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Dr. Irving Heller (1926-2003)

George Karpati, MD, FRCP(C), FRS(C), OC


Dr. Heller, c 1979

Irving Heller meant many things to many people. He was an exceptionally talented and dutiful doctor to thousands of patients. He was a distinguished professor for the Montreal Neurological Institute-Hospital and McGill University, where he was also a cherished colleague to his fellow neurologists and professors. Furthermore, he was a highly valued teacher and advisor of hundreds of students and residents. As well, he was a devoted family man. These are the reasons for which Irving is mourned and missed by multitudes of people who have been beneficiaries of his travails during a long carrier. These are the reasons that in the hall for his memorial service, space was not available for many people. Special mention is to be made about Irving's second career, which he cultivated with fervor and excellence. He became a self-taught scholar of the Old Testament of the Bible, and became a knowledgeable professor in this field at McGill. Another noteworthy item is Irving's special sense of humor and wit, even though it was often sharp and not always flattering to everybody. Irving's humor has lead to the survival of several "hellerisms". One of the more noteworthy of these is the advice that he gave to people with a problem: "Life is a fatal disease".

Irving had a rich life, even though he had to defeat with dignity several major diseases after his retirement. He was respected and admired throughout his career and will be missed.

Words from the Editor

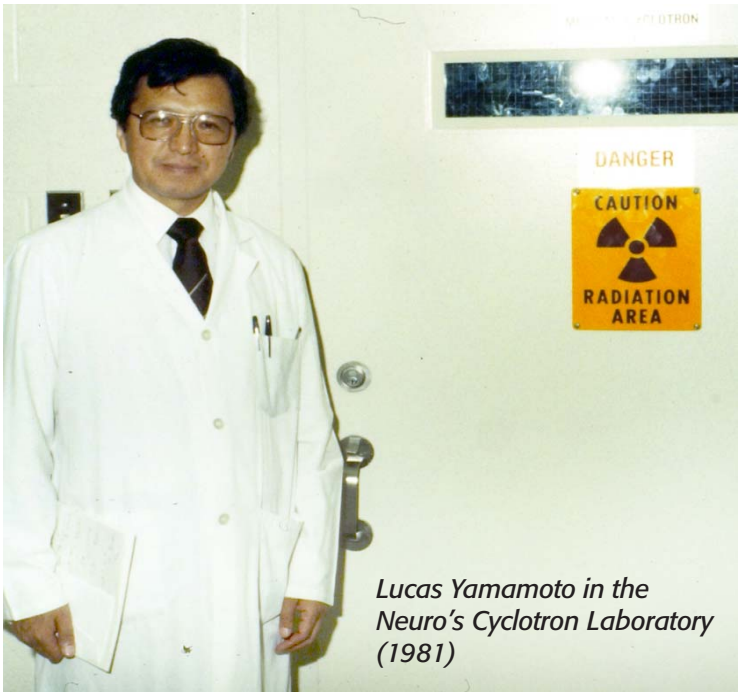
Dr. Denis Melançon

A Bonsai Tree

YAZOKAZU LUCAS YAMAMOTO, MD, PHD (1928-2003)
NEUROSURGEON, NUCLEAR PHYSICIAN AND SCIENTIST

By William Feindel, MDCM, and Mirko Diksic, PHD
Cone Laboratory for Neurosurgical Research,
Montreal Neurological Institute and Hospital, McGill Uinversity

Yazokazu Lucas Yamamoto was internationally recognized for his research in nuclear medicine and in the pathophysiology of brain disorders. It was one of nature's ironies that Lucas passed away on September 18, 2003 from a stroke, the very ailment he had researched for more than three decades.



Lucas Yamamoto in the Neuro's Cyclotron Laboratory (1981)

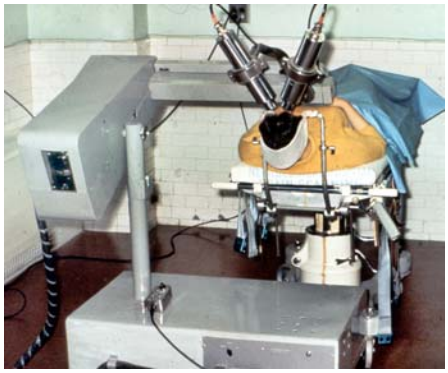
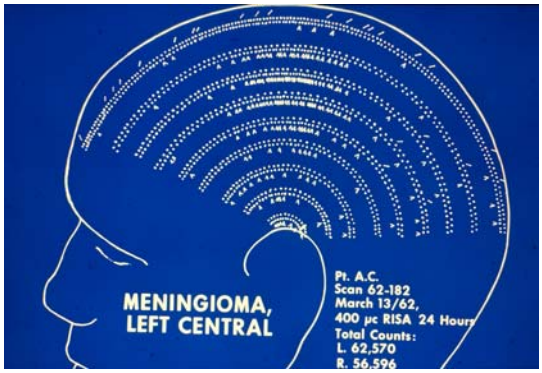
EDUCATION

Born on 19 January 1928 at Shibetzu-shi, Hokkaido, Japan, Lucas completed his medical course in 1952 at Hokkaido University Medical School. After two years of surgery at the International Catholic Hospital, Tokyo, he studied neurosurgery from 1954 to 1958 in Washington, DC, at Georgetown Medical Center and neuropathology at the Armed Forces Institute of Pathology under Dr Webb Haymaker, a former Fellow of the Montreal Neurological Institute. From 1958 to 1961 at the Medical Research Center of Brookhaven National Laboratory, Long Island, NY, Lucas participated as a neurosurgeon with Dr. Lee Farr and his research team in the thermal neutron-capture project using Boron-10 to treat brain tumors. For research on the biological effectiveness of thermal neutrons, Lucas gained his PhD in Radiobiology in 1961 from Yokohama University, Japan.

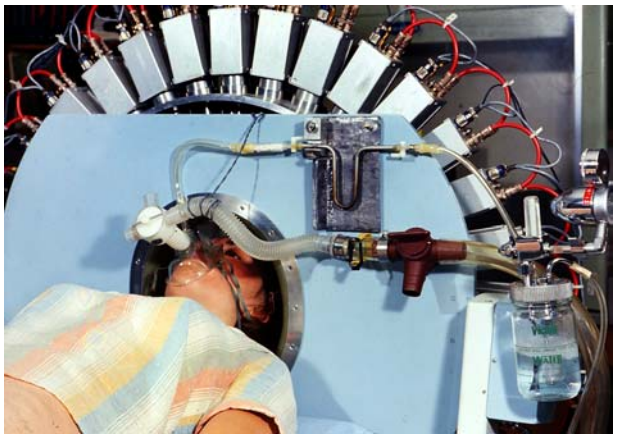
CANADA'S FIRST PET UNIT

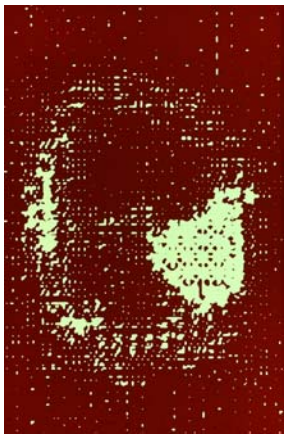
One of us (WF), while visiting Brookhaven in 1960 met Dr. Wenceslao Calvo, a former MNI Fellow who mentioned that Lucas Yamamoto, about to end his term of study in the USA, was interested in coming to Canada. Lucas had by this time married a paediatric nurse, Jeanine Zollner who hailed from Nova Scotia.

So in 1961 Lucas came to the MNI where his training in nuclear medicine and neuro-surgery added great strength to the program already under way in the Cone Laboratory for Neurosurgical Research, utilizing an upgraded model of the MNI's Saskatoon Contour Automatic Neuroisotope Scanner (SCANS). This radioactive system eventually led to the development of PET at the MNI. Lucas was one of the few experts around at that time in neuroisotopes. Continuing summer stints at Brookhaven, he helped to develop the first cerebral blood flow studies by positron emitters using an instrument of 32 sodium iodide detectors (1967), a system later transferred on loan from the US government to the MNI. With the help of the physicists and engineers at McGill, this equipment was modified to produce in 1975 the first tomographic PET scans of a glioma and an infarct.

Saskatoon scanner at MNI (1960) and on the right, scan of a meningioma (1962)

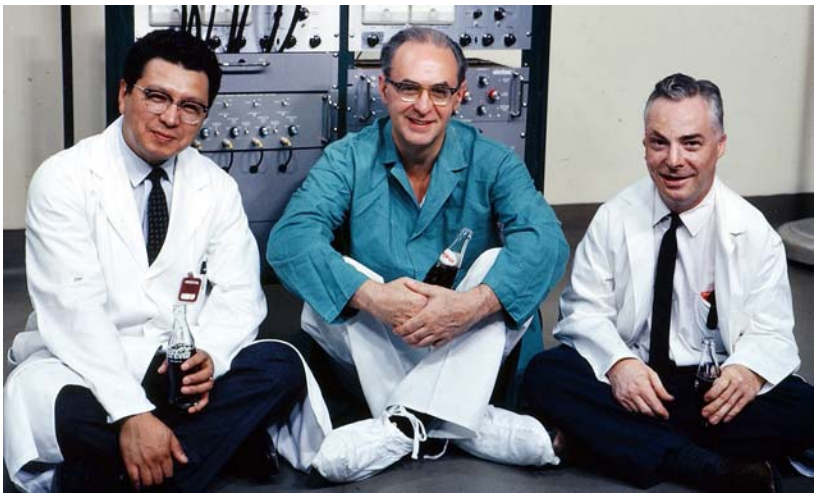




(left)Lucas Yamamoto in scanner at Brookhaven (1967); (centre) the NaI scanner after Neuro-McGill upgrade (1975). (right), the world's first PET cross section scan of a glioma (1975).

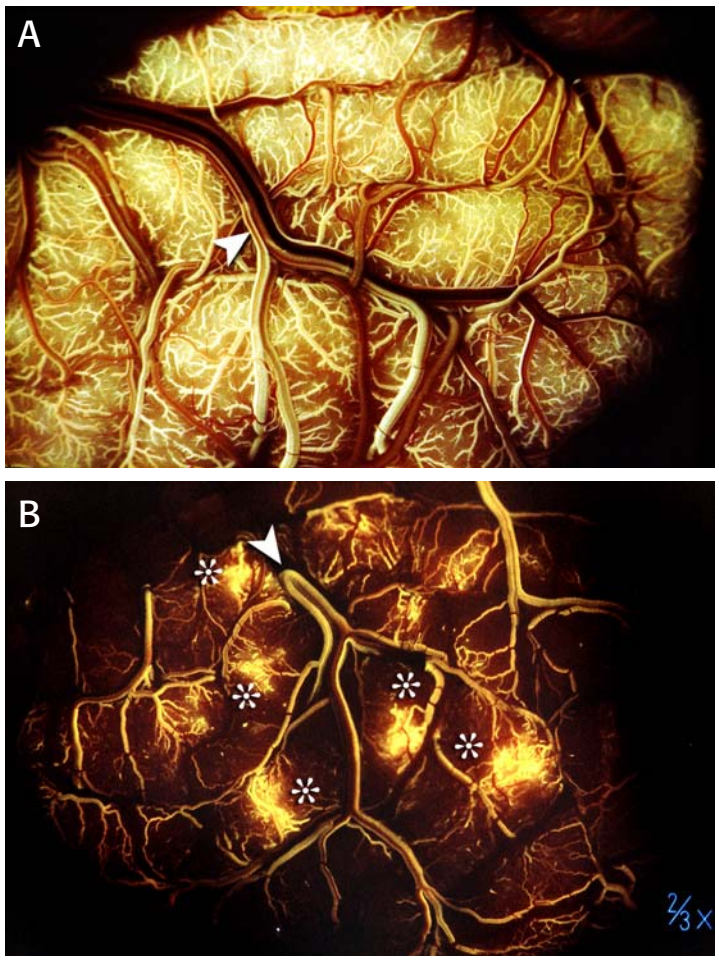
With the team in the Cone Laboratory, Lucas was a key figure in developing PET with a Germanium-68 generator and with Krypton-77 uniquely produced by the physicists and radiochemists at McGill's Foster synchrocyclotron. In 1978 the first PET scanner using Bismuth-Germanate crystal detectors was designed, constructed and tested by Christopher Thompson, Lucas Yamamoto and Ernst Meyer; it became the prototype for many scanners elsewhere. The clinical findings from this BGO scanner "The Positome" were presented at the 1st International Symposium on PET held at the Montreal Neurological Institute in 1978, at which reports from all ten of the world centres reviewed the exciting technical and clinical advances in this emerging imaging field.

CEREBROVASCULAR STUDIES



Yamamoto, Feindel and Hodge comparing the effects of coke and pepsi on cerebral blood flow (1967).

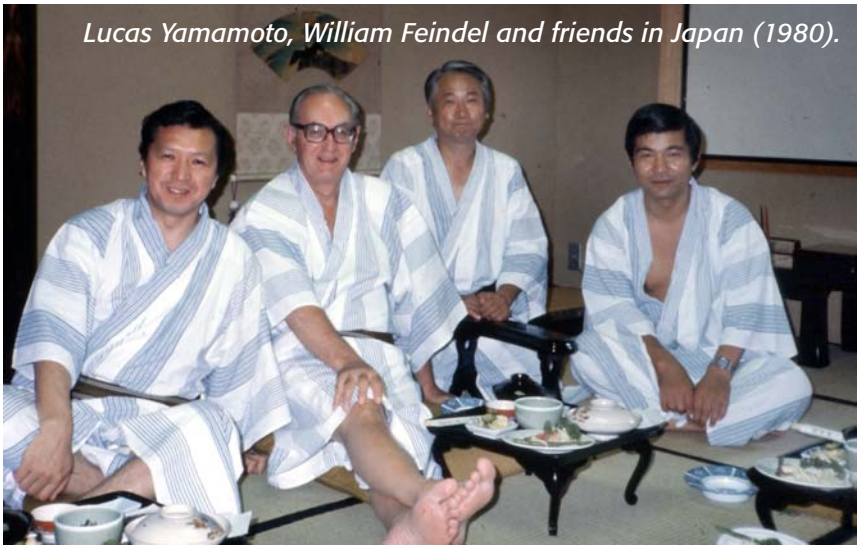
Lucas and his associates in the Cone Laboratory, carried out a vigorous schedule of research for which they devised experimental models of cerebrovascular disorders to elucidate collateral flow, hyperemia and "red veins", carotid-cerebral arterial anastomoses, cerebral vasospasm, brain edema, and the possible role of retrograde venous perfusion in treating acute cerebral ischemia. We worked closely with Charles Hodge to create (1966) fluorescein angiography of the brain both for experimental use and for application in the operating room to treat cerebrovascular malformations. Lucas was author or co-author of over 300 scientific reports.



(A) fluorescein angiogram of the dog brain showing laminar flow in veins (arrow) (B) after occlusion of midline veins, depicting reverse laminar flow (arrow) and perivenous hemorrhages (asterisks) (1968).

THE JAPAN CONNECTION

In 1981 Lucas' energy and enthusiasm played a major role in our acquisition of the first "Baby Cyclotron" outside Japan; it produced positron emitters from the atoms of O, N, C and F. This enabled us to do a wide range of innovative PET studies on the metabolism and chemotherapy of brain tumors, on epilepsy, migraine, psychoses, Alzheimer's and Parkinson's diseases and stroke. Ernst Meyer's method to display cerebral "activation" by PET – focal increase in blood flow with neuronal stimulation – led to exciting research on brain mapping by the neuropsychologists at the MNI. Negotiations for the Baby Cyclotron took our research team on several occasions to Tokyo



Lucas Yamamoto, William Feindel and friends in Japan (1980).

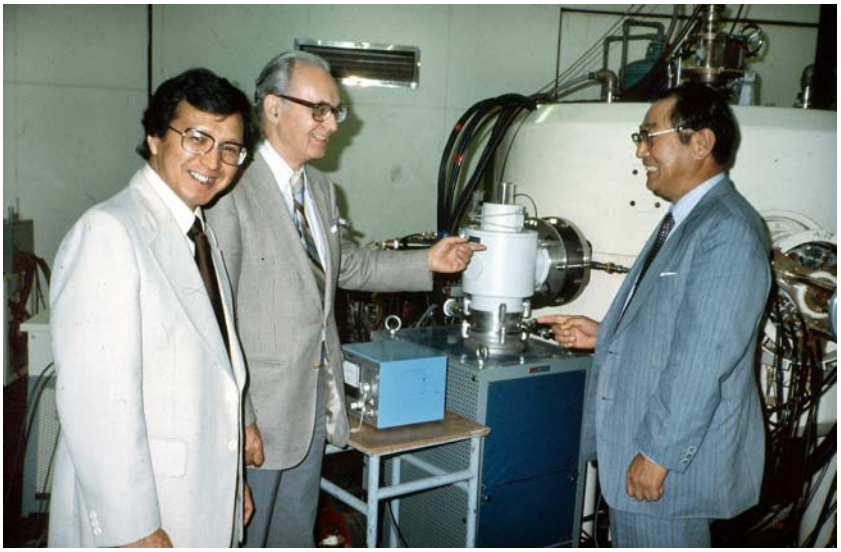
Christopher Thompson, Mirko Diksic, Lucas Yamamoto, Christopher Farr consulting in Japan on the Baby Cyclotron (1980).

and to the north island of Hokkaido to visit the Japan Steel Works at Muroran. We had stopovers in the exotic mountainous countryside with its volcanic hot springs, as in Noboribetsu where the owner of a luxury hotel, who had been a schoolmate of Lucas, treated us like visiting royalty. In the Cone Laboratory Lucas trained more than seventy research fellows from Japan; he not only supervised their professional work but gave them and their families much moral support during their stay in Canada. The annual "pool party" arranged by Lucas and his wife Jeanine for the staff of the research laboratories became legendary. Many went on to become distinguished chiefs of academic departments and directors of hospitals throughout Japan, making up a "Japan Neuro Club".

ACHIEVEMENTS

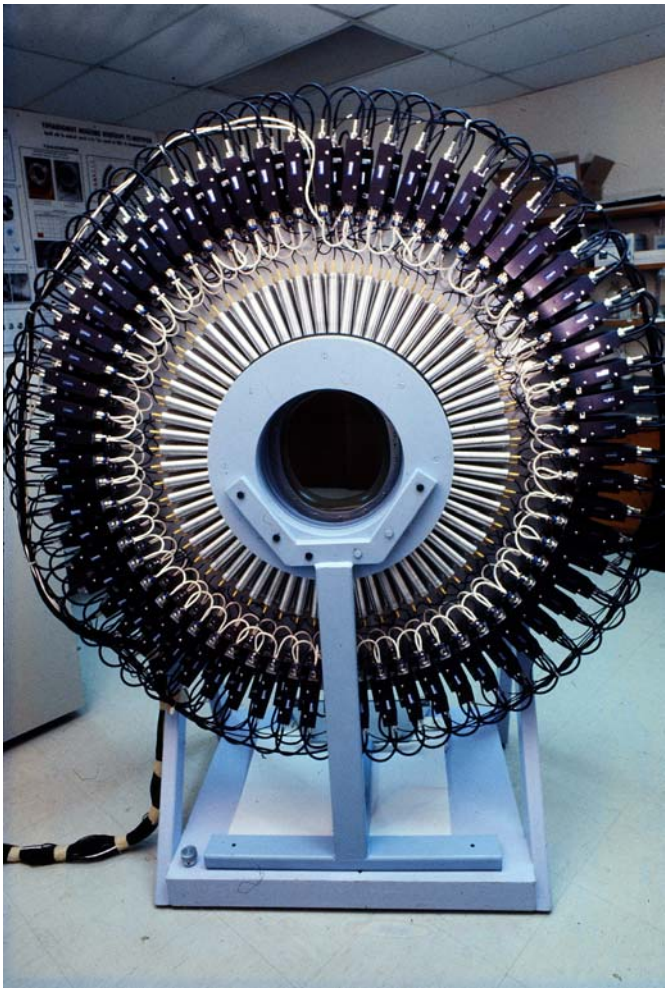
Lucas became a Canadian citizen in 1967. He advanced to Assistant Professor in 1968 and Professor in 1980 in the Department of Neurology and Neurosurgery at McGill, Director of the Neuroisotope Laboratory for Brain Scanning at the MNH from 1973, Chairman, Radiation Safety Committee of the MNI from 1979, and Co-Director of the Cone Laboratory for Neurosurgical Research at the MNI in 1984.

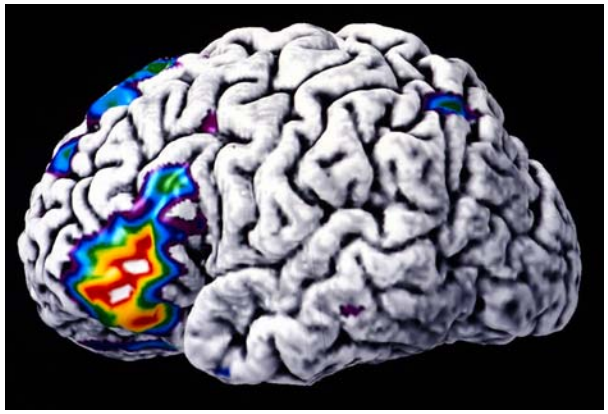
Lucas was very generous to work with, very willing to discuss and to share his knowledge with others. Christopher Thompson, his close colleague for many years, tells how Lucas and he brought the Brookhaven scanner from Long Island to Montreal in the back of a blue van, driving through a blinding snowstorm, succesfully passing through customs despite their "atomic" cargo. He noted that Lucas would deliberately arrange a break when he was flying through Boston so that he might visit Logan airport's famous oyster bar.



Lucas Yamamoto, William Feindel and President Kashihara of Japan Steel Works inaugurating the Baby Cyclotron (1981).

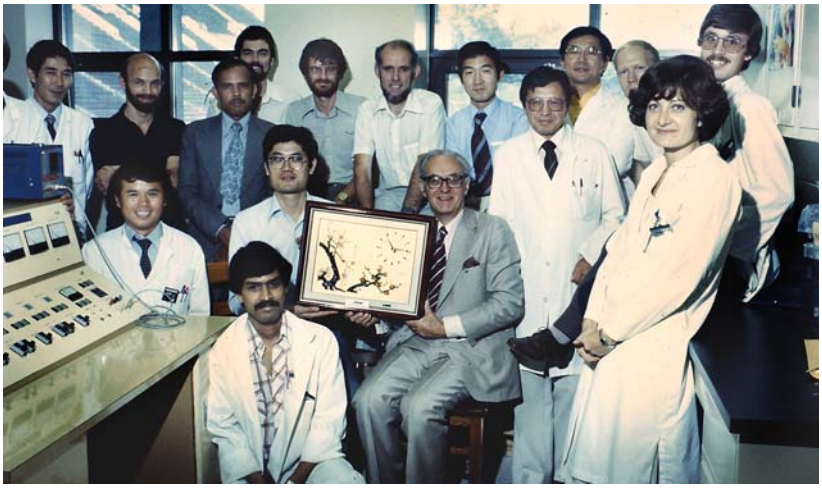
Lucas was a member of many medical associations, including the Canadian Neurosurgical Society, Canadian Association of Nuclear Medicine, and a certified specialist in nuclear medicine of the College of Physicians and Surgeons of Quebec and of the American Board of Nuclear Medicine. He was elected a senior





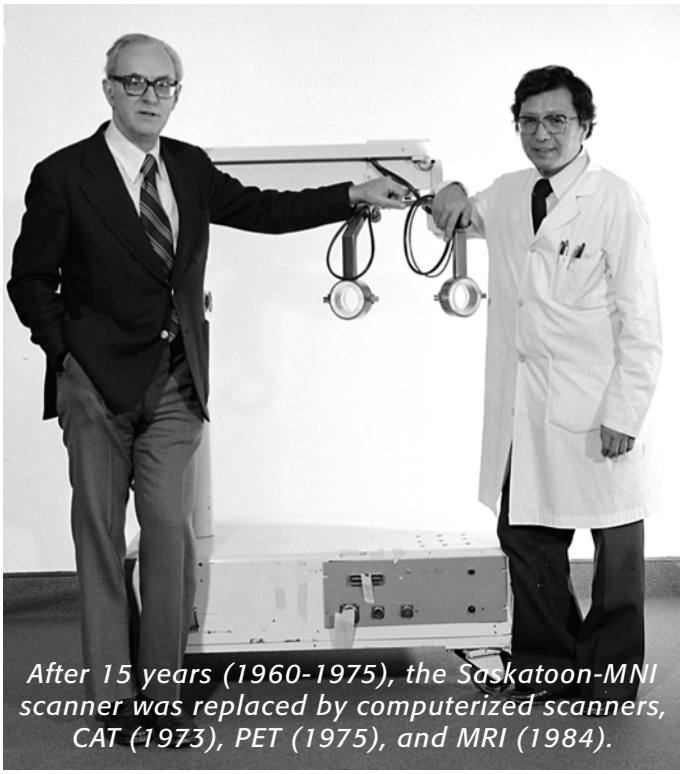
Left is the BGO Positome scanner (1978). On the right, a study by Denise Klein and Brenda Milner, showing the anterior speech area on PET combined with MRI.

Christopher Thompson and Lucas Yamamoto with the Neuro PET prize, 1981.



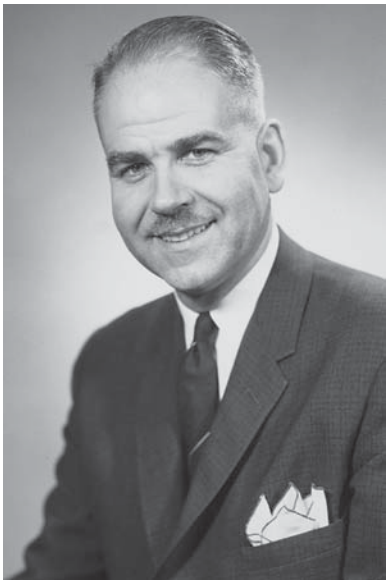
The Neuro-PET Group celebrating the installation of the Baby Cyclotron (1981)

member of the Japan Neurosurgical Society in 1992. As a family physician he provided care to the Japanese community, focusing on the elderly and new arrivals in Montreal. He and Jeanine have two daughters, Ann Marie and Grace, both business executives and a son Peter, a computer engineer. Lucas will be fondly remembered by many of his colleagues and students as a scholar, physician and friend. And with his friends in Kyoto, where they have a special way of saying "Thank you", we can all say, "Ohkini, Lucas"



After 15 years (1960-1975), the Saskatoon-MNI scanner was replaced by computerized scanners, CAT (1973), PET (1975), and MRI (1984).

The family of Lucas Yamamoto has kindly suggested that memorial donations may be directed to the Neuro-Library Fund, and sent to the Director, Montreal Neurological Institute, 3801 University Street, Montreal H3A 2B4



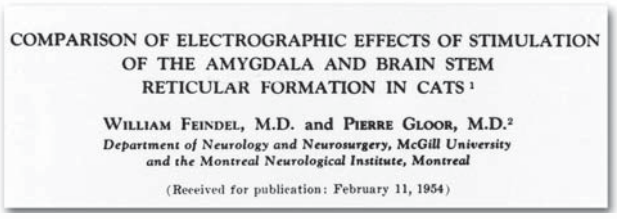
PIERRE GLOOR (1923-2003)
Physician, Brain Scientist & Historian
by William Feindel, MDCM, DPhil, FRCS(C)

Peter Gloor fulfilled many roles as medical doctor, brain scientist, teacher, author, humanist and, to so many of us, a good friend. He added his own particular dimension to the Neuro over the past fifty years.

Peter completed his medical studies at the University of Basel, Switzerland, in 1948 when he was 25. He studied neurology and neurosurgery at Hôpital Louis-Pasteur in Colmar for the next three years, a period reflected in his first twelve publications on neurosurgical problems and the monitoring of general anesthesia by EEG. Awarded a Fellowship of the Swiss Academy of Medical Sciences, Peter came to the Montreal Neurological Institute early in 1952.

Herbert Jasper and Fellows in the Neurophysiology Laboratory (1952). Peter Gloor stands in the back row, 2nd from the left.

Shortly after his arrival he joined me in the Neurophysiology Laboratory to elucidate the role of the amygdala in epilepsy. Dr Penfield and I had shown a few months earlier in the operating room, working with Dr Jasper, that the amygdala played a crucial role in the generation of seizures coming from the temporal lobe and characterized by automatism and amnesia. As I wrote earlier about our experiences in that 7th-floor lab, "those were exciting days – and nights. Peter and I would start the experiments in the early morning and continue well past midnight, often seeing the sunrise before we folded up reams of EEG paper regurgitated from an ancient Offner machine that recorded our results." Peter at that time was fond of "Cizane" cigarettes with their strong mixture of Turkish tobacco. One of our serendipitous findings was a spontaneous seizure discharge from the electrode in the amygdala which we soon attributed to a smoking Cizane that lay on the operating table and was evidently stimulating the animal's olfactory system. We never published that observation.



COMPARISON OF ELECTROGRAPHIC EFFECTS OF STIMULATION OF THE AMYGDALA AND BRAIN STEM RETICULAR FORMATION IN CATS [1]

WILLIAM FEINDEL, M.D. and PIERRE GLOOR, M.D.[2]
Department of Neurology and Neurosurgery, McGill University and the Montreal Neurological Institute, Montreal

(Received for publication: February 11, 1954)

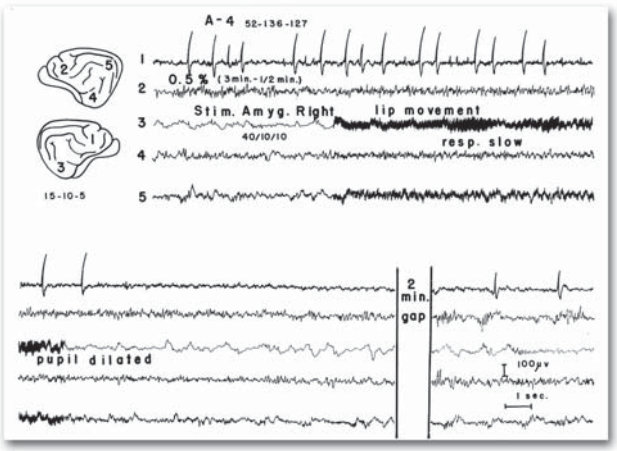


Figure 9 from our original paper shows seizure activity and arrest of strychnine spikes from the cortex with stimulation of the amygdala.

As two residents in training, scheduled soon to go on the clinical services, we worked hard for four months and published our findings in the EEG Journal in 1954. In that paper we listed fewer than twenty references found from an assiduous literature search in the Neuro Library. This morning on the Web, I clicked onto "Google" for the subject "Amygdala". I received "about 173,000" hits and the interesting piece of information, "Search took 0.26 seconds". Quite a change in fifty years! Peter pursued this topic for his PhD thesis, and continued these studies to become a world authority on the anatomy and physiology of the amygdala.

Peter Gloor and Hanna Pappius (c.1955)

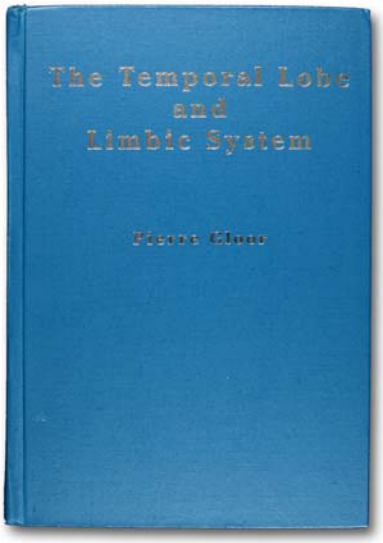


(Left) *Mary Roach and Peter Gloor (c.1955)* ***(Centre)*** *Peter in the O.R. Gallery (1980)* ***(Right)*** *Herbert Jasper, Massimo Avoli, Peter Gloor (1992)*

A decade later Peter succeeded Herbert Jasper as Chief of the Laboratory of Electro-encephalography then as Director of the Neuro-physiology Laboratory at the Neuro. The latter was efficiently supervised by Nurse Mary Roach, to whom Peter paid tribute on her retirement. In the meantime I returned from Saskatoon to become the Cone Professor of Neurosurgery. So we once again worked together on the amygdala during epilepsy surgery, when Peter would provide advice from the OR gallery on electrocorticography. And we co-authored a long review in 1963 on "Affective behavior and temporal lobe". It was published in a multi-author volume, edited by Professor Monnier from Basel and printed in Stuttgart, so aside from Peter and myself, I expect there were few in North America who became aware of our industrious effort.

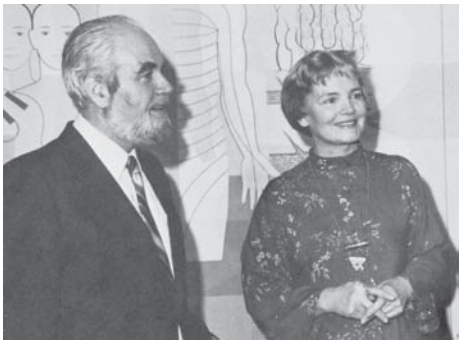
Peter lecturing in the Hughlings Jackson Amphitheatre (1990)

Peter was a dedicated teacher of numerous young physicians and scientists and for the technical staff of the EEG Laboratory. He contributed critical studies on experimental epilepsy triggered by penicillin, observations on depth stimulation of the mesial temporal structures and early efforts with Lucas Yamamoto, Jean Gotman and myself to examine epilepsy using PET imaging. An avid historian, Peter was responsible for translating and publishing the pioneer papers of Hans Berger, the psychiatrist who invented EEG.

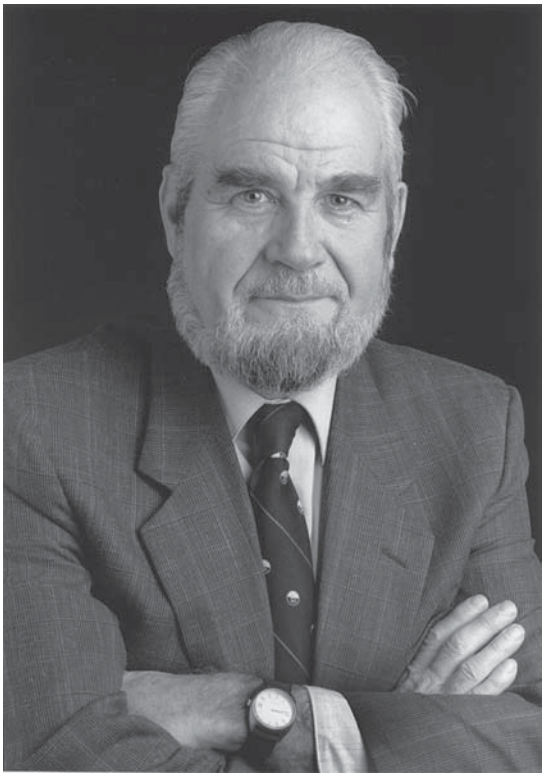


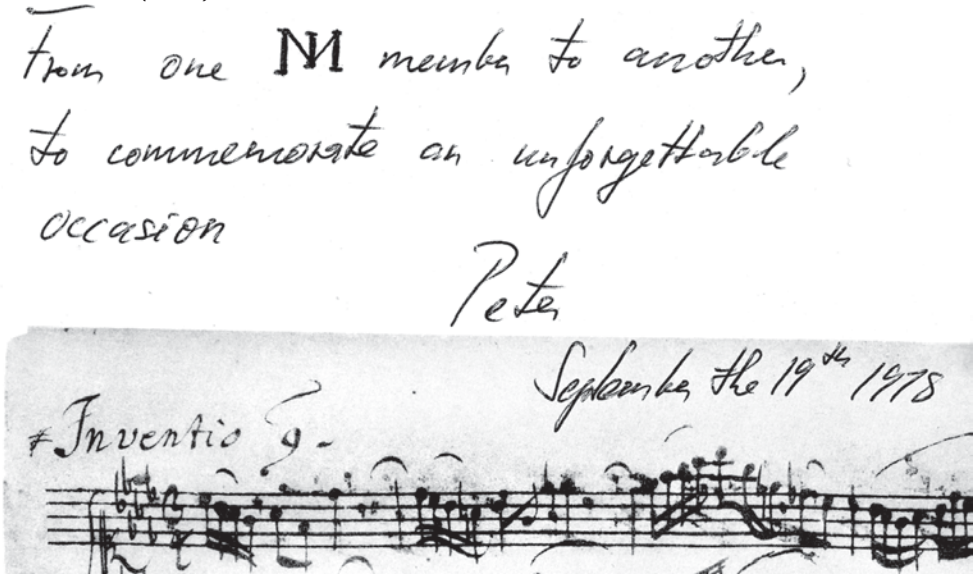
In the late 1980s, Peter focused on a major project – to marshal a full-scale scholarly critique of the vast canon of literature dealing with the temporal lobe and its disorders. He was in a unique position to undertake such a formidable task from his deep understanding of the physiology of the amygdala and hippocampus, and his continuing involvement in the medical and surgical treatment of patients with temporal lobe epilepsy. Unfortunately, as we all know, in September of 1994, before he had finished this task, a severe stroke disabled his language function. Those of us who realized how much effort he had devoted to this project, and what a fundamental contribution it would make to neurology, devised a plan to complete and prepare his manuscript for publication as an expression of our esteem, admiration and friendship for Peter. This definitive monograph, published in 1997, by Oxford University Press, will continue to speak for Peter Gloor for years to come.

Luba Genush Gloor, an acclaimed artist, created a splendid mural for the Penfield Pavilion in 1954 in which she depicted features of the nervous system and neurotechnology.



Peter and Luba at unveiling of her mural "The Neurosciences"– (1954)





The portrait of Peter on your Program, that you see here on the left, has caught the sparkle in his eyes and his smile, which on occasion could break into hearty laughter. The watch on his wrist we can take as symbolic of his Swiss origin and his precisely organized work schedules. And he wears the Neuro tie with the logo we devised for our Third Foundation in 1978 when we celebrated the opening of the Penfield Pavilion. At that time, quite unexpectedly, I received the fine Dover facsimile edition of Johann Sebastian Bach's *Two- and Three-part Inventions* for the pianoforte. It was inscribed "From one M member to another, to commemorate an unforgettable occasion" and was signed "Peter, September the 19th 1978". I was deeply touched by this gift and flattered by Peter's high expectations of my strictly amateur talents as a Bach performer. Today I cherish even more this small book as a symbol of our long and wonderful friendship. Peter and Luba have one daughter, Irene a McGill graduate, married to Eric Shoubridge, Director, Neurogenetics Laboratory at the Neuro, and a son Daniel, an engineering graduate, married to Donna, a teacher.

As Rainer Maria Rilke wrote in one of his poems in *Das Stundenbuch* [Book of Hours]:

> *Ich höre jeden in mir schreiten*
> *und breite meine Einsamkeiten*
> *von Anbeginn zu Anbeginn.*

Victor Epp has kindly translated these lines:

> *I hear everyone stride within me*
> *and I spread out my loneliness*
> *from beginning to beginning.*

(Adapted from remarks at a memorial service on 3 December 2003 in Birks Chapel, McGill University; with illustrations added from the Neuroarchives. The family of Peter Gloor has kindly suggested that memorial donations may be directed to the Neuro-Library Fund, and sent to the Director, Montreal Neurological Institute, 3801 University Street, Montreal H3A 2B4)

Peter, Luba and group at the Symposium on Amygdala and Seizures, Jeanne Timmins Amphitheatre, MNI, June 2003.

CREUTZFELDT-JACOB DISEASE

Ribeiro, LT; Matos, ALM; Simão, GN; Takaynagui, O and Santos, AC.
Center of Imaging Sciences and Medical Physics, University of São Paulo (USP) – Brazil.

INTRODUCTION

Creutzfeldt-Jacob disease is a rare dementing illness caused by an agent called a prion. This entity is classified into four main subtypes: familial forms (fCJD), sporadic forms (sCJD), iatrogenic forms (iCJD) from cadaveric hormones-related transmission or neurosurgical procedures, and the recently described variant form (vCJD) from animal contaminated food products (1). Approximately 90% of the cases are sporadic, without any detectable cause (1).

Probable clinical diagnosis is suggested by rapidly progressive dementia, myoclonic jerks and periodic sharp-wave electroencephalographic (EEG) activity. But the classical symptoms may be lacking in as many as 25% of the patients, and the antemortem diagnosis remains problematic (1, 2). The cerebrospinal fluid may have evidence of 14-3-3 brain protein, however false positives and negatives may occur (3). Histological analysis of the brain tissue provides the definite diagnosis. The histopathological findings of sCJD are characterized by astrocytosis and neuron loss, being particularly prominent in the putamen and head of caudate nucleus associated with cytoplasmatic vacuole accumulation (4).

Recently, many authors reported cases of sCJD with typical findings at magnetic resonance imaging (MRI) in 67 to 79% of cases. The patients studied showed classic hyperintensity of the cerebral cortex and basal ganglia (putamen and caudate head) on T2-weighted images (T2W), FLAIR and diffusion-weighted (DWI) sequences, in both the early and late stages (more than 4 months after the onset of symptoms). This finding is indicative of restricted water diffusion and is seen mainly in the basal ganglia. The T1-weighted sequences are usually normal but may demonstrate slight hypointensity in the basal ganglia. The contrast enhancement does not occur (2, 4-10). According to these authors, these typical imaging findings, mainly the DWI findings, in conjunction with the classical clinical presentation, could facilitate the antemortem diagnosis of this rare entity.

We report radiological aspects, structural and diffusion MRI, of two cases with sCJD typical clinical presentation. In the second case, we show the MRI evolution a year and ten months after the onset of the symptoms.

CASE 1

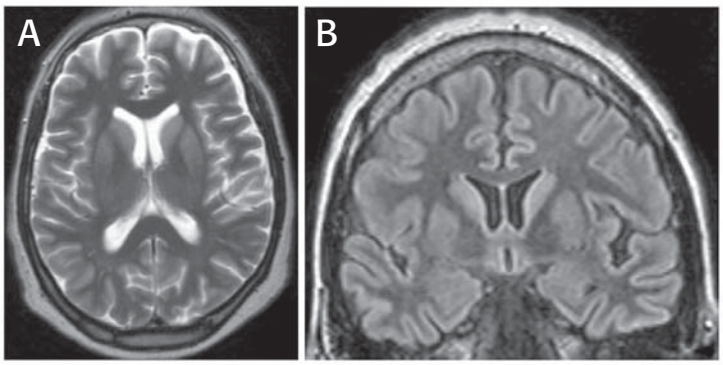


Figure 1. *A. axial T2W and B. coronal FLAIR sequences demonstrate hyperintensity of the basal ganglia (putamen and caudate head).*

The patient was a 64 year old man. In June of 2001, the patient began to experience apathy and sleep disturbance. The family noticed memory impairment, with a rapidly progressive course. Three months into the disease process, the patient presented to the neurologist with severe apathy with no verbal contact. The neurological exam revealed quadraparesis associated with extrapyramidal rigidity, global hypereflexia, bilateral Babinski sign and facial myoclonic jerks. Cerebrospinal fluid (CSF) was normal and the EEG showed typical periodical sharp-wave activity.

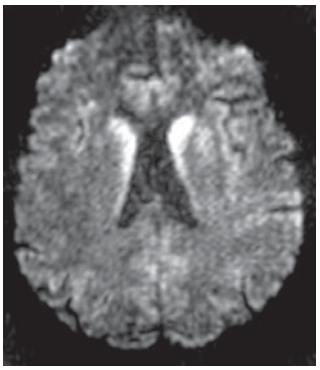


Figure 2. *An axial DW with (b = 1000 mm2/s) demonstrates restriction of water diffusion in the basal ganglia (putamen and caudate head).*

The MRI was performed 3 months after the onset of symptoms and demonstrated marked hyperintensity in the basal ganglia on T2W, FLAIR and DWI sequences (Figure 1 and 2). Hyperintensity in the basal frontal cortex was also noted. The T1W images showed a faint hypointensity of the basal ganglia and no contrast enhancement. The apparent coefficient diffusion (ADC) in the striatum was 0,52 x 10-3 mm2/s (mean 0,74 ± 0,12 x 10-3 mm2/s in voluntary subjects, 64,9 ± 14,2 years) (5).

Basic biochemical and serological exams were performed and were negative, including HIV and VDRL.

The patient was lost to follow up in October 2001.

CASE 2

The patient was a 61 year old man who presented in March of 2001 with apathy, dysarthria and ataxic gait. Rapidly progressive memory impairment was noticed by family members. The neurological examination, performed two months after the onset of symptoms revealed sporadic myoclonic facial jerks, dysarthria, quadraparesis associated with extrapyramidal rigidity, global hypereflexia, bilateral Babinski sign and cognitive impairment.

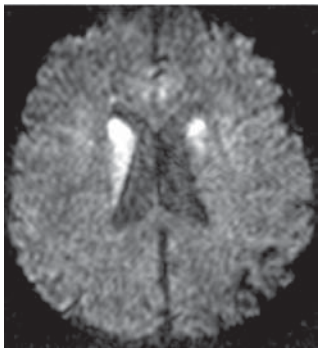


***Figure 3**. A. axial T2W and B. axial FLAIR sequences demonstrate hyperintensity in the basal ganglia (putamen and caudate head).*

At that time, the protein P 14-3-3 was positive in the cerebrospinal fluid (CSF), but the rest of the CSF analysis was normal. The EEG showed typical periodical sharp-wave activity.

The MRI was performed at the presentation and revealed hyperintensity of the basal ganglia on T2W, FLAIR and DWI sequences. The T1W sequence showed a slight hypointensity of the basal ganglia and no contrast enhancement (Figure 3 and 4). The ADC was 0,44 x 10-3 mm2/s in the putamen and 0,41 x 10-3 mm2/s in the caudate head, confirming restriction of water diffusion (5). Basic biochemical and serological exams were performed and were negative.

At 6 months, the patient developed a complete irresponsible state, requiring respiratory assistance.

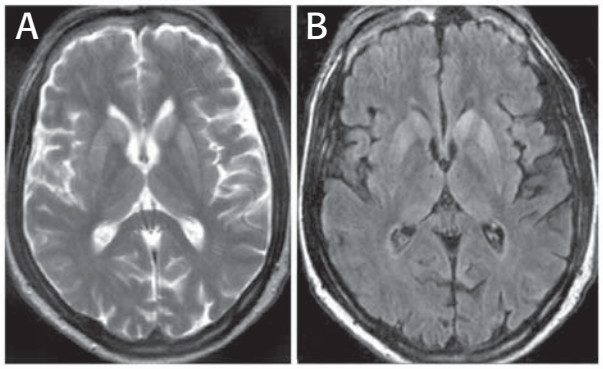


***Figure 4.** DWI (b = 1000 mm2/s) sequence showed demonstrates restricted water diffusion in the basal ganglia (putamen and caudate head).*

In January of 2003, a year and ten months after the onset of symptoms, the patient was kept under intensive care and underwent a second MRI. The T1W images demonstrated diffuse marked brain atrophy associated with frontoparietal subdural effusions (figure 5). The signal abnormalities in the basal ganglia were no longer apparent on T2W or DWI images (Figure 6).

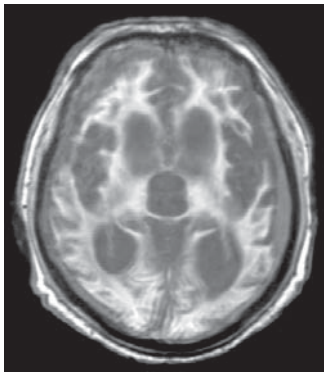


***Figure 5.** Diffuse brain atrophy on axial T1W image associated with bilateral extra-axial bifrontoparietal effusions.*

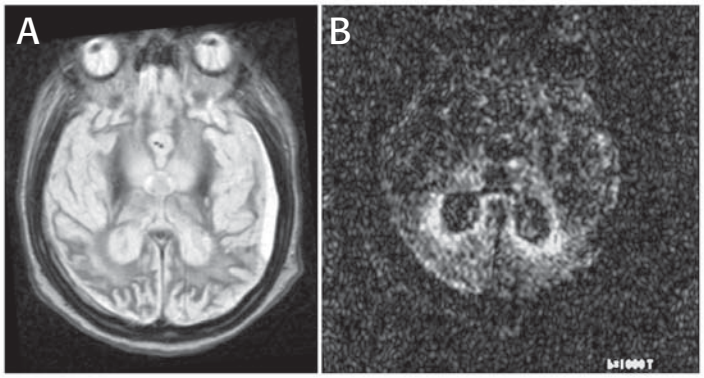


***Figure 6.** A. axial T2W shows diffuse significant atrophy of the brain and B. supranormal water diffusion on DWI sequence (b = 1000 mm2/s).*

DISCUSSION

The classic triad of CJD includes rapidly progressive dementia, typical EEG findings (periodic sharp wave) and myoclonic jerks. Both patients manifested these classical symptoms, fulfilling the clinical diagnostic criterias for CJD (1, 2).

Moreover, both patients showed signal changes on T2W and FLAIR in the putamen and caudate nuclei early in the course of the disease (2 to 4 months after first symptoms). In addition, the DWI sequence demonstrated restriction of the water motion in the striatum. The striatal hyperintensity could be seen early in the evolution of sCJD and supported the clinical diagnosis (2, 5-7, 9). The DWI is the most specific for the diagnosis of sCJD (2). Many diseases could manifest with basal ganglia hyperintensity on T2W, DWI and FLAIR images (ischemic, edematous, metabolic and toxic lesions, inborn errors of metabolism, Leigh's disease and mitochondrial encephalomyopathies in general), but these findings are transitory and observed in the acute phases of these diseases only (9). The persistence of the DWI hyperintensity images associated with typical clinical signs greatly suggests sCJD (2). Although, there are few reports about late phase of sCJD and we do not know for how long the restriction of water motion could be noted on the DWI.

Murata et al. (2002) discussed in their study the persistence of striatal and cortical hyperintensities on FLAIR and DWI images in the early and late stage of sCJD (2). In their study, the authors define the late stage of sCJD as more than 4 months after the onset of symptoms. Since the patient studied in our paper has such a long disease evolution and such profound brain damage, this might explain the disappearance of the striatal hyperintensities on T2W and DWI images. These suggest that the changes in the late stage of sCJD are unspecific and compatible with any encephalopathic process (8).

The origin of the basal ganglia diffusion abnormality in the sCJD is not well-known to date. Some authors have suggested that the vacuolar accumulation in the cellular cytoplasm could explain the restriction of the water motion on DWI (5, 6).

In conclusion, further studies are necessary to understand the pathophysiological mechanisms involved in the basal ganglia diffusion restriction in patients with sCJD. The role of MRI, mainly the T2W and DWI sequences is well established in the earlier stages of the disease process. In more advanced phases of the disease, when severe brain atrophy is often observed, the DWI sequence fails to demonstrate a specific pattern.

Bibliography available upon request.

Gratefulness: We thank Dr. Jeff Chankowsky for reviewing the manuscript and suggestions.

"The Year in Review"

T'is just weeks before New Years and here sit we
reflecting back on the year 2000 and 3.

In January Alyson arrived on the hill
and Eric and Stef received chairs from McGill.

The snows of February caste a pall on CIHR,
but with 75% success the MNI was a star.

The Penfield lecture in March was never a bore
as Jeff Hawkins did Handsprings across the floor.

In April salary awards poured in to many of you
from lots of places, including the FRSQ.

We also had candidates stream through the doors.
They gave some great talks; they wandered our floors.

In May there were prizes to break any gloom -
two Brain Stars for students; an award for this room.

In June more candidates from far, far away;
finally we settled on J-F Cloutier.

In July more CIHR results came to pass
and once again the MNI kicked ass.

And George went to Italy to accept a prize.
He returned with his suitcase which was quite a surprise.

Much of the summer was quiet as a mouse
except for the media coverage of Paus.

And newspapers, they wrote many a story
about Alonso, Colman, Kennedy, Milner, Petrides, Sossin...
and, of course, Zatorre.

In the fall a lot happened and most of it was swell -
A great lecture from Tilman; another by Kandel.

In London I witnessed as hundreds came
for Bill Feindel's induction to the Hall of Fame.

And the fame of Fred Andermann was also revealed
when he received the Prix de Penfield.

In September the Glen Yards again came to fore
and Dave warned the Dean – there could be a war.

To Mr. Culver he reasoned, "You don't have the money,
so leave us alone, this is no longer funny."

In October the faculty got into the fray,
and everyone voted "the Neuro should stay."

In November Board members each caste their vote.
We reached part of our goal but it's no time to gloat.

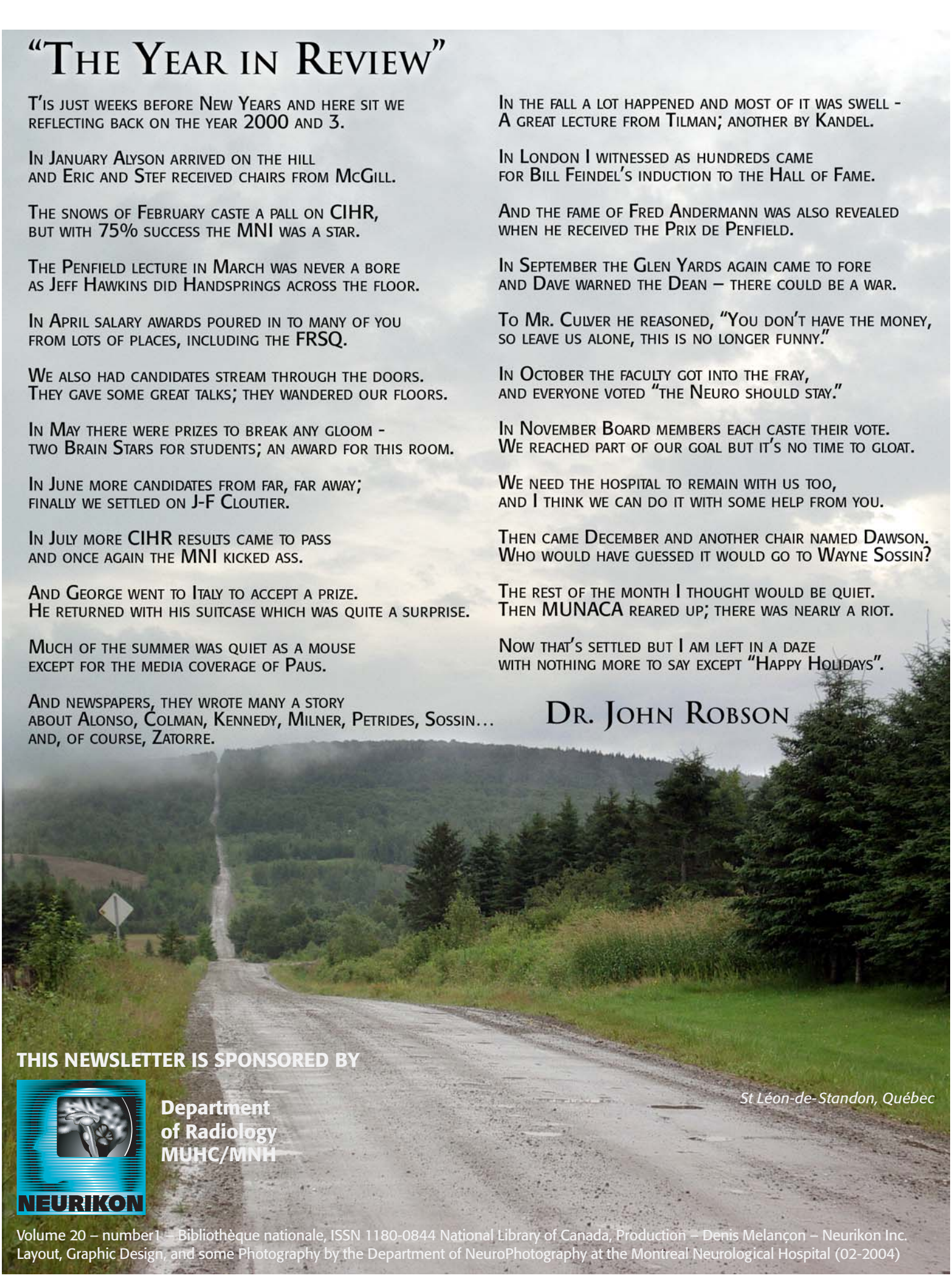
We need the hospital to remain with us too,
and I think we can do it with some help from you.

Then came December and another chair named Dawson.
Who would have guessed it would go to Wayne Sossin?

The rest of the month I thought would be quiet.
Then MUNACA reared up; there was nearly a riot.

Now that's settled but I am left in a daze
with nothing more to say except "Happy Holidays".

Dr. John Robson



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