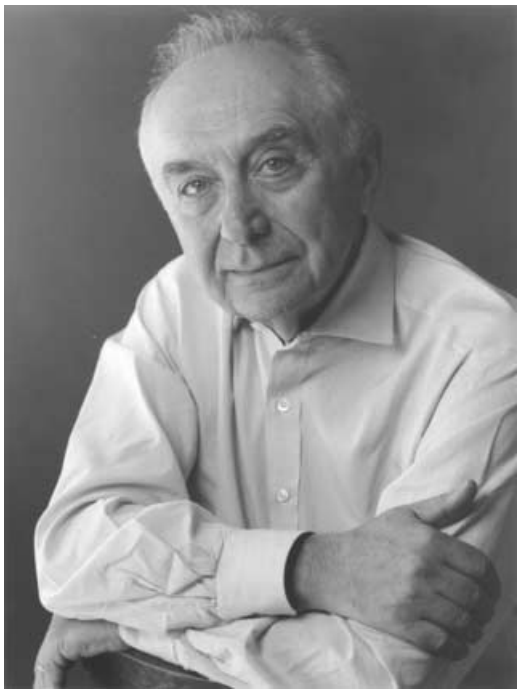
IN MEMORIAM



Mircea Steriade—August 20, 1924–April 14, 2006

In the early hours of April 14, 2006 Mircea Steriade died of lung cancer.

Mircea Steriade was born in Bucharest, Romania on August 20, 1924. He started his investigations of the nervous system during his first year of medical school (1945), and continued this work with relentless determination until a few months before his death. The only individual he ever recognized as a mentor, Frédéric Bremer, identified this feature of his personality, this intense striving, by calling him *l'infatigable Monsieur Steriade*. Mircea Steriade's long list of publications constitutes a vivid reflection of the exciting evolution of neurosciences through the second half of the twentieth century. He started his professional career as a researcher in the Laboratory of Neurophysiology at the Institute of Neurology in Bucharest, Romania. Then, after a short transition at the Université de Montréal, he established his own laboratory at the Université Laval in Quebec City, Canada. During the last two years of his life, he returned to Montréal, first on a sabbatical (his first sabbatical ever!) and then as a Visiting Professor.

Summarizing Mircea Steriade's career and outstanding contributions, as well as his passionate character, is difficult in a short article. Clearly, Professor Steriade will be remembered as one of the most productive neuroscientists of the last six decades. Most of his work was devoted to the understanding of thalamocortical networks and of their contribution to the genesis of brain oscillations – including epileptiform activities. He was an outstanding electrophysiologist and theorist. He made invaluable insights into the mechanisms of sleep oscillations (both physiological and pathological) and their modulation by brainstem activation systems. Many of our most influential and challenging ideas about the origins of spike-wave seizures, and about the contribution of intrinsic properties of cortical neurons to consciousness, were introduced by Mircea Steriade.

He had an unfettered belief that hard work is the main source of advancement in science. And he lived this philosophy. Professor Steriade arrived before anyone else in the laboratory, and served as an inspiring impulse for his students and younger colleagues. He remained youthful and vigorous in scientific debate, served especially well by his vast memory and encyclopedic knowledge of neuroscience. During his last years, seeing the rise of *in vitro* techniques in neuroscience, he fought passionately for the place of *in vivo* experimentation as an irreplaceable method for understanding the complex features of biological behaviors.

Through his leadership and laboratory contributions to experimental neuroscience and epilepsy, Mircea Steriade's career has touched generations of researchers. His strong personality and high standards will continue to influence the neuroscience and epilepsy research fields, through those who knew him (his many students and collaborators) and those inspired by his creative legacy.

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LETTERS/COMMENTARY

Bone Metabolism and Vitamin D Levels in Carbamazepine-treated Patients

To the Editor:

I read with great interest the paper by Mintzer et al. (1) about the vitamin D and bone turnover in epileptic patients receiving carbamazepine (CBZ) or oxcarbazepine (OXC) and found an increase in bone metabolism with low serum 25-hydroxyvitamin D (25-OHD).

In our experience (2,3), patients treated by CBZ monotherapy showed a significant increase of bone turnover: in fact, we found higher values of markers of bone formation (serum bone alkaline phosphatase, osteocalcin, and propetides of types I and III procollagen) and of bone resorption (serum telopeptide of type I collagen and urine *N*-telopeptides of type I collagen) in patients than in controls. These data confirm the results of Mintzer et al. (1) who demonstrated significant increase in the bone formation markers. It is possible that these abnormalities were secondary to induction of hepatic microsomal enzymes. In contrast with the results of these authors (1), in our patients we did not find any significant changes in serum 25-OHD levels.

There is a great debate about whether this increase in bone turnover is really due to decrease in 25-OHD. The absence of relationship between serum 25-OHD and serum concentration of CBZ shows that the increase of bone turnover can be independent of the effect of this antiepileptic drug on vitamin D metabolism, as previously suggested (4,5). Moreover, very recently, also Pack et al. (6) found numerous abnormalities in bone turnover markers with 25-OHD normal levels in their CBZ treated patients. Although biochemical changes in the metabolism of vitamin D are observed during treatment with CBZ, whether clinically apparent and/or histological osteomalacia develops during treatment with the drug is still controversial; in fact, no difference in bone mineral density between patients and controls has been reported. Therefore, we do not agree with the suggestion of Mintzer et al. (1) who encourage 25-OHD replacement in patients receiving this drug, because this preventive treatment could not be always indicated.

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Response: Bone Metabolism and Vitamin D Levels in Carbamazepine-treated Patients

To the Editor:

I thank Dr. Verrotti and his colleagues for their interest in our recent publication (1). It is always useful to engage in a dialogue with other investigators working in the same area. We appear to be in agreement that bone turnover is elevated in CBZ-treated patients, and I would also certainly agree with their statement that "there is a great debate about whether this increase in bone turnover is really due to decrease in 25-OHD." I must contend several of the points they raise, however.

First, I do not agree that the absence of a relationship between serum 25-OHD and CBZ level suggests that the drug does not affect 25-OHD. Since CBZ is known to be a rather potent inducer of cytochrome P450 enzymes, it is quite possible that even a relatively small dose of CBZ markedly induces the cytochrome P450 system, so that whether a patient is taking 5 or 15 mg/kg/day, the metabolic effects are the same. For example, even the paltry adult dose of 300 mg daily appears to be enough to markedly induce the metabolism of valproic acid (2). Using the logic of Verrotti and his colleagues one would conclude that CBZ does not affect bone turnover either, since there was no relationship between CBZ dose (or serum level) and any of the bone turnover markers in our study, nor in theirs (1,3).

Second, I disagree with their assessment of the study of Pack et al. (4). The CBZ-treated patients in that study had mean 25-OHD levels of 21 (normal range: 20–60). This was 30% lower than that in the group treated with the noninducing drug lamotrigine. These numbers are strikingly similar to the numbers obtained in the CBZ and control groups in our study. This is clearly a clinically

meaningful difference; in fact, about half the CBZ-treated patients in each of these studies were frankly 25-OHD deficient. We mentioned in our paper that 5 different studies have shown relative decreases in 25-OHD in CBZ-treated patients ranging from 12% to 42% (1,4–7). Another study, recently presented in abstract form, also demonstrated 25-OHD reduction of about 30% in CBZ-treated patients relative to controls (8). The fact that a difference of this magnitude did not quite reach significance in the study of Pack et al. is simply a function of statistical power; this too was mentioned in our discussion (1).

The real question is why the two studies by Verrotti et al. (3,9) showed no change in 25-OHD levels with CBZ treatment in spite of the considerable and otherwise consistent evidence to the contrary. I could only speculate on the reasons for this, but in any case it is unquestionably true that further work needs to be done to clarify this point; it is of considerable clinical importance, because if CBZ-induced changes cannot be counteracted using 25-OHD supplementation, then the drugs may pose an unavoidable risk of bone loss over time. Which brings me to a third point of disagreement: it is difficult to see the objection to 25-OHD supplementation in light of the evidence suggesting that clinically relevant 25-OHD deficiency might occur, particularly since such supplementation is both inexpensive and free of any adverse effects. I believe that Verrotti et al. are in the minority on this point, since two recent reviews on the subject also recommended prophylactic 25-OHD supplementation in this population (10,11).

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Hypermotor Seizure Arising from Insular Cortex

To the Editor:

Epilepsia recently published an article of three cases of drug-resistant nocturnal hypermotor seizures associated with an insular seizure onset (1). MR images of all three patients did not show any abnormalities in brain parenchyma but frontobasal arachnoid cyst in one case. Data of stereoelectroencephalography (SEEG) revealed interictal and ictal abnormalities at the anterosuperior insula in all three patients. The epileptic discharge spread over the mesial frontal structures such as the supplementary motor area and the cingulate gyrus. The histological diagnoses were not performed because surgery had been refused.

This article supports our recent findings about hypermotor seizures arising from insular cortex (2). We reported two surgical cases of hypermotor seizures. One of them had seizures mainly in the night and another in the daytime. Both of our two cases showed a minute high signal change in the right posterior ventral insular cortex in fluid-attenuated inversion recovery (FLAIR) studies of MRI. The posteroventral insula and lateral temporal cortices were resected after subdural electroencephalographic monitoring, resulting in complete seizure freedom. The histological diagnoses were focal cortical dysplasia in one and gliosis in another.

The insular lobe has efferent projections into cingulate areas (3), which are associated with hypermotor seizure (4). We estimate that nocturnal hypermotor seizures in three cases of this article (1) could be associated with a neural connection between the insula and medial frontal lobe for the onset of their symptoms.

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WORKSHOP REPORT

Proposed Diagnostic Scheme for the Classification of Epileptic Seizures and Epilepsies (ILAE, 2001): Proposal from Japan Epilepsy Society

A workshop on the newly proposed diagnostic scheme for the classification of epileptic seizures and epilepsies (ILAE, 2001) was held at Asahikawa, Japan on 14 October 2005 during the 39th Annual Meeting of Japan Epilepsy Society (JES). This workshop was planned by the JES Commission of Classification and Terminology of Epilepsy and chaired by Drs Kazuie Iinuma and Kiyoshi Morimoto. The main contents of the workshop are reported.

1. Application of the proposed new classification (ILAE, 2001) in neuroepidemiological study of childhood epilepsy (Tomoyuki Akiyama, Okayama)

Evaluation of the usefulness and validity of the newly proposed ILAE classification in the field of epidemiology was attempted by applying the scheme to the same subject population classified in our previous neuroepidemiological study conducted in Okayama Prefecture, Japan, in 1999.

- *Classification of epilepsy syndromes:* Only 269 patients (12.1%) with specific epilepsy syndromes were identified from the sample of 2,220 epileptic children, including 26 with newly defined syndromes. This figure did not change markedly in comparison with the 13.4% in our previous study based on the 1989 classification. According to the explanation for Axis 3, "a syndromic diagnosis may not always be possible." In reality, however, a syndromic diagnosis cannot be made for most cases in the general population. The reason is that the ILAE Task Force has adopted a method of classification that focuses mainly on specific epileptic syndromes. No matter how many specific syndromes may be defined and added to the classification in the future, it is very likely that most children with epilepsy in the general population will remain unclassified by Axis 3. Besides, in the current ILAE classification (1989), epilepsies can be at least classified into major categories such as idiopathic, symptomatic, or cryptogenic localization-related. This approach is useful for inferring the clinical characteristics of an individual patient. However, in the new classification scheme, epilepsies that do not belong to specific epilepsy syndromes cannot be classified.
- *Classification of seizure types:* Axis 2 (seizure type classification) was applied to 1,761 patients who could not be classified into specific syndromes. In 102 of them (5.8%), at least one seizure type could not be classified because they are not listed in the new classification. Most of them were seizures with clouding of consciousness as the only symptom or the main symptom, and seizures with only autonomic symptoms. These seizure types should be added to the new classification. Among the 1,153 cases of focal seizures, secondarily generalized seizures (89.8%) that do not reflect specific anatomical substrate were the most common. In the 2001 classification, Axis 2 is supposed to be used instead of Axis 3 in the diagnosis of patients without specific epilepsy syndromes. However, the new concept that seizure type is "a diagnostic entity with etiologic, therapeutic, and prognostic implications" does not apply to most cases, because even in children with focal seizures, most seizures do not represent a unique pathophysiologic mechanism and anatomic substrate. In addition, if interictal EEG findings are excluded from the diagnostic criteria of seizure types, most of the generalized seizures and secondarily generalized seizures cannot be diagnosed without ictal EEGs. This can cause a great problem, because a significant number of children with epilepsy will remain unclassified based on both Axes 2 and 3.

2. Application of the newly proposed ILAE diagnostic scheme for seizures and epilepsies (2001) to adult patients with epilepsy (Akio Ikeda and Masako Kinoshita, Kyoto)

In order to clarify the clinical validity of the new diagnostic scheme for epileptic seizures and epilepsies proposed by ILAE (2001), Axes 1–4 were applied to the adult patients with epilepsy treated at the neurology clinic, and also to the patients who underwent long-term video-EEG monitoring for intractable seizure disorders.

- *Patients and methods:* The subjects comprised 100 consecutive patients with a diagnosis of epilepsy who visited the Neurology Clinic, Kyoto University Hospital in June 2005 (group 1) and 100 consecutive patients with intractable epilepsy who underwent long-term video-EEG monitoring for the purpose of diagnosis or presurgical evaluation at the Department of Neurology, Kyoto University Hospital during the past 3 years (group 2). In both groups, Axes 1–4 of the ILAE proposed scheme (2001) were applied and the results were compared with the diagnoses established by the current ILAE seizure (1981) and epilepsy (1989) classifications.
- *Results:* In group 1 (mean age of 35 years), 27% of patients had seizures more than once a month, 146 seizures could be identified [44% were generalized tonic–clonic seizures (GTCS), 29% complex partial seizures (CPS), and 22% simple partial seizures (SPS)], and 62% of patients had localization-related epilepsy according to the conventional classifications. Applying Axis 1 (glossary) of the ILAE proposed scheme (2001) to group 1, 186 items (7 motor, 3 nonmotor, 2 modifier, 1 postictal) were listed. The glossary was useful to describe the seizure semiology independent of EEG findings. When Axis 2 (seizure type) was applied, 137 seizures were diagnosed [48% were GTCS, 27% focal motor seizures (FMS), and 13% focal sensory seizures (FSS)] and 7 seizures could not be classified other than as so-called CPS. Twenty-four of 36 FMS and 11 of 17 FSS were identified as CPS according to the 1981 classification. By applying Axis 3 (epilepsy syndromes), one patient with familial temporal lobe epilepsy (TLE) was newly diagnosed, but five patients with symptomatic generalized epilepsy according to the 1989 classification could not be classified. By applying Axis 4 (etiology), specific etiologies such as hippocampal abnormalities, space occupying lesion, cortical malformation, hereditary, tuberous sclerosis, SLE, head trauma, stroke and encephalitis were well described, but four patients with so-called cryptogenic epilepsy remained unclassified. In group 2 (mean age of 29 years), 84% of patients had seizures more than once per week, and 89% of patients had localization-related epilepsy. By applying Axis 1 (glossary) of the ILAE proposed scheme (2001), 437 items (10 elementary motor, 8 automatism, 5 nonmotor, 2 modifier, 1 prodrome, 3 postictal) were listed. Especially, items not listed in group 1 but listed in group 2 mainly belonged to elementary motor (Jacksonian, atonic, dystonic, versive) and automatism (verbal dysphasic, vocal, hyperkinetic, hypokinetic, oroalimentary, manual or pedal).
- *Conclusions:* (1) Axis 1 in the ILAE proposed scheme (2001) is helpful to document the patients' clinical state precisely, even without EEG information. It is also useful in clinical practice for a precise diagnosis of the epileptic state in candidates of epilepsy surgery. (2) Axes 1 and 2 may provide redundant information. (3) Axes 2 and 3 are not either supplementary to or replaceable of the 1981 and 1989 classifications, respectively. (4) Axis 4 is useful to understand the patients' current and possibly future states.

3. Practical use of Axis 5 in the diagnostic scheme for people with epileptic seizures and with epilepsy (Mana Kurihara, Kanagawa)

The Proposed Diagnostic Scheme for People with Epileptic Seizures and with Epilepsy (2001) is divided into five axes. Axis 5 is recommended for use as a parameter for evaluating the impairment in patients with epilepsy on the basis of the International Classification of Functioning, Disability and Health (ICF). The impairment in 132 patients with epilepsy aged over 18 years was checked according to the criteria of ICF. The subjects were divided into three groups according to the requirement of help in daily living activities; group I: 84 patients requiring full help, group II: 17 patients requiring some help, and group III: 31 patients requiring no help. The impairments in groups I and II were dependent on mental retardation and physical disability, and the impairments could be easily diagnosed using the components of body functions (b) and activities and anticipation (d). The impairments in group III could not be readily diagnosed, but were indicated in self-care (d5), major life areas (d8), and other components. Many patients with epilepsy apparently exhibit no impairment in their daily lives, although they have some problems. Axis 5 is useful for objective evaluation of such patients.

Comments, questions, and suggestions on our proposal are always welcome.

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Response

It is important that diagnostic schemes and classifications proposed by ILAE, or any other group or individual, be field-tested. We, therefore, appreciate the fact that members of the Japanese Epilepsy Society have attempted to evaluate the clinical efficacy of the diagnostic scheme proposed by the ILAE Task Force on Classification and Terminology in 2001. Their report, however, requires commentary on several levels:

1. The discussion of this report refers to the proposed diagnostic scheme as a "new classification." The diagnostic scheme was never intended to be a classification. It merely sets out a standardized format for describing individual patients. It should not, therefore, be compared to either the 1981 Classification of Epileptic Seizures or the 1989 Classification of Epilepsies.
2. The failure to make a syndromic diagnosis in a high percentage of patients reflects on the 1989 classification and list of currently accepted syndromes, but not on the diagnostic scheme per se. The data presented, in any event, are inconsistent with several other studies that have found that between 40% and 65% of children could be given a specific syndromic diagnosis (1–5). The low percentage in this report could be due to a variety of reasons that have been discussed elsewhere (6), and there is incomplete information to permit determination, for instance, of how well these patients were evaluated and by whom, whether attempts at diagnosis were made only at the first visit, or whether they may have had only a single seizure. The percentage of patients who were given a syndromic diagnosis also depends on the willingness of the diagnosing physician to make differential diagnoses that need to be confirmed over time.
3. The ILAE Task Force has not ". . . adopted a method of classification that focuses mainly on specific epileptic syndromes . . . ," as these investigators claim. The Task Force has stated repeatedly that a syndromic diagnosis cannot be made in many patients, but contends that when this is not possible, diagnosis of one or more seizure types as diagnostic entities can be made which have etiologic, therapeutic, and prognostic implications. These investigators also indicate that the 1989 classification at least permitted categorizing patients into approximate categories (e.g., localization-related cryptogenic epilepsy) but that the 2001 approach does not. The list of syndromes provided in Table 4 of the 2001 report is not a classification but merely an updated list of syndromes that have gained general acceptance in the epilepsy community. Although Table 5 in that report provides an example of an alternative classification, the 1989 categorization should still be used.
4. Although there is some redundancy between Axis 1 descriptions of seizure semiology, and Axis 2 seizures as a diagnostic entity, the concepts are entirely different. It is almost always possible to obtain some description of seizure semiology, but this often provides no information about pathophysiology and anatomic substrate. It should also almost always be possible to diagnose one of the diagnostic seizure types listed in the 2001 report, although these investigators were not able to do this for at least one seizure type in 5.8% of patients, most of whom had "seizures with clouding of consciousness as the only symptom or the main symptom, and seizures with only autonomic symptoms." They suggest that these seizure types should be added to a new classification. Perhaps they mean that they should be added to the list of diagnostic seizure types. We are aware that the list of focal seizure types in the 2001 report is incomplete, in that it does not include a category where cognitive deficits and autonomic symptoms are the only ictal manifestations. An entirely different approach for focal seizures is suggested in a report of the Core Group of the ILAE Task Force on Classification and Terminology to be published in Epilepsia in September.
5. It is true that we do not at this point know the pathophysiological mechanisms and anatomical substrates of all of the seizure types listed in the 2001 report, nor can we be absolutely certain at this point whether each of these represents a unique diagnostic entity. Nevertheless, the statement ". . .even in children with focal seizures, most seizures do not represent a unique pathophysiologic mechanism and anatomic substrate" cannot be true. All seizures have pathophysiologic mechanisms and anatomic substrates. The new ILAE Commission on Classification is currently taking an evidence-based approach toward examining and testing the uniqueness of seizure and syndrome diagnostic entities. This approach will be flexible and responsive to new information as it becomes available, and is described in the report to be published in September.

The new ILAE Commission on Classification welcomes suggestions from clinicians, particularly when they are based on quantitative observations. More details about the actual studies reported by these authors would be helpful. Nevertheless, it is important to understand that there is

as yet no new classification of epileptic seizures or epilepsies, and that neither the 2001 ILAE report, nor the report to be published this September, should be considered as a replacement for the 1981 Classification of Epileptic Seizures and the 1989 Classification of Epilepsies. These reports provide updated lists of syndromes, seizure types, and etiologies, and discuss some anticipated changes in development that could eventually lead to new approaches for organizing our knowledge about seizures and epilepsies and classifying their various forms.

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NEXT MONTH IN *Epilepsia*

The featured theme of the October issue is **Epilepsy Genes and Genetics**. The theme will be introduced by three critical reviews. The first, from Sara Shostak and Ruth Ottman, deals with the "Ethical, Legal, and Social Dimensions of Epilepsy Genetics." These authors have introduced a number of very important issues that we must consider as we become more knowledgeable and technically sophisticated about the genetics that influence epilepsy and seizure susceptibility. We've invited commentary on this paper, not only from epilepsy (and other) geneticists but also from social scientists; their letters, and responses from Shostak and Ottman, will also appear in Gray Matters of the October issue.

Two other reviews on the genetic theme focus on "gene profiling." Frank Sharp and colleagues present a general overview of this powerful technique–including opportunities and technical difficulties inherent in gene profiling from both brain tissue and blood. Albert Becker and Peter Crino then review the studies that have been carried out with gene profiling techniques, to assess gene changes/abnormalities in temporal lobe epilepsy and in various forms of epilepsy-associated cortical dysplasia.

These reviews will be followed by a set of Original Research papers and Brief Communications that focus on:

Genetics of febrile seizures
Genetics of myoclonic seizures
Genetics of Rolandic epilepsy
$GABA_A$ receptor subunits
Sodium channel mutations

There are also articles on non-genetic themes, amongst which is an article entitled "Epilepsy in North America". This report, from this ILAE Commission on North America, provides a comprehensive summary of regional facilities and concerns.

ANNOUNCEMENTS

Esther A. & Joseph Klingenstein Fund

The Esther A. & Joseph Klingenstein Fund is pleased to announce its Fellowship Awards in the Neurosciences for 2007. The purpose of these awards is to support young investigators, in the early stages of their careers, engaged in basic or clinical research that may lead to a better understanding of the etiology, treatment, and prevention of epilepsy. Studies of interest range from the molecular and cellular levels to integrative systems function to clinical investigations. Up to ten Klingenstein Fellows will be appointed in 2007, and each will receive an award of $150,000 over three years. Applicants must hold a Ph.D. and/or M.D. degree, and have completed all research training (including postdoctoral). The deadline for applications is December 8, 2006. For additional information, see www.klingenstein.org or write to The Klingenstein Fund, 787 Seventh Avenue-6th Floor, New York, NY 10019-6016 or call (212) 492-6181.

Michael Prize 2005/2006

The Michael Prize is one of the most highly regarded international awards for the best contribution to scientific and clinical research promoting further developments in epileptology. It is awarded biannually and specially designed to attract younger scientists (under 45 years of age). Applications should be submitted in English or German and the prize money is 15.000 Euros. Publications which have appeared in 2005/2006 or papers of the same period not yet published will be considered. Members of the Jury are Colin Binnie, London, UK; Uwe Heinemann, Berlin, Germany; and Solomon Moshé, New York, USA. Since 2006 the Michael Prize has been sponsored by UCB International. Articles and papers, together with a curriculum vitae should be submitted in triplicate to Stiftung Michael by December 31, 2006. For applications or for more information contact Stiftung Michael, Muenzkamp 5, D-22339 Hamburg, Germany. Tel.: +49 (0)40 5388540 Fax: +49 (0)40 5381559 e-mail: stiftungmichael@t-online.de

CALENDAR OF MEETINGS

September 2006

10th Congress of the European Federation of Neurological Societies (EFNS)
2–5 September 2006
Glasgow, UK
http://www.efns.org/efns2006

4th Latin American Congress on Epilepsy
6–9 September 2006
Guatemala
http://www.epilepsyguatemala2006.org/en/index.html

5th INMED/TINS Conference -"Physiogenic and pathogenic oscillations: the beauty and the beast"
9–12 September 2006
LaCiotat, France
http://inmednet.com/2006-conference.html

8th Eilat Conference on New Antiepileptic Drugs (Eilat VIII)
10–14 September 2006
Sitges, Spain
http://www.eilat-aeds.com/viii/index.asp

October 2006

131st Annual Meeting of the American Neurological Association (ANA)
8–11 October 2006
Chicago, Illinois, USA
http://www.aneuroa.org/index.php?submenu=AnnualMeeting&src=gendocs&link=AnnualMeetingProgram&category=2005%20San%20Diego

November 2006

6th Asian & Oceanian Epilepsy Congress (OAEC)
16–19 November 2006
Kuala Lumpur, Malaysia
http://www.epilepsykualalumpur2006.org

December 2006

60th Annual Meeting of the American Epilepsy Society (AES) - First North American Regional Epilepsy Congress
1–5 December 2006
San Diego, California, USA
http://www.aesnet.org/Visitors/AnnualMeeting/index.cfm

March 2007

First International Congress on Epilepsy, Mind & Brain
29–31 March 2007
Prague, Czech Republic
http://www.kenes.com/epilepsy

April 2007

International Symposium on Biology of Seizure Susceptibility (ISBSS): (10th Annual Meeting of Infantile Seizure Society)
7–8 April 2007
Tokyo, Japan
www.iss-jpn.info

First London Colloquium on Status Epilepticus
12–14 April 2007
London, UK
http://www.conference2k.com/statusconf.asp

American Academy of Neurology (AAN) 59th Annual Meeting
28 April–5 May 2007
Boston, MA, USA
http://www.aan.com

June 2007

17th European Neurological Society (ENS) Meeting
16–20 June 2007
Rhodes, Greece
http://www.ensinfo.com/

July 2007

IX Workshop on Neurobiology of Epilepsy (WONOEP 2007)
3–6 July 2007
Langkawi Island, Malaysia
http://www.wonoep2007.univ-mrs.fr

27th International Epilepsy Congress
8–12 July 2007
Singapore
http://www.epilepsysingapore2007.org