International School of Neurology

7th International Summer School of Neurology

1 - 5 July 2012 | Hotel Europa | Eforie Nord | Romania

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International
School of Neurology

The Society for the Study of
Neuroprotection and Neuroplasticity
www.ssnn.ro

"Iuliu Haţieganu" University of Medicine
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www.umfcluj.ro

Tel Aviv University
www.tau.ac.il

World Federation for NeuroRehabilitation
www.wfnr.co.uk

Romanian Academy of Medical Sciences
www.adsm.ro

Romanian Society of Neurology
www.neurology.ro

UPPSALA
UNIVERSITET

Uppsala University
www.uu.se

Association of Parkinsons
Related Disorders

EFNRS
European Federation
Neurorehabilitation Societies

European Federation of Neurilogical Societies
www.efns.org

Faculty
/in alphabetical order

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Heinrich Binder/Austria
Dana Boering/Germany
Natan Bornstein/Israel
Volker Hömberg/Germany
Arnon Karni/Israel
Dafin F. Mureşanu/Romania
Gelu Onose/Romania
Hilleke Hulshoff Pol/The Netherlands
Laurenţiu M. Popescu/Romania
Bogdan O. Popescu/Romania
Leopold Saltuari/Austria
Hari Shanker Sharma/Sweden
Mihaela Simu/Romania
Stephen D. Skaper/Italy
Daniel D. Truong/USA
Johannes Vester/Germany
Pieter E. Vos/The Netherlands
Erik Ch. Wolters/The Netherlands

General Information

ANA Hotels – Eforie Nord - Europa and Astoria Hotels

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Scientific Secretariat

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Registration Desk

All materials and documentation will be available at the registration desk located at SSNN booth.
The staff will be pleased to help you with all enquiries regarding registration, materials and program. Please do not hesitate to contact the staff members if there is something they can do to make your stay more enjoyable.

Language

The official language is English. Simultaneous translation will not be provided.

Changes in program

The organizers cannot assume liability for any changes in the program due to external or unforeseen circumstances.

Name Badges

Participants are kindly requested to wear their name badge at all times. The badge enables admission to the scientific sessions and gala dinners.

Final Program & Abstract Book

The participants documents include the program and abstract book which will be handed out together with the congress bag at the registration counter.

Coffee Breaks

Coffee, tea and mineral water are served morning and afternoon coffee breaks free of charge to all registered participants.

Mobile Phones

Participants are kindly requested to keep their mobile phones turned off while attending the scientific sessions in the meeting rooms.

Currency

The official Romanian currency is RON.

Electricity

Electrical power is 220 volts, 50Hz. Two-prong plugs are standard.

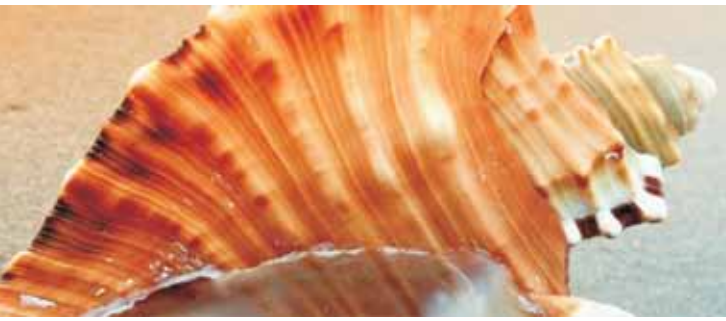
Time

The time in Romania is
Eastern European Time (GMT+2).

CONTACT:

If you need further information on technical details, please contact:
Ovidiu Selejan/e-mail/ovidius@ssnn.ro
For updates and details please visit our website www.ssnn.ro

SCIENTIFIC PROGRAM

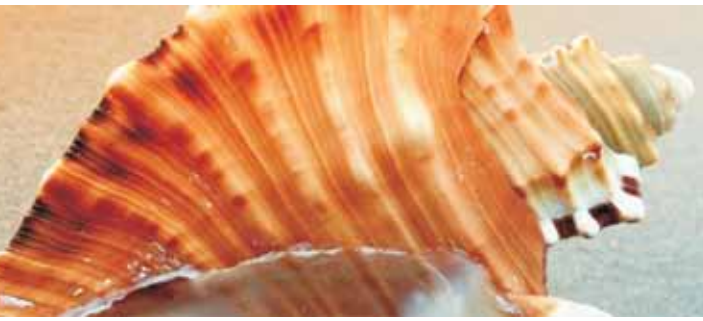


Sunday, 1st of July – DAY 1		
18:00-20:00	CNS Injury and Repair Module coordinators: L.M. Popescu (Romania); Hari Shanker Sharma (Sweden)	
18:00 – 18:30	Hari Shanker Sharma (Sweden)	Potential Role of Nanodrug Delivery of Neurotrophic Factors in Central Nervous System Injury and Repair
18:30 - 19:00	Stephen D. Skaper (Italy)	Physiopathology and Pharmacology of the Endoneural Microenvironment
19:00 - 19:30	Pieter E. Vos (The Netherlands)	Functional Connectivity Changes After Mild Traumatic Brain Injury
19:30 – 20:00	Hilleke Hulshoff Pol (The Netherlands)	Plasticity of Human Brain Structure Throughout Life
20:30	Dinner	

Monday, 2nd of July – DAY 2

08:45 – 09:00	Welcome Address:	Dafin F. Mureşanu (Romania), Natan Bornstein (Israel), L. M. Popescu (Romania), Volker Hömberg (Germany)
09:00-14:00	Neurorehabilitation Approved by WFNR and EFNRS Module coordinators: Volker Hömberg (Germany); Heinrich Binder (Austria)	
09:00 – 09:30	Volker Hömberg (Germany)	Clinical Examination
09:30 – 10:00	Volker Hömberg (Germany)	Motor Rehabilitation: Physical Therapy
10:00 - 10:30	Volker Hömberg (Germany)	Motor Rehabilitation: Training Techniques
10:30 – 11:00	Coffee Break	
11:00 – 11:30	Dana Boering (Germany)	Motor Assessments
11:30 – 12:00	Dana Boering (Germany)	Aiding Devices
12:00 – 12:30	Heinrich Binder (Austria)	Basics of Motor Control
12:30 – 13:00	Coffee Break	
13:00 – 13:30	Leopold Saltuari (Austria)	Robotics
13:30 – 14:00	Gelu Onose (Romania)	Main Orthotic Devices and Their Use in Neurorehabilitation
14:00	Lunch	
18:00-20:00	Clinical Trial Statistics for Non-Statisticians Module coordinator: Johannes Vester (Germany)	
18:00 – 20:00	Johannes C. Vester (Germany)	Hypothesis Testing and Statistical Significance: The Basic Concept of a Statistical Test
20:30	Welcome Dinner	

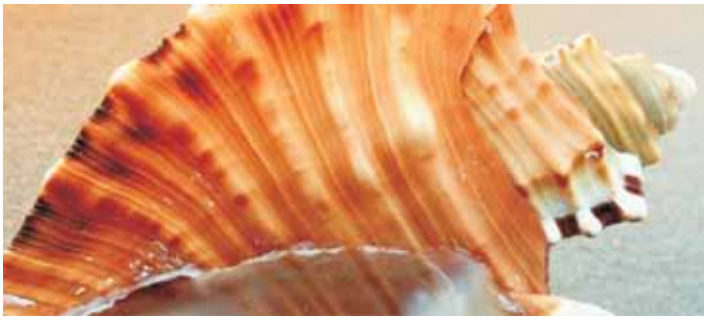
	Tuesday, 3rd of July – DAY 3		
09:00-11:00	**Clinical Trial Statistics for Non-Statisticians** Module coordinator: Johannes Vester (Germany)		
	09:00 – 10:00	Johannes C. Vester (Germany)	P-Values, Effect Sizes and Confidence Intervals: Definition and Handling in Superiority and Non-Inferiority Trials
	10:00 – 11:00	Johannes C. Vester (Germany)	Proper Interpretation of Study Results: Examples from Recent TBI Trials
	11:00 – 11:30	Coffee Break	
11:30-13:30	**Neuroimmunology** Module coordinator: Arnon Karni (Israel)		
	11:30 – 12:00	Arnon Karni (Israel)	New Pharmacologic Approach to Multiple Sclerosis (I)
	12:00 – 12:30	Arnon Karni (Israel)	New Pharmacologic Approach to Multiple Sclerosis (II)
	12:30 – 13:00	Arnon Karni (Israel)	The Immune Responses and Dysregulation in Autoimmune Diseases
	13:00 – 13:30	Arnon Karni (Israel)	Case Presentation
	14:00	Lunch	
18:00-20:00	**New Perspectives in Multiple Sclerosis** Chairmen: Ovidiu Băjenaru (Romania), Dafin F. Mureşanu (Romania)		
	18:00 – 18:40	Ovidiu Băjenaru (Romania)	A New Approach in MS therapy, the Nrf2 Pathway
	18:40 – 19:20	Mihaela Simu (Romania)	Treating Walking Impairments in MS
	19:20 – 20:00	Dafin F. Mureşanu (Romania)	Considerations on Treatment Window of Opportunity in MS
	20:30	Dinner	



	Wednesday, 4th of July – DAY 4	
09:00-12:30	**Stroke** Module coordinators: Natan Bornstein (Israel); Dafin F. Mureşanu (Romania)	
09:00 – 09:45	Natan Bornstein (Israel)	Secondary Stroke Prevention
09:45 – 10:30	Natan Bornstein (Israel)	The Heart's Effect on the Brain - Atrial Fibrillation and Stroke Prevention-Update
10:30 – 11:00	Coffee Break	
11:00 - 11:45	Natan Bornstein (Israel)	Time is Brain, TIA as an Emergency
11:45 – 12:30	Dafin F. Mureşanu (Romania)	An Integrated Approach in Brain Protection and Recovery in Stroke Therapy
13:00	Lunch	
18:00 – 20:00	Case presentations	
20:00	Dinner	

	Thursday, 5th of July – DAY 5	
09:00-12:30	**Neurodegenerative disorders** **Supported and Approved by the WFNA-PRD** Module coordinators: **Ovidiu Băjenaru** (Romania); **Erik Ch. Wolters** (The Netherlands)	
09:00 – 09:30	**Ovidiu Băjenaru** (Romania)	Clinical and Biological Spectrum of Fronto-Temporal Dementia
09:30 – 10:00	**Daniel D. Truong** (USA)	Multiple System Atrophy
10:00 – 10:30	**Erik Ch. Wolters** (The Netherlands)	Parkinson's Disease Revisited
10:30 – 11:00	Coffee Break	
11:00 - 11:30	**Bogdan O. Popescu** (Romania)	Comorbidities in Parkinson's Disease
11:30 – 12:00	**Angelo Antonini** (Italy)	Clinical Challenges in Parkinson Therapy Including Apomorphine
12:00 – 12:30	**Ovidiu Băjenaru** (Romania)	Interventional Treatment of Continuous Dopaminergic Stimulation in Advanced Parkinson's Disease
13:00	Lunch	
15:00 – 16:00	Examination	
20:00	Farewell Dinner	

ABSTRACTS



CLINICAL CHALLENGES IN PARKINSON THERAPY INCLUDING APOMORPHINE

ANGELO ANTONINI

Director Parkinson Unit IRCCS San Camillo, Venice – Neurology Clinic, University of Padua, Italy

Apomorphine is the oldest dopaminergic medication and was initially known for its emetic properties. It was initially used for Parkinson's disease over 60 years ago but later ignored for many years following levodopa introduction. It is also the most potent dopamine agonist and its administration can provide symptom relief comparable to levodopa. Apomorphine exerts its antiparkinsonian effect by direct stimulation of striatal postsynaptic dopamine D1 and D2 receptors. The drug has a rapid absorption after subcutaneous injection (Cmax 20 min), and a short half-life (almost 43 min), and this is consistent with its rapid onset of action, with effects apparent within 5–15 minutes of subcutaneous administration. Clinical studies and evidence-based reviews generally support a role for apomorphine infusion as an effective option for patients with PD and severe fluctuations, poorly controlled by conventional oral drug treatment with an improvement in OFF-time between 50% and 80% as well as dyskinesia. While the benefit on off time is consistent across all studies, dyskinesia improvement generally occurs after a few weeks or months of continuous dopaminergic stimulation as a result of wider therapeutic window. Moreover it can be best achieved with apomorphine monotherapy that may require high infusion doses.

Intermittent subcutaneous apomorphine (penjet) may instead be suitable for the long-term acute treatment of OFF episodes in patients with advanced PD. Apomorphine injections can be a particularly helpful in the management of patients who undergo surgical procedures and cannot take medication by mouth or to treat additional severe non-motor symptoms occurring during OFF periods.

References:

Antonini A, Tolosa E Apomorphine and levodopa infusion therapies for advanced Parkinson's disease: selection criteria and patient management. Expert Rev Neurother. 2009 Jun;9(6):859-67

Antonini A, Isaias IU, Rodolfi G A 5-year prospective assessment of advanced Parkinson disease patients treated with subcutaneous apomorphine infusion or deep brain stimulation J Neurol. 2011 Apr;258(4):579-85

INTERVENTIONAL TREATMENT OF CONTINUOUS DOPAMINERGIC STIMULATION IN ADVANCED PARKINSON'S DISEASE

OVIDIU BĂJENARU

University Hospital of Emergency Bucharest, Department of Neurology,

„Carol Davila" University of Medicine and Pharmacy, Bucharest,

Romania

Parkinson's disease (PD) is a progressive multifocal neurodegenerative disease characterised by multiple synaptic dysfunctions which clinical impact impairs motor behaviour, sensory, neurocognitive, autonomic functions, sleep, mood and other components of human behaviour. Among these, motor dysfunction is one of the most important factors which impairs patients' daily life activity and quality and the best studied due to its relation to disturbed dopaminergic modulation in the cortico-striatal circuits (even if in these patients also dysfunctions of other structures of the brain - not always related to dopamine modulation - impair the motor behaviour). In advanced stages of the disease these clinical impairments are enhanced in symptomaticaly treated patients by the dopaminergic agents, in particular oral levodopa leading to motor and non-motor complications. These treatment complications are mostly related to the non-physiologic pulsatile stimulation by oral drug administration which are oposed to the tonic, continuous, physiologic dopaminergic synaptic modulation of the indirect striopallidal pathway and probably also of the non-synaptic modulation of neuronal intracellular signaling pathways related to neuroplasticity. In advanced Parkinson's disease in patients whom symptomatic control of their clinical manifestations cannot be obtained any more with the best oral drugs combinations, particular therapeutic approaches are available today. Among these, continuous levodopa administration by an electronic pump directly in the jejunum using a transcutaneous laparoscopic gastrostoma using a special tubular device or deep brain stimulation with a high frequency from an electronic stimulator using a special electrode system stereotaxically inserted bilateraly in the subthalamic nucleus (or in the internal part of the globus pallidus) are common nowadays. We shall present also our personal clinical experience using these methods in the Department of Neurology of the University Emergency Hospital Bucharest.

CLINICAL AND BIOLOGICAL SPECTRUM OF FRONTO-TEMPORAL DEMENTIA

The data accumulated during the last 10 – 15 years related to fronto-temporal dementia show that this terminology refer to a large group of clinical entities represented by neurodegenerative diseases, which initial clinical manifestations are behavioural disorders with a suggestive pattern (related to the impairment of social cognition - as a function of the frontal lobe) or primary language dysfunction (not explained by another pathology). More recently it has been documented that the pathology of these neurodegenerative diseases is characterized by some well defined types of hystopathologic modifications due to alterations of some cellular functional proteins some of which are genetically identified and correlated to the phenotype expression. During the last few years, it has been noticed that some of these hystopathologic changes are present also in other neurologic clinical entities considered till recently as different diseases not related among them (some forms of amyotrophic lateral sclerosis, progressive supranuclear palsy, cortico-basal dementia), but which clinical features are often recognized also in patients with fronto-temporal dementia. The actual clinico-biologic classification of fronto-temporal dementia is based on the complex correlation between the neurologyc phenotype (often with different patterns of combination among the clinical features mentioned before) and the defining hystopathologic features which place the frontotemporal dementia in the group of proteinopathies.

BASICS OF MOTOR CONTROL

HEINRICH BINDER

Landsteiner Institute for Neurorehabilitation and space medicine

Vienna, Austria

The term motor control signifies for a function of the central nervous system to accomplish posture and movement. Both are inseparable and interact. On one side movements can regarded as transition from one posture to another. On the other hand movement is unconceivable without basic posture. Humankind is born with humble basic configuration. Only during infancy he learns all these motor skills necessary for sitting, walking, grasping and so on - whatever necessary for daily life and human behaviour. This learning process comprises of how it feels on the one hand and the experience of the result. The crucial point is that without feedback motor control and learning is unthinkable. And this happens continuously with the aid of mainly kinaesthetic and somatic input at various levels in different networks. Since the body is represented in each of these networks the brain knows the body he resides in at any times in any situation. This knowledge is the basis for whatever motor activity. It is continuously adapted accordingly achieving objectives. Furthermore it is improved by continuous practice and stored in the form of motor programs and motor plans with consequent skilled movements.

AIDING DEVICES

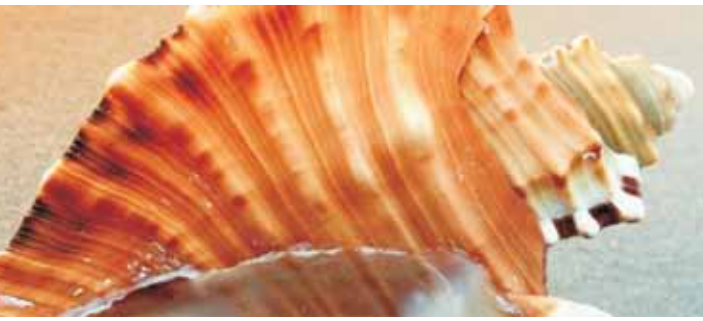
DANA BOERING

St. Mauritius Therapieklinik Meerbusch, Germany

Assistive devices for people with impaired motor control translate or amplify remaining functions to allow actions which are otherwise impossible. People with disabilities are entitled to access assistive technology to facilitate their effective participation in society and may reasonably expect to be central to the decision making processes of the services providing these technologies.

There is an improvement in knowledge and resources available for aiding device delivery inthe majority of European countries but there are still barriers to their effective provision.

The talk will summarize the mainfields of applicability of aiding devices: mobility, communication, environment adaption/ daily comfort. It will present some aspects of actual mobility technology research i.e. the closer integration with the user, decreasing user burden while increasing user capability, the further development of wearable sensors and pervasive systems improving home rehabilitation, further progresses in rehabilitation technology enriching clinical practice. It will also point out some factors limiting assistive technology advancements: the need for therapy technology that can be used at home is largely unmet, improvements in actuators and power supplies that have not progressed as quickly as those in sensors, the relative immaturity of control algorithms. Multidisciplinary teams that work closely with users and are embedded with scientists with a profound knowledge of motor learning and postlesional plasticity are best positioned toproduce transformative mobility technology.



MOTOR ASSESSMENTS

Currently we are provided with multifaceted therapy techniques with high evidence of efficiency for the treatment of upper/ lower limb central paresis. Hence we have principally the possibility to work in an evidence based way in a certain clinical context.

Scientifical guidance is for the further development of therapeutic techniques fundamental.

Because of shortened therapy duration in neurological rehabilitation it is usually not possible to discern empirically which evidence based therapy technique helps most to an individual patient with his individual background.

Therefore it is of eminent importance to develop a systematic research to point out which therapeutic technique helps which patient in a certain stage of his postacute development.

This could lead to the further definition of a clear sequence of therapeutic techniques for a particular postacute development stage of the patient.

Prerequisite for this development is the systematic comparison of rehabilitation outcomeswhen using certain therapy techniquesin a certain well defined patient group.
Only a thorough sequential assessment of each of these patients can constitute the groundwork for this undertaking.

This well-defined assessment approach allows proofing the efficiency of the used therapy techniques by comparing the rehabilitation outcomes of patients with similar assessment parameters at their rehab admission.

The talk will focus on a detailed presentation of the most used motor assessments for the principal mobility related activities: changing and maintaining posture, carrying and moving objects, walking and moving around.

A rational outcome definition always implies a broad evaluation of all information related to a definite patient including the medical and personal biographical background and special individual aspects.

This means that beyond the necessary orientation of rehabilitation practice on applied neuroscience, beyond all evidence and guideline based rehabilitational approaches, goals of rehabilitation outcomes must always be individually shaped.
(EBM vs Individualised Medicine)

SECONDARY STROKE PREVENTION

NATAN BORNSTEIN

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Sackler Faculty of
Medicine, Israel

Vice President of the
World Stroke
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Head of
Stroke Unit at the
Tel-Aviv
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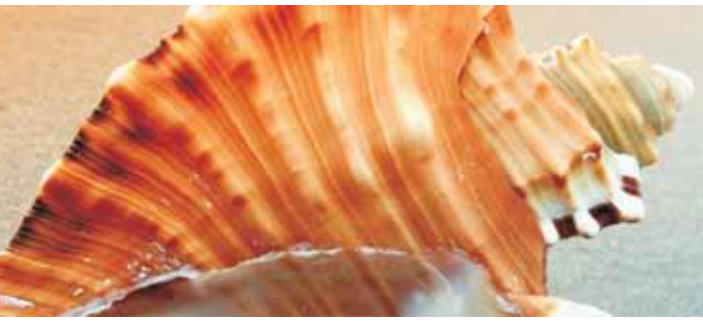
Chairman of the
Israeli Neurological
Association

Patients with TIA or ischemic stroke carry a risk of recurrent stroke between 5 and 20% per year. In patients with TIA or ischemic stroke of noncardiac origin antiplatelet drugs are able to decrease the risk of stroke by 11-15% and the risk of stroke, MI and vascular death by 15-22%. Aspirin is the most widely used drug. It is affordable and effective. Low doses of 50-325 mg aspirin are as effective as high doses and cause less gastrointestinal side effects. Severe bleeding complications are dose-dependent. The combination of aspirin with slow release dipyridamole is superior to aspirin alone for stroke prevention (ESPS-2 and ESPRIT1). Both studies have shown approximately 20%-24% relative risk reduction (RRR) of stroke and death. Clopidgrel is superior to aspirin in patients at high risk of recurrence by about 8.7% RRR (CAPRIE2). The combination of aspirin plus clopidogrel is not more effective than clopidogrel alone but carries a higher bleeding risk (MATCH3 and CHARISMA4). None of the antiplatelet agents is able to significantly reduce mortality. The recent results of the PRoFESS trial 5,6 showed no difference between clopidogrel and aspirin with slow release dipyridamole in secondary stroke prevention.

References

1. Lancet 2006;367:1665-73
2. Lancet 1996;348:1392-1339
3. Lancet 2004;364:331-337
4. N Eng J Med 2006;354(16):1744-6
5. Cerebrovasc Dis 2007;23:368-380
6. N Engl J Med 2008;359:1238-51



TIME IS BRAIN, TIA AS AN EMERGENCY

Transient Ischemic Attack (TIA) should be considered as an emergency and work-up has to be done within 24 hours like acute unstable angina pectoris. It is known that about 23% of stroke are preceded by TIA.Several studies have shown that the risk of subsequent stroke in the first 2 weeks after a TIA is about 1% per day. In 2 published well conducted studies, EXPRESS (P. Rothwell) and SOS_TIA (P. Amarenco) it was shown that very early management in a TIA clinic will reduce the risk of subsequent stroke by 80% at 3 months. Therefore, work-up evaluation has to be performed with in 24 hours in a dedicated organized structure.

Several stroke registries reported that carotid stenosis is the cause of embolic stroke in about 25%-30% of all ischemic strokes. Current guidelines recommend immediate intervention either by carotid endarterectomy (CEA) or stenting (CAS) in patients with symptomatic carotid stenosis greater than 50%.

Carotid duplex is a reliable, non-invasive, accessible tool for evaluation of carotid stenosis with very high level of accuracy. Therefore, carotid duplex should be the first line tool for rapid evaluation of every patient with TIA in order to detect a potential treatable carotid stenosis for stroke prevention. It is recommended to establish an “Acute TIA clinic” equipped with immediate accessible Duplex device to enable rapid evaluation of the carotid system in order to detect potential treatable carotid stenosis.

THE HEART'S EFFECT ON THE BRAIN
ATRIAL FIBRILLATION AND STROKE PREVENTAION-UPDATE

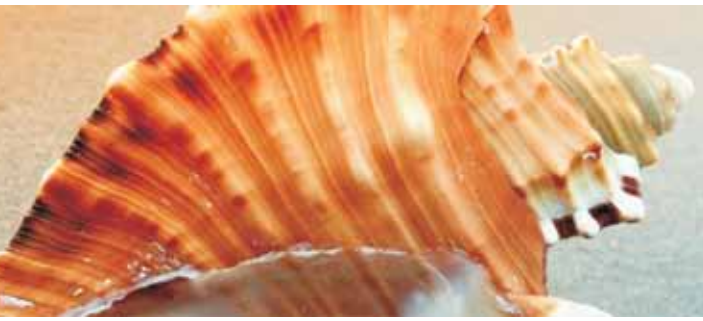
Approximately 20%-25% of all ischemic strokes are cardioembolic stroke.

Atrial fibrillation (AF) is the most frequently found arrhythmia with a prevalence of 0.4 – 0.7% in the general population. The prevalence of AF rises to approximately 6% in population older than 65 years, and up to 10% in people older than 75 years.
AF related stroke comprises approximately 45% of all cardioembolic strokes.

AF is a well-established independent risk factor for stroke, leading to 5.6-fold increase of risk. Risk for recurrent stroke in AF patients without antithrombotic treatment is 12% per year.An ischemic stroke will occur during lifetime of about 35% non-anticoagulated AF patients.

According to Class I evidence, adjusted-dose warfarin reduces risk of stroke in AF patients by about 70% and aspirin by only 20%. Treatment with warfarin is recommended with target INR of 2.5 (range 2.0-3.0). Newly developed devices to occlude the left atrial appendage are currently being developed and tested in clinical trials.

Recently new oral direct thrombin inhibitor, Dabigatran., was proven to be similarly effective in reducing embolic events and strokes in NVAF patients with lower rate of major hemorrhage [NEJM 2009; 361:1139-51]. Several other anti-factor Xa drugs are in phase III clinical trials and the results are awaited in the near future



MOTOR REHABILITATION:TRAINING TECHNIQUES

VOLKER HÖMBERG

Dept of Neurology,
Heinrich Heine University
Düsseldorf
Germany

This lecture will summarize the most important motor training techniques and strategies in neurorehabilitation and critically discuss their application according to individual patients`problems

Evidence based techniques (e. forced use training,treadmill training etc) which follow elementary learning rules will be contrasted to conventional physiotherapeutic schools such as Bobath,Vojta ,PNF etc.

Also the differential therapeutic usefulness of mechanical therapy devices ("robots") will be demonstrated.

CLINICAL EXAMINATION

In this course the art of a rational neurological examination will be taught:

More than in any other clinical discipline the history and examination in neurology are the most informative source of information for the clinician.This is of course due to the fact that structure and function of central and peripheral nervous system are clear and informative.

Clinical skills for optimal examination of cranial nerves,motor and sensory functions and screeningapproaches for cognitive and linguistic analysis will be presented .So the students will soon learn that neurologic examination is much more than just looking at "reflexes"

MOTOR REHABILITATION:PHYSICAL THERAPY

This lecture will summarize the most important physical therapeutic techniqueused inneurorehabilitation for improvement of motor function and discuss their differential clinical usefulness for special patients`problems.

This list will include the most useful electrical and magnetic stimulation methods,aspects of hydrotherapy and application of heat and cold.

These techniques will also be classified according to their impact on neuromodulation.

NEW PHARMACOLOGIC APPROACH TO MULTIPLE SCLEROSIS

ARNON KARNI

Faculty: Medicine, Tel Aviv University Hospital/Department: Tel Aviv Sourasky Medical center, Department of Neurology,

Israel

Recent years have broadened the spectrum of therapeutic strategies and specific agents for treatment of multiple sclerosis (MS). While immune-modulating drugs such as interferon-b and glatiramer acetate remain the first-line agents for MS predominantly due to their benign safety profile, the understanding of the immunopathological processes of MS had led to the development of new agents with specific targets. One concept of these novel drugs is to hamper migration of immune cells towards the affected central nervous system (CNS). The first oral drug approved for MS therapy, fingolimod inhibits egress of lymphocytes from lymph nodes; the monoclonal antibody natalizumab prevents inflammatory CNS infiltration by blocking required adhesion molecule (VLA4). The second concept is to deplete T cells and/or B cells from the peripheral circulation using highly specific monoclonal antibodies such as alemtuzumab (anti-CD52) or rituximab/ocrelizumab (anti-CD20). Moreover, there are new emerging symptomatic therapies as modafinil reduces fatigue, dalfampridine improves walking difficulty, dextromethorphan hydrobromide /quinidine sulfate (Neudexta) reduces pseudobulbar symptoms and a cannabis extract-based product (Sativex) decreases pain and spasticity. All of these novels therapies are a substantial addition to therapeutic armamentarium for MS. We will discuss their advantages and shortcomings in treatment of MS.

THE IMMUNE RESPONSES AND DYSREGULATION IN AUTOIMMUNE DISEASES

The immune system aims to protect the host in danger condition. The immune responses are regulated in order to achieve the appropriate response in different conditions of antigen management (non-self vs. self, etc.) Autoimmune diseases results from the defective regulation of the immune system leading to tissue damage. We will discuss these topics and describe the type of immune responses (innate and adaptive responses) the modes of regulation of the immune responses, the putative mechanism of autoimmune attack via activation of Th1, Th17 and B cells and will critically evaluate the immunological basis of multiple sclerosis.

AN INTEGRATED APPROACH IN BRAIN PROTECTION AND RECOVERY IN STROKE THERAPY

DAFIN F. MUREŞANU

Department of Clinical Neurosciences, University of Medicine and Pharmacy "Iuliu Haţieganu", Cluj-Napoca, Romania

President of the Society for the Study of Neuroprotection and Neuroplasticity (SSNN)

This presentation briefly reviews some of the mechanisms involved in the pathogenesis of neurological diseases, i.e. damage mechanisms, and their interactions and overlap with protection and reparatory processes (i.e., endogenous defense activities). A relationship between damage mechanism (DM) and endogenous defense activity (EDA) regarding therapy principles will also be described.

Currently, it is difficult to find the correct therapeutic approach for brain protection and recovery, especially because we do not fully understand all of the endogenous neurobiological processes, the complete nature of the pathophysiological mechanisms and the links between these two categories. Moreover, we continue to use a simplistic and reductionist approach in this respect.

Endogenous neurobiological processes, such as neurotrophicity, neuroprotection, neuroplasticity and neurogenesis, are central to protection and recovery and represent the background of EDA.

The biological reality of the nervous system is far more complex. In fact, there is an endogenous holistic process of neuroprotection and neurorecovery that should be approached therapeutically in an integrated way.

The current tendency to exclusively frame drug activity in terms of single mechanisms and single focus effect might distract from other paradigms with greater explanatory power and hinder the development of more effective treatment strategies. A change of concept is required in pharmacological brain protection and recovery. This presentation will also highlight the current and future considerations in stroke therapy, including an integrated pharmacological approach, focusing on drugs with multimodal activity and pleiotropic neuroprotective effect which are biological drugs, rather than single mechanism drugs, which usually are chemical drugs.

MAIN ORTHOTIC DEVICES AND THEIR USE IN NEUROREHABILITATION

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Orthotic device(s) or orthosis(es) - also found/ designated in literature by (common) terms of more specific/ restricted coverage, such as braces (that allow/ favor) or splints, or respectively - naming with a decreasing frequency used - corsets (which both - basically but not absolutely - do not allow) related articular movements, represent "any externally applied device used to modify structural and/or functional characteristics of the neuromuscular, skeletal system" (ISO, AAOP, AOPA - cited by Redford JB, Basmajian JV, 1995).
Accordingly, they have valuable and quite large (often syncretic, i.e. for more than one single purpose) applicability, including in NeuroRehabilitation. Because of their related wide usefulness, orthotic devices hold - aside the situations of many in cases with a more simple neural/ muscle-skeletal pathology - a very important intermediate/ key (considering also for continuity) systematic position within the dynamics of the - mandatory - intricate/ integrative: care and rehabilitative endeavors, that characterize the complex approach of patients with severe neurological - and consequent neuro-myo-arthro-kinetic - impairments. In this latter respect, utilization of orthoses can be framed both: within Rehabilitation Nursing (RN) - which "center on: life processes, well-being and/or optimum functioning" (Hoeman SP, 2008) - and also, respectively among effectively rehabilitative programs (applied when, considering the general evolution of the patient, they become possible - Onose G, 2011).
The lecture aims to synthetically present orthotics' basics, including with practical examples of the orthotic devices' use in NeuroRehabilitation. This includes: brief data of historical kind, items as regards their denomination - considering some main determinants, such as: anatomical region to which it/they is/are designated for application, morph-functional correction aim, (eventual) pathological condition/ therapeutic state, (commonly but more and more seldom introduced) the inventor's proper name, institution or geographic region of emergence, etc., attempts to standardize the connected naming and discussion/ examples of prescription (subsequent to a minute evaluation, resulting in a correct clinical/ functional diagnosis, in order to settle the impairment and consequent disability status - as objective/s to be corrected) -, principles of action/ related biomechanics and of materials/ design/ construction, specific indications with concise methodological guidance and comments for the applying of different orthotic devices in some more frequent neuro-myo-arthro-kinetic needing conditions, following the principal (simplest and most often used) classification criterion - the approximate big anatomical/ topographical region's one, i.e.: for the upper, respectively for the lower, limb(s) or for the spinal column.

Key words:
Orthotic device(s), orthosis(es), orthotics, splint(s), brace(s), corset(s), neuro-rehabilitation

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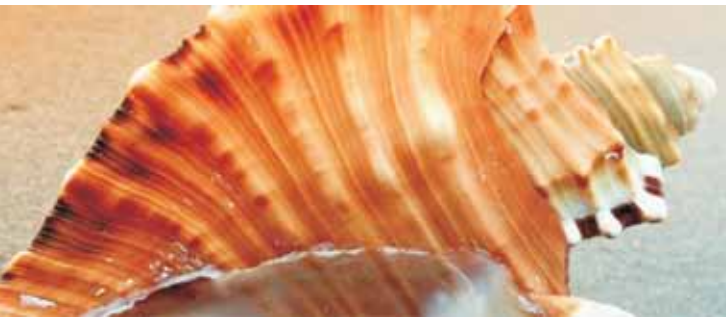

PLASTICITY OF HUMAN BRAIN STRUCTURE THROUGHOUT LIFE

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Evidence is accumulating that human brain structure is plastic throughout life. Longitudinal magnetic resonance imaging (MRI) allows us to measure individual differences in brain plasticity in children and adults. A wave of growth occurs during childhood/adolescence, where around 9 years of age a 1% annual brain growth is found which levels off until at age 13 a gradual volume decrease sets in. During young adulthood, between 18 and 35 years of age, possibly another wave of growth occurs or at least a period of no brain tissue loss. After age 35 years, a steady volume loss is found of 0.2% per year, which accelerates gradually to an annual brain volume loss of 0.5% at age 60. The brains of people over 60 years of age show a steady volume loss of more than 0.5% (Hedman et al, 2011). Brain plasticity is associated with intelligence and is at least in part heritable (Brans et al, 2010).Understanding the mechanisms underlying these plastic brain changes may contribute to distinguishing progressive brain changes in psychiatric (Brans et al, 2008; Hulshoff Pol and Kahn, 2008)and neurological diseases from healthy aging processes and aid in developing new treatments.

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COMORBIDITIES IN PARKINSON'S DISEASE

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Even though in the last decade a large progress was achieved in recognition and evaluation of non-motor signs of Parkinson's disease (PD), much less attention was payed to identification of comorbidities in PD patients, and frequently there is some confusion about these two separate entities. In this paper I will review diseases that are more frequent in parkinsonians compared to non-parkinsonian control population, such as cerebrovascular diseases, peripheral neuropathies or bone fractures. I will also try to give some clues for distiction of non-motor signs and comorbidities, which I believe are important for the PD clinician, the add-on value being here that not all complains of PD patients are due to their neurodegenerative disease and there is room for improving the quality of life and for prevention beside the anti-parkinsonian treatment.

ROBOTICS

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The neurophysiological background of Robotics in Neurorehabilitation is the evidence that intensive training (frequency and duration) and task-specific training improves significantly the neurological outcome. There are several Robotic devices on the market, more or less complex, for upper and lower limbs, with different approaches (Exoskeleton, Endeffector System). Although several critical reports the robotic training seems at least equal to intensive conventional rehabilitative therapy.

In our Rehabilitation Department we started to use Robotic gait training since 2002 and we developed different devices to improve muscle tone and motor control of upper limbs and the trunk. The clinical experience and the data will be discussed.

POTENTIAL ROLE OF NANODRUG DELIVERY OF NEUROTROPHIC FACTORS IN CENTRAL NERVOUS SYSTEM INJURY AND REPAIR

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Central nervous system (CNS) injury is caused by a variety of external and internal factors leading to serious mental and physical dysfunction [1-3]. Thus, CNS injuries caused by external factors e.g., trauma and/or neurodegenerative diseases i.e., Parkinson's, Alzheimer's, Huntington's Diseases, Multiple sclerosis, stroke, ischemia, infarction or other kinds neurological injuries require urgent neuroprotection strategies using pharmacotherapy of potential drugs in combination with neuroregenerative approaches e.g., treatment with neurotrophic factors either alone or using nanobiotechnology for enhanced drug delivery within the CNS. A combination of drug therapy using nanotechnology will enhance therapeutic measures [1-5]. Thus, the future therapeutic approaches in clinical settings will be a multimodal approach usinga combination of drugs to restore functions of the CNS in patients [4]. Our laboratory is engaged in neuroregenerative therapy in different animal models of CNS injuriesusing nanotechnological approaches for drug delivery [5-8]. Our observations in the last 9 years shows that multiple com-binations of neurotrophic factors derived from neurons, e.g., brain derived neurotrophic factor (BDNF), or glia derived neurotrophic factor (GDNF), nerve growth factor (NGF), ciliary neurotrophic factor (CNTF) when administered exogenously following CNS injury caused by trauma or hyperthermia induces remarkable restoration of neuronal, glial and endothelial cell structure and functions [1-3, 5-8]. However, similar or even improved results could be obtained by administering neurotrophic factors in such circumstances as com-pared to different combinations of neurotrophicfactors applied separately. This sug-gests that suitable combination of neuroregenerative therapy, leads to marked neuroprotection. Interestingly, the therapeutic efficiency of these neurotrophic factors is further enhanced when they are administered using nanowired technology [6]. Although, nanowired drug delivery did not alter the fundamental principles of neurotrophic factor therapy. Thus, thecombination of neurotrophicfactors that did not influence neuroprotectionwas not effective in inducing neuroprotection-following their nanodrug delivery. However, nanowired drug delivery of neurotrophins that induces profound neurorestoration is markedly enhanced by their nano-drug delivery. The functional significance of neuroprotective therapy using neurotrophic factors with or without nanodrug delivery is discussed.

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PHYSIOPATHOLOGY AND PHARMACOLOGY OF THE ENDONEURAL MICROENVIRONMENT

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The endoneurial microenvironment, delimited by the endothelium of endoneurial vessels and a multi-layered ensheathing perineurium, is a specialized milieu intérieur within which myelinated and unmyelinated axons, associated Schwann cells and other resident cells (fibroblasts, mast cells, and microvessels surrounded by pericytes) of peripheral nerves function. Regulation of the endoneurial microenvironment is achieved by two specialized interfaces: blood-nerve barrier (or blood-nerve interface) formed by endoneurial microvessels, and the perineurium. The endothelium and perineurium restrict as well as regulate exchange of material between the endoneurial microenvironment and the surrounding extracellular space. Input to and output from the endoneurial microenvironment occurs via blood-nerve exchange and convective endoneurial fluid flow. If capillary permeability to albumin increases slightly, endoneurial albumin concentration will rise and thus draw more fluid from the vascular compartment into endoneurial interstitium. The resulting endoneurial edema will elevate endoneurial hydrostatic pressure, which can negatively impact nerve conduction. From this perspective, pathophysiological changes of the nerve microenvironment can be view as a consequence of altered endoneurial homeostasis. In this regard, mast cells may be of particular note. Mast cells are tissue resident immune cells that participate in a variety of allergic and other inflammatory conditions. In most tissues, mast cells are found in close proximity to nerve endings of primary afferent neurons that signal pain (i.e. nociceptors) and also within the endoneurium. Activation of mast cells causes the release of a plethora of mediators (e.g. histamine, serotonin, heparin, proteases, pro-inflammatory cytokines, eicosanoids, chemoattractants) that can activate these nociceptors and promote pain. Further, mast cell activation can provoke edema in nervous tissues and, conceivably, contribute to the dynamic nature of the blood-nerve interface including nerve conduction block and neuropathic pain. Moreover, mast cell action can be amplified via interaction with microglia. Inhibiting mast cell (and microglia) activation could thus be of therapeutic benefit in peripheral neuropathy. This will be discussed in terms of the N-acylethanolamines, a class of naturally occurring lipidic mediator molecules, and palmitoylethanolamide in particular, which is produced on-demand within the lipid bilayer and has been described as possessing anti-inflammatory, analgesic and anti-convulsant properties.

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MULTIPLE SYSTEM ATROPHY (MSA)

MSA encompasses the three originally described disorders: olivopontocerebellar atrophy (OPCA), Shy Drager syndrome (SDS), and striatonigral degeneration (SND). The three conditions later will merge intoparkinsonism, autonomic dysfunction, and cerebellar dysfunction. These syndromes were determined to be manifestations of the same disease process and are now known as MSA.

a) Epidemiology

MSA presents on average at age 54 and median survival after diagnosis is approximately 6 years. Both sexes are affected equally. Prevalence estimates range from 2to5 per100,000 persons. Incidence is less than 1 per100,000 persons.

b) Neuropathology

Neuronal loss and gliosis is seen in the striatum, substantianigra, locus ceruleus, Edinger-Westphal nucleus, middle cerebellar peduncles, cerebellar Purkinje cells, inferior olives, intermediolateral cell columns, and Onuf's nucleus. The involvement of these nuclei and tracts correlates with the clinical findings of parkinsonism, cerebellar dysfunction, and autonomic failure.

Microscopically, MSA is characterized by glial cytoplasmic inclusions (Papp-Lantos inclusions), seen primarily in the nuclei of oligodendrocytes. Inclusions stain heavily for α-synuclein, marking MSA as a synucleinopathy. The mechanism of cell loss is unknown.

c) Clinical Features

MSA-P, the more common presentation of MSA, is characterized by parkinsonism and symmetric onset of motor signs; tremor is not a prominent feature. Dyskinesias are reported earlier than in PD. Initially, MSA-P may be difficult to distinguish from PD; however, with the passage of time, autonomic and cerebellar signs emerge.

MSA-C is more common than MSA-P in Japan, although the reasons for this are unclear. Gait ataxia appears first, followed by limb dysmetria, dysarthria, and eye movement abnormalities, including nystagmus, ocular dysmetria, slow saccades, and interruption of smooth pursuit. Essentially all patients develop some evidence of autonomic dysfunction. Urinary symptoms are common, with incontinence (71%) being more common than retention (27%). Urogenital symptoms often present early. Men develop impotence (96%), and women may experience reduced genital sensitivity and

a variable decrease in libido. Orthostatic hypotension occurs in 43% to 88% of patients. Most patients experience mild postural symptoms and approximately 15% have severe orthostatic hypotension leading to recurrent syncope. Other manifestations of autonomic dysfunction include constipation, disordered breathing, and problems with thermoregulation.

d) Investigations

Diagnosis is clinical (Tables 1 and 2), but ancillary testing may be useful. Diagnosis of possible MSA requires one criterion (b) plus two features (a) from separate domains listed in (see Table 1). Probable MSA requires autonomic failure/urinary dysfunction plus poorly levodopa-responsive parkinsonism or cerebellar dysfunction. Definitive diagnosis of MSA is pathologically confirmed by the presence of glial cytoplasmic inclusions (GCI) in association with a combination of degenerative changes in the nigrostriatal and olivopontocerebellar pathways. Tilt table testing may reveal subtle autonomic dysfunction. Electromyography/nerve conduction studiesmay show a subclinical polyneuropathy and denervation of the external anal sphincter. Neuroimaging with MRI is more useful than CT, as MRI may show cerebellar and brainstem atrophy as well as characteristic T2 abnormalities in the putamen.

e) Management

There is no therapy to slow progression of disease. Thirty percent of patients have a sustained clinical response to levodopa for parkinsonism symptoms. If there is no response to 1000 mg/day of levodopa, then the drug should be titrated down and discontinued. If orthostatic symptoms are not adequately managed by increased sodium intake and fluids, pharmacologic therapy with fludrocortisone (mineralocorticoid) or midodrine (α-agonist) may be employed. Incontinence may respond to anti-cholinergics.

Table 1: Clinical Domains, Features, and Criteria Used in the Diagnosis of MSA

I. Autonomic and urinary dysfunction

a. Autonomic and urinary features
   i. Orthostatic hypotension (by 20 mm Hg systolic or 10 mm Hg diastolic)
   ii. Urinary incontinence or incomplete bladder emptying

b. Criterion for autonomic failure or urinary dysfunction in MSA -Orthostatic fall in blood pressure (by 30 mm Hg systolic or 15 mm Hg diastolic) or urinary incontinence (persistent, involuntary partial or total bladder emptying, accompanied by erectile dysfunction) or both

II. Parkinsonism

a. Parkinsonian features
   i. Bradykinesia (slowness of voluntary movement with progressive reduction in speed and amplitude during repetitive actions)
   ii. Rigidity
   iii. Postural instability (not caused by primary visual, vestibular, cerebellar, or proprioceptive dysfunction)
   iv. Tremor (postural, resting, or both)

Criterion for parkinsonism in MSA – Bradykinesia plus at least one of items 2 to 4

III. Cerebellar dysfunction

a. Cerebellar features
   i. Gait ataxia (wide based stance with steps of irregular length and direction)
   ii. Ataxic dysarthria
   iii. Limb ataxia
   iv. Sustained gaze-evoked nystagmus

Criterion for cerebellar dysfunction in MSA - Gait ataxia plus at least one of items 2 to 4

IV. Corticospinal tract dysfunction

a. Corticospinal tract features
   i. Extensor plantar responses with hyperreflexia

Corticospinal tract dysfunction in MSA – no corticospinal tract features are used in defining the diagnosis of MSA

A feature (a) is a characteristic of the disease and a criterion (b) is a defining feature or composite of features required for diagnosis.

Table 2: Exclusion Criteria For the Diagnosis of MSA
I. History
Symptomatic onset under 30 years of age
Family history of a similar disorder
Systemic diseases or other identifiable causes for features listed in Table 2
Hallucinations unrelated to medication
II. Physical examination
DSM criteria for dementia
Prominent slowing of vertical saccades or vertical supranuclear gaze palsy*
Evidence of focal cortical dysfunction such as aphasia, alien limb syndrome, and parietal dysfunction
III. Laboratory investigation
Metabolic, molecular genetic, and imaging evidence of an alternative cause of features listed in Table 2

*In practice, MSA is most frequently confused with PD or PSP. Mild limitation of upward gaze alone is non-specific, whereas a prominent (>50%) limitation of upward gaze or any limitation of downward gaze suggests PSP. Before the onset of vertical gaze limitation, a clinically obvious slowing of voluntary vertical saccades is usually easily detectable in PSP and assists in the early differentiation of these 2 disorders.

CLINICAL TRIAL STATISTICS FOR NON-STATISTICIANS

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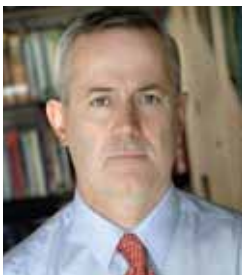
The primary goal of the statistical lectures is to provide non-statisticians with an basic understanding of the interconnections and relationships which are important in practice and the ability to implement and apply this basic knowledge in the proper interpretation of study results.

The lectures will adress the following issues:

1. Hypothesis testing and statistical significance: the basic concept of a statistical test. One-sided and two-sided tests. Definition and interpretation of P-values. Level of significance. Correct and false interpretation of significance through examples from the literature.

2. Effect sizes and confidence intervals: Basic principles and interpretation. Relationship with significance tests. Definition and handling in superiority and non-inferiority trials. Why confidence intervals rather than P-values?

3. Proper interpretation of study results: examples from recent TBI trials. Pros and cons of dichotomization. Univariate vs. multidimensional approach. Current recommendations. What can we learn from „failed“ studies.



FUNCTIONAL CONNECTIVITY CHANGES AFTER MILD TRAUMATIC BRAIN INJURY

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Traumatic brain injury is a heterogeneous disease that may affect people of all ages. Severity may range from mild with a low frequency (1 per 100) of life threatening intracranial hematoma that needs immediate neurosurgical operation and very low mortality (1 per 1000) to severe with a high likelihood of life threatening intracranial hematoma (up to 1 per 3) and a 40% case fatality rate and a 50-60% disability rate in survivors. TBI is heterogeneous in definitions, pathology, age of onset and in the presence of additional injury to other body regions.

Traumatic Brain Injury (TBI) encompasses the functional disturbances and structural damage of the brain caused by direct impact, by external acceleration, deceleration and/or rotation forces to the head.
Pathophysiologically, TBI is characterized by diffuse damage of grey matter and white matter tracts in the brain, and by contusion, laceration and intracerebral or extracerebral haemorrhage signifying focal and/or diffuse damage (primary damage). Secondary brain injury consists of the damage that occurs in the hours-dayas post injury. Both intracranial and systemic insults (e.g. hypoxia and/or hypotension) may exacerbate secondary damage.
The incidence of TBI is high, in the international literature varying between 100 and 300 per 100,000, with the highest incidence occurring in men, aged 15 to 24 years. The average age of patients with TBI is 30-40 years(1;2). Recent data indicate an increase in average age and a larger contribution of elderly patients with TBI. Approximately 90–95% of all TBIs are considered mild. Intracranial complications of mild traumatic brain injury (MTBI) are infrequent but potentially life-threatening, and may require neurosurgical intervention in a minority of cases (0.2–3.1%). Because of the importance to exclude the small chance of a life-threatening complication in large numbers of individual patients much research has been dedicated to the prediction of these complications.
The use of new MRI techniques like DWI, SWI, DTI and resting state MRI in the acute stages of TBI demonstrating the pathological heterogeneity of TBI may open ways for new intervention studies.(3;4)
It is increasingly recognized that outcome from mild TBI may be influenced by other factors than injury alone and that the patients previous history or pre-injury characteristics may modify the response of injured individuals. A central theme in MTBI is whether prolonged posttraumatic complaints have a structural or more functional basis.

We undertook a study to evaluate whole-brain resting-state networks in a homogeneous group of patients with acute MTBI and to identify alterations in functional connectivity induced by MTBI.

Methods: Thirty-five patients with acute MTBI and 35 healthy control subjects, matched in age, gender, handedness, and education, underwent resting-state fMRI, susceptibility weighted imaging, neuropsychological, and postconcussive symptom assessments. We ensured the homogeneity of the patient group by limiting the injury mechanism to fronto-occipital impacts. Alterations in functional connectivity were analyzed by using data-driven independent component analysis, which is not biased by a priori region selection.

Results: We found a decrease in functional connectivity within the motor-striatal network in the MTBI group. At the same time, patients showed deficits in psychomotor speed as well as in speed of information processing. We propose that although disorders in motor function after MTBI are rarely reported, injury still has an effect on motor functioning, which in its turn may also explain the reduction in speed of information processing. Further, we found a cluster of increased functional
connectivity in the right frontoparietal network in the MTBI group. We suggest that this abnormal increased connectivity might reflect increased awareness to external environment and explain excessive cognitive fatigue reported by patients with MTBI. It might also underlie the physical postconcussive symptoms, such as headache and increased sensitivity to noise/light.

Conclusions: We proved that whole-brain functional connectivity is altered early (within 4 weeks) after MTBI, suggesting that changes in functional networks underlie the cognitive deficits and postconcussive complaints reported by patients with MTBI.

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PARKINSON'S DISEASE REVISITED

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Parkinson's disease is a multi-system alpha-synuclein inclusion body process, involving both the central and peripheral nervous system and affecting not only the dopaminergic but also the noradrenergic, serotoninergic and cholinergic transmitter systems.

This synucleinopathy-related Lewy body pathology was found to start at clearly defined sites (medulla oblongata, pontine tegmentum, olfactory bulb and anterior olfactory nucleus) and to progressively advance in a topographically predictable sequence through the nuclear grays of the basal midbrain (including the nigral substance) and forebrain into the neocortex. The clinical expression of this process depends of the (variable) extent of the local pathology and of the local safety margins.

Clinical symptoms in Parkinson's disease (PD) comprise both motor and non-motor symptoms. In this disease, synucleinopathic-induced, nigral dopamine deficiency-related dysfunction of the basal ganglia is held responsible for the characteristic levodopa-responsive motor signs and symptoms (bradykinesia, hypokinesia, rigidity), known as parkinsonism and essential for clinical diagnosis in PD, as well as subtle emotivational and cognitive dysfunctions. Some motor symptoms, such as tremor and postural instability, and most non-motor symptoms, however, are not fully levodopa-responsive, and suggested to manifest extranigral pathology. These symptoms include autonomic, sleep, sensory and neuropsychiatric symptoms, which in some cases may precede the first signs of motor parkinsonism, closely correlating with the progression of Lewy body pathology in PD.

On top of this, (non)motor extranigral symptoms in PD might also be of iatrogenic origin, whether directly as indirectly. During conventional, oral, dopaminomimetic treatment, the progressive loss of striatal dopaminergic nerve endings with the loss of cerebral dopamine storage capacity, renders the cerebral dopamine level fully dependent of the plasma levodopa levels, thus changing dopaminergic receptor stimulation from continuous to a more pulsatile pattern. Supposedly due to this process, neuroplastic changes in (sub)cortical dopaminergic pathways might cause therapeutic response fluctuations: motor and nonmotor fluctuations with anxiety- and panick- attacks and/or mood swings, dyskinesias and punding. Finally, dopaminomimetic pharmacotherapy may also induce extranigral non-motor drug-related direct adverse effects, such as impulse control disorders.

As of yet, clinical diagnosis is only possible when significant synucleinopathic-induced, nigral dopamine deficiency-related dysfunction of the basal ganglia with characteristic signs and symptoms of levodopa-responsive motor parkinsonism (bradykinesia, hypokinesia, rigidity) becomes overt.

In many patients, though, non-motor symptoms, closely correlating with the progression of Lewy body pathology in PD, such as hyposmia, autonomic dysfunctions, mood disorders, sleep disorders, sensory disorders and/or cognitive deficits may antedate the first motor symptoms.

The recognition and treatment of these symptoms is important, as they have more impact on the quality of life in PD patients as compared to motor parkinsonism. On top of this, recognition of these manifestations in the premotor phase of PD is critical to early diagnosis and treatment, as disease-modifying drugs, once identified, should be initiated as soon as possible, preferably in this premotor phase.

CURRICULUM VITAE

ANGELO ANTONINI /ITALY

Angelo Antonini joined the Parkinson Institute in Milan, Italy in November 1997. He is now medical coordinator for Information Technology and Clinical Research at the Department of Neuroscience of the Hospital 'Istituti Clinici di Perfezionamento' in Milan, Italy. He is also aggregate Professor of Neurology at the University of Milan-Bicocca. He earned his medical degree from the Università degli Studi di Roma 'La Sapienza', Rome. In November1990 he completed his neurology training with honors and then undertook a visiting fellowship at the PET Department Paul Scherrer Institute, Villigen, Switzerland before starting his PhD in neuroradiology under the supervision of Professor Klaus Leenders. In 1995 he received the first award from the National Parkinson Foundation for 'young researchers in Parkinson's disease'. In 1996 he was awarded the Junior Faculty Award 1996/97 from United Parkinson Foundation and Parkinson's Disease Foundation for his research in the field of Parkinson's disease. His research interests include neuroimaging as well as cognitive and behavioral aspects of Parkinson's disease. His research also focuses on the use of continuous subcutaneous infusion of apomorphine and subthalamic nucleus deep brain stimulus (STN-DBS) in the treatment of serious motor fluctuations and dyskinesia of patients suffering from advanced Parkinson's disease. During his academic career he has published over 150 peer-reviewed manuscripts, over 200 abstracts and several book chapters. He serves as reviewer for the main neurology journals and is on the editorial board of Movement Disorders.

OVIDIU BĂJENARU /ROMANIA

1983 : M.D. at the Faculty of Medecine of University of Medecine and Pharmacy "Carol Davila" Bucharest
1989 : specialist in neurology, confirmed by the Ministery of Health of Romania
1993 : Ph.D. at the University of Medecine and Pharmacy "Carol Davila" Bucharest
1999 (since) : Professor of Neurology at the University of Medicine and Pharmacy " Carol Davila" Bucharest, Chairman and Head of the Neurology Department of the University Hospital of Emergency Bucharest
2000-2004 : Vice-Dean of the Faculty of Medecine - University of Medecine and Pharmacy "Carol Davila" Bucharest
2001(since) : President of the Romanian Society of Neurology
2003 – 2009 : member of the Scientific Committee of ECTRIMS
2004 - 2009 : Member of the Executive Committee of the European Society of Neurology
2008 (since) : Romania official delegate in UEMS – EBN (Board of Neurology)

*sept. 2010: elected Sectretary of the Executive Committee of UEMS-EBN

2011 (since): Director of Department of Neurology, Neurosurgery and Psychiatry of the University of Medicine and Pharmacy "Carol Davila" Bucharest

Post graduate training :
1992 - 1994 : post graduate training in clinical neurology and functional investigations of the nervous system at University " Rene Descartes"(Paris)

Fields of interest for the scientific research
- stroke, dementia and neurodegenerative diseases (in particular Alzheimer and Parkinson's disease), multiple sclerosis
- more than 300 scientific papers published and reported in different national and international scientific meetings, 5 medical books and monographies (published in Romania), co-author (1 chapter) to the "International Neurology - A Clinical Approach", Wiley-Blackwell, 2009; Principal Investigator in 12 research grants from the Romanian National Council for Science and Research, Country Principal Investigator in an International Program of Research for genetic factors in stroke patients; Country Principal Investigator – in more than 30 international, multicentric clinical trials; Principal Investigator of the research site – in more than 30 international and national multicentic trials

HEINRICH BINDER /AUSTRIA

EDUCATION:

1965 - 1972	Faculty of Medicine at the University Vienna MD since (promotion on) 1972, June 6th
1972 - 1978	University Hospital for Neurology, graduated in Medical Specialist for Neurology and Psychiatry
9/1982	Docent for neurology, a title corresponding to PhD
since 1988	Professor for Neurology, University Vienna founding member of the Austrian Society for Neurorehabilitation
5/1989	Head of the Neurological Hospital "Maria Theresien-Schlössel"
1994-2007	Head of Ludwig Boltzmann Insitute for Restorative Neurology and Neuromodulation
Since 2008	Deputy Head of Landsteiner Institute for Neurorehabilitation and Space Medicine
since 2002	Head of the Neurological Center, Otto Wagner Hospital, Vienna. Main focus: Patients with severe neurological/ neuropsychological deficits and invasive neurorehabilitation methods

currently
President of
- Austrian Society for Neurorehabilitation (OEGNR)
- European Federation NeuroRehabilitation Societies (EFNRS)

Member of
- Management Committee of the World Federation NeuroRehabilitation (WFNR)
- Managing Board of the International Danube Symposium
- Editorial Board of "Journal of Medicine and Life":

Chairman of
- Special Interest Group/WFNR "Spinal Cord Injury"
- Special Interest Group/WFNR "Early Rehabilitation"
- Scientific panel/EFNS "Brain recovery and Rehabilitation"
- Special Branch / International Danube Symposium: "NeuroRehabilitation"

Main topic of research: Neurorehabilitation, brain injury, spinal cord injury, vegetative state/ apallic syndrome (more than 140 publications)

DANA
BOERING
/GERMANY

EDUCATION:

1. Secondary School I. Slavici Arad, Romania
2. Medical School: Facultatea de medicina si Farmacie I.M.F. Cluj- Napoca, Romania

ACADEMICAL QUALIFICATIONS:

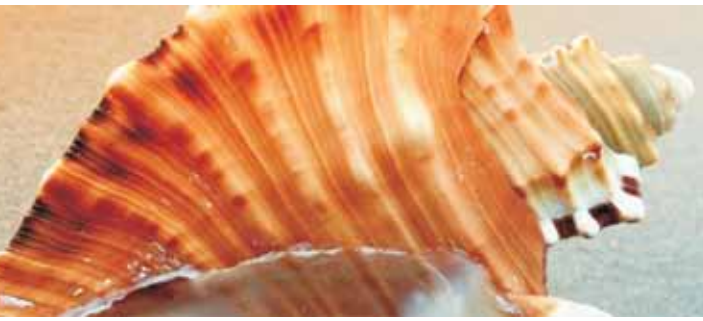
1. Dr. medic : I.M.F. Cluj Napoca 1981
2. German acknowledgement as Dr. med. 1987
3. Specialty qualification: Neurologist 1994
4. Further specialty qualification: Neurorehabilitationist 2001, Neurophysiologist 2002

EMPLOYMENT:

St. Mauritius Therapieklinik Meerbusch since 2002
Professional appointments, scientifical activities:
1994-2002 Collaboration with the University of Essen in the field of plasticity after stroke, with an emphasis on the role of theerebellum in motoric learning tasks
Since 2002 Collaboration with the University of Düsseldorf in the field of plasticity after stroke
2009 Collaboration with the Coma Science Group Liege/Belgium
2010 Collaboration with the Neuroradiology of the Wake University Winson- Salem U.S.A. in a study on network properties of DOC patients

NATAN BORNSTEIN /ISRAEL

EDUCATION
1970-73 University of Sienna, Medicine, Sienna, Italy
1973-79 Technion Medical School, Hifa, Medicine, MD, 1979
Date of receiving specialixation certificate: 11 September, 1984
Title of Doctoral dissertation: Dextran 40 in acute ischemic stroke
Name of Supervisor: Dr. Jacob Vardi
FURTHER EDUCATION
1978-83 Tel-Aviv University, Sackler Faculty of Medicine, neurology (residence), Israeli Board certified in Neurology, 1983
1979-83 Tel-Aviv University, Sackler Faculty of Medicine, Post graduate studies in Neurology
1984-87 Sunnybrook Medical Center, University of Toronto, M.R.C stroke, Fellowship
ACADEMIC AND PROFESSIONAL EXPERIENCE
1982-1995 Tel-Aviv University, Neurology, instructor
1991-present European stroke Conference (ESC), Executive committee
1995-1999 Tel-Aviv University, Neurology, Senior lecturer
1995 Eliprodil CVD 715 clinical trial, Steering Committee
1995-1997 International Stroke Study (IST), Steering Committee
1995-1999 American Academy of Neurology, Member of the International Affairs Committee
1996 Asymptomatic Carotid Stenosis and Risk of Stroke(ACSRS), Advisory Committee
1996-present The Mediterranean Stroke Society (MSS), President
1996-2002 EFNS, Management Committee
1997-2009 Israeli Neurological Association, Secretary
1999-present Tel-Aviv University, Neurology, Associated Professor
2001- present European Society Neurosonology and Cerebral Hemodynamics (ESNCH) Executive committee
2005-present Neurosonolgy Research Group, Executive committee
2006-present European Master in Stroke Medicine, Member of faculty
2006-2008 NEST II clinical Trial, Steering Committee
2006-present SENTIS clinical Trial, Steering Committee
2006-present CASTA Trial, Steering Committee
2006-present Brainsgate clinical Trial, Steering Committee
2008- present World Stroke Association (WSO), Vice president
2009-present Israeli Neurological Association, Chairman
2009-present European Stroke Organization (ESO), Member on the board of directors
2010- NEST III clinical Trial, Steering Committee



PROFESSIONAL ACHIEVEMENTS- EDITORIAL BOARD
1991-present Neurological Research Journal, Guest Editor
1991-present STROKE, Member of the editorial board
1998-present European Journal of Neurology, Member of the editorial board
1999-present Journal of Cerebrovascular disease, Member of the editorial board
2000-present Journal of Annals of Medical Science, Consulting Editor
2001-present Journal of Neurological Science (Turkish), Member of the editorial board
2001-present Acta Clinica Croatica, Member of the editorial Counsil
2003-present Italian Heart Journal, International Scientific Board
2003-present Journal of Neurological Sciences, Guest Editor
2004-present Turkish Journal of Neurology, International Advisory Board
2005-present Archives of Medical Sciences (AMS) , Member of the Editorial Board
2006-present Journal of Cardiovascular Medicine, International Scientific Board
2006-present International Journal of Stroke, Editorial Board
2006-present Acta Neurologica Scandinavica, Editorial Board
2009-present American Journal of Neuroprotection& Neurogeneration (AJNN) Member of the Editorial Board
2010 Neurosonology, International Editorial Board
2010 Frontiers in Stroke, Review Editor
PROFESSIONAL ACHIEVEMENTS- REVIEWER
1998-present Lancet, Ad Hoc reviewer
1998-present Diabetes and its complications, Ad Hoc reviewer
1999-present Journal of Neuroimaging, Reviewer
1999-present Journal of Neurology, Ad Hoc reviewer
2000-present Neurology, Ad Hoc reviewer
2003-present Israeli Medical Association Journal (IMAJ), Reviewer
2003-present Acta Neurologica Scandinavica, Ad Hoc reviewer
2006-present Journal of Neurology, Neurosurgery & Psychiatry, Reviewer
2010- European Neurology, Ad Hoc reviewer
MEMBERSHIP IN PROFESSIONAL SOCIETIES
1977-present Israeli Medical Association
1983-present The Israeli Neurological Association
1985-present Stroke Council of the American Heart Association (Fellow)
1986-present American Academy of Neurology
1986-present Neurosonology Research Group of the World Federation of Neurology
1987-present Stroke Research Group of the World Federation of Neurology
1990-2008 International Stroke Society
1995-2008 European Stroke Council
1995-present Mediterranean Stroke Society (MSS)
1998-present European Neurosonology Society
2005-present World Stroke Organization (WSO)
2008-present Fellow of the European Stroke organization (FESO)

VOLKER HÖMBERG /GERMANY

MEDICAL DIRECTOR

St. Mauritius Therapy Hospital Meerbusch

PERSONAL DATA

Born 25 July 1954
Married to Priv.-Doz. Dr. Kristina Müller, paediatric neurologist

MEDICAL CAREER

1973 - 1980	School, Universities of Düsseldorf and Freiburg; Elective in Neurology at Boston City Hospital, Boston, Mass.; National Hospital for Nervous Diseases, London
since 1975	Junior researcher in the Department of Neuropsychology at the C. & O. Vogt Institute for Brain Research, Düsseldorf and the Department of Neurology, Freiburg (Prof. R. Jung)
1980 - 1981	Research fellow in the Department of Neuropsychology (Prof. G. Grünewald) at the C. & O. Vogt Institute for Brain Research, Düsseldorf
since 1981	Clinical training in the Department of Neurology (Prof. H.-J. Freund), Heinrich- Heine-University Düsseldorf
since 1985	Senior registrar in the Department of Neurology, Heinrich-Heine-University Düsseldorf
since 1987	Senior investigator for the German Research Council Special Task Force in Neurology at Heinrich-Heine-University (SFB 200 and SFB 194)
1987-2005	Medical director of the Neurological Therapy Center (NTC), Heinrich-Heine-University Düsseldorf
since 1988	Board examiner for Neurology at the local examination board (Ärztekammer Nordrhein)
1989-1997	Vice president of the German Society for Neurological Rehabilitation
1993	Habilitation in Neurology, Heinrich-Heine-University Düsseldorf
since 1995	Board examiner for physical medicine and rehabilitation (Ärztekammer Nordrhein)
1997-2005	Medical director of the Neurological Therapy Center, Cologne
1998-2004	President of the German Society for Neurological Rehabilitation
since 2000	Medical director and head of neurology, St. Mauritius Therapy Hospital, Meerbusch
since 2003	Secretary General World Federation for NeuroRehabilitation (WFNR)
since 10/2004	Vice president of the German Society for Neurological Rehabilitation
since 2005	Panel-Chairman Neurorehabilitation for European Federation Neurological Societies (EFNS)

ARNON KARNI
/ISRAEL

A. Education:
- Years:1984-1985, Degree: first year of the program, Institution: Natural Science School, Tel-Aviv University, Tel-Aviv, Israel.
- Years:1985-1991, Degree: M.D, Institution: Medical School - Sackler's medical faculty, Tel Aviv University, Tel-Aviv, Israel.
- Year: 1999- 2002, Title: National MS Society Research Fellow, Place of Training: Center for Neurologic Diseases, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, U.S.A. Mentor: Prof. Howard L. Weiner
- Year 2010, Degree: Ph.D. Institution: The Hebrew University of Jerusalem. Topic: The basis for immune dys-regulation in multiple sclerosis. Mentors: Prof. Oded Abramsky and Prof. Howard L. Weiner

B. Academic and Professional Experiences:
- Years: Feb. 1. 1992- Feb. 1.1993, Title: Internship, Discipline: Medicine, Place of Training: Tel-Aviv Sorasky Medical Center. Tel-Aviv, Israel.
- Years: 1993-1998, Title: Residency, Specialty: Neurology, Place of Training: Department of Neurology Hadassah University Hospital, Jerusalem, Israel.
- Year: 1996 (6 month), Title: Basic Science, Discipline: Neuroimmunology, Mentor: Prof. Avraham Ben-Nun, Place of Training: Department of Immunology, Weizmann Institute of Science, Rehovot, Israel.
- Years: July 1999- November 2002 – Research fellowship, Center for Neurologic Diseases, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, U.S.A
- Year: 1/1/2003- Head of Neuroimmunology Clinic and senior physician in Department of Neurology at Sourasky Tel-Aviv Medical Center, Tel Aviv.

C. Academic Appointment:
Year 2006: Academic Title: Lecturer in Neurology, Faculty of Medicine, Tel Aviv University
Year 2011: Academic Title: Senior Lecturer, Israel Academic College, Ramat Gan

D. Membership:
Israle Neurology Society
Istaeli Neuroimmunology Society – member of the executive committee
ECTRIMS - council member

E. Academic and Professional Awards
Year: 1993 – The Dean Prize for excellency, Natural Science School , Tel-Aviv University, Tel-Aviv, Israel.

Yaer: 1997 – A grant from The Joint Research Fund of The Hebrew University and Hadassah, Saare Tzedek, Bikur Holim and Kaplan Hospital

Year: 1997 - The Medicine Faculty - Hebrew University Prize for Excellency in original research
Year: 1997 - The Prize for the Best Presentation among the Residents., The Israel Society of Neurology - Annual meeting 1997. (Linkage analysis of HLA class II genes in Jewish multiplex families with multiple sclerosis).

Year: 1999 – The United States National Multiple Sclerosis Society Fellowship Award –3 years fellowship.

Year 1999 – American Physicians Fellowship (APF) for Medicine in Israel – APF's Dr. Manuel and Helen Glazier memorial Fund.

Year 2001: Travelling award for the NIH chemokine receptors and multiple sclerosis workshop, Washington , DC, U.S.A

Year 2002: Travelling award for federation of clinical immunology societies (FOCIS) meeting, San Francisco, CA, U.S.A
Year 2010: Israel Scientic Foundation research grant: The role of BMPs and their antagonist in multiple sclerosis

F. Reviewer

2007 – Present, Journal of Neuroimmunology - Ad voc reviewer
2010 - Present, Journal of Neurological Sciences - Ad voc reviewer
2010 – ISRN Neurulogy – editorial board

DAFIN F. MUREŞANU /ROMANIA

Mureșanu Fior Dafin, MD, PhD, MBA, is Professor of Neurology, Chairman of the Clinical Neurosciences Department, University of Medicine and Pharmacy "Iuliu Hatieganu" Cluj-Napoca, member of the Academy of Medical Sciences, Romania. He is also President of the Society for the Study of Neuroprotection and Neuroplasticity. In these roles, he acts as coordinator in international educational programs of European Master type (European Master in Stroke Medicine, University of Krems), organizer and co-organizer of European and international schools and courses (Eastern European Neurology Summer School for Young Neurologists - www.ssnn.ro, European Stroke Organisation Summer School, Danubian Neurological Society Teaching Course). His activity includes his involvement in many clinical studies and research projects, his membership in the executive board of many national and international societies, participations as invited speaker in national and international congresses, and a significant portfolio of scientific articles, contributions in monographs and books published by prestigious international publishing houses. Prof. Dr. Muresanu has been honoured with the Faculty of Medicine, University of Medicine and Pharmacy "Iuliu Hatieganu" Cluj-Napoca "Octavian Fodor Award" for the best scientific activity of the year 2010 and the 2009 Romanian Academy of Medical Sciences "Gheorghe Marinescu Award" for advanced contributions in Neuroprotection and Neuroplasticity.

GELU ONOSE
/ROMANIA

Dr. Gelu Onose - 55 years; graduated, in 1982, from the Faculty of General Medicine, within the Inatitute of Medicine and Pharmacy, in Bucharest, Romania

- Professor at the (State) University of Medicine and Pharmacy (UMP) "Carol Davila", in Bucharest
- Doctoral/ Post-Graduate Tutor - at the (State) University of Medicine and Pharmacy "Carol Davila" (UMPCD), in Bucharest

- MD; - PhD; - MSc

- Senior Physician of : - Physical & Rehabilitation Medicine (PRM) and
- Gerontology & Geriatrics (G-G)
Competences in : - General Echograpy
- Management of sanitary services

- Chief of the of the UMPCD PRM Discipline and of the (neural-muscular) Clinic Division - the National Reference Center for NeuroRehabilitation - and of its RDI Nucleus, of theTeaching Emergency Hospital"Bagdasar-Arseni" (TEHBA), in Bucharest

- President Co-Founder of the Romanian Society for Neurorehabilitation (RoSNeRa) - affiliated to the World Federation for NeuroRehabilitation (WFNR) - member of the Management Committee - and respectively, of the Romanian Society for Spinal Cord Pathology, Therapy and Rehabilitation (RoSCoS) - affiliated to the International Spinal Cord Society (ISCoS) and to European Spinal Cord Injury Federation (ESCIF)

- A member of the Scientific Committee, afferent to the Prezidium of the world Academy for Multidisciplinary Neurotraumatology (AMN)

- Selected and invited - as among "Highly-specialized scholars" - by Thomson Reuters to participate in the invitation-only "Academic Reputation Survey", within its related partnership with Times Higher Education's influential World University Rankings: 2010, 2011, 2012

- Invitated Peer-Reviewer (March 2010) by the "Journal of Molecular Histology" and (March, 2012) by the "Spinal Cord" journal (both ISI Thomson Reuters rated)

- A member of the Board of the Romanian Society of Physical and Rehabilitation Medicine

- Gest Editor within the Special Issue, Second Edition, Vol. 4 of the Journal of Medicine and Life, 2011

- 8 published medical books - one of them : "The Spondyloarthropathies", and received, in 2002, the "Iuliu Hatieganu" Award of The Romanian Academy)

- 2 chapters within medical books

- About 200 scientific works and papers - communicated within national and international scientific meetings and/or published in peer-reviewed or non peer-reviewed medical journals - and professional interviews/ articles, in mass-media

- 3 Patents/ Invention Certificates (plus an Utility Model), appointed by the State Office for Inventions and Marks (SOIM/ OSIM)

- Main awards: the "Iuliu Hatieganu" Award of The Romanian Academy (2002); the Award of the (Romanian) National Authority for Scientific Research for the RDI project acronymed "ACTUAT" (2006); the Gold Medal at the International Saloon of Inventions, Geneve/ Switzerland for the RDI project acronymed "MOD" (2008)

- A member of the Scientific Council/ Editorial Board of medical journals:
  - "Journal of Medicine and Life" (rated in Index Medicus, Medline)
  - "Infomedica"
  - (Romanian) "Rehabilitation, Physical Medicine and Balneology"
  - "Romanian Neurosurgery"
  - "Industria Textila" (ISI Thomson rated journal)

- A member of the :
  - Romanian Medical Association (RMA)
  - Romanian Society of Physical and Rehabilitation Medicine (PRM) - including of its Board
  - Romanian Society of Neurosurgery (RSN)
  - Romanian Society of Biomaterials (RSB)
  - Balkan Medical Union (BMU),
  - International Society of Hydrothermal Technique (SITH - the National Council of the Romanian Section SITH - RS),
  - British Society of Gerontology (BSG)
  - The International Spinal Cord Society (ISCoS)
  - The European Spinal Cord Injury Federation (ESCIF)
  - World Academy for Multidisciplinary Neurotraumatology (AMN) - a member of the Scientific Committee, afferent to the Prezidium
  - World Federation For Neurorehabiliation (WFNR) - a member of the Council, Management Committee
  - (International) Association for the Study of Medical Education (ASME, UK)

HILLEKE HULSHOFF POL /THE NETHERLANDS

Hilleke Hulshoff Pol is professor of neuroscience with a particular emphasis on psychiatric disorders and head of the Neuroimaging research group at the Department of Psychiatry of the University Medical Center Utrecht (UMCU), theNetherlands. She uses Magnetic Resonance Imaging to study structural and functional plasticity of the human brain in health and in psychiatric diseases, particularly schizophrenia. She studies the influences of genes and environmental factors longitudinally in healthy twin-pairs and in twin-pairs concordant and discordant for psychiatric diseases. She has published over 120 articles in international scientific journals, and is VIDI-laureate of the Netherlands Organisation for Scientific Research (NWO) and received a High-Potential grant of the Utrecht University (UU).

BOGDAN O. POPESCU /ROMANIA

Academic Education and Appointments

1996	MD, 'Carol Davila' University School of Medicine, Bucharest, Romania
1997 - 2002	Resident in Neurology, University Hospital Bucharest
2000 - 2009	Assistant Professor, 'Carol Davila' University School of Medicine
2001	PhD, 'Carol Davila' University School of Medicine - suma cum laudae
2002 - 2008	Neurologist, University Hospital Bucharest
2004	PhD, Karolinska Institute, Stockholm, Sweden
2005 -	Head of Laboratory of Molecular Medicine, 'Victor Babeş' National Institute of Pathology, Bucharest, Romania
2008-	Senior Neurologist, University Hospital Bucharest
2009 -	Lecturer, 'Carol Davila' University School of Medicine
2009 -	Senior Researcher, 'Victor Babeş' National Institute of Pathology, Bucharest, Romania

Awards

1999	Beaufour-Ipsen prize for the best research study in neurology
2000	Young histochemist award - International Society of Histochemistry and Cytochemistry
2004	Diploma of scientific merit – 'Victor Babeş' National Institute of Pathology
2007	Romanian Academy award for medical research
2010	'Science and Art National Foundation Award of Excellence for research in the field of Neuroscience and Neuropathology

Other current activities

Guest editor for Alzheimer's review series at Journal of Cellular and Molecular Medicine
Executive editor of Romanian Journal of Neurology
Secretary General of the Romanian Society of Neurology
Research director of the Society for the Study of Neuroprotection and Neuroplasticity
Director, Victor Babeş' National Institute of Pathology, Bucharest, Romania

LEOPOLD SALTUARI /AUSTRIA

After completing his study of Medicine in Innsbruck, Austria, he was a resident in the speciality of Neurology at the University of Pavia, Italy, from 1978 to 1983. Further study in the specialization of Physical Medicine and Rehabilitation was completed in 1986.

From 1983 to 1995 Dr. Saltuari was Head of Department on the Neurology Ward IIS/IV at the University Clinic in Innsbruck, specializing in post-acute rehabilitation for stroke and brain-injury patients. During this period, eight physicians completed their residency in Neurorehabilitation under his tutelage
Dr. Saltuari introduced new rehabilitation techniques such as cortical facilitation in Austria and developed new therapeutic techniques, e.g. intrathecal application of Baclofen in patients with supraspinal spasticity.
The government of South Tyrol (Italy) appointed Dr. Saltuari in 1985 to the Commission for Development of National Laws for Rehabilitation.

From 1988-1995 he served as Director of Therapy (Physical, Occupational, and Speech Therapy) in the Department for Neurology in the University Clinic in Innsbruck.

In 1988 Dr. Saltuari was appointed as Medical Director of the School for Occupational Therapy, where he introduced new functional aspects to the educational course. He was active in the "Project Group for Neurological Rehabilitation", reporting to the government of Tyrol in 1992.

Between 1988 and 1995 he was Director of the Laboratory for Evoked Potentials at the University of Innsbruck.

In 1987 and in 1988 he was in residence for several months at Baylor College of Medicine in Houston, Texas. The main area of this research assignment was the treatment of spasticity and pain in hemiplegic and spinal cord injured patients, as well as the treatment of pain by techniques of restorative neurology.

In 1992 Dr. Saltuari was awarded the Venia legendi in Neurology with the theme "Baclofen in Spasticity", in which the efficacy of intrathecal application of Baclofen in cases of supraspinal spasticity was described for the first time.

Dr. Saltuari has been Medical Director of the Department of Neurology in the Hochzirl Hospital since 1995. He is also Vicepresident of the Austrian Neuromodulation Society – AUNS.)
Since 1988 he has been active in the further education for Physical Therapists in Neurorehabilitation at the Scientific Academy of Lower Austria. He was elected President of the Austrian Society for Neurorehabilitation in 2002 and is the actual Past-president.
Dr. Saltuari has submitted over 200 publications dealing with neurorehabilitative subjects as well as with acute neurological topics.

Since 1986 Dr. Saltuari has been Lecturer for Neurorehabilitation and Evoked Potentials at the University for Medicine in Innsbruck and since 1995 on the staff of the Institute for Sport Science. Since October 2009 he is the Director of the Research Department for Neurorehabitation South Tyrol, Bolzano, Italy.
Since 2012 Prof. Saltuari is member of the Editorial Board of Functional Neurology.

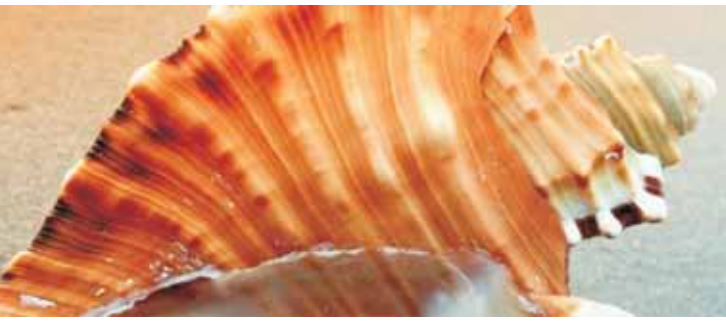
HARI SHANKER SHARMA /SWEDEN

Hari Shanker Sharma, (Swedish Citizen), Docent in Neuroanatomy (UU); Professor of Neurobiology (MRC), is currently working in Uppsala University Hospital, Department of Surgical Sciences, Division of Anesthesiology & Intensive Care Medicine, Uppsala University, Sweden.

Career History on Research in Neuroscience

Hari Sharma was born on Jan 15, 1955 in an Industrial town Dalmianagar (Bihar), India in a well-reputed family: Father Shri Ram Rup Sharma, M.Eng. (Cal), and one of the founders of Paper Factory under Rohtas Industries Ltd. Hari Sharma did his Higher Secondary Schooling in 1969 from Dalmianagar and enrolled in Bihar University, Muzaffarpur for higher studies. He did his Bachelor of Science with Honors from the prestigious L S College Muzaffarpur in 1973 and secured 1st position in his batch. He obtained his Master Degree from Bihar University with special expertise in Cell Biology in 1976 and awarded Gold Medal of Bihar University for securing 1st potion in the 1st Class. Having a knowledge in cell biology with special interest in the central nervous system, Hari Sharma joined the group of Professor Prasanta Kumar Dey, a neurophysiologist by training in the Department of Physiology, Institute of Medical; Sciences, Banaras Hindu University, Varanasi in 1977 to obtain Doctor of Philosophy Degree (D. Phil) in Neurosciences. In the lab he conducted experiments on morphine dependence and withdrawal in relation to body temperature regulation, behavioral changes and neurochemistry in rat and mice models. In addition he was trained as neurophysiologist to record electrophysiological activity in relation to stress, hyperthermia and drugs of abuse. Hari Sharma was always fascinated by the role of blood-brain barrier (BBB) in various experimental conditions and wanted to know whether brain disease has any relation with the spontaneous disruption of the BBB. His curiosity about the role of the BBB breakdown in stress condition leading to mental diseases was the basis of his Doctoral studies on “Blood-Brain Barrier in Stress” in which he for the first time showed that long or short term stress can disrupt the BBB and disrupts the EEG activity. These changes can be altered by drugs capable to modulate neurochemical metabolism of serotonin, prostaglandins and opioids in the CNS. On this work, he was awarded Ph D in 1982, that was examined and approved by the renowned team of experts on the BBB, namely: the father of Blood-Brain Barrier Research, Stanley I Rapoport of NIH, Bethesda, Maryland, USA; a pioneer on BBB in hypertension Professor Barbro Johansson, Department of Neurology of Lund University, Lund, Sweden; and noted Neuroanatomist with special regard to BBB Erik Westergaard, University of Copenhagen, Copenhagen, Denmark.

Hari Sharma after carrying out several Govt. of India Research Projects on the BBB and brain dysfunction (1982-1987), joined the lab of Neuropathology at Uppsala University with Professor Yngve Olsson in 1988 to expand his knowledge on the passage of tracer transport across the BBB in stress caused by traumatic insults to the Brain and Spinal cord at light and electron microscopy. Dr Sharma awarded the prestigious Alexander von Humboldt Foundation Fellowship of German Govt. (1989-1991) to work on hyperthermia induced BBB dysfunction at the ultrastructural level in the laboratory of Professor Jorge Cervós-Navarro (recognized as living “Legends in Neuropathology in Europe”, World Federation of Neuropathology in 1990, Kyoto, Japan, and later awarded with the German Govt. highest Civil Award, Bundestag by German Chancellor in 1996).



After that Dr Sharma came back to Uppsala to continue his research on Neurotrauma and established a network of collaboration on "Experimental CNS Injury Research Group" with key collaborators in various parts of Europe, USA, and Australia including his parent Institutions in India that is still continuing. The works carried out by Dr Sharma on the pathophysiology of blood-brain barrier in hyperthermia using immunohistochemistry and electron microscopy in the Neuroanatomy Department of Uppsala University (1995-1999). On his work on hyperthermia Dr Sharma was decorated with prestigious Neuroanatomy award "Rönnows Research prize" of Uppsala University for "best neuroanatomical research of the year 1996" followed by the Award of the Degree of Doctor of Medical Sciences of Uppsala University in Neuroanatomy in 1999 (examined and approved by another legend of Blood-brain barrier Research, Professor David Begley, Kings College London, UK). The Uppsala University Thesis of Dr Sharma was also selected for the Best Thesis Award of the Medical faculty, "The Hwassers Prize" of 1999. Subsequent research of Dr Sharma in Uppsala University on the neurobiology of hyperthermia in relation to the Blood Brain barrier and Brain edema (2000-2003) has earned the prestigious title of Docent in Neuroanatomy of Medical Faculty, Uppsala University (approved and recommended by eminent Neuroanatomist, Professor Ole Petter Ottersen, University of Oslo, Norway) in April 2004.

Academic positions:

Director of Research, CNS Injury & Repair (since 1991-)
Professor of Neurobiology (MRC) (since 1999-)
Docent in Neuroanatomy (since 2004-)
Visiting Professor Uppsala University (1988-1989)
Humboldt Fellow, Berlin Free University (1989-1991)
Research Scientists Grade A Banaras Hindu University, India (1987-1989)
Research Associate Banaras Hindu University, India (1982-1987)

STEPHEN D. SKAPER /ITALY

STUDIES:
B.S. (chemistry) Illinois Institute of Technology (1969); Ph.D. (biochemistry) University of South Dakota (1973); Laurea in chemistry, University of Padova (1990)

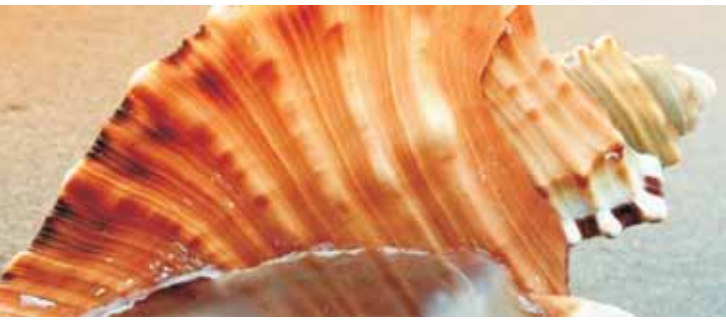
CAREER:
NIH Postdoctoral Fellow, Department of Medicine, University of California, San Diego (1973-1976); Fellow in Human Genetics, Department of Pediatrics, Case Western Reserve University, Cleveland, Ohio (1977); Postgraduate Research Biologist, Department of Biology, University of California, San Diego (1978); Assistant Research Biologist, Department of Biology, University of California, San Diego (1979-1982); Associate Research Biologist, Department of Biology, University of California, San Diego (1983-1987); Head, Laboratory of Neuropharmacology, Neuroscience Research Laboratories, Fidia S.p.A. - Abano Terme, Italy (1987-1993); Principal Scientist and Head, Laboratory of Cell Biology, Researchlife S.c.p.A. (a Lifegroup Company), Biomedical Research Center, St. Thomas Hospital, Castelfranco Veneto (TV), Italy (1993-1996); Visiting Scientist, Department of Pharmacology, University of Padova, Padova, Italy (1997); Assistant Director, Molecular Neurobiology Research, SmithKline Beecham Pharmaceuticals, New Frontiers Science Park, Harlow, United Kingdom (1998-2001); Senior Team Leader, Migraine and Stroke Research, Neurology & GI Centre of Excellence for Drug Discovery, GlaxoSmithKline R & D Limited, Harlow, United Kingdom (2002-2003); Senior Team Leader, Neuro Cell Sciences/Neurodegeneration Research, Neurology & GI Centre of Excellence for Drug Discovery, GlaxoSmithKline R & D Limited, Harlow, United Kingdom (2004-2007); Senior Team Leader, Target Validation Dept (Cognition and Pain), Centre of Excellence for Drug Discovery, GlaxoSmithKline R&D Limited, Harlow, United Kingdom (2008); Adjunct Professor, Department of Pharmacology and Anesthesiology, University of Padova, Padova, Italy (2009-present).

PROFESSIONAL MEMBERSHIPS:
Sigma I (The Scientific Research Society); Phi Lambda Upsilon (honorary chemistry society); Alpha Chi Sigma (professional society in chemistry/chemical engineering); Society for Neuroscience

JOURNALS EDITED:
Editor-in-Chief, CNS & Neurological Disorders – Drug Targets; Associate Editor, American Journal of Neuroprotection and Neuroregeneration; Councilor, International Association of Neurorestoratology

REVIEW PANELS:
The Wellcome Trust (UK), Medical Research Council (UK), Biotechnology and Biological Sciences Research Council (BBSRC) (UK), Austrian Science Fund (ad hoc review panel to evaluate interdisciplinary doctoral programmes in neuroscience), Dutch Internationale Stichting Alzheimer Onderzoek (The Netherlands), National Science Foundation (US), The Alberta Heritage Foundation (Canada)

RESEARCH INTERESTS:
Molecular biology and cellular mechanisms of cell death in CNS aging and neurodegenerative disorders and neuroinflammation.Track record of drug discovery project leadership in kinases, ion channels, G-protein-coupled receptors, DNA repair enzymes,



growth factors, identification and optimization of tools for target validation studies, utilising RNAi, conditional and viral knockdown\outs\ins, transcriptomics, proteomics and in vitro cell-based disease or mechanism relevant assays in rodent systems.

PUBLICATIONS: OVER
230 publications in the neurosciences, including book chapters and symposia proceedings.

PATENTS:
Pharmaceutical compositions containing monosialoganglioside GM1 or derivative thereof suitable for the treatment of Parkinson's disease (Patent No.: US 6,620,792 B1), use of CRF receptor agonists for the treatment or prophylaxis of diseases, for example neurodegenerative diseases (US 2003/0186867 A1), treatment of conditions with a need of GSK-3 inhibition (PCT WO 02/062387 A1), use of CRF receptor agonists for the treatment or prophylaxis of diseases, for example neurodegenerative diseases (PCT WO 01/72326 A1), use of monosialoganglioside GM1 or N-dichloro-acetyl-lyso-GM1 for preventing or reversing neuronal degeneration induced by long term treatment with L-DOPA in the therapy of Parkinson's disease (EP 0 770 389 A1)

REVIEWER FOR JOURNALS:
Journal of Neuroscience, PNAS, Nature Reviews, The FASEB Journal, Journal of Neurochemistry, Journal of Cell Biology, Neurobiology of Disease, Neurobiology of Aging, Experimental Neurology,Molecular & Cellular Neuroscience, Journal of Pharmacology and Experimental Therapeutics, Neuroscience Letters, British Journal of Pharmacology, Neuropharmacology, European Journal of Pharmacology, Biochimica et Biophysica Acta, Biochemical and Biophysical Research Communications, Brain Research/Molecular Brain Research

DANIEL D. TRUONG /USA

Dr. Daniel Truong received his training in Movement Disorders by Professor Stanley Fahn at Columbia University and David Marsden at the National Hospital for Nervous Disease at Queen Square, London, UK. He founded the program for Parkinson Disease and Movement Disorders at the University of California, Irvine in 1991, before leaving to form The Parkinson and Movement Disorder Institute at the Orange Coast Memorial Medical Center also in California. Dr Truong serves on many committees of national and international professional organizations. He currently chairs the educational committee of the World Federation of Neurology Association for Parkinsonism and Related Disorders. Dr. Truong serves as editor on the editorial board of different professional journals and is the author of over 130 peer review articles. He is also active in different layman organizations.

JOHANNES C. VESTER /GERMANY

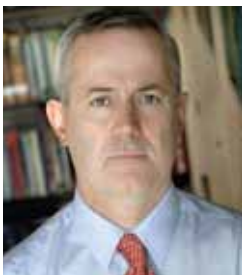
Born, 1952, he specialized in Veterinary Medicine between 1971 and 1974 at the University in Munich, then changed to the University in Cologne in 1974 and specialized in Human Medicine from 1974 to 1980. In 1976 to 1979, he additionally studied biometric methods for pharmacology and clinical research at the Institute for Data Analysis and Study Planning in Munich.

While studying human medicine, he completed research work on pattern recognition in the visual brain and developed a pharmacodynamic Neuron Simulation Model at the Institute for Medical Documentation and Statistics of the University at Cologne.

From 1985 to 1995, he was member of the Ultrahigh Dexamethasone Head Injury Study Group and leading biometrician of the German GUDHIS Study.

Since 1982 he holds advanced training courses on biometry for professionals in clinical research and university establishments. His work also involves human engineering of biometric software and GCP-compliant tutorials for biometric appraisal of clinical studies.

Since 1995 he cooperates closely with the Institute for Data Analysis and Study Planning as Senior Consultant for Biometry & Clinical Research. He planned and evaluated about 150 randomized clinical studies worldwide and is member of various international advisory boards including participation as biometric expert in regulatory authority panels and in FDA, EMEA, and BfArM hearings.



PIETER E. VOS /THE NETHERLANDS

Pieter Vos is neurologist at the Institute of Neurology at Radboud University Nijmegen Medical Centre in the Netherlands. Research activities over the last 15 years have been dedicated to traumatic brain injury. Focus of the research activities is aiming to unravel the clinical, biochemical and genetic determinants of neuroplasticity and recovery after mild, moderate and severe traumatic brain injury. Pieter Vos is founder of the Dutch working group on Neurotraumatology. Current international activities: chairman of the scientist panel on neurotraumatology and head of the task force mild traumatic brain injury, both residing under the European Federation of Neurological Societies. He is a member of the editorial board of the European Journal of Neurology and treasurer for the Academia Multidisciplinaria Neurotraumatologica.

ERIK CH. WOLTERS /THE NETHERLANDS

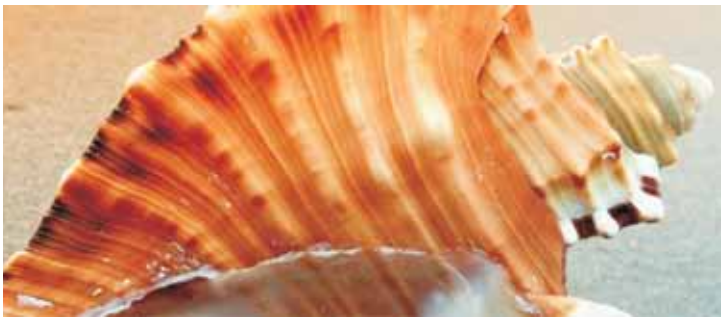
Prof. Erik Wolters is a member of the Dutch Association of Neurology, who served initially (1978-1984) at the University of Amsterdam (AMC) and later (from 1984-2010) at the VU University Medical Center in Amsterdam. In 1986 he worked during a year at the Dept. of Neurology at the University of British Columbia in Vancouver where he got a special training in basic neuroscience, related to the field of Movement Disorders. Subsequently, he was directing a clinic for Movement Disorders at the VU-UMC where he was appointed to full professor in 2000. His research comprises premotor symptoms (hyposmia, autonomic failure, sleep disorders, mood disorders, cognitive dysfunction, dementia) and premotor diagnosis in Parkinson's Disease (PD) as well as non-motor symptomatology in Parkinson's Disease, in particular PD-related depression, dementia and psychosis. After his retirement in 2010, professor Wolters joined the department of neurology of the University of Zurich, Switzerland, as well as Amarna Therapeutics, Leiden, the Netherlands, to work on Parkinson's disease-related genetic therapeutical strategies and stem cells.

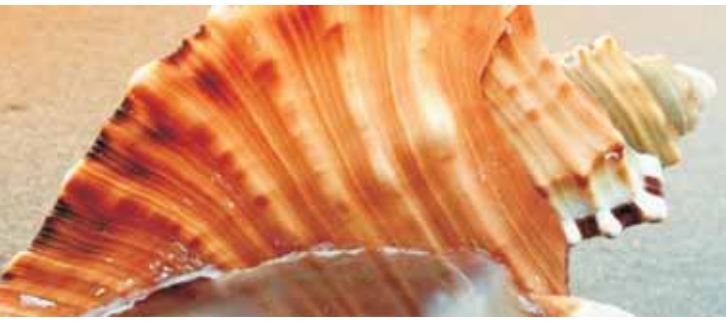
Dr Wolters wrote over 250 peer reviewed medical articles, mainly dealing with Parkinson's Disease, as well as many book chapters, and various textbooks on Neurology and Movement Disorders. He also (co)organised various international congresses, among them the first, third and fifth International Congress on Mental Dysfunctions in Parkinson's Disease (Amsterdam), and the 17th (Amsterdam), 18th (Miami),19th (Shanghai) and the upcoming 20st (Geneva) WFN World Congress on Parkinsonism and Related Disorders.

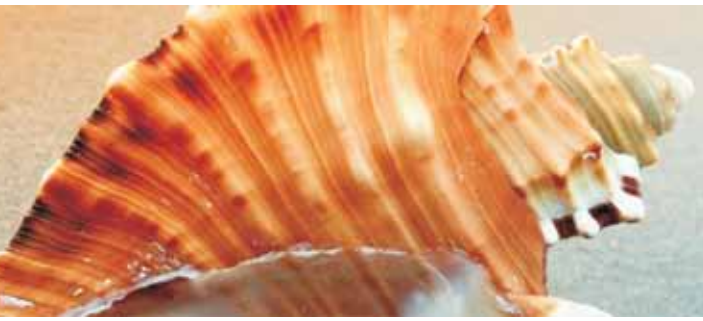
In 2007, Dr Wolters was elected, and in 2011 re-elected, as president of the World Federation of Neurology Association of Parkinsonism and Related Disorders. He is also a member of the International Scientific Committee of the WFN and the CINP, and a corresponding member of the American Neurological Association.

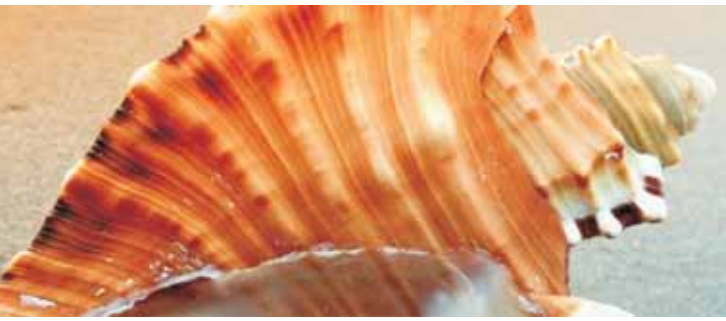
Prof. Wolters received the gold medal of the Italian Society of Neurology (1982), the Charles E. Smith award (Jerusalem, 2008), the Hans Lakke award (Amsterdam, 2010) and the Senator Burda award (2010). He is an honorary member of the ESCNP board as well as the Dutch Parkinson Association.

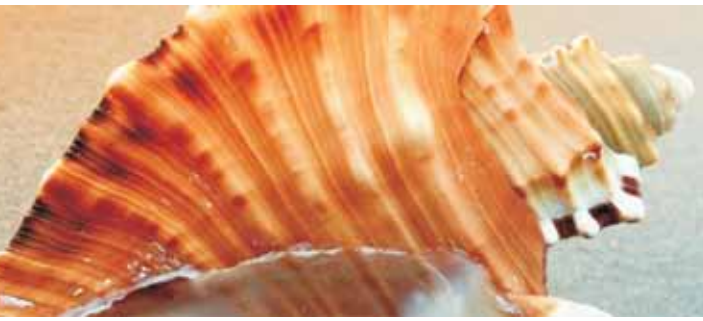
NOTES

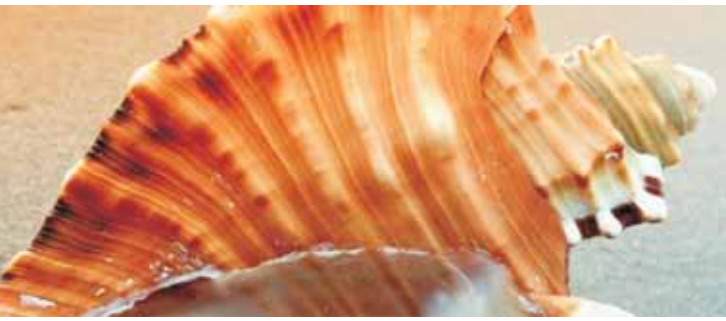


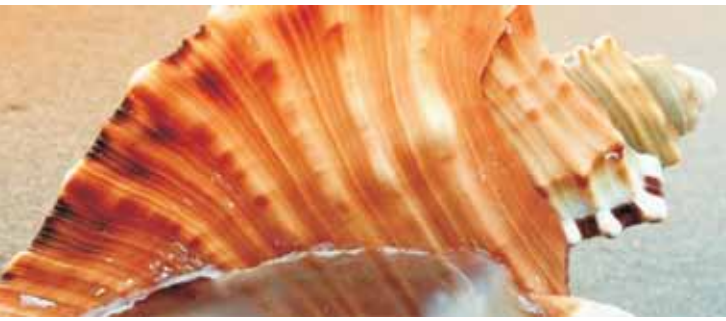


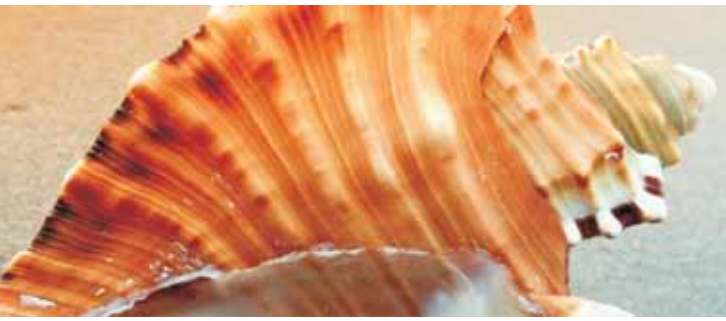










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