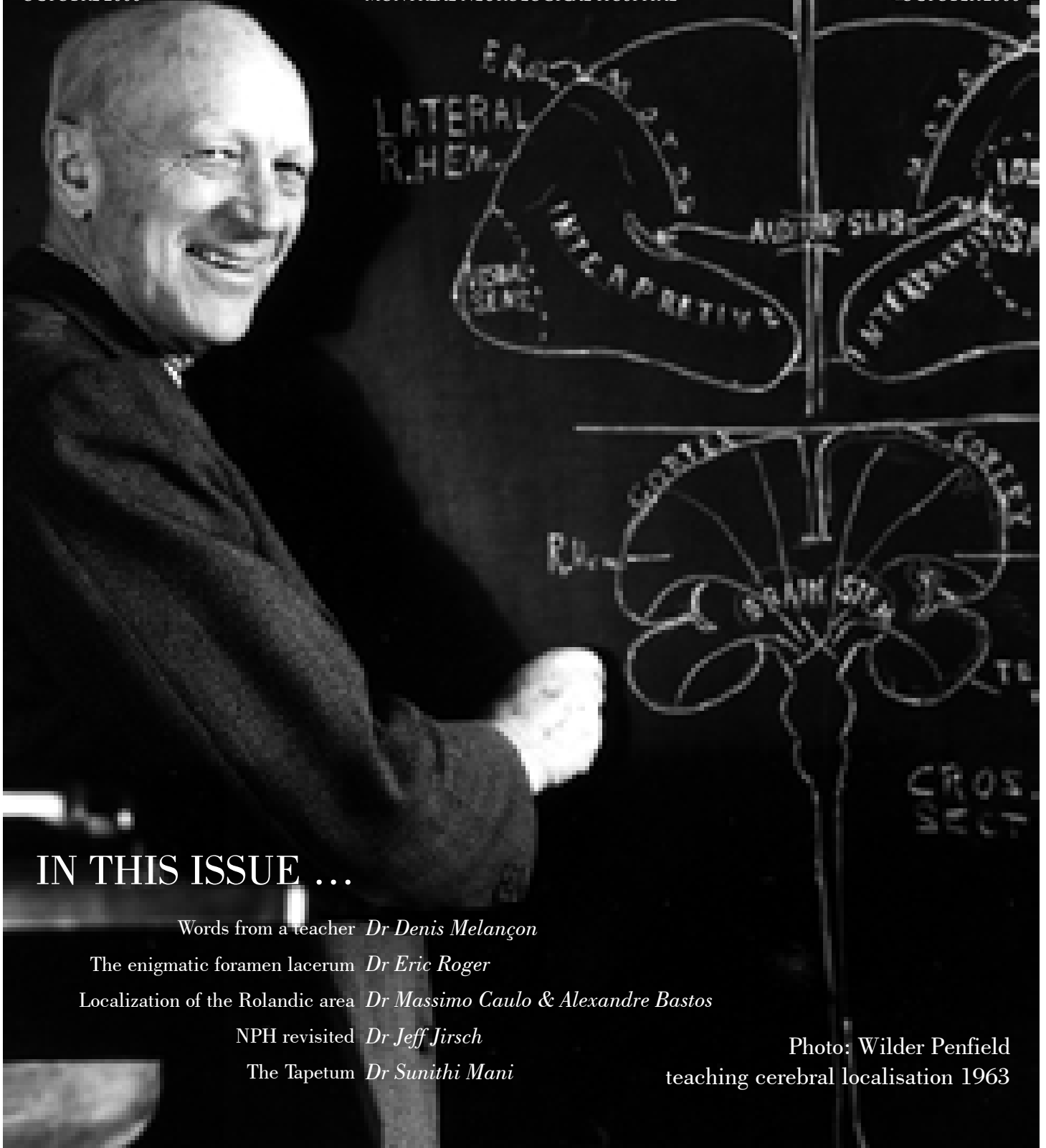


NEUROIMAGE

VOLUME 16, NUMERO 3
OCTOBRE 2000

HÔPITAL NEUROLOGIQUE DE MONTRÉAL
MONTREAL NEUROLOGICAL HOSPITAL

VOLUME 16, NUMBER 3
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Photo: Wilder Penfield
teaching cerebral localisation 1963

WORDS FROM A TEACHER...

For the past three months, we had the opportunity to accommodate a large class of students. As the teacher, I was asked regularly, and often, the usual difficult questions: why such a signal?... , what do you think it is?... , could it not be....?, the book says..., do you agree with Dr Atlas or Dr Osborne?, and so on.

I decided to reverse the trend and ask my students to prepare topics of interest and of some debate, and to present them at our regular Scientific meetings.

Everyone got down to work and produced very interesting Power Point presentations. They intend to eventually present these at other meetings and publish papers.

With their permission, I have included abstracts of their works in this issue of Neuro Image.

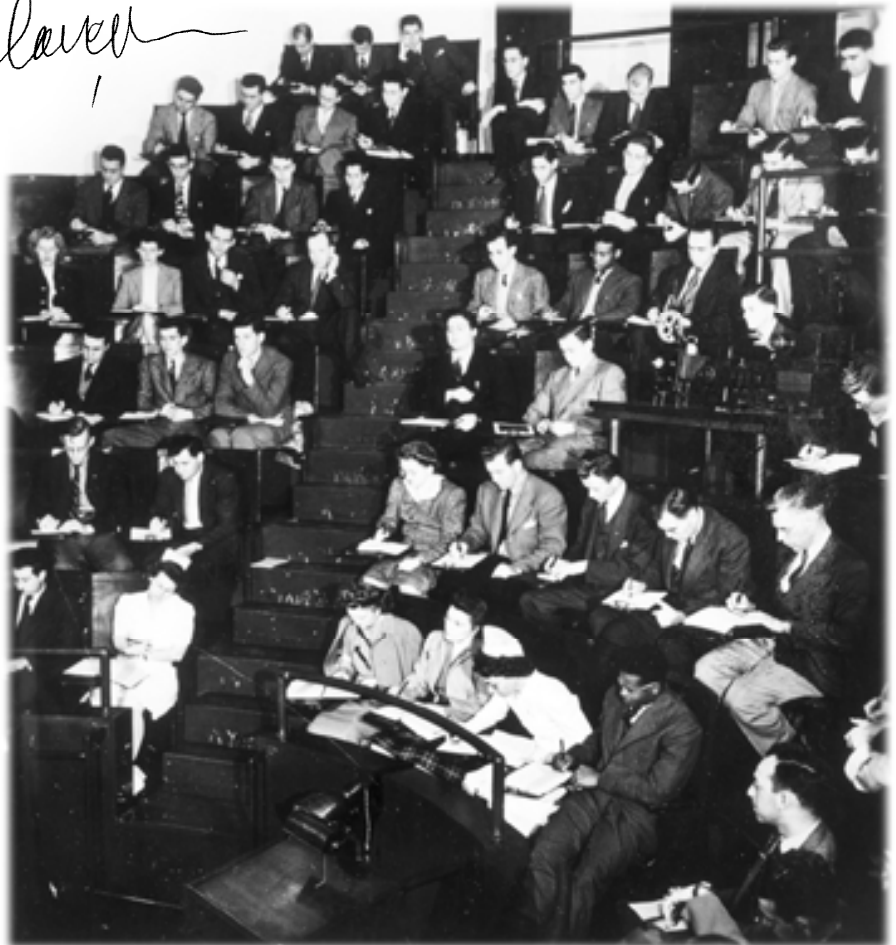
The teacher has demonstrated that students can often answer their own questions, as he is often short of answers.

Denis Melanson

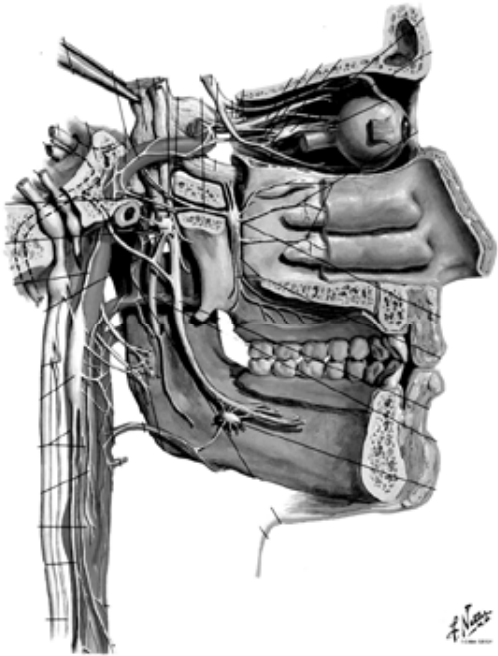


Dr. Roger is a fellow in Neurosurgery, Dr Jeff Jirsch fellow in Neurology, while Drs Mani, Bastos and Caulo are visiting Radiology fellows, respectively from Vellore, India; Sao Paulo, Brazil; and L'Aquila, Italy.

It is a pleasure having them with us and this issue of Neuro Image will remain as a memorial.



Old Hughlings Jackson Amphitheatre



THE FORAMEN LACERUM: SOURCE OF DEBATE

DR. ERIC ROGERS

Question...

What structure runs through to make it a true foramen?

What runs through?

- Osborn: meningeal branches of ascending pharyngeal artery, and inconsistent Vidian artery
- Moore: ICA and sympathetic + venous structures
- Netter: ICA and internal carotid nerve plexus
- Lasjaunias and Berenstein: recurrent artery of foramen lacerum
- Melançon: sympathetic branch from ICA to GPN
- Tauber and al.: nothing « significant »

The best answer is...

As suggested by Tauber and al., (1999),

- The foramen Lacerum has an upper and a lower part
- The lower part is filled with fibrocartilage and contains the Greater Petrosal Nerve, as well as a ? sympathetic contribution from the Deep Petrosal Nerve
- The upper part is in fact a canal continuous with the carotid canal
- And should in fact be called the "Lacerum" portion of the carotid canal
- The term Foramen Lacerum should be restricted to the fibrocartilage filled fenestration (inferior part)

P.S. The paper by Tauber & al. came to our attention as this debate was going on. It obviously helped closing our session " questions and answers " on this topic, leaving further debate to the readers of the Neurosurgery 1999 feb.; 44(2) 386-91; 391-3

LOCALIZATION OF THE CENTRAL AREA OF THE BRAIN: HOW CAN IT BE DONE ?

DRS MASSIMO CAULO & ALEXANDRE BASTOS

Despite the use of sophisticated imaging techniques, the complexity and individual variation of the brain surface make it often difficult to recognize the pre-central gyrus (preCG) in patients with space-occupying lesions. In this study, we report a method for improved identification of the central region based in the utilization of curvilinear mutiplanar reformatting, a novel method of image processing.

The anatomical landmarks used were reviewed from the literature:

- The hand knob of the preCG corresponding to a small digitation situated in the middle portion of the central sulcus
- The posterior directed hook-like process where the pars opercularis of the inferior frontal gyrus (IFG) merges with the preCG
- The arrangement of the medullary branches
- The junction between the superior frontal gyrus and the pre-central sulcus

Thirty patients with brain tumors and fifteen patients with normal MRI were retrospectively evaluated. All patients had 3D MRI using gradient-echo technique. Curvilinear multiplanar reformatted images were obtained using an in-house developed software (Brainsight, Rogue Research. Montreal, Canada) that provided symmetrical curved slices of the gyral structure.

Normal brains. The preCG was identified in all the examinations studied. Using the hand knob as landmark, the preCG was recognized in all the normal brains. The preCG was localized in 100% of cases, by looking at the relationship between the posteriorly directed hook-like process of the pars opercularis of the IFG and the precentral sulcus (2). In 100% of cases, the preCG was localized from the deepest curvilinear reconstruction image by following the medullary pattern of the white matter (4). In 90% of cases, it was possible to recognize the relationship between the superior frontal gyrus and the preCS (4).

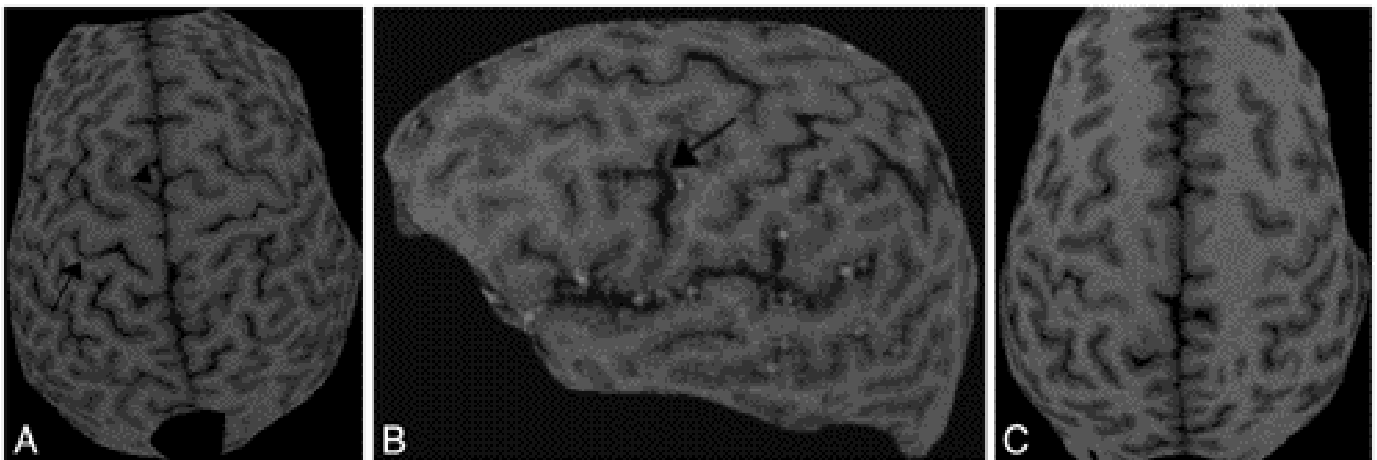


Fig.1 CMPR normal anatomy. In a) The "omega" shape of the hand knob (arrow); relationship between the SFG and the preCSulcus (head of arrow). In b) the pars opercularis of the IFG merges with the preCS (arrow). In c) medullary patterns of the white matter with the constant relationship between the SFG, middle frontal gyrus and preCG.

Diseased brains. It was possible to identify at least one landmark of the pre-central gyrus in all the affected brains. Manipulation of the CMPR images in space allowed to identify and to follow the course of the pre-central gyrus, to establish its relationship with the tumor. Tumors involving the low-middle convexity were localized by the anatomical landmarks of the high convexity. In case of parasagittal tumors, on the contrary, the preCG was identified mainly using the landmark of the middle-low convexity.

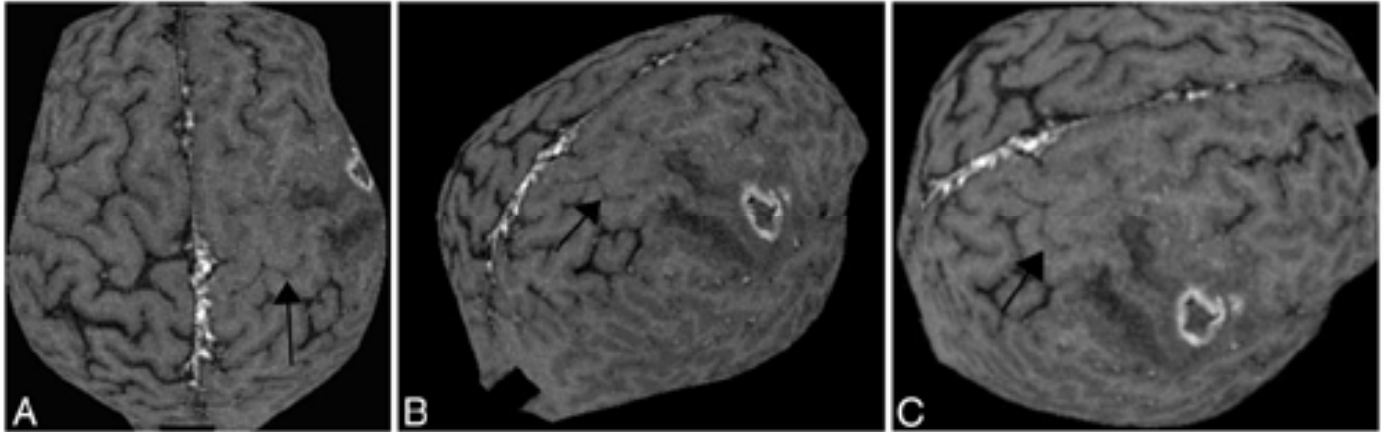


Fig.2 CMPR in patient with grade IV astrocytoma. In a)b)c) three different angles of view at a depth of 6mm from the brain surface. It is possible to recognize the "omega" and to follow the preCG up to the tumor.

Answer to question:

The localization of the central area of the brain can be achieved with accuracy using the usual anatomical landmarks combined with CMPR.

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This has been abstracted from a publication in progress: "Curvilinear Multiplanar Reformatting (CMPR) from 3D MRI for the localization of the motor area in brain tumor disease". M Caulo, A Bastos, D Tampieri, D Melanson et al.

SHUNTING

NORMAL PRESSURE HYDROCEPHALUS IS IMAGING THE ANSWER?

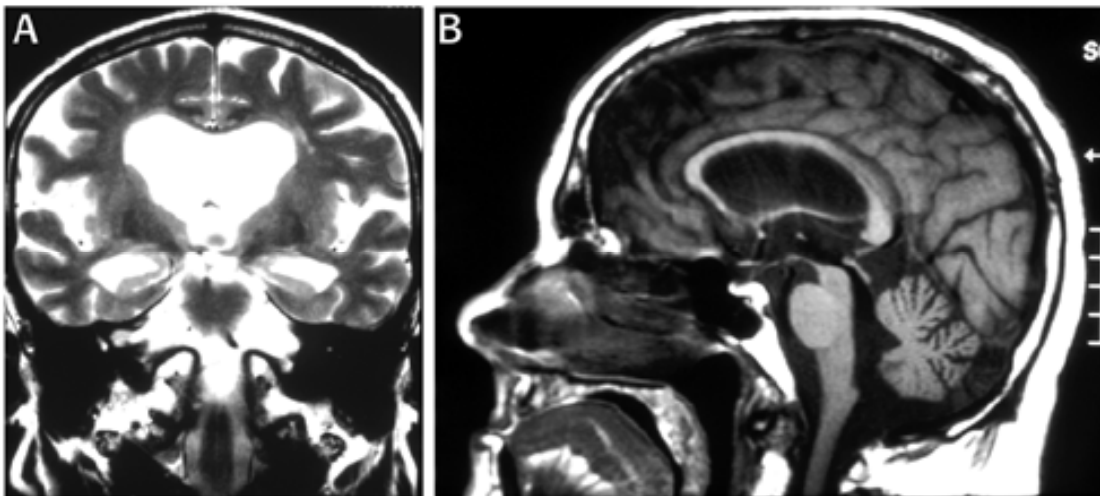
DR JEFFREY JIRSCH

The Problem:

Clinical: 1) Gait disturbance
 2) Cognitive decline
 3) Urinary Incontinence

Pathophysiology: – 30% secondary (e.g. head injury, SAH, meningitis)
 – 70% idiopathic (weak ventricular wall, secondary to ischemia)

Diagnosis: 1) Clinical Triad
 2) LP: opening pressure <18 cm H₂O
 3) Imaging CT and MRI
 – hydrocephalus out of keeping with cortical atrophy (Fig A)
 – trans-ependymal CSF migration, increased flow void in aqueduct (Fig B)



Treatment: Shunting -Vanneste et al. 1992 Meta analysis:
 – 47% no improvement in idiopathic NPH vs. 32% symptomatic NPH.
 – 73% shunt complications, 6% shunt-related mortality.

Results: – Improved outcome when treated early, in patient without significant comorbid
 neurologic disease.
 – Stringent selection criteria needed to avoid unnecessary shunting.

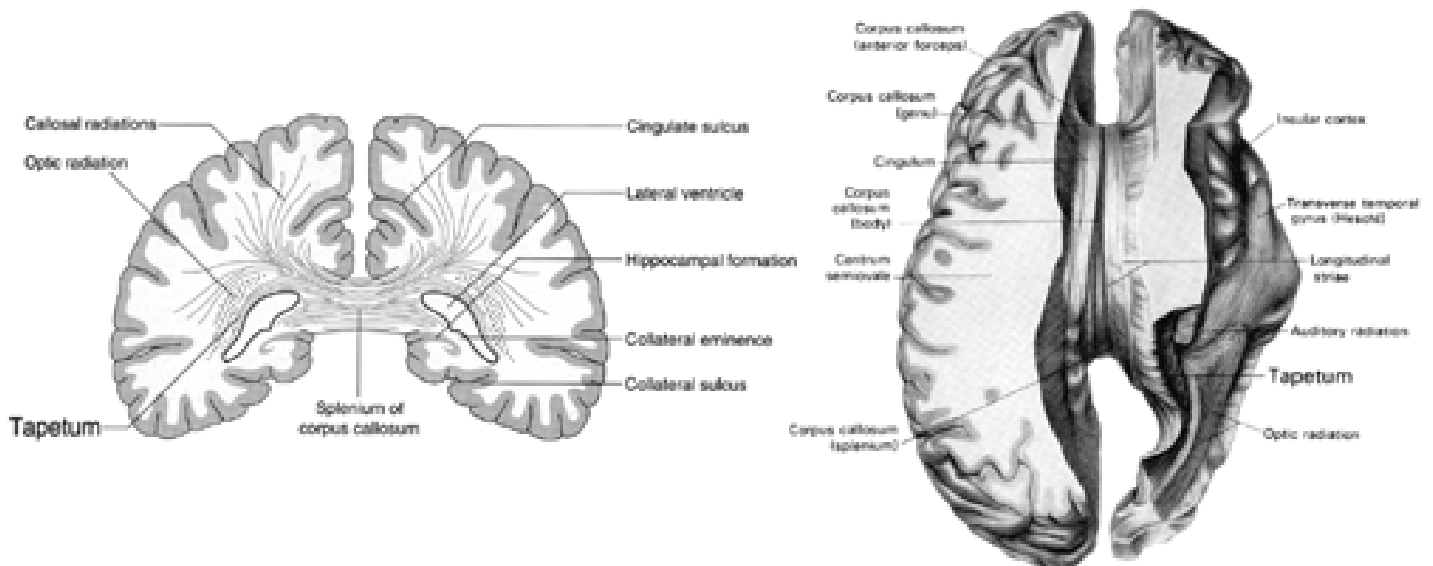
Answer to question:

Imaging is not the decisive factor to decide shunting. CSF Tap study with psychometric and timed gait testing is superior.

THE TAPETUM ... IS IT REALLY THERE?

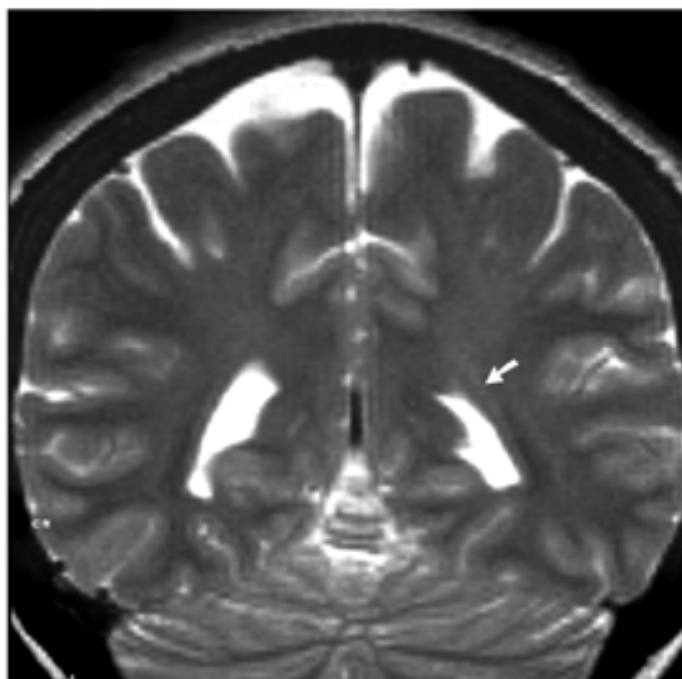
DR. SUNITHI MANI

The tapetum was described by Reil (1759-1813), who also described the insula. The name means carpet or tapestry, and was applied to the fibers of the corpus callosum that pass over the lateral ventricle. In addition to the major and minor forceps, and the fibers that cross the corona radiata, there are other fibers from the splenium that go to form the lateral wall of the temporal horn and the lateral wall and roof of the occipital horn.

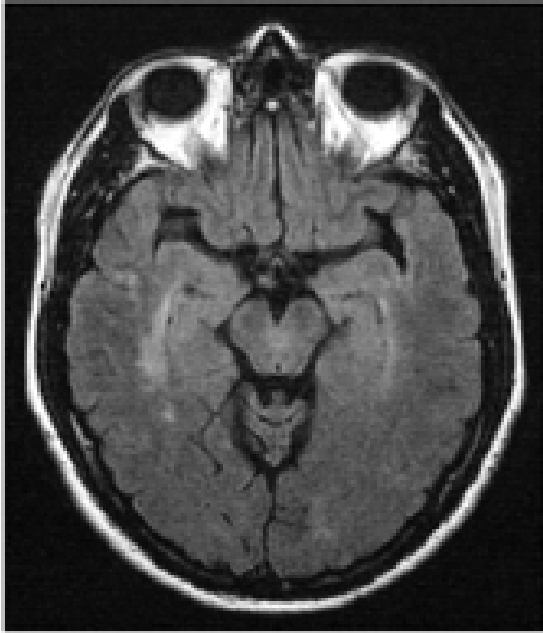


The fibers of the tapetum connect the inferior temporal lobes on both sides, as part of the corpus callosal function. The tapetum is supplied by the long penetrating branches of the middle cerebral artery and also by the branches of the posterior cerebral artery around the trigone.

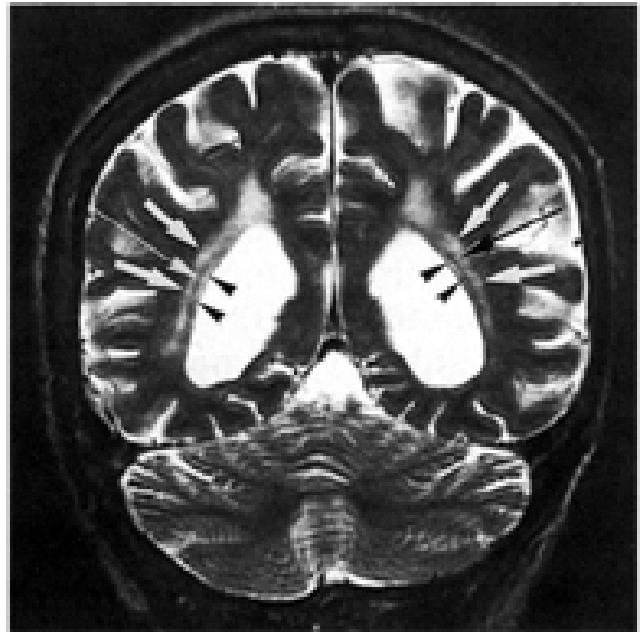
On MRI, the tapetum appears hypointense on T2W images, due to the high axonal density, and the adjacent optic radiations appear hyperintense due to the less axonal density.



The significance of the peritrigonal hyperintensities on T2W sequences in elderly patients was studied, by Kitajima et al. using MRI of 160 patients, their clinical presentation, and gross pathology specimens in patients who had died of non neurologic causes, but had hyperintensity in the same regions. The study concluded that the hyperintensities are due to myelin pallor, infarcts or ischaemic changes, perivascular spaces, and that these changes do not cause any visual deficits or other clinical symptoms.



Seen in M.S.
Hyperintensity in the tapetum bilaterally



Seen in elderly patients
Hyperintensity in the tapetum and optic radiations bilaterally

Answer to question

The Tapetum really does exist. Its signal is different from that of the adjacent optic radiations. It is often a site of plaques of Multiple Sclerosis, in addition to those in the radiations of the corpus callosum and the forceps.

This is a part of my tribute to a great teacher, who reminds us not to forget the basics.

If you wish to contribute to our education fund and the making of this bulletin, send your donations to:

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