Candidates’ Statements for President

VLADIMIR HACHINSKI, C.M., M.D.
FRCPC, D.S.C., DR. HON. CAUSA
Introduction
I was born in Ukraine and raised in Venezuela. I graduated in medicine from the University of Toronto, Canada. I studied cerebral circulation physiology at the National Hospital for Nervous Diseases at Queen Square in London, England, and followed that with a research fellowship at Bispebjerg Hospital in Copenhagen, Denmark. I returned to Toronto, where I began my career and was later appointed the Richard and Beryl Ivey Professor and Chair of the Department of Neurological Sciences at the University of Western Ontario in London, Canada.

I began the Controversies in Neurology section in the Archives of Neurology, which engages a range of contentious issues in current neurology and which has become the journal’s most popular feature.

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Introduction
I have served the World Federation of Neurology in various capacities for almost 25 years and plan to continue serving it with the same dedication and devotion. I am grateful to the WFN Nominating Committee for considering my candidacy for the office of President.

Having worked as a neurology teacher, researcher, and clinical neuropyschiatrist for almost 5 decades and having spent most of my life in a developing nation, I know what it is like to live in a society where people have no access to neurological services. As a result, I understand the extent to which neurological education and services are needed in developing and underdeveloped countries.

My main aim as President will be to strengthen both of these essentials—education and services—in developing countries, and to raise awareness of neurology. I am eager to bring all member countries of the United Nations into the WFN fold and to maintain its close ties with the World Health Organization, American Academy of Neurology, and European Federation of Neurological Associations.

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Suicidality Alert for AEDs May Do More Harm Than Good

BY MICHÈLE G. SULLIVAN
Elsevier Global Medical News

For years, scientifically sound data have informed clinicians that patients with epilepsy have an increased risk of depression. But the U.S. Food and Drug Administration’s recent warning of an increased risk of suicidal ideation in patients on antiepileptic drugs, and the European Medicines Agency’s recommendation to disclose such risk in product information for some of the drugs, are based on much less rigorous data, and could do more harm than good, say several experts on epilepsy.

The FDA’s decision, announced last December, requires manufacturers of any medication in the class of antiepileptics to add warnings about suicidal thoughts or behavior in prescribing information or labeling and to develop medication guides for patients.

“Following the initial FDA alert reporting increased suicidal ideation with anticonvulsant drugs, a number of my patients called to ask if they should discontinue their medications. They did not realize, as I did, that although any risk of suicidal ideation was very small, the risk of stopping medications, with possible uncontrolled seizures, was much greater. Fortunately I easily convinced them of this, but I do have colleagues who report that some patients stopped medications,” said Dr. Carl W. Bazil, professor of clinical neurology at Columbia University, New York.

The EMEA only recommended that the risks should be mentioned with the product information for the antiepileptic drugs (AEDs) levetiracetam, pregabalin, lamotrigine, and zonisamide. The European agency also said prescribers should be advised to monitor their patients for suicidal ideation or behavior.

See Suicidality • page 4

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See Suicidality • page 4
EDITOR IN CHIEF'S COLUMN

Risky Research and Sharing Stories

By Mark Hallett, M.D.

Carleton Gajdusek has died, and one of his students and colleagues, Lev Goldfarb, has written an obituary in this issue of World Neurology (see p. 14). Carleton was brilliant and a very colorful character whom I got to know in the National Institute of Neurological Disorders and Stroke (NINDS) intramural program at the National Institutes of Health in Bethesda, Md., U.S.A. His lectures were incredible and spellbinding. Back in the days before PowerPoint, I recall one of his lectures when he had three screens—dual slides and a movie—all running simultaneously. He loved to tell stories, and he always had a few jokes.

It is worthwhile to consider the work he did in discovering a new class of infectious agents, which earned him the 1976 Nobel Prize for Medicine. In travels to the island of New Guinea north of Australia, he lived with the Fore tribe and studied tribe members who were inflicted with kuru disease. He characterized the properties of the infectious agent were unusual. His work opened an entirely new area of biochemistry and medicine. If he had embarked on his research today, would he have been able to get a grant to do so? Was his work hypothesis directed? Could he have gotten funding for longer than 5 years? It is easy to imagine how every, the NINDS intramural program took the risk for a potentially high payoff. Dr. Murray Goldstein, the director of NINDS, tells the story of how Carleton once needed transport to get around in the Pacific region in order to continue his work. He simply hired a ship and sent the bill to Dr. Murray—who paid it.

It makes one wonder if today’s funding agencies have become too conservative and short sighted. Sometimes it seems that the requirement for preliminary data means that the work is already half done at application time. Many funding agencies have recognized this and are trying to find ways to bypass the conservative approach and identify some risky projects that might succeed. This is certainly difficult but well worth doing.

But back to Carleton, he was also the source of many stories. He was always crying wolf about how the mice in Bethesda were going to shed the virus that would unleash a plague of hemorrhagic fever with renal syndrome. The Hantavirus was unknown in the United States—except for that one mouse that his children caught on his property. So I was not surprised when he called me in the late 1980s (I was wearing an administrative hat at that stage) to say that he had just been to England as a consultant and that it appeared that some cows had come down with a scrapie-like disease. He predicted that this was going to be big news, having important health consequences, and cost millions of dollars. I thanked him politely and then forgot about it, until the mad cow disease stories hit the press. He was right that time. (Actually, he was sort of right about the hemorrhagic fever with renal syndrome as well. Many years later, there was just such an epidemic in the southwestern United States.)

In thinking about Carleton, it occurred to me that one thing neurologists enjoy when they get together is swapping stories, often about old times, their mentors, or great teachers. Many of these exchanges are part of our oral tradition, but I thought it would be fun and possibly even valuable—if some of those stories were put down on paper and sent to us for publication. So, as with my call for you to submit articles relating to our specialty, I also invite you to share a favorite story with us.

I am grateful to David Dawson who is kicking off this feature in the current issue. On page 4, he tells the story of Soma Weiss, the brilliant young doctor who diagnosed his own ruptured cerebral aneurysm in the days before the clinical pathological correlation was well known. This issue, we have our first Letter to the Editor—also on page 4. Keep those coming as well.

Do you have a idea for a story? Do you want to comment on something you’ve read recently in WORLD NEUROLOGY? Or maybe you’d like us to share news of your research or upcoming event with the global neurology community? Send us an e-mail at worldneurology@gmail.com. We’d love to hear from you.
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Dr. Soma Weiss, a physician and biochemist, played an important role in academic medicine in the mid-20th century despite his untimely death at the age of 43.

Although he had not trained as a neurologist, he was acutely aware of the contributions neurosurgeons, notably Harvey Cushing, had made to the study of neurological disease, and of his many published papers, more than a dozen were about epilepsy, vascular disease of the brain, beri-beri, carotid sinus sensitivity, and other conditions.

Dr. Weiss had studied physiology and chemistry in Europe before emigrating to New York when he was 21. Soon after arriving in the United States, he went to study at the Cornell Medical School in upstate New York, where he established himself as a postdoc.

He continued his medical training at the Boston City Hospital as part of the Harvard Medical School academic program. After completing his training, he advanced rapidly in his field and succeeded Dr. Henry Christian as chief of the department of medicine at the Peter Bent Brigham Hospital and as Hersey Professor of the Theory and Practice of Physick, the oldest professorship at Harvard Medical School. His appointment to this prestigious position, at the age of 39, was a striking testament to his brilliance.

Those who knew Dr. Weiss always commented on his enthusiasm, warmth, drive, and legendary teaching ability. His ward rounds were known to attract was many as 100 students and doctors. He would go from floor to floor, seeing as many patients as were presented to him.

In late December 1941, Dr. Weiss was returning by train to Boston from Washing- ton. He was an advisor to President Franklin D. Roosevelt, and on this occasion planning for the civilian aspects of the war was the reason for his visit. During the trip, he experienced a sudden blinding headache, vomiting, and stiff neck. He himself diagnosed a ruptured aneurysm, which his physicians later confirmed.

Dr. Weiss was cared for at home and visited daily by one or the other of his junior faculty, John Romano and Charles A. Janeway. A friend, Dr. H. Houston Merritt, also attended. No neurosurgeon was consulted; in any case, surgical procedures for aneurysm were in their infancy at that time.

Dr. Weiss improved slowly, but after a restless night in January 1942 he had a second hemorrhage and died the next day. An autopsy showed an anterior communicating aneurysm, with rupture into the frontal lobes.

His death at such an early age, and at the beginning of his career at the Peter Bent Brigham Hospital, was widely mourned. Each year, in remembrance of his contributions as a teacher, clinician, and advocate for research, the Harvard Medical School hosts the Soma Weiss Student Research Day, at which students present their research.

Albert Saiz and Dr. Francesco Graus discussed the pathogenic significance of autoantibodies against glutamic acid decarboxylase 65 in stiff person syndrome (WORLD NEUROLOGY, December 2008, p. 16).

Glutamic acid decarboxylase 65 (GAD65) catalyzes the synthesis of gamma-aminobutyric acid, and glutaminergic and GABAergic neurons express high levels of GAD65 antibodies (GAD65-Ab) could possibly alter the balance between excitatory and inhibitory neurotransmitters. However, GAD65 is located in the cytosol and is therefore not readily accessible to antibodies. Nevertheless, transfer of immunoglobulin G (IgG) from GAD65-Ab-positive stiff person syndrome (SPS) patients induces an SPS-like phenotype in rats (Ann. Neurol. 2007;61:544-51).

We have recently shown that GAD65-Ab from cerebrospinal fluid (CSF) of GAD65-Ab-positive patients with SPS has high avidity, and persists for years (Eur. J. Neurolog. 2008;9:973-80). This suggests they are produced by clonally expanded B cells that have received T-cell help. We found it possible to clone GAD65-specific T cells from the CSF of three patients with, but not from a single patient without, prominent intrathecal synthesis of GAD65-Ab (J. Autoimmun. 2009;34:124-32). We therefore conclude that clonally expanded GAD65-specific B cells and T cells coexist intrathecially.

Dr. Saiz and Dr. Graus state that they cannot draw conclusions from some autopsy reports. This does not disqualify T cells from the pathogenesis of SPS. There are few histological reports, and some have even shown discrete lymphocyte infiltration (Clin. Neuropathol. 1986;5:40-6 and J. Clin. Neurosci. 2002;9:328-9). Moreover, absence of evidence is not evidence of absence. The clonal expansion of B cells secreting GAD65-Ab to the CSF must reside somewhere. Lack of overt inflammation in CNS specimens may suggest that the inflammatory process is small and few, rather than nonexistent.

Intrathecal synthesis of oligoclonal IgG is a hallmark of MS. In MS, the mechanism of IgG production is difficult to study because the specificity of the oligoclonal IgG is largely unknown. The finding that clonally expanded B cells and T cells specific for the same autoantigen exist intrathecially in SPS should encourage the study of T-B cell collaboration also in MS.

Trygve Holmøy M.D.
Institute of Immunology, University of Oslo and the Department of Neurology, Ullevål University Hospital, Oslo, Norway

Dr. Saiz and Dr. Graus reply: We agree with Dr. Holmøy’s points. He provides interesting data on the need to study T-B cell collaboration. However, we cannot add any comment since our studies in this matter were clinical.
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Stopping AEDs Poses Greater Risk

Suicidality • from page 1

while being treated with those drugs.

"The suicidality warnings have made very little difference to our clinical prac-
tice," said Prof. Martin J. Brodie, direc-
tor of the epilepsy unit at the Western Infirmary, Glasgow, Scotland.

"It is well known that around a third or perhaps even more patients with re-
fractory epilepsy will have comorbid de-
pression as part of a panoply of brain dysfunction. This is associated with a small increased risk of suicide. A num-
ber of antiepileptic drugs can produce depression in a small number of pa-
ients," Prof. Brodie said.

"As it stands, epileptologists have long been aware of the importance of check-
ning for potential suicidality in this popu-
lation. However, I do not routinely warn patients of the possibility when intro-
ducing a new antiepileptic drug," Prof. Brodie said.

On the surface, Dr. Bazil said that the FDA’s alert does sound frightening. Pa-
tients receiving AEDs have twice the risk of suicidal behavior or ideation that of those taking placebo. But even with this doubling, the risk remained small—
less than 6.5% of patients taking the drugs. Moreover, the FDA recommenda-
tion was based on a retrospective analy-
sis of 199 separate AED trials. None of these trials was de-
signed to examine the risk of suicidal ideation; instead, this information was collected dur-
ing patient self-re-
ports of adverse events.

These reports, determined by a series of open-ended questions posed by the investigator, have a notorious po-
tential for bias. If the patient complains of one adverse event—depression, for ex-
ample—the investigator will automati-
cally ask more questions, including ques-
tions about suicidality. And because it’s known that drugs always evoke more ad-
verse events than do placebos, once a pa-
tient reports an adverse event, investi-
gators will have increased vigilance for the possibility of another adverse event—suicidality—could be recorded more often in the drug group than in the placebo group, even if the rates were similar in the two groups, according to Dr. Bazil, who is a member of the speaker’s bureau for UCBS Pharma, Pfizer, and GlaxoSmithKline.

Although the risk of suicidality is small, the risk associated with stopping medications is much greater.

DR. BAZIL

In the trials that generated the suicidi-
ality warning, both the active and place-
bo treated patients were asked the same question, two, or three baseline antiepileptic drugs. Accordingly, the cause and effect rela-
tionship is by no means clear," Prof. Brodie said.

In the absence of prospective study us-
ing specially validated psychological measures, physicians should make sure that patients understand this is not a high risk, and that the drug is very safe when taken as directed, Dr. Bazil concluded.
Introduction

The prevailing discrepancies in the practice of neurology throughout the world present great challenges for the World Federation of Neurology. In Europe and North America, neurology has advanced substantially with the advent of modern technology and advances in research in molecular biology, and these advances translate into more accurate diagnoses and effective therapies for patients. But these considerable advantages are available to a relatively small percentage of the world’s population—in many regions of Africa, Asia, and Oceania, the practice of neurology is either inadequate or simply not available.

BY LEONTINO BATTISTI, M.D.

Background

I was born in 1939. I received my medical degree from the University of Padua, and from 1967 to 1970, I was a visiting research fellow at Columbia University’s Research Institute of Neurochemistry in New York, U.S.A. On my return to Italy, I was assistant professor in the department of neurology at Padua’s medical school. After tenures as associate and then full professor in neurology, I was a named director of the school’s Neurological Clinic. In 2002, I became head of the department of neurosciences.

In addition, I have been a member of the financial committee of the European Federation of Neurological Societies; president of the Italian Society of Parkinson’s Disease and the European Society for Clinical Neuropharmacology; and organizer and chair of world congresses on neurorehabilitation and clinical pharmacology.

WFN Activities

I have had the privilege of serving the WFN as a participant in the Parkinsonian & Related Disorders, the Dementia, and the Organization and Delivery of Neurological Services research groups; as a member of the federation’s Nominating Committee, as its Vice-President for Europe; and as Organizer and Chair of its congress on Parkinson’s disease in Rome in 1994.

Goals and Objectives

The federation faces many obligations as it contemplates the future. It needs to:

- Develop and expand the practice of neurology worldwide;
- Support educational programs;
- Cooperate with national and international health organizations, such as the World Health Organization;
- Establish a policy for providing training and a support network for young neurologists; and
- Promote neurological and neurorehabilitation centers.

I believe collaboration and collegial decision making and am ever mindful that we need to be realistic in executing projects in a timely manner and without wasting resources. To achieve these goals, I would work to:

- Strengthen the organization by engaging a professional agency;
- Adopt a policy of transparency and ensure that all information about its activities reaches its members quickly; and
- Hold congresses, teaching courses, and scientific events throughout the world.

WMcCullar Carroll, M.D., B.S., M.D., FRACP, FRCP

Introduction

Over the last 10 years, I have seen the World Federation of Neurology expand its role to a truly global strategic organization and embark on sustained endeavors in education and advocacy. The most recent of these has been the Africa initiative. The WFN faces a considerable challenge to continue such initiatives, while at the same time not neglecting other regions such as Central and Southeast Asia and parts of Eastern Europe. In these financially strained times, it is the internal strength of organizations that determine continued growth and influence. Successful use of modern communications can bolster our organization by improving members’ proximity to the organization and its leadership. The reformed WORLD NEUROLOGY, modernized Web site, and move to a biennial world congress have begun this process.

Background

Below are listed some of the positions that I have held, or currently occupy, followed by a summary of my WFN activities, which I believe support my candidacy:

2008-present Vice-President, Asian & Oceania Association of Neurology
2007-present Vice-President, Pan-Asian Committee for the Treatment and Research in Multiple Sclerosis
2004-present WFN Director, World Neurological Foundation
2003-present Editor (Asia & Pacific), Multiple Sclerosis 1998-present Chair, Research Management Council of Multiple Sclerosis Research Australia
1996-2004 Neurology Editor, Journal of Internal Medicine
1992-2000 President and Councillor, Australian Association of Neurologists (now the Australian and New Zealand Association of Neurologists)
1948-present Head, Department of Neurology, Sir Charles Gardiner Hospital, Perth, Australia.

WFN Activities

My association with the WFN began when I served as president of the Australian Association of Neurologists in 1999. At the 17th World Congress of Neurology in 2001 in London, I presented the successful bid for the AAN to host the 2005 18th World Congress of Neurology in Sydney. In 2001, I was also elected as a WFN Trustee and served two terms. As President of the 2005 World Congress of Neurology in Sydney, I was proud to be associated with its success, especially financially, with considerable funds accruing to the federation. I have chaired the WFN Fundraising Committee, and I serve on the Membership and the Publication & Website committees, as a member of the Editorial Board of WORLD NEUROLOGY, and as an invited member of the Task and Advisory Force for Neurology in Africa.

Aims and Objectives

- To enhance the WFN’s ability to communicate with neurologists and neurological associations worldwide.
- To maintain existing initiatives in Africa and Central America and develop others elsewhere in the world.
- To develop an equitable formula for subscriptions for countries of differing economic circumstances.
- To cement the WFN’s financial stability.

Werner Hacke, M.D., Ph.D.

Introduction

My three main objectives as vice president of the World Federation of Neurology would be to improve neurological care and research; to ensure that the advances in the diagnosis and treatment of neurological diseases are made available in all regions of the world, and to make neurology visible in the field of medicine.

Background

I am a neurologist and a psychologist by training. I am 61 years old and have been chair of the department of neurology at the University of Heidelberg (Germany), for more than 20 years. My main interest areas of interest in neurology have always been stroke and critical care neurology, though I also have a major interest in higher brain functions and neuropsychology.

International Activities

During the past 20 years I have devoted much time to international teaching activities. Many delegates will know me from their national or regional neurology and stroke conferences, at which I have had the honor to speak.

I have received honorary membership from several neurological societies, including the American Neurological Association, the French Neurological Society, and the All-Russian Society of Neurologists.

Currently, I am president of the European Stroke Organisation and in the past, I have been president of the German Neurological Society, the German Stroke Society, and the German Society for Neurological Critical Care Medicine.

WFN Activities

I became involved in the WFN very early in my career. In 1995, I became secretary to the World Congress of Neurology (WCN) president, the late Professor Klaus Pocock. I was the founder and founding president of the WFN’s Research Group for Critical Care Neurology. In 2006, I became an elected trustee with the WFN, and I have worked actively on the Board of Trustees since then.

As the chair of the Conference Oversight Committee, I worked on the future structure of the WCN and its change to a biennial conference.

Goals and Objectives

- To continue to work on the new format of the World Stroke Conferences with special a special emphasis on geographic distribution and input.
- To pursue greater collaboration with our colleagues in neuromedicine—with neurosurgeons, neuroradiologists, neuropaediatricians, and neuropathologists.
- To help raise the profile of neurology. Our specialty has not received the public attention it deserves as a discipline that works with our most precious organ. Considering the amount of funding and public interest that is given to cancer, AIDS, and heart disease, we have a long way to go.
- And most importantly, to promote teaching and training activities for neurologists in developing regions of the world and to support broader training and qualification programs.
We don’t know about you, but when we were choosing our careers, it didn’t occur to us that we would find ourselves in the crosshairs of terrorism. We weren’t becoming police officers, after all, or even high-earning bankers whose children might be at risk for kidnapping.

We were going to become researchers, which meant years of education and commitment to postdoctoral programs and residencies for the privilege of competing for meager federal funding from the U.S. government. We were picking a profession in which our goal would be to understand the workings of neural systems, so that we could help correct the defects—biochemical and anatomical—that cause disease.

In addition to benefiting humankind, we knew that animals would also benefit by the work that we were doing.

When we were students, extremist acts against animal research were happening only in England; they posed no threat to most researchers in the U.S.A. But that has changed.

The British research community has succeeded in educating the public about the importance of its work and, because of that, the number of extremist acts has all but disappeared. In contrast, extremism against animal research has increased in other countries such as Europe, Canada and the United States, Asia, Australia and South America.

Today, many of those engaged in neurological research are aggressively targeted by these extremists. Being targeted means having their homes with bullhorns and distributing pamphlets that claim that we torture animals. The leaflets include our home addresses and phone numbers with the gentle suggestion that the reader express “outrage” in what we do. Targeting means stalking our children and spray painting or firebombing our homes and cars.

These extremists demonize and misrepresent both what we do and its value. They distribute “educational” materials in many schools, claiming that our research is worthless and that researchers are actually engaged in federally supported animal torture.

How do we respond to this? We don’t, thereby making ourselves the best of all possible targets: One that doesn’t fight back. We work in our labs and clinics believing, wrongly, that the average person understands and appreciates the long hours and sacrifices we make to learn about neural systems, work that, as we have mentioned, is so essential to diagnosing, treating, and curing disease.

When we and our colleagues became targets of extremism against animal research, we were amazed that the public—and, in fact, many of our colleagues outside of our state—were not aware of what was happening. We wrote a book, “The Animal Research War” (New York: Palgrave Macmillan, 2008), to raise public awareness of the benefits of research. We realized that young people, seeing what is happening to their mentors and observing the lack of public support, are making alternative career choices. Some of our colleagues quit productive careers out of concern for the well-being of their families.

The public buys medicines from drug companies, but because it doesn’t understand the role of research in developing those drugs or in bringing them through safety testing and to market, it is prone to accept activists’ arguments that the use of animals in drug development can be replaced by cell cultures or computers. The public seldom realizes that researchers actually developed these techniques and currently use them. But computers model only data, and cell cultures measure only part of the response of a living organism to a drug. Even if a drug were to come directly from cell culture, laws in the United States and in most countries require that drugs first be tested in animals before they can be used by humans. Most people don’t understand the federal regulation of research; when they are told about that level of oversight, many become supportive of our efforts.

What can you do? Talk with nonscientists about what you do and show them your lab and animal facilities; they need to know about the benefits of the research and that your research is both humane and closely regulated. Offer to visit schools and have that same conversation with the students. Invite field trips to your institution and suggest age-appropriate projects that engage students in research.

We know that those activities may not help researchers get tenure or research funding. This means that if the “you” we are addressing is an administrator, consider giving incentives to your staff to encourage them to take part in an educational mission to guarantee the survival of animal-based health research.

Neuroscience and other basic science-related organizations have been working through their animal research committees to raise members’ awareness of these threats and help them and institutions understand and respond to the threats.

However, some major medical organizations have not recognized the seriousness of these issues and have not warned their members or patients. Much more needs to be done. This will not be an inexpensive task. Hundreds of millions of dollars are donated to animal rights organizations each year. A recent, single television advertisement brought in U.S.$10 million.

The World Federation of Neurology needs to recognize the severity of the issue and develop programs to encourage medical organizations to join the efforts to bring the dangers of the animal rights terrorist to the attention of their patients and the public.

It was with a strong sense of déjà vu that I read this very stirring article by Dr. Conn and Dr. Parker. Researchers and doctors in the United Kingdom have endured similar abuse from the so-called animal liberationists for many years.

My main concern would be that despite setbacks and abuses, the scientific community must continue to work hard to improve openness about what we do and why we do it. We must continue to try to engage in a dialogue with those opposed to the use of animals in research.

However, this is only possible with those who agree to debate in a peaceful manner, and not with those who force their views upon others through violence and fear. The fear instilled in researchers by the illegal intimidatory actions is a long-term threat to neurological research—activists’ efforts to increase the legal rights of animals. They hope eventually to grant animals’ personhood. If that were to occur, the likely interested party could bring legal action against researchers and institutions engaged in animal research. The increase in legal threats has come in large part through the rapid expansion of courses in animal law at the major law schools in the United States.

The U.S.-based Society for Neuroscience has increased its efforts to educate the public about the importance of animal-based health research. We have increased our educational efforts both within the United States and internationally to encourage others to join our efforts to educate the public about the benefits of animal research and that your research is both humane and closely regulated.

By Roger L. Lemon, Ph.D.

BY ROGER L. LEMON, PH.D.

Although the animal rights terminology may have changed over the years, there is an even greater long-term threat to neurological research—activists’ efforts to increase the legal rights of animals. They hope to eventually grant animals legal rights and, as such, may soon be challenged in the courts.

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Study Sheds Light on Features of Brain AVM Rupture

BY DOUG BRUNK
Elsevier Global Medical News

SAN DIEGO — Disabling neurological deficits caused by ruptured brain arteriovenous malformations seem to occur more frequently in patients with exclusive deep venous drainage and in those that bled into the brain parenchyma, results from a single-center study demonstrate.

“Hemorrhage is the most dangerous complication in the natural history of untreated arteriovenous malformations [AVMs] of the brain,” Dr. Christian Stapf said at the International Stroke Conference. “We expected that AVM bleeding in eloquent locations would cause more functional damage to the brain, but we were unable to prove that in our study.”

In an effort to determine the demographic, clinical, and morphological AVM characteristics associated with disabling neurological outcomes after acute AVM rupture, Dr. Stapf, of Columbia University, New York, U.S.A., and his colleagues studied 80 patients with an AVM who presented with intracranial hemorrhage at Hôpital Lariboisière in Paris, France, from 2003 to 2008. The mean age of the patients was 40 years, 45% were women, and their median Rankin score was 2, with a range of 1-4.

Dr. Stapf reported that of the 80 patients, 54 (68%) had intracerebral bleeding, 17 (21%) had a subarachnoid component and 29 (36%) had an intraventricular hemorrhage component, including overlapping bleeding locations in 20 cases. The mean AVM size was 20 mm, 67% were located in the lobar region of the brain, and 52% had deep venous drainage pattern.

Retrospective studies cannot explain how bleeding may change the AVM’s angiographic configuration. Dr. Buis

There is some clinical plausibility that the fiber disruption when you bleed into the brain may cause more harm than bleeding into already existing external spaces, meaning the ventricles or the subarachnoid space,” Dr. Stapf said at the conference, which was sponsored by the American Heart Association. Dr. Stapf had no conflicts of interest in regard to the study, which was funded by the American Medical Association.

When the researchers compared the 48 patients with a median Rankin score of 2 or less (group 1) with the 32 patients with a median Rankin score of greater than 2 (group 2), the only statistically significant differences between them were parenchymatous location (88% in group 1 vs. 54% in group 2) and a “deep only” venous drainage pattern (44% in group 1 vs. 21% in group 2).

“As a clinician, what should the consequence of this finding be?” Dr. Stapf asked. “If AVMs with exclusive deep venous drainage have higher spontaneous bleeding risk and unfavorable functional outcome after hemorrhage, should this be the target for preventive intervention before bleeding occurs?”

In an interview, Dr. Osvaldo Fustini, a professor at the University of Buenos Aires, noted that the “if the treatment risks outweigh the benefits, treated patients with unruptured AVMs are more likely to end up more severely handicapped” than if their unruptured AVM was left untreated.

Some evidence suggests that invasive treatment strategies are significantly associated with a more than threefold increased risk of AVM hemorrhage, and an increased risk of clinical impairment, said Dr. Fustini. “A Randomized Trial of Unruptured Brain Arteriovenous Malformations (ARUBA), led by researchers at Columbia University, and supported by the U.S. National Institutes of Health and the National Institutes of Neurological Disorders and Stroke in Bethesda, Md., will shed light on further understanding of this complication. The trial is currently enrolling international sites to participate (www.arubastudy.org).”

In an interview, Dr. Dennis R. Buis of VU University Medical Center, in Amsterdam, the Netherlands, said the ARUBA trial will increase the understanding of risk factors for bleeding from AVMs that have not previously bled because studies with a retrospective design, such as the current study, cannot account for how “bleeding may change the angiographic configuration of the AVM due to kinking of its vessels or compression on the AVM.”

Neither Dr. Fustini nor Dr. Buis was involved in the study.
BEFORE THE RESEARCH IS PUBLISHED...

BEFORE THE DRUG IS APPROVED...

BEFORE THE GUIDELINE IS ISSUED...

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Clinical Neurology News

We Write Medicine’s First Draft

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Malawi’s Radiological Advances Take Root

Before Westerners settled in central sub-Saharan Africa, medical subspecialties, especially those that were technology-based such as radiology, were virtually nonexistent. The resource-poor Malawi serves as an illustration of the emergence of radiology in this region. There are 13 million people in Malawi, of which 87% live in rural areas. It is ranked in the bottom 10 countries on the United Nations Development Programme per capita income scale. Malawi, HIV, and respiratory and diarrheal diseases comprise the bulk of the disease burden, so preventive medicine is a health priority.

The development of radiology in Malawi followed the general trend of the growth of medical institutions in the country. The overall growth of medical services in Malawi falls into three periods: preindependence (pre-1964), postindependence (post-1964), and recent (the last 20 years). During the colonial era, medical services (and radiology) were concentrated in the cities of Blantyre, Zomba, and Lilongwe. Since British rule, the Queen Elizabeth Central Hospital (QECH) in Blantyre has been the largest medical facility in the country.

In the preindependence period, radiology services consisted of plain radiography and limited use of oil-based contrast agents. General physicians interpreted the studies without assistance from specialists. QECH trained para-medical staff, including nurses and radiography assistants. There was no medical or radiology school. There were specialists in fields such as pediatrics, but not in radiology. Apart from chest and skeletal films, the department performed selected contrast examinations, chief among them being myelography, cholecystography, hysterosalpingography, barium meal, and intravenous urography. Zomba and Lilongwe had similar radiology set-ups.

Later independence from Britain in 1964, health services were expanded and better equipped hospitals were built in most of the country’s districts. Each of these hospitals had an x-ray room and manual film processing equipment, and the radiology department would be operated by a radiologic technologist. But the interpretation of the films remained in the hands of the general physician. The most common examinations were chest films for coughs and limb x-rays for trauma. In the 1970s, QECH, Zomba, and Lilongwe hospitals were further improved to include ultrasound and angiography (at Lilongwe). QECH had a tomography x-ray unit. In the late 1970s, the Lilongwe School of Health Sciences was established for training paramedical personnel, including radiologic technologists. It remains the country’s only training institution for radiographers. It has produced over 100 radiographers, and the training has improved from basic training in radiography to the inclusion of ultrasound and CT skills.

Malawi has always been short of radiologists—only one or two have ever worked in the country at a time. Lilongwe Central Hospital had one radiologist from the late 1970s to the mid-1980s. From 2003 to 2004, the hospital was staffed by a German volunteer radiologist. Presently, there is one radiologist at QECH and two in training in South Africa and Kenya. There has been a single radiologist at QECH since 1996. It remains a standard practice of clinicians with varying levels of expertise to request and read radiologic examinations.

Malawi acquired its first— and only—MRI machine in 2008. It is also used for patients from neighboring states.

Ultrasound examinations also are performed by clinicians. Since 1998, radiology in Malawi has experienced accelerated growth. QECH, Zomba, and Lilongwe hospitals acquired improved x-ray units and automatic film processors. QECH acquired a Philips single-slice CT scanner in 1998. Around that time, the private hospitals of Mwawi and Blantyre Advenist Hospitals also acquired CT scanners. These scanners altered the practice of medicine in the country. Their impact was particularly noticeable because they reduced referrals to South Africa for neurological diseases and spared up diagnosis of intracranial lesions, especially traumatic bleeds.

In 2008, Malawi opened its first MRI service, courtesy of the generuous efforts of the Malawi Government, GE Healthcare, Michigan State University in the United States, and the U.S. National Institutes of Health. The service uses a 0.35T GE Sigma Ovation scanner with the latest software technology. This is the country’s only MRI scanner, and it serves patients from neighboring countries. It will provide imaging in neuroAIDS, skeletal trauma, orthopedics, and better imaging for children with cerebral malaria. It will also help in early detection and diagnosis of disease so patients need not leave the country.

Neurological Pearl: Parsing Essential Tremor

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essential tremor is one of the most common neurological disorders. To neurologists, the word “essential” may seem cryptic, but to patients, it is irksome, be-cause it seems to suggest the disorder is in some way unknown. In this sense, the use of “essential” to describe the tremor embodied the notion of a constitutional property that it was not under the control of the body that they were considered an intrinsic part of the individual make up. Hence, the word “essential” seemed appropriate.

Although “essential” and “idiopathic” are conceptual-ly similar, essential—which could be equated with familial or constitutional—diseases contrasted with idio-pathic, which meant that the underlying cause was unknown. In this sense, the use of “essential” to describe the tremor embodied the notion of a constitutional property of an individual. The difference is subtle, but it pro-vides an important clue as to how this emerging tremor disorder was viewed. Although the term has evolved in meaning over the last century, it has stuck, much to the chagrin of many confused E.T. patients.

Dr. Louis is at the Taub Institute for Research on Alzheimer’s Disease and the Aging Brain at Columbia University, New York, U.S.A.

Dr. E. Brusselle, Dr. C.G. Goetz, Dr. P. Knack, Dr. P. Kaufmann, and Dr. F. Mazzoni contributed to this and an earlier article on this topic (Neurology 2008;71:836-9).
Daniel Carleton Gajdusek (1923-2008)

BY LEV G. GOLDFARB, M.D.

Daniel Carleton Gajdusek, who died on Dec. 12, 2008, at age 85, was an active contributor to the fields of neurovirology and neurogenetics. He received his highest recognition in 1976, when he won the Nobel Prize for Medicine for the discovery of a new class of infectious agents, later named prions. His work outlined a group of neurodegenerative disorders known as spongiform encephalopathies, which include kuru, Creutzfeldt-Jakob disease, familial fatal insomnia, and several other disorders shown to be transmissible.

Dr. Gajdusek was born in 1923, in Yonkers, New York, U.S.A., into a family of immigrants from Slovakia and Hungary. He attended the University of Rochester in New York, then Harvard Medical School in Boston, Mass., U.S.A., and spent several postdoctoral years first at Harvard and later at the California Institute of Technology in Pasadena, U.S.A., where he worked with Nobel laureates Linus Pauling and Max Delbrück.

During his military service at the Walter Reed Army Medical School in Washington D.C., U.S.A., he was sent to post-war Germany on a mission to save orphand children. He later travelled to Iran, Afghanistan, and Turkey and subse- quently visited the Philippines, Indonesia, and Peru to collect data on epidemics of rabies, plague, and arbovirus infections. On these trips, he became increasingly fas- cinated with rare diseases in little-known, remote world populations.

He had trained as a pediatrician and neurologist at Harvard and started a program on child growth and development and disease patterns in remote cul- tures, which he later continued at the U.S. National Institutes of Health (NIH) in Bethesda, Maryland. In 1955, Dr. Gajdusek worked tempor- arily at the Burnet Institute in Melbourne, Australia, where the research fo- cus is infectious diseases. While he was there, he travelled to the tropical Australian tribes and subsequently went to New Guinea, where he discovered a previously un- known deadly disease that locals called kuru. He set up systematic studies of disease, built a hospital for kuru patients, and spent months in the field docu- menting each of several thousand cases of the disease and performing autopsies.

Kuru had emerged in the early 1950s in a small indigenous population of the New Guinean Eastern Highlands and reached epidemic proportions in the mid-1950s. It is a subacute neurodegenerative disease presenting with limb ataxia, dysarthria, and shivering tremor. Speech deteriorates as the disease ad- vances. Dystonic movements and my- oclonus-type jerks are present in the trunk, limbs, neck, and jaw. Dementia may not be evident until later stages of illness. The disease progresses to com- plete motor and mental incapacity and death within 6 to 24 months. Kuru neu- ropathology is represented by severe neuronal loss, spongiform change, as- trocytic proliferation and astrogliosis, and microglial proliferation, highly characteristic (“kuru-type”) amy- loid plaques, and prion protein deposits in the cerebral cortex and cerebellum. Astrocytic proliferation and astroglia are widespread and intense, but no in- flammatory changes are observed. The disease was prevalent in women and children, but rare in adult males.

During a 1959 visit to Dr. Gajdusek’s laboratory at the NIH, veterinary neu- ropathologist James R.M. Innes noted similarities between neuropathology findings in kuru and scrapie of sheep, a slow viral infection in which susceptibil- ity is genetically determined. Another veterinarian neuropathologist, William J. Hadlow, also suggested in a letter to the Lancet a possible relationship between kuru and scrapie and recommended that the transmissibility of kuru be tested in laboratory primates. These suggestions stimulated the ongoing attempts to find a microbial etiologic agent.

In 1968, Dr. Gajdusek, J. Gibbs, and M. Alpers reported the transmission of kuru to chimpanzees (Nature 1966:209: 794-6). Follow-up studies showed kuru agent is uniquely resistant to physical and chemical treatments that would other- wise destroy “conventional” viruses. The higher prevalence in women and children, was explained by their preferential participa- tion in cannibalistic funeral rituals, dur- ing which the tissue of kuru patients was consumed or rubbed into children’s skin.

The fact that a neurodegenerative dis- ease such as kuru was caused by an in- fection agent prompted the analysis of several other progressive neurogenera- tive disorders. The interest first focused on Creutzfeldt-Jakob disease (CJD). In 1968, Dr. Gajdusek and his co-investigators re- ported that an infectious agent similar to kuru was transmitted to chimpanzees from CJD brains after a 12-month incuba- tion (Science 1968:161:388-9). The find- ings that purely neurodegenerative dis- ease had an infectious origin and the subsequently described cases, were com- pletely new and unexpected. The latest member of this group, vari- ant Creutzfeldt-Jakob disease that is linked to the transmission of bovine spongiform encephalopathy (mad cow disease) to hu- mans, shows features similar to kuru.

Several unrelated neurological disor- ders have been successfully investigated at Dr. Gajdusek’s NIH laboratory: tropical spastic paraparesis, amyotrophic lateral sclerosis/parkinsonian dementia complex of Guam, characterization of spino- cerebellar ataxia genes, and the discovery of the amyloid precursor protein gene play- ing a critical role in Alzheimer’s disease.

Dr. Gajdusek’s interests spanned clas- sic literature, music, the fine arts, histo- ry, ethnography, and human develop- ment. He was a warm and kind man. He gave his staff and students complete free- dom and encouragement. Over the years, he took 48 orphaned teenagers from the Southern Pacific region back to the United States. He adopt- ed them and educated them; some sub- sequently returned to their homeland to offer services at the highest levels of gov- ernment and society.

Having worked closely with Dr. Gaj- dusek for almost 30 years, I remember his exceptional qualities—the clarity, speed, and depth of his thinking, his at- tention to detail, and his ability to orga- nize and direct his thinking.

He made lengthy daily entries in his di- aries, using a typewriter he carried with him, or in later years, dictating into a tape recorder. The public has inherited his pub- lished books, more than 1,000 research pa- per, and the multivolume set of notes of a passionate and productive scientist, thinker, and generous human being.

Dr. GOLDFARB is the head of the Clinical Neurogenetics Unit at the National Institute of Neurological Disorders at the NIH in Bethesda, Md., U.S.A.

Charting the Course of the World Neurology Foundation

T
e World Neurology Foundation was incorporated in 1999 as an independent nonprofit organization to serve as a charitable arm of the World Federation of Neurology in the United States of America. (WFN is a registered charity in the United Kingdom.)

Dr. James Toole, a former WFN President, conceived of the idea to allow for tax-deductible donations to be made in the United States to help fund the WFN’s international projects. Canadian donors ben- efitted because of the tax treaties between the two countries.

Under the presidencies of Dr. Toole and Dr. Antonio Culebras, the foundation secured funding for four en- dowed lectureships to be presented at the World Con- gress of Neurology in Bangkok honoring Dr. Bhim Singh- hal (India), Dr. Eddy Bharucha and his wife, Dr. Piloo Bharucha (India), Dr. Melvin Yahr (U.S.), and Dr. Richard Masland and his wife, Dr. Mary Masland (U.S.). This sup- port will continue for the biennial congresses. Since 2005, the foundation has accepted responsibilities in two new areas. The first initia- tive is based on data in the 2004 At-лас of Country Resources for Neu- rological Disorders by the WHO and WFN, which showed a global need for better neurological ser- vices, especially in Africa.

On assuming the WFN presi- dency, Dr. Johan A. Aarli recog- nized those needs and committed resources to improving the ser- vices. He included the federation in the coalition of or- ganizations that have worked on the Africa Project. As president of the foundation and together with an able board of directors, I have supported this new initiative.

African neurologists requested basic tools for the neu- rological exam. In response, the foundation conceived of the Neurologist’s Tool Kit, consisting of a 128 Hz tuning fork, a stethoscope, a collapsible Queen Square reflex hammer, a pen light, an NIH stroke scale, and a Snellen chart, stored in a portable case. Last year, 100 kits were distributed at meetings in Senegal and Cameroon and to Zambian trainees. The foundation is seeking funding for kits for neurologists in Uganda, Ethiopia, and Nigeria.

The second initiative grew out of the success of the American Academy of Neurology’s Palatucci Advocacy Leadership Forum, which trains three internation- al advocates a year, each of whom devises a project to improve neurological services in their home countries. The foundation has helped these individuals formulate the projects and find funding. Members of the Pakistan International Neuroscience Society and of the Associ- ation of Indian Neurologists in America have con- tributed to active programs in New Guinea and Western Papua. For more information about the foundation or to make a donation, visit www.worldneurology.org.
RESOURCE UPDATE


The editor and contributors to this new edition have approached the difficult task of providing an overview of the complex disease myasthenia gravis. Many chapters are written by international experts who have made pivotal contributions to the field, and as such, the book provides an integrative view of this disease and related disorders.

Most of the illustrations are of excellent quality, especially those representing neurophysiologic and molecular mechanisms. Some of the color plates could just as easily have been shown as black and white figures. Immunologic chapters are excellent, especially those referring to T-cell education in thymomas.

Given the substantial progress in understanding the pathophysiology of myasthenia gravis, a more detailed discussion or perhaps a separate chapter on specific clinical aspects and therapy of MuSK-myasthenia, which poses a number of therapeutic challenges, would have been desirable. The same pertains to management of malignant thymomas.

What improvements could be made? More discussion about the systematic introduction to clinical monitoring of disease via scoring (Reiter-Toyka score) and escalating therapy algorithms in severe cases would have been helpful. As with multiple sclerosis, baseline therapeutic and individualized trials with combination therapies or monoclonal antibodies can be envisioned for MG.

Treatment of MG in the intensive care unit has become rare: less than 2% of patients have a disease crisis in their lifetime. The threshold of a forced vital capacity less than 1 L only pertains to extremely slow deterioration; especially in rapid progression, vital risks may start when FVC reaches 1.5 L. Soft warning signs such as weakness of neck extensor muscles, and fluctuating swallowing and respiration should be mentioned.

The application of BiPAP to bridge respiratory symptoms has been significant progress in ventilatory management. When plasma exchange procedures have been considered in MG crisis, immunoadsorption via triethylamine columns should be mentioned as well.

In accord with the personal approach noted by Dr. Kaminski, I will also make some personal comments. The title picture may be slightly misleading, since associated glycogenolysis rather points to nonremitting medical conditions or Kennedy syndrome. Having studied with the late Dr. Hertel Mertens, I would like to see an acknowledgement of his introduction of azathioprine mentioned with the reference to the 1981 paper in the Archives of the N.Y. Academy of Sciences. The disorder is often associated with Mertens and Isaacs, who described neuromyotonia almost simultaneously.

FROM THE JOURNAL OF THE NEUROLOGICAL SCIENCES

AMM and Diabetic Polyneuropathy

BY ALEX TSELSIS, M.D., PH.D.

Dr. Tselis is an associate professor of neurology at Wayne State University, in Detroit, Michigan, U.S.A. He is the book review editor for the Journal of the Neurological Sciences.

Many systemic diseases are known to affect peripheral nerves diffusely. The most common is diabetes mellitus, but there are many other causes of different classes of disease. Such examples would include metabolic problems such as uremia, toxic neuropathy from alcohol or medications, nutritional conditions due to thiamine and vitamin B12 deficiency, and infections such as HIV disease.

In each case, although the association between the systemic disease and polyneuropathy is well known, there is a dearth of detailed data to characterize the pathogenesis. Clearly, these are sick nerves; their fragility leaves them vulnerable to the effects of pressure, and these patients are commonly affected by carpal tunnel syndrome, ulnar and peroneal neuropathy, or other pressure-related neuropathies.

A fruitful approach to investigating this problem would be to identify at-risk patients with mild asymmetric disease and study them in detail. In that way, a cohort of patients with diabetic polyneuropathy (DPN) could be evaluated for a pressure neuropathy to which they are especially susceptible. Such a cohort can be divided into those with and those without a pressure polyneuropathy, and the two groups could be compared.

The study, reported in a paper by Dr. Eleftheriou Stamboulis and colleagues, illustrates one of the first steps in this type of strategy (J. Neurol. Sci. 2009;278:41–3). Dr. Stamboulis, who has been investigating diabetic neuropathy for 30 years, and his team recruited 100 diabetic patients who were participating in a clinical trial and had electrodiagnostic diagnosis of an asymmetric median mononeuropathy (AMM) of the non-dominant arm. They were classified into four grades of DPN and according to the presence or absence of AMM.

The analysis of these patients was instructive. The researchers found that AMM was present in 28% of these patients overall. It was significantly more common in those with more severe DPN, but it did not depend on the duration or nature (type 1 or type 2) of diabetes. However, there were some unexpected results. Some patients with mild DPN had AMM (18% of those with stage 0 DPN), and some with severe DPN were free of AMM (35% of those with stage 3).

These results, if robust, have pathogenetic implications. Thus, although DPN is likely a result of relative tissue ischemia, proportional to the total exposure to hyperglycemia, other factors contribute to the fragility, given that some patients had unexpected AMM and others did not. This implies that other factors contribute to the risk and severity of disease. One possibility is that of mitochondrial involvement (as is likely the case for the neurotoxic effects of certain antiretroviral drugs).

These results will need replication, but they clearly suggest that multiple factors affect the diabetic nerve. Further research will be needed to characterize these factors and allow the design of specifically targeted therapy.
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