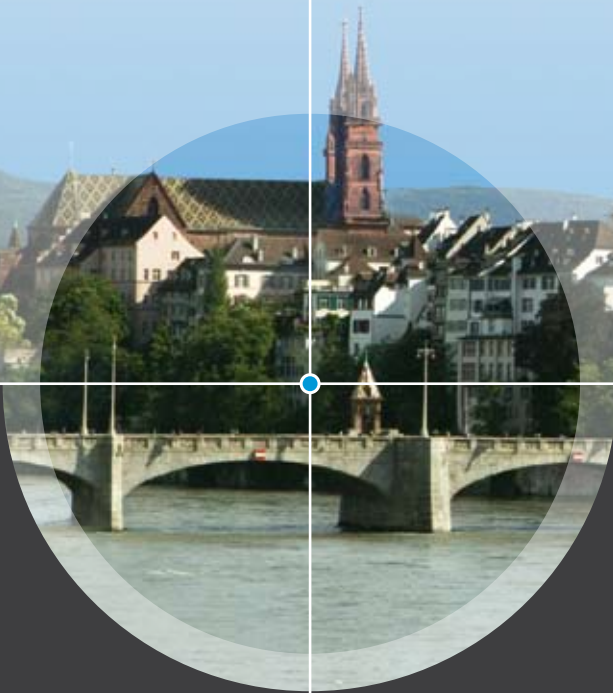


Swiss Neurological Society SNS  
Swiss Society of Neurosurgery SSN  
Swiss Society of Clinical Neurophysiology SSCN  
Swiss Society of Neuropediatrics SSNP  
Swiss Society of Neuroradiology SSNR  
Swiss Society of Neuropathology SSNPath



Abstractbook

**1st Congress  
Swiss Federation of  
Clinical Neuro-Societies  
SFCNS**

June 2 - 4, 2010  
Congress Center Basel

## Dear colleagues

The Joint Meeting of six clinical neuroscience societies in April 2008 in Montreux, which commemorated the hundred years of the Swiss Neurological Society, marked the beginning of a new cooperative effort to strengthen the clinical neurosciences in Switzerland. Following this great success and overwhelming approval, the Swiss Neurological Society (SNS), Swiss Society of Neurosurgery (SSN), Swiss Society of Clinical Neurophysiology (SSNC), Swiss Society of Neuropaediatrics (SSNP), Swiss Society of Neuroradiology (SSNR), and the Swiss Society of Neuropathology (SSNPath) decided to close ranks and form a Swiss Federation of Clinical Neuro-Societies (SFCNS) which should be open also to other medical groups pursuing clinical neuroscience interests. The federation should formalise and structure the joint medico-political, educational, and clinical research efforts and hold common meetings on a regular basis which should facilitate mutual interactions. It should also be a cooperative partner of the Swiss Society for Neuroscience.

It is thus my great pleasure to invite you to the first meeting of the newly founded Swiss Federation of Clinical Neuro-Societies which will be held on 2nd to 4th June 2010 in Basel. This first meeting will be dedicated to cerebrovascular diseases, epilepsy, and disorders of the spine & spinal cord as main topics. The scientific committee of the congress spares no effort to organize an attractive program which will interest a great majority of the societies involved.

I am looking forward to welcoming all of you in Basel in June 2010!



Christian W. Hess, MD, PhD Professor  
Chairman of the Scientific Committee



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**Andreas Raabe, MD, Professor of Neurosurgery**

Inselspital, Neurochirurgie  
CH-3010 Bern

**Curriculum vitae**

Andreas Raabe, MD, is Professor of Neurosurgery and Chairman of the University Department of Neurosurgery in Bern, Switzerland. He was trained at the neurosurgical departments and universities in Chemnitz, Leipzig, Cambridge, Munich and Phoenix, Arizona. Before his appointment as director in Bern, he was associate professor of Neurosurgery at the Johann Wolfgang Goethe University in Frankfurt am Main.

Professor Raabe's fields of expertise are in operative microneurosurgery and endoscopy with main focus on vascular neurosurgery, tumor surgery, and spinal microsurgery. His clinical research focus on the use of intraoperative technologies during vascular and tumor surgery such as image guidance, fMRI and tractography, intraoperative imaging, fluorescence diagnostics and intraoperative electrophysiological monitoring and mapping techniques.

His vascular research interest are intracranial aneurysms, especially the assessment of growth and rupture risk, epidemiology, management or unruptured aneurysms and counselling of incidental or familial aneurysm patients. It also includes development of new intraoperative techniques such as infrared angiography, vascular navigation and more. He is also active in researching diagnosis and treatment of cerebral vasospasm after subarachnoid hemorrhage and is member of the steering committee of CONSCIOUS-2 and CONSCIOUS-3 study investigating the effect of a selective endothelin-A receptor antagonist clazosentan in clipped and coiled patients.

Prof. Raabe has published more than 150 original scientific papers, review articles or technical notes and is author of several book chapters. He is a member of 13 national and international societies and reviews for more than 20 international journals. Prof. Raabe is married and happy about his 11-year old boy and his 16-year old daughter.

**Intracranial aneurysms: new aspects of formation, rupture and outcome**

Rupture of an intracranial aneurysm is the third most common cause of stroke. Contrary to ischemic stroke it affects younger patients during their socially and professionally active life period. About 50% of patients die from subarachnoid haemorrhage (SAH) and only 10% of patients return to their previous personal, social and professional life. From a socioeconomic standpoint, the loss of productive life years caused by SAH is comparable to ischemic stroke. Therefore, prevention of aneurysm formation and rupture are logical first line research strategies.

Major risk factors for SAH are hypertension, smoking, alcohol consumption, family history of SAH or aneurysms and certain genetic diseases such as adult polycystic kidney diseases. They increase the risks by 2 to 6 and the former play an important role as modifiable risk factors. Recent research has confirmed the role of genetic predisposition, but the individual risk prediction remains a challenge.

Outcome after aneurysm rupture is poor and is determined most often by the severity of the subarachnoid haemorrhage. Medical treatment is virtually excluded during the minutes between rupture and clotting of the leak at the aneurysm. Early aggressive management of vital functions, intracranial pressure, hydrocephalus, space occupying hematomas and prevention of rebleeding by early clip or coil obliteration remain the mainstays of treatment. Most aneurysms are treated endovascularly (60-70%) and microsurgical clipping is mainly performed in complex configured aneurysms that preclude coiling. Advances of endovascular and microsurgical intraoperative developments will be presented. Prevention and treatment of delayed neurologic deterioration caused by cerebral vasospasm is reshaping currently with increasing endovascular therapies. Selective endothelin A-receptor antagonists are currently investigated in large international randomized trials. Outcome has improved in recent years but remains far from acceptable.



**Karl Schaller, MD, Professor of Neurosurgery**

HUG, Service de Neurochirurgie  
24, Rue Micheli du Crest, CH-1211 Genève

**Curriculum vitae**

Karl Schaller is Professor and chairman of the Department of Neurosurgery at the University of Geneva Medical Center (HUG). He did his pre-graduate studies at the University of Tübingen, and was trained as a neurosurgeon by Werner Hassler in Duisburg, and by Johannes Schramm in Bonn. He has a strong clinical focus on cranial neurosurgery in the area of neurovascular disease, epilepsy, intrinsic brain tumors, and tumors of the skull base. His research activities relate to the pathophysiology of cerebral blood flow, and to the co-registration of imaging and electrophysiological data for the refinement of surgical planning and for real-time intra-operative adjustment. At the HUG he is supported by a young team of specialist neurosurgeons, and he cooperates closely with epileptologists, basic neuroscientists, and IT-scientists, both - locally and internationally.

**Intracerebral haemorrhage**

Spontaneous intracerebral haemorrhage (ICH) is a frequent disease, particularly in the elderly. Its incidence in the general population accounts for 12-15/100.000 per year, whereas it reaches its peak of app. 100/100.000 persons per year in people >75 years of age. The various patient groups differ not only demographically, but also in dependance of the origin of their ICH. Alcohol abuse, elevated arterial blood pressure, elevated cholesterine level, and previous ischemic stroke predispose for the development of ICH. Most ICH, particularly in elderly patients are located deeply, or ganglionic, and relate to long-standing arterial hypertension with concomitant atherosclerosis of small and fragile end-arteries. Even lobar ICH in the elderly may have its origin in a degenerative process of small arteries, so-called cerebral Amyloidosis. ICH in the elderly has a poor prognosis with an estimated one-year mortality rate of 40%. What is particularly unfortunate concerns the fact, adequate treatment regimen does not exist. Thus, it doesn't make a difference, whether these patients are treated surgically (via open craniotomy, stereotactic aspiration and hematoma lysis, or endoscopically), or if they receive intensive care treatment according to a conservative internistic/neurological regimen. This is the reason for most neurosurgeons to refrain from surgical therapy. This almost fatalistic attitude has been substantiated by the first part of an international multi-center study (STICH trial). All this applies to the management of supratentorial ICH. The situation with infratentorial, cerebellar, ICH is different, as surgical management of those hematomas bears a better prognosis. Chronic diseases of advanced age contribute negatively by increased rates of secondary complications. Recombinant factor VIIa may improve the prognosis of patients suffering from ICH, but is very costly. Management and prognosis of young patients presenting with ICH is entirely different, as treatable pathologies are the underlying cause mostly, e.g. vascular malformations (AVM, cavernomas etc.), or aneurysms, to name a few.



**Geoffrey A. Donnan, MD, Professor of Neurology**

Florey Neuroscience Institutes, University of Melbourne, Level 2 Alan Gilbert Building,  
161 Barry Street, AUS-Carlton South Victoria 3053

**Curriculum vitae**

Geoffrey A. Donnan, MD is Director of the Florey Neuroscience Institutes and Professor of Neurology, University of Melbourne (Department of Medicine) in Australia. He was founder of the National Stroke Research Institute, co-founder of the Australian Stroke Trials Network (ASTN) and Neurosciences Trials Australia (NTA). His major interests are in neuroimaging and clinical trials. Professor Donnan has published over 400 papers in peer reviewed journals, over 60 book chapters, edited 4 books and has been Lecturer or Visiting Professor in numerous countries. He is Past President of the Stroke Society of Australasia, the Australian Association of Neurologists and the World Stroke Organization. He was the recipient of the American Stroke Association William Feinberg award for excellence in clinical stroke research in 2007. He has also delivered the Priscilla Kincaid Smith Oration for the Royal Australasian College of Physicians in 2007 and received the MJ Eadie Award for Career Achievement in Neuroscience Research at the Australian and New Zealand Association of Neurologists in 2008. He was awarded the Bethlehem Griffiths Research Foundation medal for outstanding contribution to international stroke research in 2008.

**New Insights in the pathophysiology of stroke**

Eighty-five percent of strokes are of ischemic origin. The mechanisms range from large artery to artery embolism, cardiac embolism, small vessel leukemic or borderzone infarction. Regardless of this, the final common pathway is arterial obstruction and haemorrhagic compromise, energy failure and disruption of homeostatic mechanisms. The concept of the ischemic penumbra is critical to an understanding of the subsequent evolution of ischemic tissue to infarction or salvage. Fortunately, modern imaging techniques allow us to understand this process much better. There is evidence to suggest that penumbra duration may be up to 48 hours post stroke onset and that the "life expectancy" of the penumbra may be linked to arterial collateralisation among other factors. A better understanding of the ischemic penumbra is essential if we are to successfully extend the time window for interventions such as thrombolytic therapy.



**Hans Lassmann, MD, Professor of Neuroimmunology**

Medical University of Vienna, Center for Brain Research  
Spitalgasse 4, A-1090 Vienna

**Curriculum vitae**

Hans Lassmann graduated from Medical School at the University of Vienna in 1975. He then joined the Institute of Neurology of the University of Vienna for training in clinical and experimental neuropathology. In addition he spent one year as a post doc at the Institute for Basic Research in Developmental Disabilities in New York. In 1990 he became director of the Research Unit for Experimental Neuropathology of the Austrian Academy of Science and in 1993 Professor for Experimental Neuropathology in the University of Vienna. From 1999 to 2007 he was the founding director of the Center for Brain Research of the Medical University of Vienna. He is currently Professor for Neuroimmunology at the Medical University of Vienna. He has received many research awards, including the Charcot Award of the International Federation of Multiple Sclerosis Societies for Life Long Achievement in Multiple Sclerosis Research.

**Inflammatory Demyelinating Myelopathies: Pathology and Pathogenesis**

Classical inflammatory demyelinating diseases, which affect the spinal cord are multiple sclerosis, neuromyelitis optica and acute disseminated encephalomyelitis. All these diseases have in common an acute or chronic inflammatory reaction within the tissue, which is associated with demyelination and tissue injury. Multiple sclerosis lesions may appear at any site of the myelon, although the cervical cord is more frequently affected than other spinal cord segment. Lesions affect both the white and grey matter, although the lateral columns of the cord are most frequently involved. Primary demyelination with variable axonal loss and reactive gliosis is the hallmark of MS lesions. As in the brain lesions in the spinal cord may also get remyelinated and may appear as shadow plaques. Different immunological mechanisms appear to be involved in the formation of MS lesions, the most important one seems to be mediated by macrophages and activated microglia, which induce tissue damage through radical formation and mitochondrial injury. Active lesions in neuromyelitis optica are characterized by massive deposition of immunoglobulins and complement at the sites of active damage. This appears to reflect the pathogenic role of auto-antibodies against aquaporin 4. In line with this concept, aquaporin 4 is widely lost within the lesions and astrocytes are impaired or destroyed prior to demyelination. Transfer of aquaporin 4 antibody containing human immunoglobulin from NMO patients induces NMO lesions in experimental animals, when they reach the central nervous system at sites of T-cell mediated inflammation. A currently unresolved question relates to the pathogenesis of disease in NMO patients, who have no aquaporin 4 antibody response. So far no pathological data are available from such patients. Finally, acute disseminated encephalomyelitis may involve the spinal cord and, when severe may lead to transverse myelitis. It is likely that acute disseminated encephalomyelitis represents a clinical entity, which includes several pathogenetically different diseases with both, autoimmune or infectious background. Its hallmark is severe inflammation, which is associated with small rims of perivascular demyelination. In fulminant cases profound tissue destruction dominates, while primary and selective demyelination is sparse.



**Brian Weinschenker, MD, FRCP(C), Professor of Neurology**  
Mayo Clinic, 200 First St SW  
US-Rochester MN 55905

### Curriculum vitae

Dr. Weinschenker is a Professor of Neurology at Mayo Clinic, Rochester MN. His principal interests are the spectrum of inflammatory demyelinating disease of the central nervous system, especially neuromyelitis optica; the natural history of MS; treatment of acute, severe attacks of inflammatory demyelinating disease; and the genetics of complex diseases, especially multiple sclerosis and neuromyelitis optica. His major academic accomplishments are:

1988-2001: characterization of the natural history of MS in a cohort of 1100 patients in Middlesex County, Ontario, and application of multivariate analysis to prediction of clinical outcome

1995-2002: establishment of a plasma exchange as an effective therapy for acute severe attacks of inflammatory demyelinating disease in a randomized, double-masked, sham-controlled study

2002 until the present: characterization of clinical aspects of neuromyelitis optica and differentiation from MS; establishment of effective diagnostic criteria; collaborator in identification of NMO-IgG (aquaporin-4-specific antibodies) as a specific diagnostic test for neuromyelitis optica; characterization of an extended spectrum of illness and insights into pathogenesis of neuromyelitis optica; genetic analysis of a variety of candidate genes for MS, neuromyelitis optica and other complex genetic diseases, including lung cancer.

### Acute Myelopathy: Advances in Diagnosis and Management

Evaluation of acute myelopathy had been limited by the lack of specificity of clinical features, limited imaging resolution, lack of specificity of cerebrospinal fluid investigations and the eloquence of the spinal cord that usually precludes biopsy as a diagnostic modality. Class I and II data that permit a diagnosis based on demographic, clinical and investigations are lacking, and it has been difficult to convincingly develop evidence-based guidelines for the efficient and comprehensive evaluation of patients with this clinical syndrome that consistently yields a specific and accurate diagnosis. However, advances in neuroimaging, CSF diagnostic techniques (e.g. PCR), and neuroimmunology (in particular the discovery of a highly specific marker for neuromyelitis optica spectrum disorders), have enhanced the neurologist's ability to more specifically diagnose patients with acute myelopathy. The proportion of patients with a final diagnosis of "idiopathic transverse myelitis" after appropriate investigations is declining and it is possible to reliably identify those at risk for MS from those with either idiopathic transverse myelitis or a neuromyelitis optica spectrum disorder. The majority of those patients with an inaugural neuromyelitis optica spectrum disorder are identifiable at initial presentation by virtue of seropositivity for autoantibodies to aquaporin-4 (NMO-IgG). Such patients benefit from immunoprophylaxis at an early stage of their illness with immunosuppressive drugs to reduce the risk of relapse.

The evaluation begins with confirmation that a myelopathy is present and excluding acute polyneuropathy, brainstem or brain disorder presenting with spasticity. Determination of the time course is extremely important and dictates the differential diagnosis. Spinal cord infarction develops over minutes to hours. Transverse myelitis develops over days to weeks, and reaches a nadir deficit within 3 weeks; patients with transverse myelitis may have an infectious (typically viral), parainfectious or demyelinating disorder. Conversely, patients who experience progression of myelopathy beyond this interval likely do not have a demyelinating transverse myelitis, but a progressive myelopathy, the differential diagnosis of which is more extensive and includes vascular causes (e.g. dural arteriovenous fistula), other inflammatory or atypical infectious causes (e.g. neurosarcoidosis, schistosomiasis, paraneoplastic disorders). Determination of the pattern of myelopathy (e.g. hemicord, central cord, anterior cord, tractopathy) can further refine the diagnosis, but these patterns of clinical involvement are nonspecific, and a comprehensive evaluation of risk factors, time course, radiology, CSF and other factors may be necessary for accurate diagnosis.



**Anton Valavanis, MD, Professor of Neuroradiology**

Director of the Institute of Neuroradiology  
Universitätsspital, CH-8091 Zurich

**Curriculum vitae**

Professor Anton Valavanis, born in Athens, Greece, studied medicine at the University of Zurich and completed his training in radiology and neuroradiology at the Department of Radiology of the University Hospital of Zurich in 1981.

He started his academic career as Assistant Professor in 1984, was promoted to Associate Professor in 1985 and obtained full Professorship at the chair of neuroradiology in 1994. The same year he founded the Institute of Neuroradiology at the University Hospital of Zurich and has been its Chairman ever since. Since 1998, he is member of the Governing Council of the Neuroscience Center of the University and the ETH Zurich.

His research is focused on the further refinement of therapeutic neuroendovascular techniques and on the in-vivo elucidation of the architectonic organization of the brain by applying advanced neuroimaging modalities. He has authored or co-authored 200 original scientific publications and several books. He served as Editor-in-Chief of "Neuroradiology", the official organ of the European Society of Neuroradiology from 1991-2005 and as International Advisor to the Editor of the "American Journal of Neuroradiology". He is Corresponding Editor of "Interventional Neuroradiology" and member of the editorial board of various journals.

He delivered more than 700 invited lectures all around the world and was President of the 17th Congress of the European Society of Neuroradiology, the International Congress of Head and Neck Radiology, the 1st Congress of the World Federation of Interventional and Therapeutic Neuroradiology, Zurich 1991, and of annual meetings of the Swiss Society of Neuroradiology. In 1992 he founded the "Zurich Course on Interventional Neuroradiology", which is held annually and is recognised as one of the premier educational activities in the field.

He is founding member of the Swiss Society of Neuroradiology, the European Society of Head and Neck Radiology, the International Skull Base Society, the World Federation of Neuroradiological Societies and the World Federation of Interventional and Therapeutic Neuroradiology. He served as President of the Swiss Society of Neuroradiology and of the World Federation of Neuroradiological Societies. He is a co-initiator for the founding of the Swiss Federation of Clinical Neuro-Societies and currently Vice-President of this federation.

He received the Scientific Awards of the German, Swiss and European Societies of Neuroradiology, the Georg Friedrich Götz Award of the University of Zurich, and the James Bull Medal of the British Society of Neuroradiologists. He has been awarded Honorary Membership of the Hellenic Radiological Society, the Italian Society of Neuroradiology, the Hellenic Neurosurgical Society and the Austrian Radiological Society. He holds honorary directorships at the neuroradiology departments of the International Neuroscience Institutes of Hannover, Germany and Beijing, China and a honorary doctorate of the University of Athens.

**Vascular myelopathies**

Spinal vascular diseases manifesting themselves as acute, subacute, intermittent or chronic myelopathy caused by ischemia or hemorrhage are rare, constituting approximately 2% of all vascular neurologic pathologies. In this presentation the role of neuroradiology in the diagnostic work-up, which is primarily based on advanced, multimodal MR-imaging techniques and in the therapeutic management, which is based on superselective endovascular techniques will be discussed with emphasis on spinal arterial ischemia and spinal vascular malformations including cavernomas and the various types of arteriovenous shunts.

Thursday, 03.06.2010



**Michel Zerah, MD, Professor of Paediatric Neurosurgery**

Hôpital Necker - Enfants Malades, Service de Neurochirurgie Pédiatrique  
149, Rue de Sèvres, F-75743 Paris

**Curriculum vitae**

Michel Zerah was born in 1956 in Paris where he did his medical studies. He is graduate in Mathematics and Computer Engineering and has a PHD in Statistics. From 1985 to 1994 he has been consultant in the Department of Neurosurgery in Bicêtre where he worked with Pierre Lasjaunias. Since 1998 he is Professor of Paediatric Neurosurgery in Necker Enfants Malades Hospital in Paris where he has been director of the Department of Paediatric Surgery from 2000 to 2006. He belongs to the French National Institute for Medical Research (INSERM U745 : Genetics and biotherapy of degenerative and proliferative diseases of the nervous system). He is the Chairman of the European Course of Paediatric Neurosurgery. He is specially involved in spinal and spinal cord tumors and malformations, vascular diseases and gene therapy for genetic and metabolic diseases in children.

Friday, 04.06.2010



**Itzhak Fried, MD, PhD, Professor of Neurosurgery**

David Geffen School of Medicine at UCLA, Department of Neurosurgery  
740 Westwood Plaza, Box 957039, US-Los Angeles, CA 90095-7039

**Curriculum vitae**

Dr. Itzhak Fried is Professor of Neurosurgery and Psychiatry & Biobehavioral Sciences at the David Geffen School of Medicine at UCLA. His training was at UCLA, Stanford and Yale. His research program at UCLA centers on the neuronal substrates for visual perception and memory, auditory function and motor programming in humans. His research uses the opportunity of recording single neuron activity in neurosurgical patients undergoing implantation of intracranial electrodes for evaluation for epilepsy surgery. Some findings from his laboratory have included the discovery in humans of place cells for navigation, imagery neurons, mirror neurons, surprising hyper-narrow frequency tuning of auditory neurons, and the unraveling of a unique sparse, invariant coding in the human hippocampus. For this work he has been named a fellow of the American Association for the Advancement of Science.



**Angela Vincent, MBBS MSc FRCPath FRCP FMedSci, Professor of Neuroimmunology**

John Radcliffe Hospital, Neuroimmunology Group  
West Wing and Weatherall Institute of Molecular Medicine, UK-Oxford OX3 9DS

**Curriculum vitae**

Angela Vincent qualified as a doctor but subsequently did an MSc in Biochemistry. Working with Ricardo Miledi FRS, she became involved in some of the earliest studies on acetylcholine receptors in myasthenia gravis and congenital myasthenic syndromes, and began a long partnership with Prof John Newsom-Davis, first at the Royal Free Hospital in London and then at the newly-established Weatherall Institute of Molecular Medicine in Oxford. Since Newsom-Davis' retirement in 1998 (and unexpected death in 2007), she has led the research in Oxford. She is an honorary consultant in immunology and has established a national and international referral centre for the diagnosis of immune-mediated neurological diseases. From 2005-2008, she was Head of Department of Clinical Neurology.

She is now Emeritus Professor of Neuroimmunology in Oxford, Professor of Neuroimmunology at the Institute of Neurology, University College London, and an Emeritus Fellow of Somerville College. She has an Honorary degree from the University of Bergen (2004), and is a Fellow of the Academy of Medical Sciences. She is an Associate Editor of *Brain* and was President of the International Society of Neuroimmunology (2001-2004). She has coedited four books including one on pediatric neuroimmunology (2010). Her major clinical interest is in the role of auto-antibodies to ion channels and receptors in peripheral and central disorders, and in advancing the diagnosis of these conditions. Her research interests include neuromuscular junction disorders, models of immune-mediated CNS diseases, and the influence of maternal antibodies in neurodevelopmental disorders.

**Autoimmune channelopathies: the growing spectrum of immunotherapy-responsive neuropsychiatric disorders**

It is well known that in myasthenia gravis there are antibodies to the acetylcholine receptor (AChR) in 80% and to muscle specific kinase (Musk) in a proportion of the remaining 20%. In the Lambert Eaton myasthenic syndrome there are antibodies to the voltage-gated calcium channel (VGCC); and in acquired neuromyotonia antibodies to voltage-gated potassium (VGKC) channels. Each of these conditions, which are usually chronic and unremitting, is associated with good clinical responses to immunotherapies (in conjunction with symptomatic therapies), and the roles of the antibodies have been established by a variety of in vitro and in vivo approaches.

However, VGKC antibodies are also found in Morvan's which includes peripheral nerve hyperexcitability, autonomic dysfunction and central involvement. This condition is often associated with thymoma. VGKC antibodies are also increasingly found in patients with a predominantly non-paraneoplastic limbic encephalitis that presents as memory loss, seizures and sometimes frank psychosis, and in patients with adult-onset epileptic disorders. Most of these patients also do well with immunotherapies. A more complex encephalopathy is associated with antibodies to NMDA receptors. These patients are often younger and can be children. Females are more common than males, and may have ovarian teratomas. The disease involves seizures, cognitive and neuropsychiatric disorders, movement disorders, autonomic disturbance, reduced consciousness and other brainstem features. The patients respond slowly to immunotherapies and may relapse if not adequately treated. Finally, high levels of antibodies to glutamic acid decarboxylase (GAD) are markers not only for the stiff person syndrome (frequently) but also for other immune-mediated CNS diseases such as limbic encephalitis, and antibodies to glycine receptors are beginning to be found in patients with progressive rigidity and startle syndromes.

This area is growing rapidly and there will likely be other neuropsychiatric, psychiatric and seizure disorders with specific antibodies discovered over the coming years.



**Christoph Michel, MD, PhD, Professor of Clinical Neuroscience**

HUG, Clinique de Neurologie  
4, Rue Gabrielle-Perret-Gentil, CH-1211 Genève

**Curriculum vitae**

Born 27.8.1959, Swiss  
Neurology Clinic, University Hospital, and Neuroscience Dept., University Geneva  
Prof. Dr. sc. nat. ETH  
Associate Professor for Clinical Neuroscience, Faculty of Medicine, University Geneva

**Education and appointments:**

- 1988: PhD., Doctor of Natural Sciences of the Swiss Federal Institute of Technology (ETH), Zurich, Switzerland
- 1988-1994: Research Assistant at the Neurology Clinic, University of Zurich
- 1991: Research Fellow at the Dept. of Physics and Psychology, New York University
- 1994-2003: MER at the Neurology Clinic, University Hospital of Geneva
- 1998: Habilitation at the Faculty of Medicine, University of Geneva
- 2003: Associate Professor at the Faculty of Medicine, University of Geneva
- 2003- Head of the Functional Brain Mapping Laboratory, Clinical and Fundamental Neuroscience Department, University of Geneva
- 2005- Director of the EEG core of the Lemanic Biomedical Imaging Center (CIBM) of the Universities and Hospitals of Geneva and Lausanne

**Awards, honours, major responsibilities:**

- Receiver of the "Robert Bing Award 2000" from the Swiss Academy of Medical Sciences
- Chief-Editor of the journal Brain Topography
- Secretary of the Swiss Society for Neuroscience
- President-Elect of the International Society for Functional Source Imaging (ISFSI)
- Member of the Board of the Swiss League against Epilepsy.
- Editorial Board Member of the journals Clinical Neurophysiology, Int. J. Psychophysiology, The Open Neuroimaging Journal, and Frontiers in Integrative Neuroscience

**Major research achievements:**

- Development of Electrical Neuroimaging Methods
- EEG source imaging in Epilepsy
- EEG/fMRI combination
- Characterization of functional microstates of the brain
- Speed of cognitive processing
- State-dependent information processing

### Relevant recent publications:

Britz J, Van De Ville, D, Michel CM. BOLD correlates of EEG topography reveal rapid resting-state network dynamics. *Neuroimage*, in press.

Brodbeck V, Spinelli L, Lascano AM, Pollo C, Schaller K, Vargas MI, Wissmeyer M, Michel CM, Seeck M. Electrical source imaging for presurgical focus localization in epilepsy patients with normal MRI. *Epilepsia*, 51(4): 583 – 591.

Mégevand P, Troncoso E, Quairiaux C, Muller D, Michel CM, Kiss JZ. Long-term plasticity in mouse sensorimotor circuits after rhythmic whisker stimulation. *J Neurosci*. 2009; 29: 5326-5335.

Vulliemoz S, Lemieux L, Daunizeau J, Michel CM, Duncan JS. The combination of EEG Source Imaging and EEG-correlated functional MRI to map epileptic networks. *Epilepsia*, 2010; 51:491-505.

Ducommun CY, Michel CM, Clarke S, Adriani M, Seeck M, Landis T, Blanke O. Cortical Motion Deafness, *Neuron*, 2004, 43: 765-777.

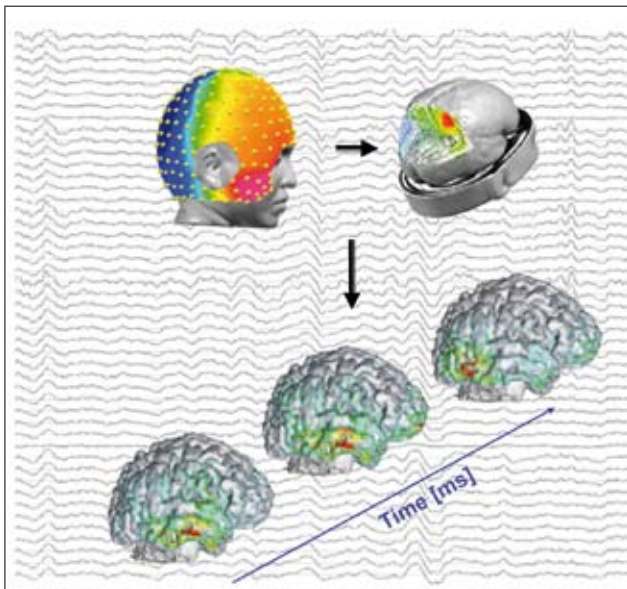
Michel CM, Murray M, Lantz G, Gonzalez S, Grave de Peralta R. EEG Source Imaging. *Clinical Neurophysiology*, 2004, 115: 2195-2222.

### Book:

Michel, C.M., Koenig, T., Brandeis, D., Gianotti, L.R.R., Wackermann, J. *Electrical Neuroimaging*. Cambridge University Press, 2009.

### Update on Advanced Neurophysiology

This talk will demonstrate that the EEG has developed to a functional imaging method. Great advancements have been made in the past years in recording and analyzing of high-resolution EEG. Powerful EEG systems have been built that allow fast and easy recording from hundreds of channels simultaneously. Sophisticated pattern recognition algorithms have been developed to characterize and to detect changes of the topography of the scalp electric fields. New powerful methods for estimating the 3-dimensional source distribution of the scalp EEG have been constructed. The incorporation of anatomical information, as obtained from magnetic resonance imaging in the individual subject, has boosted the use of electrophysiological neuroimaging in experimental studies. It has become a performing neuroimaging method that complements fMRI, because it provides the temporal resolution that allows monitoring the dynamics of the neuronal activity in real time. The high temporal resolution is crucial for the understanding of information exchange in large-scale neuronal networks. While EEG imaging techniques are becoming standard in experimental and cognitive studies, their use in clinical applications is still modest, albeit growing. This talk will show the clinical use of high-resolution EEG imaging in different neurological diseases and will show that, in combination with structural and functional magnetic resonance imaging, EEG neuroimaging provides valuable information on the localization, distribution and temporal propagation of normal as well as pathological neuronal activity in the human brain.



#### Electrical Neuroimaging:

EEG is recorded from a dense array of electrodes covering the entire head (here: 256 channels). At each moment in time the scalp potential map is reconstructed (blue: negative, red: positive). Using distributed linear inverse solutions, the three dimensional distribution of the neuronal generators in the brain is then estimated for each time point and followed over time with millisecond resolution.



**K. Bhatia, MD, PhD, Professor**

Institute of Neurology,  
7 Queen Square, Box 13, UK-London WC1N 3BG

**Curriculum vitae**

**Qualifications:**

- MBBS, Bombay University, 1982
- MD (Internal Medicine), Bombay University, 1986
- DM (Doctorate in Neurology), Bombay University, 1988
- MRCP Royal College of Physicians, London, UK, 1999
- FRCP Royal College of Physicians, London, UK, 2002

**Previous and other appointments:**

Feb 1997-Sep 2002: Senior Lecturer Clinical Neurology, Institute of Neurology, Honorary consultant neurologist, NHNN  
Oct 2002-Sep 2005: Reader as above

**Current grants (PI only):**

- 2005-2008: Astor Foundation studentship for research into Parkinson's Disease awarded for PhD student (Dr S Schneider) under my supervision, £80,000
- 2007-2008: Halley Stewart Foundation Dystonia Society UK research grant for study of endophenotypes in familial cervical dystonia, £32,000

### Recent Publications:

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# FIRST ANNOUNCEMENT



## Gemeinsame Tagung

Jahrestagung Schweizerische Gesellschaft für Klinische Neurophysiologie SGKN  
185. Tagung Schweizerische Neurologische Gesellschaft SNG  
Jahrestagung Schweizerische Hirnschlag Gesellschaft SHG - [www.neurovasc.ch](http://www.neurovasc.ch)  
& SVEPTA Fortbildung

## Réunion commune

Réunion annuelle de la Société Suisse de Neurophysiologie Clinique SSNC  
185e réunion de la Société Suisse de Neurologie SSN  
Réunion annuelle Société Cérébrovasculaire Suisse SCS - [www.neurovasc.ch](http://www.neurovasc.ch)  
& ASATEP Formation continue

**19.-21.05.2011**

Kultur- und Kongresszentrum Luzern

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Deadline abstract submission  
**21.02.2011**