PRESIDENT'S COLUMN

The World Federation of Neurology (WFN) traditionally held a Council of Delegates (COD) meeting every two years, once during the quadrennial World Congress of Neurology (WCN) and the other during the preplanning meeting (PPM) for WCN. The newly adopted Articles of Association stipulate the needs of Annual General Meetings (AGM) as part of the requirements set forth by the UK Charity Commission. We held the first such AGM after the new constitution went into effect, on July 7, 2002, at the time of the Xth International Congress on Neuromuscular Diseases (XICNMD) in Vancouver, as reported in the previous issue of World Neurology.

In preparing for this AGM, we were concerned about adequate delegate attendance because we had no previous experience of calling COD only one year after the WCN. Although some 34 member societies initially indicated their participation, only 24 representatives eventually attended, with four additional countries offering a proxy assigned to a delegate of their choice. To compound the problem, most countries were represented by one of the attendees of the Neuromuscular Congress rather than the registered delegate. Anticipating this trend of substitution, we took extra care to inform each national society of our main agenda, which included the election of the Secretary-Treasurer General and a trustee. Despite our best efforts, however, many substitute delegates came without much knowledge of WFN in general and COD in particular. Despite the lack of regular delegates, the meeting accomplished the objective at (cont. on p. 4)

DISTRIBUTION OF ‘WORLD NEUROLOGY’

Important Announcement

At a meeting of the Editorial Board of World Neurology held in Denver during the annual conference of the American Academy of Neurology, it was agreed that all readers should be consulted on how they would like to receive future copies of the publication. Because World Neurology is accessible on the WFN Website, it is proposed that each issue may either be read or downloaded from the site and that this electronic format should be the prime means of distribution to the membership. Alternatively, readers may prefer to be sent a copy as an e-mail attachment. Finally, there may be those who do not have Internet access or who choose to continue to receive a hard copy of the newsletter, for these the current arrangements will remain unchanged. A number of factors lie behind this proposal, including cost issues, delays in current distribution provisions by surface mail, etc.

Readers are asked to notify the London Office at the earliest if they wish to continue to receive future issues by either (a) e-mail attachment or (b) hard copy. Otherwise, it will be assumed that a member is willing to access his or her copy on the WFN Website. These new arrangements are likely to come into effect early in 2004. (cont. on p. 3)

ALSO IN THIS ISSUE:
- Editorial
- President’s Column
- WFN Elections
- Regional Reports
- The NAIS Study
- WFN Training Programme
- Update on Genetics of PD
- Book Reviews & Calendar
EDITORIAL

This is the third consecutive ad on the title page in reference to distribution of World Neurology. Readers are requested to respond positively to help the WFN economize on the publication and mailing costs of the newsletter by distribution through electronic media as much as possible.

WFN President Jun Kimura has raised in his column some very pertinent points emphasizing the Trustees’ concern about the strictness of UK charity commission regulations regarding the holding of Annual General Meetings (AGMs). Inadequate attendance by Delegates at the first AGM since the adoption of the new WFN constitution underlines the need for careful consideration to be given to finding some means of overcoming this difficulty in future. At the AGM held during a World Congress itself and at its Planning Meeting two years earlier the number of National Delegates attending is usually satisfactory. But it is in the interim years that the problem arises. National Neurological Associations and Societies affiliated to the WFN worldwide also need to ponder this problem and find ways of funding the travelling costs of their National Delegates to participate in the Annual General Meetings. The WFN President predicts a dilemma in future if an adequate number of Delegates does not attend the AGM, not because of the UK regulations but more importantly because there is a danger that the fundamental issue of realizing the WFN’s mission to improve care of neurological patients globally will not be fulfilled. National Delegates are the torchbearers of the WFN and are duty bound to project the aims and objects of the WFN for universal better care of patients. The mission of the WFN can only be propagated if the National Delegates attend the maximum number of WFN meetings for discussion and planning.

The WFN Long Range Planning Committee’s recommendations for future World and Regional Congresses have been accepted by the Trustees and these recommendations are reflected in these columns. The Regional Vice Presidents of the WFN will need to do their homework and initiate discussions with the National Neurological Associations of the region to fulfill the aspirations and aims of the WFN to educate and train general practitioners in the all round good care of neurologically sick patients. The Zambian Project stands as a shining example of the WFN fulfilling its mission and this needs to be replicated in other regions of the world.

The results of the Neurosonology Research Group of the WFN in acute ischemic stroke are available in this issue. Daniel Truong has also updated our knowledge about the Genetics of Parkinson’s Disease. Both write-ups are excellent, meaningful and reflect modern thinking.

The World Health Organization conducted a meeting at its headquarters in Geneva from 30th October to 1st November 2002 on Secondary Prevention of Coronary Heart Disease and Stroke in underdeveloped countries. I was invited to participate in this meeting which was mainly for evaluating recommendations on these lines. Exhaustive discussions were held, especially on the cost effectiveness of the recommendations. The participants included neurologists, cardiologists, epidemiologists and WHO officials. The recommendations are likely to be released by WHO during the next year. Stroke is devastating with considerable mortality and morbidity, and its prevention is one of the burning topics of community medicine. However, the dilemma of those who suffer from it is manifold. Secondary prevention after the first stroke, which may even be TIA, is of fundamental importance and WHO is concerned about it.

A recent report in the British Medical Journal by French scientists is revealing. The

(Distribution of WN – cont. from p. 1)

It would also be helpful to the Federation if members could supply current e-mail addresses when responding, together with the website address of their national neurological society, so that our records may be kept up-to-date. Finally, please indicate whether or not you wish to receive advertising information.

Editor-in-Chief
Based on this experience, some trustees expressed concern about the strict British rules, particularly the need for an AGM. We also wondered whether the UK’s regulations allow us the flexibility we prefer as an international organization. Some see no point in asking people to travel long distances, at great expense, for a few hours of a passive meeting in these days of modern communication. We have, however, opted to give a little more time to test the new Articles of Association, which are only one year old. Besides, they have a number of features we would like to exploit, such as: tax exemption, fundraising and protection of the WFN from liability. Also, most clauses, intentionally written 'loose' without specifics, allow us to conduct day to day operation according to the policy manual that can be readily modified based on our changing needs. We can establish a quorum (of 15) for an AGM without much difficulty and, we believe that the use of modern communication could facilitate meaningful contribution from those delegates not personally attending the meeting. We will continue to pursue possibilities to circumvent the requirement of face-to-face AGMs, exploiting other means such as electronic interactions.

The lack of delegates’ participation is an inherent problem of all international organizations, which usually have a Management Committee, composed of a small group of executive members, who run most business with limited consultation from the member societies. The WFN also operates with seven elected and co-opted trustees who have an overall control and final say on most aspects of ongoing activities, consulting primarily with standing and ad hoc committees (Constitution & Bye-Laws, Education, Finance, Long Range Planning, Membership, Nominating, Public Relations & WHO Liaison, Publications & Website, Research, Structure & Function, and WCN Liaison). Of these, the Research Committee comprises some 30 research groups, each of which represents various disciplines of neurology. We are now exploring various means to entice more active involvement of national delegations representing their countries. Otherwise, I am afraid that we will face a dilemma, which will continue to challenge us – not just the hope of getting enough delegates at an AGM, but the more fundamental issue of realizing WFN’s mission – not just the hope of getting enough delegates at an AGM, but the more fundamental issue of realizing WFN’s mission to improve care for neurological patients globally through education of physicians. Unless we devise a mechanism for active, meaningful delegate participation, we will not be able to build the organizational strength we seek.

The WFN, with some 90 member societies scattered around the world, cannot possibly operate like national or regional societies such as the American Academy of Neurology, composed of individual memberships in one nation or, the European Federation of Neurological Societies (EFNS) that consists of relatively homogeneous and closely located countries. Thus, we do have to use a very different tactic to unite our front to achieve our objectives. We must further explore various avenues such as a website and newsletters in addition to our official publications, Journal of the Neurological Sciences and World Neurology, to find out how best to establish a better liaison between the trustees and delegates from national member societies. We should develop a worldwide e-mail database so that we can directly send information. The trustees take this challenge under serious consideration and discuss various options in our monthly teleconferences in the hope that we may improve the preparation for the next COD meeting in Sydney, which is scheduled for July 6, 2003. We are open to any suggestions to enhance participation of our member societies and would appreciate it very much if you take time to write me or other trustees in this regard.

To improve the international relationship, I try to discuss WFN matters with as many colleagues as possible during national and regional conferences of neurology, which I have the privilege to attend. I was pleased to participate in the 20th Brazilian Congress of Neurology. 20th Brazilian Congress of Neurology. From left to right: Dr. Ylmar Correa Neto, Secretary of the Congress, Prof. Luiz Alberto Bacheschi, President of the Brazilian Academy of Neurology, Prof. Paulo Sa, President of the Congress, and Andre Santos, Treasurer of the Congress.

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Editor-in-Chief
WFN ELECTIONS: NOMINATING COMMITTEE RECOMMENDATIONS

The Nominating Committee of the World Federation of Neurology, having invited nominations for the single Elected Trustee post falling vacant in 2003, recommends to the membership through their representatives on the Council of Delegates those listed below as candidates in accordance with the Federation’s Memorandum and Articles of Association. The current holder, Dr. William Carroll, is eligible to stand for re-election.

Recommended candidates:

Dr. William Carroll (Australia), Prof. Alla Guekht (Russia), Prof. Marco Medina (Honduras), Prof. Donald Silberberg (USA), Prof. Imre Szirmai (Hungary).

It is open to anyone to make additional nominations with the permission of the individual(s) concerned by

• Securing the supporting signatures of five or more authorized delegates;

• Submitting the name(s) of the individual(s) in question to the Secretary-Treasurer General, c/o the London Secretariat office, at least thirty days prior to the AGM (the date of which is 6 July 2003).

FUTURE WORLD AND REGIONAL CONGRESSES

The Long Range Planning Committee of the WFN has recommended, and the Trustees have accepted, a proposal that (i) the World Congress of Neurology should continue to take place every 4 years (2005, 2009, 2013, etc.), but that (ii) in the intervening years (2007, 2011, 2015, etc.) at 4 yearly intervals the Federation should also offer its support to a geographic regional organization or national society to hold a joint geographic Regional Congress on the basis of combined planning and a sharing of any profits realized.

There would be no obligation to take up the offer and regional organizations and national societies would of course be free to arrange their own independent meeting in the same year. The aim of a joint WFN Regional Congress would be to: a) help poorer regions that might have great difficulty in hosting meetings b) allow for an academic meeting to be held free of all the usual additional events with their cost impact c) highlight topics of particular concern to the region in question; and d) perhaps secure the involvement of WHO.

The Regional Congress

The new arrangements will therefore commence with the venue for a WFN Regional Congress.

GLAXOSMITHKLINE JUNIOR TRAVELLING FELLOWSHIPS – 2003

GlaxoSmithKline are again generously providing funding for a number of Junior Travelling Fellowships - each worth up to £1,000 - for young neurologists from developing countries to attend WFN approved congresses in 2003. Applicants should hold a post not above that of Associate Professor and should not be over the age of 42 years. Applications (one clear copy of CV, a letter of recommendation from Head of Department, a covering letter giving name and date of congress for which travel funds are sought and whether the applicant is presenting a paper or poster, plus an estimate of expenses) must be sent to the WFN London Office to arrive by 7th March 2003.

Bids are now invited from regional organizations or national societies within that region, particularly in Latin America, Africa or the Pan-Arab group of countries. They should be discussed initially with the WFN Regional Vice President who will refer them to the Trustees, c/o the WFN London Office by 28th February 2003 for voting by the COD at the meeting in Sydney on 6th July 2003. Bids should be supported by full details of amenities and facilities available.

Current Regional Vice Presidents are:
- Asian-Oceanian - Dr Jin Soo Kim, South Korea
- Pan-European - Professor Leontino Battistin, Italy (leontino.battistin@unipd.it)
- Pan-African - Prof Najoua Miladi, Tunisia (miladi.mg@planet.tn)
- Pan-American - Prof Carlos Chouza, Uruguay (chouza@hc.edu.uy)
- Pan-Arab - Prof Saleh Al Deeb, Saudi Arabia (rkhnsksa@zajil.net)

REGIONAL REPORTS

WFN Project Zambia

For most developing countries for which data are available, neurologists are literally “one in a million” [1]. In Zambia, the prevalence of treatable, yet untreated neurological disease is high. The most common disorders include seizures (especially epilepsy and febrile seizures), peripheral neuropathies and infections of the central nervous system, and 10 per cent of hospital admissions may be due to neurological disease. Furthermore, stroke-related mortality exceeds 50 per cent, and neurological patients account for 30 per cent of all ICU bed days [2].

Many Zambians suffering from neurological disease do not have access to physicians for geographical, cultural and financial reasons, and since 1994, Government policies have pushed for the care of such patients, by primary health care workers. However, without any provision for additional neurological training for these workers, and with no Zambian neurologists either, the need for those with the knowledge to share it with those who do not is vital to improve the quality of medical care and education in Zambia.

To this end, Dr Gretchen Birbeck of Michigan State University, has organized the Zambia Project, one of the educational activities of the World Federation of Neurology. This project is in its infancy, but is intended to be ongoing and expanding in scope. Dr Birbeck visited Zambia as a medical student in 1994, and with subsequent visits as a Resident and Fellow, her interest and appreciation of the problems of neurological care in Zambia have grown. Where There Is No Neurologist is a practical manual she has produced targeted especially at community health care workers. Using links with the University of Zambia Medical School, Chainama College and Hospital of Health Sciences, and community-based projects, the Zambia Project bases Visiting Professors for a minimum of one month at Chainama College, Lusaka.

The first of such visits took place between August and October 2002, when Dr Enrique Wulff, a Venezuelan neurologist with wide-ranging experience in both clinical work and teaching, spent a very busy six weeks in Zambia. He succeeded in delivering an extremely varied program that included lecturing to medical and nursing students, postgraduates and staff, on clinical and adult paediatric neurology, neurological problems during pregnancy, clinical neurology, and neuropharmacology. He gave academic presentations on peripheral neuropathies, HIV/AIDS, and cerebrovascular disease, made clinical rounds, and held outpatient clinics. In addition, Dr Wulff participated in radio and television programs on cerebral palsy, mental retardation, and learning disorders.

Since much care of neurological patients takes place in the community and the prevalence of epilepsy is high, part of Dr Wulff’s program also involved meeting epilepsy sufferers and their families to re-evaluate their treatment, and making home visits in the surrounding communities with the Cheshire Home Lusaka Rehabilitation Program. Seventeen of the assistants at this program benefited from a four-hour intensive course on Epilepsy.

In addition, Dr Wulff was able to establish links for several Zambian doctors who had an interest in neurology, with colleagues in the United States who have similar interests. Links such as these are positively encouraged, and hopefully, will lead not only to an increase in Zambian neurological education and ultimately to an improvement in medical care, but also to a greater appreciation of each other’s neurological situations. The next visiting professor, Alex Moll from Victoria, British Columbia, leaves for Zambia in January, and a full program of activities is planned on similar lines. As Dr Wulff says, the health care workers in Zambia provide the care they know to the best of their ability, and it is the Zambia Project’s objective to help them improve the quality of the neurological care they can give.

WFN NEUROLOGY TRAINING PROGRAM – HONDURAS

The Honduras Neurology Training Program at the National Autonomous University of Honduras has the honour to announce the Graduation of the first neurologists in our country: Dr. Reyna Duron, Dr. Heike Hesse, Dr. Lazaro Molina and Dr. Humberto Su. This program has been supported by the Honduras Ministry of Health, the National Autonomous University of Honduras, the Honduras Association of Neurology (WFN chapter) and the Education Committee of the World Federation of Neurology.

Marco T. Medina, MD, Professor of Neurology, Chairman Neurology Training Program, National Autonomous University of Honduras

Asian Oceanian Myology Center
This is to inform you that the 2nd annual conference and scientific meeting of AOMC (Asian Oceanian Myology Center) which brought together neuromuscular experts and neuroscientists not only from Asia and Oceania but also the rest of the world to exchange their knowledge on myopathies and muscle channelopathies with particular reference to gene therapy, was held in Beijing from 17th to 19th Sept. 2002. Prof. Jun Kimura, WFN President, delivered a comprehensive lecture at the opening ceremony of the function and highlighted his role in promoting this new organization. It was a great pleasure for me to present the only scientific paper from India on this occasion. The executive body was of the opinion that, after Tokyo and Beijing, the next venue for AOMC would be New Delhi.

G.P. Burman


The European Society for Clinical Neuropharmacology held its 6th Congress in Budapest, Hungary, on October 24-25, 2002. The scientific organizer was Prof. Laszlo Vecsei, Secretary of the ESCNP and also of the Danube Symposium for Neurological Sciences and Continuing Education. The general theme was Neurodegeneration and Neuroprotection and it was divided in various Sessions regarding Stroke, Parkinson’s disease, Epilepsy, Demyelinating disorders, and Dementia.

There were two main lectures, one by Prof. Franz Gerstenbrand (Austria) on Neuroethics in Clinical Neuropharmacology and the other by Prof. Peter Riederer (Germany) on Changing the paradigm: from transmitter substitution to inhibition of protein aggregation.

A very special event was the tribute paid to Prof. Abel Lajtha (USA) on the occasion of his 80th birthday by Prof. Sylvester E. Vizi, President of the Hungarian Academy of Science, and Prof. Leonotto Battistin, President of the ESCNP; both remarked on the very outstanding personality of Prof. Lajtha, born in Budapest and living in New York, as well as on his great achievements in neuroscience.

Finally, it was decided that the 7th Congress of the ESCNP will take place on May 5–9, 2004, in Trieste, Italy.

Prof. L. Battistin

THE NAIS – NEUROSONOLOGY IN ACUTE ISCHEMIC STROKE – STUDY

A Project of the WFN Neurosonology Research Group

The NAIS Study Group

Background

The pathophysiology, determining the extension or progression of ischemic damage in the first hours after stroke, is very complex. It is unlikely that specific medications such as thrombolytic agents will have the same effect in different stroke subgroups. The current evidence does not indicate which patient, with stroke, will profit from thrombolysis or identify specific conditions favouring anticoagulation. Therefore there is a need for a diagnostic modality, which characterises subtypes of stroke, not only by severity or localisation, but by criteria for treatment based on vascular imaging and hemodynamics. The methods to be used in acute stroke should be easily available and cost-effective as well as applicable to patients who are unable to cooperate fully. In addition such tests should not expose the patient to any risk. It should also be repeatable at short intervals and possibly at the bedside in order to avoid transport of patients who require close monitoring. Diagnostic ultrasound meets many of these conditions. Furthermore it is a technique that can be used in highly developed as well as in developing countries. Stroke is a major cause of disability in all countries and societies. However, the role of diagnostic ultrasound in acute stroke has only been evaluated in a few studies with a small sample size performed by highly specialized groups [Zanette et al. 1989, Ringelstein et al. 1992, Seidel et al. 1995, Goertler et al. 1998, Alexandrov et al. 1999, Allendoerfer et al. 1999]. The NSRG will bring together researchers from different countries in order to evaluate the impact of modern diagnostic ultrasound in a large number of patients.

Aims and Purpose

The study was designed to answer the question, whether extracranial and transcranial Doppler and duplex sonography, in addition to routine clinical evaluation on admission, adds information on the prognosis of patients with an acute ischaemic stroke. Its primary aim is to determine whether the functional outcome at three months after a moderate to severe acute ischaemic stroke, in the territory of the middle cerebral artery, differs in patients who exhibit an occlusion of the middle cer-

WORLD CONGRESS OF NEUROLOGY 2009

With planning for the 2005 World Congress in Sydney underway only comparatively recently, it may seem far too soon to be thinking about the one that follows in 2009. Nevertheless, member societies that may be interested in bidding to host WCN 2009 should be giving some preliminary thought to their strategy. The Trustees hope that at least some countries will be able to set out their preliminary ideas at the Council of Delegates meeting in Sydney on 6th July 2003, adding a further reason for as many Delegates as possible to attend so that they may be informed about what lies ahead. At the time of writing, expressions of interest have been received from Brazil, Italy, Mexico and Thailand. As for previous Congresses, countries will be able to formulate their proposals right up to the 2005 Congress where full presentations, including videos etc., will be on the agenda of the Council of Delegates meeting held at that time. Any country wishing now to signal its wish to be listed as a possible candidate for the venue of the World Congress in 2009, should communicate by e-mail, fax or letter to the WFN London Office.

Visit the WFN website at http://www.wfneurology.org
ebral artery (main stem or branches) as demonstrated by transcranial ultrasound within 6 hours of symptom onset, in comparison to those with an open artery. Secondary objectives are 1) to determine the clinical outcome in patients with internal carotid artery occlusion or severe stenosis and reduced middle cerebral artery blood flow velocity in comparison to those with a normal flow velocity, 2) to determine the influence of early (6 hours after symptom onset) or late (6 to 12 hours) recanalization of an initially occluded middle cerebral artery on clinical outcome, 3) to determine the effect of the course of arterial blood pressure within the first 6 hours in patients initially presenting with middle cerebral artery occlusion, and 4) to further support the hypothesis that patients initially presenting with a patent middle cerebral artery within 6 hours of symptom onset may not benefit from intravenous thrombolysis.

**Design**

Eligible patients are men and women aged over 18 years with a clinical and CT based diagnosis of an ischaemic middle cerebral artery territory stroke of more than 30 minutes duration and a measurable neurological deficit on the National Institutes of Health Stroke Scale (NIH-SS), in whom extracranial and transcranial ultrasound can be performed within 6 hours of the onset of symptoms.

To prove the hypothesis of the primary aim only patients suffering from a moderate or severe acute ischemic stroke (NIH-Scale >5 to <25) will be compared. Patients are excluded if they have a pre-existing clinical neurological deficit potentially interfering with standardised assessment of the acute ischaemic deficit, have an insufficient temporal bone window preventing bilateral transcranial sonography, or if sonography would delay immediate thrombolysis performed in accordance with local standards. Neurological deficit and functional disability is determined at admission and on day 7 by the NIH-SS score, the modified Rankin Scale (mRS), and the Barthel Index (BI). Disability is re-evaluated 90 days after stroke by telephone interview with the patient and/or a relative (mRS, BI). For primary and secondary outcome analysis the 90-day outcome will be dichotomised into: mild disability, total independence and not requiring help in the activities of daily living (mRS 0 to 2), to varying degrees of dependence (mRS 3 to 6) or death. Sonographic examination is performed at admission on all study patients. Those with occlusion or severe stenosis of the internal carotid or middle cerebral artery on the side of the symptomatic hemisphere will be re-examined 6 hours and 12 (to 24) hours after stroke onset. In patients who were initially examined, prior to 3 hours after stroke, additional sonographic re-examination will be performed at 3 hours. Ultrasound examination includes Doppler sonography of the orbital/supraorbital arteries, colour-coded duplex sonography with Doppler spectrum registration from the common carotid and extracranial internal carotid artery, and transcranial Doppler or colour-coded duplex sonography with spectrum registration from the intracranial internal carotid, middle cerebral (M1/M2 segment), anterior cerebral (A1 segment), and posterior cerebral artery (P1 or P2 segment), performed bilaterally for each artery. Contrast enhancing agents are recommended in any case where there is insufficient signal intensity of one or both middle cerebral arteries, especially in case of suspected occlusion.

**Study course**

By May 2002, 15 participating centres had enrolled 340 patients. It is estimated by the end of 2002, we will have the inclusion of the calculated sample of 400 patients.

**References**


J. Allendörfer, G.-M. von Reutern for the NAIS study group

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**WFN FELLOWSHIP REPORTS**

I am extremely grateful to the WFN for supporting and funding me for the European Neuro-Trauma Congress in Newcastle-upon-Tyne, June 26th-29th, 2002. This enabled me to interact with international faculty and learn the perspectives on Neurotrauma prevailing worldwide. Especially as this is a very common problem in our country.

I presented my paper on traumatic CV Junction Anomalies: Experience of 110 cases over 10 years. This paper invoked a lot of interest and allowed me to take opinion from other faculty on the management of the same.

Dr. P. Sarat Chandra

India

As a result of being awarded by the World Federation of Neurology Travelling Fellowship, two Cuban young physicians, Dr. Pedro Calderon and Dr. Alexander Roussaux had the marvellous opportunity of attending the American Academy of Electrodiagnostic Medicine Meeting that was held in Toronto, October 9th to 12th. The Fairmont Royal York Hotel was the excel-
lent place chosen by the organizers to hold the meeting.

During the event, many experienced professors throughout the world presented interesting topics as courses, seminars, workshops, roundtables, special interest groups, poster sessions and videos at the videotapes learning centers. The Cuban doctors attended sessions during the event for about 20 CME credits. Of particular interest for their work were the “Entrapment Neuropathies”, the “Pelvic Floor”, the “Neurophysiological effects of aging” and the “Plexuopathies” sessions.

As an important part of the meeting, the referred physicians attended the Exhibiting Hall Hours, where the most outstanding and upgraded of the EDX technology could be found. Several top companies in EDX medicine exhibited the results of their work there.

On Saturday 12th, at the final Poster Session, Drs. Calderon and Rousseaux presented their work entitled “EEG Findings in Myotonic Dystrophy of Steinert”, which had to do with an alternative but interesting and useful tool for studying the Central Nervous System involvement in this disease.

A wonderful and developed city, one of the prettiest water falls anywhere in the world, an ancient and luxurious hotel with the presence of Her Majesty the Queen during these days, a really well-organized event with all details covered and with a very high professional standard and the tempting invitation to attend the next AAEM meeting in San Francisco next year can be summarized as the main results of this important WFN award.

Thank you very much to the Organizing Committee of the AAEM Meeting and especially to the WFN for such an opportunity.

Dr. Pedro L. Calderon
Clinical Neurophysiology Dept.
University Hospital and
Cuban Institute of Neurology and
Neurosurgery

Dr. Alexander Rousseaux
Clinical Neurophysiology Dept.
University Hospital “Caluxto Garcia”

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In pursuit of its primary mission to improve human health world-wide by promoting the prevention of neurological disease and the care of those afflicted with neurological disorders, the WFN through its Neurology Training Programme (NTP), is making support available for those countries in greatest need of assistance i.e. those designated by the World Bank as having “low income” or “middle income” economies. Emphasis will be placed on training programmes. The specific goal of the programme is to produce clinical neurologists of the highest calibre.

Eligibility

The programme is designed to support projects that result in the improved training of neurologists. Examples of projects which qualify are:

1. Resident or fellow support
2. Purchase of educational material
3. Purchase of equipment necessary to train residents adequately
4. Assistance in establishing a new neurology training programme in an area of need
5. Salary support for resident training outside of the home country

Applications

Applications may be made by Training Programme Directors or similar administrative officials. There is no application form. A brief letter of intent (1-2 pages) – providing a general description of the training programme, major personnel involved, rationale for request and potential benefits for the programme and country – should be sent to the WFN London Office. These will be reviewed and ranked by a specially appointed sub-committee of the Education Committee. Those programmes deemed worthy of further consideration will then be referred back to the applicant for a more detailed proposal to be submitted. Final award decisions will be made by the WFN Trustees.

Terms of Award

Awards to successful institutions will be for periods of 1-3 years. Yearly progress reports will be required.

AMERICAN ACADEMY OF NEUROLOGY: NEWS RELEASE

Medical Groups agree: resources for treating Alzheimer's patients are available but underutilized

St. Paul, MN (December 4, 2002) — Care of patients with Alzheimer's disease is a challenging task but can be improved, according to seven medical organizations that met to discuss strengths and potential pitfalls in the diagnosis and treatment of Alzheimer's disease.

meeting recently in Chicago, the American Academy of Neurology, Alzheimer's Asso-
to provide tools to help more physicians diagnose and treat people dealing with this devastating disease,” said Catherine Rydell, executive director and CEO of the American Academy of Neurology. Representatives of the seven organizations emphasized the following important principles in caring for patients with Alzheimer’s disease:

• Alzheimer’s disease is recognizable and can be differentiated from normal aging by clinicians; symptoms are usually first identified by family members and should be reported to their doctors.
• Alzheimer’s disease can be diagnosed as reliably as appendicitis.
• Effective care options exist and can improve quality of life for patients and their caregivers.
• Adequate resources and reimbursement for ongoing dementia care in outpatient, home care and long-term care settings are critical to ensure access to quality care.
• Resources exist in the community for people with dementia and their caregivers.

One conclusion drawn from this research is that Parkinson’s disease is a multifactorial condition that may be caused by environmental events, genetic mutations, or an interaction of the two. Advances in the genetics of Parkinson’s disease have proceeded especially rapidly over the past few years with the identification of mutations in several cellular proteins, all of which may involve the appropriate clearing of damaged or misfolded proteins from the cell.

The first mutation to be associated with Parkinson’s disease was in the gene for α-synuclein, a protein located in presynaptic nerve terminals and a major component of Lewy bodies. Identified mutations in the α-synuclein gene lead to a clinical picture of disease distinguished from the idiopathic form by earlier onset and more rapid progression, but similar in responsiveness to L-dopa and the deposition of Lewy bodies (Gasser, 2001). The normal function of α-synuclein is still under investigation, but it has been linked to synaptic plasticity and regulation of vesicular dopamine release in preclinical studies.

Under certain conditions, the secondary structure of α-synuclein may be altered, causing the protein to form misfolded aggregates. It is possible that these misfolded proteins eventually become toxic to the cell. In several animal models, incorporation of a mutant α-synuclein protein or overexpression of the wild type resulted in parkinsonian symptoms.

A recent finding suggests that dopamine itself promotes the in vitro stabilization of α-synuclein in its protofibrillar form (Conway et al, 2001). This is an important finding because α-synuclein is not specific to dopaminergic neurons and thus the selective accumulation of α-synuclein-rich Lewy bodies in these cells and their eventual death have been perplexing. Although intracellular dopamine is largely confined to synaptic vesicles, the authors speculate that newly synthesized dopamine that has not yet been sequestered could be involved in this stabilization.

Two of the other mutations that have been identified in inherited forms of Parkinson’s disease involve the ubiquitin-proteasome pathway. The ubiquitin system is designed to remove damaged or unfolded proteins from the cell. In this system, a series of ubiquitin molecules are conjugated to damaged proteins, which marks them for degradation by the 26S proteasome. If the damaged proteins are not removed, they may form aggregates that could lead to cell death (Shastry, 2001).

Parkin is a cytosolic and membrane-associated protein that functions as a ubiquitin ligase. A mutation in the parkin gene has been identified that leads to L-dopa responsive parkinsonism with diurnal fluctuations, increased tendon reflexes in the lower limbs, severe dyskinesia, and occasional dystonia (Gasser, 2001). Although this mutation promotes the degeneration of nigral dopamine neurons, no Lewy bodies are present. A mutation in the parkin gene could interfere with ubiquitination and removal of undesirable proteins, resulting in their build-up and eventual toxicity to the cell.

Another mutation that involves the ubiquitin pathway occurs in the ubiquitin carboxy-terminal hydrolase L1 gene (UCH-L1). The gene product is an enzyme that acts to produce ubiquitin monomers that can be

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**UPDATE ON THE GENETICS OF PARKINSON’S DISEASE**

Parkinson’s disease is a progressive movement disorder that affects approximately 1 million people in the United States and 1–2% of those over the age of 65. The hallmark features of the disease—bradykinesia, tremor, rigidity, and impaired balance—are believed to result from the selective degeneration of dopaminergic neurons in the pars compacta of the substantia nigra. The events that lead to the death of these neurons have been the subject of intense research for many decades.

One conclusion drawn from this research is that Parkinson’s disease is a multifactorial condition that may be caused by environmental events, genetic mutations, or an interaction of the two. Advances in the genetics of Parkinson’s disease have proceeded especially rapidly over the past few years with the identification of mutations in several cellular proteins, all of which may involve the appropriate clearing of damaged or misfolded proteins from the cell.

The first mutation to be associated with Parkinson’s disease was in the gene for α-synuclein, a protein located in presynaptic nerve terminals and a major component of Lewy bodies. Identified mutations in the α-synuclein gene lead to a clinical picture of disease distinguished from the idiopathic form by earlier onset and more rapid progression, but similar in responsiveness to L-dopa and the deposition of Lewy bodies (Gasser, 2001). The normal function of α-synuclein is still under investigation, but it has been linked to synaptic plasticity and regulation of vesicular dopamine release in preclinical studies.

Under certain conditions, the secondary structure of α-synuclein may be altered, causing the protein to form misfolded aggregates. It is possible that these misfolded proteins eventually become toxic to the cell. In several animal models, incorporation of a mutant α-synuclein protein or overexpression of the wild type resulted in parkinsonian symptoms.

A recent finding suggests that dopamine itself promotes the in vitro stabilization of α-synuclein in its protofibrillar form (Conway et al, 2001). This is an important finding because α-synuclein is not specific to dopaminergic neurons and thus the selective accumulation of α-synuclein-rich Lewy bodies in these cells and their eventual death have been perplexing. Although intracellular dopamine is largely confined to synaptic vesicles, the authors speculate that newly synthesized dopamine that has not yet been sequestered could be involved in this stabilization.

Two of the other mutations that have been identified in inherited forms of Parkinson’s disease involve the ubiquitin-proteasome pathway. The ubiquitin system is designed to remove damaged or unfolded proteins from the cell. In this system, a series of ubiquitin molecules are conjugated to damaged proteins, which marks them for degradation by the 26S proteasome. If the damaged proteins are not removed, they may form aggregates that could lead to cell death (Shastry, 2001).

Parkin is a cytosolic and membrane-associated protein that functions as a ubiquitin ligase. A mutation in the parkin gene has been identified that leads to L-dopa responsive parkinsonism with diurnal fluctuations, increased tendon reflexes in the lower limbs, severe dyskinesia, and occasional dystonia (Gasser, 2001). Although this mutation promotes the degeneration of nigral dopamine neurons, no Lewy bodies are present. A mutation in the parkin gene could interfere with ubiquitination and removal of undesirable proteins, resulting in their build-up and eventual toxicity to the cell.

Another mutation that involves the ubiquitin pathway occurs in the ubiquitin carboxy-terminal hydrolase L1 gene (UCH-L1). The gene product is an enzyme that acts to produce ubiquitin monomers that can be
A variety of additional genetic loci have been linked to Parkinson’s disease. The PARK3 locus is associated with inherited Parkinson’s disease that shows a similar age of onset and symptomatology as idiopathic Parkinson’s disease, with some affected individuals developing dementia and Alzheimer-like plaques (Gasser, 2001). The PARK4 locus is also associated with disease similar to the idiopathic form, but with a younger onset, more rapid progression, early weight loss, dementia, and vacuoles in the hippocampus (Gasser, 2001). An interesting observation that has emerged from these genetic findings is that individuals with the same mutation may show somewhat different clinical phenotypes and vice versa. For instance, several individuals with the PARK4 locus developed postural tremor as their only symptom (Gasser, 2001). It has also long been recognized that despite commonalities in motor symptoms across individuals and clinical response to L-dopa, the age of disease onset and inheritance pattern (e.g., autosomal dominant, autosomal recessive, etc.) of Parkinson’s disease is highly variable. These genetic findings help explain the observed heterogeneity.

All of the aforementioned mutations and genetic loci described thus far are quite rare. The vast majority of individuals with Parkinson’s disease lack the characterized mutations, and their disease shows no clear inheritance pattern. However, the variety of different genes associated with inherited forms of the disease suggests that many more loci have yet to be identified.

One of the primary risk factors for idiopathic Parkinson’s disease is age, leading many to postulate an interaction between susceptibility genes and environment. Alternate theories have been proposed with regard to this interaction. Some have suggested that cellular damage accumulates over time, which leads to the prediction that the probability of cell death should increase over time (Clarke et al., 2000). However, research across a number of different neurodegenerative conditions including Parkinson’s disease suggests that the risk of cell death is more likely to be constant or decrease exponentially with age (Clarke et al., 2000). The authors indicate that their findings may be best accounted for by a ‘one-hit’ model in which a genetic mutation causes damage to the neuron making it vulnerable to a single event that results in cell death.

Despite the lack of association of idiopathic Parkinson’s disease with the aforementioned genetic mutations, the progress in identifying genes responsible for inherited forms of Parkinson’s disease has spurred new interest in seeking genetic loci in the idiopathic form. With the advent of more sophisticated genetic screening techniques, identification of multiple susceptibility genes in idiopathic Parkinson’s disease seems possible. This identification may then lead to gene therapies, although several such therapies may be required if the disease is associated with more than one gene - which seems likely.

Another challenge in Parkinson’s disease research is to determine the reason for selective degeneration of midbrain dopamine neurons. Because the proteins associated with inherited forms of the disease are not specific to dopamine neurons, other mechanisms must be at work. A variety of hypotheses have been suggested with regard to this specificity but conclusive evidence has yet to be presented (Mouradian, 2002). It appears a full understanding of Parkinson’s disease will require synthesis of information from a variety of different approaches that may include molecular biology, biochemistry, and epidemiology.

References

Daniel D. Truong, MD
Member WFN, Publications & Website Committee

This is a comprehensive book in the field of critical neurology and is very informative and has covered most of the recent developments for the investigations, diagnosis and treatment of acute neurological problems. The material is updated from the first edition. The book is divided in 3 parts containing 20 chapters. Part I comprises general clinical neurological problems in the Intensive Care Unit such as coma, seizures, generalized weakness and complications of invasive procedures. Part II deals with Neurological complications in medical and surgical intensive care units and transplantation units such as bacterial infections and sepsis, neurological complications of cardiac arrest, acid–base derangements, acute renal disease, hepatic failure, acute vasculitic syndromes, critically ill pregnant patients, aortic surgery, cardiac surgery, trauma and organ transplantation. Part III consists of a final 2 chapters on central nervous system catastrophes. Much of the material in this book is original collections from Mayo Clinic and is an effort of the single author who is well experienced in critical neurology. The book is useful for all disciplines of medicine, for a personal or institutional library.
### Myoclonus & Paroxysmal Dyskinesia (Advances in Neurology, Vol. 89)

**Editors:** Stanley Fahn, Steven J Frucht, Daniel Truong, Mark Hallett  
**ISBN:** 0-7817-3759-1  
**No. of pages:** 528  
**Price:** $169.00  
**Publication Date:** March 2002  
**Publisher:** Lippincott, Williams & Wilkins

This is volume 89 in the classic series of Advances in Neurology. The book is in 6 sections, the first dealing with myoclonus in its clinical features, the second on neurophysiology, the third on genetics and the fourth on pharmacology and therapies. The last two sections consider paroxysmal disorders in relationship to epilepsy and its genetics. This is a first class state-of-the-art review of two important topics and is highly recommended.

*F. Clifford Rose*  
London, U.K.

### Clinical Neuroanatomy and Related Neuroscience

**Editors:** M J T Fitzgerald and Jean Folan-Curran  
**ISBN:** 0702025585  
**No. of pages:** 336  
**Price:** £12.95  
**Publication Date:** 2002 (Fourth Edition)  
**Publishers:** W.B. Saunders

The fourth edition of this book is most illustrative and precise. There are some 327 illustrations, all in four colours and with descriptions. The edition has been totally rewritten and the artwork is new.

*F. Clifford Rose*  
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### Calendar

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**CALENDAR**

*Meeting endorsed by the Continuing Education Committee of the WFN*

**2003**

**23rd Annual Meeting of the Australian Neuroscience Society**  
Adelaide, SA, Australia  
Website: www.sallyjayconferences.com.au/ans/  
Joint Congress – Neurological Association of South Africa & the Association of British Neurologists  
29 January – 1 February, 2003  
Cape Town, South Africa

**Cancer Neurology in Clinical Practice**

**Editors:** David Schiff and Patrick Y. Wen  
**ISBN:** 0 86903-922-6  
**No. of Pages:** 464  
**Price:** $175.00  
**Publisher:** The Humana Press

Neuro-oncology has evolved as an important discipline under the neurosciences and this book will go a long way in the understanding and management of malignancies related to the nervous system. It is a multi-author book containing 31 chapters. A large number of the 50 authors are clinical neurologists. The book is divided into seven parts and covers almost everything relevant to neuro-oncology. Neurological symptoms, direct and indirect complications of cancer are discussed in parts II–IV. Part V covers the complications of cancer therapy and neuro-oncologic complications of specific malignancies are elaborated in Part VII specially related to lung, breast, genitourinary cancers, leukemia, Hodgkin’s and non-Hodgkin’s lymphomas, benign and malignant plasma cell dyscrasias, reproductive tract cancers, gastro-intestinal malignancies, sarcomas, head and neck cancer and children with systemic cancer. This book is of great interest for the practicing neurologist and neurosurgeon and is also of great value for radiotherapists and oncologists.

*F. Clifford Rose*  
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**Website:** [www.ucr.ac.za/depts/pgc/pna.html](http://www.ucr.ac.za/depts/pgc/pna.html)

**28th International Stroke Conference**  
13–15 February, 2003  
Phoenix, AZ, USA  
**Website:** [www.strokeconference.org/index.html](http://www.strokeconference.org/index.html)

**26th Annual Meeting of the American Society of Neuroimaging and 10th Meeting of the Neurosonology Research Group**  
5–9 March, 2003  
New Orleans, LA, USA  
**Website:** [www.asnweb.org/annual/index.htm](http://www.asnweb.org/annual/index.htm)

**6th International Conference on Stroke and 3rd Conference of the Mediterranean Stroke Society**

**12–15 March, 2003**  
Monte Carlo, Monaco  
**Website:** [www.kenes.com/stroke6/](http://www.kenes.com/stroke6/)

**22nd Annual Scientific Meeting of the American Pain Society**  
20–23 March, 2003  
Chicago, IL, USA  
**Website:** [www.ampainsoc.org](http://www.ampainsoc.org)

**55th Annual Meeting of the American Academy of Neurology**  
29 March – 5 April, 2003  
Honolulu, Hawaii, USA  
**Website:** [www.aan.com](http://www.aan.com)

**The Alzheimer Society of Canada’s 25th National Conference**  
10–13 April, 2003  
Ottawa, Ontario, Canada  
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