

Supplement to

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Eighteenth Meeting of the European Neurological Society

June 7 – 11, 2008 • Nice, France

European
Neurological Society



Dear Colleagues

It is a great pleasure to welcome you to Nice, capital of the French Riviera, fifth largest city of France and one of its leading convention cities, for the 18th Meeting of the European Neurological Society.

Nice is a unique and vibrant Mediterranean city that has plenty to offer the visitor year-round. Taking a stroll in Nice, seeing the Modern Art Museum, the Old Town, the Colline du Château or the Promenade des Anglais, or enjoying a trip along the breath-taking coastline from Saint-Tropez to Menton, all sound like an invitation to enter the world-famous legends of the French Riviera.

It is a special pleasure to welcome our colleagues from the UK to Nice. Great Britain and France have both influenced the history of neurology tremendously and the Anglo-French relationship remains essential to the study of neurology. Thomas Willis, a pioneer in the research of the anatomy of the brain and nerves, with his work 'Cerebri anatomi' of 1664,

presented enormous scientific progress and coined the term neurology. A modern approach was developed through descriptions of most neurological diseases given by both English and French famous neurologists by the nineteenth century. Sharing once again the experience on the French Riviera may be one of the opportunities of this meeting.

The ENS annual meeting provides an ideal platform for continuing education in all fields of neurology, covering a broad spectrum of topics with state-of-the-art lectures by acknowledged experts. I am convinced that you will enjoy this excellent programme arranged by the ENS Executive Committee and equally convinced that you will enjoy your stay here. On behalf of the Local Organising Committee of the Congress, I am delighted to welcome you to Nice in June 2008.

*Prof C Desnuelle
Chairperson of the 18th ENS Meeting.*



Prof C Desnuelle is Professor of Neurology at the University Hospital of Nice, France. He is currently the head of the Neuroscience Unit and has a keen interest in the investigation of myoclonus and neuromuscular diseases.

European Neurological Society: a leading force in Europe Neurology: Learning, knowledge, progress and the future

The Society

The European Neurological Society (ENS) was founded in 1986, based on the initiative of Gérard Said, Anita Harding and PK Thomas. The ENS represents an effort to break away from a national representation to a membership on an individual basis. This emphasis on individuality underlines the importance of expertise in the various fields of neurology, as well as the singular expression of enthusiasm for clinical and experimental neurology. The ENS has now become the most prominent society for neurologists on the European Continent, and its members excel in the practice and teaching of neurology, including research in which neuroscience plays an important role. The official scientific journal of the ENS is the Journal of Neurology, one of the leading publications in this medical discipline.

The role of the ENS

An academic organisation such as the ENS provides the platform from which clinical and experimental neurologists of various subspecialties can interact and exchange their knowledge and expertise. The society aspires to guide neurologists in their decision-making in order to attain the best possible care for patients with neurological disorders.

The aims of the society are

- to provide continuing education in all fields of neurology
- to create a scientific forum for the presentation of original research work for all neurologists
- to guarantee a high level of scientific standard
- to support the younger generation by continuing promotions such as travel grants, fellowship stipends and the Neurologist in Training Offer

The ENS is especially eager to support, encourage and guide young neurologists. In order to facilitate international contact of young physicians among themselves and with leading experts, participation in our meetings is actively promoted. Every year, travel grants to junior abstract authors whose papers have been accepted for presentation at the meeting are distributed.

In addition, the ENS has started the very successful 'Young neurologist in training programme' in 2006, which offers a limited number of grants providing free accommodation (four nights), free registration and free admission to three teaching courses. Accordingly, the ENS annual meeting becomes an attractive forum for young scientists for learning and networking, which enhances the scope of their activities and possibilities.

Furthermore, the ENS has been supporting young neurologists with fellowships

for many years. ENS sponsors this programme to provide an opportunity for talented researchers to participate in an exchange of scientific activities between home and host institutions.

Annual meetings

The ENS organises a scientific meeting every year, which provides the ideal platform for continuing education in all fields of neurology, covering a broad spectrum of topics with state-of-the-art lectures by acknowledged experts. The ENS is dedicated to giving the congress attendees the highest quality continuing medical education as well as to open professional education opportunities. The ENS provides a wide range of programme formats, including main symposia, teaching courses, workshops, interactive case presentations and oral and poster sessions.

The ENS 2009 annual meeting will take place in Milan / Italy, 20-24 June 2009. This meeting will again be the primary stage for the latest developments in scientific research, where neurologists share innovative research.

ENS Subcommittees

The ENS Executive Committee has set up a series of subcommittees in order to increase involvement of ENS members in policy decisions representative of the diversity within the neurological field. The subcommittees aim to promote and advance the continuing education within their neurological specialities. Each ENS member can join an ENS subcommittee to actively play a part in contributing to the scientific programmes of the ENS annual meetings and to promote the growth and excellence of the subspecialty. The subcommittees serve as a place to exchange ideas and to network, an immensely important point with the increasing globalisation of the world.

European Neurological Society



PARKINSON'S & ON & ON & ON & ON & ON & ON &

APO-go: treatment for both
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symptoms of Parkinson's disease

Predictable symptoms: Use **APO-go Pen** early in treatment plan for rapid reversal of impending "off's". Simple, easy to use sc injection

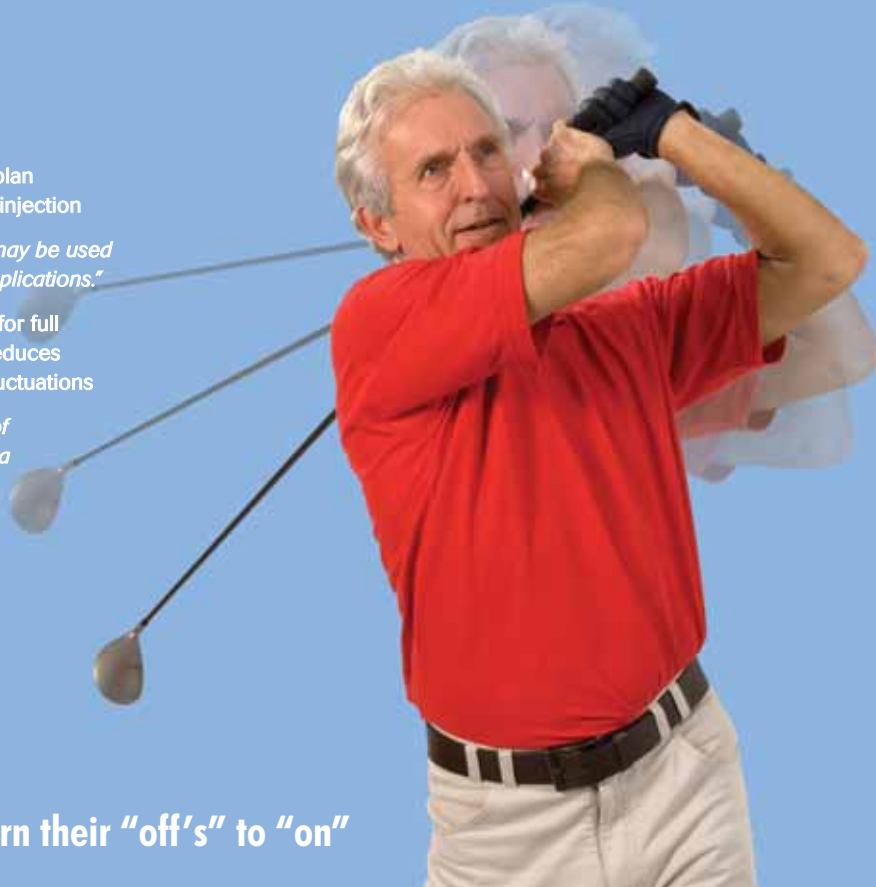
Positive NICE review: "Intermittent apomorphine injections may be used to reduce "off" time in people with PD with severe motor complications."

Unpredictable symptoms: Use continuous **APO-go infusion** for full waking-day cover. Continuous dopaminergic stimulation – reduces pulsatile treatment-related complications including "on-off" fluctuations

Positive NICE review: "Continuous subcutaneous infusions of apomorphine may be used to reduce "off" time and dyskinesia in people with PD with severe motor complications."



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ABRIDGED PRESCRIBING INFORMATION

Consult Summary of Product Characteristics before prescribing. **Uses** The treatment of disabling motor fluctuations ("on-off" phenomena) in patients with Parkinson's disease which persist despite individually titrated treatment with levodopa (with a peripheral decarboxylase inhibitor) and/or other dopamine agonists. **Dosage and administration** Apomorphine hydrochloride is administered subcutaneously either as an intermittent bolus injection or by continuous subcutaneous infusion. Its rapid onset (5–10 mins) and duration of action (about 1 hour) may prevent an "off" episode which is refractory to other treatments. Hospital admission under appropriate specialist supervision is necessary during patient selection and when establishing a patient's therapeutic regime. Please refer to the Summary of Product Characteristics for full details before initiating therapy. Treatment with domperidone (typical dosage 20mg three times a day) before and during apomorphine HCl therapy is essential. The optimal dosage of apomorphine HCl has to be determined on an individual patient basis; individual bolus injections should not exceed 10mg and the total daily dose should not exceed 100mg. **Contraindications** Children and adolescents (up to 18 years of age). Known sensitivity to apomorphine or any other ingredients of the product. Respiratory depression, dementia, psychotic diseases or hepatic insufficiency. Intermittent apomorphine HCl treatment is not suitable for patients who have an "on" response to levodopa which is marred by severe dyskinesia or dystonia. **Pregnancy and lactation** Caution should be exercised if prescribing apomorphine to pregnant women and women of childbearing age. Breast-feeding should be avoided during apomorphine HCl therapy. **Interactions** Patients should be monitored for potential interactions during initial stages of apomorphine therapy. Particular caution should be given when apomorphine is used with other medications that have a narrow therapeutic window. It should be noted that there is potential for interaction with neuroleptic and antihypertensive agents. **Precautions** Use with caution in patients with renal, pulmonary or cardiovascular disease, or who are prone to nausea and vomiting. Extra caution is recommended during initiation of therapy in elderly and/or debilitated patients. Since apomorphine may produce hypotension, care should be exercised in patients with cardiac disease or who are taking vasoactive drugs, particularly when pre-existing postural hypotension is present. Neuropsychiatric disturbances are common in Parkinsonian patients. APO-go should be used with special caution in these patients. Apomorphine has been associated with somnolence, and other dopamine agonists can be associated with sudden sleep onset episodes, particularly

in patients with Parkinson's disease. Patients must be informed of this and advised to exercise caution while driving or operating machines during treatment with apomorphine. Haematology tests should be undertaken at regular intervals as with levodopa when given concomitantly with apomorphine. Pathological gambling, increased libido and hypersexuality have been reported in patients treated with dopamine agonists, including apomorphine. **Side Effects** Local induration and nodules (usually asymptomatic) often develop at subcutaneous site of injection leading to areas of erythema, tenderness, induration, and (rarely) ulceration. Pruritus may occur at the site of injection. Drug-induced dyskinesias during "on" periods can be severe, and in a few patients may result in cessation of therapy. Postural hypotension is seen infrequently and is usually transient. Transient sedation following each dose of apomorphine may occur at the start of therapy, but this usually resolves after a few weeks of treatment. Nausea and vomiting may occur, particularly when APO-go treatment is initiated, usually as a result of the omission of domperidone. Neuropsychiatric disturbances (including transient mild confusion and visual hallucinations) have occurred during apomorphine therapy, and neuropsychiatric disturbances may be exacerbated by apomorphine. Positive Coombs' tests and haemolytic anaemia have been reported in patients receiving apomorphine and levodopa. Local and generalised rashes have been reported. Eosinophilia has occurred in only a few patients during treatment with apomorphine HCl. Patients treated with dopamine agonists, including apomorphine, have been reported as exhibiting signs of pathological gambling, increased libido and hypersexuality (especially at high doses). Apomorphine is associated with somnolence. Breathing difficulties have been reported. *Prescribers should consult the Summary of Product Characteristics in relation to other side effects.* **Presentation and Basic NHS Cost:** APO-go ampoules contain apomorphine hydrochloride, 10mg/ml, as follows: 20mg in 2ml – basic NHS cost £37.96 per carton of 5 ampoules. 50mg in 5ml – basic NHS cost £73.11 per carton of 5 ampoules. APO-go pens (disposable multiple dosage injector system) contain apomorphine hydrochloride 10mg/ml, as follows: 30mg in 3ml – basic NHS cost £123.91 per carton of 5 pens. APO-go Pre-filled Syringes contain apomorphine hydrochloride, 5mg/ml, as follows: 50mg in 10ml – basic NHS cost £73.11 per carton of 5 syringes. **Marketing Authorisation Numbers:** APO-go Ampoules: 05928/0020 APO-go Pens: 05928/0021 APO-go Pre-filled Syringes: 05928/0025 **Legal Category:** POM. **Date of Last Revision:** August 2007. For further information please contact: Britannia Pharmaceuticals Limited 41-51 Brighton Road, Redhill, Surrey RH1 6YS Version Number: APG.API.V6

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The Boundaries of Consciousness: Lessons from Coma and Related States

After severe acute brain damage patients may go through different clinical entities encompassing brain death, coma, vegetative (VS), minimally conscious (MCS) or locked-in (LIS) states. Some of these conditions are irrevocable, others may be temporary. Here, we review residual brain function as measured by EEG, event related potentials (ERP) and functional neuroimaging (e.g. positron emission tomography (PET) and functional MRI) in each of these challenging clinical entities. Consciousness is a multifaceted concept that has two dimensions: arousal or wakefulness (i.e. level of consciousness) and awareness (i.e. content of consciousness) – see Figure 1.

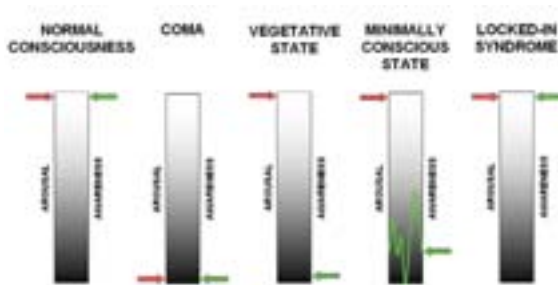


Figure 1: The two dimensions of consciousness: arousal (red arrow) and awareness (green arrow) and their alterations in coma, the vegetative state, the minimally conscious state and in the locked-in syndrome.

Brain death

The EEG in brain death (see table for clinical criteria) is isoelectric (sensitivity and specificity around 90%). This makes the EEG the preferred confirmatory test. Somatosensory evoked potentials typically show arrest of conduction at the cervico-medullary level. Cerebral angiography and transcranial Doppler sonography document with very high sensitivity and 100% specificity the absence of cerebral blood flow. Radionuclide cerebral imaging demonstrate the hollow-skull sign (see Figure 2) confirming the absence of neuronal function in the whole brain.¹

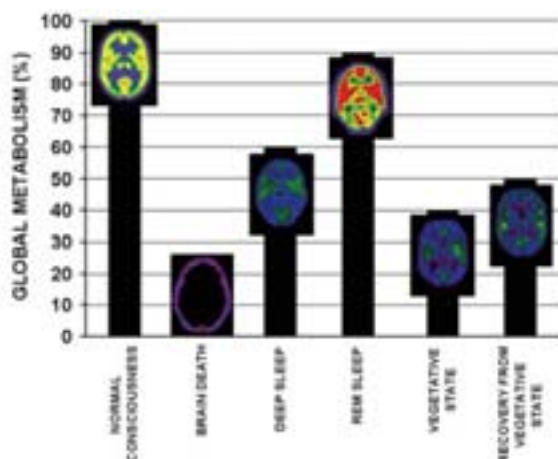


Figure 2: Cerebral metabolism in conscious wakefulness; in brain death (hollow skull sign confirming the absence of neuronal function in the whole brain); physiological (slow wave sleep) modulation of arousal reflecting massive global decreases in cortical metabolism (in REM sleep metabolic activity is paradoxically prominent); and in wakefulness without awareness (i.e., the vegetative state). Note that recovery from the vegetative state may occur without substantial increase in overall cortical metabolism, emphasizing that some areas in the brain are more important than others for the emergence of awareness.

Coma

The EEG tends to become nonreactive and slower as the depth of coma increases. Bilateral absence of cortical potentials (i.e., N20) on somatosensory evoked potentials heralds bad outcome. The presence of 'mismatch negativity' in auditory 'oddball' paradigms is predictive of recovery of consciousness (at least MCS).²

Cortical metabolism in coma survivors on average is 50-70% of normal values. A global depression of cerebral metabolism is not unique to coma. When anaesthetic drugs are titrated to the point of unresponsiveness, the resulting reduction in brain metabolism is similar as that observed in pathological coma. Another example of transient metabolic depression can be observed during slow wave sleep. In this daily physiological condition cortical cerebral metabolism can drop to nearly 40% of normal values - while in REM-sleep metabolism returns to normal waking values.³

Vegetative state

In the vegetative state the EEG most often shows a diffuse slowing, only sporadically is it isoelectric. Somatosensory evoked potentials may show preserved primary somatosensory cortical potentials and brainstem auditory evoked potentials often show preserved brainstem potentials. Endogenous evoked potentials measuring for example the brain's response to complex auditory stimuli such as the patient's own name (as compared to other names) permits one to record a P300 response. Recent data show that the P300 is not a reliable marker of awareness but rather signs automatic processing, as it could be recorded in well-documented VS patients who never recovered.⁴

In contrast to the functional decapitation observed in irreversible coma or brain death, vegetative patients show substantially reduced (40-50% of normal values) but not absent overall cortical metabolism. In some vegetative patients who subsequently recovered, global metabolic rates for glucose metabolism did not show substantial changes. Hence, the relationship between global levels of brain function and the presence or absence of awareness is not absolute. It rather seems that some areas in the brain are more important than others for its emergence.

Statistical analyses of metabolic PET data have identified a dysfunction in a wide frontoparietal network encompassing the polymodal associative cortices: bilateral lateral frontal regions, parieto-temporal and posterior parietal areas, mesiofrontal, posterior cingulate and precuneal cortices. However, awareness seems not exclusively related to the activity in this 'global workspace' cortical network but, as importantly, to the functional connectivity within this system and with the thalami. Long-range fronto-parietal and thalamo-cortical (with non-specific intralaminar thalamic nuclei) 'functional disconnections' have been identified in the VS. Moreover, recovery is paralleled by a functional restoration of this frontoparietal network and part of its thalamo-cortical connections.⁵

The most relevant question regards possible residual consciousness in 'vegetative' patients. In well-documented VS, noxious stimulation resulted in activation of brainstem, thalamus and primary somatosensory cortex but hierarchically higher-order areas of the pain matrix encompassing the anterior cingulate cortex failed to activate.⁶ Importantly, the activated cortex was isolated and functionally disconnected from the fronto-parietal network, considered critical for conscious perception. Similarly, auditory stimulation in VS was found to activate primary auditory cortices but not higher-order multi-modal areas from which they were disconnected.⁷

Minimally conscious state

Metabolic PET studies are unable to reliably differentiate VS from MCS. Functional neuroimaging can here be of value in differentiating activation patterns measured during external stimulation characteristic of either clinical entity. Complex auditory stimuli with emo-



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Melanie Boly, MD is Research Fellow of the Belgian Funds for Scientific Research at the Coma Science Group. Her interests include the study of recovery of neurological disability and of neuronal plasticity by means of multimodal functional neuroimaging (EEG-fMRI, PET and MEG) and behavioural assessment in severely brain damaged patients with altered states of consciousness.



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tional valence such as personalised narratives⁸ or the patient's own name⁹ activate cortical areas not observed during presentation of meaningless stimuli. Such context-dependent higher-order auditory processing in MCS, often is not assessable at the patient's bedside, and indicates that content does matter when talking to these patients.

However, given the absence of a thorough understanding of the neural correlates of consciousness, functional neuroimaging results must be used with caution as proof or disproof of awareness in severely brain-damaged patients.¹⁰ Recently, Adrian Owen et al.¹¹ proposed a more powerful approach to identify 'volition without action' in non-communicative brain damaged patients (rather than to use passive external stimulation paradigms), by scanning patients when asked to perform a mental imagery task. Reproducible and anatomically specific activation in individual patients during tasks that unequivocally require intentionality for their completion reflect awareness.¹² In one exceptional VS patient, task-specific activation was observed, unequivocally demonstrating consciousness in the absence of reliable behavioural motor signs of voluntary interaction with the environment. Interestingly, the patient subsequently recovered. Other studies also showed that VS patients with atypical brain activation patterns on functional neuroimaging, afterwards showed clinical signs of recovery of consciousness - albeit sometimes many months later.¹³

Investigations using MRI diffusion tensor imaging - which gauges the integrity of the white matter - are increasing our understanding of the brain mechanisms underlying recovery from VS. A team led by Nicholas Schiff, for instance, recently used diffusion tensor imaging to show the re-growth of axons in the brain of Terry Wallis, an Arkansas man in a post-traumatic MCS who started talking in 2003 after 19 years of silence.¹⁴

Locked-in syndrome

Classically, the EEG is relatively normal (or minimally slow) and reactive to external stimuli in LIS - but unreactive alpha rhythms (i.e., 'alpha coma' patterns) may also be observed. Cognitive ERPs and brain computer interfaces may document awareness and permit communication in the extremely challenging cases of complete LIS. Residual cognition in LIS has long remained terra incognita. Recently, standard neuropsychological testing batteries have been adapted and validated for eye-response communication. In classical LIS caused by a brainstem lesion, these studies have shown preserved attention, memory, executive functioning, phonological and lexico-semantic performances.

PET scanning has shown significantly higher metabolic levels in the brains of patients in LIS compared to patients in VS. Voxel-based comparisons with healthy controls showed that no supra-tentorial cortical area has a significantly impaired metabolism in classical LIS. Conversely, a hyperactivity was observed in bilateral amygdala of acute, but not chronic, LIS patients. The amygdala are known to be involved in emotions, especially negative emotions such as fear and anxiety. The absence of metabolic signs of reduced function in any area of the gray matter re-emphasizes the fact that LIS patients suffer from a pure motor de-efferentation and recover an entirely intact intellectual capacity. The increased activity in the amygdala might relate to the terrifying situation of an intact awareness in a mute but sensitive being. Health-care workers should be aware of this condition, adapt their bedside-behavior and consider pharmacological anxiolytic therapy.¹⁵

Table 1: Diagnostic criteria of clinical entities that can be encountered following coma

BRAIN DEATH

- *Demonstration of coma*
- *Evidence for the cause of coma*
- *Absence of confounding factors, including hypothermia, drugs, electrolyte, and endocrine disturbances*
- *Absence of brainstem reflexes*
- *Absent motor responses*
- *Apnoea*
- *A repeat evaluation in 6h is advised, but the time period is considered arbitrary*
- *Confirmatory laboratory tests are only required when specific components of the clinical testing cannot be reliably evaluated*

VEGETATIVE STATE

- *No evidence of awareness of self or environment and an inability to interact with others*
- *No evidence of sustained, reproducible, purposeful, or voluntary behavioural responses to visual, auditory, tactile, or noxious stimuli*
- *No evidence of language comprehension or expression*
- *Intermittent wakefulness manifested by the presence of sleep-wake cycles*
- *Sufficiently preserved hypothalamic and brainstem autonomic functions to permit survival with medical and nursing care*
- *Bowel and bladder incontinence*
- *Variably preserved cranial-nerve and spinal reflexes*

MINIMALLY CONSCIOUS STATE

Reproducible or sustained observation of at least one of the following:

- *Purposeful behaviour (including movements or affective behaviour that occur in contingent relation to relevant environment stimuli and are not due to reflexive activity)*
- *Following simple commands*
- *Gestural or verbal yes/no response (regardless of accuracy)*
- *Intelligible verbalization*

EMERGENCE FROM THE MINIMALLY CONSCIOUS STATE

Reliable and consistent demonstration (on two consecutive evaluations) of at least one of the following:

- *Functional interactive communication: accurate yes/no responses to six of six basic situational orientation questions*
- *Functional use of two different objects*

LOCKED-IN SYNDROME

- *Sustained eye opening (bilateral ptosis should be ruled out as a complicating factor)*
- *Quadriplegia or quadriparesis*
- *Aphonia or hypophonia*
- *A primary mode of communication that uses vertical or lateral eye movement or blinking of the upper eyelid to signal yes/no responses*
- *Preserved awareness of the environment*

Conclusion

At the bedside, the evaluation of consciousness in these conditions is challenging and sometimes erroneous. Electrophysiological and functional neuroimaging studies can objectively describe the cerebral activity at rest, and under various conditions of passive stimulation and during 'active' mental imagery tasks. These studies are increasing our understanding of the neural correlates of consciousness and will improve the diagnosis, prognosis and management of disorders of consciousness.

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The Scientific Programme of the ENS 2008 annual meeting is designed to offer a whole perspective on what is state-of-the-art in the field of neurology. This year the Scientific Programme includes five symposia:

Presidential Symposium Coma and locked-in syndrome

Chair: G Moonen / Liège

Topics:

- Coma and locked-in syndrome – Disorders of consciousness – G Moonen / Liège
- Multimodal neuroimaging studies in coma and related conditions – N Schiff / New York
- Detecting awareness in non-communicative patients using fMRI – A Owen / Cambridge
- Thought translation devices in the locked-in syndrome – A Kübler / Tubingen

Symposium Behavioural disorders and dementia

Chair: E Scarpini / Milan

- Physiopathological bases of behaviour – T Robbins / Cambridge
- Synucleinopathies (Parkinson, Levy body) – S Cappa / Milan
- MCI and Alzheimer – H Feldman / Vancouver
- Tauopathies (Fronto-temporal, PSP, etc.) – E Scarpini / Milan

Symposium Autoimmune disorders of the nervous system

Chair: R Hohlfeld / Munich

- Latest developments in Multiple Sclerosis – G Comi / Milan
- Autoimmune diseases of the neuromuscular junction – A Vincent / Oxford
- Pathogenesis and treatment of the Guillain-Barré syndrome – H-P Hartung / Dusseldorf
- Immunopathogenesis of inflammatory myopathies – R Hohlfeld / Munich

Symposium Multiple sclerosis: when to start a treatment and which treatment

Chairs: I Milonas / Thessaloniki, L Kappos / Basel

- The rationale for early treatment in multiple sclerosis – A Compston / Cambridge
- Starting treatment after a CIS – G Comi / Milan
- Starting treatment when definite MS is diagnosed – C Confavreux / Lyon
- Immunosuppressants as first choice, followed by immunomodulatory agents – RP Lisak / Detroit

Symposium Transient ischaemic attack (TIA): imaging and management

Chair: P Amarenco / Paris

- Diagnosis and risk assessment for TIAP – M Rothwell / Oxford
- Yield of brain and vascular imaging (MRI, ultrasound etc.) in the diagnosis of TIA – MG Hennerici / Mannheim
- Feasibility and efficacy of ultra early evaluation and intervention after a TIA – P Amarenco / Paris
- The concept of the TIA clinic – P Canhão / Lisbon

In addition to the symposia, the Scientific Programme includes five Poster Sessions and approximately 16 Oral Sessions of free communications. The ENS has always been known for the practical and education side of its annual meetings. This year again, the participants have the chance to hear about the latest techniques and tools in practice in teaching courses, workshops, interactive case presentations and practical sessions in clinical neurophysiology.

Teaching Courses

The teaching courses are led by the world's leading experts who share their personal experiences and guide the participants through all aspects of neurology. To name just a few highlights: E Melamed (Petach Tikva/IL) and I Litvan (Louisville/USA) chair the course 'Parkinson's disease and related disorders'. This course, as three others, is given in collaboration between the ENS and the AAN. The teaching course 'Practical issues in movement disorders', chaired by MJ Vidailhet (Paris / FR) and E Ruzicka (Prague / CZ), focuses on the diagnosis strategy, the useful tests, the therapeutic approach and the beneficial outcome of psychogenic movement disorders. Up-to-date and practical advice for physicians involved in epilepsy management is given in the teaching course 'Epilepsy', which is chaired by H Cock (London / UK) and T Tomson (Stockholm / SE).

Workshops

For the first time, the ENS Subcommittees have organised various workshops covering all fields of neurology. Programme topics were chosen by the subcommittees and the programme blends clinical neurology, diagnostic approaches and treatment on a variety of neurological aspects. The workshop 'Practical guidelines for the use of teleneurology and telestroke' for example, discusses the rapidly developing application of telemedicine in clinical neurology (focusing on stroke) where medical information is transferred via IT networks for the purpose of consulting or sometimes even for examining and treating. V Dietz (Zurich / CH) and W Schupp (Herzogenaurach / DE) chair the workshop 'Stroke rehabilitation – challenges and tasks'. This workshop focuses on the best strategies and therapeutic measures of stroke rehabilitation. The workshop 'Training in neurology in the USA', attempts to clarify the rules and regulations concerning subspecialty training of European neurologists in the US. This workshop is chaired by S Sergay (Tampa / USA), current president of the AAN, and is particularly interesting for your physicians.

Interactive Case Presentations – What is your decision?

The Interactive Case Presentations cover a wide range of clinical neurology topics. These sessions allow participants to interact actively in the session via an electronic voting system.

Practical Breakfast Sessions in Clinical Neurophysiology

Practical demonstrations in the field of clinical neurophysiology take place. These sessions allow participants the opportunity to practise their diagnostic skills. Basic and advanced courses are available.



Giovanni Abbruzzese	Genova, Italy	Lüder Deecke	Wien, Austria
Sharon Abrahms	Edinburgh, United Kingdom	Marcus Deschauer	Halle, Germany
Pierre Amarengo	Paris, France	Claude Desnuelle	Nice, France
Maria Pia Amato	Firenze, Italy	Feza Deymeer	Capa Istanbul, Turkey
Birgit Andersen	Copenhagen, Denmark	Marianne Dieterich	Mainz, Germany
Corrado Angelini	Padova, Italy	Volker Dietz	Zürich, Switzerland
Zohar Argov	Jerusalem, Israel	Ruth Djaldetti	Petach Tikva, Israel
Jean-Claude Baron	Cambridge, United Kingdom	BrunoDubois	Paris, France
Claudio Bassetti	Zürich, Switzerland	Anne Ducros	Paris, France
Rachid Bech-Azeddine	Copenhagen, Denmark	Mefküre Eraksoy	Capa Istanbul, Turkey
Diederik Van de Beek	Amsterdam, Netherlands	Bruno Eymard	Paris, France
Andreas Bender	München, Germany	Cristian Falup-Pecurariu	Brasov, Romania
Fabrizio Benedetti	Torino, Italy	Franz Fazekas	Graz, Austria
Thomas Berger	Innsbruck, Austria	Howard Feldman	Vancouver, Canada
Kailash Bhatia	London, United Kingdom	Christina van der Feltz	Amsterdam, Netherlands
Alexandre Bisdorff	Esch sur Alzette, Luxembourg	Cesar Fernández-de-las-Peñas	Alcorcon / Madrid, Spain
Bastiaan Bloem	Nijmegen, Netherlands	Massimo Filippi	Milano, Italy
Ulrich Bogdahn	Regensburg, Germany	Roy Freeman	Boston, United States
Thomas Brandt	München, Germany	Daniela Galimberti	Milano, Italy
Adolfo M Bronstein	London, United Kingdom	Juan CarlosGarcia Monco	Galdacano, Spain
Martin M Brown	London, United Kingdom	Thomas Gasser	Tübingen, Germany
Paul Burgess	London, United Kingdom	Jan van Gijn	Utrecht, Netherlands
David J Burn	Newcastle-upon-Tyne, United Kingdom	Peter Goadsby	London, United Kingdom
Kate Bushby	Newcastle-upon-Tyne, United Kingdom	Benjamin D Greenberg	Baltimore, United States
Patricia Canhao	Lisbon, Portugal	Wolfgang Grisold	Wien, Austria
Stefano F Cappa	Milano, Italy	Orla Hardiman	Dublin 9, Ireland
Michel Clanet	Toulouse Cedex, France	Hans-Peter Hartung	Düsseldorf, Germany
Hannah Cock	London, United Kingdom	Christoph Helmstädter	Bonn, Germany
Giancarlo Comi	Milano, Italy	Michael G Hennerici	Mannheim, Germany
David AS Compston	Cambridge, United Kingdom	Jan Herzog	Kiel, Germany
Christian Confavreux	Lyon Cedex 03, France	Khè Hoang-Xuan	Paris, France
Marinos C Dalakas	Bethesda, United States	Reinhard Hohlfeld	München, Germany
Adrian Danek	München, Germany	Guntram Ickenstein	Aue, Germany
Marianne De Visser	Amsterdam, Netherlands	Ludwig Kappos	Basel, Switzerland

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List of Invited Speakers

Giorgos Karas	Amsterdam, Netherlands
Jürg Kesselring	Valens, Switzerland
Ramin Khatami	Zürich, Switzerland
Matthew Kiernan	Randwick, Australia
Hermann Kingma	Maastricht, Netherlands
MJ Klein	Amsterdam, Netherlands
Matthias Klein	München, Germany
Hans-Helmut Kornhuber	Blaubeuren-Altental, Germany
Christian Krarup	Copenhagen, Denmark
Andrea Kübler,	Tübingen, Germany
Ron Kupers	Aarhus, Denmark
Laura Kupila	Turku, Finland
Pierre Landrieu	Le Kremlin Bicêtre, France
Steven Laureys	Liège, Belgium
Giuseppe Lauria	Milano, Italy
Thomas Lempert	Berli, Germany
Richard Lewis	Detroit, United States
Didier Leys	Lille, France
Robert P Lisak	Detroit, United States
Irene Litvan	Louisville, United States
Andrew Lockhart	Cambridge, United Kingdom
Pierre Lozeron	Le Kremlin Bicêtre, France
Catherine Lubetzki	Paris Cedex 13, France
Hadi Manji	London, United Kingdom
Marie-Hélène Marion	London, United Kingdom
Heinrich Mattle	Bern, Switzerland
Carlos Matute	Leioa, Spain
Mikael Mazighi	Paris, France
Colin T McDonald	Wellesley, United States
Vincent Meininger	Paris, France
Eldad Melamed	Petach Tikva, Israel
A David Mendelow	Newcastle-upon-Tyne, United Kingdom
Patrik Michel	Lausanne, Switzerland
David H Miller	London, United Kingdom
Ioannis Milonas	Thessaloniki, Greece
Pasquale Montagna	Bologna, Italy
Xavier Montalban	Barcelona, Spain
Gustave Moonen	Liège, Belgium
Oswaldo Nascimento	Rio de Janeiro, Brazil
Lino Nobili	Milano, Italy
Víctor Oliveira	Lisbon, Portugal
Adrian Owen	Cambridge, United Kingdom
Véronique Paquis	Nice Cedex 1, France
Davide Pareyson	Milano, Italy
Yesim Parman	Istanbul, Turkey
Julio Pascual	Salamanca, Spain
Hans-Walter Pfister	München, Germany
Daniela Pohl	Göttingen, Germany
Pierre Pollak	Grenoble, France
Mary Reilly	London, United Kingdom
Patrício F Reyes	Phoenix, United States
Wim Robberecht	Leuven, Belgium
TW Robbins	Cambridge, United Kingdom
Andrea Rossetti	Lausanne, Switzerland
Peter M Rothwell	Oxford, United Kingdom
Evzen Ruzicka	Prague, Czech Republic
Gérard Said	Charenton Le Pont, France
Elio Scarpini	Milano, Italy
Nicole Schaeren-Wiemers	Basel, Switzerland
Angelo Schenone	Genova, Italy
Pedro Schestatsky	Barcelona, Spain
Nicholas Schiff	New York, United States
Erich Schmutzhard	Innsbruck, Austria
Jean Schoenen	Liège, Belgium
Wilfried Schupp	Herzogenaurach, Germany
Stefan Schwab	Erlangen, Germany
Armando Sena	Lisbon, Portugal

Jan Senderek	Aachen, Germany
Klaus Seppi	Innsbruck, Austria
Steven Sergay	Tampa, United States
Jordi Serra Catafau	Barcelona, Spain
Vincenzo Silani	Milano, Italy
Michael Sinnreich	Montreal, Canada
Kenneth J Smith	London, United Kingdom
Riccardo Soffietti	Torino, Italy
Claudia Sommer	Würzburg, Germany
Andreas Steck	Basel, Switzerland
Guido Stoll	Würzburg, Germany
Michael J Strong	London, Canada
Vincent Timmerman	Antwerpen-Wilrijk, Belgium
François Tison	Pessac, France
Torbjörn Tomson	Stockholm, Sweden
Daniilo Toni	Roma, Italy
Antonio Toscano	Messina, Italy
Jacques Touchon	Montpellier, France
Klaus Viktor Toyka	Würzburg, Germany
Bryan J Traynor	Washington, United States
David M Treiman	Phoenix, United States
Josep Valls-Solé	Barcelona, Spain
Marie José D Vidailhet	Paris, France
Angela Vincent	Oxford, United Kingdom
Jens Volkmann	Kiel, Germany
Ruth Walker	Bronx, United States
Thomas Warner	London, United Kingdom
Wolfgang Wick	Heidelberg, Germany
Carsten I Wikkelsø	Göteborg, Sweden
Jérôme Yelnik	Paris, France

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FENS Forum 2008

Brain Awareness Week Reception: BAW and Beyond

This reception event will provide a forum to discuss Brain Awareness Week, an international week of events celebrating brain science that takes place every March. BAW in Europe is organised by the European Dana Alliance for the Brain, an organisation that promotes brain research.

Whether you're a participant in BAW or you'd just like to find out more about it, join us for a free lunch and a discussion about this international programme of events.

Venue: Room Cervin, Palexpo, Geneva

Date: Sunday 13 July 2008

Time: 11.45 – 13.45

All FENS delegates welcome!



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Pharmaceutical Industry Satellite Symposia Schedules at the ENS Meeting

Advancements in novel drug developments and treatments by the pharmaceutical industry for a wide spectrum of neurological disorders has taken on a fast pace and there is an increasing need for comprehensive Satellite Symposia. Participants will be informed as to advancements currently taking place in the pharmaceutical industry, along with insights as to the possibilities of future innovations. Topics and schedules of these Satellite Symposia are given below (as per 20 March 2008).

Pathophysiology and diagnosis of dementias: role of functional and structural techniques

Serono Symposia International Foundation – Sunday, June 8, 2008 – lunch time

Treating Alzheimer's Disease: Is time on our side?

Eisai / Pfizer – Sunday, June 8, 2008 – early evening

The Role of IVIg in Alzheimer's Disease

Baxter – Monday, June 9, 2008 – lunch time

Advancing early treatment in MS – new insights into a proven choice

Bayer Schering – Monday, June 9, 2008 – lunch time

Narcolepsy: a clinician approach review

UCB – Monday, June 9, 2008 – lunch time

Dopamine agonists – Recent advances in treating Parkinson's disease and restless legs syndrome

Boehringer Ingelheim – Monday, June 9, 2008 – early evening

Next generation solutions in neurology: new treatments, better prospects

Merck Serono – Monday, June 9, 2008 – early evening

The multidimensions of vertigo and dizziness

Solvay – Monday, June 9, 2008 – early evening

Targeting the pathophysiology of MS: Transforming

Biogen Idec / Elan – Tuesday, June 10, 2008 – lunch time

New perspectives on the use of IVIG in neurological disorders

Talecris – Tuesday, June 10, 2008 – lunch time

Advances in the Management of Friedreich's Ataxia

Takeda / Santhera – Tuesday, June 10, 2008 – lunch time

Towards a better management of advanced Parkinson's disease

Solvay – Tuesday, June 10, 2008 – early evening

Advance Programme - www.eano.eu
 Contact: Marieke Hodel, Romana Koenig
 Vienna Medical Academy, Alser Strasse 4, 1090 Vienna, Austria
 T: +43 1 40513830, F: +43 1 4078274, E: eano2008@medacad.org

Annual Scientific Meeting
 9-11th July 2008
 Apex City Quay Hotel, Dundee

Topics include:

- **Genetics and its Relevance to Clinical Practice:**
 ION Channel and Chromosomal Disorders;
 Pharmacogenetics; Complex Inheritance in Epilepsy
- **ILAE UK Chapter and Epilepsy Research UK Basic Science Session:**
 Submitted Topics
- **UK Epilepsy Surgery Network Meeting: Case Discussions**
 Epilepsy Surgery Involving the Posterior Half of the Brain; New and Innovative Treatments in Epilepsy Surgery
- **GABA and Epilepsy: From the Receptor to the Clinic:**
 GABA Receptors: A Review; Depolarization and GABA; GABAergic AEDs
- **Intracranial EEG Monitoring – Different Approaches:**
 Review of EEG Basics; Marseilles Approach; Cleveland Approach
- **Non-Epileptic Attacks and Dissociative Seizures:**
 Epidemiology; Clinical Observations; Conversational Analysis; Management
- **Provision of Care:**
 Managed Clinical Networks for Epilepsy; Measurement of Adherence

Registration includes the conference dinner at historic Guthrie Castle
 For more information please contact Denise Hickman-Rowe, Conference 2k Ltd, Capstan House, Western Road, Pevensey Bay, East Sussex BN24 6HG • Tel: 01323 740612 • denise@conference2k.com
 Full programme, registration forms, abstract and Gowers' forms are available for download at www.ilae-uk.org.uk and www.conference2k.com
ILAE UK Chapter Annual Scientific Meeting - October 2009, Sheffield.

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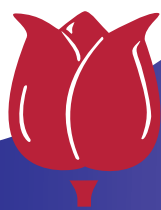
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