementia of NPH, usually involve subcortical features, diminished attention, and visuospatial dysfunction, but they can be distinguished from NPH by bradykinesia and tremor. Visual hallucinations and fluctuations in consciousness are additional hallmarks of dementia with Lewy bodies.
- Vascular dementia typically involves a stepwise cognitive decline as well as subcortical deficits with prominent impairment of executive function. Patients with vascular dementia may have significant mood problems manifested as emotional lability, impulsivity, or depression.

Urinary incontinence has many causes; distinguish urge from stress incontinence

A brief evaluation can help in determining the cause of cognitive impairment. The Mini-Mental State Examination may show deficits in calculation, concentration, sentence-writing, copying intersecting pentagons, and following a three-stage command, indicating impaired executive function. Slowed mental processing may be evident from increased test-taking time. Performance of the clock-drawing test may also be impaired, showing poor spatial planning and organization. A screening tool for depression such as the short form of the Geriatric Depression Scale can help distinguish depression from NPH.

More extensive neuropsychiatric testing can further delineate and quantify the severity of deficits. This is especially useful in defining whether an impairment is cortical or subcortical and in monitoring responses to treatment.

NEUROIMAGING CAN CONFIRM THE DIAGNOSIS

Neuroimaging of the brain is needed to confirm the diagnosis of NPH and to help rule out other causes. Ventriculomegaly can be detected by either computed tomography (CT) or MRI. The latter provides more information and can help identify obstruction as a cause of ventriculomegaly.

The Evans ratio. Ventriculomegaly is quantified by the Evans ratio, which is the maximum width of the anterior ventricular horns divided by the maximum width of the calvarium at the level of the foramen of Monro. Hydrocephalus is defined as an Evans ratio of at least 0.3, but most patients with NPH have a ratio of more than 0.4.[11,16]

Neuroimaging may also reveal:

- Focal sulcal dilatations, representing atypical reservoirs of CSF
- Periventricular areas of high intensity that typically resolve after shunting[17]
- The "CSF flow void" sign: reduced MRI signal in the aqueduct of Sylvius due to hyperdynamic flow or turbulence of CSF in the fourth and posterior third ventricles.

Cerebral atrophy as a result of aging can also cause ventricular dilatation, known as hydrocephalus ex vacuo. It is difficult to determine the significance of hydrocephalus in the face of parenchymal volume loss and to distinguish age-related changes from NPH in this context.

In patients with NPH, the peripheral edges of the brain parenchyma may appear as if the ventricles have pushed the tissues against the cranial vault and flattened them. Sulci may appear to be compressed, and ventricular enlargement may appear to be disproportional to the size of the sulci. In contrast, sulci in hydrocephalus ex vacuo are more prominent, and the periphery of the parenchyma does not appear flattened. The amount of ventriculomegaly and sulcal widening may also appear to be proportional.

Cerebrovascular disease (detected by lacunar infarcts or white matter changes on MRI) also causes atrophy and ventricular dilatation

Ventricular enlargement in normal pressure hydrocephalus

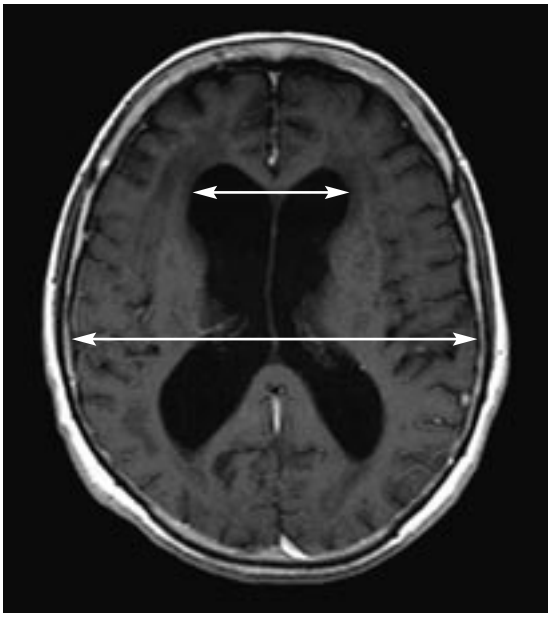

FIGURE 2. Magnetic resonance image showing ventriculomegaly in a patient with normal pressure hydrocephalus. The Evans ratio is the maximum width of the anterior horns (shorter arrow) divided by the maximum width of the calvarium (longer arrow). Hydrocephalus is defined by a ratio of 0.3 or greater; this patient's ratio is 0.4.

Depression can be hard to distinguish from NPH-associated dementia

and does not rule out NPH. If cerebrovascular disease is evident, the prognosis for clinical improvement for patients being treated for NPH is poor.[18]

The ventriculomegaly of NPH and normal aging often have overlapping findings. Identifying components of the clinical triad of NPH is key. If the diagnosis is uncertain, ancillary testing may help.

Vanneste et al,[19] in a retrospective study of 98 patients, found that the combination of the Adams triad and the criteria for diagnosing NPH by CT had a sensitivity of 71% and a specificity of 84% for predicting substantial improvement with shunting.

Case continued

In our patient, MRI of the brain (FIGURE 2) shows a communicating hydrocephalus and an Evans ratio of 0.4, indicating ventriculomegaly and the likelihood of NPH. Further testing is ordered to solidify the diagnosis and to determine whether the patient is a good candidate for shunt placement.

ADDITIONAL TESTS

Additional testing can clarify an uncertain diagnosis and determine whether CSF diversion (ie, placement of a shunt) is likely to be successful. No evidence-based guidelines exist, however, to determine whether shunt surgery is appropriate for a particular patient with NPH. Evaluation protocols vary from one institution to another, highlighting the variety of opinions that exist regarding the utility of ancillary tests.

CSF drainage tests. The CSF drainage (tap) test involves removing 40 to 50 mL of CSF by single or multiple lumbar punctures to see if symptoms improve. Alternatively, a large volume of CSF can be removed by external lumbar drainage while a patient is monitored for 1 to 5 days in the hospital: a maximum of 400 to 500 mL can be removed via continuous drainage at a rate of 10 mL per hour.

Improvement in symptoms is usually transient. Gait (speed and stride length) usually improves most obviously,[20,21] although sometimes urinary symptoms also improve.[22] Patients with dementia may report feeling more lucid.[6] Neuropsychiatric testing may reveal improved visual attention (as revealed with the symbol digit modalities test, a timed test involving pairing numbers with geometric figures using a reference key), verbal memory, and motor precision (revealed with the line-tracing test, which involves tracing over a line marked on a piece of paper without touching it).[23–25] Noted improvement in these areas during extended lumbar drainage has a positive predictive value of 80% to 90%, sensitivity 50% to 100%, and specificity 60% to 100% for diagnosing NPH.[24,26–28] NPH is not necessarily ruled out if symptoms do not improve, however.

Although CSF drainage tests can help identify good candidates for shunt treatment, they are not completely reliable for predicting success. The CSF tap test has a positive predictive value of 75% in patients who have a significant response, but it has a false-negative rate of 50%[11,29]; sensitivity ranges from 26%

to 42%, and specificity from 33% to 100%.[28]

For patients who improve only mildly after CSF removal, watchful waiting for further clinical deterioration rather than prompt treatment with a shunt is a reasonable approach.

Continuous intracranial pressure monitoring can detect B waves (spikes of increased pressure over normal intraventricular pressure), which are considered pathognomonic for NPH. Many consider B waves to be predictive of a good response to shunting[8]; conversely, if they are absent or rare, a poor response to shunting can be expected. The sensitivity of this sign ranges from 78% to 91% and the specificity from 13% to 40%.[30] Criteria for amplitude and frequency of B waves have not been well defined, and whether B waves should even be used to evaluate NPH is controversial.[31]

The CSF infusion test is performed by measuring CSF pressures during an infusion of mock CSF, Ringer's lactate, or isotonic solution via lumbar puncture. Infusion continues until a steady state between absorption and infusion is reached, and outflow resistance and compliance to CSF absorption are calculated. A steady- state pressure of 22 mm Hg (outflow resistance 13–14 mm Hg/mL/minute) or a steady increase in pressure to 40 mm Hg is considered to be a positive result.[10,22] Kahlon et al[22] calculated that the test had a positive predictive value of 80% and a negative predictive value of 16% compared with the CSF tap test, but the usefulness of the CSF infusion test in helping to diagnose NPH varies.[32]

Cine phase-contrast MRI may also help identify patients with NPH. A quantitative analysis of CSF flow between the third and fourth cerebral ventricles during systole and diastole is performed using T2-weighted MRI. Hyperdynamic flow (> 18 mL/minute) in a sinusoidal pattern is associated with NPH; Luetmer et al[33] determined that the average flow rate for patients with NPH is 27.4 mL/minute.

Radioisotope cisternography is useful in identifying blockages in CSF flow by visualizing the ventricular conducting system and evaluating CSF absorption and clearance. The test typically involves infusing a radiolabelled isotope into the conducting system via a lumbar subarachnoid injection and performing CT of the head at intervals over 4 days. If the isotope appears in the ventricles within 72 hours but has not been distributed to the convexities, this is considered to be evidence of NPH.[6] But whether this test increases diagnostic accuracy beyond information gained by clinical and neuroimaging criteria has not been proven.[34]

Positron emission tomography (PET) is used to evaluate glucose metabolism and cerebral blood flow. Initial studies show that patients with NPH tend to have reduced regional blood flow. PET has a sensitivity of 89% and a specificity of 82% in distinguishing patients with NPH from normal controls, but no characteristic pattern of reduced regional blood flow has been identified. PET scans have not been shown to predict the outcome of shunting.[11]

Single-photon emission-CT perfusion imaging and **perfusion-weighted MRI** can help in evaluating changes of cerebrovascular blood flow after removing CSF. Small studies have demonstrated improved selection of patients for CSF diversion by combining results of the CSF tap test with either of these imaging tests.[35]

Case continued

Our patient undergoes the CSF tap test and then is reevaluated, showing some improvement in gait and on neuropsychiatric testing. Because of his positive response, he is deemed an appropriate candidate for surgical intervention. Placement of a ventriculoperitoneal shunt is scheduled.

CSF drainage tests are not completely reliable for predicting success with shunt surgery

TREATMENT

Medical strategies little studied

Nonsurgical treatments for idiopathic NPH have been little studied, and no evidence exists that they help in either the short term or the long term. However, they are sometimes used in children as a temporary measure until shunting can be performed. These strategies include:

- Drugs to reduce CSF production—sodium/potassium adenosine triphosphatase inhibitors, carbonic anhydrase inhibitors, loop diuretics
- Drugs to reduce CSF volume within the brain—osmotic agents
- Drugs to increase CSF resorption—fibrinolytic therapy, steroids.[36]

TABLE 2

Likely outcomes of shunt therapy

Mean chance of substantial improvement: 30%–50%

Dementia is least likely of the Adams triad to improve, especially if severe

Rate of perisurgical or postsurgical complications: 20%–40%

Serious complications (death or severe residual deficit): 5%–8%, mainly in patients with other medical problems

Shunt dysfunction is not uncommon and almost always due to delayed outflow or obstruction of the peritoneal catheter

Ineffective (nondraining) shunt should be suspected especially in patients in whom the ventricular size does not decrease after shunt placement and in patients with transient postsurgical improvement

DATA FROM VANNESTE JA. DIAGNOSIS AND MANAGEMENT OF NORMAL-PRESSURE HYDROCEPHALUS. J NEUROL 2000; 247:5–14.

TABLE 3

Predictors of outcome of shunt placement in patients with normal pressure hydrocephalus

Predictors of good outcome

- Gait disturbances precede mental impairment (positive predictive value 39%–47%)
- Short history of mental deterioration
- Slight to moderate mental impairment
- MRI: pattern of hydrodynamic hydrocephalus and absence of substantial white matter lesions
- CSF drainage: substantial clinical improvement after one or several lumbar CSF taps or after continuous external lumbar CSF drainage
- Continuous intracranial pressure monitoring: B waves during at least half of the recording time
- Continuous CSF infusion test: resistance to outflow ≥ 18 mm Hg/mL/minute

Predictors of indeterminate significance

- Age
- Duration of symptoms
- MRI: absence of an aqueductal CSF void sign in spite of an open aqueduct
- CSF tap test: no improvement

Predictors of poor outcome

- Severe dementia is predominant symptom
- Dementia is first neurologic sign
- Gait disorder occurred after dementia
- No gait disorder (negative predictive value 83%)
- MRI: marked cerebral atrophy or widespread white matter involvement

ADAPTED FROM VANNESTE JA. DIAGNOSIS AND MANAGEMENT OF NORMAL-PRESSURE HYDROCEPHALUS. J NEUROL 2000; 247:5–14; AND HEBB AO, CUSIMANO MD. IDIOPATHIC NORMAL PRESSURE HYDROCEPHALUS: A SYSTEMATIC REVIEW OF DIAGNOSIS AND OUTCOME. NEUROSURGERY 2001; 49:1166–1186.

Symptoms of NPH improve substantially with surgery in only 30% to 50% of patients

Shunt placement: Success is highly variable

CSF flow diversion is the only generally accepted treatment for NPH. The goal of treatment is to restore functional capacity, and the decision of whether to treat should be made based on how likely that is to occur.

Physicians should define the goals of treatment by discussing with patients and families their expectations as well as realistic outcomes (TABLE 2). The success of shunt placement is highly variable, but several predictors of outcome have been identified (TABLE 3). Symptoms improve substantially in only 30% to 50% of patients, though symptoms can continue to improve slowly for up to 24 months.[2] Dementia is the least likely element of the

Adams triad to improve, especially if it is the predominant feature. Complications occur in about 38% of patients, with 22% requiring additional surgery; about 6% of patients sustain permanent brain damage or die.[11] Serious complications correlate strongly with the comorbid condition of the individual patient.[8]

If the shunt continues to function, improvements should last. A shunt should be suspected of being ineffective if the ventricular size does not decrease or if symptoms improve only transiently.

Patients who have old symptoms that recur or who develop new ones such as headache or delirium should be evaluated promptly with a CSF tap and either CT or MRI. Shunt dysfunction is not uncommon and is almost always caused by delayed outflow or an obstructed peritoneal catheter. A problem may require only a simple adjustment in the rate of CSF flow (in adjustable shunts) or may alternatively require the shunt to be completely revised. Many complications can occur, and regular follow-up is required.

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