A *MOVEMENT* DISORDER SOCIETY UPDATE

Moving Along

VOLUME 5, ISSUE 2 • SPRING 2003 • EDITORS, DR. THOMAS GASSER, DR. IRENE LITVAN

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Editorial

This editorial will break with tradition in some respects. The authors believe it is time we step into the political arena and take our message to the public. After all, the cuts in research funding directly affect us. Times of a weak economy and growing political uncertainty are, of course, not without consequences for researchers and clinicians in all fields of medicine. Many of the members of our *Movement* Disorder Society (MDS) and readers of *Moving Along*, the official MDS newsletter, will have already experienced these consequences in their work environment. Public research funding agencies like the National Institutes of Health (NIH) in the USA or the German Research Foundation (Deutsche Forschungsgemeinschaft, DFG) in Germany have to deal with serious budget cuts, which directly translate into lower grant approval rates or funding. Economic woes are also putting an increasing strain on the publicly funded health systems in many European countries. In Germany or in the US, for example, many hospitals, even university hospitals of the highest standard, are forced to cut down on staff and to focus more and more on economic issues rather than excellence in medicine and medical science.


Irene Litvan, MD


Thomas Gasser, MD

On the other hand, scientific progress in recent years in many areas of medicine, but particularly in the field of Movement Disorders, has been breathtaking. In the controversy section of this issue of *Moving Along*, for example, possible therapeutic applications of recent genetic and cell biologic discoveries are being discussed. It is questioned whether aggregation of proteins (i.e., alpha-synuclein in the case of Parkinson's disease, PD, or tau, in progressive supranuclear palsy), which are thought to play a central role in the process of neuronal cell death, can be influenced therapeutically and whether such a therapeutic target would make sense within the framework of our present concepts of neurodegeneration. Another article discusses the recent development of deep brain stimulation (DBS) in severe dystonia. This technique provides astonishing therapeutic benefit in some patients with dystonia.

Scientists and clinicians worldwide are working tirelessly and enthusiastically to further advance science and to translate this progress into benefit for our patients. As a society, we question if our role should also be to bring up to public opinion the scientific progress being made so our communities can have more information when they are setting the priorities of available resources. How can we convey the enthusiasm placed in our work and results to the public? We would like to make this newsletter a forum for ideas and initiatives to bring our message to the public. Please send us your opinions, ideas and plans on how individual members or MDS as a whole can increase public awareness and support for our work.

Why do you belong to The *Movement* Disorder Society? It is my hope that you have joined this organization to learn and exchange knowledge in the realm of Movement Disorders. By taking an active role in The *Movement* Disorder Society (MDS) you can hear and be heard.

That's what more than 400 members did when they responded to the recent MDS Member Needs Assessment Survey. They were heard. Of the nearly 2,000 MDS members from 70 countries who received the electronic survey, 419 members replied — an impressive 25% response rate.

Here are some facts about the survey respondents:

- The majority of survey respondents are practicing physicians working out of academic institutions and/or hospitals.
- One quarter of respondents have been in practice for less than 5 years while another quarter have practiced more than 20 years. The 50% remaining, lie somewhere in between.
- The majority of respondents (80%) practice in either North America or Europe/Mediterranean geographic regions.
- Of the 75% male and 25% female respondents, the majority (84%) have access to High Speed Internet that would allow for on-line video viewing.
- On average, respondents report to participate in one to ten educational activities per year.

The MDS Education Committee, chaired by Dr. Cynthia Comella, is now in the final stages of compiling statistical summations. Next, they will analyze the data to extract respondents' educational format preferences and continuing medical education topic preferences, as well as the motivators and barriers to participation.

I want to personally thank those who took the time to respond to this important survey. Your perspective is imperative as we move forward to expand our repertoire of MDS's educational offerings throughout the world.

Educational Activities Prepare for Launch

One of the main goals of MDS's 2002-2005 Strategic Plan is to build a continuous, free-standing, education program to support physicians around the world in their treatment of patients living with Movement Disorders. MDS already has a reputation for offering the highest level of educational programming and publishing the pre-eminent clinical and research journal devoted to Movement Disorders.

Now we are setting forth on a new path that began with the MDS Members Needs Assessment Survey, and will soon offer high caliber CME activities which will come in a variety of learning formats, specifically designed to meet the needs of our international membership. These formats will involve both live activities and self-study alternatives. Computer based learning formats will likely be employed to generate enduring materials to reach an even wider target audience.

Dystonia Workshops Take an Active Approach

Also this year, the American Academy of Neurology (AAN) and The *Movement* Disorder Society will jointly sponsor two educational courses entitled, *Treatment of Dystonia: Workshops Demonstrating the Use of Botulinum Toxin.*

These workshops will offer a critical overview of the clinical spectrum, pathophysiology and treatment of dystonia, with an emphasis on botulinum toxin therapy. They will include small group and live demonstration sessions and will focus on patient assessment and botulinum toxin injection for dystonia. The first workshop will take place in Milwaukee, Wisconsin, USA on May 9, 2003. The second workshop will take place in Durham, North Carolina, USA, June 7, 2003.

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President's Letter

The MDS-European Section (MDS-ES) will conduct a similar workshop, *Botulinum Toxins in Neurological Practice*, at the Institute of Neurology, Queen Square, London, United Kingdom on November 7, 2003.

Sponsoring Global Meetings

The *Movement* Disorder Society is also continuing its tradition of sponsoring meetings in fields related to Movement Disorders. Traditionally offered in odd-numbered years when MDS is not organizing the International Congress of Parkinson's Disease and Movement Disorders, MDS leaders have revised the policy to offer sponsorships every year beginning in 2004. You'll find more information on how to apply for MDS sponsorship or endorsement inside this issue of *Moving Along*.

International Congress Slated for Rome 2004

Plans are also underway for the next Congress in Rome, Italy, June 13-17, 2004. As one of the world's most beautiful cities, Rome is sure to be an ideal venue for MDS's 8th Congress. The Palazzo dei Congressi, located in the modern EUR district, will serve as headquarters for all scientific sessions, posters and exhibits.

We are planning a superb scientific program, which more than 3,000 neurologists, basic scientists, and Movement Disorder specialists are expected to attend. In line with MDS tradition, the scientific program will prove to be informative, comprehensive and innovative, including plenary session lectures, platform presentations, abstract poster presentations, video dinners and courses. We'll look forward to keeping you apprised and seeing you in Rome in 2004!

Like you, our goal is to enhance patient care by advancing scientific and medical knowledge in the field of neurology and sub-specialty of Movement Disorders. By working together, it is possible for us to achieve this worthwhile goal. Thank you for your support and active participation in The *Movement* Disorder Society.

C. Warren Olanow
MDS President 2003-2004

MDS Committee Rosters 2003-2004

Awards Committee
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Strategy and Planning Committee
Chair - Mark Hallett, MD; C. Warren Olanow, MD; Werner Poewe, MD; Andrew J. Lees, MD

Eliminating the Alpha-synuclein Aggregates?

Is Eliminating the Alpha-synuclein Aggregates (Lewy Bodies) an Appropriate Target for Therapeutic Intervention? – Yes

—John Duda, MD, Co-Director, Parkinson's Disease Research, Education and Clinical Center, Philadelphia VA Medical Center, Philadelphia, PA, USA

The role that intracytoplasmic α-synuclein inclusions, including Lewy bodies (LBs) and Lewy neurites (LNs), play in the neurodegenerative process in Parkinson's disease and dementia with Lewy bodies remains controversial. Whether they are the primary insult that begins a cascade toward eventual neuronal death or a secondary adaptive response is debated. Nevertheless, blocking the formation of LBs and LNs should remain a target for developing novel therapeutic interventions. First, the formation of LBs and LNs represents an integral component of the neurodegenerative cascade that predisposes neurons to their eventual demise. Most current pathophysiologic hypotheses suggest a combination of environmental, genetic and intrinsic factors that engender biochemical alterations within neurons leading to the conversion of α-synuclein into a crossed beta-pleated sheet structure, oligomerization and formation of insoluble filaments that aggregate to form LBs and LNs. Admittedly, there is still an open 'chicken or the egg' debate with α-synuclein aggregation and other physiological disturbances including proteasomal and mitochondrial dysfunction, but until these relationships are definitively established, all aspects of the neurodegenerative cascade should remain targets for intervention. Indeed, recent evidence is painting an ever-clearer picture of the significance of LNs (insoluble aggregates of α-synuclein within axonal and dendritic processes) by recognizing that they are the seminal pathologic hallmark of these disorders [1,2] as well as a dominant feature of the pathological burden in the nigrostriatal system [3]. It is important to recognize that even if aggregate formation has evolved as an adaptive response and is protective early in the disease process, continued aggregation becomes detrimental when normal cellular trafficking or axonal transport is disrupted.

> *"...there is no conclusive evidence that preventing α-synuclein aggregate formation leads to detrimental consequences..."*

In addition, there is no conclusive evidence that preventing α-synuclein aggregate formation leads to detrimental consequences by halting the neurodegenerative cascade at a previous step. Hypotheses that blocking synuclein aggregation would lead to a proliferation of a toxic 'protofibrillar' species[4] remain speculative. Further, an examination of the neurodegenerative process induced by genetic mutations that are thought to primarily affect proteolytic processing (i.e. mutations in Parkin that cause a comparatively well treatable form of parkinsonism with prolonged survival) would argue in favor of blocking α-synuclein aggregation even if alterations in proteasomal dysfunction were unremedied.

Finally, several groups have recently developed reproducible models of α-synuclein aggregation in rodents (Reviewed in Ref. 5) and drosophila [5,6] to test these interventions. These models have already led to important observations that may further our understanding and treatment of human disease including a possible role for chaperones [5] or β-synuclein [7] as therapeutic interventions and the specific role of axonal degeneration in neurodegeneration [8]. These newer animal models more closely resemble the age-dependant, chronic nature of human diseases than previous models including MPTP or 6-hydroxydopamine administration, and will become crucial in testing future therapeutic interventions aimed at the underlying mechanisms of neurodegenerative disease. In summary, eliminating α-synuclein aggregation remains an attractive target for therapeutic intervention not only due to the advent of reliable tools to assess the effects of doing so, but also because of accumulating evidence supporting a pathogenic role, particularly for neuritic aggregates.

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Eliminating the Alpha-synuclein Aggregates?

Is Eliminating the Alpha-synuclein Aggregates (Lewy Bodies) an Appropriate Target for Therapeutic Intervention? – No

—-Kevin McNaught, PhD, Department of Neurology, Mount Sinai School of Medicine, New York, NY, USA

The primary pathology of Pakinson's disease (PD) is degeneration of dopaminergic neurons in the substantia nigra pars compacta (SNc). Neuronal death is accompanied by the appearance of intracytoplasmic protein aggregates known as Lewy bodies. These inclusions were discovered over 90 years ago, but the mechanism underlying their formation and relevance to the neurodegenerative process have been unclear. While earlier studies raised the possibility that Lewy bodies might be toxic to neurons in which they occur, later investigations argue against this concept[1,2], and more recent studies suggest that Lewy bodies are formed, organized and serve a cytoprotective role in an aggresome-like manner[3-5]. Thus, elimination of Lewy body formation is not an appropriate therapeutic target in PD.

"...more recent studies suggest that Lewy bodies are formed, organized and serve a cytoprotective role in an aggresome-like manner."

Proteolytic stress is a state in which the levels of unwanted proteins (e.g. mutant and oxidatively damaged proteins) exceed the capacity for degradation and clearance within cells. Under such conditions, excess levels of undegraded or partially degraded proteins can aggregate, interfere with intracellular activities and induce cell death. In recent years, it has become increasingly evident that cells have a second line of defense to control unwanted proteins[6]. This begins with the active transport of undegraded or poorly degraded proteins with ubiquitin-proteasome system (UPS) elements, heat shock proteins (HSP), and other proteolytic elements, to the centrosome. These components are compartmentalized by a cage of intermediate filaments to form a proteinaceous structure designated and aggresome[6]. Aggresomes serve to segregate undegraded and poorly degraded proteins so as to avoid contact with organelles and critical molecules, and enhance the degradation of unwanted proteins. This process has been implicated in the formation of proteinaceous inclusion bodies in cells and tissues under a variety of pathological conditions[6].

There is increasing evidence that proteolytic stress occurs in and causes/contributes to degeneration of nigral dopaminergic cells in familial and sporadic PD. Recent studies have also demonstrated a striking similarity between the protein composition and organization of Lewy bodies and aggresomes[3]. Indeed, Lewy bodies display an aggresome-like distribution of the centrosome-specific markers (γ-tubulin and pericentrin), UPS components and HSP[3]. These observations have led to the suggestion that Lewy body formation is an aggresome-related response to proteolytic stress and attempts to control excess levels of abnormal and potentially cytotoxic proteins in PD. The effectiveness of this process is being investigated, but it may be sufficient to underscore the delayed and slowly progressive nature of neurodegeneration in sporadic PD. This contrasts with parkin-linked autosomal recessive juvenile parkinsonism in which the early and severe destruction of the SNc and locus coeruleus are associated with the absence of Lewy bodies. Indeed, the occurrence of Lewy bodies with minor dopaminergic cell death in the SNc of 10-15% of neurologically normal individuals over the age of 65 (incidental Lewy body disease), raises the possibility that the formation of these inclusions could be effective in preventing nigral cell death to the extent that is required (> 70%) for the onset of PD symptomology.

Thus, Lewy body formation appears to be a cytoprotective event aimed at combating proteolytic stress in PD. Consequently, elimination of this process cannot be of therapeutic value. Indeed, experimental inhibition of aggresome/inclusion body formation is associated with earlier and more severe death of cells undergoing proteolytic stress *in vitro* and *in vivo*[4,5]. Rather, we suggest that therapeutic strategies aimed at facilitating the formation and/or proteolytic activity of Lewy bodies would serve to enhance the clearance of abnormal proteins and provide neuroprotection in both familial and sporadic PD.

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MDS Prepares for its 8th International Congress in Rome, Italy

The *Movement* Disorder Society will convene its 8th International Congress of Parkinson's Disease and Movement Disorders from June 13-17, 2004 in the magnificent city of Rome, Italy. Held at the Palazzo dei Congressi, the MDS Congress will offer a unique opportunity to gain a global perspective through scientific presentations offered by experts from more than 45 different countries.

The 2004 Scientific Program Committee is preparing an informative program filled with a variety of educational formats covering the latest advances in Parkinson's disease, spasticity, dystonia, genetics, basic science and numerous additional topics that will appeal to an expected audience of over 3,000 clinicians, researchers, post-doctoral fellows and medical students interested in the current research and approaches for the treatment of Movement Disorders.

Participants will be encouraged to submit abstracts relevant to Movement Disorders for peer-review. Accepted abstracts will be eligible for poster presentation at the Congress. A Call for Abstracts will be issued with the Preliminary Program this summer. Electronic submissions will be encouraged and no fee will be charged for abstract submission.

The Palazzo dei Congressi will also host Congress exhibition for pharmaceutical and biotechnology companies, medical publishers and patient service providers to present information on the newest products and services available in the industry.

As one of the world's most historic, yet modern cities, Rome will surely appeal to all participants and their guests. The Congress Organizing Committee is planning outstanding social events and optional tours to entertain attendees and highlight all the area has to offer including world-renowned museums, ancient monuments, excellent cuisine, and shopping.

The Congress Preliminary Program will contain the scientific program, hotel and Congress registration forms, social event and optional tour listings, transportation information and additional details to help plan your Congress participation.

For more information on the MDS Congress, please contact:

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Transgenic Animal Models of Parkinsonian Disorders

— Philipp J. Kahle, PhD, Laboratory of Alzheimer's and Parkinson's Disease Research, Department of Biochemistry, Ludwig Maximilians University of Munich, Germany

Ten genetic loci have been linked to parkinsonism to date. The most common cause of hereditary parkinsonism are loss-of-function mutations in the parkin gene. Parkin deficiency is thought to promote selective degeneration of dopaminergic (DA) neurons in the substantia nigra, leading to the parkinsonian symptoms. In contrast to the autosomal-recessive juvenile parkinsonism linked to parkin, point mutations in the α-synuclein (αSYN) gene cause autosomal-dominant hereditary Parkinson's disease (PD). Although familial PD caused by αSYN mutations is exceedingly rare, this protein is of relevance to all PD in general, because Lewy bodies, the hallmark lesions of PD, are intracellular aggregates consisting primarily of αSYN fibrils. Moreover, the glial cytoplasmic inclusions which are diagnostic for multiple system atrophy are also composed of αSYN fibrils. Recent research efforts concentrated on the generation of transgenic animal models based on the over-expression of the human PD gene product αSYN.

Transgenic expression of human αSYN in *Drosophila* neurons successfully recapitulated the formation of αSYN fibrils *in vivo*[2]. DA neurons were particularly vulnerable to α-synucleinopathy, and age-dependent degeneration of DA neurons correlated with progressive locomotor deterioration of the flies[2]. The relative ease to develop a *Drosophila* model of α-synucleinopathy might be due to the fact that invertebrate genomes do not code for any synucleins. Thus, flies may not have evolved cellular defense mechanisms to suppress αSYN fibrillization. Indeed, the *Drosophila* phenotype could be rescued by gene transfer of the molecular chaperone Hsp70[1], pointing to the cellular protein folding machinery as a therapeutic target.

Pathology in early mouse models was mainly characterized by somatodendritic accumulation of granular, detergent-insoluble transgenic αSYN, and some mouse lines showed locomotor impairment[4]. More recently, three independent mouse models were presented which accurately reflect human Lewy pathology down to the molecular and ultrastructural level[3, 6, 8]. Regardless of the promoter (Thy1 vs. PrP) or αSYN mutation (A53T vs. A30P) employed, these mouse models commonly showed age-dependent formation of amyloid-like αSYN fibrils within neuronal perikarya (Lewy bodies) and dystrophic neurites. These mouse models developed a progressive locomotor phenotype characterized by dystonia, rigidity, paralysis, and ultimately death. Neurodegeneration was evident from the massive gliosis predominantly in the brainstem and spinal cord. Interestingly, however, the DA neurons were not impaired in these chronic mouse models of Lewy pathology. It required delivery of high αSYN gene doses directly into the substantia nigra of adult rats to confer adverse effects to the DA neurons. This was achieved by viral gene transfer using adeno-associated virus[5] or lentivirus[7]. However, in these acute gene delivery models, Lewy pathology did not develop over time. Rather, the virally delivered αSYN precipitated into granular aggregates. Thus, a complete rodent model of PD would combine the slow, orderly formation of αSYN fibrils and DA neuron loss. The defence mechanisms which protect DA neurons throughout the relatively short life span of mice may also be effective in humans. Such cellular defence mechanisms might lose activity in the elderly, ultimately leading to PD in affected patients. The molecular understanding of these protective mechanisms will open novel avenues for innovative therapeutic approaches of PD and related diseases.

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The Competence Network Parkinson in Germany

—Wolfgang Oertel, MD, Neurologische Klinik, Universität Marburg, Marburg, Germany

The Competence Network Parkinson is a German collaborative research effort supported by the BMBF (Federal Ministry of Education and Research) since September 1999. In October 2002, the network received further support for a second two-year funding period. The major goals of this network are the improvement of patient care and the optimization and coordination of research. By setting up this network, we attempt to create an effective interface between research and healthcare.

The network structure is composed of three interconnected parts: the central office located in Marburg, the "vertical" and the "horizontal" net. The *vertical* network consists of university hospitals, city hospitals, neurologists in private practice, general practitioners, rehabilitation institutions and industry. The aim of the vertical network is to achieve an exchange of expertise and knowledge between these levels of health care. The horizontal net encompasses collaborative research projects which deal with different topics regarding basic science, diagnosis, therapy and public health in Parkinson´s disease (PD).

In the first funding period, an electronic, entirely internet-based network for general communication as well as for the capture, storage, and handling of relevant project data at a maximum level of data safety and security (remote electronic data capture) was established. A uniform standard for patient documentation and follow-up was set up to register PD, multiple system atrophy, progressive supranuclear palsy patients and also healthy controls and tissue donors. All patients registered in this patient database may take part in other studies and projects of the network. An infrastructure for clinical studies was established to design and conduct investigator-initiated multicenter trials. The network runs a repository for biological materials (the German PD DNA-bank, GEPARD) and cooperates closely with the German "BrainNet", which is dedicated to post-mortem tissue collection. Network projects of the "horizontal" net include working groups on deep brain stimulation, neuroimaging, and clinical epidemiology as well as the development and validation of a questionnaire for early and differential diagnosis of PD. The first prospective pharmacoeconomic investigation on direct, indirect and intangible costs of therapy in PD including assessment of quality of life was conducted within the network.

With the internet-based standardized documentation of patient and research data, the German Competence Network Parkinson has created an effective interface between research and healthcare. This network also serves as a prototype for a European network ('EuroPa'), which is presently being developed. In the future, concepts have to be found which will secure a sustainability of the Competence Network in terms of a financially independent enterprise.

Call for Applications for Sponsored Meetings

The *Movement* Disorder Society is now accepting applications from meeting organizers who wish to receive MDS sponsorship for scientific meetings in the year 2004 and 2005.

Applications are available on the MDS Web site at www.movementdisordersociety.org or from the International Secretariat by contacting Gail Bast, Executive Director at + 1 414-276-2145 or by e-mail at gbast@movementdisorders.org.

The deadline for applications for 2004 meetings is June 15, 2003.
The deadline for applications for 2005 meetings is March 15, 2004.

Sponsorship requests should be e-mailed to gbast@movementdisorders.org or faxed to +1 414-276-3349.

All completed applications will be referred to the Education Committee, which will make recommendations to the International Executive Committee (IEC). The IEC will announce those meetings selected as MDS-Sponsored meetings for 2004 in July of 2003.

MDS-European Section Initiates New Activities

— Eduardo Tolosa, MD, Chairman, MDS-European Section

New Botulinum Toxins Workshop to be Launched

Following the success of the Dystonia workshops in the USA organized by The *Movement* Disorder Society in collaboration with the American Academy of Neurology, an MDS-European Section (MDS-ES) workshop on the use of Botulinum Toxins in Neurological Practice will be held at the Institute of Neurology, Queen Square, London, on November 7, 2003.

An international faculty, led by Dr. Kailash Bhatia, will give presentations on the use of botulinum toxins in a variety of neurological indications. Delegates will break into small groups for the afternoon session of practical demonstrations.

For further information, and to register for the course (numbers will be limited to 40 participants) please contact the MDS-European Section at: khenley@movementdisorders.org; Tel: +44 208 293 8515; Fax: +44 208 355 9632.

Dopamine Transporter Imaging Workshop Deemed a Success

Dopamine Transporter (DAT) Imaging was the topic of a European Section workshop organized in January 2003 by Professor Andrew Lees. An invited faculty of European Neurologists and Nuclear Medicine Experts convened in London to discuss key issues in the use and positioning of (DAT) imaging. The role of (DAT) imaging in the diagnosis and staging of Parkinson's disease, its use in diagnosing atypical tremors, and in the differential diagnosis of dementias was discussed. The value of the technique in research trials, and the cost versus health benefits in clinical practice were reviewed. Manuscripts of the presentations will be published as a supplement to *Movement* Disorders later this year. The workshop was supported by an unrestricted educational grant from Amersham International.

Professor Wolfgang Oertel, Secretary, and Professor Eduardo Tolosa, Chairman, in London for the Dopamine Transporter Imaging Workshop, January 2003.

Professor Andrew Lees and Professor Fabrizio Stocchi review case studies at the Dopamine Transporter Imaging Workshop.

European Federation of Neurological Societies Collaborates with MDS-ES

The MDS-ES organized the Movement Disorders curriculum at the European Federation of Neurological Societies (EFNS) Congress in Vienna, October 2002. This first EFNS Congress under a new three-year collaborative agreement was a great success, with all Movement Disorders sessions well-attended, and in some sessions there was standing room only for latecomers. Professor Mark Hallett presented the popular invited lecture at the European Basal Ganglia Club meeting. MDS-ES also held its first Section Business Meeting in Vienna.

MDS - European Section

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We thank Professor Andrew Lees for his visionary leadership over the first two years of the European Section, and the retiring Officers and members of the Executive Committee for their support and work on behalf of our European members.

Deep Brain Stimulation and Dystonia

—Joachim K. Krauss, MD, Department of Neurosurgery, University Hospital, Klinikum Mannheim, Mannheim, Germany

Deep brain stimulation (DBS) for treatment of medically-refractory dystonia has been introduced only a few years ago (Kumar et al., 1999; Krauss et al., 1999; Coubes et al., 2000). Since then, several studies with promising results have been published, but we have also become aware of some of the limitations of this new therapeutic option for treating dystonia. The increased interest in DBS for dystonia was reflected by the number of abstracts on this topic at The *Movement* Disorder Society's International Congress in Miami in November, 2002. The major advantage of DBS as compared to radiofrequency lesioning is that it allows performing contemporaneous bilateral surgery with relatively low morbidity in these patients. The posteroventral lateral globus pallidus internus (GPi) has become the preferred target in most instances of dystonia, thus far. It has been demonstrated that while phasic dystonic movements may improve early after surgery, the response of tonic dystonic postures to chronic stimulation may be delayed. The most beneficial results have been achieved in patients with primary genetic generalized and segmental dystonia, complex cervical dystonia, and myoclonic dystonia. Outcome has been more varied, however, in patients with other dystonic Movement Disorders, in particular those with secondary dystonia.

From a methodological point of view, there are several limitations to most studies on DBS for dystonia published thus far. These pilot studies have involved relatively short follow-up periods, some of them were retrospective or they included only a small number of patients. Therefore, it is important to validate the observations made within the frame of larger prospective study protocols. Such studies are being conducted presently in France and Germany, and another study is being planned in the UK. The French SPiDY multicenter study investigates the effect of bilateral GPi DBS in primary adult generalized dystonia including blinded assessment of clinical outcome. Preliminary results have confirmed the results from the earlier pilot studies. A German multicenter study investigates the long-term outcome of pallidal DBS for primary generalized and segmental dystonia using a randomized double-blind protocol. With regard to the preliminary experience made in patients with other dystonic Movement Disorders which were refractory to medical treatment, it appears justified and worthwhile to explore new possible indications for DBS including tardive dystonia, paroxysmal dystonia, and blepharospasm-oromandibular dystonia and other focal or segmental dystonias. Thalamic DBS may play a role in patients with secondary dystonia.

The mechanisms of DBS in dystonia remain elusive. The fact that pallidal DBS is beneficial both in hypokinetic and in hyperkinetic Movement Disorders is puzzling. It has been assumed that its crucial mechanisms rather involve the disruption of pathologic patterned neuronal activity than the net reduction of pallidal neuronal firing. This hypothesis is substantiated by recent studies on field potentials of the basal ganglia through the implanted electrodes. A multicenter study led by Peter Brown with participating centers in Berlin, Rome, Oxford, Amsterdam, and Mannheim indeed showed differences in pallidal local field potentials between patients with Parkinson's disease and dystonia.

It is good news to know that there is progress in the application for a CE mark for DBS for treatment of dystonia. Last summer, officials from Medtronic met with Philippe Coubes, Tipu Aziz and Joachim K. Krauss in Paris to review the current status of DBS for dystonia. Based on this review, representatives from Medtronic are preparing a dossier for submission to European authorities to request CE mark for DBS for dystonia. During the Paris meeting, other methodological and technical issues were also discussed. The costs of DBS for dystonia are higher in the long run as compared with PD owing chiefly to the higher energy consumption of the battery for chronic stimulation and the younger age of the patients. Therefore, it will be important to consider new algorithms for chronic stimulation.

DBS most likely will become the mainstay in the surgical treatment of dystonia in the near future. Perspectives of DBS for treatment of dystonia include the evaluation of the possible role of different targets, the exploration of new indications, the development of new technology, and the evaluation of carefully planned and conducted studies.

Do your colleagues know about MDS?

In 2002, MDS membership grew to 1,620 international members, with representation in 70 countries! This is yet another outstanding membership increase, indicating a strong future for MDS with continued growth and prosperity.

Please continue to encourage your colleagues to join MDS, so they may share knowledge, promote research and guide public policy with fellow members committed to the study and treatment of Movement Disorders.

Meetings

Psychogenic Movement Disorders Workshop

Psychogenic Movement Disorders represent an important diagnostic and therapeutic challenge yet there is little known about this group of disorders. In fact, psychogenic disorders are a significant problem in all of neurology and all of medicine.

In an attempt to deal with the challenge of Psychogenic Movement Disorders, an organizing committee consisting of academic neurologists and psychiatrists was formed with the primary aim to organize a workshop that would serve as a forum for a review of current concepts and to stimulate new testable hypotheses. The objective of the conference is to bring together neurologists, psychiatrists, psychologists, and neuroscientists and to generate ideas for future research in this long-neglected field. This will be a unique meeting, the proceedings of which will be published in a book or a journal supplement.

Abstracts are being accepted for poster presentation at the workshop. A maximum of 140 posters will be accepted. Accepted abstracts will be published in the journal, *Movement* Disorders. The abstract deadline is May 30, 2003.

The workshop is sponsored by The *Movement* Disorder Society and will take place at the Aberdeen Woods Conference Center in Atlanta, Georgia on October 10-13, 2003. If you would like additional information about attending the Psychogenic Movement Disorder Workshop or the abstract process, please visit the MDS Web site, www.movementdisorders.org., or call Jenny Kehoe at +1 414-276-2145.

Atypical Parkinsonian Disorders Meeting, Innsbruck, Austria, February 19-21, 2003

— Gregor Wenning, MD, PhD, Universitäts Klinik für Neurologie, Innsbruck, Austria

This international meeting on atypical parkinsonian disorders including MSA, DLB, PSP, CBD and atypical parkinsonism in isolates was recently held at the University Department of Neurology in Innsbruck with the generous support of The *Movement* Disorder Society. A kick-off seminar explored the role of DAT and dopamine receptor SPECT imaging in parkinsonism.

Oleh Hornykiewicz received the honorary MDS membership award at the beginning of the scientific sessions. The second day provided a mix of state of the art talks concerning the clinical diagnosis of MSA (Quinn, London), DLB (McKeith, Newcastle), PSP and CBD (Tolosa, Barcelona) as well as basic research updates regarding pathogenesis of alpha synucleinopathies (incl. Spillantini, Cambridge; Kahle, Munich) and tauopathies (incl. Goedert, Cambridge; Hutton, Jacksonville). The third day focused on current and future therapeutic strategies in atypical parkinsonism (Wenning, MSA; Poewe, DLB; Lang, PSP/CBD, Litvan, future approaches). Albert Ludolph, Ulm, Germany, reviewed the NNIPPS trial that investigates the action of riluzole in MSA and PSP. The meeting closed with an exciting session on the pacific isolates including parkinsonism-dementia complex (Steele, Guam), as well as atypical parkinsonism on Guadeloupe (Caparros, French West Indies) and in Japan (Kuzuhara).

During the meeting there were two guided poster sessions with approximately sixty posters covering a wide range of clinical research. Due to the excellent faculty including clinicians and basic scientists, the meeting attracted more than 200 attendants from almost 30 countries. Most participants thoroughly enjoyed the selection of basic and clinical presentations. It is hoped that within the next decade, there will be a steady flow of new candidate agents that might arrest the inexorable decline of parkinsonism in the atypical disorders.

The 4th Alpine Basal Ganglia Club Symposium on Movement Disorders

— Professor Maja Relja, MD, PhD, Chairman, Organizing Committee

Plitvice Lakes National Park, Croatia

The 4th Alpine Basal Ganglia Club Symposium on Movement Disorders was held in Croatia, from September 18-21, 2002 at the UNESCO protected Plitvice Lakes National Park, considered one of the most beautiful national parks in the world. This symposium has become a traditional symposium of specialists in the field of Movement Disorders from central Europe, but also from other countries as well.

The first symposium was held in Italy (Tarvisio), the next in Slovenia (Ljubljana) and the third in

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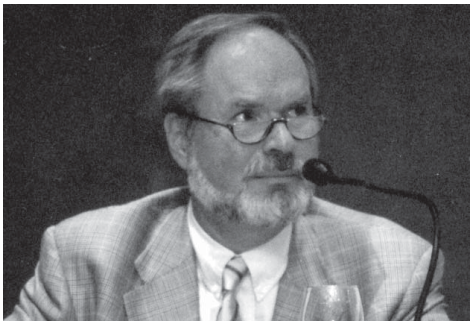
Attendees of the 4th Alpine Basal Ganglia Club Symposium on Movement Disorders, Croatia

Austria (Innsbruck). From the very beginning, the symposium was planned as a combination of pleasure and usefulness, with the aim to maintain close friendships in smaller environments in order to transfer interaction and exchange opinions from congress halls into a more relaxed atmosphere. The symposium at Plitvice Lakes surpassed the assigned framework and significantly broadened the number of participants with invited speakers from Finland, Germany, United Kingdom and Israel, achieving a markedly higher number of contributors.

Organizing this year's symposium was the Croatian Association of Patients with Movement Disorders, founded at the end of 2001 with the aim to help the development of this branch of neurology in Croatia. The introduction to the symposium on the first day was a gathering of patients, physicians, and association members gathered to see the study results found in the first Croatian study organized by the Association dedicated to dopaminergic agonists in the treatment of Parkinson's disease. Besides Professor Maja Relja, Professor Zvezdan Pirtošek from Slovenia participated in this part of the meeting as well.

The first plenary session of the Symposium was an Update on Movement Disorders, dedicated to various topics like: Gait dynamics in Movement Disorders (Nir Giladi, Tel Aviv), Dystonia today (Zvezdan Pirtošek, Ljubljana), Update in progressive supranuclear palsy (Carlo Colosimo, Rome), Huntington's disease: recent findings (Lazslo Vecsei, Szeged), and Pathophysiology and treatment of psychosis in Parkinson's disease (Erwin Ott, Graz).

The first day also included presentations on pharmacological investigations under the title "Selected topics in neuropharmacology." Afternoon poster sessions and traditional video presentations were held, as well.



Dr. Peter Riederer speaks at the 4th Alpine Basal Ganglia Club Symposium on Movement Disorders.

The second day started with the invited lectures entitled: Medical management of advanced Parkinson's disease (Werner Poewe, Innsbruck) and Dopamine agonists in the prevention and treatment: primate studies (Peter Jenner, London). The second plenary session was dedicated to Parkinson's disease. Thomas Gasser presented the newest data on genetic research in Parkinson's disease; Peter Riederer spoke on pathobiochemical and molecular mechanisms of neurodegeneration; Antonioni discussed levodopa and dopaminergic agonists, or both, from the aspect of evidence in clinical practice; Kanovsky presented subcutaneous apomorphine in the treatment of late Parkinson's disease complication; and Jech explained brain stimulation in Parkinson's disease.

The second day also included a symposium on COMT inhibitors with guests from Finland. Werner Poewe lectured on entacapone and evidence based medicine, and Ariel Gordin presented the effects of COMT inhibitors on levodopa pharmacokinetics. Maja Relja presented the first Croatian-Finnish study on entacapone. The day ended with the traditional dinner in a local restaurant "Lika House Daniela" with typical food and the renowned Croatian wines.

The last day of the symposium began with the invited lecture of David Brooks: "The developing role of imaging in Parkinson's disease diagnosis and treatment." According to the opinion of many colleagues, it was one of his best lectures. The Symposium closed with the workshop "Botulinum toxin as drug: new indications" with special review of new indications, particularly pain, hyperhydrosis and spasticity.

The organizers are particularly grateful to the invited lecturers who contributed greatly to the high scientific level of the symposium, which according to many, was one of better meetings from the field of Movement Disorders held in 2002.

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The symposium combined a high scientific level with informal gathering, and for many it also meant the discovery of, until then, an unknown part of Croatia.

The organizers are also particularly thankful to all Symposium participants. It was a significant impetus for strengthening the profession in Croatia, as well as a stimulus for further Association work. We are grateful to all contributing to the meeting and look forward to the next ABGC symposium to be held in the Czech Republic.

Frontotemporal Dementia and Pick's Disease Conference

— Dr. Andrew Kertesz, Professor of Neurology, University of Western Ontario, St. Joseph's Hospital, London, Ontario, Canada

A Conference on Frontotemporal Dementia and Pick's Disease was convened on September 13-15, 2002 in London, Ontario, Canada hosted by the Department of Clinical Neurological Sciences of the University of Western Ontario. Thirty invited speakers and 100 interested participants discussed the terminology, clinical and laboratory diagnosis, neuropathology, biochemistry, molecular biology, animal models, epidemiology, and treatment of this condition. For the first time, this Frontotemporal Dementia and Pick's Disease Conference included panels of experts in Movement Disorders and motor neuron disease, since these features are becoming increasingly recognized in the disease. Frontotemporal dementia was first described by Arnold Pick and subsequently named Pick's disease, but the eponymic term is now restricted to cases with the histological finding of characteristic silver staining inclusions (Pick bodies). The clinical disease without such distinct pathology was renamed Frontotemporal Dementia without distinctive histopathology. Frontotemporal Dementia (FTD) and Primary Progressive Aphasia (PPA) are the clinical terms used when the pathology is unknown. One of the neuropathologic varieties described by Tissot and Constantidinis in their series would be currently considered as corticobasal degeneration. However, corticobasal degeneration was considered as a separate entity until recently when the relationship to FTD was recognized.

The clinical diagnosis of FTD is improved by using behavioral inventories aimed at the personality and behavior change that defines the syndrome. The frequency of progressive aphasia, and the overlap of the behavioral and language symptoms provide a rationale for language tests. Screening tests of dementia, such as the MiniMental Status Examination or the Mattis Dementia Rating Scale are not sufficient to distinguish between AD and FTD.

A substantial number of CBD and PSP patients have a behavioral disorder (apathy, depression, or executive dysfunction). The panelists, Drs. Boeve, Lang, and Litvan felt the clinical corticobasal syndrome (CBS) needs to be differentiated from the underlying pathology (CBD) and the clinical and pathological distinctions between CBD and PSP should be maintained, but there is substantive overlap between these conditions and FTD.

CBD and PSP have a predominant 4 repeat (4R) tau and Pick' disease 3 repeat (3R) tau, but evidence presented at the conference suggests there is considerable overlap between these two disorders. There is also the most interesting finding that some of the tau negative cases (with or without moton neuron disease type inclusions) have been found to have deficiency of normal tau, therefore they could be considered tauopathies as well. Genetic studies indicated that many of the phenotypes can be seen with single common mutations such as the P301L and several different mutations can also cause the same phenotypic variant.

Low prevalence on national surveys is thought to be related to under diagnosis. In dementia clinics the incidence of FTD is estimated to be 20 percent to 25 percent of the degenerative dementias. The participants were optimistic that future research will bring better recognition, understanding, and treatment in this condition.

New MDS Course

In response to the inadequate training of many health professionals in clinical research methodology as well as the unique challenges encountered in Movement Disorder clinical trials in particular, MDS is proud to offer a new course.

Developed and organized by Professors Cristina Sampaio and Olivier Rascol, the course is entitled "Design, conduct and interpretation of clinical trials in Movement Disorders." The course will provide both a conceptual and practical foundation for health care professionals involved in Movement Disorder clinical trials, including an overview of research plan structure, design options, sample size calculation, ethics, regulatory restrictions, and the role of the Principal Investigator. The course will be offered in Lisbon, Portugal, October 8–10, 2003.

More detailed information can be obtained from Sofia Mata: smata@fm.ul.pt or sofia.mata@sapo.pt.

Announcements

Michael J. Fox Foundation for Parkinson's Research

In 2002, only its second year in operation, the Michael J. Fox Foundation for Parkinson's Research (MJFF) more than tripled its direct funding efforts from the previous year. In addition to funding its *PD Cell Line* initiative, which was launched in 2001, they successfully launched and funded three new initiatives *Protein Degradation*, *Biomarkers*, and *Fast Track 2002*. These three new programs broaden the Foundation's already diverse research portfolio, which strategically targets high-impact areas where they can significantly advance the state of science and accelerate the discovery of a Parkinson's disease (PD) cure.

MJFF launched its *Protein Degradation* initiative in June 2002 and awarded 11 grants totaling over $2.6 million to researchers investigating the role of protein degradation in Parkinson's disease. A growing body of evidence suggests that degenerative diseases of the central nervous system are characterized by the over-accumulation of abnormal protein deposits. The Foundation hopes that the proactive study of why this potentially harmful aggregation of proteins occurs with a particular focus on the way they can be broken down and removed will lead to the development of neuroprotective therapies for PD patients.

In April 2002 MJFF launched its *Biomarker* program to address the absence of a definitive diagnostic test for PD. Currently clinical diagnosis is based on a patient's medical history and neurological examinations. The misdiagnosis rate is estimated to be as high as 25 percent. MJFF designed the initiative to encourage the development of multiple types of biomarkers, reviewing applications with sensitivity toward characteristics that would allow the biomarker to be easily reproduced and translated to clinical practice. The Foundation awarded $1.6M to fund eight projects.

The Foundation granted over $4M in its second annual *Fast Track* program, an investigator-initiated program designed to stimulate novel, innovative and high-impact approaches to the field of Parkinson's research. Twenty projects were funded, comprising a comprehensive portfolio of research topics including genetic, neuroprotective and restorative studies.

MJFF launched two new programs in early 2003. *The Role of Inflammation in Parkinson's disease* invited investigator-initiated grant applications to conduct research exploring the relationship between cellular inflammation and PD. Applications are currently under review, with awards expected in June.

The second program has committed $2M to the study of dyskinesias, the involuntary, uncontrollable movements that are a frequent side effect of PD treatment. Applications are due April 18, 2003 from neuroscientists and clinicians working in fields that could potentially impact the understanding of levodopa-induced dyskinesias and finding ways to prevent or ameliorate them.

For more information on these funding opportunities or to learn more about MJFF please visit their website at www.michaeljfox.org.

Research Awards

Funds up to $150,000 are available for support of research directly related to blepharospasm or Meige's syndrome. Deadline to apply for this year is July 1, 2003. M.D. or Ph.D. degree required for principle investigator. For proposal forms apply to: Benign Essential Blepharospasm Research Foundation, Inc., P.O. Box 12468, Beaumont, TX 77726-2468

Phone: (409) 832-0788
Fax: (409) 832-0890
E-mail: bebrf@ih2000.net
or visit our web site: www.blepharospasm.org

Task Force for the Development of Rating Scales for Parkinson's Disease

The second phase of the Task Force for the Development of Rating Scales for Parkinson's Disease, the critique of the Hoehn and Yahr scale, is well underway. Chaired by Christopher Goetz, Task Force members include, Werner Poewe, Olivier Rascol, Cristina Sampaio, and Glenn Stebbins.

A group of six expert consultants have been confirmed, including Nir Giladi, Robert Holloway, Charity Moore, Gregor Wenning, Carl Counsell, and Melvin Yahr. The expert consultants' critiques on the Hoehn and Yahr scale have been completed and circulated, and the state of the art review is now being prepared by Task Force members. The final draft will be submitted to the leadership of The *Movement* Disorder Society (MDS) for review, with plans to publish the final state of the art review in *Movement* Disorders in late 2003.

The Task Force for the Development of Rating Scales for Parkinson's Disease was initiated in the year 2001 by MDS.

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Collectively, the group is charged with critiquing existing Parkinson's disease rating scales and preparing an MDS state of the art review on selected scales.

The goals and objectives of the Task Force for the Development of Rating Scales for Parkinson's Disease are:

- Generate MDS state of the art reviews on existing scales used in Parkinson's disease evaluations for research and patient care.
- Organize and conduct MDS-sponsored initiatives to modify existing scales if changes are suggested in the position statements.
- Develop and test new scales for measurements of key elements of Parkinson's disease impairment for which existing scales do not exist.

The Task Force's first project was the critique of the Unified Parkinson's Disease Rating Scale (UPDRS). The panel of expert consultants for this project included Stanley Fahn, Anthony Lang, Pablo Martinez-Martin, Barbara Tilley, and Bob van Hilten.

Task Force members and expert consultants convened for an intense one-day meeting in Chicago, IL, USA, in which the written critiques were discussed. Following the meeting, Task Force members utilized the data to draft the state of the art review, which has been accepted for publication in *Movement* Disorders, the official journal of The *Movement* Disorder Society. You may view this article online in the EarlyView section of the Wiley InterScience web site, www.interscience.wiley.com/societies/mds. The results of the project were also presented at the American Academy of Neurology (AAN) annual meeting on April 3, 2003.

Upcoming Meetings

2003

May 30-June 3, 2003
15th International Congress on Parkinson's Disease. Beijing, China. Contact: XV International Congress on Parkinson's Disease; c/o International Convention Services; Chinese Medical Association; 42 Dongsi Xidajie; Beijing 100710, China; TEL: 86-10-6524-9989 ext. 2456; FAX: 86-10-6512-3754 / 6524-4086; E-mail: xvicpd@chinamed.com.cn

***June 7, 2003**
Treatment of Dystonia: Workshop Demonstrating the Use of Botulinum Toxin. Duke, NC USA. Contact: The *Movement* Disorder Society, 611 East Wells Street, Milwaukee, WI 53202; TEL: +1-414-276-2145; FAX: +1-414-276-3349; E-mail: info@movementdisorders.org; Web site: www.movementdisorders.org

June 14-18, 2003
13th Meeting of the European Neurological Society. Istanbul, Turkey. Web site: www.ensinfo.com

June 17-21, 2003
Canadian Congress of Neurological Sciences. Quebec City, Quebec, Canada. Contact: Canadian Congress of Neurological Sciences, P.O. Box 5456, Station A, Calgary, AB, T2H 1X8 Canada; TEL: +1-403-229-9544; FAX: +1-403-229-1661; E-mail: brains@ccns.org

July 10-15, 2003
6th IBRO World Congress of Neuroscience. Prague, Czech Republic. Contact: Secretariat, 6th IBRO World Congress of Neuroscience, Guarant LTD, Opletalova 15, 110 00, Prague 1, Czech Republic; TEL: 420-2-24-21-06-50; FAX: 420-2-24-21-21-03; E-mail: ibro2003@biomed.cas.cz

August 1-4, 2003
13th Annual Course A Comprehensive Review of Movement Disorders for the Clinical Practitioner Meeting, Hotel Jerome, Aspen, Colorado, USA. Contact: Center for Continuing Education, Columbia University College of Physicians & Surgeons, 630 West 168th Street, Unit 39, New York, NY 10032; TEL: +1-212-305-3334; FAX: +1-212-781-6047; E-mail: cme@columbia.edu; Web site: http://ColumbiaCME.org

August 3-8, 2003
Joint Meeting 2003 of the International Society for Neurochemistry (ISN) and the Asian-Pacific Society for Neurochemistry (APSN), Hong Kong Convention and Exhibition Centre, Hong Kong, China. E-mail: isn2003@icc.com.hk; Web site: www.neurochem.org/HongKong/ISN-APSNmeeting.htm

August 16-19, 2003
World Congress on Huntington Disease 2003, Toronto, Canada. Organized by the Research Group on HD and the WFN, the International Huntington Association, the Huntington Study Group and the Huntington Society of Canada. For more information contact: iha@huntington-assoc.com; Web site: www.hsc-ca.org

August 22 - 24, 2003
21st Annual International Conference of the Benign Essential Blepharospasm Research Foundation, (BEBRF) Inc. Crowne Plaza Hotel, Philadelphia, PA. Contact: BEBRF; Tel: +1-409-832-0788; E-mail: bebrf@ih2000.net

August 30 – September 3, 2003
7th European Federation of Neurological Societies Congress. Helsinki, Finland. Contact: EFNS, Neurological Hospital Rosenhugel, Riedelgass 5, A-1130, Vienna, Austria; TEL: 43-1-880-00-270; FAX: 43-1-88-92-581; E-mail: headoffice@efns.org

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September 16 – 20, 2003
27th International Congress of Clinical Neurophysiology/The 50th Anniversary of the American Association of Electrodiagnostic Medicine Annual Scientific Meeting. San Francisco, California. Contact: AAEM, 421 First Avenue SW, Suite 300E, Rochester, MN, 55902; TEL: +1-507-288-0100; FAX: +1-507-288-1225; E-mail: aaem@aaem.net

October 1-4, 2003
Child Neurology Society Annual Meeting. Miami Beach, FL, USA. Contact: Child Neurology Society, 1000 West County Road E, Suite 290, St. Paul, MN 55126; TEL: +1-651-486-9447; FAX: +1-651-486-9436; E-mail: nationaloffice@childneurologysociety.org; Web site: www. childneurologysociety.org

***October 8-10, 2003**
Design, Conduct and Interpretation of Clinical Trials in Movement Disorders. Lisbon, Portugal. Offered by The *Movement* Disorder Society. Contact: Sofia Mata, Faculdade de Medicina de Lisboa-Piso 4, Instituto de Farmacologia e Terapeutica Geral, Av. Prof. Egas Moniz, 1649-028 Lisboa, Portugal; TEL: +351 21 780 21 20; FAX: +351 21 780 21 29; E-mail: smata@fm.ul.pt or sofia.mata@sapo.pt

***October 10-13, 2003**
Psychogenic Movement Disorders. Aberdeen Woods Conference Center, Atlanta, GA, USA. Sponsored by The *Movement* Disorder Society. Contact: Meeting Organizer, Mark Hallett, MD, NINDS, NIH Bldg. 10, Rm. 5N226, Bethesda, MD, 20892-1428; TEL: +1-301-496-1561; FAX: +1-301-402-1007; E-mail: mark_hallett@nih.gov

October 18-23, 2003
Congress of Neurological Surgeons 53rd Annual Meeting. Colorado Convention Center, Denver, CO, USA. Contact: Congress of Neurological Surgeons, 10 North Martingale Road, Suite 190, Schaumburg, IL, USA, 60173; TEL: +1-847-240-2500; FAX: +1-847+240-0804; E-mail: info@1cns.org

October 19-22, 2003
128th Annual Meeting of the American Neurological Association. San Francisco, CA, USA. Contact: American Neurological Association, 5841 Cedar Lake Road, Suite 204, Minneapolis, MN 55416; TEL: +1-952-545-6284; FAX: +1-952-545-6073; E-mail: lorijanderson@msn.com

November 5-8, 2003
14th International Symposium on the Autonomic Nervous System. St. Thomas, U.S. Virgin Islands. Contact: Anita Zeller, AAS Executive Secretary, American Autonomic Society, 5458 193rd St. W., Farmington, MN 55024; TEL: +1-651-463-4119; FAX: +1-651-463-2009; E-mail: zeller.anita@mayo.edu; Web site: www.americanautonomicsociety.org

November 8-13, 2003
33rd Annual Meeting of the Society for Neuroscience. New Orleans, LA, USA. Contact: Jamie Swank, Society for Neuroscience, 11 Dupont Circle, N.W., Suite 500, Washington, D.C. 20036; TEL: +1-202-462-6688; FAX: +1-202-462-9740; E-mail: info@sfn.org; Web site: http://web.sfn.org

***November 21-22, 2003**
First Symposium on Paediatric Movement Disorders. Barcelona, Spain. Contact: Marta Pla, Suport Servicio, Calvet, 30.08021, Barcelona, Spain; TEL: +34 93 2017571; FAX: +34 93 2019789; E-mail: martapla@suportserveis.com

***December 3 - 6, 2003**
2nd Parkinson's Disease and Movement Disorders Symposium. National Neuroscience Institute, Singapore. Contact: Dr. Louis Tan, 11 Jalan Tan Tock Seng, Singapore 308433; TEL: 65-6357-7171; FAX: 65-6357-7137; E-mail: louis_tan@ttsh.com.sg; Web site: http://www.nni.com.sg

***December 6 - 7, 2003**
World Parkinson's Day International Symposium. Mumbai, India. Contact: Ms. Katie M. Vania; TEL: 91-22-206-8787; FAX: 91-22-283-6926; E-mail: ktvania@vsnl.com

2004

April 24-May 1, 2004
American Academy of Neurology 56th Annual Meeting. San Francisco, CA, USA. Contact: American Academy of Neurology, 1080 Montreal Avenue, St. Paul, MN 55116; TEL: +1-651-695-1940; E-mail: web@aan.com; Web site: www.aan.com

June 8-12, 2004
Canadian Congress of Neurological Sciences. Calgary, AB, Canada. Contact: Canadian Congress of Neurological Sciences, P.O. Box 5456, Station A, Calgary, AB, T2H 1X8 Canada; TEL: +1-403-229-9544; FAX: +1-403-229-1661; E-mail: brains@ccns.org

September 4 –9, 2004
8th European Federation of Neurological Societies Congress. Paris, France. Contact: EFNS, Neurological Hospital Rosenhugel, Riedelgass 5, A-1130, Vienna, Austria; TEL: 43-1-880-00-270; FAX: 43-1-88-92-581; E-mail: headoffice@efns.org

October 3-6, 2004
129th Annual Meeting of the American Neurological Association. The Sheraton Toronto, Toronto, ON, Canada. Contact: American Neurological Association, 5841 Cedar Lake Road, Suite 204, Minneapolis, MN 55416; TEL: +1-952-545-6284; FAX: +1-952-545-6073; E-mail: lorijanderson@msn.com; Web site: www.aneuroa.org

October 23-28, 2004
34th Annual Meeting of the Society for Neuroscience. San Diego, CA, USA. Contact: Society for Neuroscience, 11 Dupont Circle, N.W., Suite 500, Washington DC 20036; TEL: +1-202-462-6688; E-mail: info@sfn.org

October 24-27, 2004
Mental Dysfunctions in Parkinson's Disease. Salzburg, Austria. Contact: Mental Dysfunctions in Parkinson's Disease, Kenes International, 17 Rue du Cendrier, P.O. Box 1726, CH-1211, Geneva 1, Switzerland; TEL: 41-22-908-0488; FAX: 44-847-127-5678; E-mail: PDment2004@kenes.com; Web site: www.kenes.com/PDment2004

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