

Pathophysiology

- Impaired CSF resorption by arachnoid villi causes communicating hydrocephalus
- Traditional theory: Increased resistance to CSF outflow
- Newer theory: Increased pulsations in intracranial pressure has been suggested as potential mechanism
- Dysfunctional CSF dynamics without increase in intracranial pressure

Pathophysiology

- One theory
 - An obstructive type of communicating hydrocephalus due to reduced CSF resorption.
- A second theory
 - Results from weakening of the ventricular wall due to periventricular white matter ischemic damage .
 - The periventricular white matter ischemic change has also been hypothesized to slow the flow of CSF through the extracellular spaces, resulting in a "back-pressure" effect, leading to ventricular enlargement.

Terminology

- It is important to note that there are many causes of communicating hydrocephalus without elevated opening CSF pressures, such as
 - Trauma
 - Prior subarachnoid hemorrhage
 - Meningitis
- This is sometimes confusingly referred to as secondary normal pressure hydrocephalus ³.
- Most clinicians will assume that one is referring to **idiopathic normal pressure hydrocephalus** if no qualifier is used.

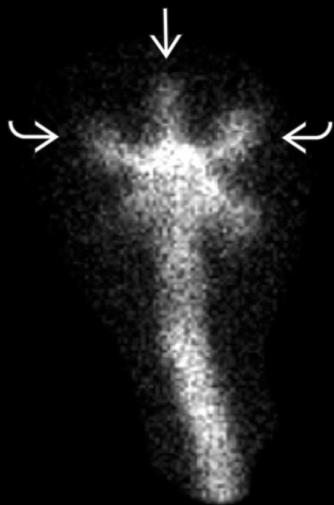
CSF movement patterns

- Type I: Radiotracer activity in cerebral convexities at 24 hours
 - Normal or noncommunicating hydrocephalus
- Type II: Delayed activity in cerebral convexities at 24 hours without ventricular activity
 - Cerebral atrophy or aging
- Type IIIa: Radiotracer activity in cerebral convexities at 24 hours with early transient ventricular activity
 - Indeterminate (can be seen with noncommunicating hydrocephalus, developing or resolving communicating hydrocephalus, or cerebral atrophy)
- Type IIIb: No radiotracer activity in cerebral convexities at 24 hours with early transient ventricular activity
 - Suggestive of NPH (communicating hydrocephalus)
- Type IV: No radiotracer activity in cerebral convexities at 24 hours with persistent ventricular activity
 - Suggestive of NPH (communicating hydrocephalus)

Imaging

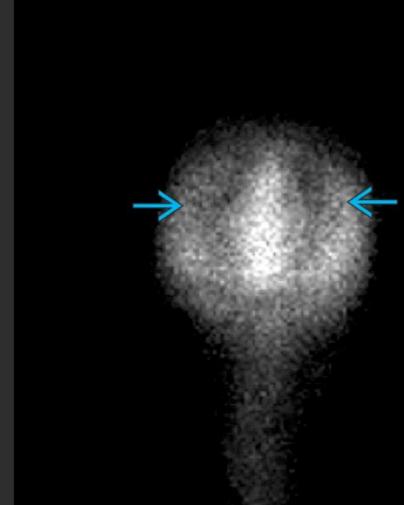
- In-111 DTPA radionuclide cisternography
 - Used in patients in whom MR is contraindicated and CT is equivocal
 - Protocol
 - Intrathecal injection of In-111 DTPA
 - Obtain planar images with gamma camera immediately after injection and at 4, 24, and 48 hours
 - Normal study
 - 1 hour: Radiotracer reaches basal cisterns
 - 2-6 hours: Radiotracer reaches Sylvian fissures
 - 12 hours: Radiotracer reaches cerebral convexities
 - 24 hours: Radiotracer reaches superior sagittal sinus and is absorbed by arachnoid villi
 - Normally no radiotracer enters ventricles, although transient activity in ventricles at 4 hours is still considered normal
 - NPH
 - Radiotracer activity in ventricles at \geq 24 hours
 - Absence of radiotracer activity in cerebral convexities by 24-72 hours
 - SPECT/CT can help confirm ventricular activity

Normal



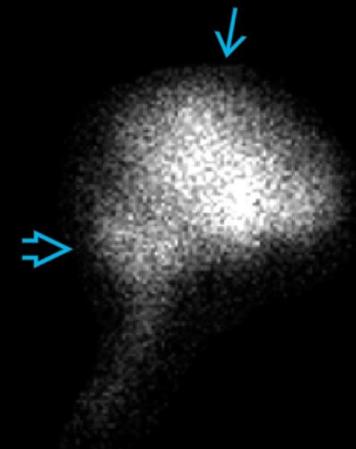
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Anterior radionuclide cisternography at 4 hours shows normal trident appearance of radiotracer in the anterior interhemispheric fissure → and Sylvian fissures ↗.



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Anterior radionuclide cisternography at 24 hours demonstrates photopenia in the region of the lateral ventricles →, a normal finding.



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Right lateral radionuclide cisternography in a normal patient at 24 hours demonstrates no ventricular activity. Activity is present in the cerebral convexities →, as well as the suprasellar and basal cisterns ⇛.



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Axial radionuclide cisternography SPECT/CT in a patient with normal-pressure hydrocephalus at 24 hours confirms activity in the lateral ventricles: Frontal horns → and occipital horns ➤.

Twenty-four hour multiplanar In-111 DTPA cisternography in a patient with NPH shows radiotracer in the lateral ventricles (cyan curved arrow) with lack of activity over the convexity (cyan solid arrow). Normally, there should be radiotracer movement over the convexities at 24 hours.

• In-111 DTPA cisternography

- Prominent ventricular activity with no flow over convexity at 24-48 hours
- High false-positive rate

