International School of Neurology

RoNEURO Institute for Neurological Research and Diagnostic

FOUNDATION OF THE SOCIETY FOR THE STUDY OF NEUROPROTECTION AND NEUROPLASTICITY

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11TH INTERNATIONAL SUMMER SCHOOL OF NEUROLOGY

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Registration Desk

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

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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 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 currency in Romania 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

11 INTERNATIONAL SUMMER SCHOOL OF NEUROLOGY

3-7 JULY, 2016 | EUROPA HOTEL | EFORIE NORD | ROMANIA

SUNDAY, JULY 3RD, 2016

MODULE COORDINATORS: Hari Shanker Sharma (Sweden), Stephen Skaper (Italy)

18:30 – 19:00	Hari Shanker Sharma, Sweden Neuroprotection and neurotoxicity of nanoparticles in the central nervous system with special reference to nanomedicine
19:00 – 19:30	Stephen Skaper, Italy Co-ultramicronized palmitoylethanolamide/luteolin promotes oligodendrocyte development, precursor cell survival and improves outcome in experimental autoimmune encephalomyelitis
19:30 – 20:00	Dana Boering, Germany Basic mechanisms of postlesional plasticity after stroke
20:00	***DINNER***

MONDAY, JULY 4TH, 2016

MODULE COORDINATORS: Volker Hömberg (Germany), Caterina Pistarini (Italy)

08:50 – 09:00	***WELCOME ADDRESS*** Dafin F. Mureșanu (Romania), Volker Hömberg (Germany), Hari Shanker Sharma (Sweden)
09:00 – 10:30	Volker Hömberg, Germany The art of neurological examination
10:30 – 11:00	Volker Hömberg, Germany Restorative neurology- do we ask the right questions?
11:00 – 11:30	***COFFEE BREAK***
11:30 – 12:00	Volker Hömberg, Germany Pharmacology in neurorehabilitation
12:00 – 12:30	Volker Hömberg, Germany Sense and non-sense of physical medicine in neurorehabilitation
12:30 – 13:00	Dafin F. Mureșanu, Romania Brain plasticity and neurorehabilitation in Parkinson's disease
13:00 – 13:30	Caterina Pistarini, Italy Brain-computer interface in neurorehabilitation in post-stroke patients
13:30	***LUNCH***

MONDAY, JULY 4TH, 2016

17:30 – 18:00	Ovidiu Băjenaru, Romania Classification and pathophysiology of dystonia
18:00 – 18:30	Ovidiu Băjenaru, Romania Diagnosis and treatment of dyskinesia
18:30 – 19:00	***COFFEE BREAK***
19:00 – 19:20	Adina Crăciunoiu, Romania Case Presentation
19:20 – 19:40	Amalia Ene, Romania Case Presentation
19:40 – 20:00	Dan Popescu, Romania Case Presentation
20:00	***DINNER***

TUESDAY, JULY 5TH, 2016

MODULE COORDINATORS: Natan Bornstein (Israel);
Dafin F. Mureșanu (Romania)

09:00 – 09:45	Natan Bornstein, Israel Time is Brain, TIA as an Emergency
09:45 – 10:45	Natan Bornstein, Israel Secondary stroke prevention. Management of symptomatic carotid stenosis CEA vs. Stent
10:45 – 11:30	Natan Bornstein, Israel Management of ICH
11:30 – 12:00	***COFFEE BREAK***
12:00 – 12:30	Dafin F. Mureșanu, Romania Motor rehabilitation after stroke
12:30 – 13:00	Dafin F. Mureșanu, Romania Cognitive rehabilitation after stroke
13:00	***LUNCH***
18:00 – 18:45	László Csiba, Hungary Desobliteration therapy in acute stroke
18:45 – 19:30	László Csiba, Hungary When the guideline does not help
19:30 – 20:30	László Csiba, Hungary Case presentations
20:30	***DINNER***

WEDNESDAY, JULY 6TH, 2016

MODULE COORDINATORS: Amos Korczyn (Israel), Antonio Federico (Italy)

09:00 – 09:30	Amos Korczyn, Israel Disease course modification in Parkinson's disease
09:30 – 10:00	Ovidiu Băjenaru, Romania The clinical profiles of patients with PD – impact on treatment options and quality of life
10:00 – 10:30	Peter Jenner, UK Wearing off, dyskinesia, and the use of continuous drug delivery now and in the future
10:30 – 11:00	Cristian Falup-Pecurariu, Romania Corticobasal degeneration – new advances
11:00 – 11:30	***COFFEE BREAK***
11:30 – 12:00	Antonio Federico, Italy Fabry disease: diagnosis, management and physiopathology
12:00 – 12:30	Antonio Federico, Italy Pompe Disease (acid alpha glucosidase deficiency): diagnosis, management and pathogenesis
12:30	***LUNCH***

WEDNESDAY, JULY 6TH, 2016

18:00 – 18:30	Bogdan Popescu, Romania Advanced therapy for Parkinson's disease
18:30 – 19:00	Bogdan Popescu, Romania Clinical cases of advanced Parkinson's disease – treatment option and patient management
19:00 – 19:30	József Szász, Romania Management of Advanced Parkinson's Disease: CDS therapies - limitations and unanswered questions (how early CDS therapies should be initiated?)
19:30 – 20:00	Cristian Falup-Pecurariu, Romania The video-case based approach to movement disorders
20:00	***DINNER***

THURSDAY, JULY 7TH, 2016

MODULE COORDINATORS: Ovidiu Băjenaru (Romania), Cristina Tiu (Romania)

09:00 – 09:30	Ovidiu Băjenaru, Romania Update in the neuropathology of MS
09:30-10:00	Anat Achiron, Israel Treatment decisions in multiple sclerosis
10:00 – 10:30	Ovidiu Băjenaru, Romania New opportunities for the immunomodulatory treatment of MS in Romania in 2016
10:30 – 11:00	Mihaela Simu, Romania Pathogenesis, diagnosis and risk stratification algorithm for Natalizumab-associated progressive multifocal leukoencephalopathy
11:00 – 11:30	***COFFEE BREAK***
11:30 – 12:00	Cristina Tiu, Romania Multiple Sclerosis before, during and after pregnancy
12:00 – 12:30	Tudor Lupescu, Romania Transcranial Magnetic Stimulation - principles, technique, applications
12:30 – 13:00	Andrea Antal, Germany Transcranial direct current stimulation (tDCS): possibilities, clinical applications and common pitfalls
13:00 – 14:00	***LUNCH***
14:00 – 15:00	Final Examination
15:00	***OFFICIAL CLOSING***
20:00	***GALA DINNER***

ABSTRACTS

TRANSCRANIAL DIRECT CURRENT STIMULATION (TDCS): POSSIBILITIES, CLINICAL APPLICATIONS AND COMMON PITFALLS

ANDREA ANTAL
Department of Clinical Neurophysiology, University Medical Center, Göttingen, Germany

Neuroplasticity became one central topic of neuroscience research in the last decades. Dynamic modifications of neuronal networks are an important substrate for learning and memory formation. Pathological neuroplasticity might be one foundation of numerous central nervous system diseases. Transcranial direct current stimulation (tDCS) was developed by our group as a non-invasive tool to induce neuroplasticity in the human cerebral cortex. tDCS as a tool aims to induce prolonged neuronal excitability and activity alterations in the human brain via alterations of the neuronal membrane potential. Accordingly, tDCS in the human is a promising tool in the treatment of diseases that are accompanied by changes of cortical excitability. tDCS seems also to be an efficient tool to alter learning and cognitive performance in healthy humans. The effects have been most extensively tested for the motor cortex stimulation. Unfortunately the results of the different studies are not always consistent. It was frequently observed that the efficacy and direction of the effects depends on the timing of stimulation, electrode arrangement, and task characteristics, besides anatomical and physiological factors. Future studies systematically probing the stimulation parameters and developing new protocols are needed to explore the reasons for the inconsistencies.

TREATMENT DECISIONS IN MULTIPLE SCLEROSIS

ANAT ACHIRON
Multiple Sclerosis Center, Sheba Medical Center, Tel-Hashomer, ISRAEL

Multiple sclerosis (MS) is a chronic, central nervous system devastating disease affecting young adults. The disease that may result in permanent disability over-time affecting patients' daily activities and quality of life. The diagnosis and management of MS continue to evolve rapidly, with an ongoing emergence of new disease-modifying drugs (DMDs), newer diagnostic criteria and revisions to MS phenotypes, and the identification of magnetic resonance imaging (MRI) measures and biomarkers capable of improving disease surveillance. Clinicians face more treatment choices and must address patient concerns about DMD side effects, treatment preferences, and drug costs, that can negatively impact adherence to treatment. Moreover, there is a window of opportunity in MS treatment which should be taken into consideration to maximize therapy benefits. Many studies have demonstrated that the earlier treatment is started, the better the results will be. In my overview lecture

I will explain the mechanisms of action, efficacy, and safety profiles of current and emerging DMDs and discuss evidence-based principles for early therapy initiation and follow-up monitoring. I will outline effective response to DMD treatments in relation to relapse rate, progression to confirmed disability, disease activity by MRI variables and brain atrophy, that are of importance to the care of MS patients in order to achieve and sustain beneficial treatment management.

CLASSIFICATION AND PATHOPHYSIOLOGY OF DYSTONIA

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

Dystonia is a movement disorder characterized by sustained or intermittent muscle contractions causing abnormal, often repetitive, movements, postures, or both. The actual 2013 MDS deffinition and classification of dystonia, is based on two main axes – the clinical phenomenology and etiology. The clinical phenomenology determined by the clinical observations accumulation during the last more than 20 years, is based on five main criteria: age at onset, body distribution, temporal pattern, coexistence of other movement disorders, and other neurological manifestations; it allows a detailed and practical possibility to have the right and complex clinical diagnosis, and it also offers an important criteron for an etiologic orietation. The etiologic classification takes into account two useful complementary characteristics: identifiable anatomical changes and pattern of inheritance. The huge progress in this direction is based on the development and clinical implementation of the spectacular evolution of imaging technologies – mainly the functional imaging of the brain, but also of neuroelectrophysiologic techniques, and in the same time by the explosive knowledge and clinical applications of cellular and mollecular genetics. These same techniques alloed also the understanding of the patophysiology of dystonia, based on the study of neuroplasticity and very fine aspects of neurodegenerative processes in the complex brain neuronal networks centered on the corticostriatal circuits and their connections. The understanding of these pathophysiological aspects and a correct diagnosis in terms of the new classification of dystonia represents today the rationale basis for the right therapeutical approach of these patients.

DIAGNOSIS AND TREATMENT OF DYSKINESIA

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

Dyskinesia refers to a category of movement disorders that are characterized by complex involuntary muscle movements, including movements similar to tics, chorea or other primary abnormal movements and diminished voluntary movements. Dyskinesia is not a disease, but a symptom of several medical disorders that are distinguished by their underlying cause. Most dyskinesia appear as medication-induced dyskinesias (which may be classified in three main subgroups: neuroleptic-induced dyskinesia, levodopa-induced dyskinesia and tardive syndromes/ dyskinesia), or – more rarely in genetic combined forms of dystonia/dyskinesias (identified in the actual MDS classification of genetic dystonia). One of the most difficult to diagnose and treat group of dyskinesia are the tardive syndromes, which today may be sudivided into more clinical subtypes: tardive dyskinesia (the most frequent form), tardive dystonia, tardive akathisia, tardive tics (tourettism), tardive myoclonus, tardive tremor and withdrawal-emergent syndrome. Patients may simultaneously have more than one tardive syndrome. In addition to movement disorders (including involuntary vocalizations), patients with TD may have a variety of sensory symptoms, such as urge to move (as in akathisia), paresthesias, and pain. Combination of different phenomenologies, which may include a movement disorder as well as sensory symptoms, the term ''tardive syndrome'' is considered by many experts more appropriate when referring to all tardive disorders; tardive syndromes often are a source of great distress, anxiety and disability to patients and may be permanent, despite discontinuing the responsible medication. The actual different definitions of these syndromes according to AAN and DSM-5 are discussed. The understanding of the pathophysiology and clinical phenomenology of these complex movement disorders also represents the rationale basis for the actual symptomatic therapeutic possibilities.

THE CLINICAL PROFILES OF PATIENTS WITH PD – IMPACT ON TREATMENT OPTIONS AND QUALITY OF LIFE

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

The clinical profiles of patients with PD are very complex and are characterized by the clinical core of motor parkinsonism, with different dominance of motor symptoms in different disease stages and the combination of a huge variety of

non-motor symptoms due to the the disease itself; to these, other symptoms are added and interfere with the natural evolution of the disease, due to the actual symptomatic treatments for PD and their complications, but also to some more frequent co-morbidities. The interference among the natural neuropathologic evolution of the disease itself - with different particularities in different diseases stages, with the pharmacologic effects of the drugs and/ or the biologic effects of deep brain stimulation (in some patients in advanced stages), with aging and comorbidities and also with some particularities of the personality structure of the patients, offer the basis of understanding the great variety of clinical profiles of PD patients in different disease stages. This understanding has to determine the doctors to make all the efforts to identify a personalized profile of each patient as a rationale for personalized and most efficient treatment options in order to minimize as much as possible the disability determined by the disease in combination with all the above variable factors to minimize the inherent adverse effects and to increase the active survival and the quality of life of the patients and their caregivers.

UPDATE IN THE NEUROPATHOLOGY OF MS

OVIDIU BĂJENARU

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

The understanding of neuropathology of multiple sclerosis recognized a great development during the last two decades and is continuously developping, due to the evolution of neuroimmunology, neurohistology and cellular and mollecular medicine, in combination with clinical and modern neuroimaging techniques. Apart of the classical demyelinating lesions, important progress has been achieved in demonstrating the extended and irreversible neurodegenerative nature of this disease and its diffuse chronic inflammatory basis mediated by complex neuroimmunologic mechanisms. The correlations among the neuropathologic evolutive patterns and neuroimmunologic mechanisms, also allowed the understanding of the pathologic heterogeneicity of the disease and the identification of at least four pathologic patterns and their clinical correlations. The most recent data also allowed the understanding that in many cases an important chronic meningeal inflammatory process and the presence of inflammatory infiltrates in the deep cortical sulci have an important role in the patogenesis of this disease, in particular in the development of cortical lesions which are frequently present, and at least in part mediated by the soluble inflammatory mediators originating in the cortical follicular inflammatory infiltrates. More detailed aspects of the demyelinating cortical lesions but also of the degenerative subtle changes at the level of the components of cortical neurons – as their dendrites and synaptic connections, have been also identified. All these aspects may correlate with different

evolutive clinical patterns of the relapsing-remitting forms and progressive forms of MS, but also with some particular sets of clinical features as neurocognitive impairment. O synthesis of these new data will be presented.

BASIC MECHANISMS OF POSTLESIONAL PLASTICITY AFTER STROKE

DANA BOERING
Medical Director, Gesundheitszentrum, Bad Wimpfen, Germany

In the last two decades, neuroscience research provided considerable evidence demonstrating that the cerebral cortex of adult mammals, including humans, possesses substantial capacity for structural and physiological plasticity after acute brain lesion. While the basic mechanisms underlying cortical plasticity are still under intense investigation, the implications for developing novel therapeutic interventions for central nervous system injury are now inescapable. Therapeutic interventions aimed to restore motor, sensitive or cognitive function after stroke are now based on hypotheses derived from our rapidly evolving understanding of brain plasticity processes.

The talk will give an overview on the lifelong mechanisms of motor learning related plasticity , lead over to a comprehensive presentation of basic mechanisms of neural recovery after stroke, and draft some of the actual plasticity modulation modalities including neurobehavioral modulation, pharmacological neuromodulation, noninvasive brain stimulation, somatosensory input modulation, brain computer interfaces, fMRI based feedback, pointing out that we are dealing with a challenging, rapidly evolving field.

MANAGEMENT OF SYMPTOMATIC CAROTID STENOSIS CEA VS. STENT

NATAN M. BORNSTEIN
Tel-Aviv University, Sackler Faculty of Medicine, Israel
Stroke Unit at 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.

SECONDARY STROKE PREVENTION. MANAGEMENT OF SYMPTOMATIC CAROTID STENOSIS CEA VS. STENT

NATAN M. BORNSTEIN
Tel-Aviv University, Sackler Faculty of Medicine, Israel
Stroke Unit at Tel-Aviv Medical Center, Israel

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

References

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

TIME IS BRAIN, TIA AS AN EMERGENCY

NATAN M. BORNSTEIN
Tel-Aviv University, Sackler Faculty of Medicine, Israel
Stroke Unit at Tel-Aviv Medical Center, Israel

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.

DESOBLITERATION THERAPY IN ACUTE STROKE

LÁSZLÓ CSIBA
Department of Neurology, Debrecen University, Hungary

1. IV Desobliteration

IV. TPA treatment for acute ischemic stroke must be applied as soon as possible.

Treatment within 3 hours:complete or near complete recovery (38 vs.21% placebo), while three-month mortality was not significantly different between the alteplase and placebo groups despite a 10-fold increase in symptomatic intracerebral hemorrhage among the alteplase group (6.4% vs 0.6%). Patients treated within 90 minutes had better outcomes than those treated 90 to 180 minutes after symptom onset.

Treatment from 3 to 4.5 hours: the trial excluded patients >80 years old, those with an nihss >25, those with a combination of previous stroke and diabetes and those on anticoagulants regardless of inr. There was a favorable outcome with (52% vs 45%) and no difference in mortality (7.7% vs. 8.4 %). The sooner intravenous alteplase treatment is initiated, the more likely it is to be beneficial. The effectiveness of IV. T-pa (4.5 to 6 hours after stroke) is neither established nor excluded by the available data (open-label ist-3 trial).

Although the recanalisation is associated with clinical benefit it is influenced with different factors including: clot age and composition (thrombolysis may be more effective for recent thromboembolic event, mechanical disruption may be more effective for older event).

Extracranial ICA occlusion, carotid t occlusion, mca occlusion (a hyperdense mca sign may still benefit from intravenous TPA treatment despite poorer overall prognosis, and early ct signs of infarction do not appear to modify the benefit of intravenous TPA treatment. Basilar occlusion: IV alteplase may be effective >6 hours who do not have extensive ischemia on baseline neuroimaging.

Factors affecting outcome: age (no reason to exclude older patients from IV. Thrombolysis), gender (conflicting data), hyperglycemia (associated with greater infarct size and worse clinical outcome). Reocclusion (high initial stroke severity and severe ipsilateral carotid disease are associated with an increased risk of early mca reocclusion after intravenous TPA therapy).

2. Mechanical thrombectomy (ca. 10 % of large artery occlusion in the anterior circulation)

Five new studies proved the efficacy if:

- NIHSS of ≥2 points and aspects score ≥6 on noncontrast brain ct,
- Angiography: occlusion of the distal intracranial ica, or the middle, (m1/m2) or anterior (a1/a2) cerebral artery,
- Within 6 hours of stroke onset,

Exclusion criteria

Arterial blood pressure >185/110 mmhg

Blood glucose <2.7 or >22.2 mmol/l

Platelet count <40,000/microl or international normalized ratio >3.0)

3. Intra-arterial thrombolysis

Remains unproven, the total dose of intra-arterial therapy is about one-third of the intravenous dose. It is a reasonable treatment option for patients with acute ischemic stroke who have contraindications to intravenous thrombolysis.

The results of five randomized controlled trials:

- Significantly increased likelihood of a good outcome
- Higher rates of partial or complete vessel recanalization the risk of symptomatic intracerebral hemorrhage was increased with intra-arterial thrombolysis (9% vs 2 %), but no increase of mortality.

Other aspects: patients with suspected or confirmed occlusion of the basilar artery who cannot be treated with IV. T-pa may still be considered for intra-arterial alteplase therapy at expert centers. Intra-arterial thrombolytic therapy can be considered for patients who are ineligible for intravenous thrombolysis with angiographically demonstrated acute basilar artery occlusion but no signs of major infarction on CT or MRI scan. The duration of time window can be up till 12 hours (individual variations).

4. The ultrasound enhanced thrombolysis combines intravenous TPA treatment and high-frequency ultrasound or transcranial color-coded doppler or low-frequency ultrasound. Another variation combines intravenous TPA with ultrasound and administration of microbubbles, which are small microspheres filled with air or gas that. The techniques remain investigational.

5. Risk of intracerebral hemorrhage

Intravenous thrombolysis

- Early CT changes: the presence or absence of early ischemic changes on CT scan can not be used to select patients for thrombolytic therapy in the conventional 3 hour time window.
- Stroke severity: stroke severity alone cannot be used to select or exclude patients for intravenous thrombolysis.
- Age: the evidences are conflicting.
- Heart diseases (heart failure, af etc.)
- Hyperglycemia, elevated hemoglobin a1c, diabetes mellitus, renal impairment, recent hypertension, preceding antiplatelet or ac therapy, borderline of low platelet
- Leukoaraiosis, size of lesion on dwi
- Persisting occlusion
- Noac: IV. Therapy only if no intake for 2 days with normal kidney function or the specific coagulation test are absolutely normal.
- Intra-arterial thrombolysis — for intra-arterial thrombolytic therapy, factors related to the risk of intracerebral hemorrhage appear to be similar to those reported for intravenous thrombolysis. In the following independent predictors of hemorrhagic transformation were found on multivariate analysis:
    - Higher baseline nihss score
    - Higher glucose levels
    - Longer time to recanalization

- Lower platelet count
- microcatheter contrast injections during intra-arterial thrombolysis
- Extravasated contrast medium on ct
- Early venous filling on angiography
- Cerebral microbleeds (small number of microbleeds <5) is not a contraindication

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WHEN THE GUIDELINE DOES NOT HELP

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Intravenous tissue plasminogen activator (IV-TPA) for acute ischemic stroke (AIS) should be dangerous in ascending aortic dissection due to the possibility of a pericardial tamponade or other bleeding complications. The physicians should be cautious if chest pain or pulseless syndrom are associated with stroke signs (especially with left sided hemiparesis). On contrary, both extra- nd intracranial dissections appear to be safe for IV. Thrombolysis and no evidence of subarachnoidal hemorrhage (SAH) could be seen on baseline imaging.

Myocardial infarct with concomottant acute ischemic infarct can be also complicated with bleeding and needs cautious evaluation. Although ca. 6-7% of IV-TPA treated patients can host an unruptured intracranial aneurysm on angiography, the risk of subarachnoidal bleeding remains low, especially if the size of aneurysm is small and no clinical sign of rupture is present. If either the clinical symptoms or the results of imaging raise the suspicion of an acutely thrombosed aneurysm presenting with ais, the thrombolysis should be withheld.

The data regarding the safety of intravenous thrombolysis in acute ischemic syndrom patients harboring arteriovenous malformations are insufficient. The presence of cerebral cavernous malformation increases the bleeding risk and mortality, therefore the lysis should be withheld. The IV. lysis in patients with benign tumor (e.g. meningeoma) is safe, while no intervention is suggested for patients suffering from either primary or secondary malignant brain tumor or for end-stage patients. Postepileptic hemiparesis (todd paresis), MS, migraine associated with hemiparesis, subdural hematoma are stroke mimics and the IV. Lysis (with the exception of subdural hematoma and malignant brain tumors) appears to be safe. IVT should be started for patients with mild stroke or rapidly improving stroke symptoms if residual deficits are considered by the patient or the treating physician as potentially disabling, because the risk of bleeding is low. Similarly, a recent transient ischemic attack in patients with stroke should not exclude TPA administration. A silent infarction on CT or MRI should not be used as a reason to exclude ivt when clinically evident strokes within the past 3 months have been excluded.

A strict blood pressure control is suggested (less than 185 hgmm) for the IV. intervention, because a moderate blood pressure substantially lowers the risk of sich. Transient orolingual edema happens in 1- 2% of lysis patients (previous ace therapy is a risk factor), the edema should be treated and the t -pa infusion continued.

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THE VIDEO-CASE BASED APPROACH TO MOVEMENT DISORDERS

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Movement disorders are common findings in clinical neurological practice, most of them being the result of abnormal functioning of the basal ganglia and their connections. The two main categories of movement disorders can be identified based on clinical observation: too little movement (hypokinesia) or an excess of movement (hyperkinesia). Phenomenology of movement disorders are taking into account important features like rhythmicity, frequency and amplitude. Hypokinesia, bradykinesia and akinesia are terms that define the paucity of movement - decreased amplitude, slowness or loss of movement – and are generally associated with rigidity, being the main features of parkinsonian syndromes. Most hyperkinetic movements are: tremor (rhythmical, regular), myoclonus (sudden, brief shock-like), chorea (irregular, non-rhythmic, rapid), tics (jerky, repetitive) and dystonia (prolonged contraction of both agonist and antagonist muscles).

The presentation will cover some key points of defining the main types of movement disorders, using video-case based approach.

CORTICOBASAL DEGENERATION – NEW ADVANCES

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The corticobasal degeneration (CBD) is a heterogeneous clinical and pathological syndrome. It belongs to the atypical parkinsonism group and it is encountered the least common from it. Probably it is the most challenging atypical parkinsonism. It is due by the abnormal aggregation of hyperphosphorylated tau proteins. In the last years there were new discoveries related to the genetics and potential link to the pathogenesis. Recently new diagnostic criteria have been published. The anatomopathological exam is the definitive diagnosis.

It comprise corticobasal syndrome (CBS), which is based on a clinical diagnosis, and CBD, which is based on a pathologically confirmed diagnosis. There is a low clinical and pathological correlation of CBD.

The main pathologically features are: narrowing of cortical gyri, asymmetric superior frontoparietal and perirolandic cerebral cortical atrophy, atrophy of the thalamus and caudate, pallor of the substantia nigra, tau-immunopositive neurons. There are data that show swollen neurons are a hallmark of CBD.

The clinical spectrum of CBD is diverse and the diagnosis is challenging. The most common symptom at initial presentation is "clumsy, stiff, or jerky arm". The arms are usually rigid and apraxic. The key feature of CBD is parkinsonism followed by dystonia. Other clinical features are postural instability and gait disturbances, speech disturbances. There is no laboratory or neuroimagistic test to confirm the diagnosis of CBD.

No causal treatment is nowadays available for CBD. Motor symptoms respond poorly to dopaminergic medication.

FABRY DISEASE: DIAGNOSIS, MANAGEMENT AND PHYSIOPATHOLOGY

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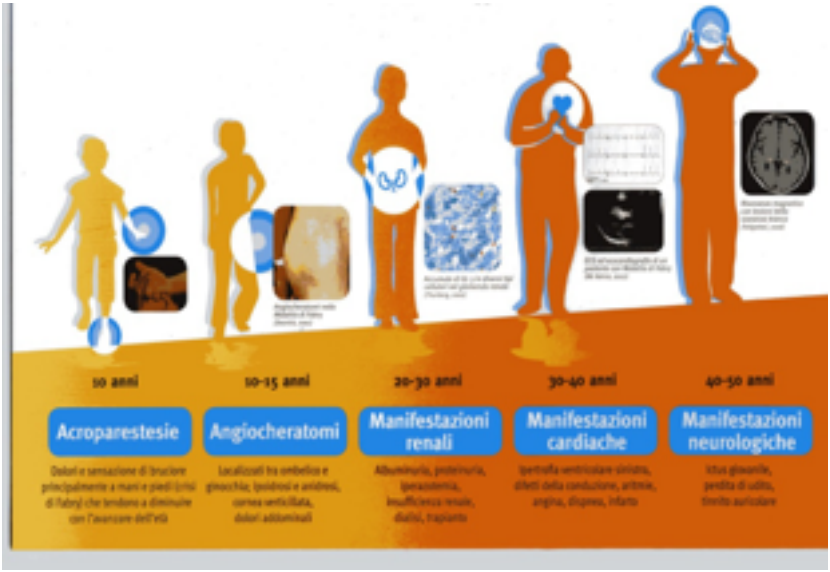
Fabry disease is an X-linked, lysosomal storage disorder caused by a mutation in the GLA gene leading to a deficiency in alpha-galactosidase A enzyme (α-Gal A) activity, which in turn results in accumulation of globotriaosylceramide in the vascular endothelium and smooth muscle cells of different organs, including kidney and heart, finally leading to impairment or failure of organ function.

The central and peripheral nervous systems are also affected leading to neurological manifestations such as cerebrovascular diseases, small fiber neuropathy (SFN), and dysautonomic disorders that may be the presenting clinical features in a proportion of patients.



The classic form of the disease, presenting in males with no detectable α-Gal A activity, is characterized by angiokeratomas, acroparesthesia, hypohidrosis, corneal opacity in childhood or adolescence and progressive vascular disease of the heart, kidneys and central nervous system. In contrast, patients with mild forms of Fabry disease and residual α-Gal A activity are usually asymptomatic until late in adulthood. Their clinical manifestations are often limited to heart and kidneys. In both cases, a single point mutation is sufficient to produce FD, affecting correct secretion of the protein at the active site, as well as the folded state of the molecule. In female carriers, a higher incidence of stroke has been described.

Conventional management consists of pain relief with analgesic drugs, nephroprotection (angiotensin converting enzyme inhibitors and angiotensin receptor blockers) and antiarrhythmic agents, whereas dialysis or renal transplant are available for patients with end-stage renal failure. With age, progressive damage to vital organ systems may lead to failure. With respect to the general population, end-stage renal disease and life-threatening cardiovascular or cerebrovascular complications limit the life-expectancy of untreated males and females by 20 and 10 years, respectively . Early detection of organ damage is important because any treatment is more successful before irreversible structural damage

Enzyme replacement therapy (ERT) and pharmacological chaperone therapy (PCT) were recently introduced. The former is based on intravenously administered α-galactosidase A, which is taken up by cells and tissues by the mannose-6-phosphate receptor pathway and delivered to lysosomes.

Here we will report the clinical aspects of the different forms, with particular interest on the neurologic symptoms and on their pathophysiologic mechanisms, and the strategies of symptomatic and pathogenetic treatments.

POMPE DISEASE (ACID ALPHA GLUCOSIDASE DEFICIENCY): DIAGNOSIS, MANAGEMENT AND PATHOGENESIS

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Pompe disease (OMIM606800) is a lysosomal storage disease characterized by deficiency of the enzyme acid alpha-glucosidase leading to myopathy, respiratory weakness, physical disability and premature death. The symptoms manifest as a continuum from birth through to adulthood, with a recognized severe infantile-onset form that is associated with cardiomyopathy and high mortality, to late-onset forms that are primarily characterized as weakness of limb-girdle and respiratory muscles but usually without cardiomyopathy. Although the overall understanding of the disease has progressed, the pathophysiology of muscle damage remains poorly understood. Lysosomal enlargement/rupture has long been considered a

mechanism of relentless muscle damage in Pompe disease. In past years, it became clear that this simple view of the pathology is inadequate; the pathological cascade involves dysfunctional autophagy, a major lysosome-dependent intracellular degradative pathway. The autophagic process in Pompe skeletal muscle is affected at the termination stage-impaired autophagosomal-lysosomal fusion. Yet another abnormality in the diseased muscle is the accelerated production of large, unrelated to ageing, lipofuscin deposits-a marker of cellular oxidative damage and a sign of mitochondrial dysfunction. The massive autophagic buildup and lipofuscin inclusions appear to cause a greater effect on muscle architecture than the enlarged lysosomes outside the autophagic regions. Furthermore, the dysfunctional autophagy affects the trafficking of the replacement enzyme and interferes with its delivery to the lysosomes.

Here we will describe the different clinical forms , with particular interest to the late onset forms, the diagnostic tools and the therapeutic approach including diet, physiotherapy and enzyme replacement therapy.

We will also report the data of a recently published guideline on the diagnosis and management (Tarnopolsky et al, 2016).

THE ART OF NEUROLOGICAL EXAMINATION

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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.

PHARMACOLOGY IN NEUROREHABILITATION

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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 should be avoided because they are known to impair brain repair mechanism.

This lecture will address the following issues:

1. A general pharmacologica overview on drug influence on brain recovery

2. It is of high 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.

3. The current status of drugs to influence states of diminished consciousness wiil be given

4. 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.

5. Most recent data from a published multicentre trial treatment on the use of neurotrophic factors in combination with rehabilitative treatment in postacute stroke will be presented (CARS trial)

SENSE AND NON-SENSE OF PHYSICAL MEDICINE IN NEUROREHABILITATION

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This lecture will summarize the most important physical therapeutic techniques used in neurorehabilitation and discuss their differential clinical usefulness for special patients`problems.

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

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

RESTORATIVE NEUROLOGY- DO WE ASK THE RIGHT QUESTIONS?

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Over the last two decades there has been a remarkable change in our thinking in the invention, design and efficacy evaluation of motor therapies in neuro-rehabilitation which can be described by three paradigmatic changes:

First there is a change from confession to profession i.e. more and more evidence based approaches rather than intuitively driven procedures have come into use.

This was accompanied by a change from "hands on" treating to "hands off" coaching approaches, which now dominate most of the evidence procedures. This change in treatment philosophy has had a marked impact also on the self-understanding of the therapists in their relation to the patient mutating from treaters to teachers .

Thirdly these developments were accompanied by a transition from intuitevely marshaled individual one to one treatments to quality proven group treatments.

In neurological rehabilitation the distinction between treatment strategies

targetted

to restore function and thereby decrese impairments contrasted to approaches to compensate function in order to improve activities is becoming more and more important.

We certainly have to ask ourselves if we really have addressed the right questions to bring the field forward.

Especially in the early postacute stage within a limited therapeutic time window (e.g. ca 3 months in stroke) restorative approaches are aimed to decrease impairment .This approach probably implies very time intensive (e.g. up to 8 hours a day multifacetted treatments. We must admit that the repertoire for impairment oriented treatment approaches still is rather limited.

So far only three major strategies have been shown to help decrease impairment in the subacute stage e.g. after stroke: The forced use or constraint induced movement therapy approach has been proven to be effectve in the multicenter prospective EXCITE trial (Wolf et al 2008)). Also the use of antidepressant agents was shown to be effective in the FLAME trial (Chollet et al 2011) . Very recently the CARS trial (Mureșanu et al 2016) documented for the first time after decades of frustrane attempts to achieve some sort of neuroprotective and/or neurorestorative effects that a mutimodal drug can improve impairment after stroke .

Possible additional candidates for a true „impairment" oriented treatment approach are neuromodulatory techniques such as peripheral neuromuscular and/ or sensory stimulation (eg. whole hand subliminal „mesh-glove" stimulation)and more and more also non invasive brain stimulation techniques such as repetitive transcranial magnetic stimulation and transcranial DC stimulation. Also the use of non fatiguable robotic devices to enable a high intensity massed movement treatment appear promising.

Probably the most important impact in facilitating impairment reduction will however have clever ,economically feasible, approches to increase the net number of therapy or activity hours per day by creating true „ enriched environment" for severely impaired patients . They should enable 6-8 hours of daytime treatment to avoid leaving our patients „inactive and alone" in future.

WEARING OFF, DYSKINESIA, AND THE USE OF CONTINUOUS DRUG DELIVERY NOW AND IN THE FUTURE'

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Dopamine replacement therapy in Parkinson's disease (PD) is highly effective in reversing motor symptoms in the early stages of the illness but with continuing drug treatment and disease progression, motor fluctuations ('wearing off') and motor complications (dyskinesia) become increasing prevalent and affect most of the patient population. To overcome these problems, two strategies can be adopted – either the avoidance of their onset or their reversal – both require an understanding of why motor fluctuations and complications occur and how the outcome is affected by the way in which drug treatment is administered.

'Wearing off' is a centrally mediated pharmacodynamics phenomenon that does not involve any change in peripheral L-dopa kinetics. It appears to be largely due to loss of presynaptic storage of L-dopa/dopamine in remaining striatal dopaminergic terminals but there is also an alteration in the motor response to the drug that relates to loss of its long duration response (LDR) and exposure of the pulsatile short duration response (SDR). However, since 'wearing off' can also occur in response to dopamine agonist treatment, a post-synaptic component is also involved but this is poorly understood.

Dyskinesia is a reflection of the extent of nigral dopaminergic cell degeneration and resulting changes in post-synaptic dopamine receptor transduction mechanisms linked to the way in which drug exposure occurs. There is also evidence that non-physiological formation and release of dopamine from serotoninergic neurons may contribute to dyskinesia induction. Dopamine agonist drugs produce less dyskinesia than L-dopa and it is claimed that shorter acting pulsatile drugs may produce less dyskinesia than longer acting compounds that result in continuous dopaminergic stimulation – however, this is not entirely true. What is certainly true is that more continuous drug delivery results in less dyskinesia whether this concept is applied to the administration of L-dopa (DuoDopa) or to dopamine agonists, notably apomorphine.

The use of continuous drug delivery in the later stages of PD has been shown to be highly effective in decreasing 'off' time and improving quality of life in those patients with marked 'wearing off' where oral therapy has proved inadequate. This is perfectly logical as the constant delivery of drug maintains levels of dopamine or dopamine receptor stimulation in the striatum that result in expression of voluntary movement. However, in patients with established dyskinesia, the continuous delivery of apomorphine by subcutaneous infusion or the direct intraduodenal

delivery of L-dopa (DuoDopa), results in a decline in the intensity of involuntary movements with time. In theory, the use of continuous drug delivery with L-dopa in early PD should prevent the occurrence of 'wearing off' and dyskinesia induction compared to its oral use, but this has yet to be tested in man. There may also be a reduction in impulse control disorders and some non-motor symptoms of PD.

At this time, the continuous drug delivery techniques that are available are reliant on somewhat invasive technologies but in development are a new generation of delivery strategies for achieving constant drug levels. These range from new routes of administration (sublingual, nasal, pulmonary, transdermal) to a new generation of delivery technologies, to the use of gene therapies and cell technologies that reinstate dopamine production in the basal ganglia in PD.

At this time, continuous drug delivery offers a way forward in the treatment of components of the later stages of PD that is not provided by standard oral therapy. It allows the 'best' drugs for the treatment of PD to be used in a way that expands their therapeutic window in this patient population. In the future, advances in drug delivery in PD will allow these approaches to be used in patients with earlier disease and might eventually become common practice when initiating treatment.

DISEASE COURSE MODIFICATION IN PARKINSON'S DISEASE

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Personalized medicine is an emerging field that encompasses the use of risk algorithms, molecular diagnostics, targeted therapies and pharmacogenomics in order to improve health care. It is expected to impact the way drugs are developed and patients are treated in many fields, including neurodegenerative diseases in the near future.

Parkinson's disease (PD) is the second most common neurodegenerative disease in man and its clinical hallmark is the motor parkinsonian features, namely rest tremor, bradykinesia, rigidity and loss of postural reflexes; These symptoms, resulting from the loss of dopaminergic neurons in the substantia nigra pars compacta, respond well to dopamine replacement therapy; The limitation of dopaminergic therapy is that patients soon develop motor fluctuations, shortening and loss of stability and predictability of the response as well as drug-induced involuntary movements termed dyskinesias; additionally they do not provide benefit for the multiple nonmotor symptoms affecting most patients' lives and decreasing patients' quality of life. Moreover they do not slow down disease progression with evolution of cumulative widespread neurological disability.

In this review we will outline the applications of personalized medicine for the

several stages from at risk populations to full-blown advanced PD.

We expect to change the way we currently define PD with molecular diagnostics, the use of DNA-, protein- or mRNA-based biological markers to predict the risk for developing PD as well as the molecular phenotype of ongoing PD through its various stages. Genomic analysis of diseases with homogeneous clinical phenotypes will unveil distinct molecular entities that require different treatment strategies for optimal outcomes. Furthermore molecular-targeted therapies that slow degeneration of both dopaminergic and non-dopaminergic neurons will replace those that simply treat PD symptoms, providing long-term disease course modification. Finally, pharmacogenomic data that predicts therapy response and limitations in the individual patient based on his genomic profile will accompany many drugs.

TRANSCRANIAL MAGNETIC STIMULATION - PRINCIPLES, TECHNIQUE, APPLICATIONS

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Transcranial magnetic stimulation is a useful neurophysiological technique that investigates the central nervous system, mainly the central motor pathways. It is used as a diagnostic tool, but also in research, therapeutics and neurorehabilitation. The method appeared 30 years ago, and has developed intensively throughout the world, so that nowadays a lot of scientific knowledge has been gathered. This presentation will try to describe the method, its physical and biological principles, and to show its major indications in clinical situations, as well as other more complex approaches regarding the central nervous system function in normal and pathological conditions.

BRAIN PLASTICITY AND NEUROREHABILITATION IN PARKINSON'S DISEASE

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Neurological disorders, especially degenerative diseases, represent a leading cause of long term disability all over the world. Many advances have been done

in the treatment of these pathologies. The need to identify therapeutic methods, able to limit brain damage or enhance recovery of motor and cognitive functions through neuroprotective and neurorestorative mechanisms, is desirable. There are many animal and human studies trying to elucidate the cellular and molecular mechanisms of plasticity of the nervous system. Neurorecovery is the positive outcome that produces clinically relevant results with immediate functional and late structural effects.

Neurorecovery depends on the adaptive plasticity of the undamaged nervous tissue, and of the non-affected elements of functional network. This process can be enhanced by pharmacological intervention, physical and cognitive activity, electromagnetic stimulation, psychological support, environmental stimulation or any demonstrated combinations of these factors capable of improving the patient's condition.

A better understanding of the mechanisms underlying the neuroplasticity will reflect in a more efficient and comprehensive treatment. This presentation will focus on the role these mechanisms in Parkinson's disease, and will give a brief overview on current neurorehabilitation procedures in this complex condition.

COGNITIVE REHABILITATION AFTER STROKE

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Cognitive impairment represents a common complication after stroke that include executive, attention, memory, visual-spatial perception dysfunction. Difficulties in speech and language are also associated cu impaired cognitive abilities, and cognitive training is more and more used as an add-on strategy to other specific rehabilitation procedure such as motor rehabilitation. Actually, one of the principles of cognitive neurorehabilitation is to combine specific interventions for motor function with a cognitive-behavioral intervention. The rationale behind this approach is based on the dynamic interconnectivity among the brain circuits involved in motor execution, executive function, attention and memory.

This presentation will focus on the principles of homeostatic mechanism that modulates all three levels of brain's organization: cellular/molecular, local circuitry and network level.

The concept of endogenous neuromodulation refers to the brain's capacity to balance anti-correlated processes, such as pro-survival signaling mechanisms versus pro-death signaling mechanisms at the cellular and molecular level, long-term potentiation versus long-term depression at the local circuit level, synchronization versus desynchronization at the dynamic network level. Every

level in turn comprises several sublevels, each of which is characterized by a multitude of anti-correlated processes.

Brain networks strength is determined by the capacity of neuronal groups to fire synchronously, modulated by synaptic communication and by resting membrane potential, which are determined by the expression of genes tightly linked to neurotransmitters and ion channels activity. This crosstalk between genetic and neuronal networks is staring to be increasingly more studied in neurological and psychiatrically pathologies; recent data showed that stroke imbalances the miRNA-genes network leading to alteration of the processes regulated by targetgenes such as MAPK signaling pathway, with important consequences upon inflammation, oxidative stress and neuroprotection .

Recent data support the idea of inter-correlation between molecular/cellular level and network level showed that an ischemic lesion, even of small dimensions, may trigger a progressive molecular disorganization of axons, even at a distance from the infarct core, possibly incriminating mechanisms being widespread inflammation and neuro-vascular unit (NVU) dysfunction.

The presentation will also highlight the current status on evidence based interventions in preventing and treating post-stroke cognitive impairment.

MOTOR REHABILITATION AFTER STROKE

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Brain damage affects all three levels of structural and functional organization: cellular and molecular level, circuitries level and dynamic network level and launches an endogenous continuous brain defense response which consists in neuroprotection (the immediate response) and neurorecovery (a later response).

Endogenous neuromodulation represents at the cellular and molecular level the optimization of common biological processes that could potentially generate cell death or promote neurodegeneration. At the circuitries and dynamic network levels, it represents the tendency in reinbalancing of functional connectivity in resting-state netwoks.

In the last years, there has been a substantial effort in understanding the brain functioning and how to enhance endogenous neuromodulation and neurorehabilitation in general, by using a large spectrum of neurotechnologies such as imaging techniques (functional magnetic resonance imaging, ligant-based positron emission tomography, diffusion-tensor imaging), quantitative electroencephalogram, magnetoencephalography, eye tracking, optogenetics, transcranial magnetic stimulation, transcranial direct current simulation, deep

brain simulation, computational neuroscience and brain-computer interfaces. The combination between these technologies provide valuable information about the structure-function relationship underling resting-state networks, about the dynamic cross-talk between networks and about the abnormalities in the functional connectivity in different pathologies.

Neurorecovery can be enhanced by pharmacological intervention, physical activity, electromagnetic stimulation, psychological support, environmental stimulation or any demonstrated combinations of these factors capable of improving the patient's condition after brain and spinal cord injuries. From the pharmacological perspective, it is clear that the focusing on molecules that are capable of mimic the function of endogenous molecules with multimodal and pleiotropic neuroprotective effects is the best approach in neurorecovery, especially when they are associated with intensive physical training.

Biological agents (e.g., neurotrophic factors and related molecules) with modulating and multimodal effects are better pharmacological agents for brain and spinal cord protection and recovery, because they usually have also pleiotropic neuroprotective effect. That is why they are capable of pharmacologically bridging acute neuroprotective processes with the long-term recovery processes.

There are many animal and human studies trying to elucidate the cellular and molecular mechanisms of plasticity of the nervous system. A better understanding of the mechanisms underlying the neuroplasticity will reflect in a more efficient and comprehensive treatment.

This presentation will focus on therapeutic effects of main interventions in neurorecovery after stroke.

BRAIN-COMPUTER INTERFACE IN NEUROREHABILITATION IN POST-STROKE PATIENTS

CATERINA PISTARINI
IRCCS Salvatore Maugeri Foundation, Pavia, Italy

A brain-computer interface (BCI) is a system that decodes brain signals generated by the user allowing specific commands ,to be executed on an external device. Therefore such an interface would enable severely disabled people to interact with their environment without the need for any muscle activation

Since the first electroencephalography (EEG) on humans, there has been much speculation about the possibility of reading thoughts and of using the brain to control devices.

It was not until the late 1980s and early 1990s that significant research activity started in this field. Since then, there has been a continuous increase in the research aimed at signal acquisition and processing, and at medical applications.

A search of the PubMed database for “brain computer interface” provided :

- 2 publications before 1993- 5 for the 1993-1998 period 38 for 1998-2003 - 357 for 2003-2008.

The input signal for a BCI system cannot be simply the EEG signal at rest. At least two different states are needed to operate an external device. Thus a cognitive task assigned to the user produces a signal containing features that are extracted and classified. Different cognitive tasks can be used to produce such features.

The operational framework used to specify them is called the paradigm.

Motor Imagery can be defined as a dynamic state during which a given action is mentally simulated by a subject. The subject can implement two different techniques: “first person perspective”, or motor-kinesthetic, and “third person”, or visuo-spatial perspective.

EEGs show event-related potentials(ERP) in response to some stimuli. Traditionally, such potentials are extracted from the EEG by presenting the stimulus repeatedly, following the neurophisiological standardized methods.The resulting waveform presents peaks of different amplitude at different latencies: the P300 component is a positive peak with a latency of about 300 ms.

Given the structure of BCI systems, it is easy to understand how BCIs can work as assistive devices.

BCIs can be used for communication , as wheelchair controllers in real or virtual environments, and to operate in invasive or non-invasive settings (i.e. pts with tetraplegia.BCI can also help recover some lost abilities.

Experimental evidence has shown that the brain is not rigidly hardwired because it has cortical plasticity, and cortical plasticity can be stimulated by learning processes.

Moreover, studies on synaptical plasticity are shedding new light on the relationship between cortical reorganization and rehabilitation, showing that rehabilitation can actually modify cortical circuitries.

Two ways of using BCI in clinical rehabilitation

1. One way is related to the use of biofeedback for controlling and modulating brain signals.
2. The other uses BCI to control an external device, like a robotic arm, to provide sensory input that can help to normalize motor control. Both approaches are based on the BCI feature : BCIs require skilled users, able to modulate their brain activity and should give such users real time feedback.

Since one of the BCI paradigms is based on the P300 potential related to cognitive events, BCI could also be used for cognitive rehabilitation. People affected by brain injury or disease often experience cognitive problems , which can seriously affect their quality of life.

While there is substantial evidence of the efficacy of cognitive therapies concerning stroke and traumatic brain injury.

In cognitive rehabilitation, the use of event-related potentials is traditionally

limited to an assessment of injuries incurred or disorder severity.

Use of BCI systems for behaviorally nonresponsive patients is a substantial technical and clinical challenge

Lack of a priori knowledge about their level of conscious awareness, cognitive capacities, and communicative intent. Multisensory feedback plays an important role in motor learning by re-establishing the sensorimotor loop that is disrupted after SN damage but before adapting a BCI in a patient with DoC, the first step would be to establish that they are able to follow commands with adequate consistency.

ADVANCED THERAPY FOR PARKINSON'S DISEASE

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Colentina Clinical Hospital Bucharest,
'Victor Babes' National institute of Pathology

Parkinson's disease (PD) is the second most prevalent neurodegenerative disease, after Alzheimer's disease. However, from all neurodegenerative entities it remains the best treated one. In the first years after clinical diagnosis, classical treatment is able to compensate the typical parkinsonian signs, but in advanced disease a specialized team is needed to choose the best therapeutic approach for the patient in order to offer the best quality of life possible. In this paper I will present the current options for treating the advanced PD, current selection criteria, efficiency and side effects of each of the advanced treatments available (levodopa/carbidopa intestinal gel, deep brain stimulation and apomorphine).

CLINICAL CASES OF ADVANCED PARKINSON'S DISEASE – TREATMENT OPTION AND PATIENT MANAGEMENT

BOGDAN O. POPESCU
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Colentina Clinical Hospital Bucharest,
'Victor Babes' National institute of Pathology

The possibility to deliver an intervention to continuously interfere with disturbed basal ganglia circuits in advanced PD patients is the target of all experienced movement disorder specialists. In this session I will present advanced PD cases which challenge the medical decision, including the proposal of continuous therapy and combination of therapies.

NEUROPROTECTION AND NEUROTOXICITY OF NANOPARTICLES IN THE CENTRAL NERVOUS SYSTEM WITH SPECIAL REFERENCE TO NANOMEDICINE

HARI SHANKER SHARMA[1]

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Nanoformulation enhances neurotherapeutic values of drugs or neurodiagnostic tools as compared to their parent compounds. However, effects of nanoparticles per se in inducing neurotoxicology, if any is still being largely ignored. Thus, role of nanomedicine is to enhance the drug availability within the central nervous system (CNS) for greater therapeutic successes. However, once the drug-nanoparticle complexes enter into the CNS, the fate of nanomaterial is largely unknown. Thus, to achieve greater successes in nanomedicine expanding our understanding of nanoneurotoxicology is the need of the hour. In our studies, we observed that intoxication of nanoparticles e.g., Ag, Au, Cu, Al, SiO2, single walled carbon nanotubes (SWCNTs) administered systemically in rats or mice induces neurotoxicity e.g., disruption of the blood-brain barrier (BBB), development of brain edema and neuronal, glial, axonal and endothelial cell damages. Furthermore, when additional traumatic brain or spinal cord injuries inflicted in these animals the magnitude of brain pathology was enhanced by 150 to 300 % depending on the type of nanoparticles used. Ag, Cu and SiO2 nanoparticles exhibited the most marked exacerbation of brain pathology following injury. In such situations, neuroprotective agents if given either in double doses or administered through nontechnology, e.g., TiO2 nanowired delivery achieved better neuroprotection than the parent compound given alone. These observations clearly indicate that nanomedicine is the need of the hour to induce clinical benefits in situation with co-morbidity factors e.g., nanoparticles exposure after central nervous system (CNS) injury.

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PATHOGENESIS, DIAGNOSIS AND RISK STRATIFICATION ALGORITHM FOR NATALIZUMAB-ASSOCIATED PROGRESSIVE MULTIFOCAL LEUKOENCEPHALOPATHY

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The ever increasing therapeutic choices -with significantly increased efficacy- available in the treatment of multiple sclerosis patients brings in the front of medical interest and concern the long term safety issue in the decision balance.

In line with this approach, understanding, recognizing and treating progressive multifocal leukoencephalopathy (PML) as a possible serious complication of MS therapies-Natalizumab but not exclusively , has become mandatory.

Progressive multifocal leukoencephalopathy (PML), a rare but potentially disabilitating and even deadly demyelinating disease of the brain is caused by the infection of oligodendrocytes by the neuropathogenic form of JC polyomavirus (JCV) . In the general population there is a high prevalence of chronic but asymptomatic JCV infection, therefore increased odds that a multiple sclerosis patient belongs to this group is expected . Transformation of the non pathogenic virus into the neuropathogenic form which is mandatory to cause PML, although with a rather low incidence, was firstly described in individuals with a compromised immune system (hematologic malignancies , HIV, post transplant therapy etc.) but lately (since 2005) also in association with MS therapies- Natalizumab being the first and most frequently incriminated up-to-date.

Strict criteria are required to definitely diagnose the disease among which the neuropathologic features consisting of a triad of demyelination, bizzare astrocytes and enlarged oligodendroglial nuclei along with documenting the presence of the JC virus with various techniques. Still, there are clinical and imaging features that along with the presence of the JC virus by PCR (polymerase chain reaction) in CSF (cerebrospinal fluid) may document the disease in the absence of any other reasonable explanation.

Since 2012 a risk algorithm for natalizumab –associated PML has been established taking into account the JC virus antibody status, therapy duration (with

a cutoff at 2 years)and prior exposure to immune suppressive therapy . This risk assessment has evolved , a new Natalizumab patient Management Plan being currently used as endorsed by the European Medicines Agency (EMA).

The presentation summarizes the current knowledge about JCV virology, PML pathogenesis, diagnosis and risk management for PML.

Keywords: Progressive multifocal leukoencephalopathy, JC polyomavirus, demyelination, natalizumab-associated PML , risk assessments for PML

CO-ULTRAMICRONIZED PALMITOYLETHANOLAMIDE/LUTEOLIN PROMOTES OLIGODENDROCYTE DEVELOPMENT, PRECURSOR CELL SURVIVAL AND IMPROVES OUTCOME IN EXPERIMENTAL AUTOIMMUNE ENCEPHALOMYELITIS

STEPHEN SKAPER
LAURA FACCI, MASSIMO BARBIERATO, GABRIELLA CONTARINI, CARLA MARINELLI, PIETRO GIUSTI
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Oligodendrocytes, the myelin-producing cells of the central nervous system are responsible for ensheathment of axons. Oligodendrocytes have limited ability to repair damage either to themselves or to other nerve cells as in multiple sclerosis (MS), a chronic neuroinflammatory demyelinating disorder of the central nervous system with a strong neurodegenerative component. MS lesions are characterized by the presence of a diminished pool of undifferentiated oligodendrocyte precursor cells (OPCs) which are unable to mature into myelin-producing oligodendrocytes. In such settings, an important strategy may be to replace the lost oligodendrocytes and/or promote their maturation or proliferation. N-palmitoylethanolamine (PEA), an endogenous fatty acid amide signaling molecule possesses analgesic, anti-inflammatory, and neuroprotective actions. Recent studies show a co-ultramicronized composite of PEA and the flavonoid luteolin (co-ultraPEALut, 10:1 by mass) to be more efficacious that PEA alone in improving outcome in experimental models of spinal cord and traumatic brain injuries. Here, we examined the ability of co-ultraPEALut to promote the progression of OPCs into a differentiated phenotype. OPCs were isolated from newborn rat cortical mixed glial cell cultures and maintained under conditions which favor either differentiation (Sato's medium) or proliferation (serum-free medium with fibroblast growth factor-2 and platelet-derived growth factor-AA ('SFM')). When maintained in Sato's medium co-ultraPEALut (10 μM) treatment of OPCs stimulated, in a time-sependent manner their morphological development, total protein content and gene expression for the major structural myelin proteins myelin basic protein (MBP) and proteolipid

protein, the enzyme 2',3'-cyclic nucleotide 3'-phosphodiesterase (thought to mediate process outgrowth in oligodendrocytes and play a critical role in the events leading up to myelination), as well as genes coding for enzymes involved in cholesterol and fatty acid synthesis and antioxidant defense (catalase). Under these conditions, co-ultraPEALut also increased the content of MBP at the protein level. OPCs, maintained in an undifferentiated state (SFM) displayed improved survival capability in the presence of co-ultraPEALut and down-regulation of Apoe, whose deletion reportedly leads to a later time of peak symptoms/disease severity and less severe demyelination/axonal damage in myelin oligodendrocyte glycoprotein (MOG35-55)-induced experimental autoimmune encephalomyelitis in female C57BL/6 mice. Importantly, co-ultraPEALut improved the clinical score in this experimental autoimmune encephalomyelitis mouse model, which is often used as a chronic monophasic model of MS. Hence, strategies intended to promote endogenous remyelination in MS patients should focus on both enhancing the long-term survival of OPCs and on stimulating these cells to proliferate and differentiate into remyelinating oligodendrocytes. Within this context, co-ultraPEALut may represent a novel pharmacological approach.

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MANAGEMENT OF ADVANCED PARKINSON'S DISEASE: CDS THERAPIES - LIMITATIONS AND UNANSWERED QUESTIONS (HOW EARLY CDS THERAPIES SHOULD BE INITIATED?)

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Parkinson's disease (PD) is one of the most important, increasingly prevalent and progressively 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 disease progression, as this changes inevitably lead to predictable and unpredictable response fluctuations, both motor and non-motor. Motor fluctuations and dyskinesias affect almost all patients with PD at some point during the disease course, with major implications in global health status. 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). 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. In spite of undisputable improvements during the last years, many patients remain significantly disabled, and a fully satisfying management of motor complications is still an important unmet need of PD therapy.

MULTIPLE SCLEROSIS BEFORE, DURING AND AFTER PREGNANCY

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Multiple Sclerosis (MS) is more frequent in young adults and more frequent in women. This leads to the common situation of a young female patient diagnosed with MS asking us a lot of questions regarding the possibility to have a baby, about the way of giving birth, breastfeeding, risk of inheritance of the disease by her child and many other questions related to pregnancy. Data available so far do not reveal a negative long-term impact of pregnancy on MS progression. During pregnancy a certain protection against relapses occurs, but this is not always true. Since several therapeutic options have been implemented with good efficiency in the disease stabilization, increasingly more patients begin to wonder about the possibility of having a child and about the possible risks of pregnancy. IFN-b and Glatiramer acetate do not expose patients and their babies to relevant adverse events; nevertheless, these drugs should be discontinued during pregnancy and before conception. In recent years, many studies and reviews have been published addressing the most relevant issues related to MS and pregnancy, with particular reference to the use of disease-modifying therapy currently used for the treatment of MS. The decision of having a baby must be taken after a complete evaluation of the patient, preferably in a stable period of the disease, and a team with a good experience formed by a neurologist and a gynecologist should carefully monitor the patient before, during and after pregnancy.

Keywords: pregnancy, multiple sclerosis, disease-modifying therapy, relapse-rate

CURRICULUM VITAE

ANAT ACHIRON
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Director, Multiple Sclerosis Center
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Anat Achiron, MD, PhD, is a full Professor of Neurology at the Sackler School of Medicine, Tel-Aviv University and the founder Director of the Multiple Sclerosis Center at the Sheba Medical Center, Tel-Hashomer, Israel, which combines holistic multidisciplinary approach targeted to the diagnosis, treatment and rehabilitation of patients with multiple sclerosis.
Prof. Achiron's research interests are within the fields of neuroimmunology, neuroimaging and cognitive function in multiple sclerosis. She has extensively studied multiple sclerosis to better understand disease-related mechanisms using gene expression technology and characterized cognitive performance especially in the very early stages of the disease. Prof Achiron was involved in research studies evaluating genetic markers associated with the diagnosis of multiple sclerosis, various disease types and prediction of disease activity and treatment response and currently is developing a new molecule for better targeted treatment of multiple sclerosis.
Prof. Achiron has published widely, with over 200 publications to her name; she has received numerous grants and scientific awards for her research work in medicine and neurology.

ANDREA ANTAL
GERMANY

Academic education

2005	Habilitation, Cognitive Neuroscience, Georg August University Göttingen
1998	PhD, Biological Sciences, Albert Szent-Györgyi University (Hungary)
1993	Dr. univ., Comparative Physiology, József Attila University of Szeged, (Hungary)
1990	Graduation in Molecular Biology and Biotechnology, József Attila University of Szeged (Hungary)

Employment Since 2010
Extraordinary Professor, Dept. of Clinical Neurophysiology, University Medical Center, Göttingen

2001 - 2010	Group Leader, Department of Clinical Neurophysiology, University Medical Center, Göttingen
1994 - 2001	Assistant Professor of Physiology, Department of Physiology, University of Szeged (Hungary)
1994	Postdoctoral Fellow, Department of Neurology, State University of New York, Health Science Center at Brooklyn, New York (U.S.A)
1993 - 1994	Postdoctoral Fellow, Department of Internal Medicine, University of Nebraska Medical Center (U.S.A)
1991 - 1992	Research Fellow, Department of Neurology, Mount Sinai Medical Center, New York, U.S.A
1990 - 1991	Research Fellow, Department of Comparative Physiology, József Attila University of Szeged (Hungary)

Research areas (present and past):
Transcranial stimulation, Neuroplasticity, Neurodegenerative disorders, Parkinson's disease, chronic pain, Migraine

Scientific	activities (selected)
Since 2015	Full member of the Göttingen Graduate School for Neurosciences and Molecular Biosciences (GGNB)
Since 2009	Member of the Program Committee / Examination Board of the 'Sensory and Motor Neuroscience' PhD program
Since 2008	Organiser of a three - day's workshop on Magnetic and electrical stimulation, every year sponsored by the German Neuroscience Society (NWG)
Since 2008	Associate member of the Göttingen Graduate School for Neurosciences and Molecular Biosciences (GGNB)

Ten most important papers related to the scientific activities:
Alekseichuk I, Diers K, Paulus W, Antal A. Transcranial electrical stimulation of the occipital cortex during visual perception modifies the magnitude of BOLD activity: A combined tES-fMRI approach. Neuroimage. 2015:01056-3.

Pisoni A, Turi Z, Raithel A, Ambrus GG, Alekseichuk I, Schacht A, Paulus W, Antal A. Separating recognition processes of declarative memory via anodal tDCS: boosting old item recognition by temporal and new item detection by parietal stimulation. PLoS One. 2015 10:e0123085

Turi Zs, Mittner M, Opitz A, Popkes M, Paulus W, Antal A. Transcranial direct current stimulation over the left prefrontal cortex increases randomness of choice in instrumental learning. Cortex, 2015; 63C:145-154.

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Moliadze V, Antal A, Paulus W. Boosting brain excitability by transcranial high frequency stimulation in the ripple range. J Physiol, 2010, 588: 4891-904.

Terney D, Chaieb L, Moliadze V, Antal A, Paulus W. Increasing human brain excitability by transcranial high-frequency random noise stimulation. J Neurosci. 2008; 28:14147-55

OVIDIU BĂJENARU

ROMANIA

1983	: M.D. at the Faculty of Medecine of University of Medecine and Pharmacy "Carol Davila" Bucharest
1983-1985	: post graduate hospital stagium in University Hospital of Emergency Bucharest
1985- 1989	: resident of neurology
1985	: assistant professor – University of Medicine and Pharmacy "Carol Davila" Bucharest - Department of Neurology of the University Hospital of Emergency 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 - senior lecturer of neurology - Head of Department and Medical Chief (University Hospital of Emergency, Bucharest
1994 - 1999	: Associate Professor of Neurology
1999 (since)	: Professor of Neurology at the University of Medicine and Pharmacy " Carol Davila" Bucharest and Chairman of the Neurology Department of the University Hospital of Emergency Bucharest
2006:	: Doctor Honoris Causa - University „Ovidius" – Constanta (Romania)
2011	: Director of Department of Clinical Neurosciences - University of Medicine and Pharmacy " Carol Davila" Bucharest
2013 (since)	: Corresponding member of the Romanian Academy of Medical Sciences

Other professional activities :

2000-2004	: Vice-Dean of the Faculty of Medecine - University of Medecine and Pharmacy "Carol Davila" Bucharest
2001-2013	: President(founder) of the Romanian Society of Neurology
2013(since)	: Honorary President ad vitam of the Romanian Society of Neurology
2003-2009	: member of the Scientific Committee of ECTRIMS
2005-2009	: member of the Executive Committee of the European Society of Neurology
2011 (since)	: member of the National Committee of Habilitation of the Romanian Ministery of Education for PhD accreditation and high academic degrees

Post graduate training :

1992 - 1994	: post graduate training in clinical neurology and functional investigations of the nervous system at University " Rene Descartes"(Paris) : C.H.U. Sainte-Anne (Neurology) and C.H.U. Cochin – Port Royal (Functional Investigations of the Nervous System) and training in neuroendocrinology
1996	: second medical competence (confirmed by the Ministery of Health of Romania) in "Diagnosis in Neurological Diseases by MRI".
1997	: assistant of clinical research in pharmaco-clinical trials (Paris)
2009, 2011	: International training for methodology in clinical research

Fields of interest for the scientific research

- dementia and neurodegenerative diseases (in particular Parkinson's disease)
- multiple sclerosis
- stroke
- experimental and clinical study of sleep disturbances in the neurological and neuroendocrinologic diseases

- more than 450 scientific papers published and reported in different national and international scientific meetings
- ISI Web of Science: h-index : 8
- 5 medical books and monographies (published in Romania)
- co-author (1 chapter) to the "International Neurology - A Clinical Approach" (eds. ROBERT P. LISAK, DANIEL D. TRUONG, WILLIAM CARROLL, ROONGROJ BHIDAYASIRI), Wiley-Blackwell , 2009
- Country Principal Investigator – in more than 20 international, multicentric clinical trials
- Principal Investigator of the research site – in more than 30 international and national multicentic trials
- Member of the Steering Committee of PRECISE trial

Other activities:

- coordinator of the Continuous Medical Education (EMC) national program of the Romanian Society of Neurology for neurologists in Romania
- coordinator and author of the Guidelines for diagnosis and treatment of neurological diseases (agreed by the College of Medecins of Romania) main author of the national guidelines for Parkinson's disease, Multiple Sclerosis and Dementia
- coordinator of the National Program of the National House of Insurance and Ministery of Health, for treatment of patients with neurological diseases (2000 - 2015)
- coordinator of the first medical team in Romania for DBS in Parkinson's disease.

- chief-editor of Romanian Journal of Neurology (the official journal of the Romanian Society of Neurology)

Scientific affiliation :
- Romanian Society of Neurology (Honoray President ad vitam)
- UEMS – European Board of Neurology (Secretary General – elected in 2010)
- European Neurological Society (ENS) – member of the Executive Committee between 2005 – 2009
- European Stroke Organization
- European Federation of Neurological Societies (EFNS) and European Academy of Neurolgy (since 2014)
- American Academy of Neurology (cooresponding member)
- Danube Neurological Association (Vice-Secretary General – elected in 2011)
- ECTRIMS (member of the Scientific Council 2003-2009)
- New York Academy of Sciences
- American Academy for Advancement in Science
- Movement Disorders Society
- Romanian Association for the Study of Pain
- Romanian Society for the Study of Neuroplasticity (founder president of honour)

2005, 2006, 2010, 2011: awarded by the Prize of Excelence in Neurology for the scientific activity in Romania (decided by a National Jury organized by the Health Chamber of the Romanian Parliament)
2008: awarded by the Romanian Society of Internal Medicine for the best scientific activity in a related medical speciality
2014: awarded by the International Brain Foundation and Romanian Academy of Medical Sciences, for excellency in the development of management of patients with multiple sclerosis in Romania
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

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After graduation in medicine at the University of Cluj Napoca clinical training in internal medicine at the University Hospital Cluj, then, after resettlement in Germany, achievement of clinical training in neurology and neurorehabilitation in Kettwig and of neurophysiology at the Alfried Krupp Hospiltal Essen.

Between 2002 and 2016 head of the early rehabilitation department at the St Mauritius Therapieklinik Meerbusch with focus on disorders of consciousness in severe brain injured patients.

Since 2016 assistant medical director at the Gesundheitszentrum Bad Wimpfen

1994-2002 Collaboration with the University of Essen in the field of plasticity after stroke, with an emphasis on the role of the cerebellum in motoric learning tasks

Since 2002 Collaboration with the University of Düsseldorf in the field of plasticity after stroke

Since 2009 Collaboration with the Coma Science Group Liege Belgium

Member of the DOC special interest group of the IBIA.

NATAN BORNSTEIN
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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)

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)

CRISTIAN FALUP-PECURARIU
ROMANIA

Cristian Falup-Pecurariu is Head of the Department of Neurology, County Emergency Clinic Hospital from Brasov, and is Lecturer of Neurology at the Transilvania University from Brașov, Romania. He received his medical degree from the University of Medicine and Pharmacy "Iuliu Hațieganu" 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.

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). He was also Secretary of the EFNS/MDS-ES Panel on Movement Disorders, member of the Educational Committee of MDS-ES and currently is member of the MDS Leadership Task Force and European Academy of Neurology Scientific Panel Movement Disorders. He is member of EUROPAR (European Parkinson's Group) and International Parkinson and Movement Disorders Society Non motor study group.

He is the initiator and Course Director of the Movement Disorders Teaching Course held in Brasov.

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

ANTONIO FEDERICO
ITALY

Prof. Antonio Federico, born in Polla (Sa) on the 25.08.48, from 1990 is full professor of Neurology at the University of Siena , Director of the Unit Clinical Neurology and Neurometabolic Disease.

He was Director of the Department of Neurological, Neurosurgical and Behavioural Sciences, University of Siena (2002-2008).

He received the degree in Medicine and specialization in Nervous and Mental Diseases, summa cum laude, at the University of Naples in 1972 and 1975 respectively. He received the Lepetit Award for the best degree dissertation in 1972.
His biological training was in the Institute of Biochemistry as student and after in Physiology of the University of Naples, and in the Centre de Neurochimie of CNRS, in Strasbourg, directed by prof. Mandel where he worked in the years 1973-75. He also collaborated with many international research groups, in different countries where he spent in the past years some times: in Montreal (Prof. Andermann, Karpati and Shoudgbridge), in London (dr A. Harding and prof. Morgan-Hughes), in Toronto (dr.Robinson), in Bonn (prof. von Bergmann) , in Paris (dr.Baumann), in Baltimore (proff. Moser and Naidu), in Oxford (prof. Matthews), etc.
His clinical formation was made at the Medical School of the University of Naples, in the Dept, Neurology, and after in Siena, where he moved on 1980 with his mentor, prof. G.C. Guazzi. Associated professor in Neurology in 1982, since 1990 he is full professor of Neurology, Medical School, University of Siena.
In 2013, he received honoris causa degree in Medicine at University Carol Davila, Bucharest, Rumania.

In the years 1990-96 he was Secretary of the Italian Society of Neurology. In the years 2006-08 was President of the Italian Society of Neurology.

He coordinated the Study Group on Clinical Neurogenetics of the Italian Society of Neurology.

He has been referee for projects evaluation in the area of Orphan drugs and Orphan diseases for Biomed Projects from EU, for MURST, CNR and Istituto Superiore di Sanità, and other national and international funding agencies, etc.

He is member of the Second Opinion Group of the American Leucodistrophy Association.

Associated editor of Neurological Sciences in the past 3 years. From 2012, he is Editor-in Chief.

He is author of more than 500 article quoted by Pubmed. He is author of a chapter on Cerebrotendinous Xanthomatosis, Vinken and Bruyn Edts, Handbook of Clincal Neurology, vol 49, Neurodystrophies and Neurolipidoses. On the book McKusick's Mendelian Inheritance in Man,. Ed.1992, Catalog of Autosomal Dominant and Recessive Phenotypes he is cited for 3 different diseases. He was editor of the book Late Onset Neurometabolic diseases (A.Federico, K. Suzuki and N.Baumann Edts), Karger 1991, and many other books from Italian and international Publishing Companies.
Recently he published (2015) Manuale di Neurologia Pratica and Neurologia and Assistenza infermieristica, for students.

His main field of interest is related to neurometabolic, neurodegenerative and rare diseases, investigated from a genetic, metabolic, neuroimaging and clinical point of vue.

Summary of the academic involvements:
- Director of the Section Neurological Sciences, Dept Neurological , Neurosurgical and Behavioural Sciences (2000-2012)
- Director of the Research Center for the Diagnosis, Therapy and Prevention of the Neurohandicap and Rare Neurological Diseases, until the 2010
- Vice-Dine of the Medical School, University of Siena (2003-2006)
- Director of the Postgraduate School of Neurology, University of Siena, from 2006 up to 2014.
- Director of the PhD School in Cognitive and Neurological Sciences, University of Siena (from 2000 up to date)
- Coordinator of the Section of the Univ. Siena of the PhD Program Neurosciences, Univ. Florence.
- Research delegate for the Dept Medicine, Surgery and Neurosciences (2013-)
- Vice-Rector of the University of Siena, from 1st april 2016.

Medical Involvements
- Director of the OU Clinical Neurology and Neurometabolic Diseases, University Hospital of Siena Medical School.
- Director of the Regional Reference Center for Rare Diseases
- Regional Coordinator of the Network for Rare Neurological Diseases, Tuscany Region.
- Member of several Ministry of Health and Regional Committees
  National and International Commitments
- President of the Italian Society of Neurology (2009-11)
- Italian delegate to the World Federation of Neurology
- Italian Delegate to the European Union of Medical Specialists (Section Neurology)
- Italian Delegate and Chairman of the Neuromediterraneum Forum and President
- Consultive Member of the European Brain Council
- Editor – in – Chief of Neurological Sciences, Springer Verlag Editor. He is in the Editorial Board of many national and international journals.

- Member of the American Panel United Leucodystrophies.
- Member of the Scientific Committee of AISM (Associazione Italiana Sclerosi Multipla)
- Chairman of the Scientific Committee of the European Academy of Neurology
- Chairman of Neuromediterraneum Forum
- Co-Chairman of Research group of WFN Migration Neurology

Member of the Scientific Societies:
- Società Italiana di Neurologia (Past Secretary, President, Past-President and Member of the Committee)
- Society for the Inborn Errors of Metabolism
- Italian Association of Neuropathology
- SINDEM (Italian Association of Dementias)
- Italian Association for Parkinson's disease
- Italian Association of Neurogeriatrics (Member of the Scientific Committee)
- Italian Stroke Forum
- European Academy of Neurology (Member of the Board and Chairman of the Scientific Committee)
- American Academy of Neurology
- World Federation of Neurology (Co-Chair Section of Migration Neurology)
- Neuromediterraneum Forum (President)

His present positions are:
full professor of Neurology, University of Siena, Medical School
- Director of Unit Clinical Neurology and Neurometabolic Diseases, Siena Hospital.
- Past-Director of the Section Neurological Diseases of the Department of Neurological and Behavioural Sciences of the University of Siena since the 2012, at the fusion of this Department in the Dept Medicine, Surgery and Neurosciences.
- Italian Delegate to the World Federation of Neurology and to European Academy of Neurology Council.
- Past- President of the Italian Society of Neurology (President years 2009-2011)
- From 1995 he is Director of a PhD Programme on Applied Neurological Sciences at University of Siena, from 2004 of the European PhD Programme and European School of Doctorate of Applied Neurological Sciences. Since 2011 he is director of the PhD Programme on Cognitive and Neurological Sciences at University of Siena.
- He is Italian member of the Committee of European Union of Medical Specialists, in the section Neurology.
- Delegate for Research in the Dept. Medicine, Surgery and Neurosciences.
- Coordinator for the Tuscany Region of the Network on Rare Neurological Diseases.
- On 2013, he received Honoris Causa degree from the University Carol Davila, Bucharest
- Chairman of the Neuromediterraneum Forum
- Editor in Chief of Neurological Sciences, Springer-Verlag Editor.
- Co-Editor of many international journals.

- On the 2014 was nominate WHO consultant for Rare Neurological Diseases.
- From june 2014, he is Chairman of the Scientific Committee and Member of the Board of the European Academy of Neurology
- From February 2015 Co-Chairman of the Research Group Migration Neurology of the World Federation of Neurology.
- From the 1st april 2016, vice-Rector of the University of Siena.

VOLKER HÖMBERG
GERMANY

PERSONAL DATA Born 25 July 1954

MEDICAL CAREER

1973 - 1980	Medical School, Universities of Düsseldorf and Freiburg; Electives in Neurology at Boston City Hospital, Boston, Mass.; National Hospital for Nervous Diseases, London
1975-1980	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
1981-1986	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
1987-1996	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
2000 to 2010	Medical director and head of Neurology, St. Mauritius Therapy Hospital, Meerbusch
Since 10/2011	Head of Neurology SRH Gesundheitszentrum Bad Wimpfen
10/2004 to 12/2010	Vice president of the German Society for Neurological Rehabilitation
2005 to 2010	Panel-Chairman Neurorehabilitation for European Federation Neurological Societies (EFNS)
Since 12/2010	Member of the board (DGNR)
Since 2003	Secretary General World Federation for NeuroRehabilitation (WFNR)
Since 2011	Secretary General European Federation of Neurorehabilitation Societies (EFNR)
Since 2015	Vice President of EFNR

Areas of scientific interest	Motor control Neuropsychology, Brain plasticity Epistemology of rehabilitation sciences Pharmacology in neurorehablitation
Publications	more than 200 original articles in peer reviewed journals

PETER JENNER

UK

Peter Jenner is currently Emeritus Professor of Pharmacology at King's College London. He was previously Head of the Division of Pharmacology and Therapeutics at King's College and Director of the Neurodegenerative Diseases Research Centre and National Parkinson Foundation Centre of Excellence. Peter has worked on the cause, treatment and potential cure of Parkinson's disease for more than 30 years and he is a key opinion leader in the field. He has published more than 700 peer reviewed papers and frequently speaks at national and international meetings. His main expertise lies in understanding current and future drug treatment of motor and non-motor symptoms of Parkinson's disease. He has worked closely with the pharmaceutical industry in developing new approaches to therapy and he has experience of developing novel drug treatments from their discovery in the laboratory through to their use as medicines in people with Parkinson's disease.

AMOS KORCZYN
ISRAEL

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, and the incumbent of the Sieratzki Chair of Neurology at Tel-Aviv University, 1995-2010. Professor Korczyn has a particular interest in neurodegenerative diseases. He has authored or co-authored over 600 articles in peer-reviewed journals, as well as chapters in books, etc. He edited several books and Special Issues in Journals, and is co-Editor of the Journal of the Israeli Neurological Association (JINA) since 2009. He is or has been an Editorial Board member of 20 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. Professor Korczyn also served on advisory boards in several drug discovery programs.

Professor Korczyn is the Chairman of the Scientific Administrative Board of the Israeli Alzheimer's disease association (EMDA), and member of the SAB of Alzheimer Disease International, and has been the chairman of the WFN Research Committee for Neuropharmacology.

Professor Korczyn is an honorary member of the neurological societies of Israel, Serbia, Poland and Russia.

Professor Korczyn's H-index is 39.

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 F. MUREȘANU
ROMANIA

Dafin F. Mureșanu, MD, PhD, MBA, FANA
Professor of Neurology, Senior Neurologist, Chairman of the Neurosciences Department, Faculty of Medicine, University of Medicine and Pharmacy "Iuliu Hatieganu" Cluj-Napoca, President of the Romanian Society of Neurology, President of the Society for the Study of Neuroprotection and Neuroplasticity (SSNN), member of the Academy of Medical Sciences, Romania, secretary of its Cluj Branch. He is also member of 13 scientific international societies (being member of the American Neurological Association (ANA) - Fellow of ANA (FANA) since 2012) and 7 national ones, being part of the executive board of most of these societies. Professor Dafin F. Mureșanu is a specialist in Leadership and Management of Research and Health Care Systems (specialization in Management and Leadership, Arthur Anderson Institute, Illinois, USA, 1998 and several international courses and training stages in Neurology, research, management and leadership). Professor Dafin F. Mureșanu is coordinator in international educational programs of European Master (i.e. European Master in Stroke Medicine, University of Krems), organizer and co-organizer of many educational projects: European and international schools and courses (International School of Neurology, European Stroke Organisation summer School, Danubian Neurological Society Teaching Courses, Seminars - Department of Neurosciences, European Teaching Courses on Neurorehabilitation) and scientific events: congresses, conferences, symposia (International Congresses of the Society for the Study of Neuroprotection and Neuroplasticity (SSNN), International Association of Neurorestoratology (IANR) & Global College for Neuroprotection and Neuroregeneration (GCNN) Conferences, Vascular Dementia Congresses (VaD), World Congresses on Controversies in Neurology (CONy), Danube Society Neurology Congresses, World Academy for Multidisciplinary Neurotraumatolgy (AMN) Congresses, Congresses of European Society for Clinical Neuropharmacology, European Congresses of Neurorehabilitation). His activity includes involvement in many national and international clinical studies and research projects, over 350 scientific participations as "invited speaker" in national and international scientific events, a significant portfolio of scientific articles (134 papers indexed on Web of Science-ISI, H-index: 15) as well as contributions in monographs and books published by prestigious international publishing houses. Prof. Dr. Dafin F. Mureșanu has been honoured with: the Academy of Romanian Scientists, "Carol Davila Award for Medical Sciences / 2011", for the contribution to the Neurosurgery book "Tratat de Neurochirurgie" (vol.2), Editura Medicala, Bucuresti, 2011; 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.

CATERINA PISTARINI
ITALY

Director of Neurorehabilitation Unit – Coma Unit – Spinal Cord Unit
Salvatore Maugeri Foundation – Pavia Institute - Italy

Degree in Medicine (1980) Postgraduate degree in Neurology (1984) and Postgraduate degree in Physical and Rehabilitation Medicine (1987) – University of Pavia – Italy.
Current Position:
since 2003 she is the Director of Neurorehabilitation Unit – Coma Unit – Spinal Cord Unit at IRCCS Salvatore Maugeri Foundation, Pavia Institute.

Previous assignments:
From 2000 to 2003 Director of Spinal Cord Unit at IRCCS Salvatore Maugeri Foundation, Montescano (PV) Institute.
From 2000 to 2003 Director of Neurorehabilitation Unit at IRCCS Salvatore Maugeri Foundation, Montescano (PV) Institute.

She attended the Healthcare Management Qualification Course since 2000, updated in 2010, and obtained the Management degree at IREF (Institute of Research, Statistics and Training) - School of Health Management in Milan – Italy; she is actively engaged in the management and organizational processes of the Units with particular attention to the control of the Quality System in compliance with current directives applied in health services (ISO 2001).

Since 2007 she is a Contract Professor in Physiotherapy Disciplines and in Occupational Therapy Disciplines at the University of Pavia – Italy.

Scientific Positions since 2008 to now:

- Since April 2015, President of the Italian Society of Neurological Reahbilitation (SIRN) Until April 2013, Vice-President
- Past President of the Italian Society of Rehabilitation of High Specialization (SIRAS). (President 2008-2011)
- Coordinator of the International Rehabilitation Network Development Education Network (REHADE) (since 2011)
- Associated Member of the European Neurotraumatologic Academy (EMNR) (since 2010)

- Chair of the World Federation of Neuro Rehabilitation (WFNR) 's Special Interest Group on Mild/ Severe Brain Injury (since 2008)
- Founding member of the Robotic and Rehabilitation Interest Group (RoRIG) (since 2007)
- Member of Physical Medicine and Rehabilitation Italian Society (SIMFER) (since 1984)
- Partecipation to the editorial board "New Frontiers in Clinical Rehabilitation: Advances in Assessment and Care for Disability", of the Hindawi online Scientific Journal (dal 06/2013)

Awards for scientific activity:

- Premio SAPIO per la Salute ed il Sociale 2003 "Reduced plasma levels of tyrosine, precursor of brain catecholamines, and of essential amino acids in patients with severe traumatic brain injury after rehabilitation"
- Premio Franco Michele Puca "Sonno e Stato Vegetativo"
  Congresso AISM Parma 2012
- Premio Congresso MIE PISA 2012 "E-Learning for Neurological Bladder Management"
- Award of excellence ESPRM Tessaloniki-Grecia 2012 " Cognitive Impairment and ....The GeHA Europ. Project

Use and commercialization of patents:
- Evocare - Evoling 2: computerized theraphy in speech patologies (software)
- Sensorized garment for motor rehabilitation ("My Heart Project")

Principal subjects/occupational skills covered:
Stroke; Traumatic Brain Injury and Spinal Cord Injury Rehabilitation; Intensive rehabilitation of patients with consciousness disorder after acquired head injuries.
Theoretical and practical competences according to specific criteria for the best knowledge and experience in Neuro-Rehabilitation proposed by the Italian Society of Physical and Rehabilitation Medicine (SIMFER), by the Italian Society of Neurological Rehabilitation (SIRN) and the World Federation of Neuro-Rehabilitation (WFNR):
- Knowledges on Therapies in rehabilitation medicine;
- Knowledges on Clinical and functional assessment of patients with neurological disabilities;
- Application of Specific Rehabilitation Treatments on Brain injury, Spinal Cord Injury and Stroke (Rehabilitation of motor and cognitive functions, Neuro- rehabilitation in paediatric conditions, Urological and Sexual Rehabilitation of old People, Pain management.....).

Since 1984, participation to Congresses and national/international rehabilitation Courses on the above mentioned topics and presentation of many scientific contributions.

Organizator and promoter of national/international Congresses on the topics on neurological rehabilitation.
Promoter and partecipant to national and european research projects in particular on

Acquired Brain Injury Neuro-rehabilitation and Telerehabilitation.
Collaboration in drawing formal documents on the best practice's clinical and organizational criteria.
Definition of Rehabilitation Guidelines for Health Professional practice on behalf of National Ministry of Health (both for general rehabilitation activities and for rehabilitation activities in VS and MCS patients).

H Citation Index calculated according to WEB on SCIENCE = 10

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.

Research activity: 40 ISI full text articles, 780 ISI citations, Hirsch index 17.

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 Babeș' National Institute of Pathology, Bucharest, Romania
2008-	Senior Neurologist
2009 - 2012	Lecturer, 'Carol Davila' University School of Medicine
2009 -	Senior Researcher, 'Victor Babeș' 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 Babeș' National Institute of Pathology

2007 Romanian Academy award for medical research

2010 'Science and Art National Foundation Award of Excellence for research in the field of Neuroscience and Neuropathology

Other current activities

Guest editor for Alzheimer's review series at Journal of Cellular and Molecular Medicine

Executive editor of Romanian Journal of Neurology

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 Babeș' 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, Băjenaru O, Mureșanu 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, Băjenaru 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;66:122-3

HARI SHANKER SHARMA

SWEDEN

Hari Shanker Sharma, Director of Research (International Experimental Central Nervous System Injury & Repair, IECNSIR), University Hospital, Uppsala University is Professor of Neurobiology (MRC), Docent in Neuroanatomy (UU) and is currently affiliated with Department of Surgical Sciences, Division of Anesthesiology and Intensive Care Medicine, Uppsala University, Sweden. Hari Sharma was born on January 15, 1955 in an Industrialist town Dalmianagar (Bihar), India. 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. 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 and was awarded Ph.D. in 1982 on "Blood-Brain Barrier in Stress." Hari Sharma after carrying out a series of Government of India funded 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 investigate passage of tracer transport across the BBB caused by stress or 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 Government (1989–1991) to work on hyperthermia induced BBB dysfunction at the ultrastructural level in the laboratory of Professor Jorge Cervós-Navarro (a living "Legend in Neuropathology in Europe"). Dr. Sharma joined again Uppsala University and established a network of collaboration on "Experimental CNS Injury Research Group" as a lead investigator with eminent collaborators in various parts of Europe, USA, and Australia (1991–). On his work on hyperthermia Dr. Sharma received the 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 and selected for the Best Thesis Award of the Medical faculty, "The Hwassers Prize" of 1999. On his meticulous works on the Blood Brain barrier and Brain edema (2000–2003) Dr. Sharma earned the prestigious title of "Docent in Neuroanatomy" of Medical Faculty, Uppsala University in April 2004. Currently his main research interest is Neuroprotection and Neuroregeneration, in relation to the Blood-brain barrier in stress, trauma, and drugs of abuse in health and disease.

Dr. Sharma on his research on brain pathology and neuroprotection in different models received the prestigious awards from The Laerdal Foundation of Acute Medicine, Stavanger,

Norway, in 2005 followed by Distinguished International Scientists Collaboration Award by National Institute on Drug Abuse (NIDA), Baltimore, MD (2006–2008). His recent work on 5-HT3 receptor mediated neuroprotection in morphine withdrawal induced neurotoxicity won the coveted prize of Best Investigator Award 2008 and Best Scientific Presentation by European Federation of the International Association for Study of Pain (ISAP), and Awarded during their VI Annual Meeting in Lisbon, September 9–12, 2008. His recent research is aimed to find out the role of nanoparticles in Neurodegeneration and Neuroprotection using various treatment strategies that is supported by European Aerospace Research and Development (EOARD), London, UK and US Air Force Research Laboratory, Wright Patterson Air Force Base, Dayton, Oh, USA. On his works on Blood–brain barrier in hypertension and diabetes together with Romanian colleagues, University of Medicine and Pharmacy "Iuliu Hatieganu," Cluj-Napoca, Romania awarded Dr. Sharma with Honorary Doctorate of Medical Sciences in 2009. Dr. Sharma's work over 30 years on the blood-brain barrier and brain edema won him the US Neurosurgeon Dr. Anthony Marmarou Award (2011) by the International Brain Edema Society at their 15th Congress in Tokyo, Japan, November 20–24, 2011. His works on Nanoneuroscience and development of nanomedicine to treat the CNS injuries has won accolades at various Government and International Scotties or Organization across the World. Accordingly Dr Sharma was decorated with the most prestigious "Hind Rattan Award 2012" (Jewel of India) on the eve of Republic Day of India 25th January 2012 and Mahatma Gandhi Pravasi Gold Medal on October 12, 2012 in House of Lords, London, UK. Based on his outstanding contribution in Nanoneuropharmacology and nanodrug delivery to treat central nervous system (CNS) diseases including Neurodegenerative diseases such as Alzheimer's and Parkinson's Hari Sharma bestowed with Prestigious Gujarat Govt. International Visionary Award 2012 in a glittering function in Ahmedabad, Gujarat on Nov 23, 2012. His further research on co-morbidity factors e.g., hypertension or diabetes may alter pathophysiology of brain injuries and require higher drug dose or nanodrug delivery of neuroprotective agents to minimize brain dysfunction is recognized by Govt. of India by presenting him one of the coveted "Bharat Jyoti Award 2013" (Glory of India) by His Excellency Governor Balmiki Prasad Singh in Hotel Le Meridien, New Delhi on Jan 12, 2013. Dr Sharma also received the highest Award of the Govt. of India "Navrattan Award 2013" (Nine Jewels of India) on the eve of 64th Republic Day of India (25th January 2013) by His Excellency Governor Bhishma Narain Singh, in Ashok Hotel, New Delhi. Hari Sharma is Founding President of the Global College of Neuroprotection & Neuroregeneration (2004-); Elected President of International Association of Neurorestoratology (IANR) (2014-); and selected Senior Expert of Asia-Pacific CEO Association, Worldwide (APCEO) (2012-) for his contribution to uplift scientific research in many countries Globally that may have better economic and social benefit for the mankind. Hari Sharma awarded coveted National Award "Sword of Honor" 2015 by Govt. of India on the eve of 66th Republic Day of India 25th January 2015 in New Delhi Eros Hotel International during the 34th Non-resident Indian (NRI) conclave by Speaker of Lok Sabha (Indian Parliament) the Hon'ble Mrs Meira Kumar of Indian national Congress (INC) Party for the continued extraordinary achievement in nanomedicine for public health awareness and possible therapeutic measures.

Based on his expertise in Nanoneuroscience, Hari Sharma was also invited to organize and chair Nanosymposium in Society for Neuroscience meetings in Chicago (2009), San Diego (2010), Washington DC (2011), New Orleans (2012), San Diego (2013) and Washington DC

(2014, Nov 15-19, 2014); Chair Neurobiology Symposium 14th Int. Amino Acid & Peptide, Vienna, Austria; Keynote speaker & Chair Nanotechnology-2015, Frankfurt, Germany. Hari Sharma is also the recipient of Prestigious US TechConnect Global Innovation Award 2013 at the National Innovation Summit & Innovation Showcase, Washington DC May 12-16, 2013 on his work on Nanowired cerebrolysin in Neuropathic Pain. Hari Sharma Served as one of the Poster Judges in 2014 180th Annual Meeting of American Association of Advancement of Science (AAAS) Held in Chicago, IL, USA Feb 13-17, 2014 followed by 181st Annual Meeting of American Association of Advancement of Science (AAAS) held in San José, CA, USA Feb 12-16, 2015. Hari Sharma has published over 350 research papers and 85 reviews, 14 monographs, and 80 international book chapters and edited 18 book volumes with Current H-index = 38 (ISI Database) as of today. He served as Guest Editor of Curr. Pharm. Desig. (2005, 2007, 2010–); J Neural. Transmiss. (2006, 2011–) and is the founding Editor-in-Chief of Int. J. Neuroprotec. Neuroregen. (2004–), UK and the European Editor of Central Nervous system-Neurological Disorders Drug Target (2013-). Dr. Sharma is on board of various International Journals including CNS and Neurological Disorders-Drug Targets, USA (2010), Journal of Neurodegeneration and Regeneration, USA (2009–); Austin Journal of Nanomedicine & Nanotechnology (2014-); and is associate editor of Journal of Nanoscience and Nanotechnology (Nanoneuroscience 2006–), USA, Review Editor—Frontiers in Neuroengineering (2007–), Frontiers in Neurorestoratology, and Associate Editor of Frontiers in Aging Neuroscience (2008–), Frontiers of Fractal Physiology (2010–), Switzerland, Journal of Neurorestoratology, Dove Medical press, London, UK (2012–), WebMD Central, Neurology Faculty, Advisory Board Member (2010–), World Journal of Pharmacology (2011–), Journal of Physical Medicine and Rehabilitation, USA (2012–). Dr. Sharma served as volume editor of several progress in Brain research series (Volumes 104, 115, 162 and 180), International review of Neurobiology (Volume 82 and 102) and other Springer Volumes on Spinal cord injury (1988) and Handbook of Neurochemistry (2009) apart from stand alone books (Elsevier, Springer and Academic Press since 1994). Dr. Hari Sharma is invited to join several National Academies of repute including New York Academy fo Science, USA (since 1994–); International Academy of Stress, New York (2003–), Swedish Academy of Pharmaceutical Sciences (2010–). Dr. Sharma has served as an expert evaluator and advisor to various Boards, Councils and Institutions for their Research Grants including Wellcome Trust, London, UK (2011–); Catalan Agency for Health Information and Quality, TV3 (2010–), European Commission Projects (2002–), European Nanomed Council (2009–), Ministry of Health Science Foundation; Medical research Council and University Commission of Grants in various countries in Europe, USA, UK, Canada, Hong Kong, Singapore and in Australia. Some of the notable organizations include: Australia and New Zealand Health Council (2000–); University Commission of Grants, Hong Kong (2002–), Singapore Medical Council, Singapore (2003–); UK Charity Organization "Research on Ageing: Help the Aged" (2003–); Euro Nanomed (2010–). Dr. Sharma is designated as ambassador of the City of Uppsala 2007, by Uppsala County administration and Uppsala Tourism for promoting Uppsala, Sweden as International Research Collaboration/Meetings and Conference Destination. Dr. Hari Sharma is married to Aruna Sharma (nee Bajpai) since 23rd April 1979 and has two sons. Dr Sharma is designated as Visiting Professor, University of Basque Country, Bilbao, Spain supported by Basque Govt. Foundation. His political affiliation belongs to Swedish Social Democrat Party (Socialdemokraterna, Sverige) where he is associated with the development of Education and Research matters in Sweden actively.

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 SKAPER
ITALY

STUDIES: B.S. (chemistry) Illinois Institute of Technology (1969); Ph.D. (biochemistry) University of South Dakota (1973); Laurea in chemistry, University of Padua (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 Padua, Padua, Italy (1997); Assistant Director, Molecular Neurobiology Research, SmithKline Beecham Pharmaceuticals, New Frontiers Science Park, Harlow, United Kingdom (1998-2001); Senior Group Leader, Migraine and Stroke Research, Neurology & GI Centre of Excellence for Drug Discovery, GlaxoSmithKline R & D Limited, Harlow, United Kingdom (2002-2003); Senior Group Leader, Neurodegeneration Research, Neurology & GI Centre of Excellence for Drug Discovery, GlaxoSmithKline R & D Limited, Harlow, United Kingdom (2004-2007); Senior Group Leader, Target Validation (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 Padua, Faculty of Medicine, Padua, 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; Associate Editor, American Journal of Neuroprotection and Neuroregeneration; Editorial Board Member, 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 ageing, neurodegenerative disorders and neuroinflammation, astrocyte-microglia interactions, pharmacological modulation of oligodendrocyte precursor maturation and demyelinating diseases. 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 300 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, Neuroscience, Apoptosis, PLoS One Biology, Journal of Pharmacology and Experimental Therapeutics, British Journal of Pharmacology, European Journal of Pharmacology, Journal of Neurological Sciences.

JÓZSEF SZÁSZ
ROMANIA

PERSONAL DATA:
- Surname: Szász
- First name: József Attila
- Date and place of birth: 02.APR.1967, Sighisoara, Romania

EDUCATION:
- University of Medicine and Pharmacy (UMPh), Tirgu-Mures, Romania (1986-1992)
- PhD thesis: Motor complications and therapy in advanced Parkinson's Disease (2005)
  University of Medicine and Pharmacy, Tirgu-Mures, Romania

WORK EXPERIENCE :
- Resident in Neurology (1992-1998)
- Neurologist (1998-2003)
- Senior neurologist (2003-)
- Assist. Prof. at the Department of Neurology UMPh Tg.Mures (1999-2009)
- Senior Lecturer at the Department of Neurology UMPh Tg.Mures (2009-)

TEACHING ACTIVITY

IN ROMANIAN: clinical practice in neurology for students and resident doctors (1999-)

IN HUNGARIAN: lectures in adult neurology (2005-)

CLINICAL TRIALS

Principal investigator in 10, investigator in 6, phase III, clinical studies

THE MOST IMPORTANT PUBLICATIONS:

1. Kerenyi L, Kardos L, Szász J, Szatmari S, Bereczki D, Hegedus K, Csiba L. Factors influencing hemorrhagic transformation in ischemic stroke: a clinicopathological comparison. European Journal of Neurology 2006 Nov;13(11):1251-1255. ISSN 1351-5101 IF: 2,244
2. Szatmari S, Pascu I, Mihalka L, Mulesa SV, Fekete I, Fulesdi B, Csiba L, Zselyuk G, Szász J, Gebefugi J, Nicolescu S, Vasiesiu D, Smolanka VI, Bereczki D: The Mures-Uzhgorod-Debrecen study: a comparison of hospital stroke services in Central-Eastern Europe. European Journal of Neurology 2002;9:1-4 ISSN 1351-5101 IF: 1,565
3. Rupam Borgohain, Jozsef Szász, P. Stanzione, et al. Randomized trial of safinamide add-on to levodopa in Parkinson's disease with motor fluctuations. Mov Disord, 2014, 29:229–237

4. Rupam Borgohain, Jozsef Szász, Paolo Stanzione, et al. Two-Year, Randomized, Controlled Study of Safinamide as Add-on to Levodopa in Mid to Late Parkinson's Disease Mov Disord, 2014, 29: 1273–1280
5. Fekete K, Szatmari S, Szőcs I, Szekeres C, Szász J, Mihálka L, Smolanka V, Kardos L, Csiba L, Bereczki D. Prestroke alcohol consumption and smoking are not associated with stroke severity, disability at discharge, and case fatality. J Stroke Cerebrovasc Dis. 2014 Jan;23(1):e31-37 IF: 1.984

FIELDS OF INTEREST: movement disorders, dementia, stroke, chronic pain, epilepsy,

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 Băjenaru. 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...

‘‘RoNeuro’’

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