

Idiopathic normal pressure hydrocephalus diagnosis

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Diagnosis is based on the evaluation of [Idiopathic normal pressure hydrocephalus clinical features](#) combined with an [MRI](#) assessment, evaluation of [cerebrospinal fluid dynamics](#) by different methods such as a [tap test](#), [lumbar infusion test](#) (LIT), and [External lumbar cerebrospinal fluid drainage](#) (ELD).

[Screening](#) for [idiopathic normal pressure hydrocephalus](#) in the elderly presenting after [falls](#) can possibly identify iNPH patients in the earlier stage who may benefit more from surgical treatments ¹⁾.

Patients often present to the [neurosurgeon](#) frustrated and desperate after a long preoperative course. It is important to acknowledge the uncertainty regarding [Idiopathic normal pressure hydrocephalus diagnosis](#) ²⁾.

There is no accurate test for diagnosing [normal pressure hydrocephalus](#) or for screening for patients who will benefit from shunt surgery.

Shunting is possibly effective in iNPH (96% chance subjective improvement, 83% chance improvement on timed walk test at 6 months) (3 [Class III](#)). Serious adverse event risk was 11% (1 [Class III](#)). Predictors of success included elevated Ro (1 [Class I](#), multiple [Class II](#)), impaired cerebral blood flow reactivity to acetazolamide (by SPECT) (1 [Class I](#)), and positive response to either [External lumbar cerebrospinal fluid drainage](#) (1 [Class III](#)) or repeated lumbar punctures. Age may not be a prognostic factor (1 [Class II](#)). Data are insufficient to judge efficacy of radionuclide cisternography or aqueductal flow measurement by MRI.

There is limited Class I evidence that impaired cerebral blood flow (CBF) reactivity to acetazolamide is a predictor of successful CSF shunting, but Single-photon emission computed tomography (SPECT) is not a practical screening tool for NPH.

Imaging

[Idiopathic normal pressure hydrocephalus Imaging](#)

see [Disproportionately enlarged subarachnoid space hydrocephalus](#).

Fewer [centrum semiovale enlarged perivascular spaces](#) may be a [diagnostic biomarker](#) for [idiopathic normal pressure hydrocephalus diagnosis](#). This pattern may be caused by mechanical [obstruction](#) due to upward displacement of the brain leading to reduced [glymphatic](#) clearance ³⁾.

Psychomotor Tasks

Although gait is the primary indicator for treatment candidacy and outcome, additional monitoring tools are needed. [Line Tracing Test](#) (LTT) and Serial Dotting Test (SDT), two psychomotor tasks, have been introduced as potential outcome measures ⁴⁾.

Lumbar puncture for idiopathic normal pressure hydrocephalus diagnosis

see [Lumbar puncture for Idiopathic normal pressure hydrocephalus diagnosis](#).

Pressure recording

see [Idiopathic normal pressure hydrocephalus intracranial pressure monitoring](#).

[Alzheimer disease](#) (AD)-related pathology was assessed in cortical biopsy samples of 111 patients with idiopathic normal-pressure hydrocephalus. Alzheimer disease hallmark lesions [amyloid beta](#) (A β) and hyperphosphorylated [tau protein](#) (HPTau)-were observed in 47% of subjects, a percentage consistent with that for whole-brain assessment reported postmortem in unselected cohorts. Higher-immunostained area fraction of AD pathology corresponded with lower preoperative [mini mental state examination](#) scores. Concomitant A β and HPTau pathology, reminiscent of that observed in patients with AD, was observed in 22% of study subjects. There was a significant correlation between A β -immunostained area fraction in tissue and A β 42 (42-amino-acid form of A β) in [cerebrospinal fluid](#) (CSF). Levels of A β 42 were significantly lower in CSF in subjects with concomitant A β and HPTau

pathology compared with subjects lacking pathology. Moreover, a significant correlation between HPTau-immunostained area fraction and HPTau in CSF was noted. Both HPTau and total tau were significantly higher in CSF in subjects with concomitant A β and HPTau pathology compared with subjects lacking pathology. The 42-amino-acid form of A β (A β 42) and HPTau in CSF were the most significant predictors of the presence of AD pathology in cortical biopsies. Long-term follow-up studies are warranted to assess whether all patients with idiopathic normal-pressure hydrocephalus with AD pathology progress to AD and to determine the pathologic substrate of idiopathic normal-pressure hydrocephalus ⁵⁾.

Biopsy

[Biopsy for Idiopathic normal pressure hydrocephalus diagnosis.](#)

Biomarkers

[Idiopathic normal pressure hydrocephalus Cerebrospinal fluid biomarkers](#)

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⁴⁾

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