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

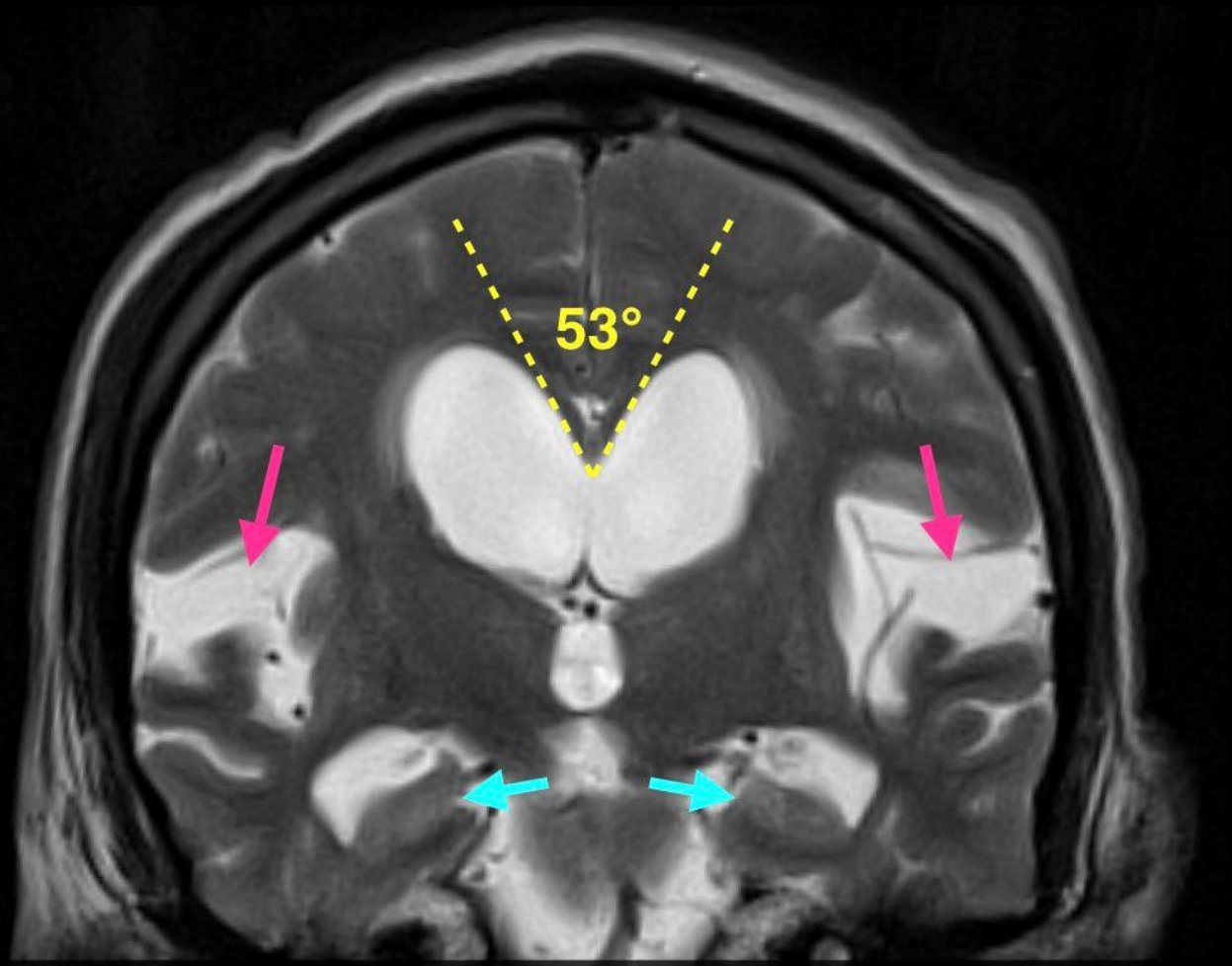
- One theory
  - An obstructive type of communicating hydrocephalus due to reduced CSF resorption.
- A second theory
  - Results from weakening of the ventricular wall due to periventricular white matter ischemic damage .
  - The periventricular white matter ischemic change has also been hypothesized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

Terminolgy

- 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.

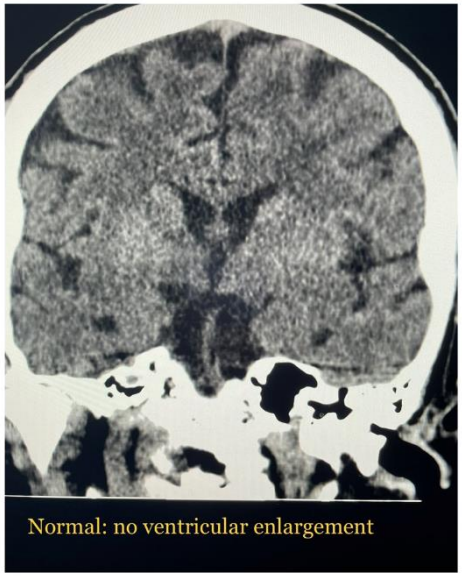
Imaging

- Enlarged lateral & 3rd ventricles, 4th ventricle relatively normal
- **Evans index** (ratio of widest diameter of frontal horns to widest diameter of brain on same axial slice) ≥ 0.3
- **Callosal angle** (angle between lateral ventricles on coronal image at level of posterior commissure) < 90°
- Disproportionately enlarged subarachnoid space hydrocephalus (**DESH**) (particularly sylvian fissures & basal cisterns)
- **Cingulate sulcus sign**: Narrowing of posterior cingulate sulcus compared with anterior
- Tight high convexity with effacement of parafalcine sulci
- Aqueductal flow void
- Aqueduct stroke volume (ASV) > 42 µL

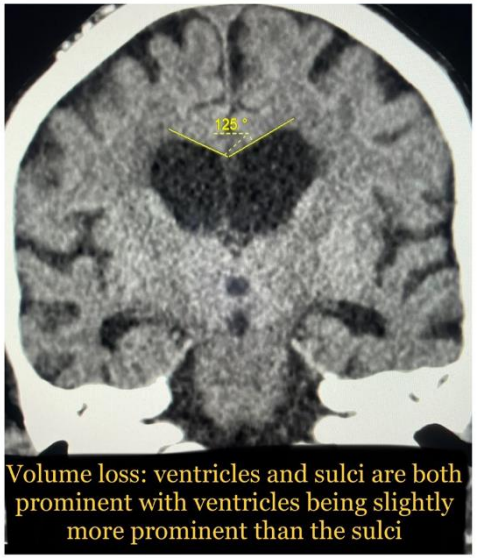


Normal Pressure Hydrocephalus

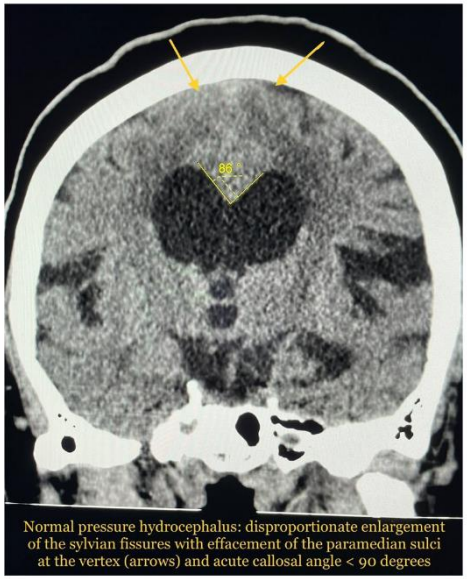
Narrowed callosal angle < 80° and prominent Sylvian fissures relative to other sulci with preserved hippocampal and temporal lobe volume.



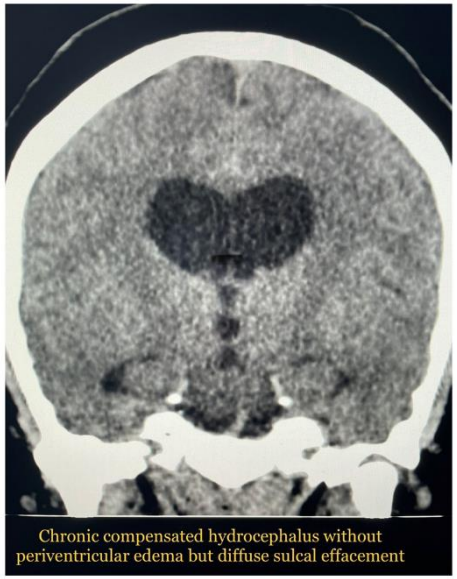
Normal: no ventricular enlargement



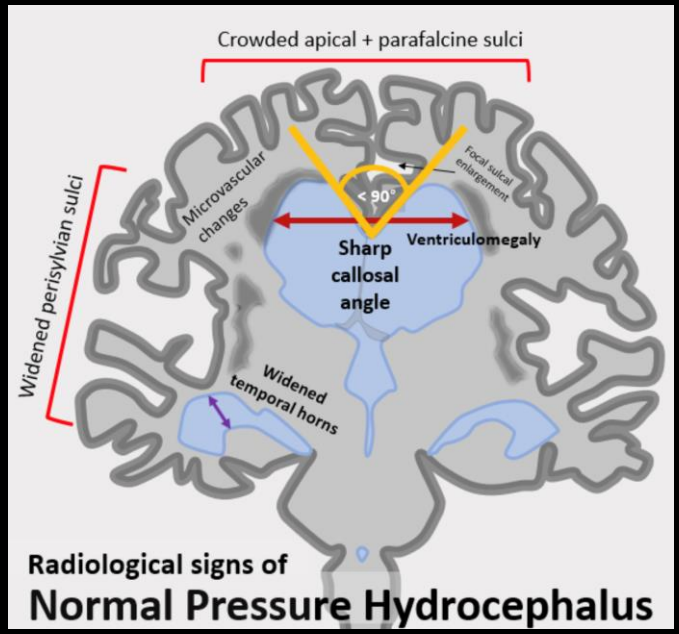
Volume loss: ventricles and sulci are both prominent with ventricles being slightly more prominent than the sulci



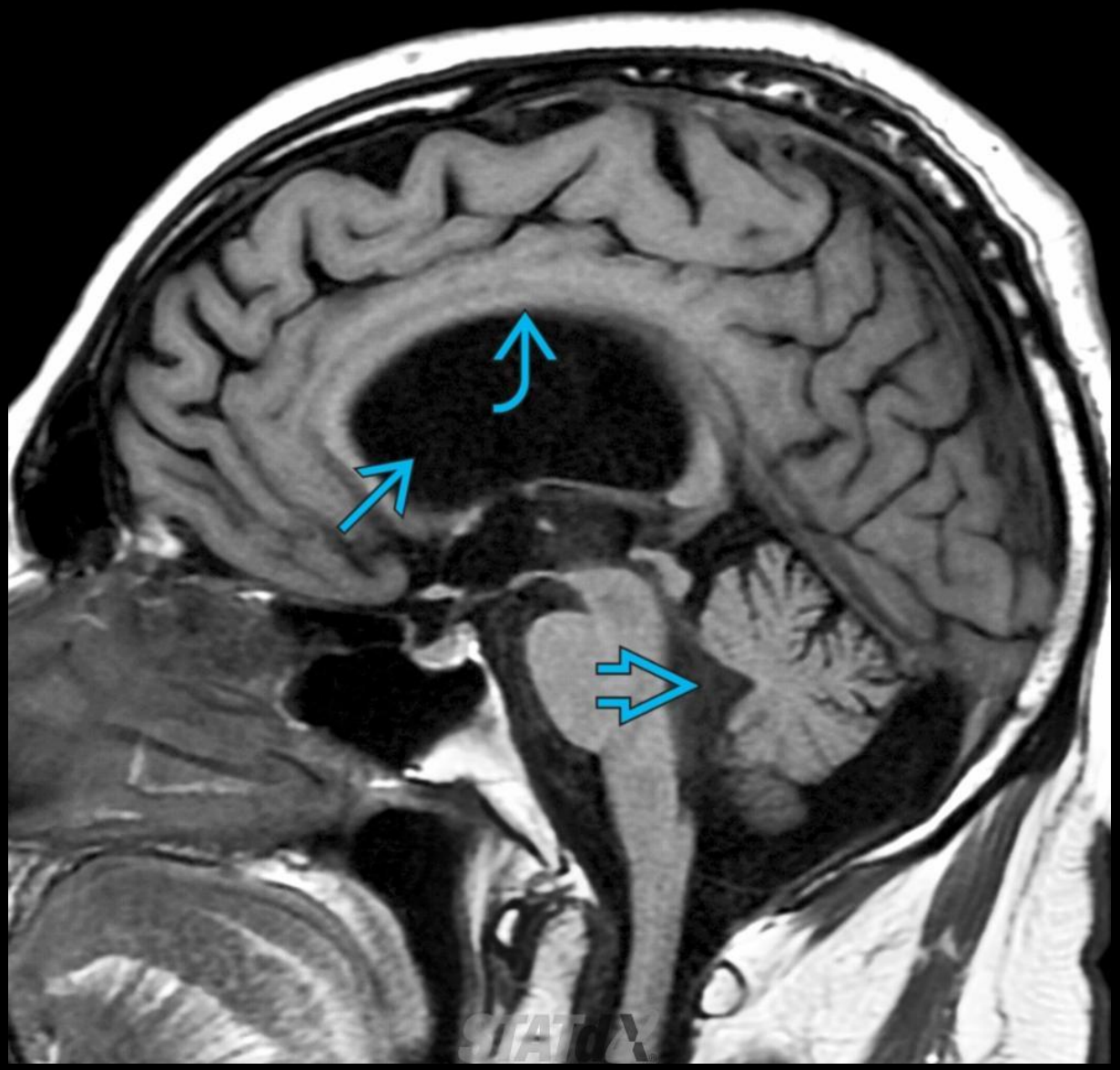
Normal pressure hydrocephalus: disproportionate enlargement of the sylvian fissures with effacement of the paramedian sulci at the vertex (arrows) and acute callosal angle < 90 degrees



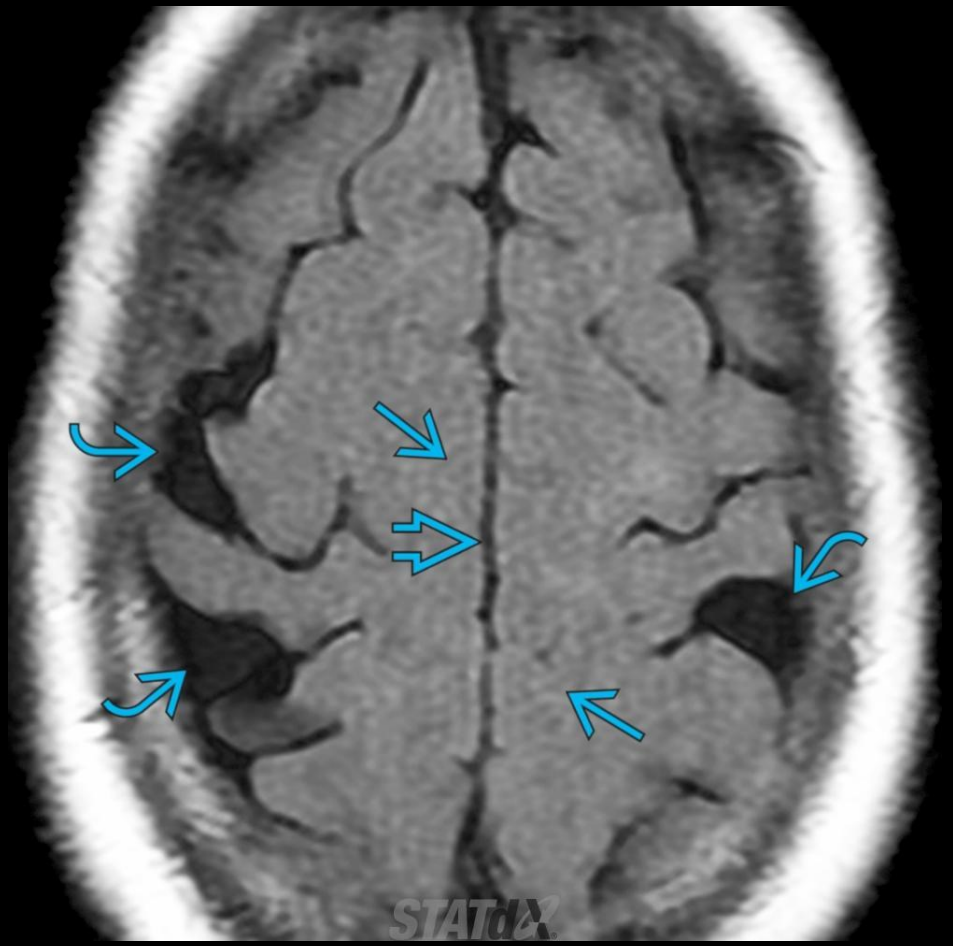
Chronic compensated hydrocephalus without periventricular edema but diffuse sulcal effacement

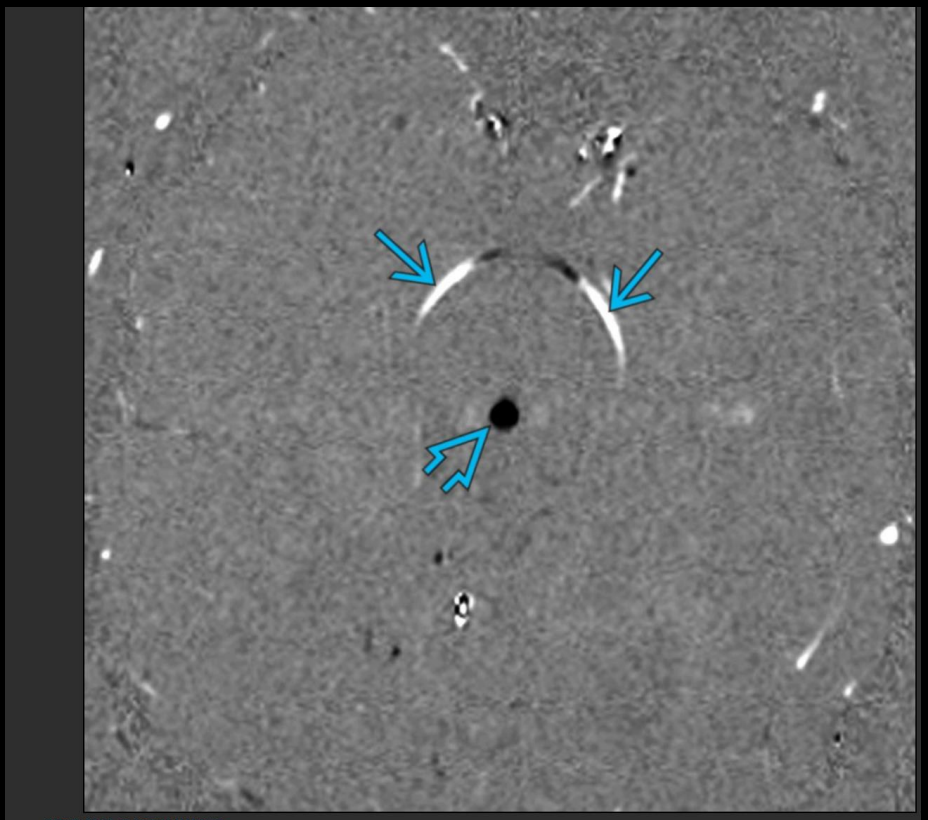


- Large lateral ventricles (cyan solid arrow),
- Thinning of the corpus callosum (cyan curved arrow)
- Relatively normal 4th ventricle (cyan open arrow) in a patient with iNPH.



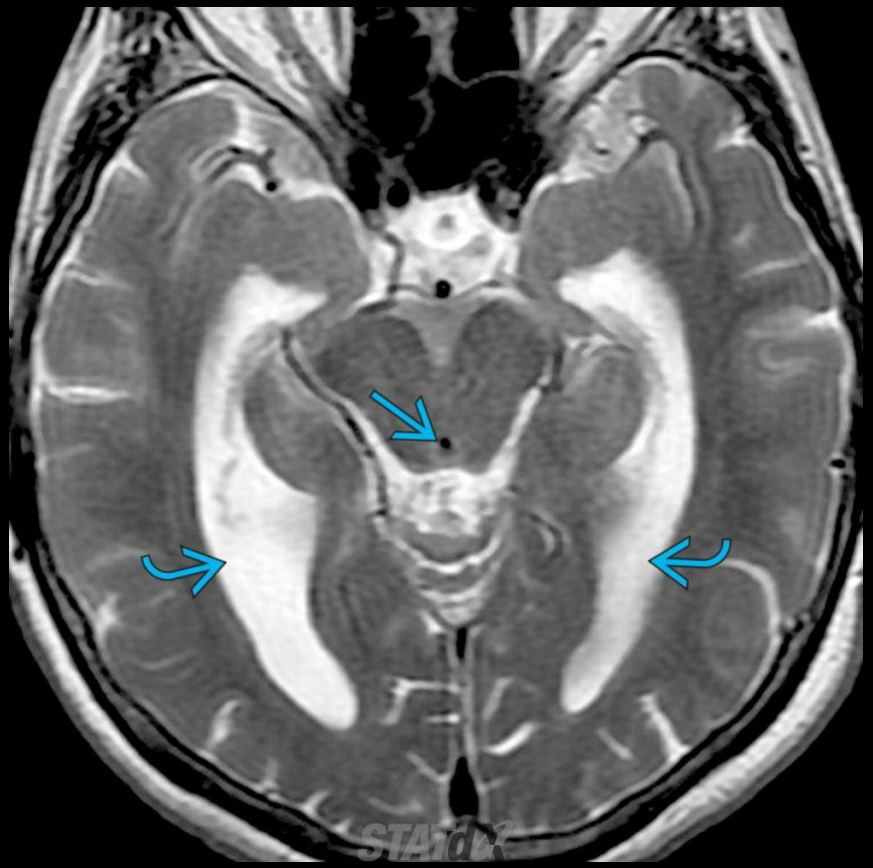
Axial FLAIR MR in the same patient demonstrates disproportionately enlarged subarachnoid spaces (cyan curved arrow), consistent with DESH, and narrowing of the sulci and subarachnoid spaces (cyan solid arrow) over the high convexity parasagittal frontoparietal regions with a tight interhemispheric fissure (cyan open arrow).



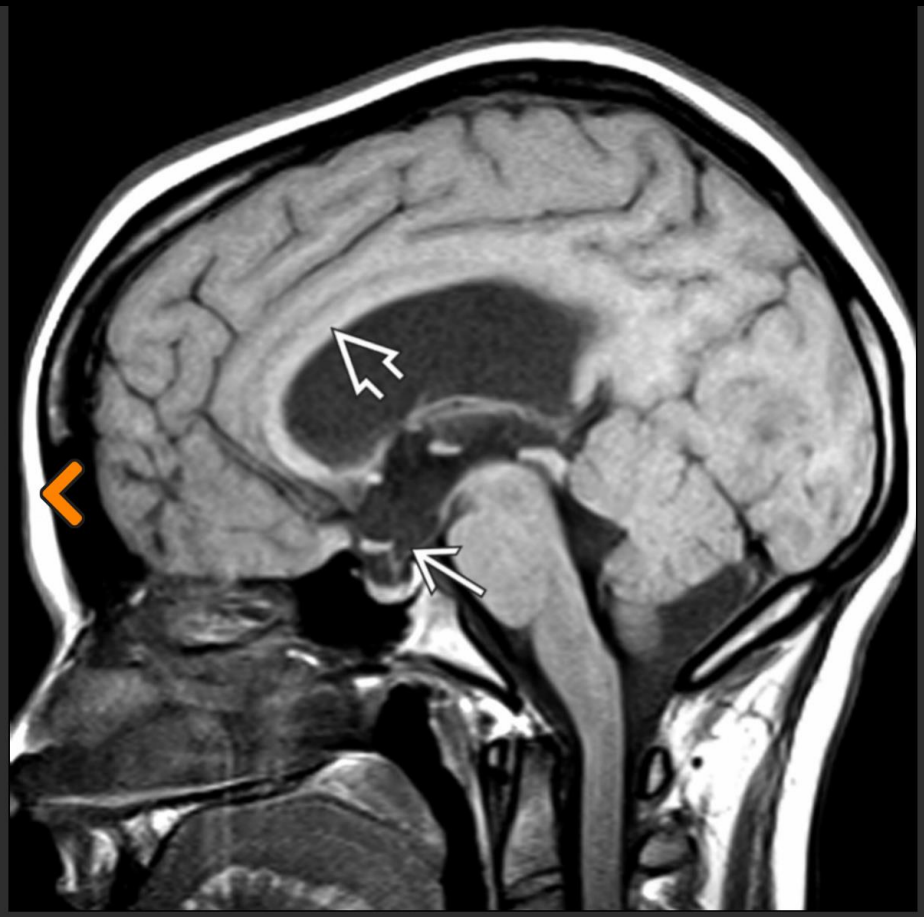


View Full Screen Image

Axial phase-contrast cine MR CSF flow study shows increased velocity of CSF through the dilated aqueduct ⇨. There is more hyperdynamic flow through the aqueduct than the cisterns, where no high-velocity signal change is seen. Flow is incidentally noted in the posterior cerebral arteries →.

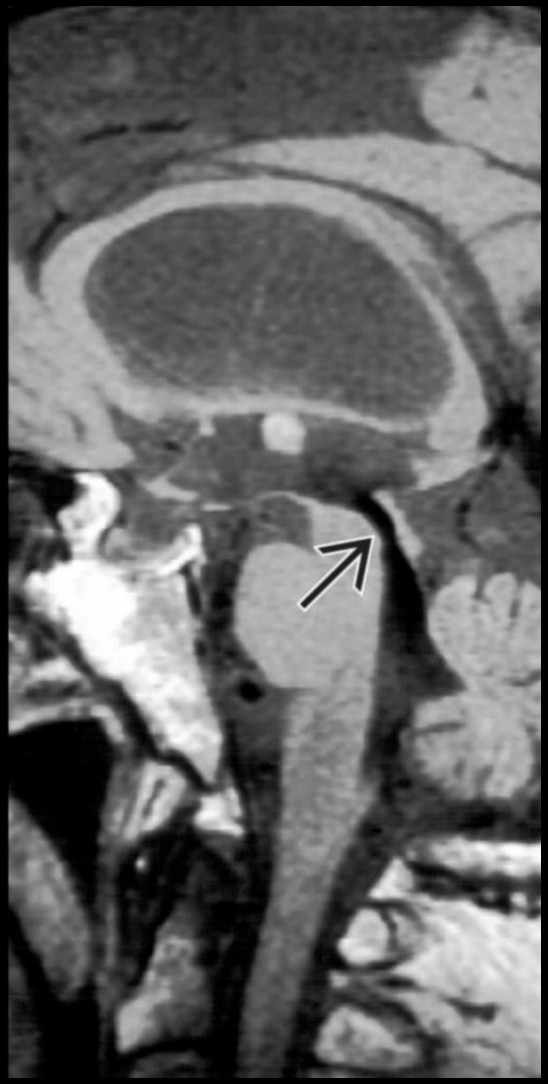


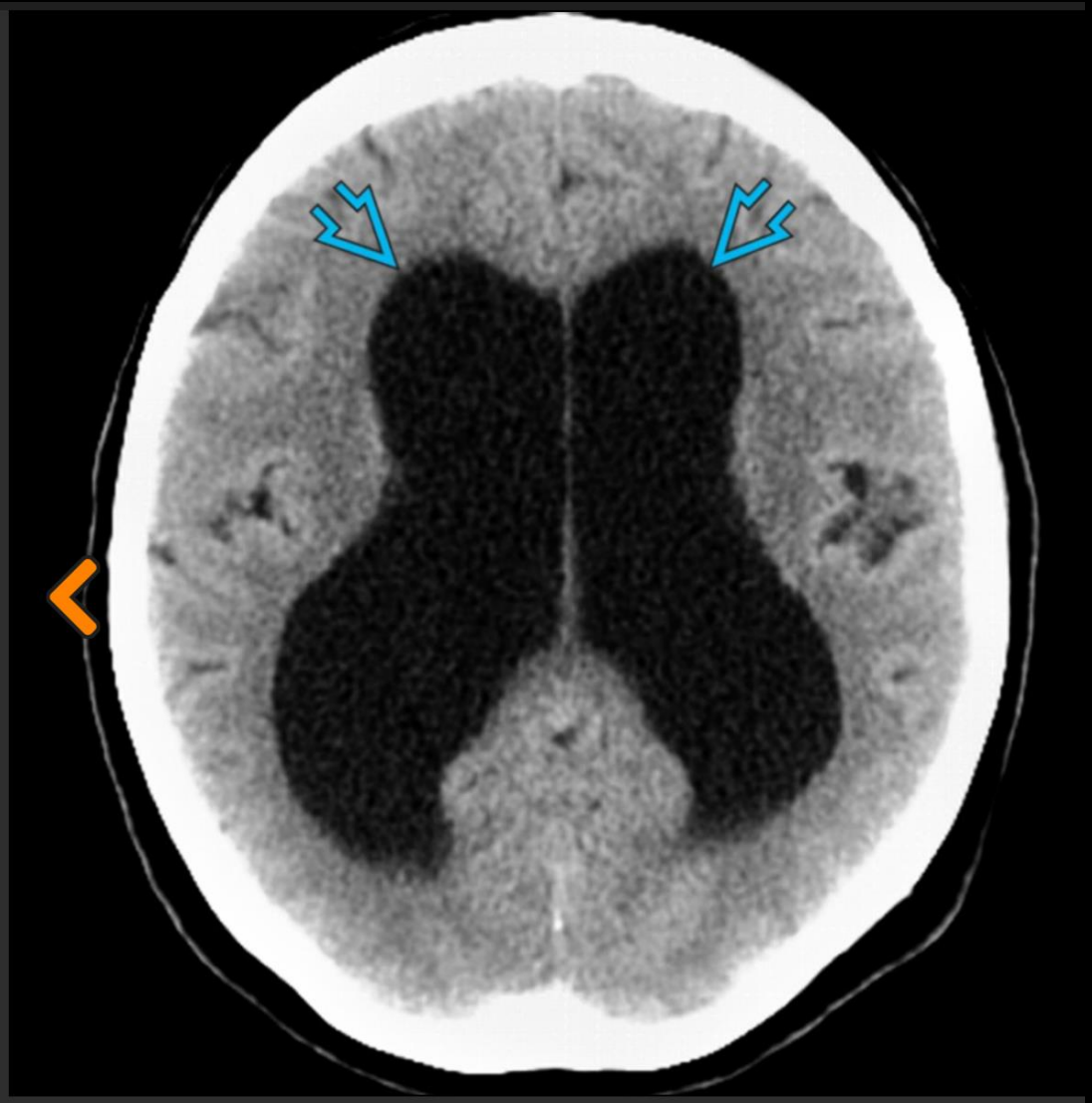
Axial T2 MR in 65 year old with NPH shows dilated temporal horns (cyan curved arrow) and low-signal flow void (cyan solid arrow) in the aqueduct caused by hyperdynamic flow of CSF.



View Full Screen Image

Sagittal T1WI MR shows enlargement of the 3rd and lateral ventricles. The infundibular recess → is enlarged and bulges downward. Note mild thinning of the corpus callosum ⇨.

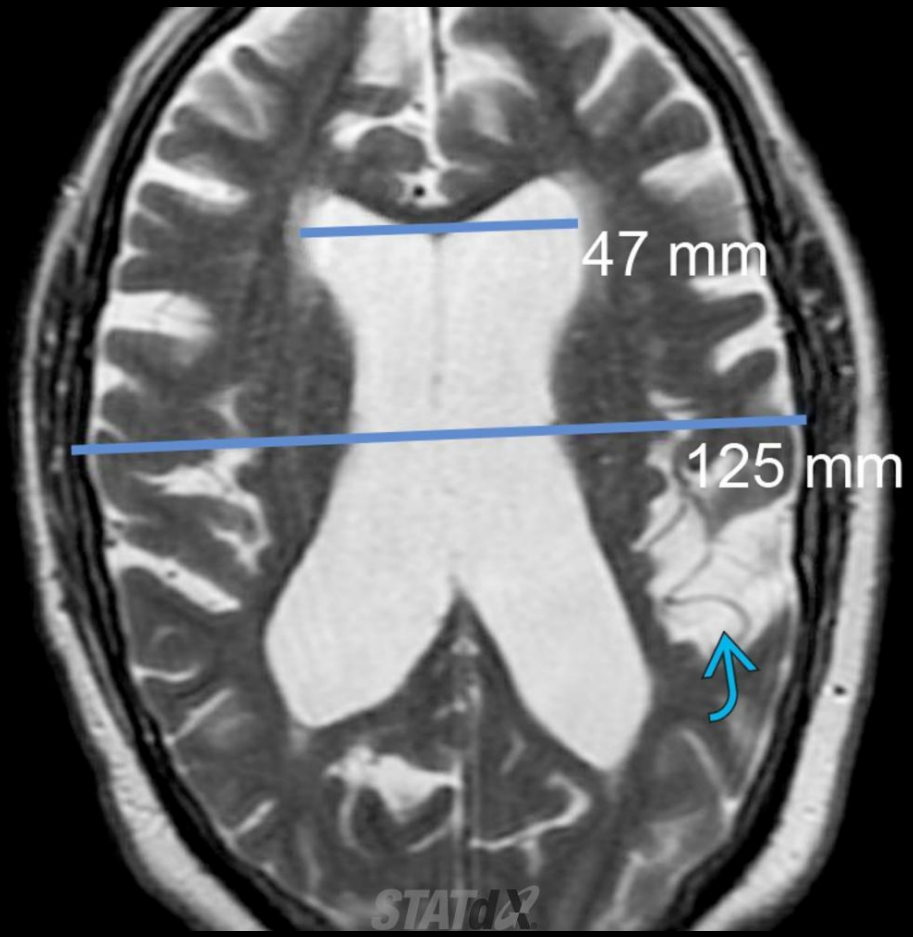




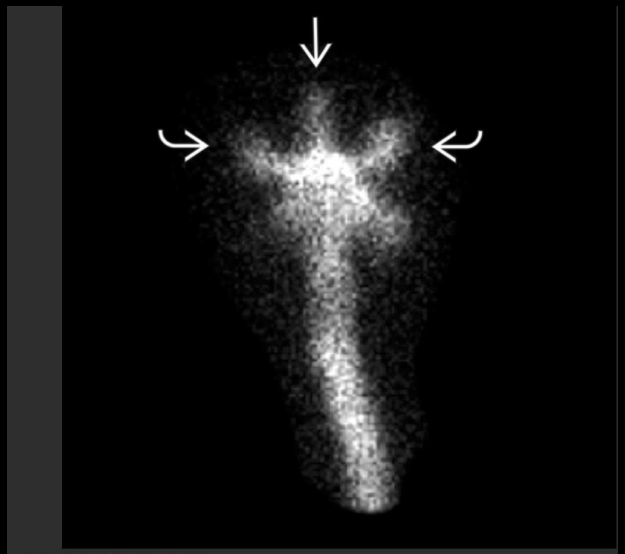
View Full Screen Image

Axial NECT shows large ventricles out of proportion to the sulcal prominence with a rounded appearance of th frontal horns ⇨.

- Demonstrates lateral ventricular enlargement and disproportionately enlarged sylvian fissure (cyan curved arrow) (DESH).
- Evans index, which is the ratio of the maximum width of the frontal horns to the maximum internal diameter of the skull at the same level, measures 0.38.
- Normal Evans index is < 0.3.

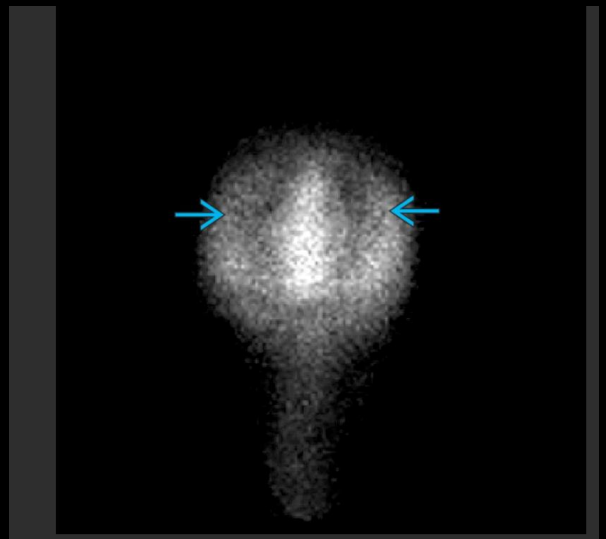


Normal



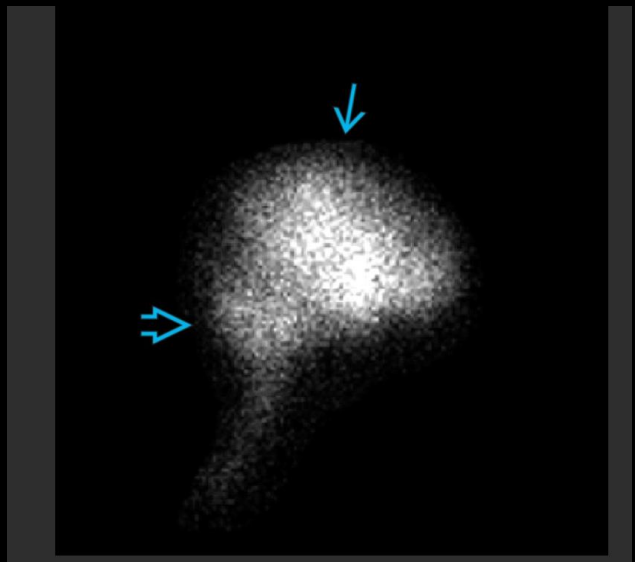
View Full Screen Image

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



View Full Screen Image

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



View Full Screen Image

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 ⇨.

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