

WORLD NEUROLOGY

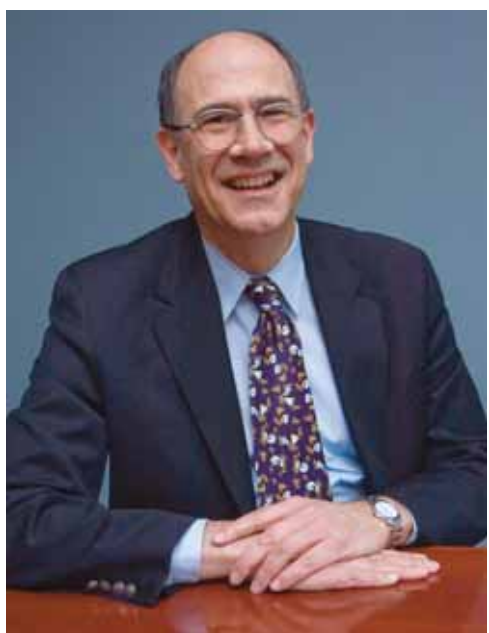
THE OFFICIAL NEWSLETTER OF THE WORLD FEDERATION OF NEUROLOGY

Welcome to the New WORLD NEUROLOGY!

Starting with this issue, WORLD NEUROLOGY will have new content and a new look. As the newsletter of the World Federation of Neurology (WFN), WORLD NEUROLOGY is the only publication going to all neurologists in the world.

The newsletter will continue to feature news from the WFN. However, the new version will have other content of interest to neurologists, including some neurology news, and will be published by Elsevier. We are all grateful to Prof. Jagjit Chopra, who has done a spectacular job of editing WORLD NEUROLOGY for the past decade.

So who am I? I am a neurologist at the National Institute of Neurological Disorders and Stroke, at the National Institutes of Health in Bethesda, Md., where I do clinical research. My interests are clinical neurophysiology and movement disorders. I recently completed an 8-year term as editor in chief of Clinical



New WORLD NEUROLOGY editor in chief Dr. Mark Hallett plans to revamp the newsletter.

Neurophysiology. Presently, I served as an associate editor for the journal *Brain* as well as for a new journal, *Brain Stimulation*.

Although I hope to assemble an editorial board to help me in the coming months, as the new medical editor, I will be looking for input from everyone. So please send in local news, or news of interesting medical and

scientific advances. What interests you may well interest others.

In this inaugural issue, you will find some new features. One of these is "Profiles in Neurology." In it, I hope to spotlight different medical care practices from around the world, giving one neurologist his or her chance at "fifteen minutes of fame," as the expression goes. I am grateful to Dr. Rawiphan Witoonpanich of Mahidol University, Bangkok (Thailand), for starring in the first of these.

We're also interested in news from the National Societies that make up the WFN. For example, the German Society just celebrated its 100th anniversary. In this issue, Dr. Günther Deuschl and his colleagues present a brief history of the society. We will highlight other national societies in upcoming issues.

This issue also includes the first "Neurologic Pearl." This column will give an in-depth

view of a brief, little-known topic of interest in clinical neurology. The first installment is on the Ramsay Hunt syndromes. Can you describe all four without cheating?

We also will publish cases. Some of these may be disorders that are seen only in certain parts of the world or that are more common in particular places. Others may be rare but important to keep in mind. So send in your suggestions!

We expect that cases, as well as the other articles, will be illustrated with photographs or other images—an interesting MRI, for example. And with all articles, we will want to have a photograph of you, too.

WORLD NEUROLOGY should be a useful and educational newsletter. Send your feedback and ideas to worldneurology@elsevier.com. Let's keep each other informed and knowledgeable, so we can bring good neurologic practice to our patients and have some fun along the way.

*Mark Hallett
Editor in Chief
World Neurology*

INSIDE

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Oral Agent for Form of Multiple Sclerosis Shows Promise

BY DOUG BRUNK
Elsevier Global Medical News

Patients with relapsing remitting multiple sclerosis who took daily oral laquinimod over a 36-week period achieved a 40% reduction in gadolinium-enhancing lesions compared with patients who took placebo, according to phase II trial results.

"Overall, the efficacy and safety profile emerging from this and from a previous phase II clinical trial, in combination with the oral route of administration, make laquinimod a promising therapeutic opportunity for patients with relapse remitting multiple sclerosis," reported the researchers, led by Dr. Giancarlo

Comi of the institute of experimental neurology at the Vita-Salute San Raffaele University, Milan.

Developed by Teva Pharmaceutical Industries Ltd., laquinimod is an oral immunomodulatory drug that is believed to exert anti-inflammatory activity in the relapsing remitting form of MS by the Th1-Th2 shift.

To date, approved drugs for MS are all injectable and include glatiramer acetate, interferon- β , natalizumab, and mitoxantrone.

Prof. Alastair Compston, head of the department of Clinical Neurosciences at the University of Cambridge (England), who was unaffiliated with the study, told WORLD NEUROLOGY in an in-

terview, "This is a first step in the direction of identifying a therapy for use early in the course of multiple sclerosis which is convenient, safe, and effective. Whether oral laquinimod meets these characteristics remains to be seen."

In a study conducted at 51 centers in nine countries, Dr. Comi and his associates randomized 102 patients with relapsing remitting multiple sclerosis to placebo, 98 to laquinimod 0.3 mg daily, and 106 to laquinimod 0.6 mg daily for 36 weeks (*Lancet* 2008;37:2085-92).

Patients were aged 18-50 years and underwent brain MRI and clinical assessments 4 weeks before the study started, at baseline, and then every 4 weeks for 9 months. The researchers measured the number of gadolinium-enhancing (GdE) lesions at weeks 12, 16, 20, 24, 28, 32, and 36.

The primary end point of the

study was the mean cumulative number of GdE lesions per scan in the last four scans.

The researchers reported that patients in the laquinimod 0.6 mg daily group demonstrated a 40% reduction in the mean number of GdE lesions over the last four scans, compared with those in the placebo group (a mean of 2.6 vs 4.2, respectively). Patients in the laquinimod 0.3 mg daily group had a mean number of GdE lesions similar to the placebo group (3.9). The latter finding

surprised the researchers, considering that the 0.3-mg dose demonstrated efficacy, compared with placebo, in a previous study (*Neurology* 2005;64:987-91).

One possible explanation could be that the previous study used a triple dose of gadolinium, "which increases the harvest of active multiple sclerosis lesions by 60%, and, as a consequence, increases the statistical power of MRI-monitored trials," the researchers

See **Multiple Sclerosis** • page 2



DR. GIANCARLO COMI

New Editor in Chief Mark Hallett Takes Over

The process of reformatting WORLD NEUROLOGY is over, and the inaugural issue, which you now hold in your hand, is the new link that will connect neurologists all over the world.

Until the Chinese Society of Neurology joined the World Federation of Neurology last year, we served a membership of about 25,000.

That number is now considerably higher, and I am proud to say that the

world. WORLD NEUROLOGY will serve as a support mechanism to a sense of solidarity, of knowing what is going on in neurology everywhere.

Therefore, WORLD NEUROLOGY will provide information about current developments in modern neurology, with dissemination of therapeutic advances and discussions on hot topics in the neurologic community.

We are delighted to have Mark Hallett as our new editor in chief. Because he was the chair of the finance committee until he took over his new position, Mark Hallett knows the World Federation of Neurology very well. Dr. Hallett did his neurology training at Massachusetts General Hospital, Boston, and has a broad background in neurology and clinical neurophysiology. He currently serves as the chief of the Medical Neurology Branch of the U.S. National Institutes of Health, and chief of its Human Motor Control Section.

Dr. Hallett has also been the president of the Movement Disorder Society and vice president of the American Academy of Neurology.

In conclusion, it is a real honor for the World Federation of Neurology to have Mark Hallett as the editor in chief of our international newsletter, especially at this crucial time when our organization is expanding and coming to new crossroads.

Johan A. Aarli
President

The World Federation of Neurology

**THE NEWSLETTER'S
FUNCTION IS TO CONVEY
INFORMATION FROM AND TO
WFN'S CENTRAL
ADMINISTRATION.**

World Federation of Neurology has become a global organization with members in 102 countries, in which more than 50 different languages are spoken.

The function of the newsletter, WORLD NEUROLOGY, is to conduct news to and from our many global members. The redesigned WORLD NEUROLOGY will help to distribute information from and to the central administration, with its various research groups and committees, as well as inform the regional neurologic communities.

However, the newsletter also aims to be a medium through which the individual neurologist can stay in contact with colleagues around the

GdE Lesions Fell 40%

Multiple Sclerosis • from page 1

explained. "GdE lesions visible only on triple-dose scans are usually smaller and with a less-severe blood-brain barrier dysfunction than those enhanced after a standard dose."

The current trial used a standard dose of gadolinium.

In terms of safety, two adverse events potentially attributable to laquinimod occurred. One patient in the laquinimod 0.6 mg daily group had Budd-Chiari syndrome and was found to be heterozygous for the factor V Leiden mutation, "which is associated with up to 30% of cases of venous thrombosis," the researchers noted. "This case highlighted the possibility of an increased risk for thrombosis in patients with pre-existing thrombophilia exposed to laquinimod, which needs to be further explored."

One patient in the laquinimod 0.3 mg daily group had persistent elevation in liver enzymes during the early phase, but did not show clinical signs of liver injury.

In an accompanying editorial, Dr. B. Mark Keegan and Dr. Brian G. Weinschenker of the department of neurology at the Mayo Clinic, Rochester, Minn., noted that many patients would prefer an oral medication such as laquinimod and recommended that a head-to-head analysis of the drug with existing immunomodulatory medications be conducted (Lancet 2008;37:2059-60). They also called for careful investigations of safety, "because seri-

**THIS IS THE FIRST STEP
TOWARD DEVELOPING A
CONVENIENT, SAFE, AND EFFECTIVE
THERAPY FOR MS.**

ous adverse effects are commonly not evident until phase II studies are started (as with linaclumab) or until after approval (as with natalizumab)."

Teva Pharmaceuticals sponsored and funded the study. Dr. Comi disclosed receiving compensation from Teva and other pharmaceutical companies. ■

WFN Committee Has Named Candidates for Trustee Post

The following candidates are recommended by the Nominating Committee of the World Federation of Neurology for the Elected Trustee post, which is falling vacant in 2008 as Prof. Marianne de Visser, from the Netherlands, retires.

The nominees are recommended to the membership through their representatives on the Council of Delegates,

in accordance with the Federation's Memorandum and Articles of Association.

It is open to anyone to make additional nominations by securing the supporting signatures of five or more authorized delegates and submitting the name(s) of the individual(s) in question to the following address: Secretary-Treasurer General, c/o the London Secretariat Office, 12

Chandos St., London, W1G 9DR, United Kingdom.

Nominations must be received by September 22, 2008.

Recommended candidates:

- ▶ **Prof. Natan Bornstein** (Israel)
- ▶ **Dr. Antonio Culebras** (Spain/USA)
- ▶ **Dr. Gustavo Román** (Colombia/USA)
- ▶ **Dr. Stephen Sergay** (USA)



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WORLD NEUROLOGY

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THE PRESIDENT'S COLUMN

WFN and Regional Neurologic Groups Share a Close Link

The World Federation of Neurology (WFN) is one of the neurologist's member organizations, membership to which is obtained through his or her national neurologic association. However, most neurologists also are usually members of local, regional professional organizations. An Indian neurologist, for example, might be a member of the Indian Academy of Neurology, the Asian Oceanian Association of Neurology (AOAN), and the World Federation of Neurology. A Norwegian neurologist, on the other hand, might be a member of the Norwegian Neu-



BY JOHAN A. AARLI
PRESIDENT, WFN

rological Association, the European Federation of Neurological Sciences (EFNS), and the World Federation of Neurology.

The regional neurologic associations, including the American Academy of Neurology, the AOAN, the EFNS, the Latin American Neurological Region, the Pan African Association of Neurological Sciences, and the Pan Arab Union of Neurological Sciences, are strong and inde-

pendent organizations. They organize annual or biennial regional congresses. The presidents of the regional associations are ex-officio WFN regional directors

and have the possibility of closer contact with the corresponding regional World Health Organization offices in Brazzaville (Congo), Cairo (Egypt), Copenhagen, Manila, New Delhi, and Washington.

The World Federation of Neurology looks forward to closer contact with the regional neurologic organizations in part because of the coming geographical rotation of world congress venues.

The 2009 World Congress of Neurology, as you likely know, is set for Bangkok, in Thailand. The 2011 Congress will take place on the African continent. In 2013, the World Congress of Neurology is scheduled to be held somewhere in Europe, and in 2015, in Latin America.

There are two bids for the 2011 Congress. One possible venue is Cape Town, South Africa. The other is Marrakesh, Morocco.

'WE LOOK FORWARD TO CLOSER CONTACT WITH THE REGIONS AND TO FUTURE EXCHANGE PROGRAMS.'

Both are extremely fascinating and attractive cities. The ultimate decision of congress venue will be made at the Council of Delegates Meeting in New Delhi in October, 2008.

As I mentioned, the World Congress of Neurology in 2013

will take place in Europe. The European Federation of Neurological Sciences, which up to now has organized successful annual regional congresses, has decided that the World Congress that year will also serve as an EFNS congress; there will be no independent EFNS meeting in 2013. The venue for that joint meeting has not yet been chosen; the EFNS will have the strongest voice in that decision, and we will let our members know.

The ultimate goal is to increase the visibility of the global responsibilities of the World Federation of Neurology. We look forward to closer contact with the regions and to future exchange programs for neurologists from different parts of the world. ■

World Neurology Foundation Launches New Web Site

It is my pleasure as webmaster to announce the launch of the new Web site for the World Neurology Foundation. The Web site was released on April 27, 2008. For immediate access and to check out what's new, please visit www.worldneurology.org.

The World Neurology Foundation was incorporated in 1999 as a charitable arm of the World Federation of Neurology.

The new Web site will serve the organization internally by providing information about the mission, program, and meetings of the foundation to the organization's staff, its 12-member volunteer board of directors, and the six-member advisory council.

The new Web site will also provide an essential external marketing function by engaging others in the foundation's mission and projects, thus promoting financial support.

Users can expect the following highlights when accessing the new Web site:

- ▶ **A home page** that emphasizes the World Neurology Foundation's central mission—to serve as a catalyst for the promotion of neurological care and ed-

ucation in countries in need—and neurologic care purposes.

- ▶ **An About Us section** that contains content regarding mission, foundation overview, leadership, and staff profiles.

- ▶ **A 2008 Projects feature**, which describes the World Neurology Foundation's projects, projects' locations, overviews on project toolkits for Africa, Neuroshare, and 2008 affiliate membership.

- ▶ **Information about Partnership Programs**, including lectureships, continuing medical education, and certification training.

- ▶ **Opportunities for you to make donations** that will go towards helping support various foundation projects, including the capacity to donate for use in specific programs.

Congratulations to the World Neurology Foundation on this new Web site.

Please don't hesitate to direct any questions about the site to the World Neurology Foundation's executive director, Carrie Becker, who can be reached at: Cbecker@worldneurology.org.

John Johnson

World Neurology Foundation
Webmaster

European Board Examination In Neurology Is Scheduled To Commence in 2009

The European Union of Medical Specialists' Section of Neurology and the European Board of Neurology will establish and administer a European board examination in neurology, beginning in 2009. This certification exam is part of a broader European effort to improve training in neurology.

To further assure the quality of neurology training, the UEMS/EBN has formed a committee to visit neurology departments throughout Europe. Residents of the centers that are visited will receive a reduction in the examination fee.

The examination will consist of two parts. One will contain both written and multiple choice questions; the other will be an oral examination in which the candidates will answer structured questions about four specific cases.

Candidates can earn extra points by presenting either one of their own cases or results from scientific research done during their training.

Scientific panels from the European Neurological Society (ENS) and European Federation of Neurological Societies (EFNS) will collaborate to develop the exam. Scientific committees from the ENS and EFNS have teamed with the UEMS/EBN to form a joint European examination committee, which will have an advisory role to the UEMS/EBN.

The application for the examination is available from the UEMS/EBN Web site (www.uems-neuroboard.org/ebn).

At the time of the implementation of the examination, only those candidates from EU/EEA (European Economic Area) countries and who have either passed their national examination or have confirmation from their national society

that they are eligible can be admitted.

Because their practical skills will be confirmed by the candidates' national training society, the examination will be a multi-step process.

Although many European countries have their own certification system, and the UEMS/EBN examination will have no

SUCCESSFUL EBN EXAMINATION CANDIDATES WILL BE AWARDED THE TITLE OF FELLOW OF THE EUROPEAN BOARD OF NEUROLOGY.

legal power, it is hoped that countries without a system will adopt this examination as a standard.

The EBN examination is being developed as a measure of excellence, such as those already established by many other UEMS sections and boards.

The successful candidates will be awarded the title of Fellow of the European Board of Neurology.

In cooperation with the big European neurologic societies (EFNS and ENS), the UEMS/EBN will launch this important step in training in neurology.

For more information, visit the UEMS' Web site at www.uems-neuroboard.org, or e-mail the VMA office in Vienna: manuel.hoetendorfer@medacad.org. ■

By Prof. Wolfgang Grisold, who is chair of the education committee at the EFNS and president of the EBN and Prof. Svein Mellgren, who is the EBN chair.

Please Visit the Updated 2009 World Congress of Neurology Web Site

The Web site for the official 19th World Congress of Neurology, to be hosted by the World Federation of Neurology, October 24-30, 2009, in Bangkok, Thailand, has now been updated. Information is now available regarding the preliminary program and



general information about the Congress. The Web site will be updated continuously as the Congress approaches, so check back often as more is added. Visit the Web site today, at www.wcn2009bangkok.com, to see what's new.

Meeting Report: WFN Junior Traveling Fellowship 2008

From Shanghai to Chicago, the WFN supports its members around the globe.

Editor's Note:

This year, Dr. Xiang-Jun Chen was one of several young neurologists to receive a World Federation of Neurology Traveling Fellowship, which he used to attend the 2008 annual meeting of the American Academy of Neurology in Chicago. At the request of WORLD NEUROLOGY, he has written a report, which appears below, on the experience and what he gained as a result of his award.

Last year, I completed my postdoctoral training in the field of neurogenetics at the University of Chicago and began my long-pursued career in academic neurology as a faculty member in the neurology department at Fudan University Huashan Hospital (Shanghai, China). To me, attending the American Academy of Neurology (AAN) annual meeting is always an excellent way to refresh and update my knowledge of clinical neurology. However, as a young neurologist now living in Shanghai, the expense of traveling to the United States to attend this meeting was intimidating. Fortunately, the generous gift of a Junior Traveling Fellowship

2008 from the World Federation of Neurology made it possible for me to attend.

The AAN annual meeting is among one of the world's largest neurology meetings. This year, the AAN celebrated its 60th anniversary meeting in Chicago from April 12 to 19, bringing together more than 10,000 neuroscience professionals from all over the world. As planned, I partook of all five intriguing plenary sessions featuring the latest neurologic research. Among the popular Integrated Neuroscience Sessions, the "Mitochondria in Diseases" session was most attractive to me because I have been working on a genetic neuropathy mouse model with a defect of mitochondrial transport. During this session, I met many of my neurologist peers involved in neuroscience research and exchanged scientific viewpoints with them in our fields of common interest. Through the introduction of Dr. Raymond Roos of the University of Chicago, I also had the opportunity to attend an ALS Association-sponsored Drug Company Working Group Meeting. It was from this small

group meeting, along with the "Anterior Horn" platform sessions, that I learned about up-to-date research and therapeutic progress in the MND/ALS field that will bring hope to my patients.

The Junior Traveling Fellowship helps young neurologists in low- and lower-middle income countries attend an international meeting, reflecting the spirit of the WFN mission. The professional knowledge gained from this memorable meeting will definitely improve my practice in the future. More importantly, the spirit of the WFN will be in my mind throughout my career in neurology.

Xiang-Jun Chen, M.D., Ph.D.
attending neurologist,
Huashan Hospital,
Fudan University, China



Pictured from right to left: Dr. Chen, WFN Secretary Susan Bilger, WFN Executive Director Keith Newton, and Jane Aberle, the development associate of the World Neurology Foundation.

WFN Traveling Fellowships Application Criteria

Each year, the World Federation of Neurology offers 10 Junior Traveling Fellowships for young neurologists from countries classified as low- or lower-middle income by the World Bank to attend an international meeting. The candidates for the awards must hold a post no higher than associate professor and must be under 42 years of age. Applicants must state the names and dates of the proposed meeting they wish to attend; send a re-

sumé, bibliography, and an estimate of expenses, to a maximum of £1,000; and enclose a letter of recommendation from the head of the department, plus an abstract if a paper or poster is to be presented. Awards are assessed by the Executive Committee of the WFN's Education Committee.

Nominations for the 2009 fellowships are not yet being considered. Check WORLD NEUROLOGY for updates on when applications will be accepted.

Vladimir Hachinski Is a Leader of Many Accomplishments

BASED ON AN INTERVIEW WITH
DR. VLADIMIR HACHINSKI

Since Dr. Vladimir Hachinski, professor of neurology and Distinguished University Professor at the University of Western Ontario, London, was elected first vice president of the WFN, he has done some impressive work for the WFN. But Dr. Hachinski has long been one of the more prestigious members in the field of neurology.

Born in Europe and raised in Latin America, Dr. Hachinski received his MD from the University of Toronto in 1960. After his residency there, he was a fellow at the National Hospital for Nervous Diseases at Queen Square, London (England), and the University of Copenhagen. After his initial return to the University of Toronto in the early 1970s, he joined the faculty at Western Ontario in 1980. With John Norris, he established the first acute stroke unit in Canada for the urgent care and systematic study of patients with "brain attack." He codesigned the Toronto Stroke Scale with John Norris, and discovered the role of the right insula of the brain in mediating cardiac complications of stroke, including sudden death.

Dr. Hachinski proposed the concept of "multi-infarct dementia" and later that of



VLADIMIR
HACHINSKI, M.D.

"vascular cognitive impairment" to encompass the broad spectrum of cognitive impairment caused by or associated with vascular factors. He developed the Hachinski Ischemic Score (2,150 citations) to identify the vascular component of cognitive impairment, and led, along with Gabrielle Leblanc, a group of experts in developing common standards for describing and studying cognitive disorders. He also helped develop a model to study the interaction between cerebrovascular disease and Alzheimer's.

Dr. Hachinski has also published 17 books and more than 600 scientific papers, book chapters, editorials, and other works. As editor in chief of Stroke, he initiated its translation into Spanish, Russian, Italian, Portuguese, Chinese, Japanese, and Korean, and a special edition for readers in India. He also started the Author Mentoring Program, through which he welcomes manuscripts from contributors in developing countries and, at an author's request, will assign a member of the editorial board. He arranged free subscriptions for readers in select countries.

In 2004, Dr. Hachinski led a working group that resulted in the establishment of World Stroke Day (Oct. 29) and the development of a world stroke agenda.

Dr. Hachinski received an ScD from the

University of London (England), and three doctor honoris causa from the universities of Salamanca, Buenos Aires, and Cordoba, respectively. He was the first recipient of the Trillium Award, which is given to the best clinical researcher in the province of Ontario, and he also received the international Mihara Award, given every 4 years to someone who has made major contributions to the field of stroke.

As Canada's delegate to the WFN and chairman of its steering committee for two terms, Dr. Hachinski was presented with a diploma whose citation reads in part, "Brought parity and fairness in the interrelationship of the WFN with regional and national groups."

As first vice president, he initiated the process that led to the decision to hold a world congress every 2 years in a differ-

ent part of the world—the premise being that such meetings have the greatest impact in the region in which they are held, and that we need to reach neurologists or potential neurologists who cannot travel internationally. The greater frequency of meetings also allows a platform for increased activities in the years between them.

Dr. Hachinski currently is fostering cooperation between the WFN and other international organizations, specifically a collaboration with the International Brain Research Organization (IBRO).

He is encouraging a survey of the membership, particularly of national delegates, to identify how the WFN can better serve its members, broaden the base of those participating in its activities, and involve neurologists in the organization. ■

Dr. Hachinski Named to Order of Canada

Dr. Vladimir Hachinski has recently been named to the Order of Canada, the country's highest honor, for his lifetime contributions in neurology. The award was announced just as WORLD NEUROLOGY went to press.

"When I learned that the government was trying to track me down, I was in China and a few unpleasant possibilities crossed my mind," joked

Dr. Hachinski. "When I was informed that I was to be named to the Order of Canada, I was surprised, and felt honored and humbled to join such a distinguished company of Canadians," he commented.

The Order of Canada recognizes a lifetime of outstanding achievement, dedication to the community, and service to the country.

Is Gene Therapy Feasible for Parkinson's Disease?

BY MICHAEL J. AMINOFF, M.D.

Pharmacologic therapy is the mainstay of the symptomatic treatment of Parkinson's disease; deep brain stimulation is reserved for those with complications. An alternative to both may be gene therapy.

Gene therapy works by inserting a normal gene into a nonspecific location within the genome in order to replace a non-functional gene or to establish a new function or role for the cell. Release of the gene product then acts on the affected or neighboring cells. The approach has advantages. Depleted neurotransmitters can be replaced in a more physiologic manner so that certain complications are less likely. Therapies can be delivered in a site-specific way, so that other side effects (like the mental side effects of dopaminergic drugs) are avoided. One treatment may provide sustained benefit. And unlike DBS, there are no device complications.

Gene therapies have been studied in animal models of parkinsonism for the last 20 years, but only in the last 5 years have clinical studies been undertaken in humans. The usual vector for gene delivery to the target cells is adeno-associated virus type 2 (AAV2). This virus is not associated with human disease, can infect

nondividing human cells, shows low immunogenicity, and is not associated with oncogenesis. Most humans have been infected naturally, but the mode of infection is unclear. The virus, when injected into the striatum, is taken up selectively by neurons.

Gene therapy by transfection of the subthalamic nucleus (STN) with AAV containing the gene for glutamic acid decarboxylase (GAD) leads to synthesis of the inhibitory neurotransmitter γ -aminobutyric acid. This reduces STN output and hence its inhibitory effect on the thalamus. The rationale is thus similar to treatment by DBS of the subthalamic nucleus, but the gene therapy avoids hardware-based complications, programming and maintenance.

In an open-label safety study, 12 patients with advanced PD received unilateral infusion of AAV-GAD into the subthalamic nucleus (four patients each in three dose cohorts). Patients were assessed clinically on and off medication (*Lancet* 2007;369:2097). There were significant improvements in motor Unified Parkinson Disease Rating Scale scores contralateral to surgery at 3 months. These persisted to

12 months; fluorodeoxyglucose-positron emission tomography (FDG-PET) showed reduction in thalamic metabolism on the treated side at 1 year, similar to after DBS. No adverse effects were related directly to gene therapy.



MICHAEL J. AMINOFF, M.D.

An alternative approach involves the gene for aromatic l-amino acid decarboxylase (AADC), which converts levodopa to dopamine. As PD advances, many patients require increasingly more levodopa to maintain benefit. In a phase I study, 10 patients with moderately advanced PD and response fluctuations received either a low or high dose of AAV-AADC into the

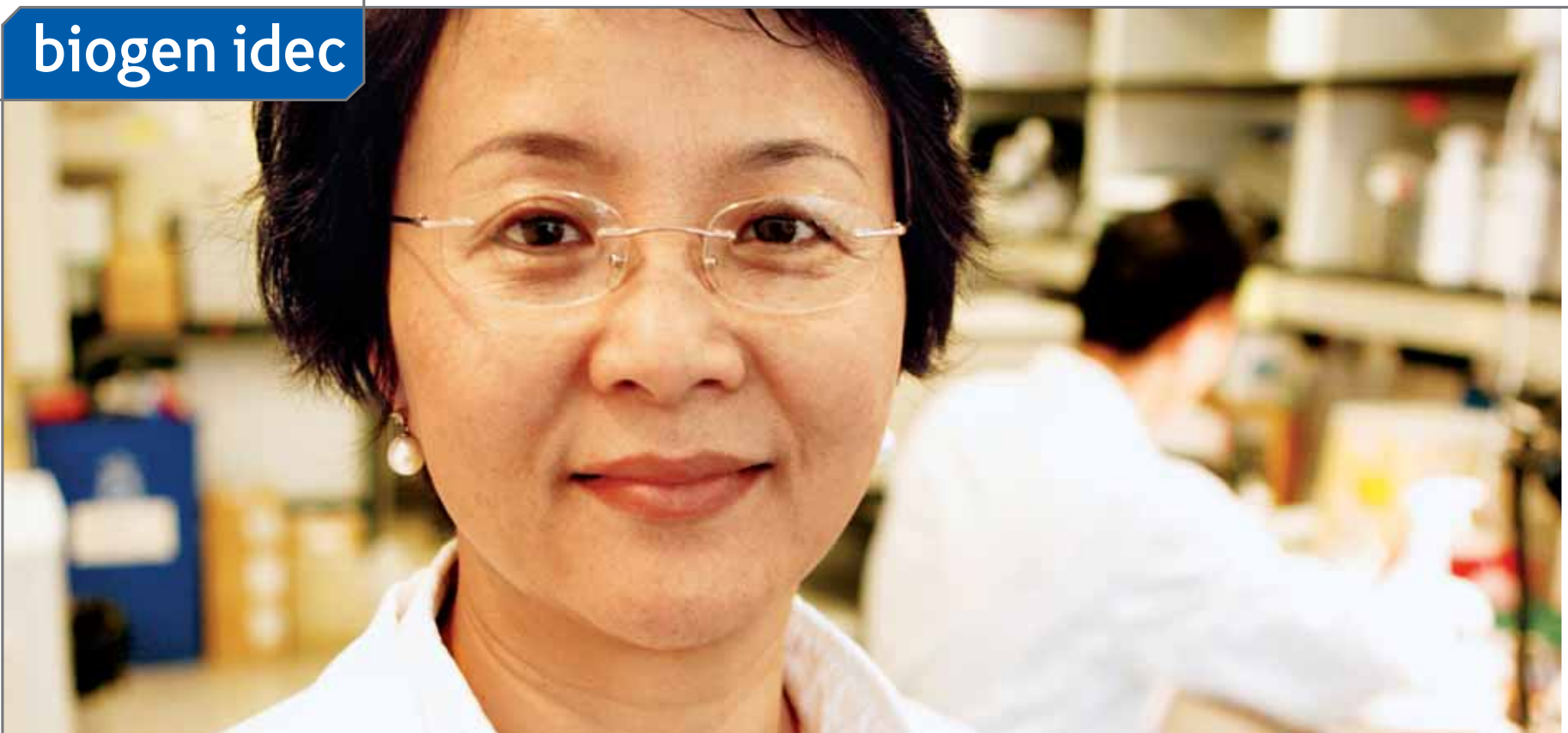
postcommissural putamen bilaterally (*Neurology* 2008;70:1980-3). Patients were assessed clinically and by PET imaging with fluoro-metatyrosine (FMT), a tracer that is more specific than fluorodopa for AADC activity. Clinical and PET scan improvements were noted. There were no adverse effects related directly to the therapy, although the surgical procedure led to intracranial hemorrhage in two instances (asymptomatic in one). It was possible to reduce dopaminergic therapy in several patients without loss of therapeutic benefit.

A third approach involves delivery of a trophic factor to the nigrostriatal system, in the hope of slowing disease progression. Neurturin (NTN) is a naturally occurring structural and functional analogue of glial-derived neurotrophic factor; it supports survival of dopaminergic cells in vitro and, in animal models of PD, prevents degeneration of nigrostriatal neurons and improves function. In a phase I study, AAV-NTN was delivered intraputaminally bilaterally in a low or higher dose to 12 patients with stage III or IV PD and response fluctuations (*Lancet Neurol.* 2008;7:400-8). There were no major adverse events, and significant clinical improvement was seen, compared with baseline.

These results suggest that gene therapy for PD is feasible and appears safe. Whether it is efficacious remains to be demonstrated. And the question remains: is it really necessary to develop gene therapy simply to duplicate what can already be achieved by a standard operation? ■

DR. AMINOFF is director of the Parkinson's Disease Clinic and Research Center and the executive vice chair of the department of neurology at the University of California, San Francisco.

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Chemo Plus Radiation Cuts Glioma Recurrence at 2 Years

Living beyond 2 years was significantly more likely for patients on radiotherapy plus PCV.

BY KERRI WACHTER
Elsevier Global Medical News

CHICAGO — Progression-free survival begins to improve after 2 years when patients with high-risk, low-grade glioma are treated with chemotherapy and radiation rather than with radiotherapy alone, according to the results of a phase III trial.

Progression-free survival rates were similar at 2 years, whether patients were treated with radiation alone or with radiation plus a chemotherapy regimen of procarbazine/lomustine/vincristine (PCV)—74% and 75%, respectively.

The groups began to separate after 2 years, however. Progression-free survival at 5 years was 46% for the radiotherapy arm and 63% for the combination arm (hazard ratio 0.60, $P = .06$).

Overall survival showed a similar trend. Two-year overall survival was compar-

able for the two groups (85%-87%), but at 5 years, overall survival was 63% for the radiotherapy arm and 72% for the combination arm (HR 0.72, $P = .33$), Dr. Edward G. Shaw reported at the annual meeting of the American Society of Clinical Oncology.

He presented these findings from the Radiation Therapy Oncology Group (RTOG) protocol 9802 study for high-risk patients who had newly diagnosed adult supratentorial low-grade gliomas (World Health Organization grade II) and a Karnofsky performance score of at least 60. High risk was defined as either being at least 40 years of age or being a patient of any age with an incomplete resection/biopsy. Results for low-risk patients were previously reported.

A total of 251 patients were randomized either to radiation therapy alone (126 patients) or to radiation plus six 8-

week cycles of chemotherapy (125 patients). Radiation was given to localized treatment fields and consisted of 30 1.8-Gy fractions (54 Gy total). Postradiation chemotherapy consisted of 60 mg/m² oral procarbazine (Matulane) on days 8-21, 110 mg/m² oral lomustine (CeeNU) on day 1, and 1.4 mg/m² IV vincristine on days 8 and 29 (no greater than 2 g per dose).

Median follow-up was 6 years. Median progression-free survival for the radiotherapy group was 4.4 years, but has not been reached for the combination arm. Likewise, median overall survival for patients in the radiation therapy arm was 7.5 years; median overall survival has not yet been reached by patients in the combination arm. Patients who survived beyond 2 years tended to be younger (less than 40 years), to have undergone resection (versus biopsy), and to have oligodendrogliomas/oligoastrocytomas, said Dr. Shaw, who is chair of the radiation oncology department at Wake Forest University, Winston-Salem, N.C.

The researchers also performed a conditional probability analysis for overall survival, asking what the likelihood of living longer than 2 years was for those patients who had lived to 2 years. "The likelihood of living beyond 2 years was significantly better for patients who were treated with radiotherapy plus PCV versus radiation therapy alone [HR 0.52, $P = .03$]," said Dr. Shaw.

A similar analysis for progression-free survival resulted in an even more pronounced advantage to those on combination therapy (HR 0.44, $P = .002$).

Severe toxicity (grade 3/4) was more common for the combination arm. Grade 3 toxicities occurred in 8% of patients in the radiotherapy arm, compared with 51% in the combination arm. Likewise, grade 4 toxicities occurred in 3% of patients in the radiotherapy arm, compared with 15% in the combination arm. "Most of the toxicities were hematologic and reversible," said Dr. Shaw.

Dr. Shaw reported that he had no conflicts of interest. ■

Psoriasis Independently Increases Stroke Risk

BY BRUCE JANCIN
Elsevier Global Medical News

KYOTO, JAPAN — Severe psoriasis appears to be a potent risk factor for stroke independent of the traditional stroke risk factors, Dr. Rahat S. Azfar said at an international investigative dermatology meeting.

Dr. Azfar presented a case-control study drawn from the U.K. General Practice Research Database (GPRD) in which she found that severe psoriasis was associated with an excess stroke risk amounting to one additional stroke per 530 patients per year beyond background levels of traditional stroke risk factors.

"Given the prevalence of psoriasis worldwide, these numbers carry a potentially significant impact on public health," observed Dr. Azfar, of the University of Pennsylvania, Philadelphia.

Psoriasis affects roughly 2.5% of the population worldwide, in-

cluding an estimated 4.5 million U.S. adults.

Five percent of psoriasis patients have severe disease as defined by a need for systemic therapy or phototherapy.

Dr. Azfar and her coinvestigators had previously shown psoriasis to be an independent risk factor for acute MI, also using the GPRD. But the relationship between psoriasis and stroke had never before been studied.

The GPRD is an extensive electronic medical record including more than 9 million U.K. patients under the care of general practitioners/family physicians in 450 primary care practices. Dr. Azfar reported on 129,143 patients with mild psoriasis in 1987-2002 and 496,666 contemporaneous controls without psoriasis, along with 3,603 patients with severe psoriasis and 14,330 separate controls. The mean follow-up was about 4 years.

As found in other studies, pa-

tients with severe psoriasis had higher rates of obesity and smoking than did controls, while rates of these and other traditional cardiovascular risk factors were similar in patients with mild psoriasis and in controls.

After adjustment for the major stroke risk factors—diabetes, hyperlipidemia, smoking, obesity, hypertension, age, and gender—patients with mild psoriasis were found to have a statistically significant 6% per year increased relative risk of stroke.

In contrast, the stroke risk in patients with severe psoriasis was increased by 43% per year, compared with matched controls.

The attributable risk of stroke in patients with mild psoriasis was 2.4 strokes per 10,000 person-years, and with severe psoriasis it was 1.9 strokes per 1,000 person-years.

A caveat: Data audit suggested up to 15% of patients categorized in the GPRD as having mild psoriasis may actually have had moderate disease.

If so, truly mild psoriasis may not be associated with any significant excess in strokes, according to Dr. Azfar.

The working hypothesis is that the link between psoriasis and stroke—and MI as well—lies in Th1/Th17-mediated systemic inflammation, a prominent shared feature, she explained at the meeting, sponsored by the European Society for Dermatological Research, the Japanese Society for Investigative Dermatology,

and the Society for Investigative Dermatology.

To examine the possibility that the excess stroke risk seen in severe psoriasis was a function of toxicities of treatments for the disease rather than being intrinsic

of the rates of cardiovascular, cerebrovascular, and peripheral vascular disease in the same study population.

The rationale for this additional analysis was that MI and stroke are acute thrombotic events, and

it would be informative to see if psoriasis is also associated with increased rates of chronic atherosclerotic diseases as reflected in the appropriate diagnostic codes, as well as procedure codes for coronary revascularization, carotid endarterectomy, and peripheral vascular intervention.

This indeed proved to be the case. As for stroke, the associated risks generally were greater with severe than with mild psoriasis, noted Mr. Shin, a medical student at the university. (See chart.)

There was, however, one glaring exception to the broad trend. How to explain the lack of association between severe psoriasis and increased peripheral vascular disease?

"I've asked some of my colleagues in cardiovascular medicine, and they think peripheral vascular disease is significantly underdiagnosed," commented Dr. Joel M. Gelfand, who is the senior investigator in the GPRD studies and medical director of the clinical studies unit in the department of dermatology at the university.

The ongoing GPRD studies are partially funded by an unrestricted grant from Centocor.

The investigators reported having no conflicts of interest. ■

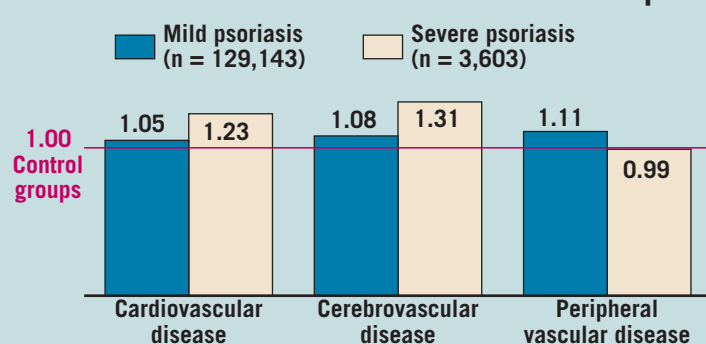


DR. RAHAT S. AZFAR



MR. DANIEL B. SHIN

Odds Ratios of Atherosclerotic Diseases In Psoriasis Patients at 4-Year Follow-Up



Notes: Odds ratios adjusted for traditional factors. There were 496,666 controls for mild psoriasis and 14,330 controls for severe psoriasis. Source: Mr. Shin

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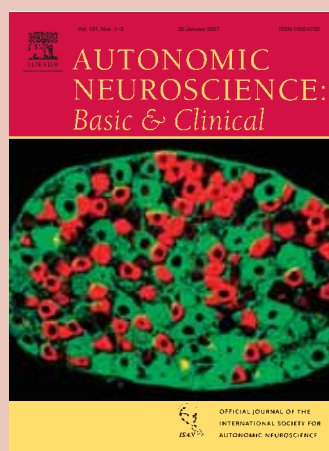
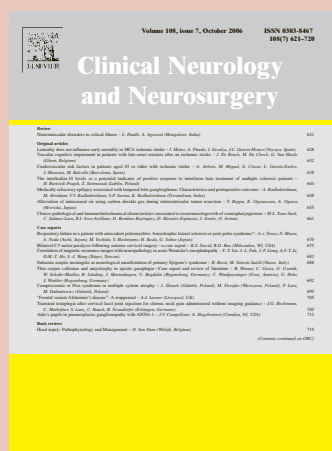
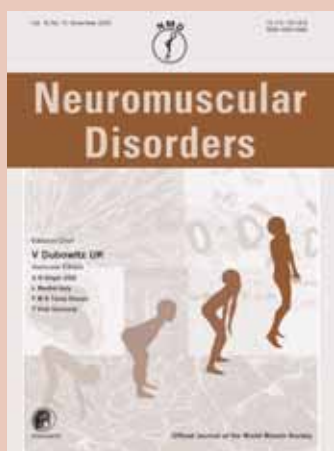
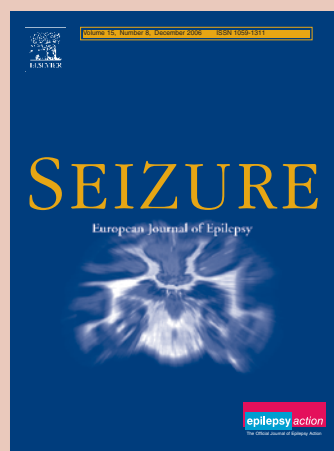
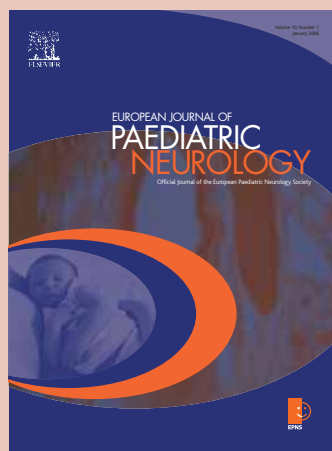


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NEWS FROM THE NATIONAL SOCIETIES

German Society for Neurology Celebrates 100th Anniversary

BY DR. STEFAN BRANDT,
DR. GÜNTHER DEUSCHL,
AND DR. AXEL
KARENBERG

Editor's note: As the German Society for Neurology celebrates its 100th year, we present a look back.

The Founding Years

"We call ourselves neurologists (Nervenärzte) and are proud to declare ourselves representatives of this science. We are united by our love of the profession." When Hermann Oppenheim spoke these words on Sept. 14, 1907, to open the first meeting of the Association of German Neurologists (Gesellschaft Deutscher Nervenärzte), the discipline could already look back on a long tradition. In 1840 Moritz H. Romberg published the world's first textbook that described all of the disorders of the nervous system that were then known.

In subsequent decades, physicians interested in neurology produced impressive research results. In 1874, Carl Wernicke described sensory aphasia and in 1884, Wilhelm Erb discussed muscular dystrophy. In 1891, Heinrich Quincke reported the first successful spinal tap; in 1906, Alois Alzheimer published a case of presenile dementia.

Shortly before World War I, Max Nonne in Hamburg and Otfried Förster in Breslau set up independent departments. In contrast to those in France, Great Britain, and the United States, German medical schools and community hospitals long considered clinical neurology a sub-discipline of psychiatry or internal medicine.

During the first quarter-century, membership of the Association of German Neurologists grew quickly, to 720. Many scientific papers from international authors were published in German. The main topics of the annual meetings between 1916 and 1930 reflected the questions of the day: shell shock (Kriegszitterer), brain trauma, neurosyphilis, epilepsy, and new diagnostic methods. It was in this period that Hans Berger described the EEG, implementation of which in clinical practice was an international scientific achievement.

The Dark Years

The advent of Nazism in 1933 had a dramatic affect on scientific leaders, morality, the quality of clinical care, and the science of German neurology. For Jewish neuroscientists, this was the beginning of the distressing history

of expulsion from the profession. Some were deported to concentration camps or chose suicide. The long list of emigrants reads like a who's who of Central European neurology: Josef Gerstmann, Kurt Goldstein, Sir Ludwig Guttmann, Friedrich Heinrich Lewy, Adolf Wallenberg, and Robert Wartenberg.

Because the Third Reich sought to stop or even reverse medical specialization, the neurologic association was forcibly "reunited" with psychiatry in 1935, a step that thwarted any further aspirations of autonomy.

More appalling was the involvement of German brain researchers, neuropathologists, and neurologists in the ideologically



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guilty of crimes against humanity, the hospitals were destroyed, and our international reputation in the field had been lost. With Germany divided after the war, the Cold War limited contact between neurologists on the two sides of the country. In West Germany, neu-

rologists to be introduced to modern techniques of clinical and experimental research. German neurologists have done pioneering research, particularly in the field of stroke, movement disorders, pain, multiple sclerosis, epilepsy, and many other fields; clinical neurophysiology is a closely related discipline whose research is promoted by the German Society for Clinical Neurophysiology.

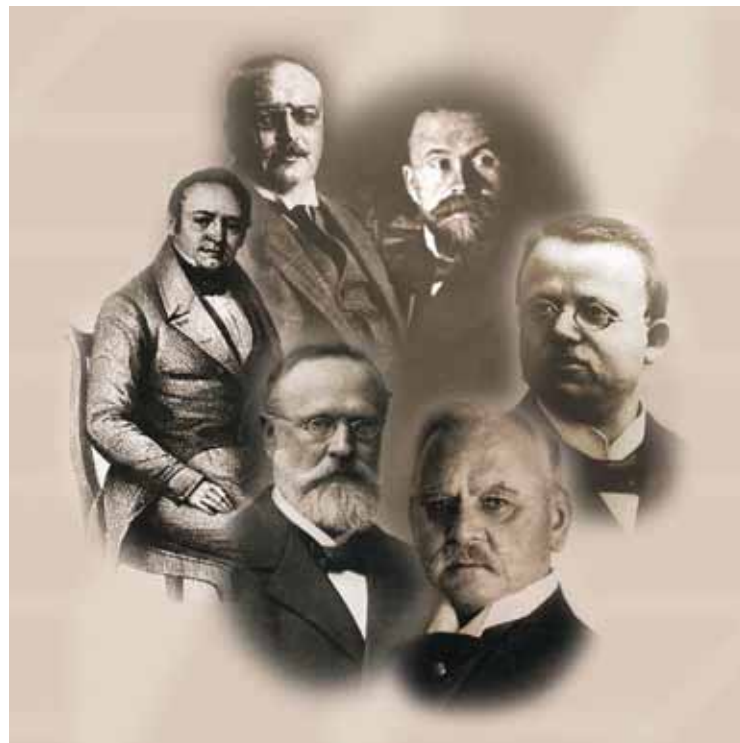
The Years of Prosperity

The Joint Congresses, first with French neurologists in 1966-1970 and then with British neurologists in 1968-1971, were among the first indications of the growing acceptance of German neurology after the war. Many of the 41 university departments in 36 German universities were established between 1960 and 1980. During that time, neurology increasingly was accepted as an academic specialty and a desperately needed discipline for daily patient care. In 1968, neurology became an autonomous medical speciality in West Germany, with board examination and certification, while in East Germany, neurology and psychiatry remained combined as a single discipline. Two new West German neurology journals (EEG-EMG and Aktuelle Neurologie) were founded around 1970 and the number and size of German-language neurology textbooks grew.

German Neurology: The 1980s

Since the 1980s, the DGN has promoted recognition that care for stroke was a critical need. Recent research and the development of care facilities have led to the advent of a nationwide network of stroke units to provide primary care for stroke patients. This was only possible through the installation of almost 400 departments of neurology at large and small community hospitals.

A second important advance was the development of intensive care neurology as a major focus of neurologic care. Many university or large community hospitals now have their own in-



Founders of the German Society for Neurology. Clockwise from left: Heinrich-Moritz Romberg (1795-1873), Alois Alzheimer (1864-1915), Carl Wernicke (1848-1905), Hermann Oppenheim (1858-1919), Max Nonne (1861-1959), and Wilhelm Heinrich Erb (1840-1921).

motivated and systematic murder of the mentally ill. After 1940, brains from patients who were murdered within the framework of the so-called euthanasia program were made available to researchers. Since the end of World War II, German science has had to come to terms with this horrific component of its past, through trenchant historic research and an appeal for forgiveness to the victims. The role played by neuropathologic research in Nazi Germany has made clear to all that there can be no progress without morality and humanity.

After World War II

In 1945, German neurology reached a nadir in its history: The best neurologists had left the country or had been murdered, some of the remaining neurologists were

rechristened in 1950 as the German Society for Neurology (DGN). In East Germany, the legal successor to Gesellschaft Deutscher Nervenärzte was the reconstituted Society for Psychiatry and Neurology, in which neurologists and psychiatrists remained in the same organization. The recovery of neurologic care, education, and society was an outstanding achievement of the first two generations of neurologists after the war.

Since then, German universities continue to be among the most important institutions for neurologic research; our researchers build global partnerships, particularly in the United States and Europe. Successful exchanges sponsored by the Deutsche Forschungsgemeinschaft and other German funding organizations have allowed Ger-

tensive care units or provide emergency neurologic treatment in interdisciplinary intensive care wards.

Another noteworthy aspect of the growth of neurology in Germany is the ongoing development of neurologic rehabilitation as a subspecialty. Many severely handicapped patients, particularly those with stroke, receive additional treatment in rehabilitation hospitals because of the shortening of the inpatient period after acute neurologic events. Rehabilitation is also becoming more popular as a research field at German universities. A major breakthrough came with the invitation of the WFN to host the 13th World Congress of Neurology in Hamburg in 1985.

The DGN Today

The most important event of the second half of the last century for the country as well as the DGN was the reunification of Germany in 1989. The DGN today has almost 6,000 members and holds annual congresses. In 2007, more than 5,000 people came to the Anniversary Congress in Berlin. Our meetings are combined with those of a national Academy of Neurology and cover all aspects of the specialty. The DGN is engaged in setting the standards for neurologic care. It has developed and constantly updates guidelines for therapy and diagnosis of many neurologic diseases.

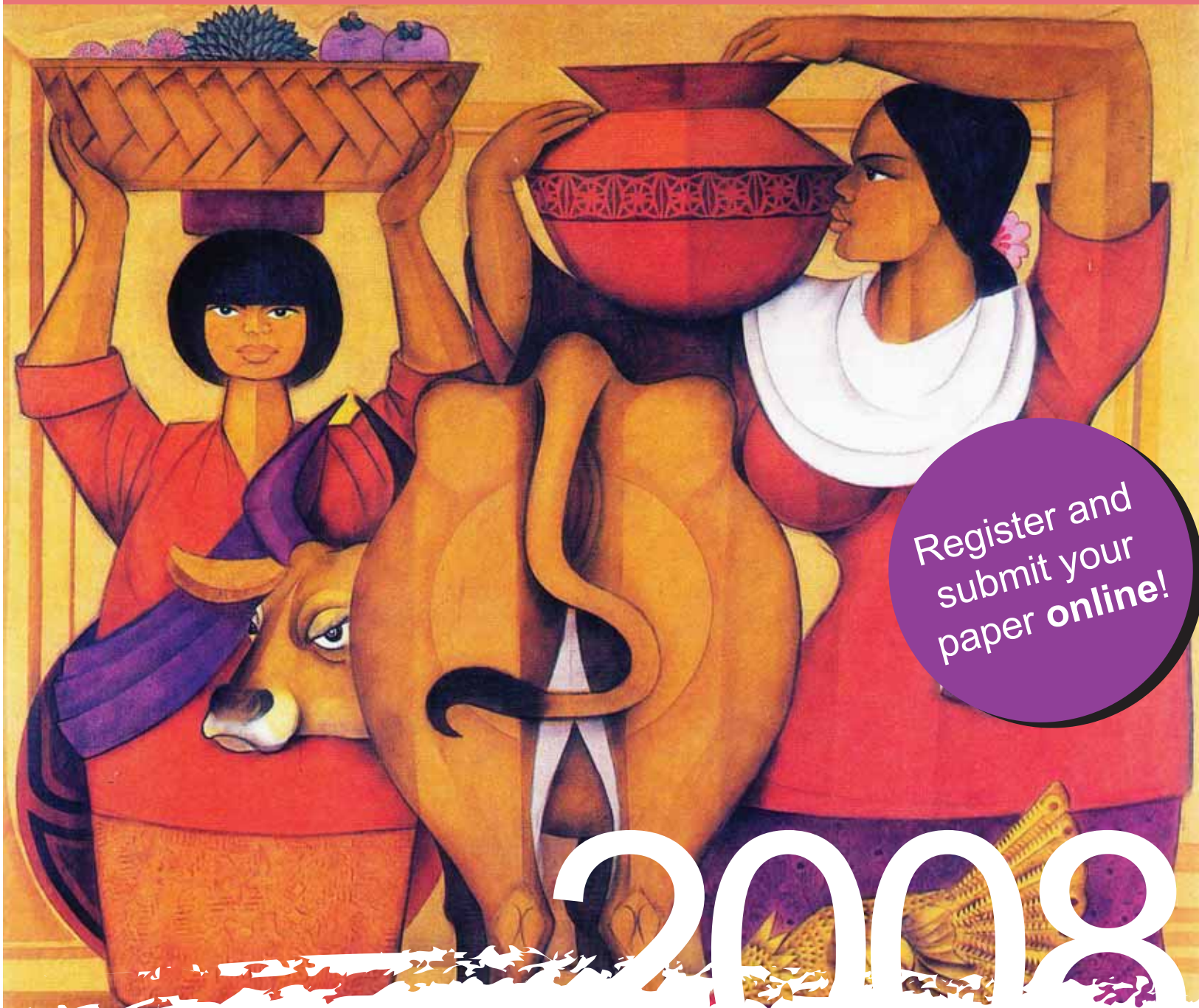
During recent years, the DGN changed its internal structure and now is organized as a contemporary society with its permanent professional headquarters in Berlin-Mitte. Our future challenge is to maintain high standards of care for the increasing number of neurologic patients, despite budget cuts for medical care in Germany. The future development of neurology and neurologic care will depend on the success of research to cure or postpone the handicaps of neurologic disease. This task is an international one, and the DGN is open for international collaboration. ■

DR. BRANDT is of the department of neurology at Humboldt-University Berlin.

DR. DEUSCHL is the chairman of the department of neurology at, Christian-Albrechts University, Kiel (Germany), and also is president of the German Society for Neurology.

DR. KARENBERG is from the Institute for the History of Medicine and Medical Ethic at the University of Cologne (Germany).

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HIGHLIGHTS FROM THE JOURNAL OF NEUROLOGICAL SCIENCES: Dengue Fever and the Central Nervous System

BY ALEX TSELIS, M.D., PH.D.

Dengue fever (DF) is an acute febrile illness found in tropical and subtropical parts of the world. It is caused by a mosquito-borne flavivirus, and characterized by high fevers, headaches, rash, and joint pains. So why does it concern neurologists?

In years past, DF resembled severe influenza, in which prostration was thought to be part and parcel of the general “toxic” effect. Physicians now realize that many patients are frankly encephalopathic, which raised the question: Does the virus invade the brain and cause encephalitis?

Dr. R.B. Domingues and colleagues evaluated 85 consecutive adults with dengue fever during the 2002-2003 summer season dengue epidemic in Brazil. They found 18 (21%) with prominent neurologic findings not attributable to other conditions.

Most had changes in mental status, and, usually, confusion rather than drowsiness;

a small number also showed incoordination, Babinski signs, hemiparesis, and nuchal rigidity. More than half of the 13 patients who had their cerebrospinal fluid (CSF) tested had positive polymerase chain reactions for viral RNA. In three, the CSF PCR was positive while the blood was negative (*J. Neurosci.* 2008;267:36-40).



DR. R.B. DOMINGUES

The authors emphasize what isn't known about DF, such as whether there are characteristic findings on imaging and the pathogenesis of the neurologic complications. It also is not known whether a direct viral infection of neurons occurs or if there is an immunopathogenetic mechanism, such as acute parainfectious demyelination.

Contacted by *WORLD NEUROLOGY*, Dr. Domingues of the department of pathology at the Escola Superior de Ciências da Saúde de Vitória (EMESCAM), Vitória (Brazil), noted, “Dengue virus infection is a very common disease in the developing world.

“In 2008, there were more than 120,000 dengue cases registered in Brazil, with more than 600 cases of dengue hemorrhagic fever and almost 50 deaths. [Considering the fact that,] during epidemics, many cases of patients with neurological involvement are expected to be seen, neu-

rologists must be familiar with the potential neurological manifestations.” ■

DR. TSELIS is an associate professor of neurology at Wayne State University, Detroit. He is the Reviews and Book Reviews Editor for the *Journal of the Neurological Sciences*.

Facts About Dengue Fever

Dengue fever is carried by *Aedes* mosquitoes. They had been suppressed, in the past, as a result of campaigns to eradicate yellow fever, which is caused by another flavivirus. However, for various reasons, these campaigns are no longer pursued with the same vigor. And with changes in populations and climate, the opportunity for further spread of the disease increases.

The incidence of dengue fever is significant and growing considerably. In 2005, the Pan American Health Organization reported 427,627 cases in the Americas, and the World Health Organization reported that the number of

cases rose from 46,458 in Southeast Asia in 1986 to 218,821 cases in 1998. By 2006, that number had risen to 188,684, with an overall upward trend since 2003.

More recently, new and more malignant forms of DF have emerged, in which multiple organ failures and hemorrhagic manifestations have assumed a prominent role. These are known as dengue shock syndrome (DSS) and dengue hemorrhagic fever (DHF). While DSS and DHF probably occurred sporadically during dengue epidemics in the past, these have formed a substantial proportion of the total number of cases.

New Study Will Combat Stigma of Epilepsy in Zambia

BY DENISE NAPOLI
Elsevier Global Medical News

Dr. Gretchen Birbeck first became aware of the pervasive stigma experienced by Zambians with epilepsy in 1994, while working in a clinic in the small, southern African country. People with epilepsy would come in for treatment all the time—for a burn or another injury experienced during a seizure—but not for the disease itself. Having epilepsy was not something to be publicized, even to health care professionals.

Many epilepsy patients around the globe complain of stigma associated with the disease. In the western world, “Data ... strongly suggest that the stigma people with epilepsy have is often self-inflicted. They have life limitations that they've often self-imposed,” said Dr. Birbeck. But in Zambia, the limitations experienced by epileptics are especially dramatic. And very real.

For example, “People with epilepsy are very disadvantaged in education and employment but also in terms of marriage,” said Dr. Birbeck. It's difficult to find a partner, but perhaps even more troubling is that even after marriage, “the women, especially, were abandoned.” Findings from an observational study conducted by Dr. Birbeck and her colleagues from 2003-2007 also revealed that single women with epilepsy felt vulnerable to sexual assault, which prevented them from seeking health care—and not unduly (*Lancet Neurol.* 2007;6:39-44). “When we did our quantitative assessment, the rate for rape for epilepsy was 20%, compared to less than 3% in our comparison group.”

There is even evidence of discrimination



From left to right: Dr. Alan Haworth, Henry Kansembe, Dr. Elwyn Chomba, Edward Mbewe, Dr. Gretchen Birbeck, Dr. Masharip Atadzhonov, Dr. Phillimon Ndubani.

against epileptic children by their own parents. In a comparison of epileptic children's food intake and nonaffected children's rations across households of similar means, “relative to the child in the other household, the child in the household with epilepsy has food deprivation,” said Dr. Birbeck, associate professor and director of the International Neurologic and Psychiatric Epidemiology program at Michigan State University.

Now Dr. Birbeck and her colleagues from Michigan State, the University of Zambia, and several Zambian health care institutions will have an opportunity to affect some of that stigma faced by Zambians with epilepsy. The Epilepsy-Associated Stigma in Zambia study, to be led by Dr. Bir-

beck, will implement social, educational, and economic interventions that her team developed after analysis of the causes of stigma and discrimination in her last study.

The interventions will be specifically targeted to each of several “power groups”—those people with the most influence in society. “We know that health care workers, clerics, police officers, and teachers propagate [the stigma against epilepsy]. But those are also the individuals who could have the largest positive impact,” said Dr. Birbeck.

In a targeted approach to overcoming discrimination by teachers, Dr. Birbeck will employ the observation made in her previous study that teachers who personally knew someone with epilepsy were less likely to stigmatize other epileptics. Dr. Birbeck

plans to set up a “somewhat bogus” week-long intervention program, where the teachers will learn about the disease from an educator they have never met. At week's end, “the teachers will find out that this person they've been working with all week actually has epilepsy.” She hopes that developing a bond with the educator, perhaps more than the lessons themselves, will break the teachers of discriminatory habits.

Another example involves Zambia's traditional healers. Dr. Birbeck's previous research showed that one of the factors in their discrimination against epilepsy patients is whether they believe the disease is medical or the result of witchcraft. She hopes to teach healers the scientific causes of epilepsy.

Intervention in households, however, will be more difficult, and will not be a focus of the present study. According to Dr. Birbeck, after her last study, “I don't think we understand that dynamic well enough to know.”

For example, she said, sometimes it is not uncommon for parents of children with epilepsy to elect not to send their child to school. “Sometimes it's because they don't want to waste money” on a child who, they presume, won't amount to much.

But “sometimes it's because they're worried the child isn't safe at school.” In both cases, the child loses, but they are “very different motivating factors. We need to understand that better, so we can intervene,” said Dr. Birbeck.

Her upcoming study will be funded with a \$1.38 million grant from the National Institute of Neurological Disorders and Stroke of the National Institutes of Health. ■

NEUROLOGIC PEARL

The Ramsay Hunt Syndromes

BY MARK HALLETT, M.D.
Editor in Chief

For reasons that are not always clear, sometimes people's names get attached to neurologic syndromes. One of the champions in this regard is James Ramsay Hunt, who has at least four syndromes named after him. Perhaps he gained this recognition because of the superb detail of both his clinical reports and correlative pathological studies.

The four Ramsay Hunt syndromes are sufficiently confusing that they are now designated by type.

Hopefully, the following list will help keep these often-perplexing syndromes straight.

► Ramsay Hunt syndrome I is a progressive hereditary neurodegenerative disease characterized by ataxia, myoclonus, seizures, and cognitive decline.

Dr. Hunt believed that he was describing cases similar to those described before him by Unverricht and Lundborg, but some people

started using Dr. Hunt's name to describe patients with these symptoms. The use of Ramsay Hunt syndrome to describe this disorder is confusing since there are so many etiologies for this syndrome (Hunt JR. *Dyssynergia cerebellaris myoclonica*—primary atrophy of the dentate system. A contribution to the pathology and symptomatology of the cerebellum. [Brain 1921;44:490-538]).

► Ramsay Hunt syndrome II, also called Herpes zoster oticus, is a herpes zoster infection of the geniculate ganglion with pain in the ear, herpetic blisters of the skin of the ear canal or auricle, and facial paralysis. There can also be dysfunction of the sensory portion of the seventh nerve and dysfunction of the eighth nerve with vertigo, hearing loss, and tinnitus. This was an original description, and it is called type II even though its description preceded that of type I by 14 years (Hunt JR. On herpetic inflammations of the geniculate ganglion: a new syndrome and its complications. [J. Nerv. Ment. Dis. 1907;34:73-96]).

► Ramsay Hunt syndrome type III can be described as a compression neuropathy of the deep palmar branch of the ulnar nerve from an occupational cause (Hunt JR. Occupational neuritis of the deep palmar branch of the ulnar nerve: a well defined clinical type of professional palsy of the hand. [J. Nerv. Ment. Dis. 1908;35:673-89]).

► Finally, Ramsay Hunt syndrome type IV, which is also called Ramsay Hunt paralysis, is a form of juvenile Parkinson disease with the pathological substrate of neuronal degeneration in the globus pallidus. Juvenile Parkinson disease had been described, but at the time of this article there had not been any prior neuropathologic correlation (Hunt JR. Progressive atrophy of the globus pallidus [primary atrophy of the pallidal system]. A system disease of the paralysis agitans type, characterized by atrophy of the motor cells of the corpus striatum. A contribution to the functions of the corpus striatum. [Brain 1917; 40:58-148]).

WFN Now Accepting Nominations for Two New Medals

The Trustees of the World Federation of Neurology have decided to establish two new WFN medals, one for Service to Neurology and one for Achievement in Neurology.

Each award will carry an honorarium of \$5,000. Nominations, which may be made either by World Federation of Neurology member societies or by individual members of a member society, should be seconded by at least five neurologists.

Three of these individuals should be from other WFN member societies.

The nominated individual should have approved the nomination and the principal proposer should write a citation of no more than 300 words in support of the nomination.

The Medal Committee will be made up of the current president of the WFN and two previous World Federation of Neurology presidents or World Federation of Neurology trustees of the president's choice.

The first medals will be awarded during the 2009 World Congress of Neurology in Bangkok (Thailand). All nominations should be sent to:

The Medal Committee
c/o the World Federation of Neurology London Office
12 Chandos St., London, W1G 9DR, United Kingdom

Nominations must arrive by October 10, 2008.



12th Asian Oceanian Congress on Neurology & 16th Annual Conference of the Indian Academy of Neurology

(AOCN-IANCON 2008)

Organized under the aegis of:

Asian Oceanian Association of Neurology (AOAN),
Indian Academy of Neurology (IAN)
World Federation of Neurology (WFN)
Delhi Neurological Association (DNA)



In association with

Association of Indian Neurologists in America (AINA)

Pre-Conference workshops on EEG,
Multiple Sclerosis, Movement Disorder & Advocacy
October 22, 2008 (Wednesday)

Main Conference
October 23-26, 2008 (Thursday-Sunday)

CME Accreditation

The 12th Asian Oceanian Congress of Neurology (AOCN 2008) & 16th Annual Conference of the Indian Academy of Neurology (IANCON 2008) have applied to Delhi Medical Council for accreditation of CME hours for the AOCN - IANCON Workshops and the Main Conference.

Venue: The Ashok Hotel, New Delhi, India

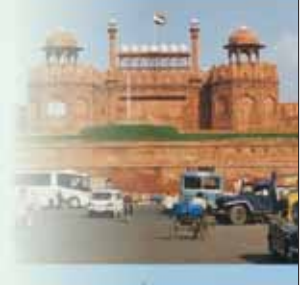
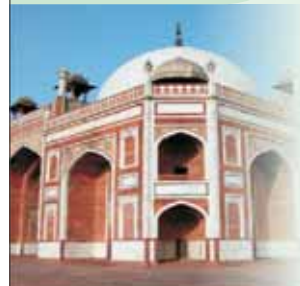
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PROFILES IN NEUROLOGY

My Practice in Bangkok, Thailand

This issue's column takes us to Bangkok, Thailand, and the practice of Dr. Rawiphan Witoonpanich, who is a neurologist and associate professor and consultant neurologist in a medical school. Dr. Witoonpanich obtained her medical degree from Ramathibodi Hospital in Thailand, where she began her training. She had further training in England, where she worked at Newcastle General Hospital, St. Thomas' Hospital, and the National Hospital for Neurology and Neurosurgery. Dr. Witoonpanich specializes in neurophysiology and has expertise in electromyography (EMG). Dr. Witoonpanich is also interested in neuromuscular disorders, particularly myasthenia gravis. She currently teaches at Mahidol University, Bangkok, and is on the faculty of medicine at Ramathibodi Hospital.

My job consists of teaching, patient care, and research. I teach medical students and neurology residents and look after outpatients and inpatients. I also perform and teach EMG.

I enjoy teaching and I always participate in academic activities, which are essential parts of residency training. I tend to spend much time talking to and examining a patient to make an accurate clinical diagnosis before sending the patient for various tests. I am rather obsessive about this clinical approach and try to emphasize this in my teaching.

Communicating with the patient can, to a certain extent, protect a physician from being sued as well. Being better educated and increasingly health conscious, Thai people have a growing tendency to sue their doctors for malpractice.

I see a wide variety of patients because we do not have enough attending staff doctors for everyone to see only patients of one's own subspecialty. In recent years, apart from ordinary epilepsy and stroke, I have seen more motor neuron disease, especially amyotrophic lateral sclerosis, multiple sclerosis, and Alzheimer's disease.

In the field of neuromuscular disorders, we have many patients with Guillain-Barré syndrome, myasthenia gravis, and inflammatory myopathy. Other common diseases include diabetic polyneuropathy, chronic inflammatory demyelinating polyneuropathy, connective tissue diseases with mononeuropathy multiplex, muscular dystrophy, thyrotoxic myopathy, periodic paralysis, and mitochondrial myopathy.

Central nervous system infections, which used to be very com-

mon in our region, have become less common nowadays because people are more aware of hygiene and health maintenance. Infections that we used to see quite often were pyogenic meningitis, viral meningoencephalitis, tuberculous meningitis, cryptococcal meningitis, neurocysticercosis, eosinophilic meningitis from *Angiostrongylus cantonensis*, eosinophilic meningoencephalitis caused by *Gnathostoma spinigerum*, and neurosyphilis. However, opportunistic infections such as tuberculosis, cryptococcosis, and toxoplasmosis are still prevalent among patients with AIDS.

There are also occasional outbreaks of food-borne botulism. The largest occurred 2 years ago and affected more than 100 people. The implicated food was improperly processed home-canned bamboo shoots.

There are opportunities to do clinical research here in Bangkok. Research is encouraged but not compulsory in our institution; however, academic positions and promotions depend on the amount of research completed, having a certain number of publications and book chapters. There is some limitation on advanced research because of a shortage of trained personnel and funding. I have one or two half-days a week spared for my research, which is mainly on myasthenia gravis, some muscle



Dr. Witoonpanich examines a patient with Kennedy's disease at Ramathibodi Hospital as three neurology residents look on.

diseases, and the clinical application of EMG.

In the past, I have served as president of the Neurological Society of Thailand and organized a few international meetings, including the Asian and Oceanian Symposium on Clinical Neurophysiology in February 2005 in Chiang Mai, also in Thailand. I am a member of the executive committee of the Asian and Oceanian Myology Center and president of the Asian and Oceanian Chapter, as well as a member of the rule committee of the International Federation of Clinical Neurophysiology.

The salary of a government officer is quite low here. Therefore, most doctors have to practice in the evening and over the

weekend in private hospitals. This combination of hard work together with family commitment means there is not much time left for each individual to relax or exercise. I am no longer in private practice now and, as a result, I am able to spare 3 evenings a week for exercise, usually lasting 2 hours at a time.

I devote most of my time to teaching and taking care of patients, with some hours spared for research. I insist on giving patients plenty of time so I can make an accurate clinical diagnosis, provide good care, and establish a good doctor-patient rapport. I enjoy my professional life and try to relax and exercise regularly. ■

Nelly Chiofalo, M.D., 1929-2008

BY OSVALDO OLIVARES, M.D.
AND RENATO VERDUGO, M.D.

Dr. Nelly Chiofalo died in Santiago, Chile, on April 18, 2008. Dr. Chiofalo was one of the pioneers in the development of clinical neurophysiology in Latin America.

Born on March 24, 1929, in Mendoza, Argentina, Dr. Chiofalo studied medicine at the National University of Cordoba, Argentina, where she obtained her medical degree in 1954. In 1956, she moved to Chile and gained admission into the Instituto de Neurocirugía. Working with Professor Carlos Villavicencio, she studied neurology and electroencephalography there until 1959. In 1960, she continued her education in electroencephalography at Harvard Medical School, Boston, and graduated in 1961. She returned to the Instituto de Neurocirugía, also assuming a leading position in the Chilean League Against Epilepsy.

In the late 1970s, Dr. Chiofalo became the first person in Chile to develop techniques to measure blood levels of antiepileptic drugs. She also introduced continuous electroencephalographic monitoring and, supported by Professor Jean Bancaud of Sainte-Anne in Paris, secured



Dr. Nelly Chiofalo died on April 18, 2008.

training for a team of neurosurgeons and neurologists to develop epileptic surgery. In 1978, the International League Against Epilepsy gave her the title of ambassador, which allowed her to organize symposia and congresses. In the late 1980s, after 4 years as president of the league, she developed the study of sleep disorders and introduced polysomnography to the country.

The Chilean Society of Clinical Neurophysiology, which Dr. Chiofalo founded,

advanced the field by organizing professionals dedicated to electroencephalography, electromyography, evoked potentials, and sleep disorders. Her concern for the specialty was evinced by her successful efforts in Chile for the professional development and continuing education of electrophysiology personnel. Through the society, she promoted the creation of the Latin American chapter of the International Federation of Clinical Neurophysiology (IFCN), founded in Guatemala during a symposium she organized in 1995. The statutes of the chapter were approved by the IFCN in Buenos Aires in 2001.

In 1977, Dr. Chiofalo organized a pioneering workshop on brain death in Chile, the conclusions of which were the basis for a transplant law—at a time when the subject was still controversial in Latin America and the world.

During 1978 and 1979, she carried out the first epidemiologic studies on epilepsy in Chile. Her works on the electroencephalographic abnormalities in Creutzfeldt-Jakob disease are considered classic. In addition, Dr. Chiofalo published several papers on pediatric epilepsy.

Dr. Chiofalo showed enduring interest

in the professional development of her collaborators. She was always searching for educational opportunities for Latin American neurophysiologists, encouraging them to apply for scholarships and awards, and urging them to improve their foreign language performance.

Dr. Chiofalo was married to Dr. Luciano Basauri, a prestigious neurosurgeon. The two were unstoppable travelers, visiting every continent, including Antarctica, and climbing the Himalayas. He preceded her in this final trip by only 2 months.

The couple is survived by their two sons, Cristian, an engineer, and Rodrigo, a publicist; and eight grandchildren, two of whom are in medical school.

Latin American neurologists and neurophysiologists acknowledge her importance in the development of the field in our countries, and will long remember her and miss her. ■

DR. OLIVARES is from the neurology unit at the Hospital San José, at the Universidad de Santiago (Chile). DR. VERDUGO is an associate professor of neurology on the faculty of medicine, Universidad de Chile, in Santiago.

Hereditary Neuroblastoma Tied to ALK Gene Mutations

Three novel anaplastic lymphoma kinase germline variations are identified.

BY LAUREN SCOTT
ZOELLER

Elsevier Global Medical News

CHICAGO — Mutations within the anaplastic lymphoma kinase gene are associated with hereditary neuroblastoma, and acquired ALK mutations may be related to the development of nonfamilial neuroblastoma, researchers reported.

These findings have important implications in terms of genetic screening and genetic counseling, and provide a novel target for the development of therapies that disrupt ALK signaling, according to Dr. Yael P. Mosse, a pediatrician at the Children's Hospital of Philadelphia, and her coinvestigators.

The vast majority of familial neuroblastomas are associated with mutations in the tyrosine kinase domain of ALK, which is located on chromosome 2p24-23, said Dr. Mosse at the annual meeting of the American Society of Clinical Oncology.

Mutations of the ALK tyrosine kinase domain have also been shown to have oncogenic functions in several other cancers and are involved in transformation, typically via translocation events, she noted.

ALK plays a role in regulating the development of the central and peripheral nervous systems, and is a highly conserved orphan receptor.

The investigators performed genomewide linkage studies using a panel of approximately 6,000 single nucleotide polymorphisms (SNPs). This produced evidence of linkage to a 16.1 megabase region of c2p23-24 (log score = 4.23) containing 104 genes, including ALK and MYCN. (MYCN amplification is associated with poor prognosis in neuroblastoma.)

Recombination events were then mapped to this region to determine potential neuroblastoma predisposition genes. Genetic and pedigree analysis (based on a mean of four individuals with neuroblastoma per family) indicated that heritable mutations of the ALK tyrosine kinase domain (exons 21-28) were present in 8 of the 10 families studied.

Three novel variations of ALK germline mutations were identified: R1275Q ($P = .91$), which activates the BRAF oncogene and other protein kinases; G1128A ($P = .95$), which also activates BRAF; and R1192P ($P = .98$), which has an undetermined function.

Neuroblastoma is passed on through the germline in only 1% of the children affected, however.

Therefore, the investigators sought to determine the role of ALK mutations in somatically acquired neuroblastoma.

They speculated that acquired

somatic mutations in the ALK gene can occur during evolution to the high-risk form of the disease. The notion that acquired mutations of ALK are likely to activate oncogenes such as BRAF is consistent with the two-hit model of oncogenesis.

In a panel of 491 tumors assessed, 112 had focal unbalanced gains of ALK, 16 of which had high levels of ALK amplification. ALK gain or amplification was associated with both high-risk disease (P less than .0001) and increased disease-related mortality ($P = .0003$).

A resequencing of the tyrosine kinase domain of ALK in 167 of the 491 primary neuroblastoma tumor samples showed that 24 of them had ALK mutations, including eight distinct single-base missense substitutions. Of nine tumors matched for germline DNA, eight had acquired and one had germline mutations of ALK.

Further gene knockdown experiments using small interfering RNA techniques showed a 40%-80% inhibition of ALK mutated or amplified neuroblastoma cell lines. Results suggested multiple mechanisms of ALK activation. Furthermore, the vast majority of somatically acquired ALK mutations fell within mutational "hot spots" observed in other cancers (for example, MET, ERBB2, and EGFR kinase).

Dr. Brian Kushner, a pediatric oncologist specializing in neuroblastoma at Memorial Sloan-Kettering Cancer Center, New York, discussed the study, saying that "ALK is a very important marker in these familial neuroblastomas and a promising target for therapy."

In terms of developing new ALK-targeted drugs, Dr. Mosse said, "Fortunately, although neuroblastoma is a rare disease, we are not starting from scratch. ALK is an oncogene which is implicated in other human cancers, such as anaplastic large cell lymphoma. So there are many pharmaceutical companies who are very interested in developing ALK inhibitors; some have actually developed ALK programs."

ALK is homologous to other kinases such as MET, an inhibitor of which is now being tested in phase I studies in adults, she added. Researchers working in Dr. Mosse's laboratory have cloned all of the key ALK mutations they discovered, and are performing ongoing transformation assays. ■

Migraine With Aura Linked To Increased CVD Risk

BY PATRICE WENDLING
Elsevier Global Medical News

CHICAGO — Migraine frequency appears to be an indicator of increased risk of cardiovascular disease in migraineurs with aura, according to findings from a large cohort analysis.

The study, which used data from the Women's Health Study involving 27,798 women with no history of cardiovascular disease, identified a mixed association between migraine frequency and major cerebrovascular disease.

Compared with women without migraine, women with at least weekly migraines were almost three times more likely to experience a stroke. Those with migraines less than once a month were one-and-a-half times more likely to have a heart attack, Dr. Tobias Kurth reported.

However, the increased risk of disease according to migraine frequency was apparent only for migraineurs with aura.

"The clinical implications of our data at this point are unclear," Dr. Kurth said at the annual meeting of the American Academy of Neurology. "The strongest evidence from our study and others is that migraine without aura is not associated with increased risk of cardiovascular disease. Future studies should focus on identifying patients with migraine and aura who are at particular increased risk of CVD."

At the start of the study, 3,568 women (mean age 55 years) had migraine based on self-reported questionnaires, of which 1,468 (41%) women reported had aura. Overall, 2,315 women reported migraine less than once a month, 1,073 reported one migraine per month, and 180 reported at least weekly migraines.

After an average of 12 years follow-up, there were 706 cerebrovascular events, 305 heart attacks, and 310 ischemic strokes.

In a multivariate analysis that adjusted for age, hypertension, smoking status, body mass index, total cholesterol, and history of myocardial infarction, the risk for major CVD was increased among those with at least weekly migraines (hazard ratio 1.90), as compared with women with migraine less than once a month (HR 1.54) and those with one migraine per month (HR 0.97).

A different emphasis emerged for risk of ischemic stroke and MI, said Dr. Kurth of Brigham and Women's Hospital and Harvard Medical School, both in Boston.

Women with the lowest migraine frequency were at signifi-

cantly greater risk of MI (HR 1.64), compared with those reporting one migraine per month (HR 0.94) or at least weekly migraine (HR 1.49).

Women with the highest migraine frequency had significantly increased risk of stroke (HR 2.74), although a nonsignificant increase was also seen among the lowest frequency group (HR 1.44). The finding of increased stroke risk in high-frequency migraine patients is consistent with previous studies, he said.

When analyzed based on aura status, the risk for stroke was significantly increased in the high- and low-frequency migraine groups with aura, compared with their counterparts without aura. The risk for MI was significantly increased in the low-frequency group, compared with their counterparts without aura.

An audience member observed that patients with low migraine frequency were still at increased risk for stroke, suggesting that migraine prevention may actually make things worse.

Dr. Kurth said the study raises many questions and that further research is needed to validate or refute its findings. Earlier in the presentation, he cautioned that the data do not support the conclusion that migraine prophylaxis will affect the risk of CVD.

Audience members also questioned Dr. Kurth about the fact that migraine status was defined based on a series of questions rather than through standardized definitions. He acknowledged that the study's classification of migraines was "problematic," but noted that only those patients who checked a box for aura were counted in that subgroup, suggesting that any misclassification potentially could have underestimated the associations observed in the study.

Finally, the pivotal question from the audience was whether it is aura frequency rather than migraine frequency that matters, as only migraineurs with aura had significantly increased disease risk when compared with migraineurs without aura. "I agree with you that it's the aura and not the frequency," Dr. Kurth said.

The study was supported by grants from the Donald W. Reynolds Foundation, the Leducq Foundation, and the Doris Duke Charitable Foundation. Dr. Kurth disclosed relationships with the National Institutes of Health, Bayer AG, McNeil Consumer & Specialty Pharmaceuticals, Wyeth Consumer Healthcare, and i3 Drug Safety. ■

DR. CHOPRA WINS AWARD



Dr. Jagjit Chopra was awarded the Padma Bhushan, one of the highest civilian awards in India, by the President of India Mrs. Pratibha Patil on May 5, 2008. Dr. Chopra is the only Indian neurologist to receive this award, which was given for Service to Medicine and Neuroscience. Dr. Chopra served as Editor in Chief of WORLD NEUROLOGY for the last decade.

COURTESY DR. JAGJIT CHOPRA/LIBERTY NEWS PICTURES/GOVERNMENT OF INDIA

Calendar of International Events

2008

NeuSIG Satellite to the Glasgow 2008 World Congress on Pain

August 13-15, 2008; London
www.kenes.com/neuropathic2008

XXIII Congresso Brasileiro de Neurologia

August 16-21, 2008; Belém, Brazil
www.neuro2008.com.br

12th World Congress on Pain

August 17-22, 2008; Glasgow, Scotland
www.iasp-pain.org

12th Congress of the European Federation of Neurological Societies

August 23-26, 2008; Madrid
www.kenes.com/efns2008

6th International Conference on Frontotemporal Dementia

Sept. 3-5, 2008; Rotterdam, The Netherlands
www.ftd2008.org/site

6th International Congress on Meningiomas and Cerebral Venous System

Sept. 3-7, 2008; Boston
www.themeningiomaconference2008.org

European Headache and Migraine Trust International Congress 2008

Sept. 4-7, 2008; London
www.ehmtcongress2008.com

14th World Congress of Psychophysiology

Sept. 8-13, 2008; St. Petersburg, Russia
www.world-psychophysiology.org/iop2008

6th International Congress on Autoimmunity

Sept. 10-14, 2008; Porto, Portugal
www.kenes.com/autoimmunity

World Congress on Treatment and Research in Multiple Sclerosis (ACTRIMS+ECTRIMS+LACTRIMS)

Sept. 17-20, 2008; Montreal
www.msmonreal.org

7th Mediterranean Congress of Physical and Rehabilitation Medicine

Sept. 18-21, 2008; Portorose, Slovenia
www.medcongress.prm08.org

Xth International Symposium on Thrombolysis and Acute Stroke Therapy

Sept. 21-23, 2008; Budapest, Hungary
www.kenes.com/tast2008

8th European Congress on Epileptology

Sept. 21-25, 2008; Berlin
www.epilepsyberlin2008.org

5th World Congress for NeuroRehabilitation

Sept. 24-27, 2008; Brasília, Brazil
www.sarah.br/wfnr-rio2008

6th World Stroke Congress

Sept. 24-27, 2008; Vienna
www.kenes.com/stroke2008

XXVII Annual Congress of the European Society of Regional Anaesthesia and Pain Therapy

Sept. 24-27, 2008; Genoa, Italy
www.kenes.com/esra

36th Annual Meeting of the International Society for Paediatric Neurosurgery (ISPN)

Oct. 12-16, 2008; Cape Town, South Africa
www.ispn2008.org

6th International Congress on Mental Dysfunctions and Other Non-Motor Features in Parkinson's Disease

Oct. 16-19, 2008; Dresden, Germany
www.kenes.com/pdment2008

Dystonia Europe 2008

Oct. 17-19, 2008; Hamburg, Germany
www.dystonia-europe-2008.org

2nd World Congress on Controversies in Neurology

Oct. 23-26, 2008; Athens
www.comtecmed.com/cony

9th International Congress of Neuroimmunology

Oct. 26-30, 2008; Fort Worth, Texas
www.isni2008.org

19th International Symposium on ALS/MND

Nov. 3-5, 2008; Birmingham, England
www.mndassociation.org

Child Neurology Society Annual Meeting

Nov. 5-8, 2008; Santa Clara, Calif.
www.childneurologysociety.org/annual_meeting/accomodations

Neuroscience 2008

Nov. 15-19, 2008; Washington
www.sfn.org/am2008

2009

2009 AAN Regional Conference

Jan. 16-18, 2009; Orlando, Fla.
www.aan.com/go/education/conferences

7th International Symposium of Asian and Pacific Parkinson's Association (APPA)

Feb. 15-16, 2009; New Delhi
www.aopmcindia.com/appa/appa_invitation.html

2nd Asian and Oceanian Parkinson's Disease and Movement Disorders Congress (AOPMC)

Feb. 15-17, 2009; New Delhi
www.aopmcindia.com

2nd European Brain Policy Forum

Feb. 25-26, 2009; Brussels
www.kenes.com/ebpf2009

9th International Conference on Alzheimer's and Parkinson's Diseases: Advances, Concepts and New Challenges

March 11-15, 2009; Prague
www.kenes.com/adpd

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The Federation of European Neurological Societies

The EFNS has expanded its activities, further promoting neurologic research and training.

BY JACQUES L. DE REUCK
European Regional Director of the WFN

The European Federation of Neurological Societies (EFNS), which represents 42 countries, has successfully extended its international cooperation across the Mediterranean.

The resulting collaboration among European, African, and Arabic countries will benefit us all as neurologists and advocates for the specialty.

The EFNS has adopted as associated members the adjoining neurologic societies of the Mediterranean basin: the

new member societies include those from Morocco, Algeria, Tunisia, Libya, Egypt, Lebanon, Syria, and Jordan.

These societies now share the same practical advantages as those in Europe. Neurologists in training

can participate in all the education and exchange programs of the EFNS, and are eligible to apply for travel bursaries to participate in EFNS congresses when their research is accepted for presentation. They can apply for the department-to-department exchange program and participate in the EFNS Academy of Young Neurologists.

Starting in Madrid, Aug. 23-26, 2008, and at every subsequent congress, a special Mediterranean session will be organized with European and North African participants speaking on a topic of common interest.

EFNS scientists also have been invited as guest speakers to the congresses of the Pan Arab Union of Neurological Societies (PAUNS). At the 2007 PAUNS meeting held on Oct. 25-27 in Yasmine Hammamet, Tunisia, attendees organized a common session on peripheral neuropathies.

At the 9th Cairo International Neurology Conference, held by the Egyptian Neurological Society on Feb. 13-15, 2008, a common PAUNS-EFNS session was on the program.

The EFNS has joined WFN President Johan Aarli in his efforts to help develop departments of neurology at universities throughout Africa.

Because the EFNS can only sponsor educational programs, it joined with the WFN and IBRO to organize the first African Regional Teaching Course (RTC) in Dakar, Senegal on June 26-28, 2008, for Senegalese neurologists and those from the surrounding French-speaking countries.

The local organizer was Gallo Diop, Ph.D. The topics, selected by the African colleagues, were peripheral neuropathies and neurodegenerative diseases.

The EFNS has traditionally organized randomized clinical trials in Eastern Europe, where neurologic departments are poorly developed and neurologists have difficulties participating in scientific seminars outside their countries.

This program has been very successful for many years.

One of the EFNS's main commitments is to help expand neurologic departments and to increase the quality of neuroscience research within and outside of Europe.

The EFNS supports the European Brain Council (EBC), which promotes neurologic research programs within the European Union. The council, launched by Jes Olesen, M.D., is supported in its brain research initiatives by various scientific societies; pharmaceutical companies; and the European Federation of Neurological Associations (EFNA), a patient organization.

The successful collaboration between the EFNS and the EFNA is demonstrated by EFNA's representation at EFNS congresses, where they organize sessions for patients.

The EFNS also supports international collaboration among established neuro-

logic societies. If your organization would like to work with us in our efforts to promote the importance of neurologic research to the World Health Organization, please contact the EFNS head office, Breite Gasse 4-8, A-1070 Vienna, Austria, or e-mail us at headoffice@efns.org. Our Web site is www.efns.org.

The European Federation of Neurological Societies

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www.efns.org



JACQUES L. DE REUCK

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DR. MARK HALLETT

Welcome
to the



19th World Congress of Neurology

October 24th-30th, 2009
Bangkok, Thailand



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