# NEUROIMAGE

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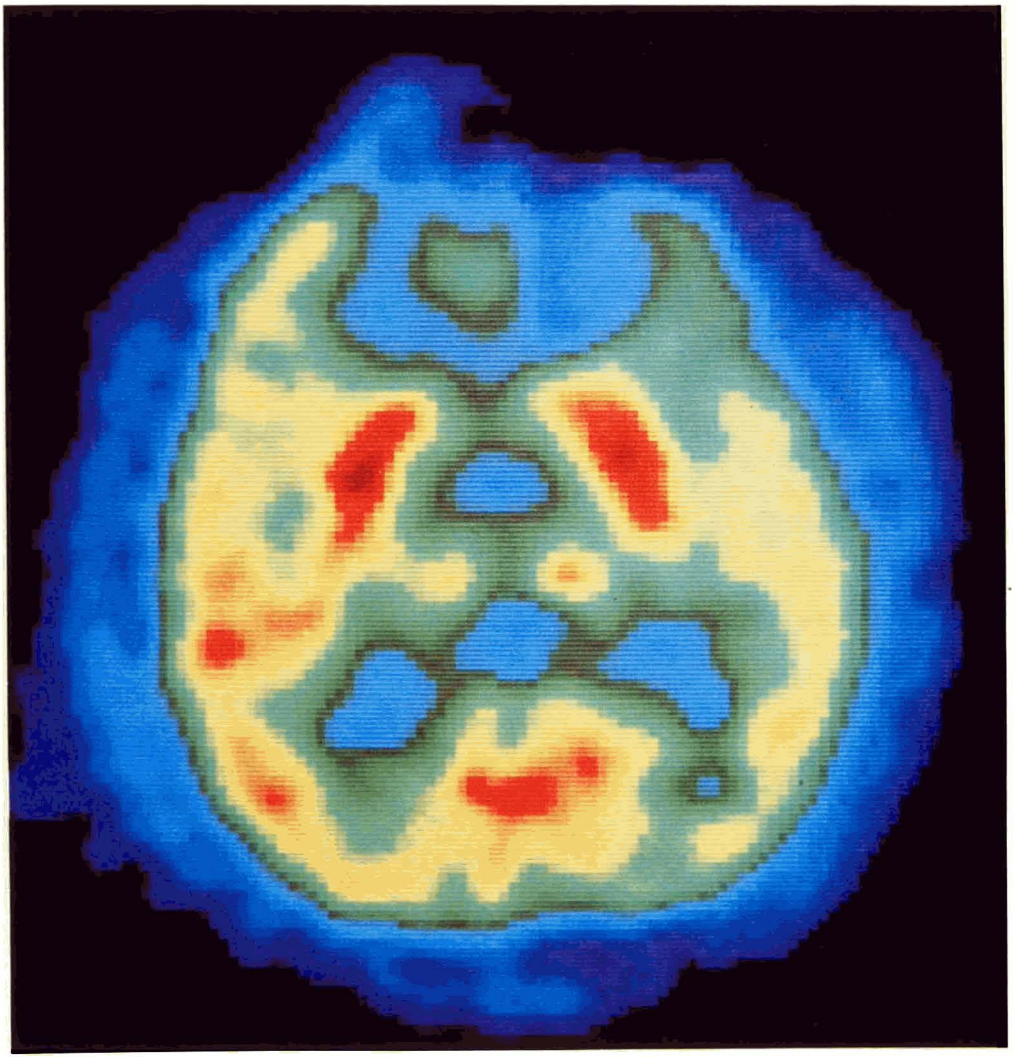
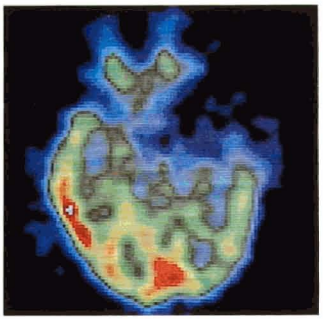
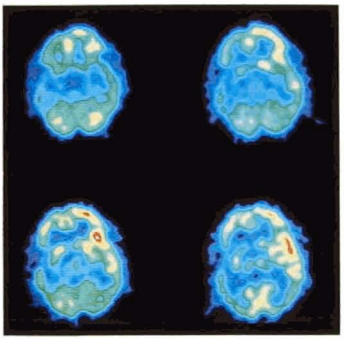
HÔPITAL NEUROLOGIQUE de MONTRÉAL
MONTREAL NEUROLOGICAL INSTITUTE





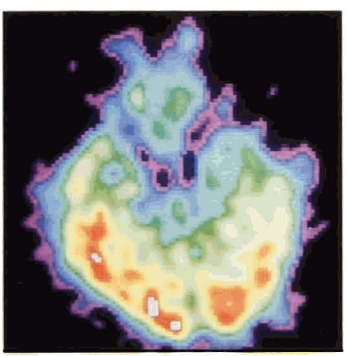
Type I
Unilateral focal hypometabolism is seen in the right anterior temporal lobe.



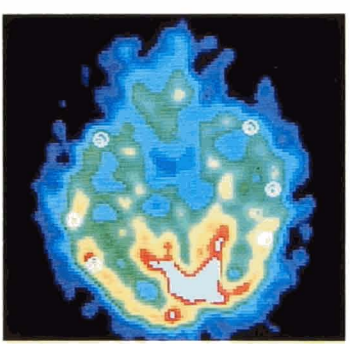
Type II
Focal hypometabolism is seen in the left temporal lobe, with extension to the left parietal area.

# Correlation of EEG and Positron Emission Tomography Studies in Partial Epilepsy

Jane Tyler, m.d.



Type IV
Focal hypometabolism is seen in the anterior right temporal lobe, with relative hypermetabolism in the mid and posterior left temporal regions.



Type V
Bilateral diffuse hypometabolism is seen, with rates of glucose utilization in both temporal lobes of one half the normal rates.

Type III (Front page)
Focal hypometabolism is seen bilaterally in the frontal lobes.

Complex partial seizures are often incompletely controlled despite optimal doses of anti-epileptic medications. In many centers, such as here at the Montreal Neurological Institute, surgical intervention is frequently considered for these patients. Scalp recorded electroencephalograms may be inconclusive in localizing the seizure focus, and computed tomography often shows no abnormalities.

Positron emission tomography (PET) allows us to investigate regional cerebral metabolism; glucose utilization can be studied using the glucose analog deoxyglucose labelled with Fluorine-18, and cerebral blood flow and oxygen metabolism can be examined using oxygen and carbon dioxide labelled with Oxygen-15. Patients are scanned at four levels, with three slices obtained at each level.

In the studies of glucose metabolism, fasting patients are scanned approximately 40 minutes after the intravenous injection of 5 mCi of $^{18}$FDG. Arterialized venous blood samples are obtained during the 90 minutes after injection of the radioisotope, from a vein in the opposite hand, which is heated to 42° in a hot-water glove box developed by Phelps et al (1). Once scanning is completed, images are reconstructed and combined with cross-calibration factors, blood glucose concentration, and blood radioactivity measurements to yield quantitative results for regional cerebral metabolic rates for glucose (rCMRGl).

The studies of cerebral blood flow and oxygen utilization are performed in three procedures. First, the patient is scanned while breathing oxygen-15 labelled molecular oxygen, to study oxygen extraction (rOER) and oxygen utilization ($rCMRO_2$). Next, for measurement of regional cerebral blood flow (rCBF), the patient is scanned while breathing oxygen-15 labelled carbon dioxide. Finally, for measurement of cerebral blood volume (rCBV), the patient is scanned after breathing a small amount of carbon monoxide labelled with oxygen-15 or carbon-11. Arterial blood samples are withdrawn during the study. rCBF, rOER, $rCMRO_2$ and rCBV are quantified with equations developed by Frackowiak et al (2).

EEG recordings are made from the scalp by telemetry during the PET exams. The EEG data is analyzed both visually and by computer analysis for epileptiform sharp waves and spikes.

PET studies at the MNI to date have shown a reduction of rCMRGl, rCBF and $rCMRO_2$ in circumscribed areas of the brain in patient's with partial epilepsy in the interictal state. In the majority of cases, there is good correlation between the epileptogenic focus demonstrated on ictal and interictal EEG studies, and the PET abnormalities. In most cases, the area of hypometabolism seen on the PET study was somewhat more extensive than the area defined by electrophysiological parameters.

In preliminary studies, five different patterns of rCMRGl hypometabolism have been observed (3). Type I showed unilateral focal hypometabolism which correlated closely with maximum epileptic discharges in EEG recordings during the interictal phase. Type II was characterized as focal hypometabolism with ipsilateral local extension of hypometabolism. Type III demonstrated bilateral focal hypometabolism. Type IV showed unilateral focal hypometabolism with contraleral focal hypermetabolism. Type V was characterized by bilateral diffuse hypometabolism with unilateral maximum focal hypometabolism. These rCMR findings are in agreement with those of Engel et al (4) and add further quantitative information with regard to the metabolic changes observed in partial epilepsy. The interictal zone of hypometabolism seen on PET does not identify the epileptic nature of the lesion. Without ictal PET studies, EEG is needed to demonstrate epileptogenicity. These two exams are not redundant, but rather are complimentary. In combination, they should provide more information about neuronal dysfunction in epilepsy than either test used alone.

In addition to studies of patients with partial epilepsy, on-going projects are evaluating patients with generalized epilepsy, patients scheduled for depth electrode implantation, and patients with epilepsia partialis continua.



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# The difficult diagnosis of lymphomatoid granulomatosis

John Milton, m.d.
Denis Melanson, m.d.

**Lymphomatoid granulomatosis is a poorly understood inflammatory angiitis of uncertain pathogenesis with histological similarities to Wegener's granulomatosis and atypical lymphoma. Pulmonary, constitutional, cutaneous, and CENTRAL NERVOUS SYSTEM manifestations predominate, but clinical signs are extraordinarily diverse. Clinical, roentgenographic, and laboratory abnormalities are nonspecific and the diagnosis is frequently missed or delayed (1).**

A 38 year old right-handed male presented with a five week history of decreased memory, confusion, drowsiness and unsteady gait. Five weeks prior to admission he had been involved in a minor motor vehicle accident in which he experienced no loss of consciousness. However, the family noted that he did not remember this event. Over the next few weeks he became progressively drowsy, confused and had unsteadiness of gait. He also had "bizarre behaviour" as, for example, putting his comb in his mouth. One day prior to admission he stopped speaking, became very drowsy and had urinary incontinence. His past medical history was unremarkable except for pneumonia one year ago and frequent episodes of tonsillitis. On admission, the patient was drowsy with little speech output and his examination revealed mild-sided signs: hyperreflexia, up-going toe, facial asymmetry and pronator drift. On day of admission a CAT scan revealed mild hydrocephalus and a space occupying lesion in the region of the left thalamus which appeared to extent along the choroid plexi of both lateral ventricles (fig. 1). The patient was started on steroids. An angiogram was performed which, aside from showing no tumor blush, provided little more insight into the nature of these lesions. A chest x-ray showed a right costophrenic nodule (fig. 2): two other nodules were suspected. Curatorship was obtained and the patient was worked up for a non-lung primary tumor. This work-up included an abdominal ultrasound, intravenous pyelogram, liver-spleen nuclear scan and human chorionic gonadotropin, all of which failed to identify an abnormality. On day 19 a transthoracic needle aspiration of one of the lung lesions was attempted. Cytology showed necrotic tissue containing no organisms and did not grow any organisms on bacterial or mycology culture. On day 22, the possibility of an open lung biopsy was discussed, but it was felt that because of the proximity of the intracranial lesion to the foramen of Monroe and the hydrocephalus, that there would be significant risk to the patient with this procedure. Therefore, on day 27 a repeat transthoracic needle biopsy was performed which again showed only necrotic tissue. Days 36-41 the patient was prepared for stereotaxic needle biopsy of the intracranial mass (including intraventriculoperitoneal shunt) and when this was performed, the cytology again showed necrotic tissue with no organisms growing in the tissue or CSF. In summary, at day 43, it was known that the lesions were not responsive to steroids, that they did not represent an infectious process and that it was not a treatable tumor such as testicular germinoma. Therefore, the plan was made to irradiate the intracranial mass and, if it improved, then to attempt open lung

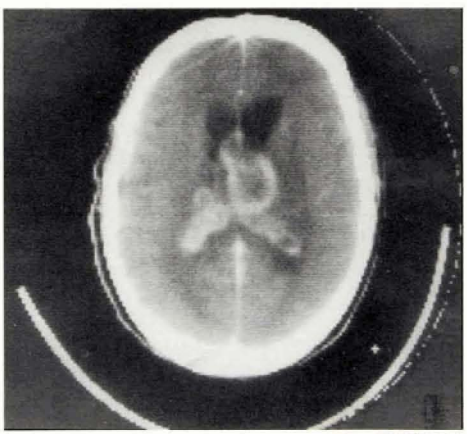

FIGURE 1

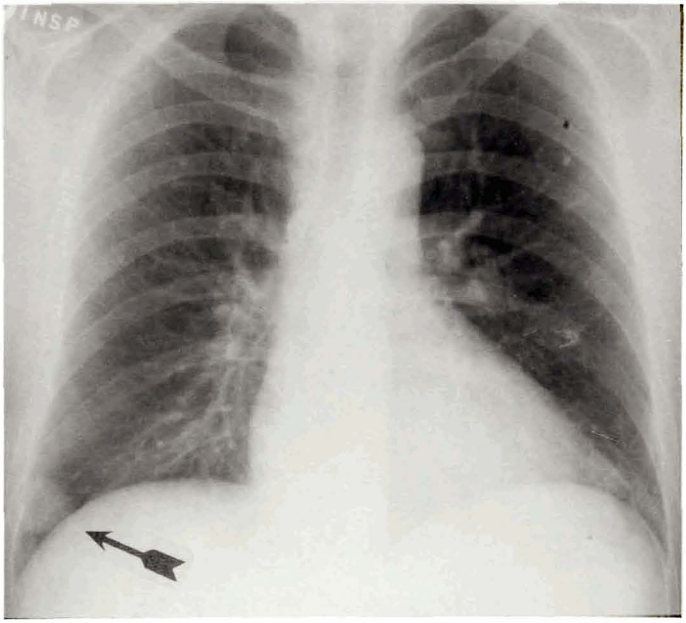

FIGURE 2

biopsy for tissue diagnosis. The patient then received a total of 5600 Rads of radiation in two courses. Repeat CAT scan then showed decrease of the intracranial mass and no hydrocephalus (fig. 3). On day 105, an open lung biopsy was performed. Pathology of the tissue was lymphomatoid granulomatosis. The patient was started on cyclophosphamide and steroids and has showed slow, but steady improvement.

The patient was readmitted, a few months later, for fever, cluths and upper respiratory tract infection. No source of infection was found. He was then restarted on cyclophosphamide and showed a slow improvement, with more initiative and interest. However, he has become a placement problem.



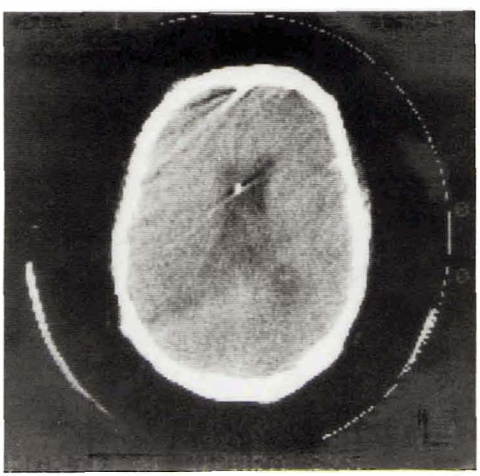

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FIGURE 3

Figures 1 and 3 are not entirely comparable because of accent of contrast in the latter. The patient had a serious reaction to the contrast on early scan and was not reinjected in follow-up examination.

# Secondary syringomyelia

Denis Melanson, m.d.

The various forms of secondary syringomyelia are rare, and their pathogeneses are poorly understood. Of this group, post-traumatic syringomyelia is the most common, and even, it is a rare lesion: the spinal cord becomes relatively fixed at the site of trauma by arachnoid adhesions. This may lead to rupture of the pia (if not already pulled away) at the root entry zone, and eventual communication with the subarachnoid space. We believe that there need not be an existing cavitation from the time of the injury but that it can be created secondarily from the outside penetration. Neurological deterioration usually occurs many years after the initial trauma, as daily life local stress causes the cyst extension upwards and downwards.

This mechanism also explains the formation of a syrinx with the presence of arachnoid adhesions from other causes than trauma, v.g. surgery, bleed, infection. In the case of bleeding, it often occurs at the time of trauma, but without damage to the spinal cord itself. We wish to report an example of such a presentation.

The patient is a 51 year old female who had numerous surgical procedures to her lumbar spine for chronic back problems. Lumbar laminectomies were done in 1969, 1970 and 1971. She also had a $L_5$-$S_1$ rhizotomy in 1973. In 1975, she had peroperative insertion of dorsal stimulators on her lower thoracic cord. More recently, 1981 and 1982, she had repeated myelography.

She was referred to us in December 1983 for recent onset of cervical myelopathy. She underwent Metrizamide myelography and computed tomography.

The Metrizamide myelogram disclosed an abnormality of the spinal cord in the vicinity of the dorsal stimulators (fig. 1): the cord was thin immediately above (fig. 2), and distended below (fig. 3). The cervical cord appeared grossly normal. There was no subarachnoid block. Following the myelogram, a CT scan was performed disclosing the same findings, and showing no abnormality at the foramen magnum. However, on the delayed scan (8 hours) two small cavitations were suspected at the cervical levels.

To disclose the abnormality more readily, a spinal cord percutaneous puncture was made at the level of the lower thoracic cord, where maximal distension had been demonstrated on myelography. Clear fluid was aspirated and Metrizamide was injected (1 cc - 175 mg %). The contrast accumulated in a distended syrinx below the dorsal stimulators (fig. 4). Delayed computed tomography showed it to progress to the cervical cord and to demonstrate two paramedian cavities (fig. 5 and 6), the same cavities suspected on delayed Metrizamide myelography.



FIGURE 1

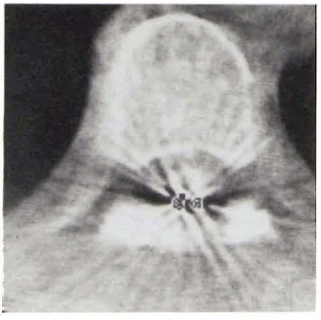


FIGURE 2

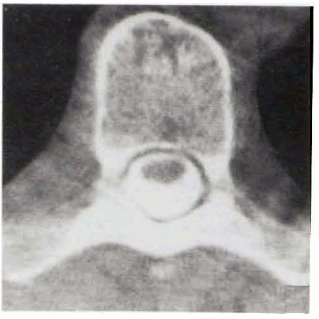
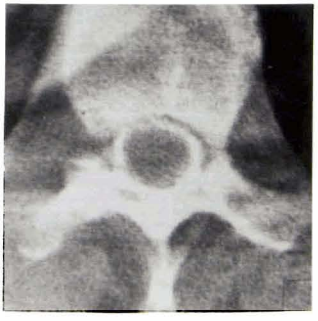


FIGURE 3



## Conclusion

In summary, we have demonstrated with Metrizamide myelography, computed tomography and endomyelography, the cavities of secondary syringomyelia. We have had the opportunity of studying similar such cases (since) and we believe that secondary syringomyelia can develop over the years from any arachnoid adhesions which prevent a normal modus vivendi of the spinal cord in its fluid cushion.

FIGURE 4

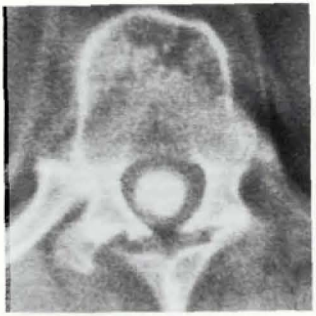
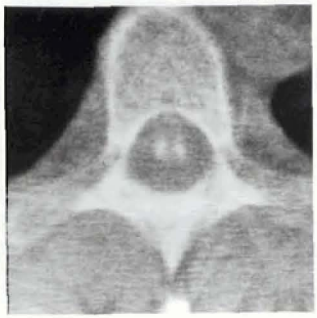


FIGURE 5



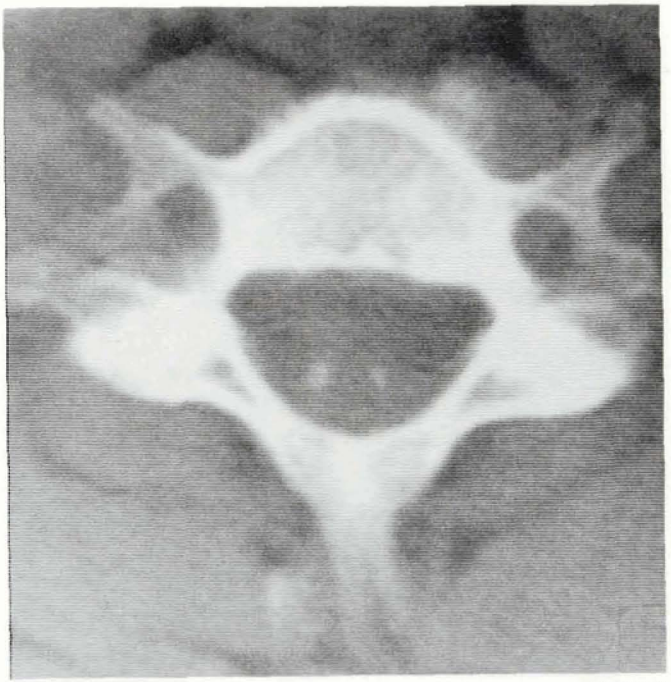


FIGURE 6



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Les sommes recueillies serviront à la promotion de l'enseignement en Neuroradiologie, au développement de nouvelles techniques et à l'organisation des conférences McRae consacrées à l'imagerie Neurologique.

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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.

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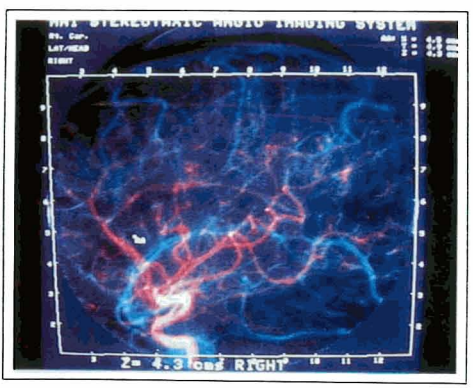


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