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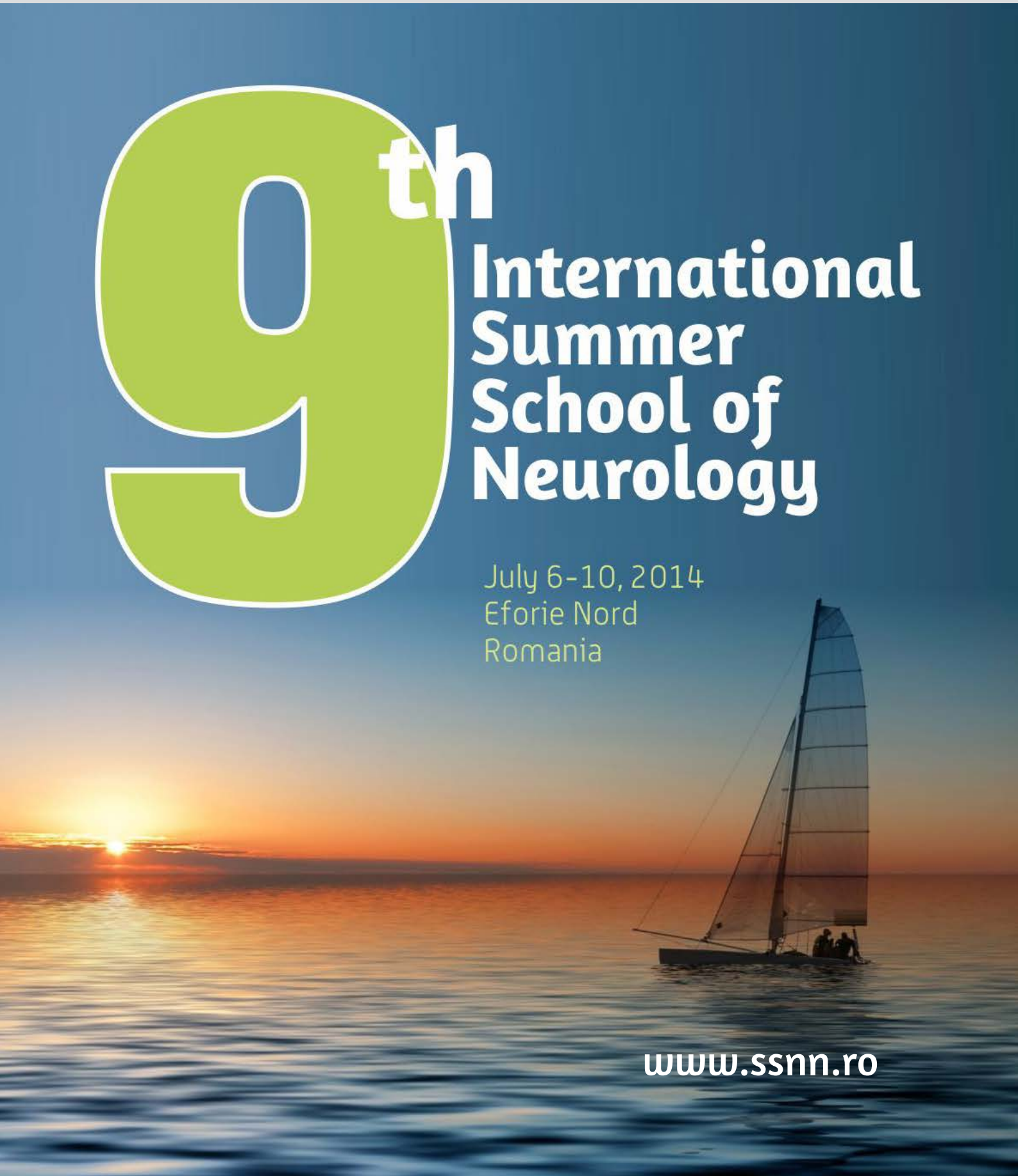
9th International Summer School of Neurology

July 6-10, 2014
Eforie Nord
Romania



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9th INTERNATIONAL SUMMER SCHOOL of NEUROLOGY

July 6-10 | 2014 | Hotel Europa
Eforie Nord | Romania

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Natan M. Bornstein

Professor of Neurology at the Tel-Aviv University
Sackler Faculty of Medicine, Israel

Vice President of the World Stroke Organization (WSO)

Head of Stroke Unit at the Tel-Aviv Medical Center

Chairman of the Israeli Neurological Association

Dafin F. Mureşanu

President of the Romanian Society of Neurology

Professor of Neurology, Chairman Department of Neurosciences
University of Medicine and Pharmacy "Iuliu Hatieganu",
Cluj-Napoca, Romania

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

The Society for the Study of Neuroprotection and Neuroplasticity
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Ovidiu Băjenaru | President

Honorary President of the Romanian Society of Neurology
University of Medicine and Pharmacy "Carol Davila" Bucharest
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Faculty of Medicine,
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Cluj-Napoca, Romania

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Past- President of the Romanian Society of Neurosurgery

Bogdan O. Popescu | Director of Research

Department of Neurology, School of Medicine, 'Carol Davila'
University of Medicine and Pharmacy,
Colentina Clinical Hospital Bucharest, Romania

ORGANIZERS

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

Tel Aviv University
www.tau.ac.il

"Iuliu Haţieganu" University of Medicine and Pharmacy Cluj-Napoca, Romania
www.umfcluj.ro

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General Information

ANA Hotels – Eforie Nord
Europa Hotel

Phone: 0040241 / 741.710, fax:
0040241 / 741.720
Republicii Street no 13, Eforie Nord,
Constanta – Romania

Scientific Secretariat

Society for the Study of
Neuroprotection and Neuroplasticity
37 Mircea Eliade Street, 400364,
Cluj-Napoca, Romania
Office phone: +40745255311
E-mail:office@ssnn.ro

Contact Details

Mrs. Doria Constantinescu,
mobile: +40757096111

Mrs. Diana Biris,
mobile: +40755080820

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 dinners.

FINAL PROGRAM & ABSTRACT BOOK

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

COFFEE BREAKS

Coffee, tea and mineral water are served during morning coffee breaks and are 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, 50 Hz. Two-prong plugs are standard.

TIME

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

SCIENTIFIC PROGRAM

SCIENTIFIC PROGRAM

Sunday, July 6th, 2014

ADVANCES IN NEUROFUNDAMENTALS
Module coordinators: Laurențiu M. Popescu (Romania); Hari Shanker Sharma (Sweden); Stephen Skaper (Italy)

18:00 – 18:30	Hari Shanker Sharma (Sweden)	TiO2-Nanowired Delivery of neurotrophic factors Enhances Neuroprotection in Traumatic and Concussive Brain Injuries
18:30 – 19:00	Stephen Skaper (Italy)	The Endoneurial Microenvironment: Anatomy, Pathophysiology and Therapeutic Target
19:00 – 19:30	Volker Hömberg (Germany)	Practical Pharmacology in Neurorehabilitation
20:00	Dinner	

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Monday, July 7th, 2014

08:45 – 09:00	Welcome Address:	Dafin F. Mureşanu (Romania); Volker Hömberg (Germany); Heinrich Binder (Austria); Ioana Ispas (Romania); Laurenţiu M. Popescu (Romania)

NEUROREHABILITATION; PERIPHERAL NEUROPATHY

Module coordinators: Volker Hömberg (Germany); Heinrich Binder (Austria)

09:00 – 09:30	Volker Hömberg (Germany)	Motor Neurorehab - State of the Art
09:30 – 10:00	Heinrich Binder (Austria)	Treatment of Low Back Pain. Do We Always Take Appropriate Decision? What You Need to Know about Anatomy and Pathophysiology
10:00 – 10:30	Dana Boering (Germany)	Nomenclature and Assessments in Diminished States of Consciousness
10:30 – 11:00	Coffee Break	
11:00 – 11:30	Vitalie Lisnic (Moldova)	Multidimensional Evaluation of the Patient with Polyneuropathy
11:30 – 12:00	Tudor Lupescu (Romania)	Diabetic Neuropathy – Diagnosis and Symptomatic Treatment
12:00 – 12:30	Doina Catrinoiu (Romania)	Diabetic Neuropathy – Pathogenetic Treatment
13:00	Lunch	
18:00 – 20:00	Volker Hömberg (Germany)	Basic Course in Neurologic Clinical Examination
20:00	Dinner	

Tuesday, July 8th, 2014

STROKE
Module coordinators: Natan Bornstein (Israel); Dafin F. Mureşanu (Romania)

09:00 – 09:45	Natan Bornstein (Israel)	Time is Brain, TIA as an Emergency
09:45 – 10:30	Natan Bornstein (Israel)	The heart's effect on the brain. Atrial fibrillation and stroke prevention-update
10:30 – 11:15	Natan Bornstein (Israel)	Management of Symptomatic Carotid Stenosis - CEA vs. Stent
11:15 – 11:45	Coffee Break	
11:45 – 12:30	Dafin F. Mureşanu (Romania)	Advances in Brain Protection and Recovery in Stroke Therapy
12:30 – 13:15	Dafin F. Mureşanu (Romania)	The Impact of Neurorehabilitation in Stroke Recovery
13:15 – 14:00	László Csiba (Hungary)	Secondary Stroke Prevention, 2014 (new and modified recommendations of AHA/ASA guideline)
14:00	Lunch	
18:00 – 18:30	Anca Buzoianu (Romania)	A Clinical-Genetic Algorithm for Calculating the Stable Therapeutic Dose of Acenocoumarol
18:30 – 20:00	Case presentations | Stroke	
20:00	Dinner	

Wednesday, July 9th, 2014

PARKINSON'S DISEASE
Module coordinators: Amos Korczyn (Israel); Ovidiu Băjenaru (Romania)

09:00 – 09:30	Amos Korczyn (Israel)	Personalized medicine: Disease modification in Parkinson's Disease
09:30 – 10:00	Ovidiu Băjenaru (Romania)	Etiopathogeny of PD
10:00 – 10:30	Cristina Panea (Romania)	Recommendations for Clinical Diagnosis and Standardized Assessment in Parkinson`s Disease
10:30 – 11:00	Cristian Falup-Pecurariu (Romania)	Non motor symptoms in Parkinson's disease
11:00 – 11:30	Coffee Break	
11:30 – 12:00	Bogdan O. Popescu (Romania)	Dopamine agonist treatment in advanced Parkinson's disease
12:00 – 12:30	Mihaela Simu (Romania)	The Concept Of Integrated Multidisciplinary Approach In Parkinson's Disease Management
12:30 – 13:00	Dafin F. Mureşanu (Romania)	Treatment impact on quality of life in advanced Parkinson's disease patients
13:00 – 13:30	József Szász (Romania)	Management of Advanced Parkinson's Disease
14:00	Lunch	
20:00	Gala Dinner	

Thursday, July 10th, 2014

MULTIPLE SCLEROSIS

Module coordinators: Ovidiu Băjenaru (Romania); Dafin F. Mureşanu (Romania)

09:00 - 09:30	Ioana Ispas (Romania)	European funding opportunities in area of neurosciences – from Horizon 2020 to Joint Programming Initiatives
09:30 – 10:00	Ovidiu Băjenaru (Romania)	Therapeutic Strategies In Relapsing-Remitting Multiple Sclerosis
10:00 – 10:30	Mihaela Simu (Romania)	Patients' Management in MS Treatment
10:30 – 11:00	Coffee Break	
11:00 – 11:30	Cristina Tiu (Romania)	Imagistic Aspects in Multiple Sclerosis
11:30 – 12:00	Cristina Panea (Romania)	Cognition and fatigue in MS: assessment and therapeutic options
12:00 – 12:30	Dafin F. Mureşanu (Romania)	Managing Mobility in MS Patients
12:30 – 13:30	Lunch	
14:00 – 15:00	Final Examination	
15:00	Official closing	
20:00	Farewell Dinner	

ABSTRACTS

THERAPEUTIC STRATEGIES IN RELAPSING-REMITTING MULTIPLE SCLEROSIS

OVIDIU BĂJENARU

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

Director of the Department of Neurology, Neurosurgery and Psychiatry
Chairman and Head of Dept. Neurology - University Emergency Hospital, Bucharest, Romania

Multiple sclerosis is a chronic inflammatory demyelinating disease of the central nervous system. Within the last decades considerable success has been made in a deep understanding of this disease. The development of novel strategies is based on the pathophysiological cascade of multiple sclerosis and on the current understanding of the autoimmunity and T cell activation and regulation, respectively.

The inflammation and oxidative stress are central to the pathophysiology of multiple sclerosis and they are associated with significant damage to the blood brain barrier myelin and axon damage, resulting in clinical symptoms. Beyond inflammation, the studies suggest that oxidative stress is linked to neurodegeneration. It is reasonable to pursue oxidative stress as a therapeutic target in MS by enhancing intrinsic antioxidant pathways.

This has resulted in a significant progress for the treatment of MS and several agents have been approved, especially for the treatment of relapsing-remitting MS.
These treatments have demonstrated both clinical and MRI efficacy in the course of the disease as well as a slow of disability progression.
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ETIOPATHOGENY OF PD

OVIDIU BĂJENARU

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

Director of the Department of Neurology, Neurosurgery and Psychiatry
Chairman and Head of Dept. Neurology - University Emergency Hospital, Bucharest, Romania

Parkinson's disease is a progressive multifocal neurodegenerative disease, characterized by mutiple synaptic systems impairment, both dopaminergic (not only the nigro-striatal circuit) and non-dopaminergic: Ach, NA, 5-HT, others. The lesions are localized in CNS (including RETINA) and the peripheral autonomic nervous system. To date, mutations in several genes have been identified to cause predominantly early-onset parkinsonism, which typically follows mendelian inheritance, but familial forms of PD represent just up to 15% of all patients with PD. The majority of these patients is represented by PD without obvious familial aggregation ("idiopathic PD"), whose etiology is likely governed by a variety of genetic and non-genetic factors that define an individual's risk to develop the disorder, as well as onset age, clinical presentation and progression. The major breakthrough in recent years in PD research has been the mapping of 18 genetic loci, named PARK1–18, and the subsequent cloning of several genes involved in familial PD. The most studied genes code for: alpha-synuclein (SNCA), ubiquitin carboxy-terminal hydrolase L1 (UCH-L1), parkin (PRKN), leucine-rich repeat kinase 2 (LRRK2 or dardarin), PTEN-induced putative kinase 1 (PINK1), DJ-1 and ATP13A2. Extensive mutation screening of these causal genes revealed both simple mutations (missense, nonsense, silent, splice site, and untranslated region (UTR) mutations, small insertions and deletions (indels), and copy number variations (genomic rearrangements) leading to PD.

To date, molecular genetic analyses have identified over 500 distinct DNA variants in five disease genes associated with familial Parkinson disease. The knowledge acquired of the functions of their protein products has revealed pathways of neurodegeneration that may be shared between inherited and sporadic PD.

The main primary genetic lesions lead to abnormalities in the ubiquitin-proteasome system function, or to mitochondrial respiratory chain abnormalities or to abnormalities in the mithocondrial life cycle (related to mitochondrial fusion, fission, biogenesis and degradation); no matter what is the first step in the pathogenetic chain of events, the initial abnormality leads during the next steps to abnormal functions in the other pathogenetic pathways. Mutations or altered expression of the proteins encoded by the recessive PD genes contributes to PD pathogenesis through common mechanisms that result in mitochondrial impairment, oxidative stress, and protein mishandling implying cell dysfunction or increased vulnerability to neurodegeneration.

Genetic variation in genes encoding proteins along several pathways, such as the UPS, the mitochondrial respiratory chain or alpha-synuclein handling, would predispose individuals to low levels of chronic, relative dysfunction of these molecular processes. There is growing evidence from genetic association studies that genetic variation in such

genes may contribute as susceptibility factors in sporadic PD. Common genetic variation plays an important part in the cause of Parkinson's disease. Genome-wide association studies (GWAS) have emerged as a powerful approach to identify susceptibility loci. These studies, involving testing of genetic polymorphisms in large series of cases versus controls, are able to identify low-penetrance alleles that cannot be detected by linkage studies. Environmental factors may have also an important role in PD etiopathogenesis. Several epidemiological studies have shown that exposure to pesticides, in particular the widely used organochlorines and the bipyridyl herbicide paraquat, is associated with increased risk of developing PD. Emerging evidence indicates that epigenetic changes, protein aggregation and autophagy are important cellular and molecular correlates of neurodegenerative diseases resulting from chronic neurotoxic chemical exposure.

The current belief is that dopaminergic neuronal degeneration in PD is likely to result from a combination of multiple interlinking signaling pathways rather than from a single unifying mechanism. The etiopathogenesis of sporadic PD, the most common form of parkinsonism, is complex with variable contributions of genetic susceptibility and environmental factors. Cumulative 'normal' stress over a long period of time, such as the ageing process (which is known to be associated with reduction in the UPS function and increased levels of oxidative stress) or exposure to toxins, could then tip the balance from a genetically determined stressed cellular state to programmed cell death in that particular individual. This explanation would unite all the molecular pathways implicated in Mendelian forms of PD into a complex multifactorial model for sporadic PD.

TREATMENT OF LOW BACK PAIN. DO WE ALWAYS TAKE APPROPRIATE DECISION? WHAT YOU NEED TO KNOW ABOUT ANATOMY AND PATHOPHYSIOLOGY

HEINRICH BINDER

Landsteiner Institute for Neurorehabilitation and Space Medicine
Vienna, Austria

High prevalence of back pain and bearing on individual life quality as well as its adverse effect on costs is a matter of common knowledge. Its talked a lot about all risks whatsoever and how to prevent and treat particularly concerning avoidance of chronification. Two subjects are in the centre stage: Whether to operate or not and how to alleviate pain. Looking through the literature many publications concerning management including recommendations for different types of treatment are apparent. Except apparent cases of pathophysiological correlations it is almost always neglected to seek and refer back pain to a clear defined cause or to discuss established alternatives. To be content to do depends on the professional competence and associated thought pattern. Therefore it depends if a neurologist, neurosurgeon, physiatrist, pain therapist or psychiatrist is involved how explicitly will be examined and if an operation among other things is considered. Anyway - each treatment requires proceeding evaluation taking into account all contemplate differential diagnoses. Certainly an interdisciplinary approach is obvious. But it is not possible to split responsibility for appropriate treatment. This basically means that we need a specialists in any discipline who serve as a kind of case manager. It is their task to examine carefully, to make a primary diagnosis, to reflect differential diagnoses and to initiate corresponding confirming or refusing examinations in particular concerning spinal column. A prerequisite, however, is knowledge of all possible causative pathophysiologic processes and careful examination of spinal column with related joints, ligaments and muscles, of neurological signs, not at least of viscera and also of mental situation. The interpretation of complaints in correlation to functional and morphologic findings at the musculoskeletal system issues a particular challenge. To make a clear diagnosis protects the patient from unnecessary operation and feared failed back surgery syndrome.

NOMENCLATURE AND ASSESSMENTS IN DIMINISHED STATES OF CONSCIOUSNESS

DANA BOERING

St. Mauritius Therapieklinik
Meerbusch, Germany

Over the last two decades there was a capacious development of consciousness science, from the implementation and international acceptance of standardized neurobehavioral assessment tools of disorders of consciousness, especially the Coma Recovery Scale Revised, which uncovered a high rate of misdiagnosis, to sophisticated ancillary techniques as brain imaging and electrophysiological examinations. These enhanced our scientific understanding of recovery of consciousness in the human brain following severe brain damage and demonstrated that patients with little or no behavioral evidence of conscious awareness may retain critical cognitive capacities and harbor latent potential for further recovery.

This raises questions regarding the phenomenon of "minimal" consciousness: When is minimal consciousness enough to call a patient conscious? What is the moment of no consciousness and how can we objectively measure this in another human being? This problem is emphasized in the renaming of the vegetative state into unresponsive wakefulness syndrome, reminding physicians to remain careful when making inferences regarding consciousness based on behavioral assessment. Furthermore there is a need to give a name to those patients who are behaviorally unresponsive but follow commands like motor imagery tasks as demonstrated by recent brain imaging paradigms.

The talk will encompass an overview of actual behavioral and ancillary assessment methods available for scientific use and everyday work in early neurorehabilitation, their respective pros and cons as well as the challenge all of them represent in our effort to deal with DSC patients and enhance their recovery and concluding that in the recent years it has become ever clearer that the separate sub conditions: coma, unresponsive wakefulness syndrome, minimally conscious state fit into the percept of gradually recuperating consciousness.

MANAGEMENT OF SYMPTOMATIC CAROTID STENOSIS CEA VS. STENT

NATAN BORNSTEIN

Tel-Aviv University
Sackler Faculty of
Medicine, Israel

Head of
Stroke Unit at the
Tel-Aviv
Medical Center
Israel

Symptomatic severe carotid stenosis (>70%) carries a high risk of subsequent stroke of about ~ 30% over 2 years.
Carotid endarterectomy (CEA) was proved to reduce the risk of stroke significantly, with Relative Risk Reduction (RRR) = 65% and Number Needed to Treat (NNT) = 6 if performed safely (perioperative
S&D =5.8%) and should be executed within 2 weeks of TIA or minor stroke (NASCET & ECST).

For carotid stenting to replace CEA we need to know the comparative safety, durability and efficacy of the procedure. Only a few randomized, controlled studies comparing CEA and stenting were conducted (CAVATAS, SAPPHIRE, EVA-3 and SPACE) with inconclusive results. There are still several ongoing studies (CREST in the USA and ICSS in Europe and Australia). Until more data will be available carotid stenting should be performed only in a selected group of patients with specific indications like: re-stenosis of the CEA, post neck radiation, inaccessible lesion for CEA and contra-indications for CEA.

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 PREVENTION-UPDATE

NATAN BORNSTEIN

Tel-Aviv University
Sackler Faculty of Medicine, Israel

Head of
Stroke Unit at the Tel-Aviv Medical Center
Israel

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.

Three novel anticoagulants (NOACs)-dabigatran etexilate, rivaroxaban, apixaban- have been approved in many countries for stroke prevention in atrail fibrillation, because they are associated with the same or lower rates of stroke, bleeding (particularly intracranially) and death compared with warfarin; and unlike warfarin, they can be given in fixed doses without routine coagulation monitoring. The effects of NOACs compared with warfarin are consistent in almost all populations and patients subgroups studied.

The lack of antidote to the NOACs in patients who experience major bleeding has not yet been associated with worse outcome among patients treated with NOACs compared with warfarin in secondary analysis. Multiple guidelines for the management of AF now recommend the NOACs for stroke prevention among atrial fibrillation (AF) patients at risk for stroke.

A CLINICAL-GENETIC ALGORITHM FOR CALCULATING THE STABLE THERAPEUTIC DOSE OF ACENOCOUMAROL

ANCA BUZOIANU[1]
Ştefan Cristian Vesa[1]
Adrian Pavel Trifa[2]
Sorin Crişan[3]

1 Department of Pharmacology, Toxicology and Clinical Pharmacology, "Iuliu Haţieganu" University of Medicine and Pharmacy Cluj-Napoca, Romania

2 Department of Medical Genetics, "Iuliu Haţieganu" University of Medicine and Pharmacy Cluj-Napoca, Romania

3 5th Department of Internal Medicine, Municipal Hospital of Cluj-Napoca, "Iuliu Haţieganu" University of Medicine and Pharmacy Cluj-Napoca, Romania

Aim: To develop and validate an algorithm for calculating the stable dose of acenocoumarol in patients diagnosed with acute deep vein thrombosis, atrial fibrillation or valvular prostheses.

Material and methods: The study included 301 patients that necessitated treatment with acenocoumarol for a prolonged time (> three months). The patients were selected from those admitted within the internal medicine, geriatric and cardiology wards of Municipal Hospital of Cluj-Napoca and the Heart Institute "Niculae Stănciou" in Cluj-Napoca, Romania, between October 2009 and December 2011. For each patient we recorded demographic, clinical and pharmacological data that could have influenced the stabile dose of acenocoumarol. The genetic analysis included genotyping the CYP2C9 gene and the VKORC1 gene. Through randomization, patients were included in the algorithm group (200 (66.4%) patients) and in the validation groups (101 (33.6%) patients).

Results: The age and body mass index were responsible for 18.8% (R2 coefficient) of the acenocoumarol weekly dose variability in patients within the algorithm group. After the inclusion of CYP2C9 and VKORC1 mutations, the R2 coefficient increased at 43.1%. For the algorithm group we calculated a mean error of -0.6 (±6.4) mg/week and a mean absolute error of 5 mg/week (0.71 mg /day). In the validation group, the clinical parameters explained 22.2% of the acenocoumarol weekly dose variability, and, after adding the genetic factors, the R2 coefficient increased at 32.8%.

Conclusion: We created and validated an adequate algorithm for the prediction of acenocoumarol therapeutic stable dose.

Key words: acenocoumarol, algorithm, CYP2C9, VKORC1

DIABETIC NEUROPATHY – PATHOGENETIC TREATMENT

DOINA CATRINOIU

Constanta Medical University and Pharmacy, Romania

Causal treatment combined with symptomatic agents appears to be the optimal treatment in Diabetic Neuropathy, however advanced neuropathy justifies two causal treatments concomitantly. Purely symptomatic therapy may lead to the subclinical progression of the neuropathic damage.

Benfotiamine and Alpha Lipoic Acid are considered causal treatments, with a documented analgesic action in addition. The two causal agents interfere through different mechanisms in the pathogenesis of Diabetic Neuropathy.

Benfotiamine is a lipid-soluble thiamine precursor having much higher bioavailability than genuine thiamine. Growing body of evidence revealed that benfotiamine alleviates the severity of diabetic complications such as neuropathy, nephropathy and retinopathy by inhibiting the formation of advanced glycation end products (AGEs). Benfotiamine prevents the progression of diabetic complications by increasing tissue levels of thiamine diphosphate, which enhances the transketolase activity that directs the precursors of AGEs to pentose phosphate pathway, resulting in the reduction of tissue levels of AGEs.

ALA is an ideal antioxidant which acts as a scavenger for reactive oxygen species, regenerator for other antioxidants (vitamin C, glutathione, and alpha-tocopherol) and chelator of free metal ions.

ALA is both water and fat soluble and therefore cross biological membranes easily, thus reaching all the compartments of the cell. By neutralizing mitochondrial ROS, alternative pathways of glucose metabolism are being blocked with an early intervention in the chain responsible for complications of Diabetes mellitus.

Beneficial effects are achieved with low micromolar levels of ALA, suggesting that some of its therapeutic potential extends beyond the strict definition of an antioxidant.

SECONDARY STROKE PREVENTION, 2014 (NEW AND MODIFIED RECOMMENDATIONS OF AHA/ASA GUIDELINE)

LÁSZLÓ CSIBA

Department of Neurology, Debrecen University, Hungary

Hypertension
Initiation of BP therapy is indicated for previously untreated patients with ischemic stroke or TIA who, after the first several days, have an established BP ≥140 mm Hg systolic or ≥90 mm Hg diastolic (Class I; Level of Evidence B). Initiation of therapy for patients with BP <140 mm Hg systolic and <90 mm Hg diastolic is of uncertain benefit (Class IIb; Level of Evidence C). Resumption of BP therapy is indicated for previously treated patients with known hypertension for both prevention of recurrent stroke and prevention of other vascular events in those who have had an ischemic stroke or TIA and are beyond the first several days (Class I; Level of Evidence A). Goals for target BP level or reduction from pretreatment baseline are uncertain and should be individualized, but it is reasonable to achieve a systolic pressure <140 mm Hg and a diastolic pressure <90 mm Hg (Class IIa; Level of Evidence B). For patients with a recent lacunar stroke, it might be reasonable to target a systolic BP of <130 mm Hg (Class IIb; Level of Evidence B).

Dyslipidemia
Statin therapy with intensive lipidlowering effects is recommended to reduce risk of stroke and cardiovascular events among patients with ischemic stroke or TIA presumed to be of atherosclerotic origin and an LDL-C level ≥100 mg/dL with or without evidence for other ASCVD (Class I; Level of Evidence B).Statin therapy with intensive lipidlowering effects is recommended to reduce risk of stroke and cardiovascular events among patients with ischemic stroke or TIA presumed to be of atherosclerotic origin, an LDL-C level <100 mg/dL, and no evidence for other clinical ASCVD (Class I; Level of Evidence C).Patients with ischemic stroke or TIA and other comorbid ASCVD should be otherwise managed according to the ACC/AHA 2013 guidelines, which include lifestyle modification, dietary recommendations, and medication recommendations (Class I; Level of Evidence A).

Glucose disorders
After a TIA or ischemic stroke, all patients should probably be screened for DM with testing of fasting plasma glucose, HbA1c, or an oral glucose tolerance test. Choice of test and timing should be guided by clinical judgment and recognition that acute illness may temporarily perturb measures of plasma glucose. In general, HbA1c may be more accurate than other screening tests in the immediate postevent period (Class IIa; Level of Evidence C).

Nutrition
Routine supplementation with a single vitamin or combination of vitamins is not recommended (Class III; Level of Evidence A). It is reasonable to recommend that

patients with a history of stroke or TIA reduce their sodium intake to less than ≈2.4 g/d. Further reduction to <1.5 g/d is also reasonable and is associated with even greater BP reduction (Class IIa; Level of Evidence C). It is reasonable to counsel patients with a history of stroke or TIA to follow a Mediterranean-type diet instead of a lowfat diet. The Mediterranean-type diet emphasizes vegetables, fruits, and whole grains and includes low-fat dairy products, poultry, fish, legumes, olive oil, and nuts. It limits intake of sweets and red meats (Class IIa; Level of Evidence C).

Sleep apnea

A sleep study might be considered for patients with an ischemic stroke or TIA on the basis of the very high prevalence of sleep apnea in this population and the strength of the evidence that the treatment of sleep apnea improves outcomes in the general population (Class IIb; Level of Evidence B). Treatment with continuous positive airway pressure might be considered for patients with ischemic stroke or TIA and sleep apnea given the emerging evidence in support of improved outcomes (Class IIb; Level of Evidence B).

Carotid disease

CAS is indicated as an alternative to CEA for symptomatic patients at average or low risk of complications associated with endovascular intervention when the diameter of the lumen of the internal carotid artery is reduced by >70% by noninvasive imaging or >50% by catheterbased imaging or noninvasive imaging with corroboration and the anticipated rate of periprocedural stroke or death is <6% (Class IIa; Level of Evidence B). It is reasonable to consider patient age in choosing between CAS and CEA. For older patients (ie, older than ≈70 years), CEA may be associated with improved outcome compared with CAS, particularly when arterial anatomy is unfavorable for endovascular intervention. For younger patients, CAS is equivalent to CEA in terms of risk for periprocedural complication (ie, stroke, MI, or death) and long-term risk for ipsilateral stroke (Class IIa; Level of Evidence B). CAS and CEA in the above settings should be performed by operators with established periprocedural stroke and mortality rates of <6% for symptomatic patients, similar to that observed in trials comparing CEA to medical therapy and more recent observational studies (Class I; Level of Evidence B). Routine, long term follow-up imaging of the extracranial carotid circulation with carotid duplex ultrasonography is not recommended (Class III; Level of Evidence B). For patients with recurrent or progressive ischemic symptoms ipsilateral to a stenosis or occlusion of a distal (surgically inaccessible) carotid artery, or occlusion of a midcervical carotid artery after institution of optimal medical therapy, the usefulness of EC/IC bypass is considered investigational (Class IIb; Level of Evidence C).

Intracranial atherosclerosis

For patients with recent stroke or TIA (within 30 days) attributable to severe stenosis (70%–99%) of a major intracranial artery, the addition of clopidogrel 75 mg/d to aspirin for 90 days might be reasonable (Class IIb; Level of Evidence B). For patients with stroke or TIA attributable to 50% to 99% stenosis of a major intracranial artery, the data are insufficient to make a recommendation regarding the usefulness of clopidogrel alone, the combination of aspirin and dipyridamole, or cilostazol alone (Class IIb; Level of Evidence C). For patients with a stroke or TIA attributable to 50% to 99% stenosis of a major intracranial artery, maintenance of systolic BP below 140 mm Hg and high intensity statin therapy are recommended (Class I; Level of Evidence B). For patients with a stroke or TIA attributable to moderate stenosis (50%– 69%) of a major intracranial artery, angioplasty or stenting is not recommended given the low rate of stroke on medical management and the inherent periprocedural risk of endovascular treatment (Class III; Level of Evidence B). For patients with stroke or TIA attributable to severe stenosis (70%–99%) of a major intracranial artery, stenting with the Wingspan stent system is not recommended as an initial treatment, even for patients who were taking an antithrombotic agent at the time of the stroke or TIA (Class III; Level of Evidence B). For patients with stroke or TIA attributable to severe stenosis (70%–99%) of a major intracranial artery, the usefulness of angioplasty alone or placement of stents other than the Wingspan stent is unknown and is considered investigational (Class IIb; Level of Evidence C). For patients with severe stenosis (70%– 99%) of a major intracranial artery and recurrent TIA or stroke after institution of aspirin and clopidogrel therapy, achievement of systolic BP <140 mm Hg, and high-intensity statin therapy, the usefulness of angioplasty alone or placement of a Wingspan stent or other stents is unknown and is considered investigational (Class IIb; Level of Evidence C).

AF

For patients who have experienced an acute ischemic stroke or TIA with no other apparent cause, prolonged rhythm monitoring (≈30 days) for AF is reasonable within 6 months of the index event (Class IIa; Level of Evidence C). VKA therapy (Class I; Level of Evidence A), apixaban (Class I; Level of Evidence A), and dabigatran (Class I; Level of Evidence B) are all indicated for the prevention of recurrent stroke in patients with nonvalvular AF, whether paroxysmal or permanent. The selection of an antithrombotic agent should be individualized on the basis of risk factors, cost, tolerability, patient preference, potential for drug interactions, and other clinical characteristics, including renal function and time in INR therapeutic range if the patient has been taking VKA therapy. Rivaroxaban is reasonable for the prevention of recurrent stroke in patients with nonvalvular AF (Class IIa; Level of Evidence B). The combination of oral anticoagulation (ie, warfarin or one of the newer agents) with antiplatelet therapy is not recommended for all patients after ischemic stroke or TIA but is reasonable in patients with clinically apparent CAD, particularly an acute coronary syndrome or stent placement (Class IIb;

Level of Evidence C). For patients with ischemic stroke or TIA and AF who are unable to take oral anticoagulants, aspirin alone is recommended (Class I; Level of Evidence A). The addition of clopidogrel to aspirin therapy, compared with aspirin therapy alone, might be reasonable (Class IIb; Level of Evidence B). For most patients with a stroke or TIA in the setting of AF, it is reasonable to initiate oral anticoagulation within 14 days after the onset of neurological symptoms (Class IIa; Level of Evidence B). In the presence of high risk for hemorrhagic conversion (ie, large infarct, hemorrhagic transformation on initial imaging, uncontrolled hypertension, or hemorrhage tendency), it is reasonable to delay initiation of oral anticoagulation beyond 14 days (Class IIa; Level of Evidence B). The usefulness of closure of the left atrial appendage with the WATCHMAN device in patients with ischemic stroke or TIA and AF is uncertain (Class IIb; Level of Evidence B).

MI and thrombus

Treatment with VKA therapy (target INR, 2.5; range, 2.0–3.0) for 3 months may be considered in patients with ischemic stroke or TIA in the setting of acute anterior STEMI without demonstrable left ventricular mural thrombus formation but with anterior apical akinesis or dyskinesis identified by echocardiography or other imaging modality (Class IIb; Level of Evidence C). In patients with ischemic stroke or TIA in the setting of acute MI complicated by left ventricular mural thrombus formation or anterior or apical wall-motion abnormalities with a left ventricular ejection fraction <40% who are intolerant to VKA therapy because of nonhemorrhagic adverse events, treatment with an LMWH, dabigatran, rivaroxaban, or apixaban for 3 months may be considered as an alternative to VKA therapy for prevention of recurrent stroke or TIA (Class IIb; Level of Evidence C)

Cardiomyopathy

In patients with ischemic stroke or TIA in sinus rhythm who have left atrial or left ventricular thrombus demonstrated by echocardiography or another imaging modality, anticoagulant therapy with a VKA is recommended for ≥3 months (Class I; Level of Evidence C). In patients with ischemic stroke or TIA in the setting of a mechanical LVAD, treatment with VKA therapy (target INR, 2.5; range, 2.0–3.0) is reasonable in the absence of major contraindications (eg, active gastrointestinal bleeding) (Class IIa; Level of Evidence C). In patients with ischemic stroke or TIA in sinus rhythm with dilated cardiomyopathy (LV ejection fraction ≤35%), restrictive cardiomyopathy, or a mechanical LVAD who are intolerant to VKA therapy because of nonhemorrhagic adverse events, the effectiveness of treatment with dabigatran, rivaroxaban, or apixaban is uncertain compared with VKA therapy for prevention of recurrent stroke (Class IIb; Level of Evidence C).

1. Stroke. 2014 Jul;45(7):2160-236

PRACTICAL PHARMACOLOGY IN NEUROREHABILITATION

VOLKER HÖMBERG

Dept of Neurology,
Heinrich Heine University
Düsseldorf,
Germany

Beside the use of training techniques and other behavioral interventions neurological rehabilitation can be augmented significantly by the use of pharmacological agents: Beside the necessary pharmacological treatments for risk factors such as hypertension and hyperlipidemia and secondary prevention, drugs can also be used to facilitate brain recovery. On the other hand certain drugs have to be avoided because they are known to impair brain repair mechanism.

This lecture will address the following issues:

1. It is of critical importance to avoid so called "detrimental" drugs defined from animal experimental as well as from clinical catamnestic studies to interfere with brain plasticity.In contrast amphetamines L-dopa ,reboxetin and antidepressants may facilitate the effect of rehabilitative techniques.
2. A survey of the current status of Drugs to alleviate states of diminished consciousness wiil be given
3. The use of particular drugs will be discussed which can be used for neuro-protection and brain repair. The concept of monomodal vs. multimodal action will be discussed.
4. Most recent data from a multicentre trial on the use of neurotrophic factors in postacute stroke will be presented (CARS trial)

BASIC COURSE IN NEUROLOGIC CLINICAL EXAMINATION

VOLKER HÖMBERG[1]
Heinrich Heine[2]

1. Dept of Neurology,
Heinrich Heine University
Düsseldorf,
Germany

2. University of Duesseldorf
SRH Health Center Bad Wimpfen
Germany

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.

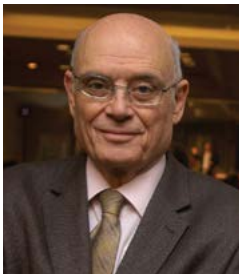
Clinical skills for optimal examination of cranial nerves, motor and sensory functions and screening approaches for cognitive and linguistic analysis will be presented .So the students will soon learn that neurologic examination is much more than just looking at "reflexes"
Also fields notoriously estimated as being difficult (such as eye movements ,nystagmus ,diplopia etc)will not be spared but elucidated in an "easy to understand and remember" mode.

EUROPEAN FUNDING OPPORTUNITIES IN AREA OF NEUROSCIENCES – FROM HORIZON 2020 TO JOINT PROGRAMMING INITIATIVES

IOANA ISPAS

European Affairs Advisor for Bioethics, Genomics and Health International Cooperation Department
Ministry of National Education
Bucharest, Romania

In December 2013 has been launched the European Union Framework Programme Horizon 2020 the largest-ever research and innovation programme, focused on three pillars. Excellent science, Industrial Leadership, Societal Challenges -- all are areas for new ideas and projects that the EU is funding, with many opportunities for neurosciences. The differences between the new programme and its smaller predecessors will be described. The focus for this presentation is calls in Horizon 2020 Health, Demographic Changes and Wellbeing Programme within the area Societal Challenges as well as Widening Participation and Spreading Excellence Programme where a competitive call for Excellence Centers is open. What guidance can the Commission and Romanian Ministry of National Education offer to applicants? In addition, will be briefly mentioned the rules and procedures for applying for the research funding opportunities offered in a coordinated way by Member States through Joint Programming Initiative in the area of neurodegenerative diseases .



PERSONALIZED MEDICINE: DISEASE MODIFICATION IN PARKINSON'S DISEASE

AMOS KORCZYN

The Sieratzki Chair of Neurology, Sackler School of Medicine

Tel-Aviv University, Ramat-Aviv, Israel

The diagnosis of Parkinson disease (PD) is based on clinical criteria, and is needed for useful symptomatic therapy. However, it became quite clear in recent years that the same features can result from different etiopathogenic mechanisms. Thus, it is accepted now that what is called PD is the result of phenotypic convergence. Even pathological diagnosis of PD, based on the demonstration of typical distribution of alpha-cynuclein deposits, is a manifestation of phenotypic convergence at the tissue level.

Since the clinical manifestations of PD can be the result of quite divergent mechanisms, it is unlikely that an intervention can be developed which will be able to influence the development of the disease in all patients. Such disease-modifying therapy should be based not on clinical but rather on understanding the underling pathogenetic processes which differ among cases.

Individualized therapy to interrupt, or at least slow, disease progression must be based on elucidation of the metabolic processes. Some patients may develop PD as a result of mitochondrial damage. Correction of these abnormalities will not affect the progression of the disease among other PD patients, in whom an identical syndrome derives from defects in the proteasome system, etc.

Precision medicine can be used now to identify the underlying pathogenic mechanisms in individual patients, paving the way to the development of real disease modification.

MULTIDIMENSIONAL EVALUATION OF THE PATIENT WITH POLYNEUROPATHY

VITALIE LISNIC

Dept. of Neurology, State University of Medicine and Pharmacy Chisinau, Republic of Moldova

Symptoms of a peripheral neuropathy are common neurologic complaints. Peripheral neuropathy has an estimated prevalence of 2 to 3% in the general population and a prevalence as high as 8% in people older than 55 years. Polyneuropathy means dysfunction or disease of many or all peripheral nerves. Long lists of causes of peripheral neuropathy make peripheral nerve disease a dry and uninspiring subject. The evaluating neurologist is faced with the knowledge that there are more than 100 potential etiologies of polyneuropathy and approximately one third of cases will remain idiopathic despite appropriate testing. Evaluation for the cause is important because diagnosis of an underlying etiology may allow treatment that prevents progression to disability and poor quality of life. An approach to the evaluation of peripheral neuropathies is valuable as it permits full characterization of the neuropathy after which the lists of possible types and tests becomes much shorter and manageable.

Despite the many similarities it is possible to limit the differential diagnosis of a polyneuropathy by determing the answers to several key questions: temporal course and progression, types of involved fibres, clinical pattern of the polyneuropathy, underlying nerve pathology, family history, associated medical illness or history of occupational or toxic exposure, response to a treatment trial.

Nerve conduction studies and electromyography play key roles in the evaluation of patients with suspected polyneuropathy. Electrodiagnostic studies are helpful in determining the location of peripheral nervous system involvement, the population of nerves affected (motor versus large fiber sensory), the severity and distribution of involvement, the portion of nerve affected (axon versus myelin), and the chronicity and regeneration status. Features suggesting demyelinating neuropathy include weakness without atrophy, a length-independent distribution with a proximal predominant or asymmetric/patchy distribution either clinically or electrodiagnostically, early involvement of proximal segments. Most neuropathies are symmetric and length-dependent and are commonly attributed to metabolic, idiopathic, inherited, or toxic conditions. Hand symptoms begin once leg symptoms have ascended toward the knees. Combined proximal and distal weakness is the hallmark of chronic inflammatory demyelinating polyradiculoneuropathy. Prominent autonomic neuropathy symptoms suggest diabetes mellitus, amyloidosis, or Guillain-Barre´ syndrome. A genetic cause of neuropathy should be considered in patients with a family history of neuropathy, lack of positive sensory symptoms, early age at onset, symmetry, associated skeletal abnormalities, or very slowly progressive course. Nerve biopsy yield is best in an acute/subacute,asymmetric, multifocal, progressive neuropathy. Skin biopsy is the best currently available test forsmall fiber neuropathy.

Distal symmetric polyneuropathy (DSP) is the most common type of neuropathy, accounting for the majority of cases. Many underlying disorders cause or are associated with DSP, with diabetes leading the list. The fasting blood glucose test, serum protein electrophoresis and the B12 test have the highest yield in the evaluation of DSP. If the fasting blood glucose level is normal, then the glucose tolerance test may be considered, especially for those with a sensory and/or painful neuropathy.

DIABETIC NEUROPATHY – DIAGNOSIS AND SYMPTOMATIC TREATMENT

TUDOR LUPESCU

Head of Neurology Department, "Agrippa Ionescu" Hospital, Bucharest, Romania

Diabetic neuropathy is not only an important medical problem, but also a real social issue, based on its high prevalence worldwide, the potential complications, and the resulting high costs of medical care. Therefore, it is recommended that neurologists are aware of essential knowledge regarding the clinical manifestations and diagnostic approach in diabetic neuropathy.

One of the most important clinical issues in the management of diabetic neuropathy is pain. Pain should be treated, and in the second part of this lecture, guidelines regarding pain management are provided.

ADVANCES IN BRAIN PROTECTION AND RECOVERY IN STROKE THERAPY

DAFIN F. MUREȘANU

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

Nowadays, it is still difficult to find the correct therapeutic approach for brain protection and recovery in stroke, 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.

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

Stroke pathological cascades contain a limited number of pathophysiological processes. It is characterized mainly by excitotoxicity, oxidative stress, inflammation and apoptotic-like processes.

Pathophysiological processes share some common mechanisms with EDA (e.g. excitotoxicity and neurotrophicity together with neuroplasticity have, as a common important driver, the NMDAR activity; inflammation has an important contribution for neuroregeneration, stimulating neuroplasticity, via trophic factors).

Every lesion in the nervous system triggers in the first minute an endogenous neuroprotective reaction. An endogenous repair process, combining neuroplasticity and neurogenesis follow this as a second answer. All these processes are initiated and regulated by endogenous biological molecules.

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.

Neurotrophic factors are produced by different players in the brain tissue and are acting in a pleotropic way against pathological cascades. The same molecules, due to a complex genetically regulated process, are able to induce, immediately after achieving the endogenous neuroprotective effect, neuroplasticity and neurogenesis as well. Therefore, they have also not only pleotropic activity but also multimodal way. Neuroprotection, neuroplasticity and neurogenesis, processes that are apparently independent, with different control, represent in fact sequences of the same process (EDA), regulated by endogenous molecules.

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 stroke therapy.

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.

The 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.

TREATMENT IMPACT ON QUALITY OF LIFE IN ADVANCED PARKINSON'S DISEASE PATIENTS

DAFIN F. MUREȘANU

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

The second most common neurodegenerative disease, Parkinson's disease (PD) is characterized by a progressive and complex neurodegenerative pattern including loss of dopaminergic neurons and the presence of α-synuclein-positive Lewy bodies within the substantia nigra. Its clinical expression consists in motor and non-motor symptoms, with a significant burden on the quality of life (QoL) of both patients and their careers. In advanced stages of PD is difficult to obtain constant plasma levodopa levels because the absorption of oral drug in the proximal small intestine is dependent on gastric emptying, which is highly variable. Continuous administration of levodopa-carbidopa intestinal gel (LCIG) leads to more stable plasma levels and can achieve continuous stimulation of striatal dopaminergic receptors. A set of recent studies pointed out the reduction of "Off" periods, the increase of "On" periods without disabling dyskinesias and statistically significant improvement in non-motor manifestations after LCIG treatment. The impact on QoL, measured by PDQ-39, PDQ-8 and Schwab & England Activities of Daily Living Scale (ADLS) in patients and by Zarit Caregiver Burden Interview (ZCBI) and Caregiver Strain Index (CSI) in caregivers revealed a significant improvement of stress and burden in caregivers correlated with patients' LCIG therapy.

THE IMPACT OF NEUROREHABILITATION IN STROKE RECOVERY

DAFIN F. MUREȘANU

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

In the last decade, we have seen an increase in efforts to establish evidence based parameters for the practice of brain protection and recovery. This effort has been placed in a broader context involving the role of theory in advancing brain protection and recovery science, particularly in relation to specifying the active components and mechanisms of action of interventions.
Rehabilitation is defined as a process through which each disabled person reaches the maximum physical, functional, cognitive and psychosocial recovery possible within the limits of their disability. Endogenous defense activity (EDA) of the nervous system is a the continuous process that simultaneously performs activities of neurotrophicity, neuroprotection, neuroplasticity and neurogenesis. Neuroregeneration (neurorepair) is the morphological outcome of the interactions between these basic neurobiological processes developed in a particular biological individual context. Neurorecovery is the positive outcome producing clinically relevant results, with immediate functional and late structural effects.
Restitution is an intrinsic process involving biochemically and genetically induced events, such as reduction of edema, absorption of heme, and restoration of axonal transport and ionic currents.
Substitution depends on external stimuli, such as practice, that, through learning, drives activity-dependant plasticity.
Compensation is targeted to improve the mismatch between patients' impaired skills and the demand of the patient or the environment.

All basic biological processes can be naturally activated endogenously or exogenously
In order to successfully compete with pathophysiological processes and support recovery, EDA effects must be enhanced by:

- pharmacological
- complex neurophyscological mood and cognitive support
- physical means
- electromagnetic stimulation
- environmental stimulation
- stem cell transplantation
- any demonstrated combinations of these factors capable of improving patient condition after stroke

This presentation will give an overview on early and late neurorehabilitation added value in stroke therapy.

MANAGING MOBILITY IN MS PATIENTS

DAFIN F. MUREȘANU

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

Multiple sclerosis is the most common non-traumatic cause of disability in young adults. Characterized by a wide range of functional impairments, still one of the most disruptive is the mobility impairment, influenced by other various deficits and symptoms associated with the disease. Either in the first years or after 15 years of disease, walking impairments is considered by the patients as the most concerning aspect related to their disease, followed in importance by visual function and thinking/ memory. The data collected by different studies and patients' registries proved that walking impairment hinders patients' ability to perform daily activities, decreases the employment and reduces health-related quality of life.

The therapeutic solutions include both pharmacological and non-pharmacological approach.

As the gait in MS is affected on many aspects, the medication is aimed to reduce spasticity, to increase speed and balance, and consequently the patients' quality of life. In the category of symptomatic treatments addressed to mobility, PR-fampridine is the first indicated specifically for walking impairments in MS adult patients with EDSS between 4.0 and 7.0. Prolonged-release (PR)-fampridine is considered a potassium channels blocker, acting on the pathological mechanism which determine the delay of electrical impulse along MS damaged axons, due to leak of potassium ions within potassium channels. The benefits proved by PR-fampridine in clinical trials have been sustain by real world experience. Patients treated with PR-fampridine showed consistent improvements in walking speed, functional walking capacity, muscle strength and spasticity. PR-fampridine was also associated with statistically significant improvements in a broad range of physical activities and mental health status measured by the individual items and scores of QoL scales (MSIS-29 PHYS and SF-36 MCS), as early as 12 weeks after initiation through 48 weeks of treatment.

One of the most important non-pharmacological intervention is rehabilitation. Even if rehabilitation does not influence directly the progression of disease, many more recent studies seem to prove that it improves daily activities and participation in social activities, and that way quality of life. Its final objective is to improve self-performance and independence through alleviate the burden of symptoms responsible for progressive impairments and handicap.

Rehabilitation is based on a comprehensive approach, considering the patient as whole, with his or her surroundings, relations and history and it is addressing to gait, balance, fatigue, exercise therapy, sensory dysfunction and neuropsychological function.

An optimal exercise program, with both endurance and resistance training, could influence many parameters related to daily activities, functional capacity and balance. Even severe disabled patients could have benefits from physical exercises.

As mobility is one of the most important aspects related to MS, the combination of a proper medication, the patients' active attitude and exercise programs may improve significantly the profile of their disability and therefore their quality of life.

RECOMMENDATIONS FOR CLINICAL DIAGNOSIS AND STANDARDIZED ASSESSMENT IN PARKINSON`S DISEASE

CRISTINA PANEA

Department of Neurology, Elias University Emergency Hospital,

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

Therapeutic options for Parkinson's disease (PD) are no more limited to symptomatic agents. There is growing evidence that individuals with mild to moderate Parkinson's disease can also benefit from neurorehabilitation that targets flexibility, strengthening and cardiovascular conditioning. Early treatment of PD is contingent upon early and accurate diagnosis of the disease, which can be challenging because there are no biomarkers or neuroimaging or other clinical tests available to confirm the diagnosis. PD diagnosis is currently based on the presence or absence of various clinical features and the experience of the treating physician. A definitive diagnosis can be made only after autopsy. Moreover, the signs and symptoms present in early PD can resemble those of a number of other movement disorders, particularly other forms of parkinsonism, such as multiple system atrophy, drug-induced parkinsonism, and vascular parkinsonism, as well as diffuse Lewy body disease and essential tremor. Nevertheless, diagnosis of PD based on clinical features and response to treatment can be achieved with a fairly high level of accuracy, particularly when made by a physician specializing in movement disorders. The presentation covers the recommendations for the clinical diagnosis of PD, the standardized assessment (movements, cognition, sleep, functioning) based on clinical scales applied immediately upon diagnosis and continue throughout the course of the disease – all serving to adequate symptomatic and physical treatment.

Key words: Parkinson`s disease, diagnosis, clinical scales

COGNITION AND FATIGUE IN MS: ASSESSMENT AND THERAPEUTIC OPTIONS

CRISTINA PANEA

Department of Neurology, Elias University Emergency Hospital,

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

Cognitive impairment, fatigue, and depression often interact in a complex way in MS. MS-related cognitive impairment is detected in 43%–70% of patients. Significant cognitive impairment has been found in 20%–30% of patients with CIS and even in 33% of children and adolescents with MS.

Cognitive decline may evolve independent of disease activity and progresses over time. Cognition can be assessed by patient and clinician reported instruments.

MS-related fatigue is related to disturbances in cognitive function, and leads to the perception of impaired general health, mental state, and quality of life. Unlike in healthy people, fatigue in MS typically comes on easily, is worsened by heat, and interferes with physical functioning and the ability to meet responsibilities. Fatigue can be assessed by patient reported instruments.

Assessment of cognition and fatigue can be used as additional measures to evaluate treatment efficacy in the individual patient.

The ENER-G study was conducted to evaluate effects of natalizumab treatment on fatigue and cognition in MS. Patients with relapsing MS treated with natalizumab demonstrated significant improvement on cognitive test scores: Index of Cognitive Efficiency (ICE) and Procedural Reaction Time (PRO), as well as significantly improved fatigue, as measured by changes in patient-reported measures: Visual Analog Scale for Fatigue (VAS-F), Modified Fatigue Impact Scale (MFIS), and Fatigue Severity Scale (FSS), for up to 48 weeks of treatment.

For all patients with MS, particularly those developing cognitive issues, it is important to evaluate the depression and treat accordingly.

NON MOTOR SYMPTOMS IN PARKINSON'S DISEASE

CRISTIAN FALUP-PECURARIU

Faculty of Medicine, Transilvania University, Brasov, Romania

Parkinson's disease (PD) is a neurodegenerative disease that has motor and non-motor symptoms (NMS). More and more recent data shows that NMS in PD is an important topic. These data gives insightes and the possibility to better understand these issues and treat PD patients. NMS in PD are common and along with motor component significanly impair quality of life. The presence of NMS conditions could precipitate or prolonged hospitalization. Some NMS are prezent when the PD is diagnozed and during early stages. Patients are reporting the presence of NMS with many years before the PD is diagnozed. Usually patients has affected some domains. In the advanced stages there are more domains of the NMS affected. Treatment can induce NMS like hallucinations, psychosis, impulse control disorders. NMS of PD are not well recognized in clinical practices and often not reported spontaneously by the patient. The spectrum of NMS involve: neuropsychiatric symptoms, sleep disorders, gastrointestinal, sensory symptoms, autonomic dysfunction, sexual dysfunction, other symptoms like fatigue, diplopia, weight loss. NMS are present in the premotor phases of PD. In the prodromal phase of PD there are some NMS linked to the futher development of PD like autonomic dysfunction, REM sleep behaviour disorders, hyposmia while others are suggestives as predictors: fatigue, apathy, excesive daytime sleepiness. The presence of these symptoms increase the chance for the patient to develop PD over time. There are some tools and instruments for NMS assessment but few are holistic. The number of NMS are increasing towards to stage of the disease. In advanced PD there are NMS diseases-related and drug-related (non fluctuating and fluctuating).

DOPAMINE AGONIST TREATMENT IN ADVANCED PARKINSON'S DISEASE

BOGDAN O. POPESCU

Department of Neurology, University Hospital Bucharest, School of Medicine, 'Carol Davila' University of Medicine and Pharmacy, Bucharest, Romania

Department of Molecular Medicine, 'Victor Babeș' National Institute of Pathology, Bucharest, Romania

Apomorphine is a non-selective dopamine agonist with a short half-life which is administered by pens or pump to advanced Parkinson's disease (PD) patients with motor fluctuations. Pen administration is useful for the fast relief of the 'off' symptomatology and end-of-dose biphasic dyskinesia. The pump continuous infusion is usually used over daytime period, being one therapeutic choice to address the continuous dopaminergic stimulation principle. Numerous clinical studies showed the efficacy of apomorphine in both pen or pump delivery and compared it to other interventions for advanced PD, such as deep brain stimulation. The most frequent side effects associated with long-term treatment with apomorphine are orthostatic hypotension, nausea and fibrotic nodules at the injection sites. However, these side-effects are less frequent with the pump delivery as compared to pen injections. In this paper I will review the most important clinical data regarding apomorphine treatment resulted from randomized clinical trials.

TiO2-NANOWIRED DELIVERY OF NEUROTROPHIC FACTORS ENHANCES NEUROPROETCTION IN TRAUMATIC AND CONCUSSIVE BRAIN INJURIES

HARI SHANKER SHARMA[1]
Aruna Sharma1
Z Ryan Tian2
Ranjana Patnaik3
Stephen Skaper4
Herbert Mössler5
Dafin F. Muresanu6

1 Laboratory of Cerebrovascular Research, Institute of Surgical Sciences, Department of Anesthesiology & Intensive Care, University Hospital, Uppsala University, SE-75185 Uppsala, Sweden,

2 Department of Biochemistry, University of Arkansas System Fayetteville, Fayetteville, AR, USA,

3 Indian Institute of technology, School of Biomedical Engineering, Dept. of Biomaterials, Banaras Hindu University, Varanasi, India

4 Dept. of Pharmaceutical and Pharmacological Sciences, University of Padova, Padova, Italy

5 Eever Neuro Pharma, Oberburgau, Austria

6 Departmetn of Clinical Neurosciences, University Hospital, University of Medicine & Pharmacy, Cluj-Napoca, Romania

Traumatic brain injuries (TBI) or concussive head injury (CHI) is quite prominent in military personnel duirng peacekeeping or combat operations across the World. The pathophysiology of TBI or CHI however is also influenced widely by the ambient temperature range where injury has occurred. Thus, suitable modulation of the therapeutic strategies is needed to contain pathophysiology of brain injuries occurring in either cold or hot environments. In this investigation we present our data on TBI and CHI at thermoneutral ambient temperature range.

Since the pathophysiology of brain injuries are complex, this is unlikely that one single drug could be able to achieve desired neuroprotection over time in various clinical situations accompanied by several other uncontrollable factors such as environmental temperatures, co-morbidity factors like hypertension, diabetes or nanoparticles intoxications. Keeping these views in mind, our laboratory is engaged to find out a suitable multimodal drug e.g., Cerebrolsyin that is a well balanced composition of several neurotrophic factors and active peptide fragments to induce neuroprotection in several animal models fo CNS injury.

In this investigation, we used TBI as well as CHI to evaluate the neuroprotective effects at room temperature. These CNS injuries induce profound edema for-mation and volume swelling in the CNS at 5 h after the insult [1,3]. The microvascular permeability disturbances to protein tracers were prominent in the CNS areas showing neuronal, glial and endothelial cell injuries. Treatment with neurotrophic factors either infused into the left lateral cerebral ventricle 30 min before or 30 min after trauma, or topically applied over the injured brain attenuated brain edema formation, volume swelling and CNS pathology. These neuroprotective effects of neurotrophic factors were dose dependent [4,5]. The microvascular permeability disturbances to protein tracers (e.g., Evans blue albumin and radioiodine) at 5 h were also considerably reduced with high dose of the drug. On the other hand, no reduction in brain edema, BBB permeability or brain pathology was seen when was administered 60 min post-trauma.

Interestingly, TiO2 nanowired neurotrophic factors when delivered 60 to 90 min after TBI or CHI, significant neuroprotection was achieved in these models. These novel observa-tions suggest that neurotrophic factors administered into the CSF or topically over the trau-matized brain in high doses during early phase of CNS injury has pronounced neuropro-tective effects. Furthermore nano-drug delivery could induce marked neuroprotection even applied longer-time intervals after the primary insults following brain trauma.

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2. Sharma HS, Johanson CE. Intracerebroventricularly Administered Neurotrophins Attenuate Blood Cerebrospinal Fluid Barrier Breakdown and Brain Pathology following Whole-Body Hyperthermia: An Experimental Study in the Rat Using Biochemical and Morphological Approaches. Ann N Y Acad Sci. 2007 Dec;1122:112-29.
3. Menon PK, Muresanu DF, Sharma A, Mössler H, Sharma HS. Cerebrolysin, a mixture of neurotrophic factors induces marked neuroprotection in spinal cord injury following intoxication of engineered nanoparticles from metals. CNS Neurol Disord Drug Targets. 2012 Feb;11(1):40-9. Review.
4. Sharma HS, Muresanu DF, Patnaik R, Stan AD, Vacaras V, Perju-Dumbrav L, Alexandru B, Buzoianu A, Opincariu I, Menon PK, Sharma A. Superior neuroprotective effects of cerebrolysin in heat stroke following chronic intoxication of Cu or Ag engineered nanoparticles. A comparative study with other neuroprotective agents using biochemical and morphological approaches in the rat. J Nanosci Nanotechnol. 2011 Sep;11(9):7549-69.
5. Sharma A, Muresanu DF, Sharma HS. Superior neuroprotective effects of cerebrolysin in nanoparticle-induced exacerbation of hyperthermia-induced brain pathology. CNS Neurol Disord Drug Targets. 2012 Feb;11(1):7-25. Review

THE CONCEPT OF INTEGRATED MULTIDISCIPLINARY APPROACH IN PARKINSON'S DISEASE MANAGEMENT

MIHAELA SIMU

University of Medicine and Pharmacy "Victor Babes" Timisoara; Head of Dept. of Neurology II, Clinical Emergency County Hospital Timisoara, Romania

Parkinson' s disease management is becoming more and more challenging in terms of optimizing the older, newer and emerging therapeutical options. As the ultimate goal of any therapy patient's side view is an improvement in his/hers health related quality of life, it is mandatory to centrally place the patient and expectations in the core of any management plan . A Parkinson's disease patient may be in an early , moderate or advanced clinical stage of the disease subjected to various therapeutical regimens. Therapy targets primarily the motor symptoms (as the core of the disease), nevertheless said symptoms are wrapped in layers of nonmotor symptoms and comorbidities that both complicate and individualize each case. Therefore a change in the paradigm of patient care from segregated multidisciplinary to integrated multidisciplinary approach has emerged as a far realistic and efficient option. We will discuss these concepts and provide as an example the multidisciplinary approach implemented in the Romanian centers for patients in advanced stages of the disease treated with the Levodopa/ Carbidopa intrajejunal infusion pump.

PATIENTS' MANAGEMENT IN MS TREATMENT

MIHAELA SIMU

University of Medicine and Pharmacy "Victor Babes" Timisoara; Head of Dept. of Neurology II, Clinical Emergency County Hospital Timisoara, Romania

In MS treatment decision, there are different aspects that should be considered in relation with patients' needs and concerns. In this sense, the communication between physician and patient plays a key role for a clear understanding of the efficacy and risks related to his or her treatment. A patient's high adherence to the therapy on all aspects of its monitoring, means at the end the optimal outcomes in terms of efficacy and safety.

With highest efficacy profile, natalizumab and alemtuzumab are infused therapy, recommended for active forms of RRMS.

On natalizumab treatment, the risk of hypersensitivity should be considered in first infusions, although the main concern remains the risk of PML. For supporting the individual evaluation for each MS patient of benefits-risk, it has been developed a risk stratification tool, based on algorithm which takes into consideration three factors, respectively the Anti-JCV Abs presence and titer, prior immunosuppressive therapy and natalizumab treatment duration. Yearly MRI provides a reference scan for suspicion of PML. Patients and caregivers should be advices to notify HCP of any worsening in function or change in behavior/cognition, as any infection or disease progression. Pharmacokinetics and pharmacodynamics effects are reversible.

For alemtuzumab the following aspects should be considered: varicella zoster testing/ vaccination prior to first dose; other vaccination prior to dosing, particularly with live viral vaccines; premedication to minimize infusion reactions associated with extensive WBC lysis; monthly monitoring of platelets and thyroid/renal function for 4 years after last infusion for early detection of potential secondary autoimmunity; expect increased incidence of common infections and herpes. Alemtuzumab pharmacokinetics and pharmacodynamics effects are long-lasting. The median time to normalize T cells is of 5 years.

Three oral MS therapy are approved now in Europe – fingolimod, teriflunomide and dimetilfumarat.

In fingolimod treatment initiation, cardiovascular and ophthalmologic evaluations are necessary. Decreased white cell count (18% grade 4) is common and the periodic monitoring is required; very low lymphocyte count requests treatment interruptions. It is important to educate the patient on risk of teratogenicity; the pregnancy should be avoid until 2 months after cessation.

On teriflunomide treatment, the patients should be informed on the risk of teratogenicity and the need for effective contraception; prior to pregnancy, drug washout is essential. Frequent liver monitoring is required and the patient should be educate on signs and symptoms of liver disease. Prior to dosing, it is important to check for latent TB infection. White cell counts must be periodically monitored. The live attenuated vaccines should be avoid and the patient has to contact physician for signs/symptoms of infection. Hair thinning and GI upset may occur, but should recover.

For dimethyl fumarate, flushing or GI symptoms are very common or common, generally mild to moderate- the solutions of decreasing these symptoms are titration and administration with food. Decreased white cell count is uncommon (6% grade 3), but regular monitoring is required, as also the liver and kidney function.

As each patient needs the right therapy in right time, in order to have the best efficacy outcomes for his or her disease, an important part of patients' management is the safety's monitoring and counseling.

THE ENDONEURIAL MICROENVIRONMENT: ANATOMY, PATHOPHYSIOLOGY AND THERAPEUTIC TARGET

STEPHEN D. SKAPER

Department of Scienze del Farmaco, Università degli Studi di Padova, Italy

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 endoneural 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. Within this context, a contribution by mast cells has received only limited attention. 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 wide range of mediators (e.g. histamine, serotonin, heparin, proteases, pro-inflammatory cytokines, eicosanoids, chemoattractants, and neuropeptides, among others) 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 lipid signalling molecules, and N-palmitoylethanolamine in particular, which is produced on-demand within the cell's lipid bilayer and has been shown to possess anti-inflammatory, analgesic and anti-convulsant properties.

MANAGEMENT OF ADVANCED PARKINSON'S DISEASE

JÓZSEF SZÁSZ

University of Medicine and Pharmacy "Victor Babes" Timisoara; Head of Dept. of Neurology II, Clinical Emergency County Hospital Timisoara, Romania

Parkinson's disease (PD) is one of the most important disabling illnesses of later life. None of the available treatments influence the progression of the disease. Since the discovery of levodopa as the mainstay of pharmacotherapy in the early 1960s, the pharmacological treatment of PD has been continuously debated and adapted, mainly as a result of the pharmacokinetic properties and changing pharmacodynamics of this drug during the progression of disease, as this changes inevitably lead to predictable and unpredictable response fluctuations, both motor and non-motor. There are now several treatment options for switching from intermittent to continuous dopaminergic stimulation (CDS) therapy. Duodenal infusion of levodopa (LCIG) or apomorphine infusions offer significant benefits for selected patients and can be considered an option prior to surgery (Deep Brain Stimulation, DBS). Both of the STN and GPI providing continuous compensation for the PD-related striatal dopaminergic denervation. The indications for using one of the available CDS therapies are similar and include: pronounced motor and/or non-motor fluctuations, dyskinesias, severe conventional oral dopaminergic therapy-related complications (ICD's). Great improvements in motor and non-motor functioning can be achieved with CDS therapy, but limitations and many unanswered questions remain (such as how early CDS therapies should be initiated).

IMAGISTIC ASPECTS IN MULTIPLE SCLEROSIS

CRISTINA TIU

University of Medicine and Pharmacy "Carol Davila" in Bucharest, Romania

Magnetic resonance imaging is an essential component of the positive diagnosis of multiple sclerosis. Brain MRI is mandatory, but spine MRI should be performed from the beginning in order to gather the requested number of lesions in a clinically isolated syndrome, or to have an innitial evaluation used further, to monitor the disease activity .

There are diffferent techniques used to examine patients suspected to have multiple sclerosis. T2 and FLAIR sequences show the total number of lesions, with the particularity of FLAIR images to better differentiate the inflammatory lesions from CSF; T1 sequences will offer information about axonal degeneration or about highly active plaques, while T1 + Gadolinium will differentiate clearly the newest, active lesions.

Appart from the routine examination, the technological development allows us to identify cortical lesions (3D-MPRAGE), to evaluate the structural changes in the optic nerves (STIR techniques), to evaluate the metabolic features of a certain lesion, in order to differentiate tumoral lesions which can mimic a hyperacute onset of multiple sclerosis.

On the side of research, measurement of atrophy was an important tool in clinical trials, and functional MRI or DTI (diffusion tension imaging) can contribute to understand the progressive phase of the disease, or the cognitive dysfunction in MS patients

Monitoring the disease activity is now inseparable from the MRI aspect, the appearance of new T2 lesions or of more than one active lesion being a sign of lack of efficacy of first line immune- modulatory drugs and a strong reason for treatment escalation

CURRICULUM VITAE

OVIDIU BĂJENARU
/Romania

1983	: M.D. at the Faculty of Medecine of University of Medicine 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 - 2013	: President of the Romanian Society of Neurology
since 2013 -	: Honorary 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)

ANCA BUZOIANU
/Romania

OCCUPATION OR POSITION HELD

- Professor in Pharmacology
- Dean of the Medical Faculty, University of Medicine and Pharmacy "Iuliu Hatieganu", Cluj-Napoca, Romania
- General Secretary of the Romanian Society for Pharmacology. Therapeutics and Clinical Toxicology

TITLE OF QUALIFICATION AWARDED

- Specialist in Pediatrics
- Specialist in Clinical Pharmacology
- Senior Clinical Pharmacologist
- PhD

AWARDS IN THE LAST 2 YEARS

- Great "Iuliu Haţieganu" Award of the University of Medicine and Pharmacy Cluj-Napoca 2007
- "Victor Papilian " University Award for fundamental sciences 2006

MAIN ACTIVITIES AND RESPONSIBILITIES

- Head of the Department of Pharmacology (medical, scientific, and administrative responsibilities)
- Chairman of University Department (teaching courses for undergraduate students, postgraduate students and PhD students)
- Dean of the Medical Faculty of the University of Medicine and Pharmacy "Iuliu Hatieganu" Cluj-Napoca (administrative tasks, universitary management, curriculum planning etc)

International journal publications cited in databases
- 4 articles

Articles published in Romanian journals, cited in international databases
- 6 articles

Papers published in Romanian journals
- 46 articles

Monographies
- 2 monographies

Chapters in published books
- 9 chapters

ORGANISATIONAL SKILLS AND COMPETENCES

- European Society of Clinical Neuropharmacology- member in International Advisory Board
- The Society for the Study of Neuroprotection and Neuroplasticity (SSNN)
- Member European Association of Clinical Pharmacology and Therapeutics
- Member in the Balkan Medical Union
- Member in the International Association for the Study of Pain
- Vice-president of the Romanian Ministry Commission of Clinical Pharmacology, Toxicology and toxic dependences
- Member in The Romanian Group for Therapeutic Guidelines Elaboration
- Executive general secretary of the Romanian Society for Pharmacology, Therapeutics and Clinical Toxicology
- Member in the Ethical Committee of the “Iuliu Hatieganu” University Cluj-Napoca
- Head of the Pharmacology Department
- President of the Deans Romanian Association of Medical Faculties

LÁSZLÓ CSIBA
/Hungary

László Csiba was born in 1952, Sajószentpéter, Hungary. Now he is the Chairman of Department of Neurology of University Debrecen and Chair of Board of Director's (European Stroke Organisation), President of European Society of Neurosonology and Cerebral Hemodynamics. He is the chair of European Cooperation Committee of EFNS.

His research interests are stroke and stroke-prone diseases, ultrasonic studies in cerebrovascular diseases and clinicopathological studies on cerebrovascular diseases. He published numerous papers on stroke and stroke-related diseases, associated editor of Frontiers on Stroke and member of editorial committee (Intern. J Stroke)

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)

IOANA ISPAS
/Romania

Ioana Rodica Ispas is Advisor for European Affairs in Genomics, Bioethics and Health at Romanian Ministry of National Education since 2003. She graduated Biochemistry at University of Bucharest and she continued her studies at the same university where he obtained his Master's degree in 1994 and has a Master degree in Molecular Biology and a second Master in European Studies and Community Rights at the University of Bucharest –Faculty of Philosophy in 2009. As scientist she was Assistant Professor in Biochemistry and Organic Chemistry at the University of Ecology in Bucharest and scientific researcher (Molecular Biology Department) at University of Bucharest. She has more than 30 papers in molecular biology and bioethics. From 2007 until 2011 she worked as General Secretary of Romanian National Research Ethics Council and member of Romania National Ethics Commission for Ethics in Life Sciences where she was actively involved in drafting regulations for research ethics and code of conduct for research in life sciences. She worked for European Commission – Brussels, between 2004-2006 and took a second appointment between 2008-2011 as National Expert for 3DGs: DG RTD, DG SANCO and DG ENV being in charge with monitoring of FP6 contracts, policy briefings, foresight in biotechnology, ethics, gender research, and environmental risk assessment for genetically modified organisms. Starting with 2001 she is Romanian representative in Management Programme Committee of European Medical Research Programme (FP5, FP6, FP7 and Horizon 2020), Innovative Medicine Initiative, Joint Programming Initiative Neurodegenerative Diseases, Joint Programming Initiative of Antimicrobial Resistance.

She has extensive experience in EU project management (more than 16 years) , currently being scientific officer for 6 FP7 projects in: neurosciences, nanomedicine, infectious diseases (NEURON II, JPI AMR, EURONANOMED II, ERASYSPAPP,HIVERA.,INFECT-ERA) on behalf of Ministry of National Education.

AMOS KORCZYN

/Israel

Professor Amos D. Korczyn is the Sieratzki Professor of Neurology at Tel-Aviv University.
Professor Korczyn graduated from the Hebrew University – Hadassah Medical School in Jerusalem in 1966 (MD), where he also received an MSc degree in pharmacology (cum laude) in 1966. He trained in neurology at Beilinson Hospital and at the National Hospital for Nervous Diseases, Queen Square, London. He was the Chairman of the Department of Neurology at the Tel-Aviv Medical Center since 1981 until 2002. Professor Korczyn has a particular interest in dementia. He has authored or co-authored over 600 articles in peer-reviewed journals, as well as chapters in books, etc. Professor Korczyn is or has been an Editorial Board member of 15 international journals, and organized several neurological conferences, mainly in the field of dementia, Parkinson's disease and other degenerative brain disorders, as well as CONy – the International Congress on Controversies in Neurology.

VITALIE LISNIC
/Moldova

Dr. Vitalie Lisnic is a Professor of Neurology at Department of Neurology of the State University of Medicine and Pharmacy „Nicolae Testemitanu", Chisinau, Republic of Moldova. He is a consultant in the Department of Vertebroneurology and Neuropathies, responsible for electromyographic examinations at the Institute of Neurology and Neurosurgery in Chisinau.

Dr. Lisnic graduated the Faculty of General Medicine of the Chisinau State Medical Institute in 1989. He passed internships in Neurology and Neurophysiology in Moscow (Russian Federation) in 1993, Charles University, Pilsen (Czech Republic) in 1994, Landesnervenklinik of Salzburg (Austria) in 1999, Emory University, Atlanta (USA) in 2002 and 2003, Vienna University (Austria) in 2008. In 2003 obtained a clinical attachment in neuropathies at the National Institute of Neurology, Queen's Square, London, UK. In 2003-2004 he was the Principal Investigator of the Moldovan team of the grant of the Moldovan Research and Development Association and U.S. Civilian Research and Development Foundation.

Dr. Lisnic other important responsibilities include the following:

- President of the Moldovan Neurological Association
- Member of the Education Committee of the European Federation of Neurological Societies
- Delegate of the Republic of Moldova in World Federation of Neurology and European Federation of Neurological Societies
- Member of the American Academy of Neurology
- Member of editorial board of 2 Moldovan medical journals

Dr. Vitalie Lisnic is the author of more than 150 scientific publications in Moldovan and International biomedical journals. Under his guidance were defended 3 Ph.D theses.

TUDOR LUPESCU
/Romania

Tudor Lupescu obtained his medical degree from “Carol Davila” University of Medicine in Bucharest, in 1989. After 3 years of training at Colentina Clinical Hospital he became Specialist in Neurology in 1994. Since 2006 he is running the Neurology Department al Agrippa Ionescu Hospital in Bucharest. 1998, he qualified as Consultant Neurologist. Since his early years of training in Neurology, Tudor Lupescu has shown a special interest in Clinical Neurophysiology. In 2000 he earned a Competence in Clinical Neurophysiology (EEG, EMG, and Evoked Potentials). 1997 he was the first to use Transcranial Magnetic Stimulation in Romania. This was also the subject of his PhD thesis presented in 2005. Since 2008, Tudor Lupescu is President of ASNER – Romanian Society of Electrodiagnostic Neurophysiology. He is also founding member and vicepresident of the the Romanian Society of Diabetic Neuropathy.

Dr Tudor Lupescu is associate member of the American Academy of Neurology, and associate member of the American Association of Neuromuscular and Electrodiagnostic Medicine. Between 2008 and 2013 he was also member of the Neurophysiology Subcommittee of ENS.

DAFIN FIOR MUREŞANU
/Romania

Muresanu Fior Dafin, MD, PhD, MBA, FANA, is the President of the Romanian Society of Neurology, Professor of Neurology, Chairman Department of Neurosciences, "Iuliu Hatieganu" University of Medicine and Pharmacy Cluj-Napoca, member of the Academy of Medical Sciences, Romania, and secretary of Cluj-Napoca regional branch. He also acts as the President of the Society for the Study of Neuroprotection and Neuroplasticity. In these roles, he is involved as member of the faculty in international educational programs of European Master (i.e. European Master in Stroke Medicine, University of Krems), organizer and co-organizer of European and international schools and courses (International School of Neurology, European Stroke Organisation Summer School, Danubian Neurological Society Teaching Courses). His activity includes involvement in many clinical studies and research projects, memberships in the executive board of many national and international societies, participations as invited speaker in national and international congresses, a significant portfolio of scientific articles (over 100 papers indexed on Web of Knowledge-ISI) as well as contributions in monographs and books published by prestigious international publishing houses. In the last 7 years, he was also invited as speaker in over 200 scientific events both national and abroad. 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 "Gheorghe Marinescu Award" for advanced contributions in Neuroprotection and Neuroplasticity.

CRISTINA PANEA
/Romania

Cristina Aura Panea has graduated the University of Medicine and Pharmacy “Carol Davila” Bucharest in 1986. She has started the neurology specialty and her university teaching career in the Neurology Department of the University Emergency Hospital of Bucharest in 1991 and has obtained her PhD in Medical Sciences in 2000. Starting with 2003, she is Associated Professor and the Head of the Neurology Department of Elias Emergency University Hospital.

The main fields in which she has activated are epilepsy, multiple sclerosis and movement disorders – fields in which she had elaborated over 100 papers and has carried out numerous clinical researches.

She is a member of the Romanian Neurology – which treasurer she was between the years 2001 to 2009; also she is a member of the European Neurology Society, American Academy of Neurology and of the International Movement Disorders Society.

CRISTIAN FALUP-PECURARIU

/Romania

Cristian Falup-Pecurariu received his medical degree from the University of Medicine and Pharmacy "Iuliu Haţieganu" from Cluj-Napoca. He hold a 1 year fellowship of the European Neurological Society in movement disorders and sleep medicine at Hospital Clinic, University of Barcelona, Spain.

He is Head of the Department of Neurology, County Emergency University Hospital from Brasov, and is Lecturer of Neurology at the Transilvania University from Braşov. During his career Cristian Falup-Pecurariu was President of the European Association of Young Neurologists and Trainees (EAYNT), EAYNT Liasion Officer with World Federation of Neurological Society, co-representative of Europe on the International Working Group for Young Neurologists and Trainees (World Federation of Neurology), Secretary of the EFNS/MDS-ES Panel on Movement Disorders and currently is member of the Educational Committee of MDS-ES.

His research focuses on non-motor aspects of Parkinson's diseases and restless legs syndrome.

LAURENȚIU M. POPESCU

/Romania

L.M. Popescu, MD, PhD, Dr. h.c.mult., is currently Professor of Cellular and Molecular Medicine, 'Davila' University of Medicine, Bucharest, Romania and Head of the National Institute of Pathology, Bucharest, Romania. He is fellow of the National Academy of Sciences. Recently, he became President of the Federation of European Academies of Medicine. He published over 125 scientific articles in international peer-review journals and is cited more than 1700 times. He has a Hirsch Index of about 30. Professor Popescu is Editor-in-Chief (and founder) of the Journal of Cellular and Molecular Medicine (Wiley/Blackwell), with a 5-year IF of 5. He is credited with the discovery of Telocytes.

BOGDAN O. POPESCU
/Romania

Bogdan O. Popescu - born March 8th, 1971 in Bucharest, Romania.
Address: Department of Neurology, School of Medicine, 'Carol Davila' University of Medicine and Pharmacy, Colentina Clinical Hospital, 19-21 Sos. Stefan cel Mare, sector 2, 020125, Bucharest, 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
2009 - 2012	Lecturer, 'Carol Davila' University School of Medicine
2009 -	Senior Researcher, 'Victor Babeş' National Institute of Pathology, Bucharest, Romania
2012 -	Associate Professor, 'Carol Davila' University School of Medicine and Head of Neurology Unit II, Colentina Clinical Hospital

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 and	'Science and Art National Foundation Award of Excellence for research in the field of Neuroscience 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
President elect of the Romanian Society of Neurology (2017-2021) and former Secretary General (2001-2013)
Research director of the Society for the Study of Neuroprotection and Neuroplasticity
Director, Victor Babeş' National Institute of Pathology, Bucharest, Romania

Selected publications
1. Popescu BO, Gherghiceanu M, Kostin S, Ceafalan L, Popescu LM. Telocytes in meninges and choroid plexus. Neurosci Lett. 2012, 516:265-9.
2. Hort J, O'Brien JT, Gainotti G, Pirttila T, Popescu BO, Rektorova I, Sorbi S, Scheltens P; EFNS Scientist Panel on Dementia. EFNS guidelines for the diagnosis and management of Alzheimer's disease. Eur J Neurol. 2010, 17:1236-48.
3. Popescu BO, Toescu EC, Popescu LM, Bajenaru O, Muresanu DF, Schultzberg M, Bogdanovic N. Blood-brain barrier alterations in ageing and dementia. J Neurol Sci, 283:99-106, 2009.

4. Cowburn RF, Popescu BO, Ankarcrona M, Dehvari N, Cedazo-Minguez A. Presenilin-mediated signal transduction. Physiol Behav. 2007;92:93-7.
5. Hansson CA, Popescu BO, Laudon H, Cedazo-Minguez A, Popescu LM, Winblad B, Ankarcrona M. Caspase cleaved presenilin-1 is part of active gamma-secretase complexes. J Neurochem. 2006;97:356-64.
6. Popescu BO, Ankarcrona M. Mechanisms of cell death in Alzheimer's disease: role of presenilins. J Alzheimers Dis. 2004;6:123-8.
7. Popescu BO, Cedazo-Minguez A, Benedikz E, Nishimura T, Winblad B, Ankarcrona M, Cowburn RF. Gamma-secretase activity of presenilin 1 regulates acetylcholine muscarinic receptor-mediated signal transduction. J Biol Chem. 2004;279:6455-64.
8. Cedazo-Mínguez A, Popescu BO, Blanco-Millán JM, Akterin S, Pei JJ, Winblad B, Cowburn RF. Apolipoprotein E and beta-amyloid (1-42) regulation of glycogen synthase kinase-3beta. J Neurochem. 2003;87:1152-64.
9. Popescu BO, Oprica M, Sajin M, Stanciu CL, Bajenaru O, Predescu A, Vidulescu C, Popescu LM. Dantrolene protects neurons against kainic acid induced apoptosis in vitro and in vivo. J Cell Mol Med. 2002;6:555-69.
10. Popescu BO, Cedazo-Minguez A, Popescu LM, Winblad B, Cowburn RF, Ankarcrona M. Caspase cleavage of exon 9 deleted presenilin-1 is an early event in apoptosis induced by calcium ionophore A 23187 in SH-SY5Y neuroblastoma cells. J Neurosci Res. 2001;66:122-34.

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 elec

tron 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)

MIHAELA SIMU
/Romania

Mihaela Simu is presently working as Professor and Chairman of the Neurology Department II of University of Medicine and Pharmacy "Victor Babes" - Timisoara.
Professor Simu is currently Vicepresident of the Romanian Society of Neurology, one of the coordinators of the National Programme for the treatment of Multiple Sclerosis in Romania, active member of ENS, EFNS, American Academy of Neurology, and MDS.
Professor Simu has been and is involved as principal investigator in more than 20 international and national multicentric trials and 4 national research grants, and is presently the Romanian project leader in the BIOMARK HURO project (cooperation between Szeged and Timisoara medical Universities). Her interests are directed mainly in clinical neurology, in particular in multiple sclerosis, Parkinson disease, dementia, cerebrovascular and focal dystonias.
As author or co-author, has published and reported more than 100 national and international scientific papers, 3 medical books and 2 neurology courses in a bilingual (Romanian /English) version.

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 Professor, 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, Faculty of Medicine, Padova, Italy (2009-present).

PROFESSIONAL MEMBERSHIPS: Sigma CI (The Scientific Research Society); Phi Lambda Upsilon (honorary chemistry society); Alpha Chi Sigma (professional society in chemistry/chemical engineering); Society for Neuroscience; International Society for Cerebral Blood Flow and Metabolism

JOURNALS EDITED: Editor-in-Chief, CNS & Neurological Disorders – Drug Targets; Editor-in-Chief, Clinical CNS Drugs; Associate Editor, American Journal of Neuroprotection and Neuroregeneration; Editorial Board Member, Nature Scientific Reports (Neuroscience); Councilor, International Association of Neurorestoratology
REVIEW PANELS: The Wellcome Trust (UK), Biotechnology and Biological Sciences Research Council (BBSRC) (UK), Austrian Science Fund (ad hoc review panel to evaluate interdisciplinary doctoral programmes in neuroscience)

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 240 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 Neuroinflammation, Neurobiology of Disease, Neurobiology of Aging, Glia, Apoptosis, Molecular & Cellular Neuroscience, Journal of Pharmacology and Experimental Therapeutics, Neuroscience, British Journal of Pharmacology, Neuropharmacology, European Journal of Pharmacology, Journal of Neurological Sciences

CRISTINA TIU
/Romania

I always considered myself an optimistic person but still there are certain things which I find depressing, and a CV is one of those things. Suddenly it is not about you anymore, but about a person who had a number of achievements which are rarely the things you find interesting about yourself, and all your life is compressed in half a page.

I have graduated the University of Medicine and Pharmacy "Carol Davila" in Bucharest in 1987 and I started my career in neurology in 1991, as a resident in the Department of Neurology of the University Hospital Bucharest, the same place where now I am Associated Professor and Head of the Stroke Unit. I have two favorite domains: vascular pathology and multiple sclerosis. My main interest is in cerebrovascular diseases, I am coordinating a teaching course for cervical and cerebral ultrasonography and I followed the European Master in Stroke Medicine Programme in Austria.

My involvement in MS field started in year 2000, when the first patients in Romania were treated with DMTs due to a constant effort (read fight) of three people: Prof. Ioan Pascu, Prof. Alexandru Serbanescu and Prof. Ovidiu Bajenaru. Since then, I have followed-up hundreds of patients with MS, and I am now the coordinator of the University Hospital Bucharest Center for the National Programme for treating the Patients with Multiple Sclerosis. I have participated, together with my colleagues in the majority of the main International Clinical Trials in MS in the last decade and we had also several original scientific work related to clinical aspects of MS patients. I am one of the two representatives of the Romanian Society of Neurology in the Board of ECTRIMS. In the end of my half page, I am looking forward to future goals: development of basic research in MS in Romania, a National MS Registry, better drugs, a better education for patients and doctors, a better me...


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