



Professor José M Ferro.

Dear Colleagues and ENS Members

It is an honour and a privilege to invite you to the 21st Meeting of the European Neurological Society in Lisbon. It is the first time that the ENS meets in Portugal. Lisbon was a natural choice not only because of the facilities available for international meetings, but also because of its charm, its unique light, its multicultural ambience and its splendid geographical location, surrounded by the River Tagus, the Atlantic Ocean and the Cascais-Sintra Natural Park.

The ENS Meeting offers an exclusive opportunity for excellent continuous education in all fields of neurology, both for practising neurologists and for young neurologists in training.

The ENS meeting is also an international stage where those devoted to clinical or translational research can present and discuss the results of their work in an open, friendly but testing environment.

Lisbon and its surroundings offer the participants of the 21st ENS Meeting a vast display of activities to enjoy in the pre- and post-meeting hours or days. Whether walking in the narrow streets of Alfama, relaxing on an esplanade by the river, dining out on delicious traditional fare or on Portuguese new-style cuisine, or exploring the vibrant night-life of Lisbon, participants will enjoy the city's warmth and atmosphere and imbibe its history and culture.

We thank the Administrative ENS Office at Congrex Switzerland for making all necessary preparations for this meeting, to ensure this is a special and memorable event for all participants.

On behalf of the Organizing Committee of the 21st ENS Meeting, it is a pleasure to welcome you to Lisbon in May 2011.

Professor José M Ferro
Chairperson of the 21st ENS Meeting

The Scientific Programme of the ENS 2011 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 seven symposia:

Joint Symposium of the ENS and the Portuguese Neurological Society:

Saturday, 28 May 2011 (17.15-18.45)

Familial amyloid polyneuropathy

Chairs: G. Said (Paris, FR), V. Oliveira (Lisbon, PT)

Clinical aspects and management – I. Conceicao (Lisbon, PT)

The genetics of FAP: A global problem – V. Planté-Bordeneuve (Paris, FR)

Treatment of FAP by liver transplantation – O. Suhr (Umea, SE)

New pharmacological treatment – T. Coelho (Porto, PT)

Presidential Symposium:

Sunday, 29 May 2011 (17.30-19.30)

Treatment of muscle diseases: The future is already here

Chair: Z. Argov (Jerusalem, IL)

Can we bypass a muscle metabolic defect? – Z. Argov (Jerusalem, IL)

Exercise therapy in muscle disease: A current overview

– T. Taivassalo (Montreal, CA)

Antisense therapy of muscular dystrophies – F. Muntoni (London, UK)

Gene therapy for myopathies – J. Mendell (Columbus, US)

Symposium: Monday, 30 May 2011 (09.00-11.00)

Molecular and cellular mechanisms of ischaemic stroke

Chair: G. Stoll (Würzburg, DE), J. Ferro (Lisbon, PT)

Novel molecular targets for acute stroke treatment – G. Stoll (Würzburg, DE)

Brain-immune interactions, infection, and inflammation in acute stroke

– X. Urra (Barcelona, ES)

Hypothermia: From animal models and translation to humans

– S. Schwab (Erlangen, DE)

Stem cells in experimental stroke: Translation to humans?

– E. Díez Tejedor (Madrid, ES)

Symposium: Monday, 30 May 2011 (09.00-11.00)

Psychiatric aspects of neurological disorders

Chair: H. Förstl (Munich, DE)

Behaviour and theory of mind in frontotemporal dementia

– H. Förstl (Munich, DE)

Psychiatric aspects of PSP, MSA and ALS – A.C. Ludolph (UlM, DE)

Psychiatric symptoms in Parkinson's disease – E. Zuckica (Prague, CZ)

Behavioural and psychological symptoms of dementia (BPSD) in

Alzheimer's disease: Update on management – R. Heun (Birmingham, UK)

Symposium: Tuesday, 31 May 2011 (09.00 – 11.00)

Metals and movement disorders

Chair: P. Taba (Tartu, EE)

Wilson's disease – W.H. Oertel (Marburg, DE)

Neurodegeneration with brain iron accumulation (NBIA) syndroms

– K. Bhatia (London, UK)

Manganese-related movement disorders – P. Taba (Tartu, EE)

Metal ions and neuro-degeneration – P. Jenner (London, UK)

Symposium:

Tuesday, 31 May 2011 (09.00 – 11.00)

Biomarkers for diagnosis, prognosis and response to treatment in MS

Chair: R. Martin (Hamburg, DE)

Transcriptomics – R. Martin (Hamburg, DE)

Genetic markers – J. Hillert (Stockholm, SE)

CSF markers – G. Giovannoni (London, UK)

Magnetic resonance imaging – M. Filippi (Milan, IT)

Symposium:

Tuesday, 31 May 2011 (17.30 – 19.30)

G rard Said Farewell Symposium

Welcome – G. Moonen

Electrophysiological studies in peripheral nerve disorders

– C. Krarup (Copenhagen, DK)

Diabetic neuropathy – G. Llewelyn

Of MAG and neuropathy – A. Steck (Basel, CH)

Is nerve biopsy still useful? – G. Said (Paris, FR)

Closing remarks – Z. Argov (Jerusalem, IL)

In addition to the symposia, the scientific programme includes four poster sessions and various oral sessions of free communications. The ENS has always been known for the practical and educational 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.

Teaching Courses

The teaching courses are led by leading European experts who share their personal experiences and guide the participants through all aspects of neurology. To name just a few highlights: “*Key differential diagnosis and management issues*” will focus on the clinically isolated syndrome which in many cases are diagnosed as early MS, stroke in the young, and the ALS-suggestive motor neuron symptoms. Especially the question who should be treated how and when, will be addressed. The “*Non-Alzheimer diseases*” course will provide an update on the diagnosis, criteria, pathophysiology and treatments of the main non-Alzheimer dementias, examining the metabolic and vascular causes of dementia, frontotemporal lobar degeneration, and dementia with Lewy bodies. Prognosis and management differ, and the differential diagnosis is necessary to assess disease-modifying therapeutic strategies. The teaching course “*Effects and complications of long-term treatment in MS*” will address the development of more treatment options which raises questions concerning their long term-effects. The course will conclude with a discussion on prospective long-term follow up studies and risk management plans. The goal of the course “*Advances in the diagnosis of seizures and epilepsy*” will be to update the audience on semiological aspects of seizures, to give information on the added value of EEG and current optimum MRI protocols and to demonstrate the current state-of-the art of MEG in epilepsy. Three teaching courses will be given in a collaboration between the ENS and AAN: “*Update in neuro-oncology*”, “*White matters: Inflammatory and genetic/metabolic leukoencephalopathies in young patients*” and “*The neurology of unconsciousness*”.

Workshops

The ENS Subcommittees have organised various workshops covering all fields of neurology. The programme blends clinical neurology, diagnostic approaches and treatment on a variety of neurological aspects. The faculty – all noted experts in their fields – will cover individual topics over a 90 – minute period.

Subjective memory complaints are an extremely frequent cause of neurological consultation, and represent the typical presentation of several neurodegenerative disorders, Alzheimer's disease in the first place. The present evidence on differential diagnoses among the causes of subjective memory complaints, neurological and psychiatric, will be reviewed in the workshop “*How to manage subjective memory complaints*”. The workshop “*fMRI to probe motor recovery from stroke rehabilitation*” will critically discuss the role of fMRI to probe motor recovery after stroke, provide information concerning the pathophysiologic underpinnings, technical possibilities and challenges and summarises the converging evidence from the studies available so far. Within the workshop “*Assessment of regional atrophy in clinical neurology*” useful technical and practical hints on the methods of analysis of regional atrophy will be given for clinicians and researchers. Suggestions on how to apply MRI-based methods and post-processing analysis tools in clinical neurology will be given. The workshop “*Pimp up your residency*” aims to discuss current challenges that neurology residents and medical students face, with a view to help their training, to give hands-on information for a training period abroad and tips for the major European fellowships. During this workshop an update on the European neurology e-learning project will also be presented.

Interactive Case Presentations

Participants are invited to present and discuss personal cases with their colleagues. Through an interactive voting system, different opinions can be collected. The following topics will be covered: Clinical pitfalls, disorder of gait, disorders of high cortical functions, neuroradiological pitfalls, paroxysmal disorders awake, and paroxysmal disorders from sleep.

Practical Sessions in Clinical Neurophysiology

Practical demonstrations in the field of clinical neurophysiology will take place in order to practice diagnostic skills. These sessions will encourage attendees to raise topics for discussion and debate on EMG, nerve conduction studies, reflex studies and SFEMG.

List of Sponsors and Exhibitors as per February 2011

Actelion Pharmaceuticals Ltd.
Biogen Idec International GmbH
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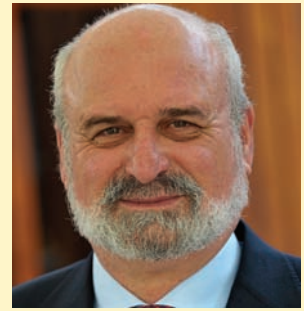
The Presidential Symposium: Treatment of Muscle Diseases: the future is already here

The aim of this symposium is to present the major developments in therapy of human hereditary myopathies.

Introduction

The field of myology has for a long time been short of therapeutic interventions. Apart from the inflammatory (autoimmune) conditions, most of the management of the hereditary myopathies was related to orthopedic complications and general physical therapy. In recent years therapy in myopathies has been a rapidly evolving field that has reached human trials.

Progress is based on accumulated knowledge from animal models and those methods that matured to be applied to patients are currently being tested. In this year's ENS Presidential Symposium, the scientific basis and the current status of metabolic therapies, exercise prescriptions, antisense oligonucleotide use and viral-mediated gene therapy will be discussed.



**Professor Zohar Argov,
ENS President 2011-2,**

is a Professor of Neurology, and Kanrich Chair of Neuromuscular Diseases at the Hadassah-Hebrew University Medical Center, Jerusalem, Israel. Also he is an Adjunct Professor of Neurology at the Montreal Neurological Institute and Chair of the Israeli Society of Neuromuscular Diseases.

Can we bypass a muscle metabolic defect? (Zohar Argov, Jerusalem, Israel)

The metabolic myopathies seem to be more amenable to treatment once the biochemical defect is identified. Some of the conditions cause mainly exercise intolerance and its improvement can be regarded as therapeutic success. In others, muscle weakness and degeneration occurs and functional and strength improvement is the goal.

Therapy of such disorders can be achieved through enzyme replacement (e.g. Pompe's

disease) or its upregulation (e.g. bezafibrate in CPT2 deficiency). Other methods include: 1. supplementation of a missing compound (e.g. CoQ10 which is effective only in primary Q10 deficiency but is given to most patients with other mitochondrial disorders); 2. use of metabolic 'cocktails' to increase the muscle oxidative capacity of muscle (combinations of oxygen species scavengers like menadione with K3, vitamin C, riboflavin, carnitine and creatine); 3. increasing availability of compen-

satory fuel sources (e.g. sucrose given before exercise or a carbohydrate-rich diet in McArdle's disease). 4. providing metabolic intermediates that are downstream to the enzymatic defective site (e.g. ManNac or sialic acid for HIBM with defects in GNE, an enzyme in the synthetic pathway of sialic acid).

Because of the rarity of metabolic myopathies, no proper double blind studies were performed to assess these therapies and recommend an agreed protocol.

Exercise therapy in muscle disease: a current overview (Tanja Taivassalo, Montreal, Canada)

Exercise intolerance is a common clinical presentation in neuromuscular disorders, resulting from the primary disease process or a secondary effect of either cardiovascular deconditioning or muscle disuse due to adoption of sedentary lifestyle. Exercise training is well-established to counter the effects of deconditioning in healthy people and various chronic disease conditions. It has traditionally been discouraged for patients with muscle disorders for fear of exacerbating symptoms as well as lack of evidence-based knowledge on the effects of strength or aerobic exercise on the given disease process. Progress has recently been made regarding the safety and efficacy of exercise training as therapy in various muscle diseases (metabolic and inflammatory myopathies and muscular dystrophies). These studies have assessed the ability of the exercise stimulus (aerobic training, AT, at 60-80% peak heart rate, 20-40 minutes 3 times per week or strength training, ST, at variable intensities 2 to 3 times per week) to reverse deconditioning and affect the disease process.

In GSD type V (McArdle's disease), moderate intensity AT improved exercise capacity (by increasing circulatory delivery

and mitochondrial metabolism of blood-borne fuels) without side effects. Despite these training adaptations, fuel availability continued to limit oxygen utilization and exercise capacity. Due to the concern for muscle injury in this population, the effects of ST on muscle strength were not evaluated.

In a handful of studies assessing exercise therapy in mitochondrial myopathies, AT and ST reversed deconditioning and increased peak exercise capacity as well as muscle strength. The specific effects of AT on the disease process (levels of mutant mitochondrial DNA in heteroplasmic defects or enzymatic deficiency in nuclearencoded defects) are still unresolved. An alternate rationale for ST in certain heteroplasmic mutations involves the activation of satellite cells in response to muscle overload or injury, and is currently being assessed as a strategy to increase levels of wild-type mitochondrial DNA.

Patients with sporadic inclusion body myositis improved aerobic capacity and muscle strength as a result of combined AT and low-intensity ST of the upper and lower limbs without unfavorable muscle symptoms or further increases in baseline CK. However, signif-

icant increases in muscle size were not detected. To achieve greater gains in muscle size and therefore strength, a novel strategy using moderate-intensity ST with vascular occlusion increased muscle cross-sectional area and strength in a single patient with sporadic IBM.

Limited studies assessing exercise training in muscular dystrophies showed that low-intensity AT improved peak exercise capacity with no signs of muscle damage in facioscapulohumeral dystrophy (FSHD), Becker's and myotonic dystrophy. In both FSHD and myotonic dystrophy, moderate-intensity ST is reported to have no negative effects, however improvements in muscle strength or size are limited or non-existent. The application of neuromuscular electrical stimulation, as a surrogate for ST in patients with severe muscle weakness, has recently been reported to be well-tolerated and resulted in improved muscle strength in FSHD.

In conclusion, despite the initial progress made thus far, there is an urgent need for larger, randomised controlled studies to confirm the safety and develop specific exercise guidelines to optimise efficacy of exercise therapy in various muscle diseases.

Antisense oligonucleotide therapy for muscular dystrophies (Francesco Muntoni, London, UK)

The improved understanding of the genetic basis and molecular events leading to muscle degeneration in muscular dystrophies, coupled with advances in antisense oligomers, has moved very rapidly in the last decade from in vitro experiments and in-vivo studies in appropriate animal models to phase I; IIa; IIb and now III clinical trials in Duchenne muscular dystrophy (DMD), the most common of the severe and childhood onset muscular dystrophies. The pace of the development of these novel genetic approaches to treat muscular dystrophies is exciting and one of the fastest in recent drug development programmes.

The research program that has paved the way to the therapeutic developments for various muscular dystrophies is the one in DMD: the first description of the use of antisense oligonucleotides to modify the splicing of the dystrophin gene in cultured mdx muscle cells was only published in 1998. Less than 10 years later in 2007,

the results of the first intramuscular 'proof of concept' study in DMD boys was published, followed by a second one in 2009. During the course of 2009-2010 two separate repeated systemic dosing studies using 2 different antisense oligomers have been completed. Two large international randomised placebo-controlled studies were recently initiated. The outcome of these studies is expected by early 2012; and additional studies are being planned.

Preclinical studies in myotonic dystrophy are also rapidly advancing, followed by also attempts to utilise antisense oligomers in other, less common muscular dystrophy variants, for example in LGMD2B. In parallel to the pioneering human studies using these 'first generation' chemistries, experimental work focused on optimising the efficacy of new generation antisense is very rapidly progressing, with unparalleled efficacy in preclinical models.

The status of the art of the clinical and preclinical work being performed in this field will be updated.

Gene Therapy: Myth, Milestones, and Momentum (Jerry R. Mendell, (Columbus, Ohio, USA)

Therapeutic options for muscular dystrophy are limited. Presently only corticosteroids have been found to benefit boys with Duchenne muscular dystrophy (DMD), although the results are modest and side effects significant. The translational research community has introduced molecular strategies to potentially enhance mutant gene expression through exon skipping, mutation suppression, and gene replacement. Each of these approaches has merit and is being tested in clinical trials. For gene replacement therapy, adeno-associated virus (AAV) has been established as a safe vehicle for gene transfer with the recognition that pre-existing exposure to this virus can present obstacles. As the most common severe form of inherited muscle disease, DMD has been a particular target for gene therapists. In the first AAV-mediated gene therapy trial for this disease, a surprising finding was that hidden epitopes of expressed dystrophin, usually found on revertant fibers, proved to be immunogenic for some individuals. It had previously been considered that dystrophin expressed on revertant fibers was tolerizing. The lesson learned

is that patients can be screened prior to participation, paving the way for safe gene transfer. Clinical experience has also demonstrated that transfer of mini-dystrophin genes must be carefully matched with endogenous DMD gene deletions to avoid expression of novel immunogenic epitopes.

A major milestone for gene therapy was the first successful gene transfer using AAV to deliver the alpha-sarcoglycan gene in one form of limb girdle dystrophy- LGMD2D. Sustained gene expression for six months was achieved following intramuscular gene transfer. Further advantages accrued through the use of a muscle specific promoter. An exception was a patient exhibiting pre-existing AAV neutralising antibody that it will be necessary to avoid in future trials. Other strategies for gene transfer are poised for clinical trials, including building muscle size and strength through AAV-mediated gene transfer of follistatin. In addition, AAV5 is uniquely capable of transferring genes that exceed the usual packaging limit of ~5kb, an important obstacle to overcome for therapy of disorders due to big gene mutation.

Pharmaceutical Industry Satellite Symposia Schedules at the ENS Meeting

Advances in novel drug development 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 advances 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 11 February 2011).

Sunday, 29 May 2011

Merck Serono – 12:00-13:30

It's a marathon, not a sprint. Clinical decision-making to support patients throughout the MS journey

Chair: M. Sandberg-Wolheim, Lund / SE; M.J. Sá, Porto / PT

UCB – 12:00-13:00

Sleep and CNS diseases

Chair: C. Bassetti, Zurich and Lugano / CH

Monday, 30 May 2011

Novartis – 11:45 13:15

Fingolimod: MS treatment transformation

Chair: J. Hillert, Stockholm / SE; J. Correia de Sá, Lisbon / PT

SSIF – 11:45 13:15

Advances in frontotemporal dementias

Chair: G. Comi, Milan / IT; S. Cappa, Milan / IT

UCB – 11:45 13:15

Restore what Parkinson's disease takes away

Chair: K. Ray-Chaudhuri, London / UK

Biogen Idec – 17:15-18:45

Addressing patient needs today and tomorrow (MS)

Chair: J. de Sá, Lisbon / PT (TBC)

Tuesday, 31 May 2011

Teva – 11:45-13:15

Laquinimod: A step closer to a novel oral treatment option for patients with MS

Chair: L. Sousa, Coimbra / PT

The ENS meeting offers the opportunity to present research results to a large number of colleagues from the entire spectrum of neurology. Specialist areas often have numerous links to various fields of inquiry and the ENS meeting offers the greatest opportunity to encounter colleagues with parallel interests. The following 3 abstracts have received the best grades. These abstracts and all other accepted abstracts for the 21st ENS meeting are published in the supplement of the *Journal of Neurology*.

Disorders of consciousness

PROgnosis of PostAnoxic Coma after treatment with hypothermia: results of PROPACII, a multi-centre prospective cohort study

A. Bouwes, J.M. Binnekade, M.A. Kuiper, F.H. Bosch, D.F. Zandstra, A.C. Toornvliet, H.S. Moeniralam, B.M. Kors, J.H. Koelman, M.M. Verbeek, H.C. Weinstein, A. Hijdra, J. Horn. Academic Medical Center (Amsterdam, NL); Medical Center Leeuwarden (Leeuwarden, NL); Rijnstate Hospital (Arnhem, NL); Onze Lieve Vrouwe Gasthuis (Amsterdam, NL); Medical Center Alkmaar (Alkmaar, NL); St. Antonius Hospital (Nieuwegein, NL); Kennemer Gasthuis (Haarlem, NL); Radboud University Nijmegen Medical Center (Nijmegen, NL); Sint Lucas Andreas Hospital (Amsterdam, NL).

Objectives: Current guidelines for outcome prediction in patients with postanoxic coma after cardiopulmonary resuscitation (CPR) are based on data from patients not treated with hypothermia. New information is required. The Aim of this study was to establish the reliability of neurological examination, neuron-specific enolase (NSE) and median nerve somatosensory evoked potentials (SEP) to predict poor outcome in patients treated with mild hypothermia after CPR.

Methods: Multicentre prospective cohort study, including adult comatose patients, admitted to the ICU after CPR and treated with hypothermia

(32–34°C). Neurological examination (Glasgow Coma Score and brain stem reflexes) was performed 72 hours after CPR. Samples for NSE levels were drawn on admission, 12 hours after reaching target temperature, 36 and 48 hours after CPR. Median nerve SEP was recorded during hypothermia and after rewarming. Neurological outcome was assessed with the Glasgow Outcome Scale (GOS), after 1 week, 1 month and six months. Primary outcome was poor outcome, defined as death, vegetative state or severe disability after six months.

Results: 391 patients were included, 53% had a poor outcome. Absent pupillary light responses

(FPR 1, 95% CI 0-7), corneal reflexes (FPR 1, 95% CI 0-7) and N20 responses in SEP after rewarming (FPR 0, 95% CI 0-18) were reliable predictors. Motor scores 72 hours after CPR and NSE levels were not.

Conclusion: Poor outcome after CPR and therapeutic hypothermia can reliably be predicted by testing brain stem reflexes and SEP. Other methods recommended in current guidelines could possibly lead to inappropriate withdrawal of treatment.

This study was supported by a research grant from The Netherlands Heart Foundation, 2007B039.

Clinical neurophysiology

Assessing cortical effective connectivity in patients with disorders of consciousness

M. Rosanova, O. Gosseries, S. Casarotto, M. Boly, A.G. Casali, MA. Bruno, P. Boveroux, G. Tononi, S. Laureys, M. Massimini. University of Milan (Milan, IT); University of Liege (Liège, BE); University of Wisconsin (Madison, US).

Objectives: Brain-injured patients are considered conscious if they can interact with the environment and unconscious otherwise. As suggested by other works, a key requirement for consciousness is that multiple, specialised cortical areas can interact rapidly and effectively. Here we employ transcranial magnetic stimulation combined with electroencephalography (TMS/EEG) in order to assess cortical effective connectivity at the bedside of brain-injured patients with disorders of consciousness.

Methods: We used a TMS-compatible 60-channels EEG amplifier to record TMS-evoked potentials in 17 brain-injured patients. A first group of 12 patients (Group I) underwent a single TMS/EEG session after one week of behavioral evaluations (Coma Recovery Scale-Revised). Five of these patients were diagnosed as vegetative state (VS),

five were minimally conscious (MCS) and two were in a locked-in syndrome (LIS). A second group of five patients (Group II) were recruited as soon as they awakened from coma and underwent longitudinal TMS/EEG measurements. Three of them recovered consciousness evolving from VS through MCS to emergence from MCS. We stimulated bilaterally the parietal and the frontal lobes in each patient.

Results: In Group I, VS patients, who were open-eyed, behaviorally awake but unresponsive, TMS triggered a stereotypic and local response indicating a breakdown of effective connectivity, similar to the one observed in sleep or anaesthesia. On the contrary, in MCS patients, who showed fluctuating signs of non-reflexive behavior, TMS triggered rapidly changing, widespread responses similar to the ones recorded in LIS and healthy

awake subjects. In Group II, a simple and local response to TMS was also recorded in all patients as soon as they transitioned from coma to VS. In the three patients who recovered consciousness and functional communication, intracortical effective connectivity resurged soon after they switched from VS to MCS as well as they emerged from MCS.

Conclusion: TMS/EEG measurements performed in Group I suggest that clear-cut differences in intracortical effective connectivity underlie the subtle clinical discrimination between VS and MCS patients. TMS/EEG measurements performed in Group II showed that cortical effective connectivity re-emerged in VS patients who recovered consciousness as soon as they recover to MCS. Thus, directly perturbing the brain to assess effective connectivity may represent a sensitive way to uncover a brain's capacity for consciousness.

Child neurology – Poster session

Using functional MRI to assess the effects of a morpheme-based training in dyslexia

D. Gebauer, A. Fink, R. Kargl, G. Reishofer, K. Koschutnig, F. Fazekas, C. Enzinger. Medical University Graz (Graz, AT); Karl-Franzens University Graz (Graz, AT); Dyslexia Institute Graz (Graz, AT).

Objectives: Dyslexia is a neurodevelopmental disorder, associated with a greater risk of school drop-out, unemployment, and emotional and behavioural problems. Approximately 15% of affected children show reading and spelling difficulties. Hence, it is of major importance to

improve the understanding of the underlying brain processes of dyslexia, in order to optimize future interventions. We here set out to assess the effects of a morpheme-based training in dyslexic children on brain function.

Methods: 42 children aged between 9 and 15 years

participated in the study. To assess a specific effect of the training, dyslexic children were divided into a 'Training-Group' (n=14) and a 'Waiting-Group' (n=14). The dyslexic groups were compared to 14 age- and intelligence-matched controls. In pretests, reading and spelling skills, intelligence and person-

ality were assessed. Structural and functional MRI scans were obtained at a 3 Tesla scanner. During fMRI, children had to identify misspelled words, correctly spelled words and pseudowords. The 'Training-Group' received a five-week intensive intervention. After this period, all three groups were again tested behaviorally and re-scanned.

Results: Compared to controls, while processing the presented words, dyslexic children showed significantly increased activation in the right hemisphere and decreased activation of the left fusiform and temporal regions prior to the training. After the intervention, an adjustment of activation pattern to controls was observed in the

'Training-Group', parallel to behavioural improvements.

Conclusion: These results support the concept of training-induced neural plasticity. Initially presumably compensatorily increased right hemispheric activation and decreased left hemispheric activation normalized subsequent to training.

Stroke

Evolution or revolution? 1-year results for mechanical thrombectomy with the Solitaire stent in acute stroke

A. Mpotsaris, M. Bussmeyer, J. Fuehrer, H. Buchner, W. Weber. *Klinikum Vest (Recklinghausen, DE).*

Background: To report the effectiveness of mechanical thrombectomy with the Solitaire stent in severe acute ischaemic stroke in conjunction with intravenous systemic thrombolysis.

Methods: Prospective, ongoing single centre study of patients with acute ischaemic stroke based on proven large artery occlusion via CT-angiography in anterior or posterior circulation. Following strict inclusion criteria patients were triaged for eligibility for mechanical thrombectomy, independently of intravenous thrombolysis with tissue plasminogen activator (rTPA). Clot retrieval was performed with the Solitaire stent (AB and FR, ev3 Inc, Plymouth, MN) with up to 4 maneuvers. NIHSS and mRS scores were assessed on admission, discharge, after 90 days and after

one year. For evaluation of outcome, patients were stratified to early, intermediate and late treatment subgroups.

Results: Up until January 2011 54 patients were eligible for mechanical thrombectomy with the Solitaire stent since October 2009. 92 % had a NIHSS score of ≥ 10 and 96% mRS 4 or 5 on admission. 40 of 54 patients received intravenous rTPA prior to mechanical thrombectomy (bridging technique), 14 were treated with thrombectomy alone. 27 of 54 had tandem stenosis and were a priori stented. Recanalisation rate was 88%; in 50% of cases the first attempt led to recanalization. There were no procedural complications. Overall 37% (20 of 54) patients had a good clinical outcome (mRS ≤ 2) in the 90 days

follow up interval. In the early treatment subgroup (n=21) with recanalization in ≤ 4.5 h from symptom onset good outcome was reached in 50%. Of 13 patients with carotid-T-occlusions 6 had a good outcome after 90 days. Patients who were treated in bridging-technique with intravenous rTPA had a higher NIHSS score reduction (p=0.06) than non-bridging patients. By May 2011 the 1-Year results of 25 patients will be available for analysis.

Conclusions: The combination of rTPA and mechanical thrombectomy is safe. The Solitaire stent can be deployed safely and quickly. The 90 day results are encouraging, especially in combination with i.v. rTPA; the Solitaire may play a key role in further improvement of outcome in severe acute stroke, especially in carotid-T-occlusions.

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 important prominent society for neurologists in Europe 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 neurologists in training programme" in 2006, which offers a limited number of grants providing free accommodation (4 nights), free registration and free admission to 3 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 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 2011 annual meeting will take place in Lisbon, Portugal, 28 – 31 May 2011. This meeting will again be the primary stage for the latest developments in scientific research, where neurologists share and discuss innovative studies.

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 specialties. 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.