

# NEUROIMAGE

VOLUME 6, NUMÉRO 2  
OCTOBRE 1989



HÔPITAL NEUROLOGIQUE DE MONTRÉAL  
MONTREAL NEUROLOGICAL INSTITUTE



VOLUME 6, NUMBER 2  
OCTOBER 1989

## Oedème papillaire et selle vide

DENIS MELANÇON

### Introduction

Au fil des années nous avons rencontré cette association d'œdème papillaire et de selle vide, sans qu'il soit possible de démontrer de lésion intracrânienne associée, soit hydrocéphalie, soit tumeur, soit pression intracrânienne du syndrome d'hypertension bénigne. Nous avons soulevé l'hypothèse à quelques reprises qu'une entité selle vide - œdème papillaire puisse exister, la selle vide représentant l'un des compartiments associés au ralentissement de la circulation de LCR à l'intérieur des gaines optiques.

Notre expérience de 4 cas, toutes des femmes, a été augmentée récemment d'un autre cas où la résonance magnétique démontre bien le LCR dans les gaines optiques. Nous croyons que la stagnation du LCR dans la gaine optique est responsable de l'œdème papillaire, et que cette stagnation existe par ralentissement du déversement du LCR dans l'espace sous-arachnoïdien, vraisemblablement en passant par l'arachnoïdocele sellaire.

### Observations

1. L'espace sous-arachnoïdien se continue dans l'orbite à l'intérieur de la gaine entourant le nerf optique. (Schwalbe, 1870 - Hayreh, 1964) Cette observation faite il y a déjà longtemps a été bien démontrée et précisée par les travaux de Hayreh.

2. L'espace sous-arachnoïdien de la gaine optique est bien démontré par le contraste hydrosoluble lors de la cisternographie. (Manelfe) (Jinkins) Mais l'espace n'est pas toujours démontré (1/3), et parfois il est plus évident d'un côté que de l'autre.

(suite à la page 2)

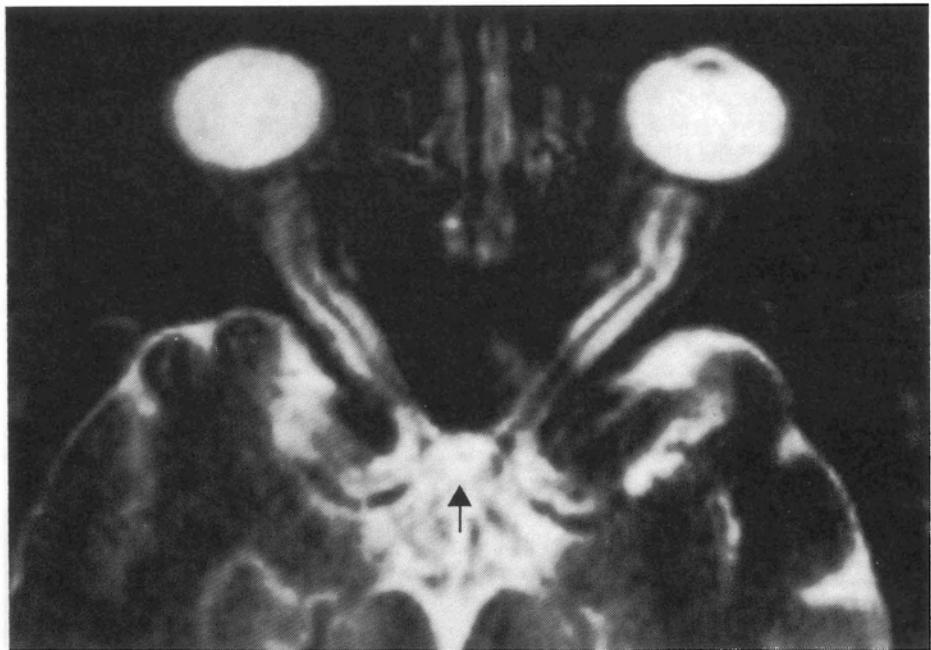


Figure 1 — Appearance of optic nerves with distended sheaths filled with CSF (intense signal in T<sub>2</sub>). Note the close relationship of the postero-medial segment of nerve sheaths with the empty sella (intense round signal on midline).↑



- Thomas Willis and his circle

WILLIAM FEINDEL

- Central Pontine Myelinolysis

R. ASHFORTH

- Kyste épidermoïde sus-calleux

ALAIN WEILL et DONATELLA TAMPIERI



**Figure 2** — The distended optic nerve sheaths show as a hypointense signal on T<sub>1</sub>.

3. L'espace sous-arachnoïdien de la gaine optique est bien démontré en IRM, en T2 surtout, (Fig.1) mais aussi en T1 (Fig.2). Quoiqu'il n'y ait que peu de publications sur le sujet, le travail récent de Azar Kia résume bien cette observation. (Poster ASNR 1989)

4. Le liquide céphalo-rachidien (LCR) de la gaine du nerf optique transmet la pression intracrânienne et cause l'œdème papillaire lorsque la pression devient anormalement élevée.

(Hayreh)

Hayreh a retenu cette hypothèse parmi toutes celles qu'il a vérifiées dans ses travaux.

5. Lorsqu'il y a œdème papillaire le nerf optique est augmenté de volume à la tomodensitométrie (Fig.3).

(J. Vignaud).

6. Parfois la gaine ne contient pas un espace liquidien et l'œdème papillaire peut être absent, ou présent d'un seul côté. (Kirkham)

Hayreh a démontré que l'espace est toujours présent mais que le degré de communication peut être variable.

Ceci explique que l'œdème papillaire peut être contralatéral à la masse intracrânienne sans avoir à invoquer le syndrome de Foster Kennedy.

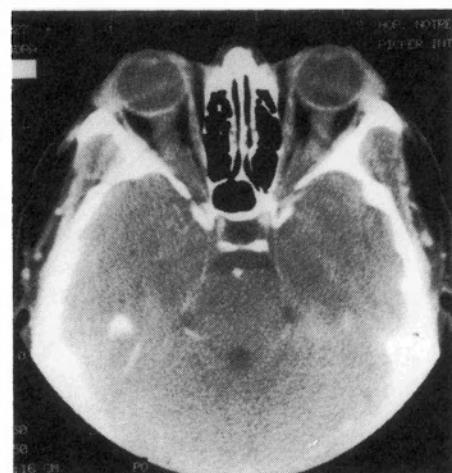
7. La selle vide est une arachnoïdocèle en relation étroite avec le chiasma optique, et donc à l'endroit de communication de l'espace sous-arachnoïdien avec l'espace péri-optique. (Fig.4) Kaufman est celui qui a le plus décrit ce phénomène de la selle vide, soit primaire, soit secondaire, et très récemment Lee (ASNR 1989) a rapporté sa très haute incidence (89%) dans 25 cas d'arachnoïdite optochiasmatique.

## Discussion

Cinq observations de patientes présentant un œdème papillaire associé à une selle vide et chez lesquelles la pression du liquide céphalo-rachidien (LCR) était normale nous ont amené à rechercher une explication reliant les deux phénomènes.

«Widening of the CSF space around the optic nerve may occur in patients without any visual complaint. These cases are usually associated with large basal cisterns and empty sella. We have seen the same change in increased intracranial pressure and pseudotumor cerebri.» (Azar-Kia)

Azar-Kia observe l'association d'espaces périoptiques élargis surtout chez des patients avec une selle vide et des citermes basales proéminentes, mais sans qu'il y ait trouble visuel.



**Figure 3** — Distended optic nerves on CT without differentiation between the nerves and their sheaths.



**Figure 4** — Empty sella demonstrated on sagittal T<sub>1</sub> image with good outline of pituitary stalk and optic chiasma

Nous croyons qu'il peut y avoir parfois troubles visuels et même de l'œdème papillaire mais ces troubles sont habituellement fluctuants. Par quel mécanisme? C'est notre hypothèse.

## Conclusion

Deux phénomènes du LCR peuvent conduire à l'œdème papillaire :

1. La transmission de la pression intracrânienne élevée dans la gaine des nerfs optiques

2. La stagnation du LCR dans ces mêmes gaines par ralentissement du flot de retour vers l'espace normal.

C'est ce deuxième mécanisme que nous invoquons. Le LCR doit réintégrer l'espace sous-arachnoïdien et le flot directionnel l'amène fort probablement vers la selle vide. Celle-ci est semblable à un kyste intra-arachnoïdien, à plus forte raison s'il existe certaines septations inhabituelles dans la région suprasellaire. (du type membrane de Liliequist - adhérences optochiasmatiques).

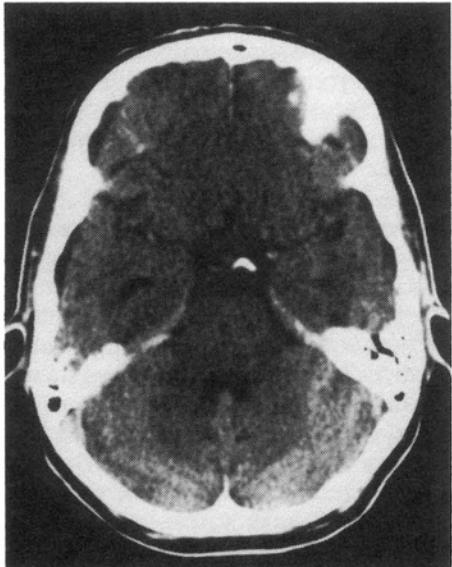
Quand on compare l'incidence générale peu élevée de l'espace péri-optique (Manelfe 33%) à l'incidence élevée de selles vides avec les adhérences optochiasmatiques (Lee 89%) ou avec les espaces péri-optiques généreux (Azar-Kia) nous pouvons spéculer que certains rares cas d'œdème papillaire soient liés à l'association de ces phénomènes.

Nous espérons que l'étude des flots du LCR en IRM viendra bientôt documenter ce phénomène.

# Central Pontine Myelinolysis – Radiographic Findings

R. ASHFORTH

In the last several years, central pontine myelinolysis has become an increasingly recognized clinical and radiological entity. A case with typical findings of this disease is presented.

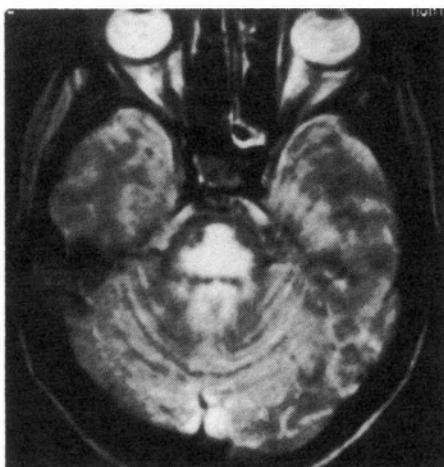
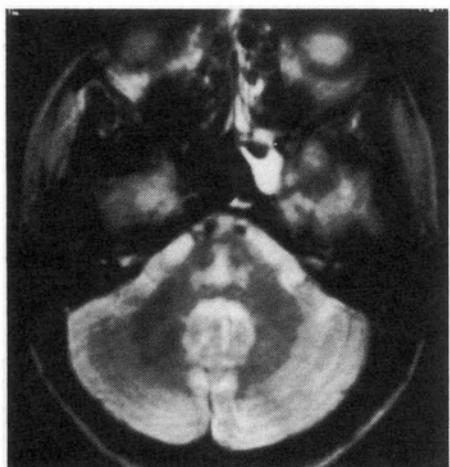


◀ Figure 1 — Nonenhanced CT scan demonstrating no pontine abnormality.



Figure 2 — Parasagittal spin-echo A) T1-weighted (TR=775msec TE=20msec) image demonstrating inhomogeneous hypointensity in base and tegmentum of pons, sparing the periphery. B) T2- weighted (TR=2000msec, TE=70msec) image showing corresponding area of high signal intensity.

Figure 3 — A) and B) Axial spin-echo T2-weighted (TR=2100msec, TE=70msec) images demonstrating trident-shaped area of increased signal in base of pons, sparing ventro-lateral portions of the pons.



## Case report:

A 43 year old previously well woman presented to hospital with lethargy, confusion and ataxia. The patient was discovered to be profoundly hyponatremic, with a serum sodium of 97 mmol/l, presumed to be secondary to antidepressant use. She was admitted to ICU for correction of hyponatremia. Subsequently, the patient developed pneumonia and hypernatremia secondary to dehydration. Within a few days of sodium replacement, the patient progressively became aphasic, quadraparetic and developed "locked-in" syndrome.

Two CT studies performed in this period, within two weeks of admission showed no definite abnormality (Fig.1). An MR study was performed approximately one month after admission, with characteristic findings in the base of the pons of hypointensity in T1-weighted and hyperintensity in T2-weighted images (Fig. 2,3), confirming the clinical diagnosis of central pontine myelinolysis. The patient gradually improved over the next few weeks, with considerable clinical recovery.

## Discussion:

Central pontine myelinolysis is of unknown pathogenesis, but has most frequently been recognized in patients with chronic debilitating disease including malnutrition, liver disease and alcoholism. Often described is a history of severe hyponatremia that has been rapidly corrected, giving rise to the name of "osmotic demyelination syndrome". (1)

Symptoms generally consist of a progressive, deteriorating level of consciousness, spastic quadripareisis or paraparesis with pseudobulbar palsies, which may progress to a "locked-in" syndrome. Death may ensue within 3-5 weeks; however, an increasing number of survivors are reported, and marked improvement in symptoms can occur.

(suite à la page 8)

# Thomas Willis and his circle – A 17th century “NEURO”

WILLIAM FEINDEL, OC, MDCM, DPhil<sup>1</sup>



Thomas Willis (1621-1675), the celebrated physician and anatomist of Oxford, published in his *Cerebri Anatome* (1664) an extensive description of the brain, spinal cord and autonomic nerves which effectively changed our knowledge of the nervous system from medieval to modern (1,2). His further writings provide some of our earliest clinical and pathological accounts of neurological and psychiatric disorders. For his many original contributions, Willis (Fig.1) has been considered as the founder of neurology (3). His books, widely published on the continent (7), can still be found in the libraries of the famous European Medical Schools (8).



Figure 1 — Thomas Willis, from the engraving by David Loggan for the frontis-piece of *Pathologia Cerebri* (1667).

## The contributions of Willis

The many medical works of Willis, already catalogued in previous historical studies, (4,5) included the detailed description of the anatomy of the human brain, and a classification of the cranial nerves (2). Among the first comparative anatomists of the nervous system, he is best known for the anatomical description of the

arterial circle at the base of the brain and his astute deduction about the significance of intracranial collateral flow. In his second great work, *De Anima Brutorum*, the souls of brutes, he described many neurological syndromes – headache, lethargy, narcolepsy, coma, apoplexy – and characterized delirium, frenzy, depression and mental retardation. Willis coined the word “Neurologie”, meaning the doctrine or teaching of the nerves. Thus our prolific range of prefixes using “neuro” all derive from Willis.

## Thomas Willis and his neuro circle

Willis had working with him in Oxford a brilliant group of “virtuosi”; many became founders of the Royal Society of London in 1662. His systematic plan for dissection of the brain and nerves was done with the help of what we would call today a research group in experimental neurology or a “Neuro” team. This included, above all, Richard Lower, the chief dissector, who later contributed important observations on the anatomy and physiology of the heart, explaining how venous blood in passage through the lungs became red when exposed to air. With Edmund King, another Oxford colleague, Lower was the first to carry out blood transfusion in man. Christopher Wren, then professor of astronomy at Oxford, devised a technique for injecting blood vessels with dyes or drugs; he drew “with his skillful hand” some of the elegant figures of the brain for Willis’s books. This was before he became one of Britain’s greatest architects. Sir Thomas Millington, a Cambridge graduate and later president of the Royal College of Physicians, was on hand to discuss with Willis his anatomical findings. Robert Hooke, later secretary of the newly founded Royal Society and one of the pioneer microscopists, worked for a time as a student with Willis. So did John Locke, who aborted his medical career to

become one of England’s most eminent philosophers. In touch with the goings on of the Oxford team was Robert Boyle, living then in London, but receiving frequent communiqués from Lower.

In one of the early European editions of Willis’s *Cerebri Anatome* published by Gerbrand Schagen of Amsterdam, an engraved title page (unique to this particular edition) illustrates the medical team of Willis grouped around the dissection table that holds a cadaver (Fig.2). On the right, the figure with the skull cap points to what appears to be a defect in the skull (a potato-sized lump to the left, apparently removed from this defect, could represent a meningeal tumor). Wells (6) in her scholarly study of this illustration, presumed this figure to be Willis. But it seems more likely to be Richard Lower, the dissector, Willis being the figure on the left, with a more commanding presence and in more elegant costume. The figure to his left, writing notes, could be John Hemmings, the apothecary, who often filled the role of amanuensis to Willis. The paraphernalia on the back wall – bodily parts, the snake, owl and surgical instruments – are appropriate to the setting<sup>2</sup>. This artist’s representation depicts faithfully the



Figure 2 — Title page of the Dutch edition of *Cerebri Anatome* (1665), depicting Willis and his Neuro-circle.

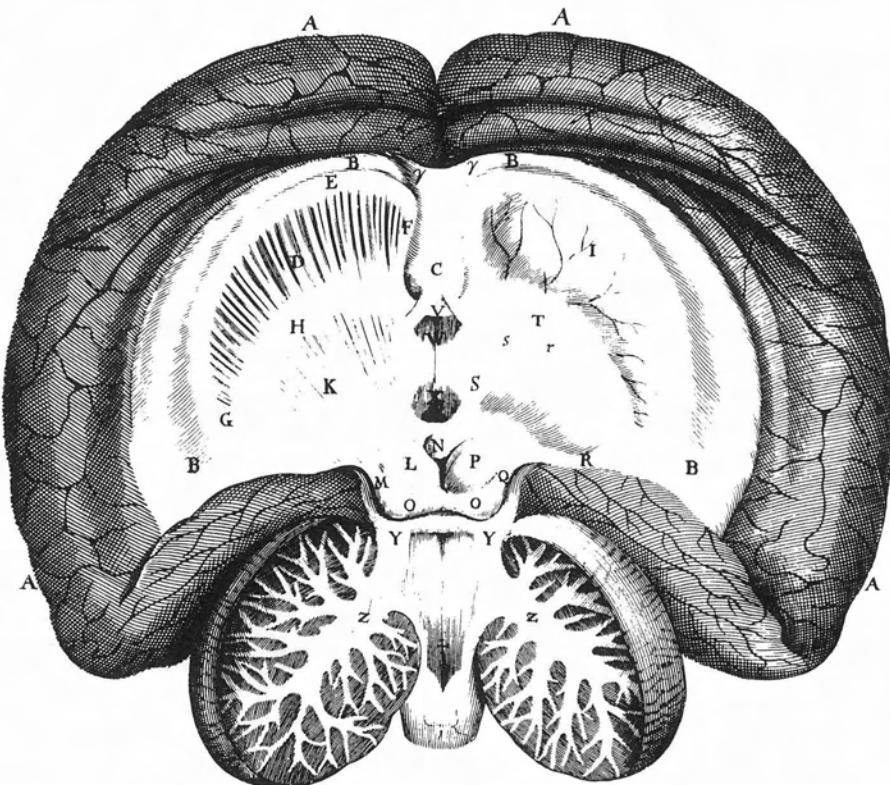
discussion, argument, consultation and recording that must have taken place among Willis and his Neuro circle during the studies that resulted in *Cerebri Anatome* (2).

#### Willis and his team as Neuro-imagers

Willis advocated a method of displaying the brain's anatomy by freeing up the hind part from the brainstem and rolling the hemispheres forward which "exposes to be seen by the eyes themselves, its whole frame or substance, to wit, whatever it hath without or within, or before and behind and also plainly detects what its habitude and respect is to the other parts". In a sense, this was an anatomical demonstration in 3-D. The splendid drawing (Fig.3) from *De Anima Brutorum* (1672), taken from "The VIII Table" shows "This new Anatomy of the Humane Brain". The corpus callosum is divided and the hemispheres laid apart to show the structures lining the ventricles. We see clearly the corpus striatum both intact with its surface vessels (I) and after scraping to show the medullary streaks or nervous tracts (D). The "optick chamber" shown in the same manner on either side (K, S), refers of course to the thalamus. We see clearly the corpora quadrigemina, the third ventricle bordered anteriorly by columns of the fornix (C) and anterior commissure (V), and posteriorly by the pineal body (N).

With their systematic survey of anatomy, Thomas Willis and his circle of neuro-imagers marked the emergence of modern explorations into the vast inner universe of the brain and mind. We can identify in them kindred spirits aware of the complex problems of examining brain and nerves "where" wrote Willis, "neither the rushings on or impressions of the animal spirits themselves, nor their tracks or footsteps, can anyways be seen. Wherefore to explicate the uses of the brain," Willis continued, "seems as difficult a task as to paint the soul of which it is commonly said, that it understands all things but itself...."

As Dr. Penfield wrote in his gracious foreword to The Tercentenary edition of the Willis Anatomy, "If it seems to neurologists today that our present understanding of the brain and the mind of man is hardly more than a beginning of science, it may be



**Figure 3** — Drawing, probably by Christopher Wren, from *De Anima Brutorum* (1672) by Willis, depicting internal structures of the human brain exposed by the Willisian technique of dissection.

reassuring to recall that our task is the ultimate one. *The problem of neurology is to understand man himself*<sup>3</sup>. We must analyse the means by which man, the creator of science, has done what he has done. This may well be the most difficult, and surely it is the most important, task of all.

#### Notes:

1 Director, The Neuro-History Project, Montreal Neurological Institute, and Curator, Penfield Archive, Osler Library, McGill University.

2 The spotted Dalmatian coach-dog in Fig.2 represents a metabolic oddity, excreting (like man) uric acid, and differing from other canines (and butterflies) that can oxidize uric acid to allantoin.

3 This sentence was selected in 1978 for lapidary inscription on the dedication plaque of the Penfield Pavillon.

© Copyright, William Feindel, 1989.

#### References

- MEYER A. Historical aspects of cerebral anatomy. Oxford University Press, London, 1971.

2. FEINDEL W. (ed.) Thomas Willis, The anatomy of the brain and nerves. Tercentenary edition, McGill University Press, Montreal, 1965 (2 vols.)

3. FEINDEL W. Thomas Willis (1621-1675). The founder of neurology. Can Med Assn J. 87: 289-292, 1962.

4. ISLER H. Thomas Willis (1621-1675). Doctor and scientist. Hafner Publishing Company, New York, 1968.

5. DEWHURST K. Thomas Willis's Oxford Lectures. Sandford Publications, Oxford, 1980.

6. WELLS E. Willis's *Cerebri Anatome*. An original drawing. J Hist Med & Allied Sci. 23: 182-184, 1967.

7. DENHAM HR. A bibliographic survey of *Cerebri Anatome*. pp. 67-104 in Vol.1: Feindel, William (ed.) Thomas Willis, The Anatomy of the Brain and Nerves. Tercentenary Edition, McGill University, Montreal, 1965.

8. FEINDEL W. The influence of Thomas Willis (1621-1675) on neurology and psychiatry on the continent. Proceedings of the 31st International Congress for the History of Medicine, September 1988. Bologna (in press).

# Kyste épidermoïde sus-calleux: une localisation rare

ALAIN WEILL et DONATELLA TAMPIERI

**À Propos d'une Localisation  
Exceptionnelle d'une Tumeur Rare  
Kyste Épidermoïde Sus-Calleux.**

**Présentation clinique:**

Patiante de 77 ans, traitée pour dépression nerveuse depuis 1962.

Devant l'apparition d'un trouble de la parole et de la marche, en plus des signes communément observés depuis 1962 (insomnie, difficulté de concentration, manque d'appétit,

tristesse), mme F. nous est adressée pour examen tomo-densitométrique au mois de février 89.

**Description radiologique:**

L'examen scanographique met en évidence une lésion kystique, bifrontale, bilobée, non rehaussée par le produit de contraste, de contenu fortement hypodense, sans œdème périlésionnel. Quelques petites calcifications pariétales sont notées (grosse flèche).

L'examen IRM réalisé par la suite démontre la localisation extra-ventriculaire de ce kyste et précise l'aspect en sablier de cette lésion (le collet se situant entre le bord inférieur de la faux et le corps calleux, les renflements de part et d'autre de la ligne médiane, dans les lobes frontaux).

Sur l'image pondérée en T1, ce kyste prend un aspect hypointense inhomogène. Lors de l'acquisition TR long/TE court (dite densité de proton) cette lésion apparaît plutôt hypointense inhomogène, entourée d'un fin liseré hyperintense, (petites flèches). Enfin sur l'image pondérée en T2 ce kyste devient hyperintense inhomogène.

Devant l'aspect scanographique, et le comportement IRM de cette masse, le diagnostic de kyste épidermoïde est évoqué. Celui-ci est confirmé anatomo-pathologiquement après ponction biopsie.

**Discussion:**

Le kyste épidermoïde est une tumeur, le plus souvent découverte chez l'adulte d'âge mur. Toutefois la littérature révèle des observations aussi bien chez le petit enfant que chez le vieillard. Une légère prédominance masculine est notée.

La fréquence des kystes épidermoïdes est estimée de (0.2% à 1%) selon les auteurs, de l'ensemble des tumeurs intracraniennes (2). La plupart de ces lésions sont en rapport avec la base du crâne (70% à 80%) (1), le nombre de kystes intraventriculaires étant également conséquent (10% à 20%) (1).

Lepoire et Pertuiset, dans leur série de 100 patients, ne rapportent que deux cas hémisphériques cérébraux non intraventriculaires. Il est curieux de constater que ces deux cas présentent la même localisation que notre observation. La pathogénie évoquée serait une migration anormale de cellules épithéliales, entraînées le long des cérébrales antérieures durant l'embryogénèse.

D'un point de vue anatomo-pathologique, le contenu de tels kystes

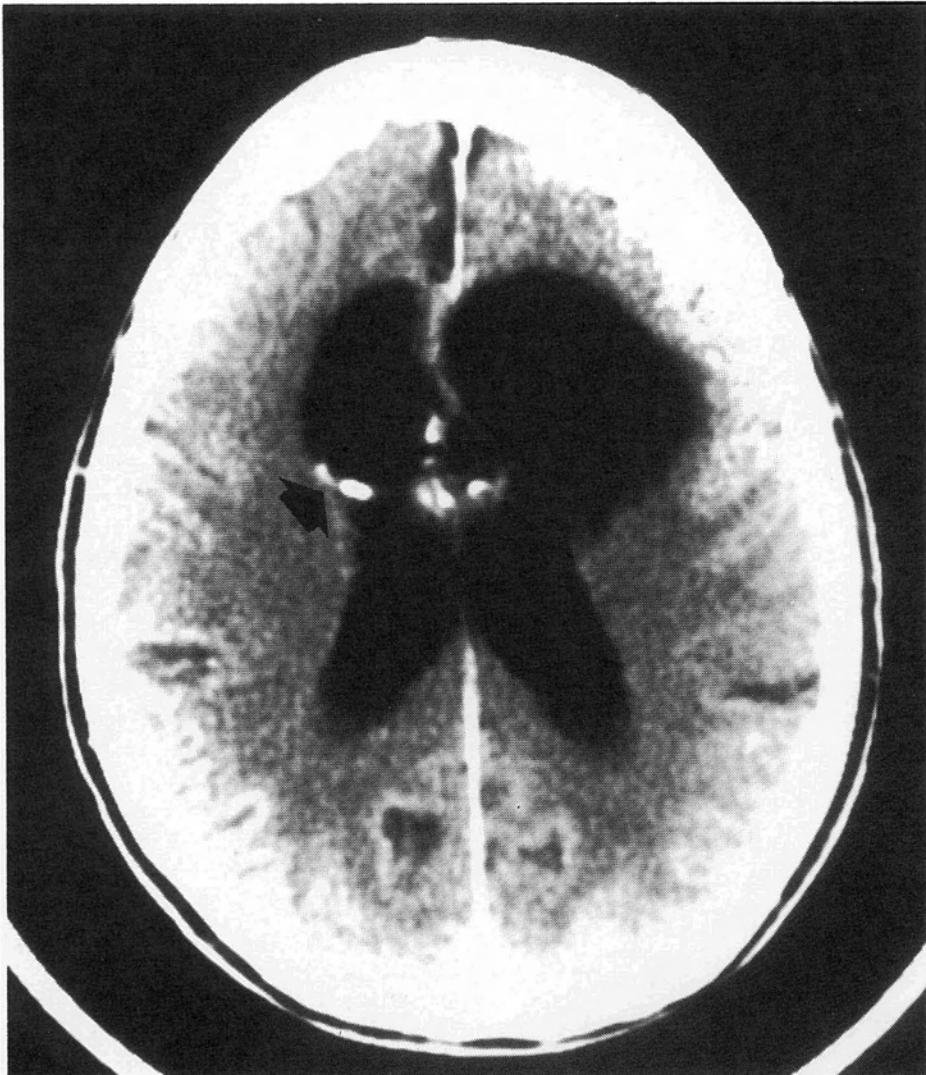


Figure 1 — Image scanographique

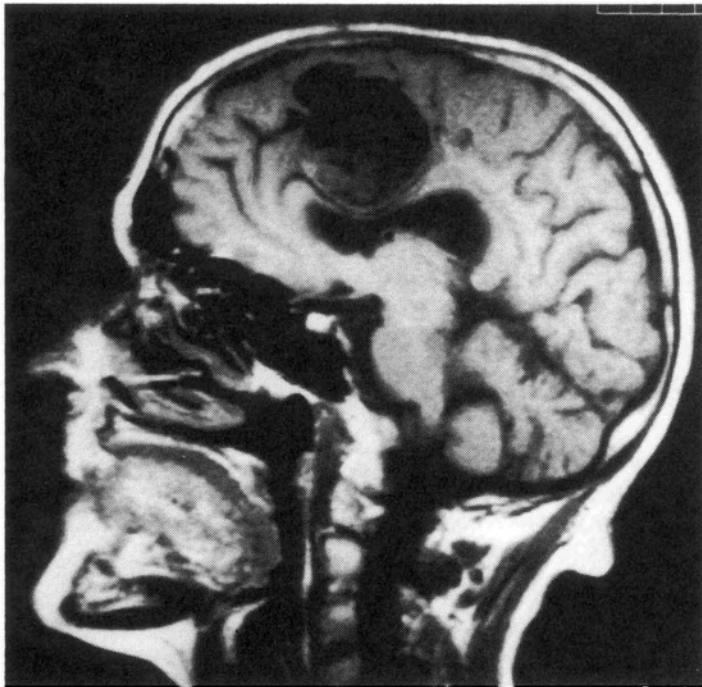


Figure 2A — IRM: sagittal T1

est classiquement blanc nacré, stratifié, certains auteurs lui donnant d'ailleurs le nom de tumeur perlée. Cette substance, parfois comparée à du fromage blanc, est riche en cristaux de cholestérol, et résulte de l'accumulation de kératine, et de la desquamation progressive des cellules épithéliales bordantes.

Si l'aspect scanographique de telles lésions est désormais bien connu, «lésion hypodense homogène non rehaussée par le contraste, sans œdème périlésionnel, avec exceptionnellement des petites calcifications pariétales», le comportement IRM des kystes épidermoïdes, n'a été que récemment décrit, et par un petit nombre d'auteurs. (4) (5) (6)

Ceux-ci mettent en évidence le caractère plus ou moins homogène, hypointense en T1, hypo/isointense en densité de proton, hyperintense en T2, du signal d'une telle masse.

À noter que dans une publication très récente (6), Dr. Tampieri et collaborateurs décrivent un fin liseré périlésionnel hyperintense, seulement visible en densité de proton dans plus de 50% des cas. Ce signal a été rattaché à une séquestration de LCR entre le kyste et le parenchyme cérébral. Notre observation retrouve ce signe.

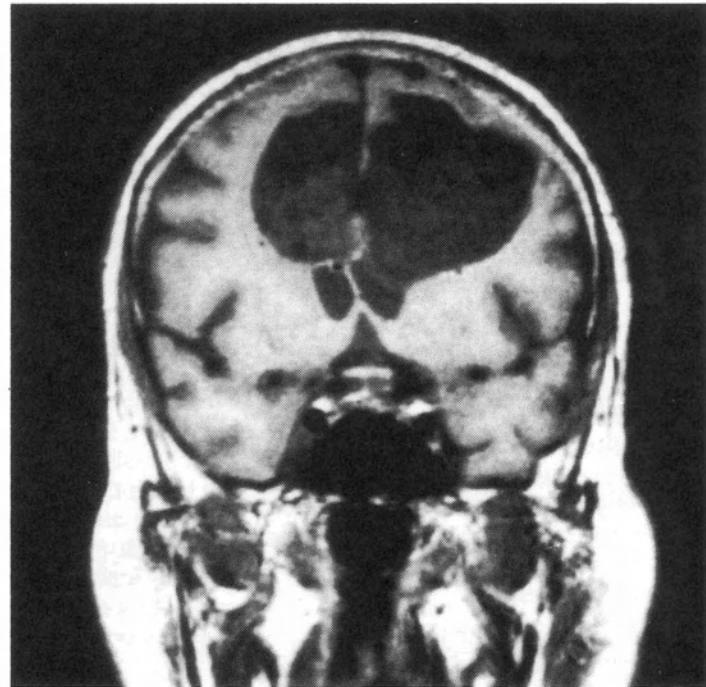


Figure 2B — IRM: coronal T1

En conclusion l'examen par résonance magnétique nucléaire nous paraît indispensable à l'exploration de telles lésions. En effet compte tenu d'un comportement scanographique et IRM relativement spécifique, il nous semble aujourd'hui possible d'évoquer le diagnostic de kyste épidermoïde même dans le cas de localisation atypique.

#### References

1. Lepoire, J. et Pertuiset, B., eds. *Les Kystes Épidermoïdes Cranio-encephaliques*. Paris: Masson et C., 1957.
2. Okezaki, M., ed. *Fundamentals of Neuropathology*. New York: Igaku - Shain, 1983.
3. Marsault, C.; Gaston, A.; Le Bras, F. *Imagerie du système nerveux «L'encéphale»*. Flammarion Médecine-Sciences, 1986.
4. Davidson, H.D.; Ouchi, T.; Steiner, R.E. *NMR Imaging of Congenital Intracranial Germinal Layer Neoplasms*. *Neuroradiology* 1985; 27:301-303.
5. Vion, Dury J.; Vincentelli, F.; Jiddane, M. et al. *MR Imaging of Epidermoid Cysts*. *Neuroradiology* 1987; 29:333-338.
6. Tampieri, D.; Melanson, D.; Ethier, R. *MR Imaging of Epidermoid Cysts*. *AJNR*: March 1989, 351-356.

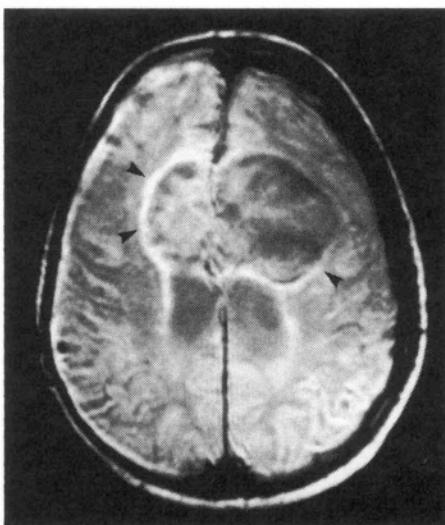


Figure 3 — IRM: Axial densité de proton

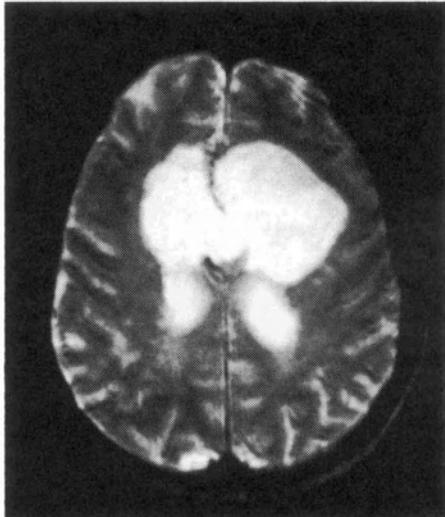


Figure 4 — IRM: Axial T2

(suite de la page 3)

Typical pathological features are symmetric demyelination of the base of the pons, with relative sparing of a rim of surrounding ventral and lateral tissue. The lesion may extend into the pontine tegmentum and midbrain. Symmetrical lesions of myelinolysis have also been found in basal ganglia, thalamus, cerebral peduncles, corticomedullary junction and spinal cord.

The CT findings of non-enhancing low density of the central pons without mass effect is characteristic. Low density lesions in basal ganglia and thalamus can also be seen. CT is frequently normal however, especially early in the course of the disease, and bony artifacts often obscure subtle pontine changes. (2)

MRI demonstrates the lesions of myelinolysis better than CT. Symmetric round or oval areas of hypointensity in T1-weighted and hyperintensity in T2-weighted images are seen in the base of the pons, sparing peripheral tissue. The lesion frequently spares corticospinal tracts and ventro-lateral regions of the pons,

resulting in a triangular or trident-shaped lesion. (3,4) Extension of the lesion into middle cerebellar peduncles can occur, and extra-pontine sites of involvement are frequently found in the lateral basal ganglia, thalamus and subcortical white matter. (5)

The lesions of myelinolysis evolve with time. (4,6) Initial MR studies in the first week of symptoms may be normal but within 1-2 weeks large lesions may be identified, likely due to a combination of demyelination and edema. Eventually, lesions become smaller and more sharply defined, representing both gliosis and some remyelination. If the patient survives, both clinical sequelae and MR findings may persist, with residual demyelination, gliosis and pontine atrophy. If the initial lesion was small, however, complete resolution may occur.

The MR findings are non-specific, and similar signal changes may be seen in pontine glioma, or with vascular leukoencephalopathic changes. (4) The presence of the typical MR findings with the appropriate clinical history should confirm the diagnosis.

## References

1. Sterns RH, Riggs JE, Schochet SS. Osmotic Demyelination Syndrome Following Correction of Hyponatremia. *N. Engl J Med* 1986; 314:1535-1542
2. Rippe DJ, Edwards MK, D'Amour PG, Holden RW, Roos KL. Case Report. MR Imaging of Central Pontine Myelinolysis. *J. Comput Assist Tomogr* 1987; 11:724-726
3. Price DB, Kramer J, Holson GC, Loh JP. Central Pontine Myelinolysis: Report of a case with distinctive appearance on MR Imaging (letter). *AJNR* 1987; 8:576-577
4. Miller GM, Baker HL, Okasaki H, Whisnant JP. Central Pontine Myelinolysis and Its Imitators: MR Findings. *Radiology* 1988; 168:795-802
5. Moriwaki F, Tashiro K, Marvo Y, Nomura M, Hamada K, Kasiwaba T. MR Imaging of Pontine and Extrapontine Myelinolysis. *J. Comput Assist Tomogr* 1988; 12:446-449
6. Ragland RL, Duffis AW, Gendelman S, Som PM, Rabinowitz JG. Case Report: Central Pontine Myelinolysis with Clinical Recovery: MR documentation. *J. Comput Assist Tomogr* 1989; 13:316-318

# McRae Research Fund Montreal Neurological Institute

Friends and colleagues of Donald L. McRae may wish to show their gratitude for his influence and teaching in the field of Neuroradiology.

The proceeds of this fund will be used to promote education in this field, specifically, to develop new techniques in Interventional Radiology and to support the McRae Lecture in Neuro-Imaging.

Send your donation to the following address:

**McRae Research Fund. Montreal Neurological Institute**  
3801 University, Montréal, Québec, Canada H3A 2B4

*A receipt for tax purpose will be mailed back to you.*

This Bulletin is sponsored by



Winthrop



HEALTH SCIENCES  
DIVISION

NEURO-IMAGE