

Neuroimaging in Clinical Neuropsychiatry

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Disclosures

Neither I nor my spouse has a relevant financial relationship with a commercial interest to disclose.

Objectives

Imaging tools to assess patients with
neuropsychiatric illness

Structural imaging

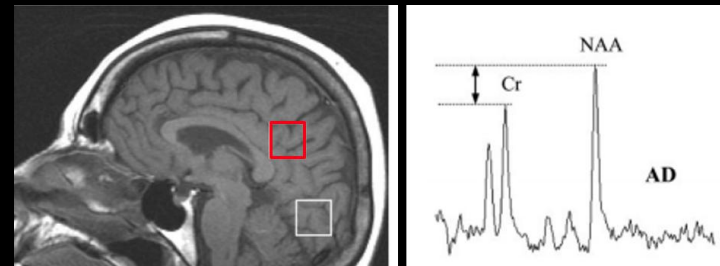
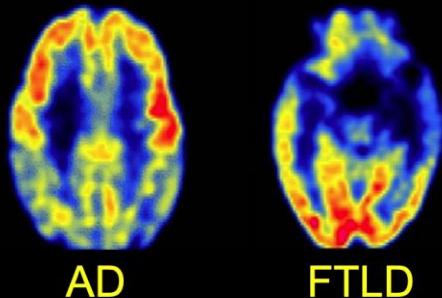
Metabolic imaging

Objectives

Imaging tools to assess patients with neuropsychiatric illness

Structural imaging (CT, MRI)

Metabolic imaging (PET, MRS)

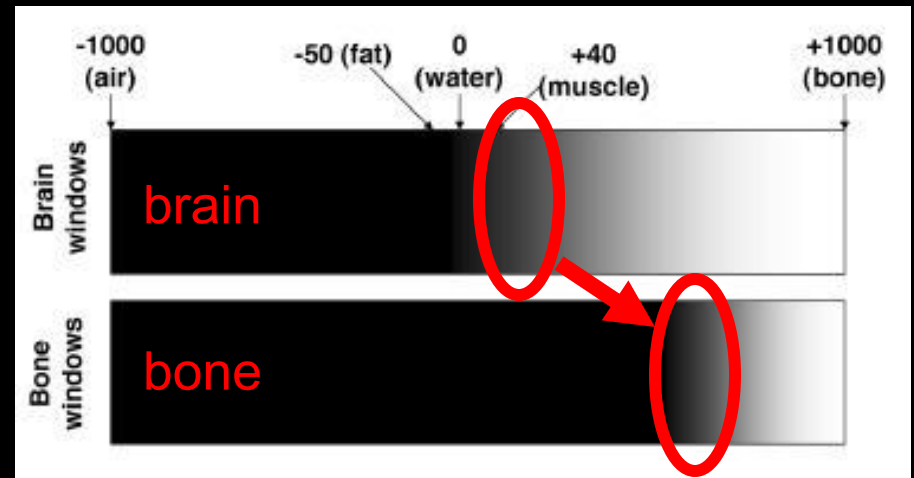


Structural imaging principles

1. *Clinical correlation is key*
2. *Assess brain volume, increased or decreased?*
3. *Increased → mass or edema, Decreased → atrophy*
4. *Is atrophy focal, regional, diffuse?*
5. *Interval changes*

CT: Hounsfield units

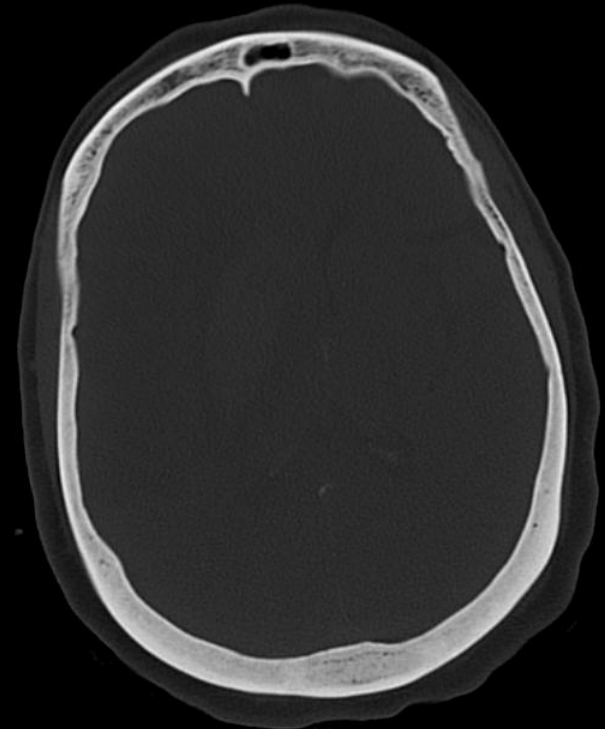
Cerebrospinal fluid	15
White matter	20 to 30
Gray matter	35 to 45
Acute hemorrhage/thrombus	60 to 100



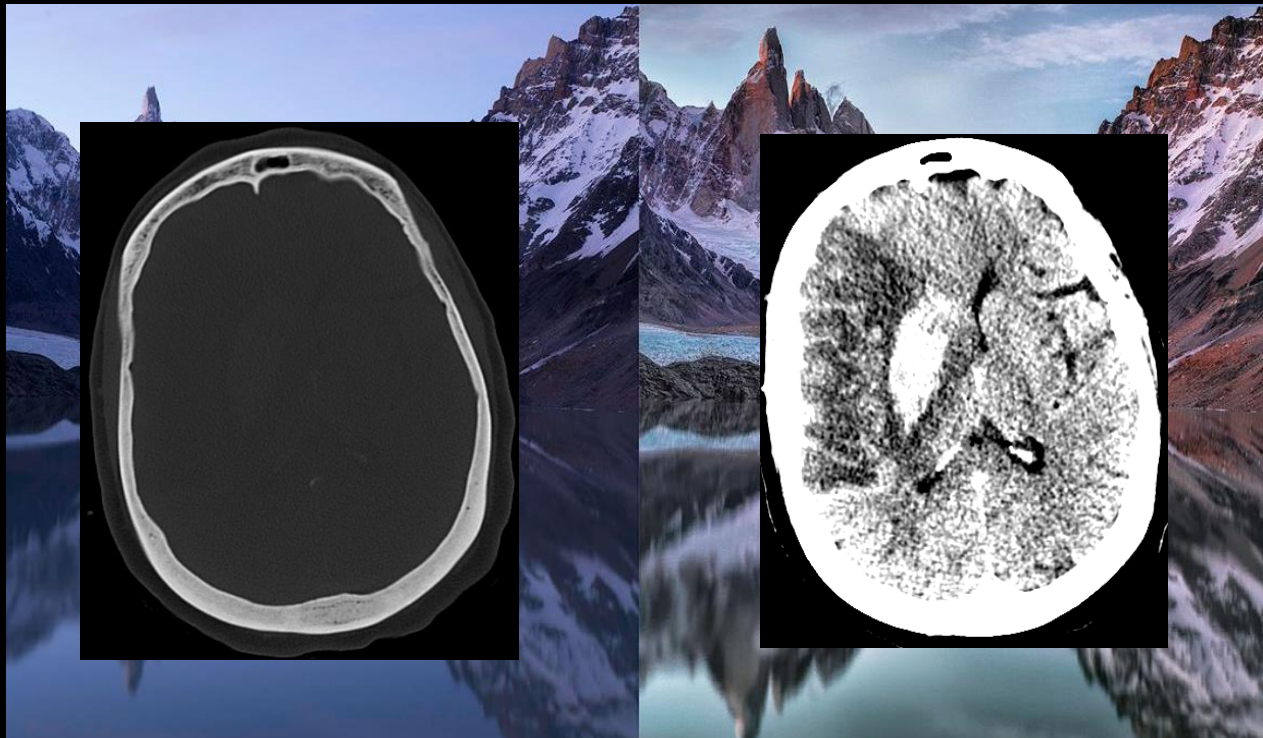
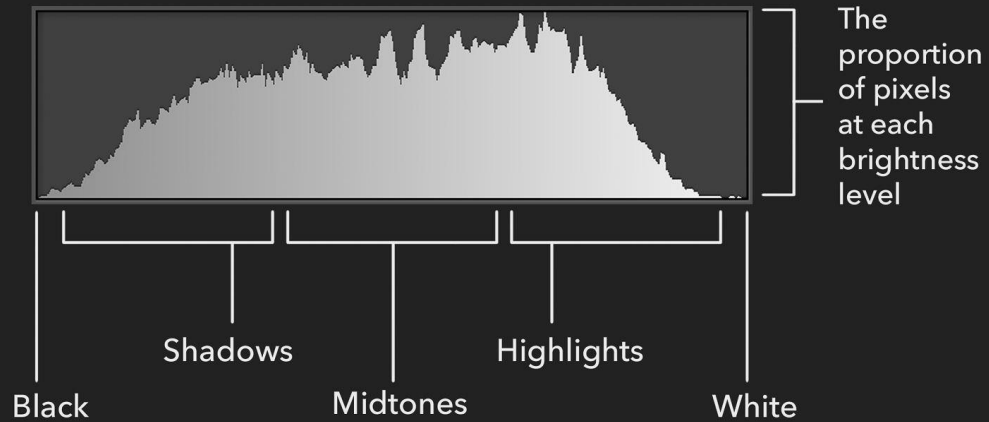
brain



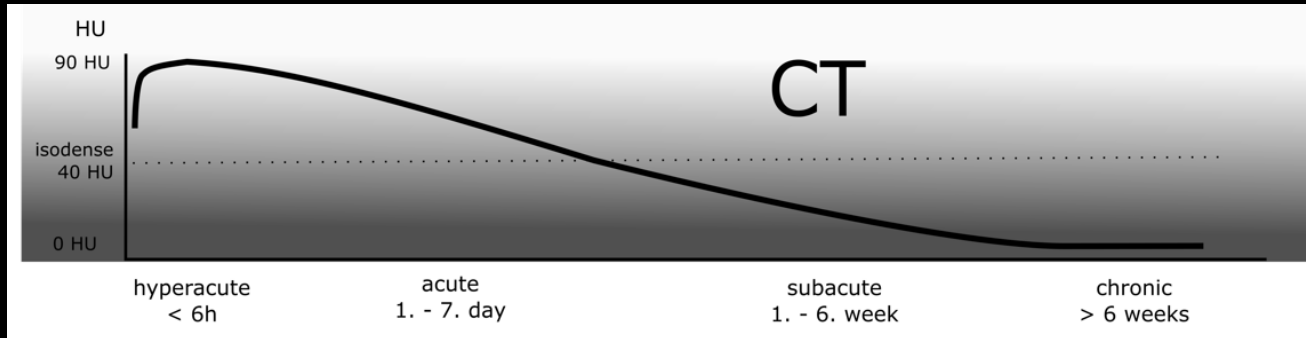
bone



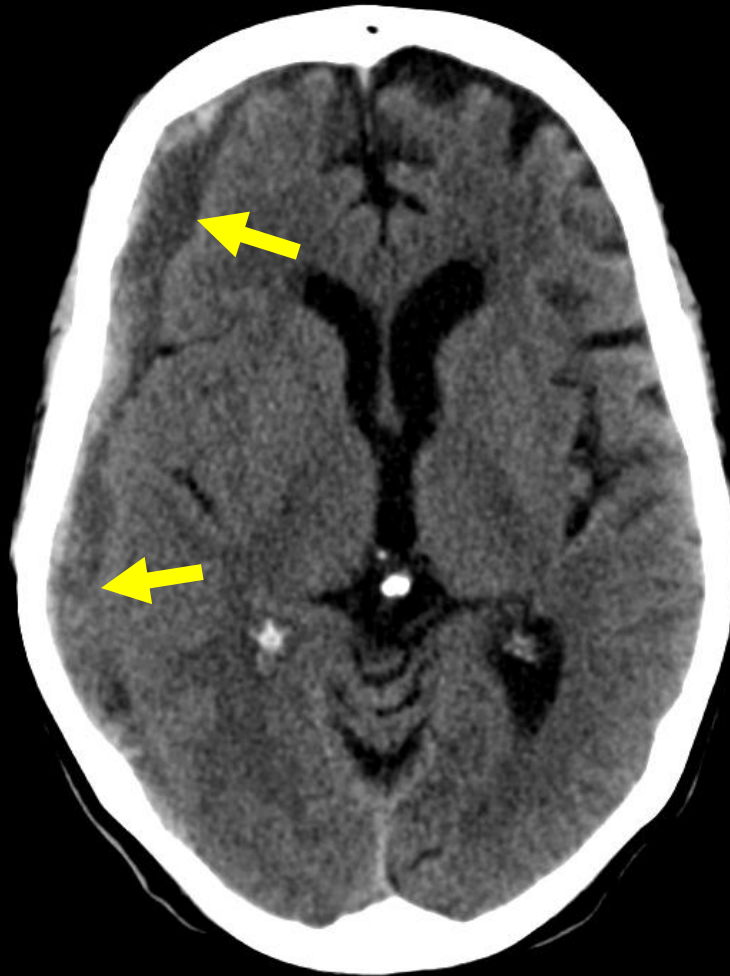
The Anatomy of a Histogram



Imaging hemorrhage on CT

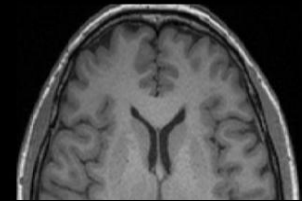


Intracranial hemorrhage, subdural

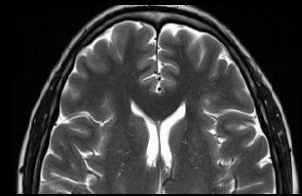


MRI

T1: fat appears brighter than water, so WM is brighter than GM. Edema and CSF are darker than normal brain.



T2: fat appears darker than water, so WM is darker than GM. Edema and CSF are brighter than normal brain.

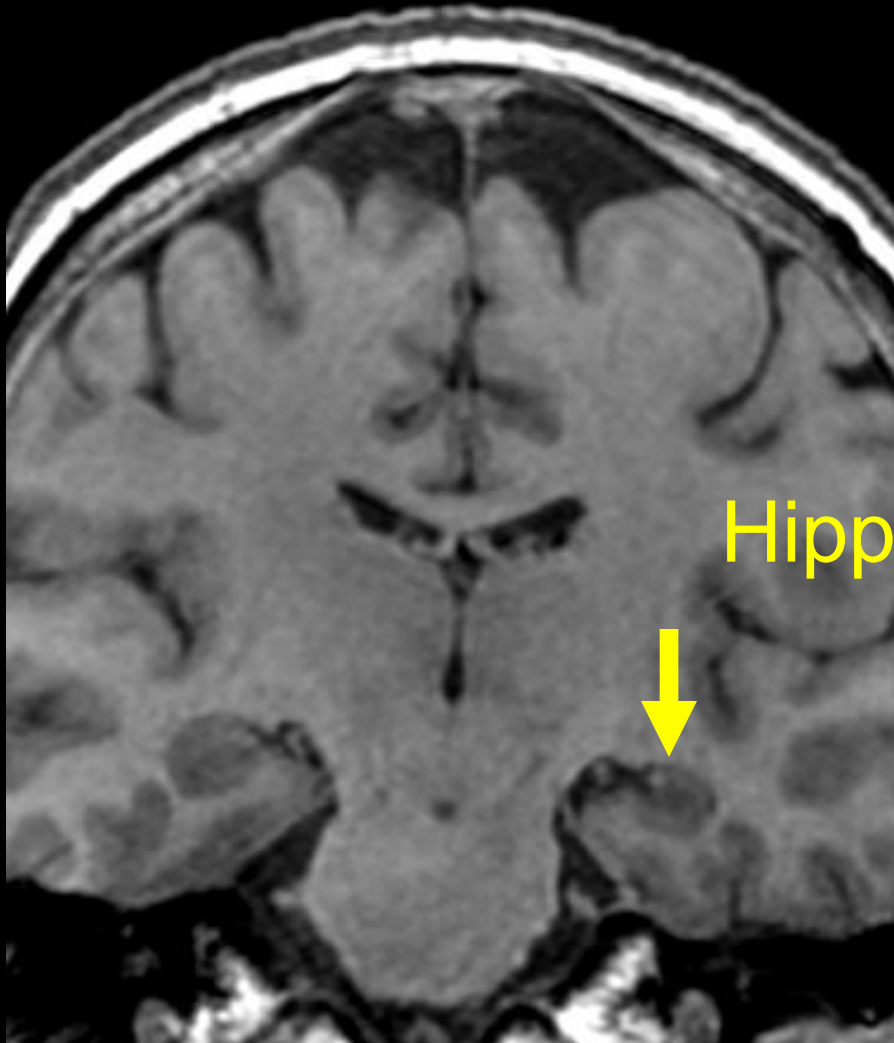




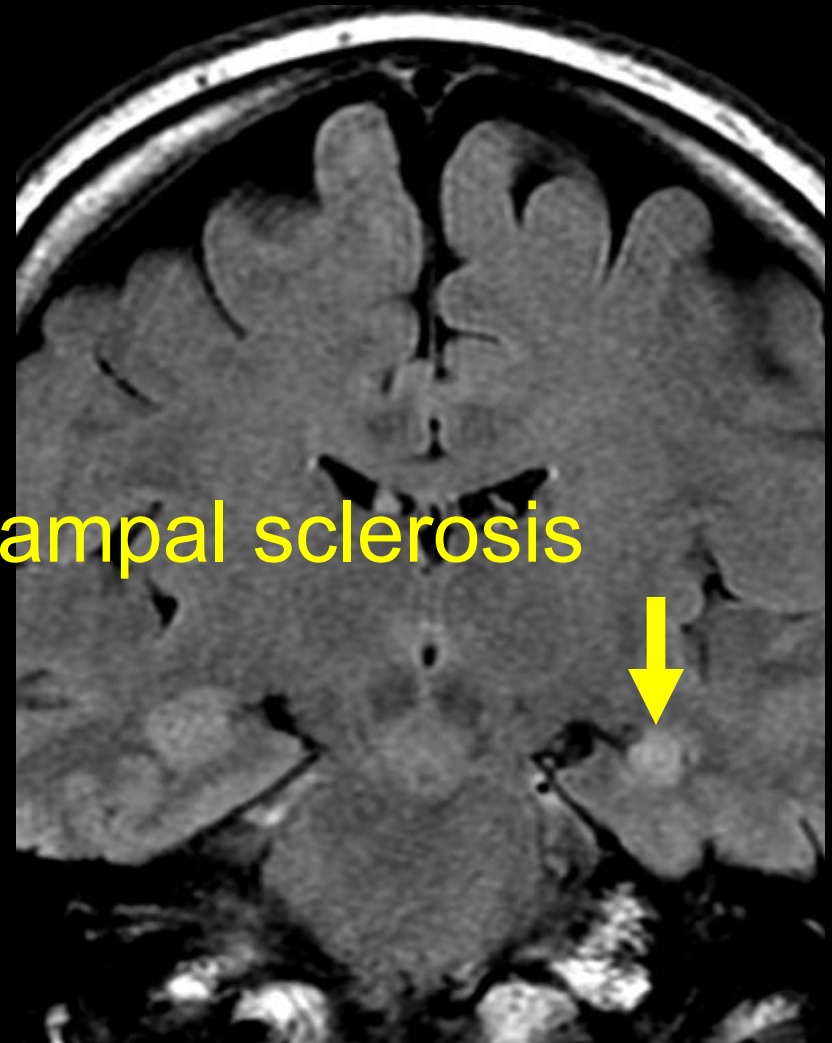
T1 “*shortening*” (hyperintensity): fat, protein, contrast, sub-acute blood, some metals

T2 “*prolongation*” (hyperintensity): edema, gliosis, sub-acute blood

Coronal T1



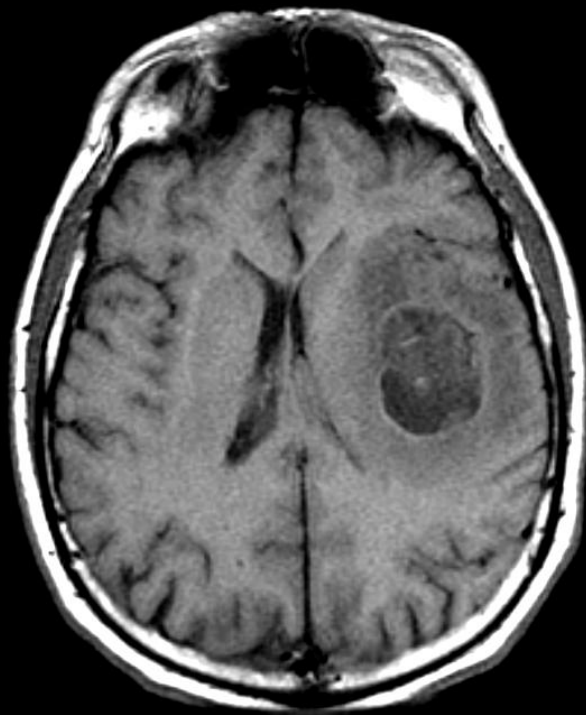
Coronal T2-FLAIR



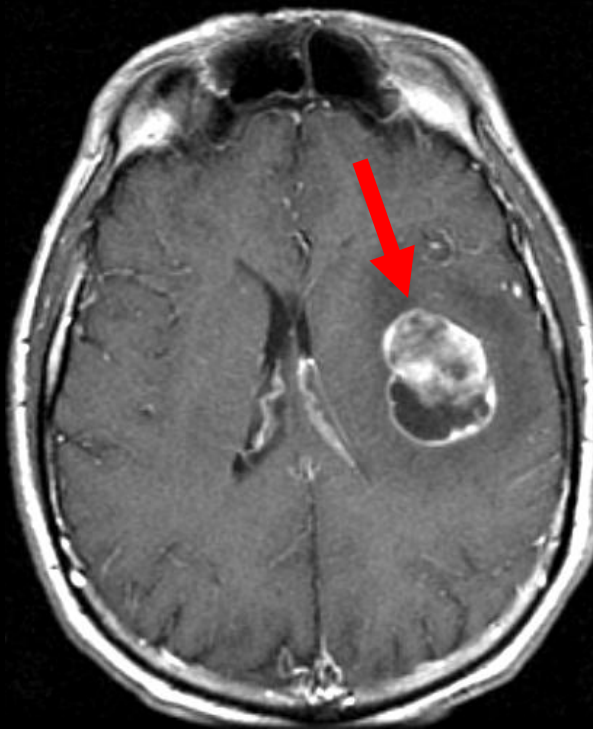
Hippocampal sclerosis

MRI

T1

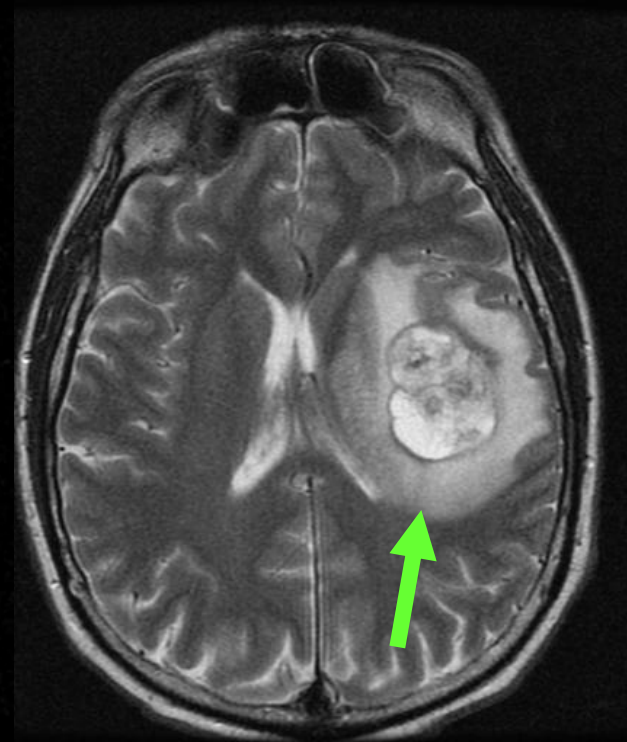


T1 with gadolinium



T1 shortening
(enhancement)

T2



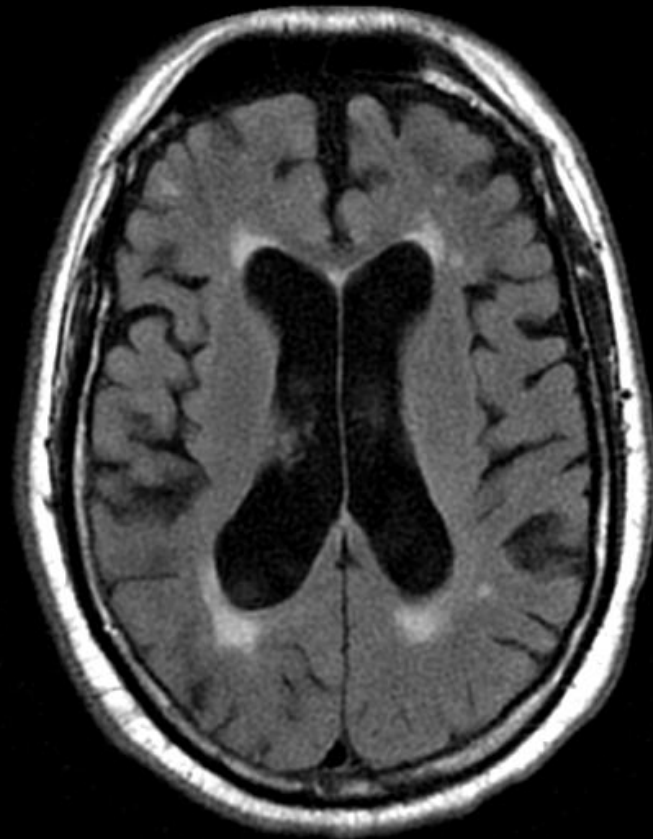
T2 prolongation
(edema)

Fluid attenuated inversion recovery (FLAIR)

T2



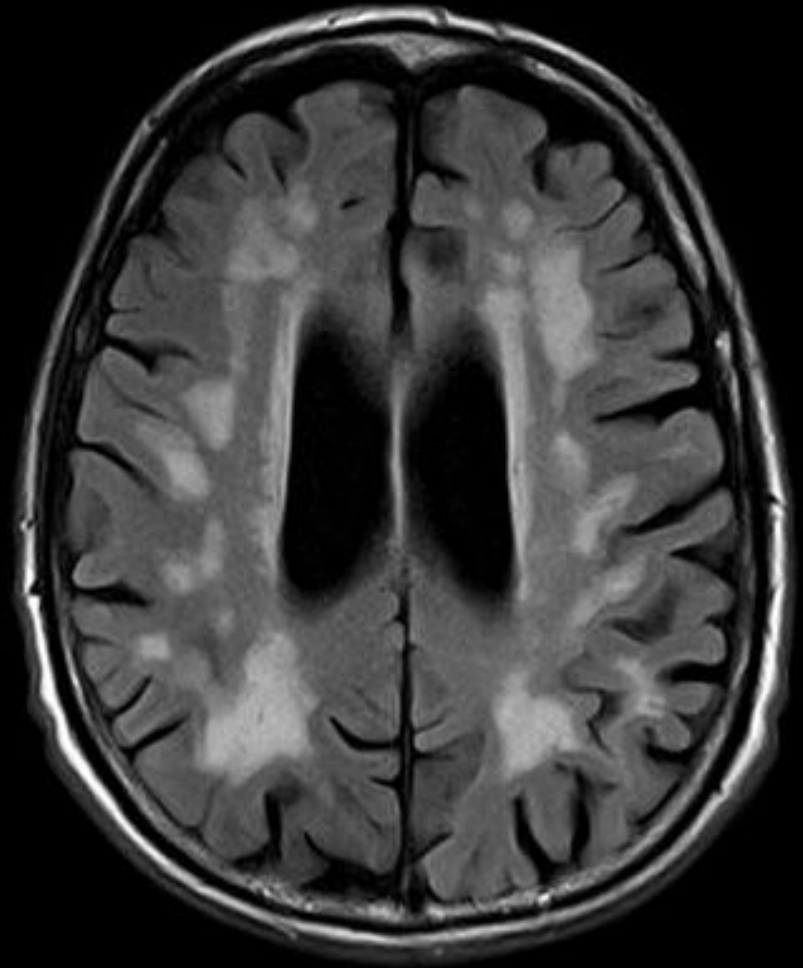
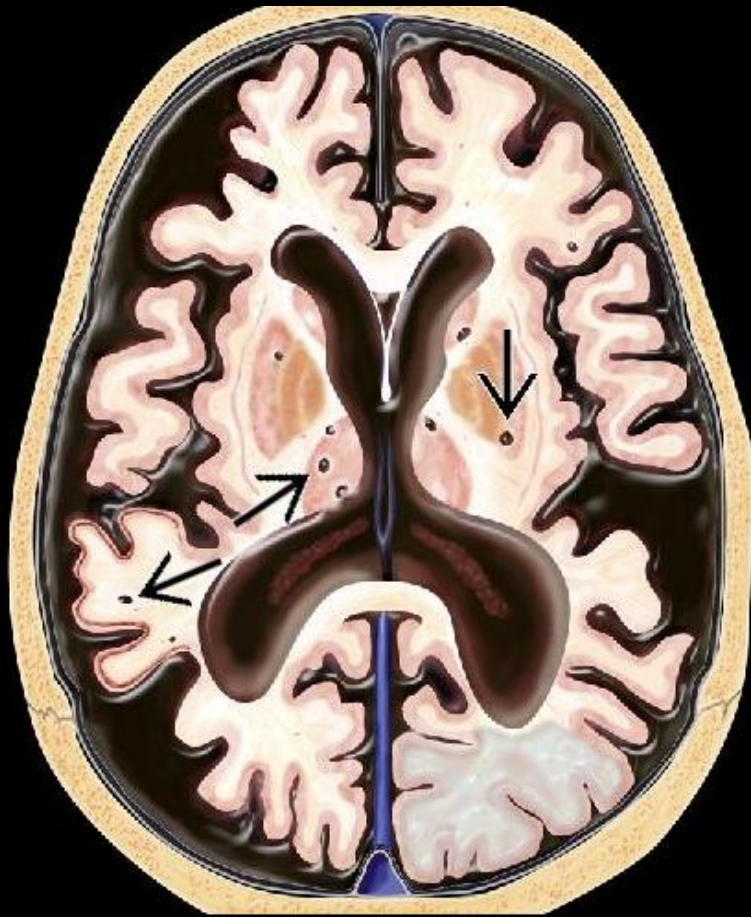
T2-FLAIR



Vascular disease in neuropsychiatry

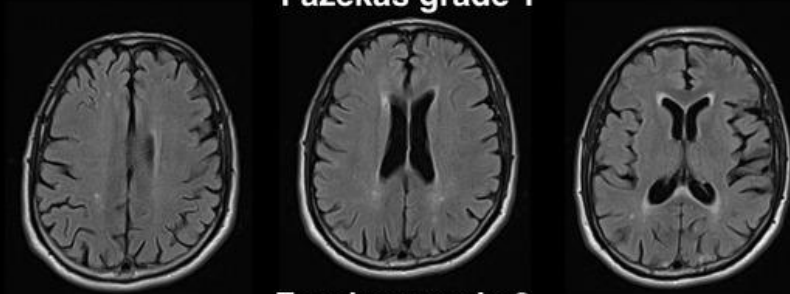
1. *Microangiopathy*
2. *Intracranial hemorrhage*
3. *Cerebral amyloid angiopathy*
4. *Superficial siderosis*

Microangiopathy

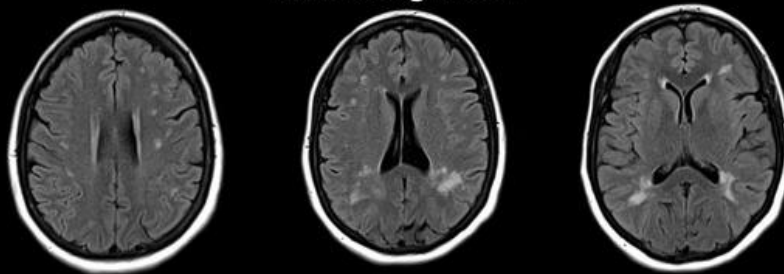


White matter lesion volume assessment

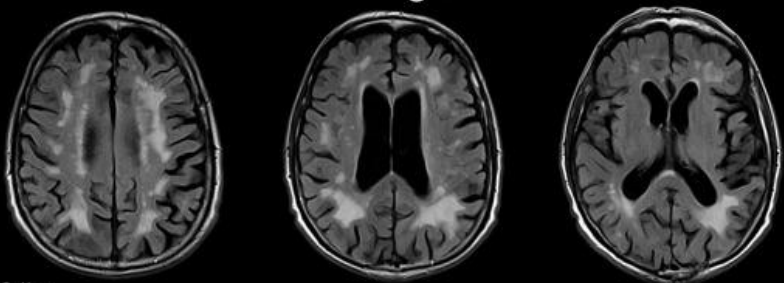
Fazekas grade 1



Fazekas grade 2



Fazekas grade 3



B. Di Muzio

periventricular white matter (PVWM)

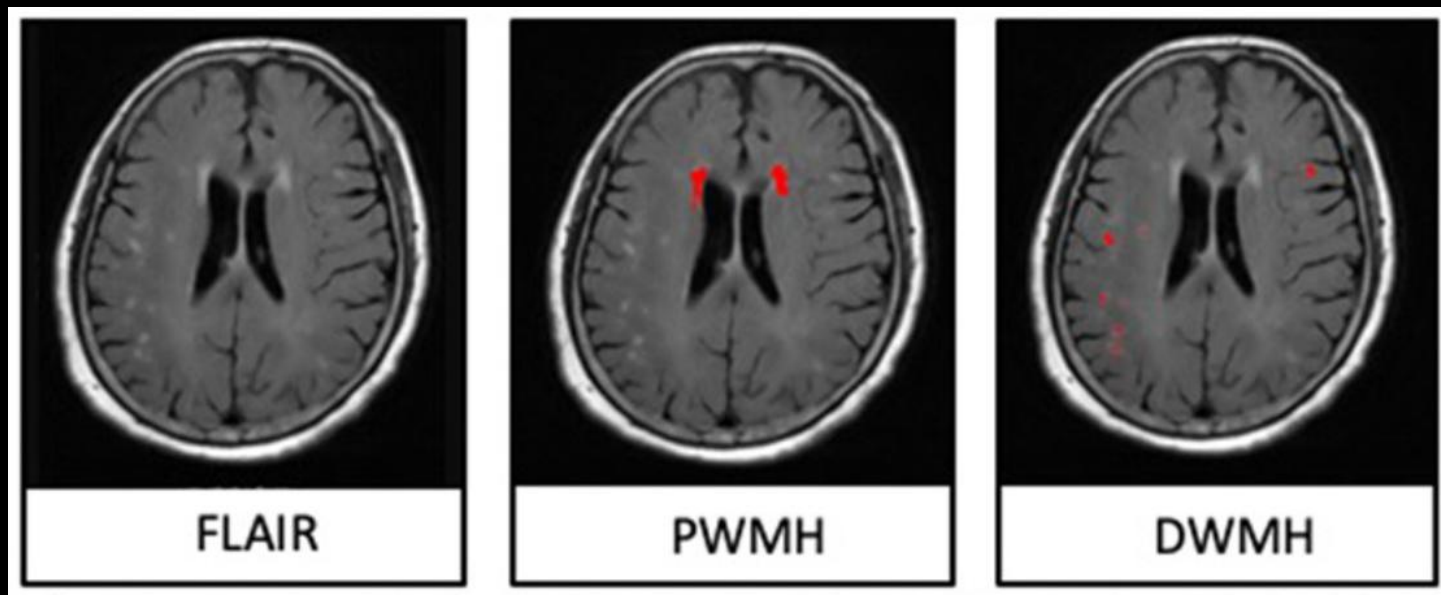
- 0 = absent
- 1 = caps or pencil-thin lining
- 2 = smooth "halo"
- 3 = irregular periventricular signal extending into the deep WM

deep white matter (DWM)

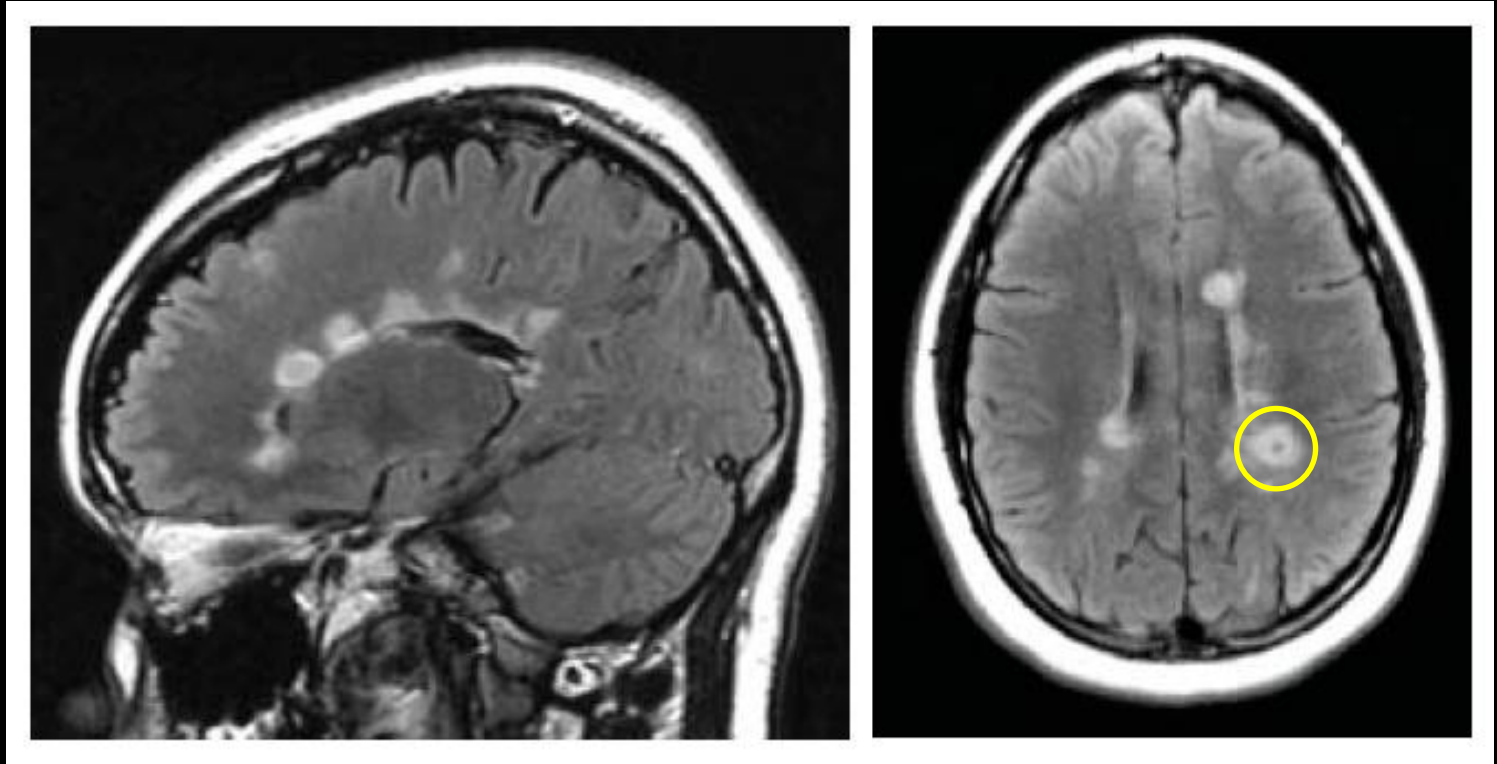
- 0 = absent
- 1 = punctate foci
- 2 = beginning confluence
- 3 = large confluent areas

Etiology of periventricular and deep white matter changes

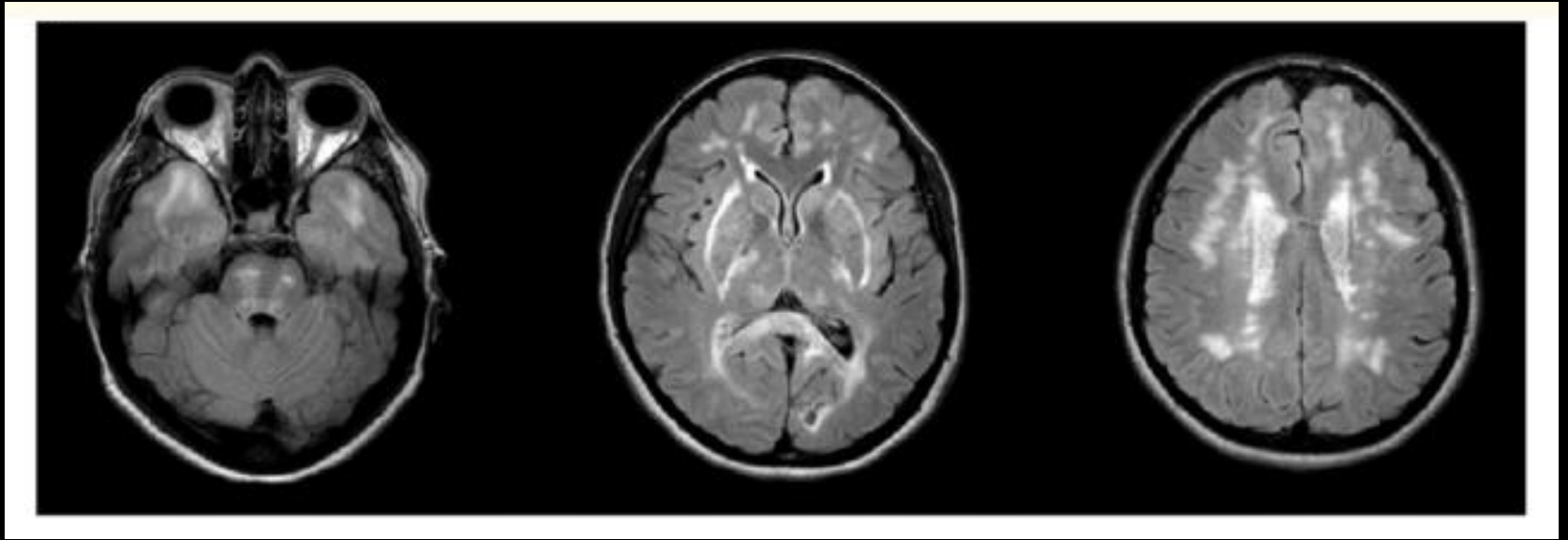
- Periventricular WM hyperintensity → combination of chronic demyelination, sub-ependymal gliosis, and small vessel ischemia
- Deep WM hyperintensity → small vessel ischemia (microangiopathy, leukoariosis)



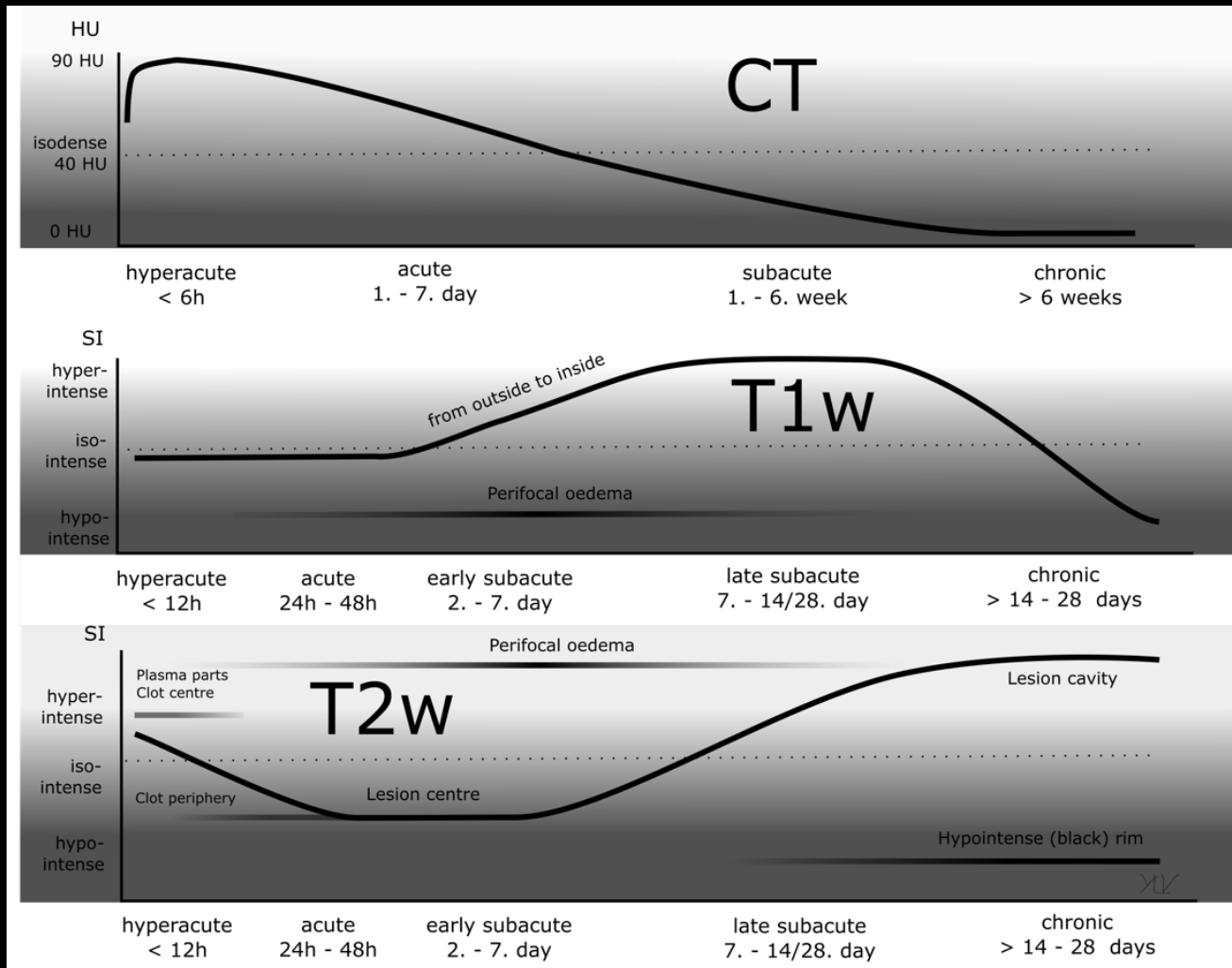
Multiple sclerosis



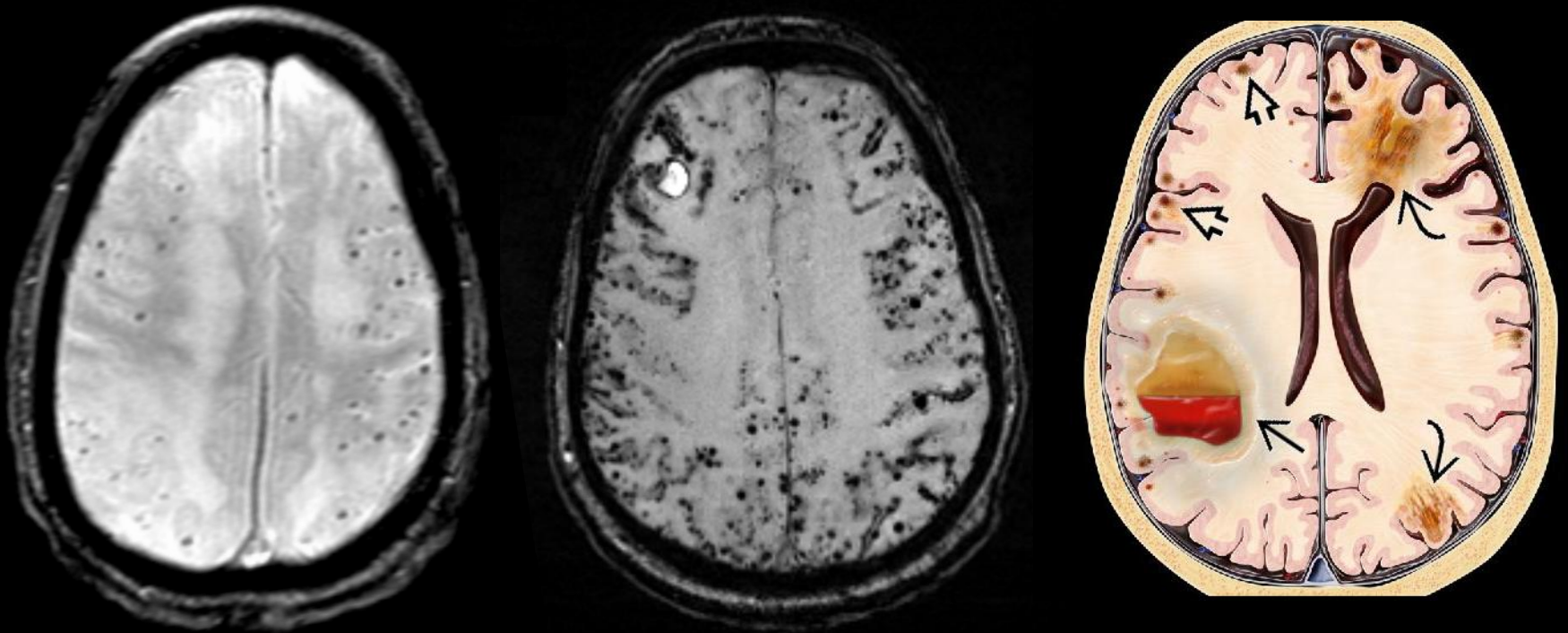
CADASIL (cerebral autosomal dominant arteriopathy with subcortical infarctions and leukoencephalopathy)



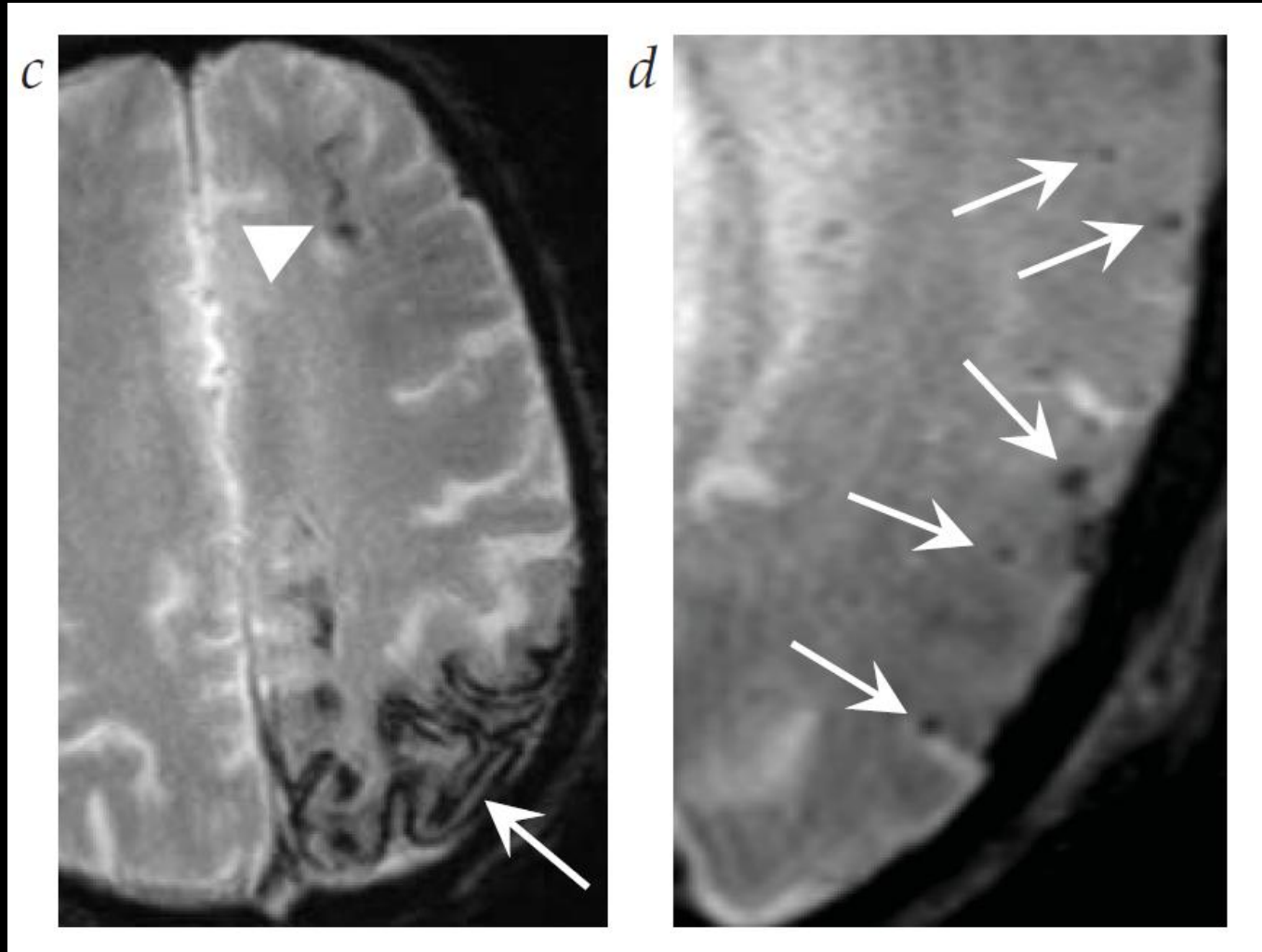
Imaging hemorrhage on CT and MRI

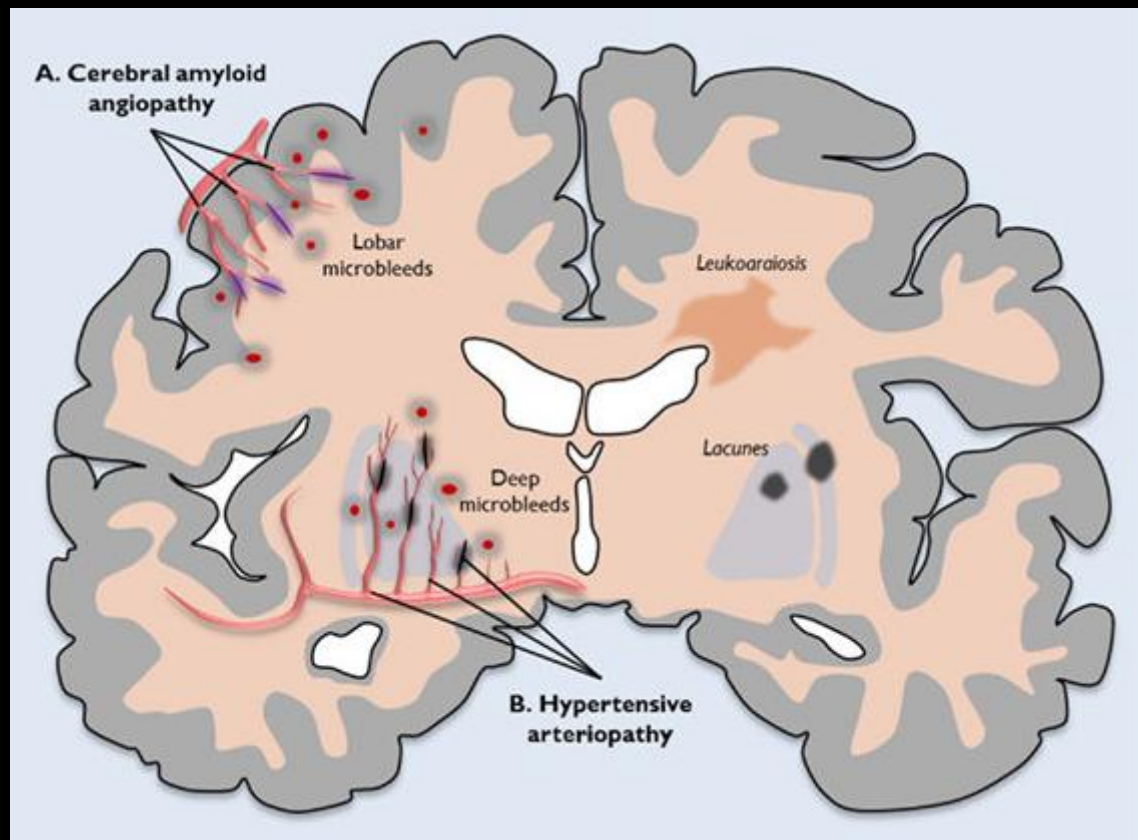


Cerebral amyloid angiopathy (CAA)

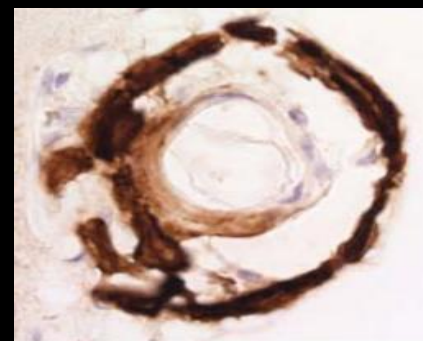


Superficial siderosis in CAA





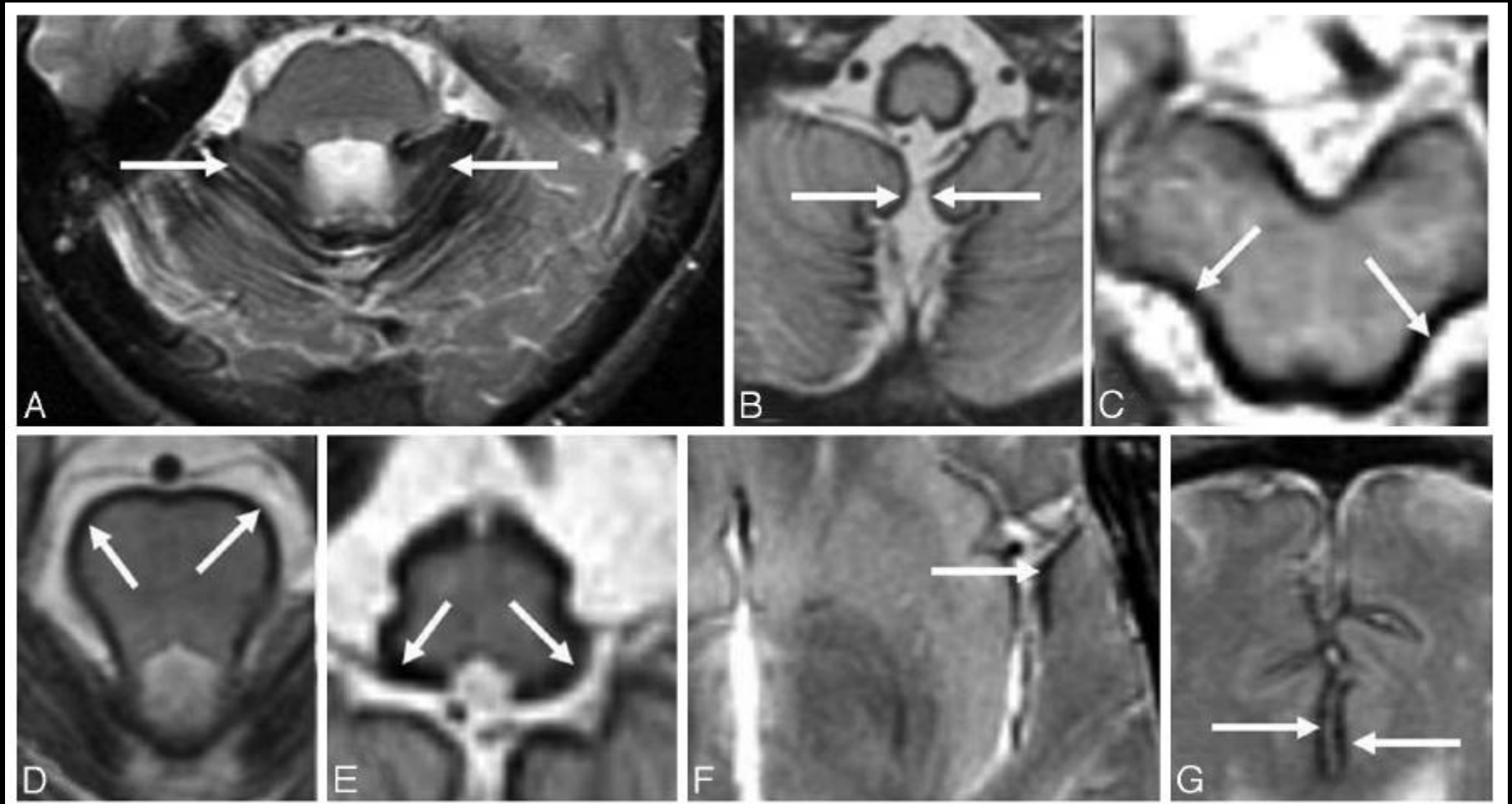
Amyloid angiopathy



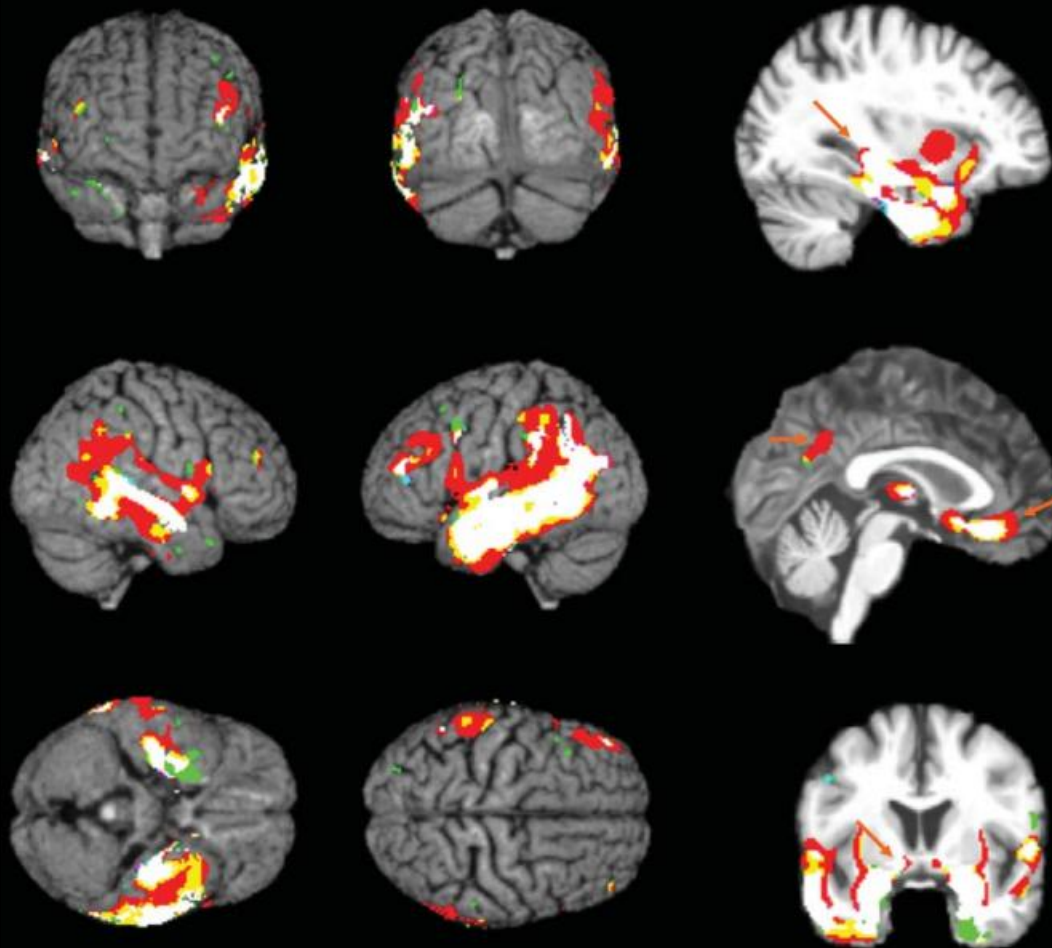
Hypertension



Superficial siderosis

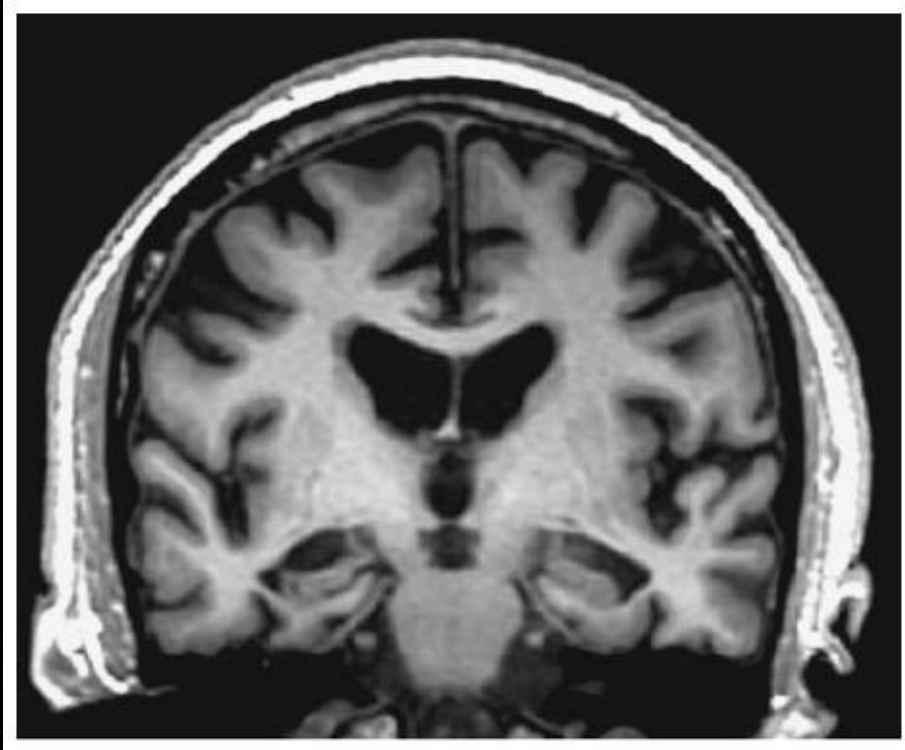


Cortical atrophy in AD, *volumetry*

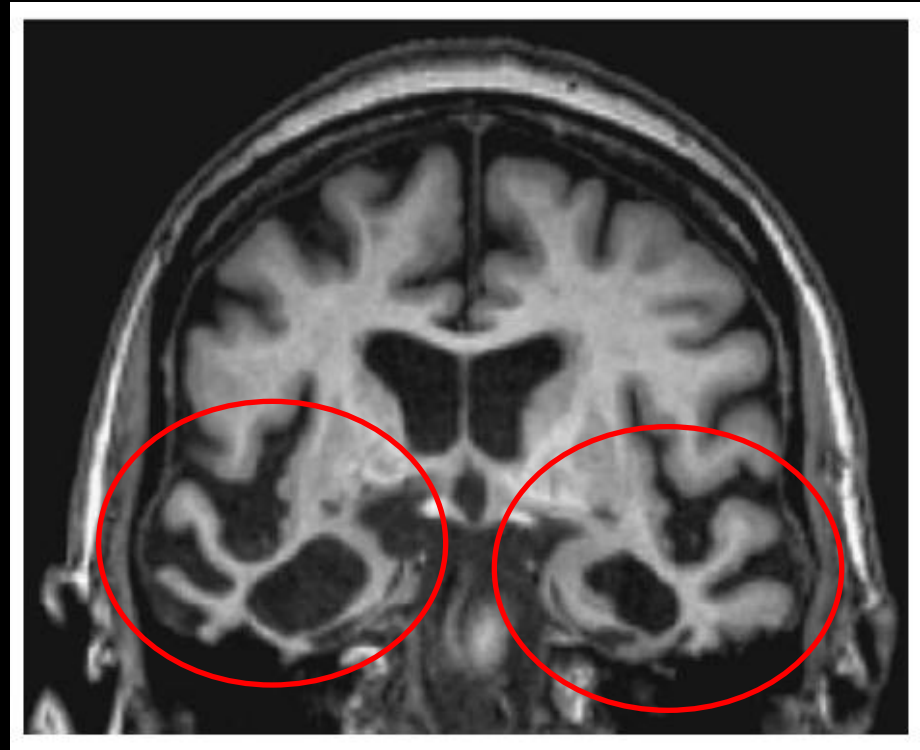


- *Hippocampus*
- *Entorhinal cortex*
- *Posterior cingulate*
- *Precuneus*
- *Basal forebrain*

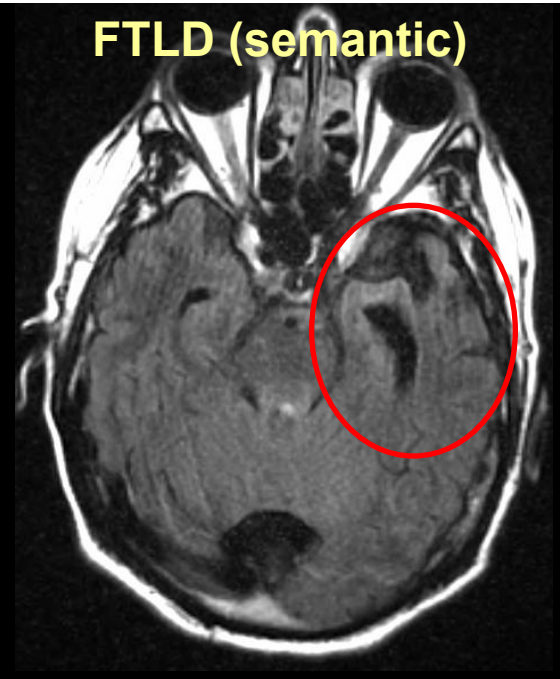
AD



FTLD



FTLD (semantic)



FTLD (behavioral)



**Lobar
atrophy**

FTLD (behavioral)

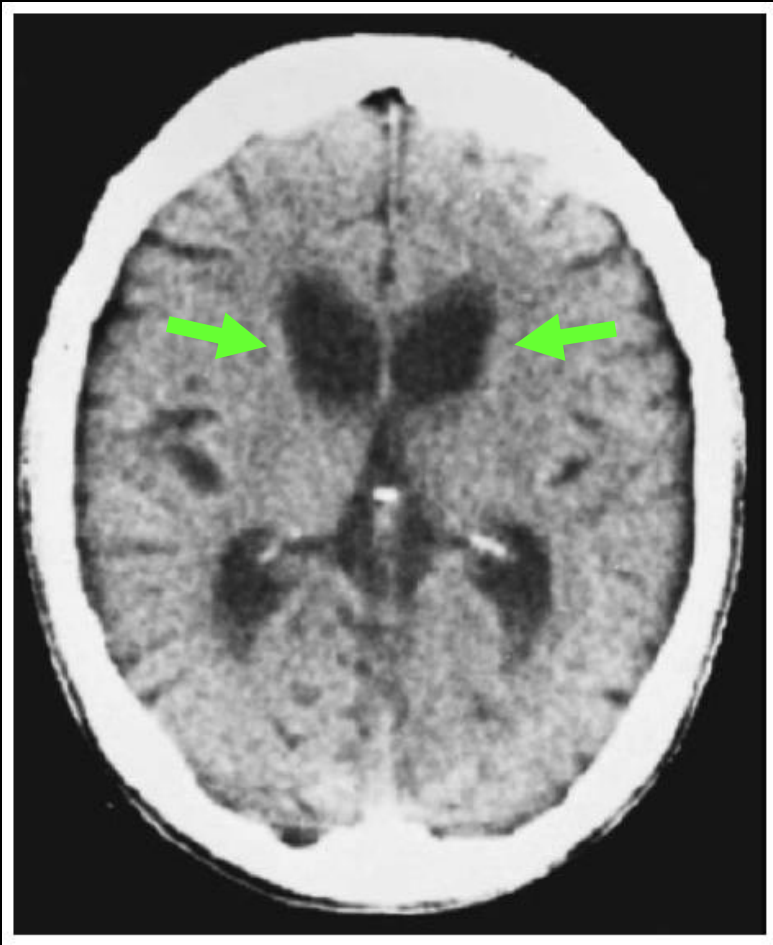


Posterior cortical atrophy

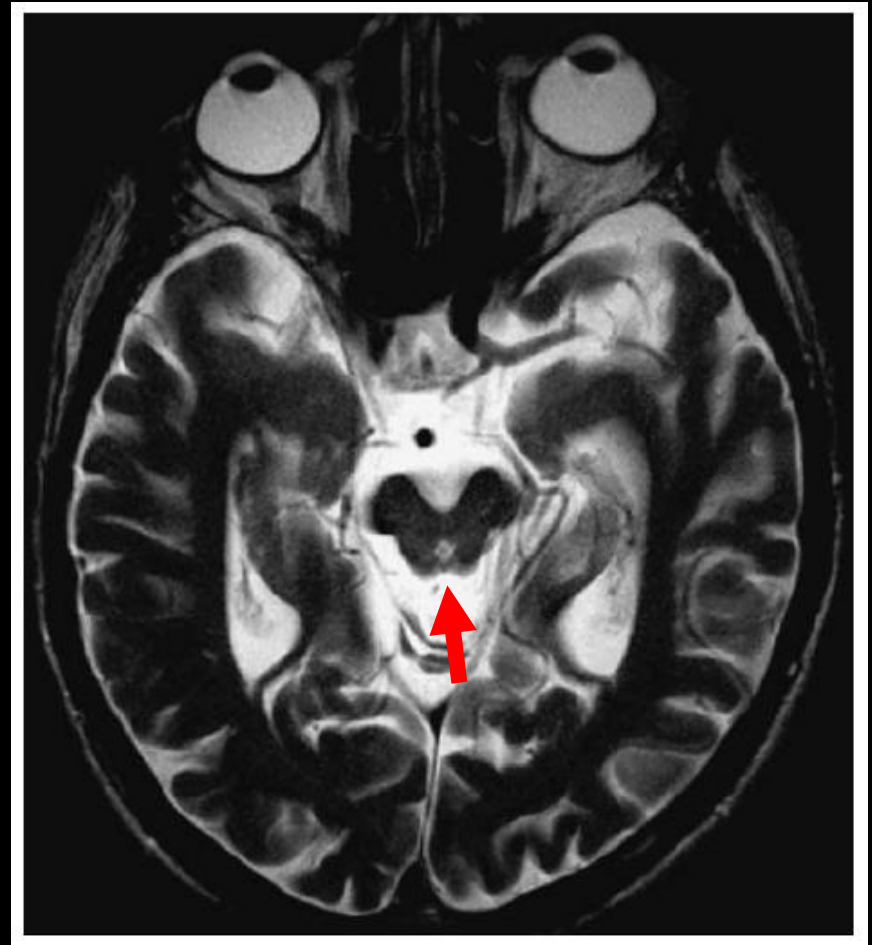


Other focal atrophy

HD



PSP



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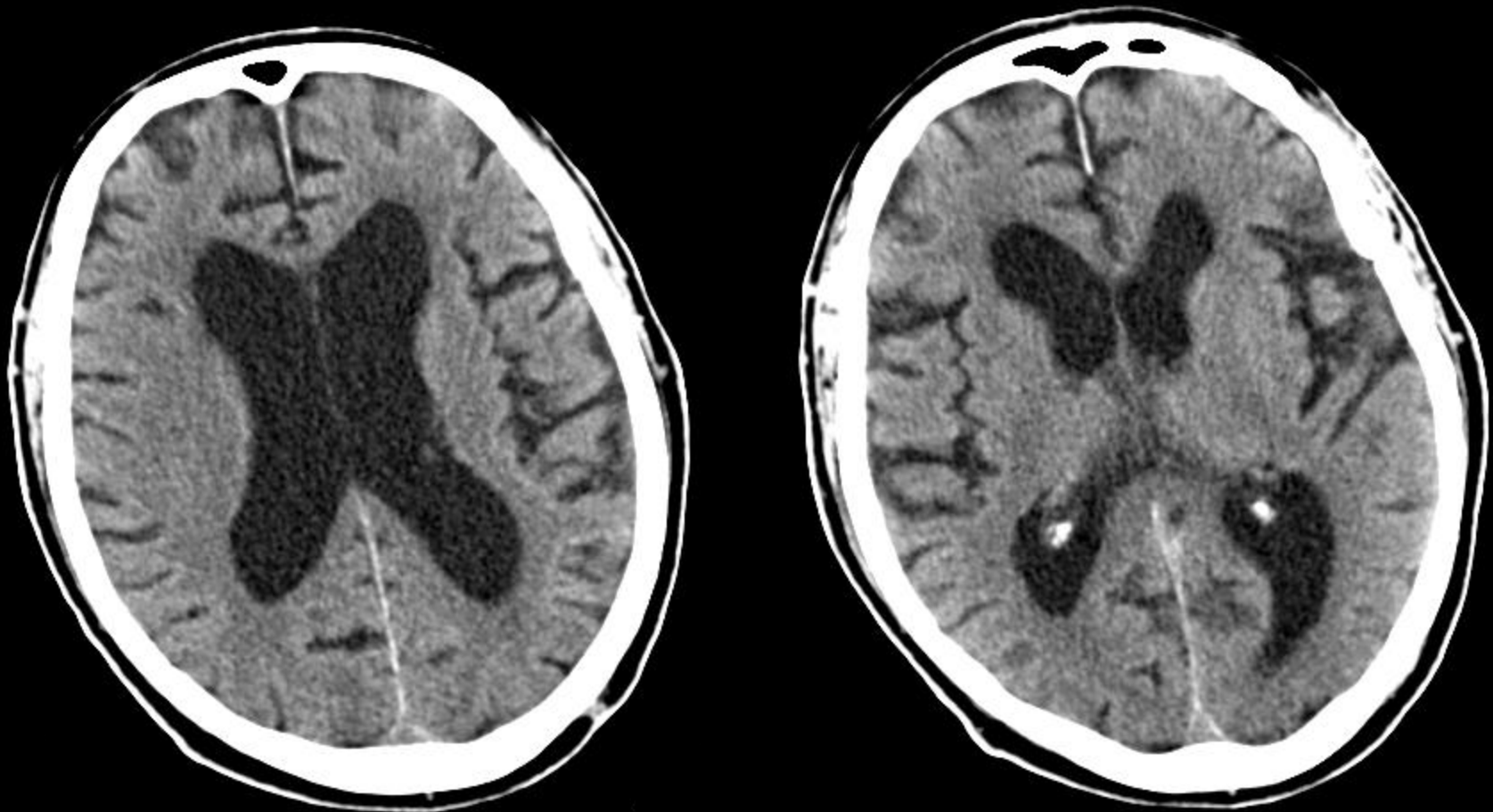
Number 3

SYMPTOMATIC OCCULT HYDROCEPHALUS WITH "NORMAL" CEREBROSPINAL-FLUID PRESSURE*

A Treatable Syndrome

R. D. ADAMS, M.D.,† C. M. FISHER, M.D.,‡ S. HAKIM, M.D.,§ R. G. OJEMANN, M.D.,|| AND
W. H. SWEET, M.D.||

BOSTON, MASSACHUSETTS, AND BOGOTÁ, COLOMBIA



Imaging abnormalities in NPH

1. No macroscopic obstruction of CSF flow
2. Ventriculo-sulcal disproportion
3. Upward bowing of the corpus callosum
4. Empty sella

Imaging abnormalities in NPH

*Ventriculo-sulcal
disproportion*

AD

NPH

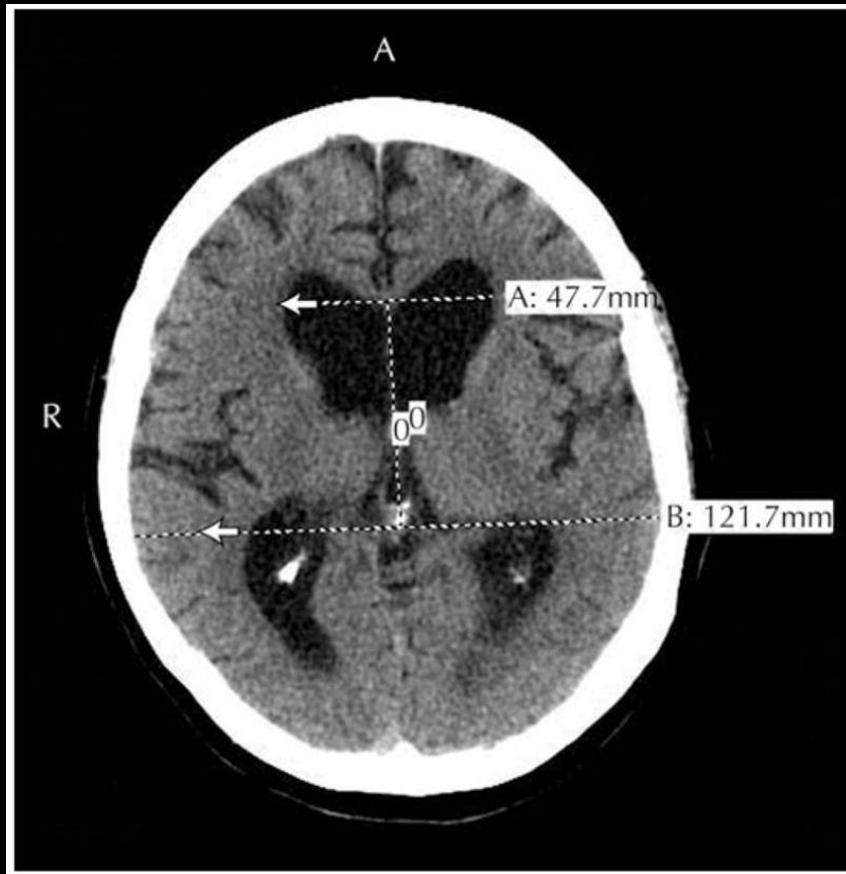


Empty sella

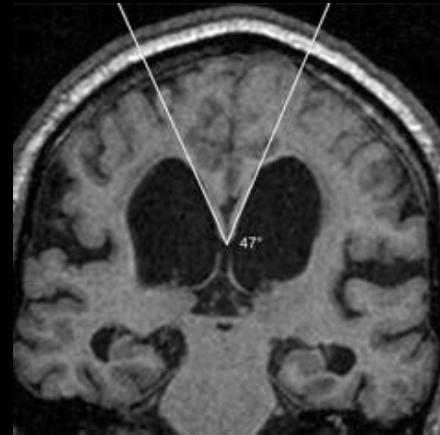


Imaging abnormalities in NPH

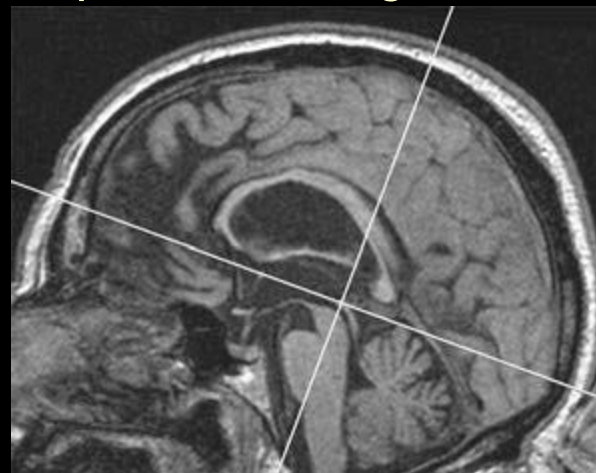
Evans ratio (>0.3)



Callosal angle (acute)



Upward bowing of CC



Conclusion

- **Neuroimaging should be hypothesis-driven.**
- **Localize the problem from the history and physical exam.**
- **Assess carefully for interval changes.**
- **Metabolic changes precede structural changes in neurodegenerative diseases.**