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European Federation of Neurological Societies
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Firas Fahoum /Israel
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Hari Shanker Sharma /Sweden
Mihaela Simu /Romania
Stephen Skaper /Italy
Cristina Tiu /Romania
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Registration Desk

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

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

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

FINAL PROGRAM & ABSTRACT BOOK
The participants documents include the program and abstract book which will be handed out at the registration counter.

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

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

CURRENCY
The official Romanian currency is RON.

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

TIME
The time in Romania is Eastern European Time (GMT+2).
SCIENTIFIC PROGRAM
## SCIENTIFIC PROGRAM

### Sunday, July 5th, 2015

<table>
<thead>
<tr>
<th>Time</th>
<th>Speaker</th>
<th>Topic</th>
</tr>
</thead>
<tbody>
<tr>
<td>16:00 – 16:30</td>
<td>Hari Shanker Sharma (Sweden)</td>
<td>Pathophysiology of the blood-brain barrier in CNS injury &amp; repair. New roles of Cerebrolysin and nanomedicine</td>
</tr>
<tr>
<td>16:30 – 17:00</td>
<td>Stephen Skaper (Italy)</td>
<td>Low-grade non-resolving neuroinflammation: age does matter</td>
</tr>
<tr>
<td>17:00 – 17:30</td>
<td>Bogdan Popescu (Romania)</td>
<td>Dyautonomia in neurodegenerative diseases – incidence, features and treatment</td>
</tr>
<tr>
<td>17:30 – 18:00</td>
<td>Dieter Meier (Austria)</td>
<td>Apomorphine in the treatment of late stage Parkinson’s Disease</td>
</tr>
<tr>
<td>18:00 – 18:30</td>
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<td>Coffee Break</td>
</tr>
<tr>
<td>18:30 – 19:15</td>
<td>Volker Homberg (Germany)</td>
<td>Neurological examination – tips and tricks (I)</td>
</tr>
<tr>
<td>19:15 – 20:00</td>
<td>Volker Homberg (Germany)</td>
<td>Neurological examination – tips and tricks (II)</td>
</tr>
<tr>
<td>20:00</td>
<td></td>
<td>Welcome dinner</td>
</tr>
</tbody>
</table>
# Monday, July 6th, 2015

**Module coordinators:** Volker Homberg (Germany), Alla Guekht (Russia)

<table>
<thead>
<tr>
<th>Time</th>
<th>Session</th>
<th>Speaker</th>
<th>Topic</th>
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<tbody>
<tr>
<td>08:30</td>
<td>Welcome Address</td>
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<tr>
<td>08:40–09:00</td>
<td>Dafin Muresanu (Romania)</td>
<td>Results from a large retrospective cohort trial in TBI</td>
<td></td>
</tr>
<tr>
<td>09:00–09:45</td>
<td>Volker Homberg (Germany)</td>
<td>Neurorehabilitation – where are we?</td>
<td></td>
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<tr>
<td>09:45–10:15</td>
<td>Dana Boering (Germany)</td>
<td>Management of dysphagia after stroke</td>
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<tr>
<td>10:15–10:45</td>
<td>Alla Guekht (Russia)</td>
<td>Treatment of the first seizure: risks and benefits</td>
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<tr>
<td>10:45–11:15</td>
<td>Coffee Break</td>
<td></td>
<td></td>
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<tr>
<td>11:15–11:45</td>
<td>Firas Fahoum (Israel)</td>
<td>Classifications of seizures and epilepsies - update</td>
<td></td>
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<tr>
<td>11:45–12:15</td>
<td>Firas Fahoum (Israel)</td>
<td>New generation antiepileptic drugs</td>
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<tr>
<td>12:15–12:45</td>
<td>Ioana Mindruta (Romania)</td>
<td>Neurophysiological exploration in epilepsy – the fundamentals</td>
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<tr>
<td>12:45–13:15</td>
<td>Ioana Mindruta (Romania)</td>
<td>Semiology of focal seizures in temporal vs extratemporal epilepsy</td>
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<tr>
<td>13:30</td>
<td>Lunch</td>
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<tr>
<td>18:00–20:00</td>
<td>Case presentations</td>
<td>Epilepsy</td>
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<tr>
<td>20:00</td>
<td>Dinner</td>
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<tr>
<td>Time</td>
<td>Speaker</td>
<td>Topic</td>
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<tr>
<td>08:45 – 09:00</td>
<td>Dafin Muresanu (Romania)</td>
<td>International School of Neurology at the Age of 10</td>
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<tr>
<td></td>
<td>Natan Bornstein (Israel)</td>
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<tr>
<td>09:00 – 09:45</td>
<td>Natan Bornstein (Israel)</td>
<td>Time is Brain, TIA as an Emergency</td>
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<tr>
<td>09:45 – 10:30</td>
<td>Natan Bornstein (Israel)</td>
<td>Secondary stroke prevention: Antiplatelet - update</td>
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<tr>
<td>10:30 – 11:00</td>
<td></td>
<td>Coffee Break</td>
<td></td>
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<tr>
<td>11:00 – 11:45</td>
<td>Natan Bornstein (Israel)</td>
<td>Management of symptomatic carotid stenosis - CEA vs. Stent</td>
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<tr>
<td>11:45 – 12:30</td>
<td>Dafin Muresanu (Romania)</td>
<td>Advances in brain protection and recovery in stroke therapy</td>
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<tr>
<td>12:30 – 13:15</td>
<td>Laszlo Csiba (Hungary)</td>
<td>2015 Advances in acute ischemic stroke and ICH</td>
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<tr>
<td>13:30</td>
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<td>Lunch</td>
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<tr>
<td>18:00 – 20:00</td>
<td></td>
<td>Case presentations</td>
<td>Stroke</td>
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<tr>
<td>20:00</td>
<td></td>
<td>Dinner</td>
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### Wednesday, July 8th, 2015

**Module coordinators:** Amos Korczyn (Israel), Vivian Drory (Israel)

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<thead>
<tr>
<th>Time</th>
<th>Speaker/s</th>
<th>Topic</th>
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<tbody>
<tr>
<td>09:00 – 09:30</td>
<td>Ovidiu Bajenaru (Romania)</td>
<td>Connectomics in neuroscience and clinical practice</td>
</tr>
<tr>
<td>09:30 – 10:00</td>
<td>Amos Korczyn (Israel)</td>
<td>Medically unexplained symptoms in neurology</td>
</tr>
<tr>
<td>10:00 – 10:30</td>
<td>Cristian Falup-Pecurarui (Romania)</td>
<td>Hyperkinetic movement disorders post stroke</td>
</tr>
<tr>
<td>10:30 – 11:00</td>
<td>Mihaela Simu (Romania)</td>
<td>Evaluation of parkinson’s disease – Features of advanced stages</td>
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<tr>
<td>11:00 – 11:30</td>
<td>Coffee Break</td>
<td></td>
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<tr>
<td>11:30 – 12:00</td>
<td>Vivian Drory (Israel)</td>
<td>Amyotrophic lateral sclerosis</td>
</tr>
<tr>
<td>12:30 – 13:00</td>
<td>Vivian Drory (Israel)</td>
<td>Chronic inflammatory demyelinating polyneuropathy</td>
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<tr>
<td>13:30</td>
<td>Lunch</td>
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<tr>
<td>18:00 – 20:30</td>
<td>Case presentations</td>
<td>The electrodiagnostic examination</td>
</tr>
<tr>
<td>20:30</td>
<td>Dinner</td>
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</table>
### Thursday, July 9th, 2015

**Module coordinators:** Ovidiu Bajenaru (Romania), Dafin Muresanu (Romania)

<table>
<thead>
<tr>
<th>Time</th>
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<th>Topic</th>
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<tbody>
<tr>
<td>09:00 – 09:30</td>
<td>Cristina Tiu (Romania)</td>
<td>Differential diagnosis in multiple sclerosis: where to start, where to end?</td>
</tr>
<tr>
<td>09:30 – 10:00</td>
<td>Ovidiu Bajenaru (Romania)</td>
<td>Optimal clinical approach of MS patients’ management</td>
</tr>
<tr>
<td>10:00 – 10:30</td>
<td>Anat Achiron (Israel)</td>
<td>Cognitive aspects in multiple sclerosis</td>
</tr>
<tr>
<td>10:30 – 11:00</td>
<td></td>
<td>Coffee Break</td>
</tr>
<tr>
<td>11:00 – 11:30</td>
<td>Mihaela Simu (Romania)</td>
<td>Measuring and managing disease activity</td>
</tr>
<tr>
<td>11:30 – 12:00</td>
<td>Cristina Panea (Romania)</td>
<td>Multiple sclerosis and pregnancy</td>
</tr>
<tr>
<td>12:00 – 12:30</td>
<td>Dafin Muresanu (Romania)</td>
<td>Symptomatic treatment in MS</td>
</tr>
<tr>
<td>12:30 – 13:00</td>
<td>Tudor Lupescu (Romania)</td>
<td>Diabetic neuropathy – Diagnosis and symptomatic treatment</td>
</tr>
<tr>
<td>13:00 – 14:00</td>
<td></td>
<td>Lunch</td>
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<tr>
<td>14:00 – 15:00</td>
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<td>Final Examination</td>
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<tr>
<td>15:15</td>
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<td>Official closing</td>
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<tr>
<td>20:00</td>
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<td>Farewell Gala Dinner</td>
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</tbody>
</table>
ABSTRACTS
Cognitive dysfunction affects many patients with multiple sclerosis (MS) and has been reported even at disease onset. This significant cognitive involvement in MS requires a comprehensively and accurately performed cognitive assessment. We have shown that computerized cognitive testing has the advantages of increased sensitivity due to precise measurement of response time and frequency of errors, minimal ceiling or floor effects due to adaptive testing designs and no learning effects. In a large cross-sectional study that included 1500 MS patients, cognitive performance was poorer than healthy age- and education- matched population norms. We identified that information processing speed and executive functions were the most frequent abnormalities in the MS population with 33.9% and 30.9% of patients performing below one standard deviation of the average, respectively. MS patients with secondary-progressive disease course performed poorly compared with clinically isolated syndrome, relapsing-remitting and primary progressive MS patients. By the fifth year from onset, 20.9% of patients performed below the 1SD cutoff for impairment, p=0.005, and 6.0% performed below the 2SD cutoff for severe cognitive impairment, p=0.002. By 10 years from onset, 29.3% and 9.0% of patients performed below the 1SD and 2SD cutoffs, respectively, p=0.0001. Regression modeling suggested that cognitive impairment may precede MS onset by 1.2 years. Better understanding the epidemiology of cognitive dysfunction and cognitive resilience in MS may help to identify patients at increased risk and facilitate the identification of possible protective factors associated with better cognitive health. Cognitive impairment differed significantly from expected normal distribution at five years from onset suggesting the existence of a therapeutic window during which patients may benefit from interventions to maintain cognitive health. Treatment option in addition to immunomodulatory drugs include methods that could lead to enhancement of neuroplasticity like cognitive rehabilitation, cognitive games and physical activity.

The essential functional structure of the central nervous system (CNS) is the neuronal network. Brain neural networks can be described and analyzed as graphs comprising a collection of nodes (describing neurons/brain regions) and a collection of edges (describing structural connections or functional relationships). A very small proportion of nodes, called hubs, are very highly connected, and over longer distances. There are two types of such networks: one type include those networks related to the accomplishment of task-specific activities; the other type of neural networks are active during the apparent resting state of the brain, and have very specific characteristics related to the different aspects of the integrative functions of the brain in relation with the environment and in the same time, with the self functional structure. The most important structural and functional difference between the human brain and the brain of other primates (not to speak about other animal categories) is the incomparable huge complexity due to extremely high interconnectivity inside and among these neural networks in the human brain. These human brain resting state neural networks have been identified and characterised during the last less than 15 years, both in normal conditions, but also in different brain diseases due to the technical advancement in FDG-PET and mainly in functional MRI. Among the best characterized resting state neural networks, are: the Default Mode Network (DMN), the Salient Detection Network, the Sensory/Motor Network, the Executive Control Network, up to three different Visual Networks, two lateralized Frontal/Parietal Networks, the Auditory Network, the Temporal/Parietal Network. These data will be presented during this conference, emphasizing the particularities of DMN which is essential in the function of the normal brain, and which impairment in different pathologic conditions is better and better identified and correlated with specific clinical manifestations. Nowadays, the possibility to study these brain networks by non-invasive means due in particular to fMRI development, has allowed the accumulation
of very interesting new data not only about the normal function of these networks, but also more and more about their functional modifications in different brain diseases — in particular in neurodegenerative diseases, but also in epilepsy, in HIV-associated neurocognitive disorders, and others. These data are a premise not only for a more detailed understanding of the pathology and dynamic dysfunctions generated in the brain by these diseases, but also to use these type of studies in their early and more precise diagnostic which could allow more efficient therapeutic interventions.

MANAGEMENT OF DYSPHAGIA AFTER STROKE

DANA BOERING
St. Mauritius Therapieklinik Meerbusch, Germany

Dysphagia affects more than 50% of stroke survivors and represents one of the first hurdles on the path of recovery after stroke, leading to a 17% increase of pulmonary infections and a 30% increase of mortality. Prompt evaluation and treatment of swallowing disorders can therefore mitigate the development of further secondary complications and foster social reintegration of stroke patients.

The talk will give an overview of swallowing physiology and neural control, of bedside screening tests, of clinical and instrumental assessment methods, a brief insight in the mechanisms of postlesional plasticity in poststroke dysphagia and in the nutritional assessment and support of the patients. It will give a detailed presentation of the different compensatory and rehabilitative techniques pointing out new trends in dysphagia management and possible future developments of this rapidly evolving field.

MANAGEMENT OF SYMPTOMATIC CAROTID STENOSIS. CEA VS. STENT

NATAN 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 =8.8%) 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

NATAN 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 ESPRIT). Both studies have shown approximately 20%-24% relative risk reduction (RRR) of stroke and death. Clopidogrel is superior to aspirin in patients at high risk of recurrence by about 8.7% RRR (CAPRIE). The combination of aspirin plus clopidogrel is not more effective than clopidogrel alone but carries a higher bleeding risk (MATCH and CHARISMA). None of the antiplatelet agents is able to significantly reduce mortality. The recent results of the PROFESS trial showed no difference between clopidogrel and aspirin with slow release dipyridamole in secondary stroke prevention.


TIME IS BRAIN, TIA AS AN EMERGENCY

NATAN 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.
1. The CADISS trial enrolled 250 subjects with extracranial carotid or vertebral dissection and randomly assigned them to antiplatelet or anticoagulant treatment for three months. Ipsilateral ischemic stroke occurred in 2% in the antiplatelet group and 1% in the anticoagulant group. There were no deaths in either group and one major bleeding event in the anticoagulation group.

2. Four multicenter, open-label randomized controlled trials (mr clean, escape, swift prime and extend-ia have demonstrated that early intra-arterial treatment with second-generation mechanical thrombectomy devices is superior to standard treatment with intravenous thrombolysis alone for ischemic stroke caused by a documented large artery occlusion in the proximal anterior circulation. The intra-arterial thrombectomy reduced disability and improved outcomes. All four trials enrolled overlapping, but not identical, patient populations and had generally similar results. The mr clean (the largest from the four trial) enrolled 500 adults within six hours of acute ischemic stroke onset caused by a proximal anterior circulation large artery occlusion and randomly assigned them to treatment with intra-arterial therapy or to usual care. Compared with usual therapy, the group assigned to intra-arterial treatment had improved outcomes, including functional independence, at 90 days.

New results from the swift prime trial show certain groups of patients with acute stroke in whom thrombectomy with a stent retrieval device was particularly effective. Mechanical thrombectomy after thrombolysis was superior to thrombolysis alone when initiated within 5 hours of stroke onset in patients with moderate to severe strokes due to large artery occlusion, according to intermediate results of the thrace study.

3. Patients with acute ischemic stroke receiving thrombectomy using the penumbra aspiration system showed a strong trend toward better outcomes than those receiving thrombolysis alone in the therapy trial.

4. Mechanical thrombectomy with a stent retriever system led to improved outcomes vs thrombolysis alone in patients with acute stroke caused by a proximal large-vessel occlusion treated within 8 hours in the revascat trial.

5. New data from a large international registry of thrombolysis in stroke suggest that tissue plasminogen activator (tpa, alteplase) is safe to use in patients with very severe strokes.

6. The first study of human neural stem cells delivered directly to the brain in patients with stroke has shown no major harmful cell-related effects over 2 to 4 years' follow-up, with a slight functional improvement.

7. New ich guideline of asa/aha may, 2015 (important statements)
   - Patients with ich should have intermittent pneumatic compression for prevention of venous thromboembolism beginning the day of hospital admission (class i; a; revised).
   - For patients with ich presenting with systolic blood pressure (sbp) between 150 and 220 mm hg and without contraindication to acute bp treatment, acute lowering of sbp to 140 mm hg is safe (i; a) and can be effective for improving functional outcome (iia; b).
   - Glucose should be monitored. Both hyperglycemia and hypoglycemia should be avoided (i; c; revised).
   - A formal screening procedure for dysphagia should be performed in all patients before the initiation of oral intake to reduce the risk for pneumonia (i; b; new recommendation).
   - Patients with cerebellar hemorrhage who are deteriorating neurologically or who have brainstem compression and/or hydrocephalus from ventricular obstruction should undergo surgical removal of the hemorrhage as soon as possible (i; b; unchanged).
   - Measures to control bp should begin immediately after ich onset (i; a; new recommendation).
AMYOTROPHIC LATERAL SCLEROSIS

VIVIAN DRORY
Neurology Department, Tel-Aviv University, Israel

Amyotrophic lateral sclerosis (ALS) is a relatively rare neurodegenerative disease of motor neurons in the brain and spinal cord that causes rapidly progressive weakness and atrophy of most muscles of the body. Although ALS is incurable and fatal, with a median survival of 3 years, treatment can extend survival and improve quality of life. Symptoms begin in most patients with limb weakness, but one quarter of patients have their first symptoms in the bulbar area (dysarthria and dysphagia). Cognitive impairment is recognized during the last decade as a relatively common feature.

There is to date no biological marker that can confirm the diagnosis of ALS, therefore a definitive diagnosis requires a period of observation to document progression and to exclude alternative diagnoses.

Hallmark findings in the electrodiagnosis of ALS are normal sensory nerve conduction studies and abnormal motor nerve conduction studies, with reduced motor compound muscle action potentials. The needle exam shows changes characteristic of ongoing denervation and reinnervation of muscles.

10% of the cases have a positive family history. In patients with familial ALS, genetic testing and genetic counseling may be appropriate.

The treatment of ALS includes riluzole that is able to slow disease progression, as well as symptomatic treatment for dysphagia (dietetic interventions and percutaneous gastrostomy), for respiratory insufficiency (non-invasive and invasive ventilation, diaphragmatic pacing), as well as medications for muscle cramps, spasticity, pain, depression, pseudobulbar affect.

Upon completion of this course attendants will improve their skills in order to diagnose and treat ALS and related motor syndromes.

CHRONIC INFLAMMATORY DEMYELINATING POLYNEUROPATHY

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Chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) is a chronically progressive or relapsing-remitting symmetric sensorimotor disorder of the peripheral nervous system. It is the chronic equivalent of acute inflammatory demyelinating polyradiculoneuropathy, the most common form of Guillain-Barré syndrome. CIDP typically starts insidiously and evolves slowly, in either a slowly progressive or a relapsing manner, with partial or complete recovery between recurrences.

Symptoms may include preceding infection, both proximal and distal limb weakness, non-predominant sensory symptoms, areflexia, autonomic disturbances.

Laboratory studies that may be helpful are cerebrospinal fluid analysis that may show elevated protein levels, electromyography (EMG) to determine whether the disorder is truly a peripheral neuropathy and whether the neuropathy is demyelinating, and in rare cases a peripheral (sural) nerve biopsy.

CIDP is an autoimmune disease that can lead over the years to significant disability, but can be treated successfully in many cases. The primary lines of treatment are corticosteroids, intravenous immunoglobulins and plasma...
exchange. In cases that are not well controlled by these treatments, rituximab, azathioprine or cyclophosphamide are indicated.

Upon completion of this course attendants will improve their skills in order to recognize and treat CIDP and other inflammatory polyneuropathies.

THE ELECTRODIAGNOSTIC EXAMINATION

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The electrodiagnostic examination is an extension of the clinical examination of the peripheral nervous system and muscles. It includes two parts: the nerve conduction study (NCS) and the muscle examination (EMG).

NCS is performed by electrical stimulation of motor and/or sensory fibers in specific nerves. The examination can contribute information whether a nerve lesion exists, if this lesion is focal or diffuse, axonal or demyelinating, and semiquantitative information regarding the degree of the injury. EMG is performed using intramuscular needle electrodes and can deliver information regarding both neurogenic and myopathic disorders that affects muscles.

During this course attendants will learn the physiologic basis of the electrodiagnostic examination, its indications and contraindications, its pitfalls and limitations, and will gain knowledge that will help to refer patients wisely to the examination and understand better electrodiagnostic reports.

CLASSIFICATIONS OF SEIZURES AND EPILEPSIES - UPDATE

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Epilepsy is a disorder of the brain characterized by the enduring predisposition to generate epileptic seizures, and by the neurobiologic, cognitive, psychological, and social consequences of this condition.

There has been a shift in the definition of epilepsy from the conceptual definition to an operational (practical) definition, adopted in 2014 by the International League Against Epilepsy (ILAE) task force. According to this update, epilepsy is now defined by any of the following conditions:

1. At least two unprovoked (or reflex) seizures occurring more than 24 hours apart
2. One unprovoked (or reflex) seizure and a probability of further seizures similar to the general recurrence risk (at least 60%) after two unprovoked seizures, occurring over the next 10 years
3. Diagnosis of an epilepsy syndrome.

From a pathophysiologic point of view, focal seizures are considered to originate within networks limited to one hemisphere whereas generalized seizure are thought to occur within and result from rapid engagement of bilaterally distributed systems.

The etiology of the epilepsies could be classified as genetic, structural-metabolic, or unknown. These terms substitute the previously used terms idiopathic, symptomatic and cryptogenic.
NEW GENERATION ANTIEPILEPTIC DRUGS

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Over the last two decades more than 15 antiepileptic drugs (AEDs) have been introduced to the global market. Randomized, placebo-controlled trials have yielded information about each drug’s efficacy, tolerability, and safety profile; however, few studies have compared the newer generation AEDs directly with the older generation. Comparative studies are not always straightforward in their interpretation, as many characteristics of drugs, both favorable and unfavorable, may not be highlighted by such studies. In general, findings from the literature suggest that the newer generation AEDs (including vigabatrin, felbamate, gabapentin, lamotrigine, tiagabine, topiramate, levetiracetam, oxcarbazepine, zonisamide, pregabalin, rufinamide, and lacosamide) have improved tolerability and safety compared with older agents such as phenobarbital, phenytoin, carbamazepine, and valproate. This is partially supported by some of the findings of the QSS and the TTA Committee of the American Academy of Neurology (AAN), whose review of four AEDs (gabapentin, lamotrigine, topiramate, and tiagabine) is discussed. The findings of the SANAD trial are also presented; the Adverse Events (AEs) rate for carbamazepine, lamotrigine, gabapentin and topiramate and valproate are compared. Such comparative trials are overall lacking for new AEDs, although some conclusions can be drawn from the available data. In the end, however, AED selection must be based on individual patient and drug characteristics.

TREATMENT OF THE FIRST SEIZURE: RISKS AND BENEFITS

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Unprovoked seizures are common, affecting approximately 4% of the population by age 80. Only approximately 30% to 40% of patients with a first seizure will have a second unprovoked seizure. The risk of a recurrence is highest during the period immediately after the initial seizure. The rate, at which first recurrences occur, drops off with increasing time since the first seizure (Hauser et al., 1990; Shinnar et al., 1996).

Decisions regarding treatment of single unprovoked seizures must balance seizure recurrence risk, the potential impact of a recurrent seizure, the likelihood of adverse effects of treatment, and patient preference. Careful history-taking and appropriate investigation together with a clear explanation provided to patient and family are important. It is essential to assure that the event, in fact, represents the first seizure. It is worth asking about partial events, absence seizures and myoclonic jerks. These may have been occurring for many years without a diagnosis having been made (Kwan & Brodie, 2000).

For the first unprovoked seizures, immediate treatment reduces the risk of seizure recurrence in the short term, but does not change the long-term prognosis for epilepsy. In fact, immediate antiepileptic drug treatment reduces the occurrence of seizures in the next 1-2 years, but does not affect long-term remission in individuals with single or infrequent seizures. (Marson et al., 2005).

Risk factors for seizure recurrence include a history of remote neurologic insult, epileptiform abnormalities on electroencephalogram, focal structural lesion on neuroimaging, and family history of epilepsy. Adult patients with these risk factors have a recurrence risk of 60% to 70% and usually should be treated with an AED to prevent seizure recurrence. Without risk factors, the recurrence risk is 20% to 30%, and treatment depends on individual risk-to-benefit ratios and patient preference (Herman, 2004).
The risks associated with prescribing antiepileptic drugs (AEDs) in a person who had a single unprovoked seizure fall into three domains: (1) the risk that treatment will not be effective in preventing seizure relapse, and the consequences thereof; (2) the risks of a person’s life being affected by the psychological, social, and legal aspects of receiving treatment for a seizure disorder; (3) the health risks associated with intake of antiepileptic medication. (Perucca et al., 2008). AEDs may cause a variety of adverse reactions, from quite mild to serious. Importantly, there are patient groups who show an increased risk of serious adverse drug reactions (ex. elderly) or special concerns (ex. women with childbearing potential - the risk of major malformations in offspring exposed prenatally to AED monotherapy is increased about twofold over the background rate) (Perucca, 2005; Morrow et al., 2006). AED interactions can have seriously adverse consequences, such as contraceptive failure in women taking oral contraceptives. The risk associated with AED interactions should be especially considered in patients with comorbidities. However, even in otherwise healthy individuals, including those not receiving any medication at the time an AED is prescribed, there is a chance of potential interaction in future, as AED treatment, as a rule, is initiated for years (Patsalos et al., 2002; Levy & Collins, 2007; Perucca, 2008).

The risk of the impact of AEDs on cognition deserves special attention. Recently an increasing evidence appeared that patients with newly diagnosed epilepsy are cognitively compromised even before the start of antiepileptic drug medication. The domains most affected are memory and psychomotor speed. It may be considered that cognitive deficits are not uncommon in patients with new-onset epilepsy since causative brain lesions, genetic influences and interictal epileptic activity are likely to exist before the first unprovoked seizure. Still, they might be caused or further aggravated by AEDs, social stigma, or a comorbid neurologic disorder (Helmstaedter et al., 2005; Taylor et al., 2010, Roshe et al., 2010). The findings of different cognitive trajectories in people with epilepsy, including recently diagnosed, and the general population were very important (Hermann et al., 2006; Seidenberg et al., 2007; Hermann et al., 2008). Baker et al (2011) demonstrated that at 12 month follow-up, people with epilepsy had significantly poorer scores for 9 of the 16 variables compared to healthy volunteers. The cognitive domains most affected were psychomotor speed, higher executive functioning, and memory. Future investigations of cognition in patients with single seizure and newly diagnosed epilepsy are needed in parallel with the experimental studies looking on the mechanisms of cognitive decline and possible preventive interventions.

Patients with single seizures should be counseled about seizure first aid and general safety measures, including precautions regarding swimming alone, engaging in high-risk activities, driving, possible seizure precipitation by photic stimuli (in generalized epilepsy), sleep deprivation, and alcohol.

NEUROLOGICAL EXAMINATION – TIPS AND TRICKS

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In this course the art of a rational neurological examination will be taught: More than in any other clinical disciplines in clinical neurology clever history taking and examination 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 as to the possible underlying disease problems. 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.
Within the last 10 years the number of survivors after stroke and traumatic brain injury (TBI) has dramatically increased due to advances in acute medical care.

In parallel the need for intensive neurorehabilitation to combat resulting impairment and handicap has increased. Fortunately also over the last 20 years neurologic rehabilitation is more and more conceived as applied neuroscience:

Dramatic progress has been made in the application of evidence based medical principles and the number of well designed randomized con-trolled trials in the field is increasing. Nevertheless there is a remaining epistemological problem in how far the rationales of EBM originally designed for pharmaceutical studies are really suited to as a source of beat evidence :Due to heterogeneity of populations ,usually comparably small sample sizes and hence also difficult to interpret metaanalyses the EBM rationale my sometimes be misleading.

Nevertheless a reasonable approach to design reasonable treatment strategies is to follow elementary rules derived from behavioural and neurosciences concerning neuroplasticity and learning mechanisms. This has resulted in the invention of better scientifically founded pro-cedures for neurological treatment of motor ,cognitive and language problems. A good example is the very successful application of the principle of forced use and avoidance of learned non use in con-strained in used movement therapy. This concept now also spreads to non motor fields as language , cognitive and perceptual rehabilita-tion.

Furthermore the use of intelligent mechanical training devices (often loosely called “robots” a has open news therapeutic windows especially in the early stage of treatment in severely impaired patients.

On the other hand pharmaceutical concepts for neuroprotection have more or less failed so fare possibly due to the selection of the wrong mostly monomodal drugs not properly addressing the complexity of the brain´s endogenous defense mechanisms at an early stage after injury. There is however a growing selection of neuromodulatory techniques such as peripheral nerve stimulation , non-invasive brain stimulation and also pharmaceutical interventions with monaminergic drugs and especially antidepressants to facilitate brain recovery within a limited time-window after stroke and TBI with the aim to reduce impairment.

In future it will be extremely important to differentiate more clearly treatment elements addressing compensation and task specific learn-ing from those elements addressing impairment reduction especially in the early sensitive period after an insult.

As treatment intensity is likely to be the key element for impairment reduction we certainly have to find clever and affordable ways: to in-crease the daily treatment time of our patients. To day even during inpatient rehabilitation treatment times hardly exceed three hours a day i.e. that we use only a small percentage of waking hours leaving long “idling” time not field by any treatment. In this sense we have to “reinvent” neurorehabilitation within this sensitive post injury period to combat impairment with high frequency treatments combined with neuromodulatory techniques (robot use, peripheral and central stimulation , pharmaceuticals).

We have to think how our rehabilitation environments should look like and can be “enriched” and how we can generate a high level of motivation and fun in patients to let them successfully participate in such high frequency treatments.

Furthermore prognostic criteria have to be worked out to enable deci-sions when to switch from impairment oriented( massed practice) to compensatory (task specific learning) strategies.
MEDICALLY UNEXPLAINED SYMPTOMS IN NEUROLOGY

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Medically unexplained symptoms in neurology (MUS) is a heterogeneous group of disorders which lack identified biological basis and are assumed to have a psychological origin. They are diagnosed by exclusion of an organic basis, as well as exclusion of feigning. MUS symptoms can be either positive (such as “epileptic” seizures) or negative (e.g. weakness). They can be accompanied by apathy (such as “la belle indifference”) or extreme anxiety (PTSD). The assumptions that all “functional” symptoms are psychogenic, and therefore can only respond to psychiatric therapy, has not been validated.

The separation of “organic” from “psychogenic” symptoms parallels the philosophical school of dualism (vs monism), implying that some processes are due to “mental” processes which are not physical.

The diagnosis of MUS requires exclusion of malingering and factitious disorders. It is almost impossible to prove the existence of feigning, and in many cases even a “maligner” may believe there is a justified source of the abnormality.

Treatment of MUS is disappointing.

DIABETIC NEUROPATHY – DIAGNOSIS AND SYMPTOMATIC TREATMENT

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

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

NEUROPHYSIOLOGICAL EXPLORATION IN EPILEPSY – THE FUNDAMENTALS

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Epilepsy is a disorder associated with hyperexcitability in the brain. Since the human electroencephalography was discovered in 1929, its potential application in epilepsy became clear and EEG continues to play the central role in diagnosis, prognosis and management of patients with epileptic disorders. However, EEG has many limitations and if we ask inappropriate questions, diagnostic errors could occur or the yield of information is poor. Epileptiform discharges in the interictal interval (IED) are highly predictive for epileptic disorders whereas background abnormalities or focal slowing are more related to the structural pathology associated with seizures. Interictal epileptiform activity could be also present in a small percentage of normal population, therefore its value
should be interpreted according with the clinical context. Misinterpretation of normal variants could also raise unwanted over-diagnosis of epilepsy. EEG studies could be applied with different protocols and have variable value during epilepsy evolitional stages. In this process we might need to indicate sleep studies or prolonged video EEG monitoring sessions. The indication of sleep recordings resides in differentiating seizures from parasomnia, studying the influence of seizures and medication on sleep architecture and diagnosing sleep co-morbidities.

Long term video-EEG monitoring is best recommended for presurgical evaluation or in difficult cases that should be differentiated from non epileptic attacks or classification challenges. In this set up, the most important target is to record seizures and analyze the clinical sequence synchronized with the EEG ictal pattern.

In focal epilepsy the most relevant ictal changes for localization occurs at the beginning of the episode during the first 10-20 seconds. Qualified personnel should apply specific tests to reveal certain clinical features as: loss of contact, aphasia, visual field deficit, post ictal paresis etc.

In conclusion: the use of different neurophysiology techniques and protocols highly depend on the disease stage and questions that we should answer in each patient case.

**SEMILOGY OF FOCAL SEIZURES IN TEMPORAL VS EXTRATEMPORAL EPILEPSY**

**IOANA MINDRUTA**

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Focal seizures are defined as occurring within networks limited to one hemisphere either very restricted or more widely distributed. The specific sequence of symptoms and signs that occur during the ictal events have a great relevance in localizing and lateralizing the seizure generators. This approach has a particular interest especially when we need to decide the pathological substrate, to differentiate between epileptic and non-epileptic events and for presurgical evaluation.

The most relevant element in the semiological picture is the signal symptoms.

These are defined as specific sensations (auras) that occur at the seizure onset and represent a hallmark of the symptomatogenic zone allowing to localize the seizure focus. The information regarding the content of auras is usually extracted from the interview and could also be tested during videoEEG monitoring sessions when recording ictal events. Some patients with focal seizures do not report auras. During the ictal sequence patients could also develop autonomic signs and/or alter the motor behavior and language or vision. These symptoms and signs might be related to ictal discharge propagation into areas distant from the ictal onset zone. These clinical manifestations are usually visible and could be described by the persons that witnessed the patient seizures. During focal seizures patients could loose consciousness and also could experience secondarily generalized seizures.

In conclusion: semiology of focal epileptic seizures is highly variable and depends on localization of the seizure focus as well as on neural networks involved in the propagation phase of the ictal discharge. The importance of each sign or symptom is established based on its sequence and specificity.
APOMORPHINE IN THE TREATMENT OF LATE STAGE PARKINSON’S DISEASE

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Once correctly diagnosed, the symptomatic treatment of early Parkinson’s disease appears to be relatively straightforward through the substitution therapy of dopamine.

With progressing disease and possibly as a result of previous therapies, later stages of Parkinson’s Disease become increasingly difficult to treat. Once “L-Dopa” induced (motor) fluctuations occur several initially oral therapies are being recommended and part of treatment guidelines.

At a subsequent stage patients may not be adequately controlled through oral or noninvasive therapies. At this stage, depending on the clinical pathology, the general conditions of the patient and the availability of the treatments a few treatment options are available which include two main avenues, deep brain stimulation (DBS) or continuous application of Levodopa or DA-Agonists.

Apomorphine is a very potent DA-Agonist and used in most countries for intermittent therapy and in large parts of Europe also for continuous therapy via subcutaneous infusion. The treatment options and possible pathways of optimization shall be discussed.

ADVANCES IN BRAIN PROTECTION AND RECOVERY IN STROKE THERAPY

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

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

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

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

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

Biological agents (e.g., neurotrophic factors and related molecules) with modulating and multimodal effects
are better pharmacological agents for brain 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 in stroke, TBI and neurodegenerative disorders.
This presentation will also focus on important therapeutic results of Cerebrolysin treatment in stroke and TBI.

RESULTS FROM A LARGE RETROSPECTIVE COHORT TRIAL IN TBI

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TBI is a field with many unmet needs in medicine and public health. It is a major cause of death and disability and
also leads to huge direct and indirect costs to society. Currently the incidence of TBI is increasing.
TBI populations are heterogeneous in terms of mechanism of disease, baseline prognostic risk factors, clinical
severity and evolution. This heterogeneity generates complex challenges.
New pharmacological approach together with more basic and clinical research is needed for better targeting TBI
therapy to the individuals.

The frequent progression of contusive brain injury indicates that this may constitute a subpopulation of TBI more
likely to benefit from acute neuroprotection (in the classic sense) by limiting processes involved in secondary brain
damage.
Other mechanisms, and consequently different approaches may be more relevant in patients with diffuse axonal
injury, and neuroprotection in a more broad sense also includes strategies and therapies aimed at promoting
regeneration or replacement of lost neuronal and glial cells, neuronal circuits, and stimulation of neuroplasticity
(neurorecovery).
The primary goal of pharmacological support in TBI is to reduce secondary damage (neuroprotection) and to enhance
repair (neurorecovery).
The current presentation will highlight the limits of monomodal drugs, the advantages of multimodal drugs and the
results of a large retrospective cohort trial with Cerebrolysin in traumatic brain injury.

SYMPTOMATIC TREATMENT IN MS

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Multiple sclerosis is the most common non-traumatic cause of disability in young adults. Characterized by a wide
range of functional impairments, still one of the most disruptive is the mobility impairment, influenced by other
various deficits and symptoms associated with the disease. Either in the first years or after 15 years of disease,
walking impairments is considered by the patients as the most concerning aspect related to their disease, followed
in importance by visual function and thinking/memory. The data collected by different studies and patients’ registries
proved that walking impairment hinders patients’ ability to perform daily activities, decreases the employment and
reduces health-related quality of life.
The therapeutic solutions include both pharmacological and non-pharmacological approach.
As the gait in MS is affected on many aspects, the medication is aimed to reduce spasticity, to increase speed and
balance, and consequently the patients’ quality of life.
In the category of symptomatic treatments addressed to mobility, PR-fampridine is the first indicated specifically for walking impairments in MS adult patients with EDSS between 4.0 and 7.0. Prolonged-release (PR)-fampridine is considered a potassium channels blocker, acting on the pathological mechanism which determine the delay of electrical impulse along MS damaged axons, due to leak of potassium ions within potassium channels. The benefits proved by PR-fampridine in clinical trials have been sustain by real world experience. Patients treated with PR-fampridine showed consistent improvements in walking speed, functional walking capacity, muscle strength and spasticity. PR-fampridine was also associated with statistically significant improvements in a broad range of physical activities and mental health status measured by the individual items and scores of QoL scales (MSIS-29 PHYS and SF-36 MCS), as early as 12 weeks after initiation through 48 weeks of treatment.

One of the most important non-pharmacological intervention is rehabilitation. Even if rehabilitation does not influence directly the progression of disease, many more recent studies seem to prove that it improves daily activities and participation in social activities, and that way quality of life. Its final objective is to improve self-performance and independence through alleviate the burden of symptoms responsible for progressive impairments and handicap. Rehabilitation is based on a comprehensive approach, considering the patient as whole, with his or her surroundings, relations and history and it is addressing to gait, balance, fatigue, exercise therapy, sensory dysfunction and neuropsychological function. An optimal exercise program, with both endurance and resistance training, could influence many parameters related to daily activities, functional capacity and balance. Even severe disabled patients could have benefits from physical exercises. As mobility is one of the most important aspects related to MS, the combination of a proper medication, the patients’ active attitude and exercise programs may improve significantly the profile of their disability and therefore their quality of life.

MULTIPLE SCLEROSIS AND PREGNANCY

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Multiple Sclerosis (MS) is the most common neurological disease of young adults that cause major disability. More than half of patients with MS develop the disease in their fertile period of life; Data available so far shows no negative long-term impact of pregnancy on MS progression.

However, a continuous short-term reduction of relapse rate

(RR) in the course of pregnancy typically occurs, followed by an increased RR after delivery. 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 presentation will summarize the most relevant results, in order to facilitate doctor’s and patient’s decisions.

Keywords: pregnancy, multiple sclerosis, disease-modifying therapy, relapse-rate
HYPERKINETIC MOVEMENT DISORDERS POST STROKE

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Movement disorders could be encountered after stroke. They could be hypokinetic or hyperkinetic. The most of them are hyperkinetic. The exact prevalence of movement disorders post stroke is not known exactly due to the fact that most reports are small case series or case reports. Temporal relationship between stroke and the onset of movement disorders is variable. Some hyperkinetic movements appear in the same time with the stroke, some appears at years after the stroke. Some patients could have two different types of movement disorders. From hyperkinetic movement disorders chorea appear earlier than dystonia.

Hemichorea after stroke is the most common hyperkinetic movement disorders. Dystonia is the second movement disorders after stroke (20%). It could be focal or hemidystonia. The great majority of hemidystonia after 50 years is post stroke. Tremor is encountered in stroke in mesencephalon, cerebellum, superior cerebellar peduncle, thalamus, and is rare in other topographical localizations. Ballism could be acute or subacute. It will discuss the phenomenology and topography of hyperkinetic movement disorders with illustrative videos.

DYSAUTONOMIA IN NEURODEGENERATIVE DISEASES
– INCIDENCE, FEATURES AND TREATMENT

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Dysautonomia is frequently encountered in neurodegenerative diseases, sometimes as an early manifestation (multisystem atrophy, Shy-Drager syndrome), other times occurring later, after years of clinical evolution (Parkinson’s disease, Alzheimer’s disease). In some disorders, such as progressive supranuclear palsy, the autonomic signs are exclusionary for initial diagnosis, however, after some time they become a rule, in this case mainly due to cardiovascular parasym pathetic system dysfunction. In amyotrophic lateral sclerosis at onset the dysautonomia is usually subclinical, but in the ventilatory failure stage hypertensive crisis and high heart rate are manifest as signs of autonomic storm. In Alzheimer’s disease the reverse might be true, since patients who are hypertensive in decades 5 or 6 tend to become apparently normotensive, actually dysautonomic, after the onset of cognitive clinical syndrome. In Parkinson’s disease the advanced stage is always characterized by a degree of dysautonomia. Multiple system atrophy is actually a spectrum disease, which balance the clinical picture on three axes: autonomic failure, parkinsonism and cerebellar syndrome.
PATHOPHYSIOLOGY OF THE BLOOD-BRAIN BARRIER IN CNS INJURY & REPAIR. NEW ROLES OF CEREBROLYSIN AND NANOMEDICINE

HARI SHANKER SHARMA1*

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Recent advancement in nanomedicine suggests that nano drug delivery using nanoformulation enhances neurotherapeutic values of drugs or neurodiagnostic tools for superior effects than the conventional drugs or the parent compounds [1,2]. This indicates a bright future for nanomedicine in treating neurological diseases in clinics. However, effects of nanoparticles per se in inducing neurotoxicology, if any is still being largely ignored [3]. The main aim of nanomedicine is to enhance the drug availability within the central nervous system (CNS) for greater therapeutic successes. However, once the drug together with nanoparticles enters into the CNS compartments, the fate of nanomaterial within the brain microenvironment is largely remained unknown. Thus, to achieve greater success in nanomedicine our knowledge in expanding our understanding of nanoneurotoxicology in details is the need of the hour.

In addition, neurological diseases are often associated with several co-morbidity factors, e.g., stress, trauma, hypertension or diabetes. These co-morbidity factors tremendously influence the neurotherapeutic potentials of conventional drugs. Thus, this is utmost necessary to develop nanomedicine keeping these factors in mind. Recent research in our laboratory demonstrated that engineered nanoparticles from metals used for nanodrug delivery significantly affected the CNS functions in healthy animals. These adverse reactions of nanoparticles are further potentiated in animals associated with heat stress, diabetes, trauma or hypertension. These effects nanomaterials were dependent on their composition and the doses used. Thus, drugs delivered using TiO2 nanowired enhanced the neurotherapeutic potential of the parent compounds following CNS injuries in healthy animals. However, almost double doses of nanodrug delivery are needed to achieve comparable neuroprotection in animals associated with anyone of the above co-morbidity factors. Thus, cerebrolysin delivered either though TiO2-nased nanowires or PLGA-nanoparticles effectively reduced brain pathology in several diverse neurological diseases often complicated with various co-morbidity factors. Taken together, it appears that while exploring new nanodrug formulations for neurotherapeutic purposes, co-morbidly factors and composition of nanoparticles require great attention. Furthermore, neurotoxicity caused by nanoparticles per se should be examined in greater details before using them for nanodrug delivery in patients.

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References
EVALUATION OF PARKINSON’S DISEASE – FEATURES OF ADVANCED STAGES

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Parkinson’s disease (PD) is a complex neurodegenerative disorder with no known etiologic treatment. Current classical therapies aim to keep striatal levels of dopamine within normal ranges. The efficacy they have in controlling symptoms is, however, variable, with afferent side effects, and known to diminish over time. Since advanced therapies available now (continuous intestinal infusion of levodopa-carbidopa, apomorphine pump and deep brain stimulation) can further control motor and non-motor symptoms in the late stages of PD, understanding the different phenotypes and key variables predicting a rapid progression towards advanced disease is important. Clinical factors such as age of onset, disease duration, motor phenotype, olfactory changes, sleep disorders, psychiatric and cognitive dysfunctions and impulse control disorders may provide information on the disease progression type. Certain biomarkers have also been linked to the progression of PD towards advanced stages—neuroimagistic studies, dopamine circuitry studies, genetic findings, and markers from serum and the cerebrospinal fluid may be risk factors for a more rapid course of the disease.

As it is important to understand the concept of advanced PD in the light of the recently available advanced therapies, further means of quantifying this stage become increasingly available. The scales for motor and non-motor symptoms (Unified Parkinson’s disease rating scale, Hoehn&Yahr, the non-motor symptom scale) are completed by patients’ questionnaires on quality of life (Parkinson’s disease questionnaire) on one hand and neuropsychological evaluation on the other hand (Beck’s depression inventory, mini mental state examination, the sleepiness scale) on the other hand.

MEASURING AND MANAGING DISEASE ACTIVITY

MIHAELA SIMU
Department of Neurology, Timisoara County Clinical Emergency Hospital “Victor Babes” University of Medicine and Pharmacy, Timisoara, Romania

Multiple sclerosis (MS) is currently defined as a chronic inflammatory autoimmune demyelinating and neurodegenerative disease of the central nervous system with a broad spectrum of morphological changes among which inflammation, demyelination, axonal damage and gliosis. Recent years have brought a significant amount of evidence that axonal damage is the major morphological substrate of permanent clinical disability, appearing from the early stage of the disease.

Epidemiological data highlight a two phase behavior of the disease in relation to reaching the cutoff EDSS 3. Phase 1, which probably corresponds to the mostly inflammatory stage of the disease, has a high temporal inter-individual variability (with a range of 5 to 25 years), while phase 2, which corresponds mainly with progressive degeneration, has a remarkably similar temporal profile for all the MS individuals (~5 years).

As the most currently used disease modifying therapies (DMTs) target the MS inflammatory mechanisms, it is justified and proved that early treatment initiation is correlated with delay of disability progression.

Therefore, it is important to evaluate each patient’s disease activity profile, the early course of the disease being predictive for the future evolution and prognosis. Clinical parameters such as relapses’ frequency and severity, disability score and also MRI parameters are currently used in monitoring and assessing the level of MS evolution. It is a priority among specialists to establish dependable, accurate and generally accepted algorithms and guidelines.
for assessing the degree of the disease’s activity.

Currently the Rio score and the Canadian MS Working Group Recommendations, are used to establish thresholds for clinical and MRI aspects that should be considered in treatment decision, as the moment of therapy initiation, the evaluation of the disease progression and treatment’s choice are key points.

LOW-GRADE NON-RESOLVING NEUROINFLAMMATION: AGE DOES MATTER

STEPHEN SKAPER
Department of Pharmaceutical and Pharmacological Sciences, University of Padua, Largo “E. Meneghetti” 2, Padua, Italy

Persistent inflammation, when manifested in the nervous system (‘neuroinflammation’) can be especially perilous, ranging from chronic and neuropathic pain to neurodegenerative diseases. Mast cells and microglia represent a key bi-directional highway linking peripheral immune signalling to the brain in an inflammatory setting. Pain in the elderly is an even more complex phenomenon than in adults due to progressive breakdown in cross-talk between the nervous and immune systems. During aging mast cells undergo a progressive modification in their reactivity (‘immunosenescence’) accompanied by increased sensitivity to pro-inflammatory mediators. Inadequate control of mast cells can lead to the release of proteolytic enzymes that adversely affect integrity and functionality of primary somatosensory neurons, leading to altered peripheral sensitization. Like mast cells, microglial activation is amplified and prolonged in the aged CNS compared with adults (senescent or “primed” microglia). ‘Aged’ microglia are capable of initiating and maintaining neuroinflammatory processes at the spinal level while modifying synaptic transmission supraspinally – especially the lateral or the median thalamus – where mast cells and their mediators are well-placed to target the sensorimotor or frontal cortex and elaborate sensation and conscious perception of pain. Within a framework of immune aging, mast cell-microglia cross-talk is positioned to create an amplification loop which interacts with peripheral and central neural structures - thereby influencing the development, persistence and intensity of neuropathic pain in the elderly. This altered mast cell / microglia reactivity may induce and maintain a state of permanent and unresolved neuroinflammation, as well. The latter is more likely to be of a lower level (‘low-grade non-resolving neuroinflammation’) but insidious all the same, as a low-grade inflammatory state may be encountered in chronic diseases which occur with particularly high frequency in the elderly (e.g. obesity, diabetes) are often co-morbid with chronic pain, and which can lead to the progressive disruption of neuro-cognitive and behavioural functions.
Multiple sclerosis is a chronic inflammatory disease of the central nervous system, that produces demyelination and axonal/ neuronal damage, resulting in characteristic multifocal lesions in the brain and spinal cord (visible on MRI), and various neurological manifestations. This polymorphism of signs and symptoms has been the hallmark of the disease since the first description, made by J.M. Charcot in the 19th century, MS being considered the great imitator of neurological diseases. Due to this complexity, the clinician facing the differential diagnosis should define to which category does his/her patient belong:

- Clinical and imagistic features typical for MS
- Initial episode typical for MS (CIS)
- Features typical for MS combined with atypical manifestations, which can be due to superimposed condition
- Atypical features (red flags) are present

The spectrum of disorders that can be confounded with multiple sclerosis include:

- Inflammatory/immune disorders: vasculitis, lupus erythematosus, Sjogren’s syndrome, Behcet’s syndrome, sarcoidosis, Wegener’s granulomatosis, Susac’s syndrome and others
- Infections: syphilis, Lyme disease, HTLV or HIV infection, mycolplasma, chlamidia
- Vascular: small vessel disease, CADASIL, migraine, SAPL
- Genetic/ degenerative: hereditary cerebellar ataxia, hereditary spastic paraparesis, Fabry disease, leucodystrophies, Nieman-Pick disease, Krabbe disease
- Metabolic: thyroid disease, vitamin B12 deficiency, copper deficiency
- Neoplastic: CNS lymphoma, intravascular lymphoma, paraneoplastic disorders, metastasis
- Spinal disorders: vascular malformations, tumor, degenerative spine disease.

The clinician will be guided by the medical history, clinical and imagistic aspects, requiring in certain situations special techniques, like MRS, or even brain biopsy, and sometimes an extensive work-up for ruling out an entire list of inflammatory or infectious disorders.

There are no perfect recipes for keeping the balance between the haste of the diagnosis, in order to start as early as possible the immunomodulatory treatment and the „wait and see” attitude, but one should try to follow an algorithm of the differential diagnosis, and in the end, take the right decision for the patient.
CURRICULUM VITAE
Anat Achiron, MD, PhD, is Full Professor of Neurology and Vice-dean for medical education 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 a 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 cognitive performance especially in the very early stages of the disease, and was involved in studies evaluating genetic markers associated with the diagnosis of multiple sclerosis, various disease types and prediction of disease activity and treatment response.

Prof. Achiron has published widely, with over 200 publications to her name, and has received numerous grants and scientific awards for her research work in medicine and neurology.
OVIDIU BAJENARU
ROMANIA

1983 : M.D. at the Faculty of Medicine of University of Medicine 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 : Ph.D. at the University of Medicine and Pharmacy “Carol Davila” Bucharest
1983-1985 : M.D. at the Faculty of Medicine of University of Medicine and Pharmacy “Carol Davila” Bucharest
- resident of neurology
- specialist in neurology, confirmed by the Ministry of Health of Romania
1993 : 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 Medicine - University of Medicine 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 Ministry of Education for PhD accreditation and high academic degrees

Post graduate training :

1996 : second medical competence (confirmed by the Ministry 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 multicentric 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 Ministry 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 Neurology (since 2014)
- American Academy of Neurology (coresponding 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)

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 multicentric trials
DANA BOERING
GERMANY

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

Academic qualifications:
1. Dr. medic : I.M.F. Cluj Napoca 1981
2. German acknowledgement as Dr. med. 1987

Employment:
St. Mauritius Therapieklinik Meerbusch since 2002

Professional appointments, scientific activities:
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
2009 Collaboration with the Coma Science Group Liege/Belgium
2010 Collaboration with the Neuroradiology of the Wake University Winson- Salem U.S.A. in a study on network properties of DOC patients
NATAN BORNSTEIN
ISRAEL

EDUCATION
1970-73 University of Sienna, Medicine, Sienna, Italy
1973-79 Technion Medical School, Haifa, Medicine, MD, 1979
Date of receiving specialization 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 (ACRSRS), 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 Neurosonology 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-present 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 Council
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 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).
Born in Romania (Galati).
Graduated in medicine from the University of Heidelberg, Germany in 1983.
Fellowship in electromyography and neuromuscular medicine at Louisiana State University Medical Center in New Orleans, LA, USA.

Since 2000 director of the Neuromuscular Service at Tel-Aviv Medical Center, Tel-Aviv, Israel (comprises EMG laboratory and neuromuscular clinic activities).

Associate Professor of Neurology at Tel-Aviv University since 2011.

My main clinical and research interests are amyotrophic lateral sclerosis (ALS) and other motor neuron diseases (Hirayama disease, primary lateral sclerosis) – genetics, epidemiology, biomarkers, nutrition, imaging aspects.
Over 100 publications in the medical literature.
FIRAS FAHOUUM
ISRAEL

Academic Education

1997-2004  MD, Hebrew University of Jerusalem, Israel
2004-2006  MSc in Neurobiology, Hebrew University of Jerusalem, Israel
2007-2009  Postgraduate Neurology Studies, Tel Aviv University, Israel
2010-2012  Epilepsy Research Fellowship, Montreal Neurological Institute, McGill University, Montreal, Canada

Clinical Experience

2007-2010  Neurology Resident, Department of Neurology, Tel Aviv Sourasky Medical Center, Israel
& 2012-2013  Senior Neurologist, EEG and Epilepsy Unit, Department of Neurology, Tel Aviv Sourasky Medical Center, Israel
2013- today  Senior Neurologist, EEG and Epilepsy Unit, Department of Neurology, Tel Aviv Sourasky Medical Center, Israel
Professor, Department of Neurology and Neurosurgery, Russian National Research Medical University and Director, Moscow Research and Clinical Center for Neuropsychiatry, Russia

Professor Guekht’s research interests are in epilepsy, neuroepidemiology and vascular dementia.

She received her MD degree from the 2nd Moscow Medical Institute and held a residency in Neurology at the same medical school where she completed PhD on EEG monitoring in carotid surgery and subsequently - doctoral dissertation on Brain plasticity and restoration after stroke.

She received several prestigious International Awards, including Bruce S. Schoenberg International Award in Neuroepidemiology for her research in post-stroke epilepsy.

Professor Guekht has authored more than 150 Pubmed-listed publications and 11 books on Neurology and Epileptology, including the National guidelines and Manual in Neurology; she serves on the Editorial Boards of several international journals.

She is the member of several Committees of the World Federation of Neurology and the European Federation of Neurological Societies, Secretary of the Commission on European Affairs of the International League against Epilepsy. Professor Guekht serves in the International Organizing / Program Committees for the several International and European Congress on neurology, epileptology, vascular dementia; she is the invited speaker at many International and European Congresses.
MEDICAL DIRECTOR
St. Mauritius Therapy Hospital Meerbusch

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

MEDICAL CAREER

1973 - 1980 School, Universities of Düsseldorf and Freiburg; Elective in Neurology at Boston City Hospital, Boston, Mass.; National Hospital for Nervous Diseases, London

since 1975 Junior researcher in the Department of Neuropsychology at the C. & O. Vogt Institute for Brain Research, Düsseldorf and the Department of Neurology, Freiburg (Prof. R. Jung)

1980 - 1981 Research fellow in the Department of Neuropsychology (Prof. G. Grünewald) at the C. & O. Vogt Institute for Brain Research, Düsseldorf

since 1981 Clinical training in the Department of Neurology (Prof. H.-J. Freund), Heinrich-Heine-University Düsseldorf

since 1985 Senior registrar in the Department of Neurology, Heinrich-Heine-University Düsseldorf

since 1987 Senior investigator for the German Research Council Special Task Force in Neurology at Heinrich-Heine-University (SFB 200 and SFB 194)

1987-2005 Medical director of the Neurological Therapy Center (NTC), Heinrich-Heine-University Düsseldorf

since 1988 Board examiner for Neurology at the local examination board (Ärztekammer Nordrhein)

1989-1997 Vice president of the German Society for Neurological Rehabilitation
1993 Habilitation in Neurology, Heinrich-Heine-University Düsseldorf

since 1995 Board examiner for physical medicine and rehabilitation (Ärztekammer Nordrhein)

1997-2005 Medical director of the Neurological Therapy Center, Cologne

1998-2004 President of the German Society for Neurological Rehabilitation

since 2000 Medical director and head of neurology, St. Mauritius Therapy Hospital, Meerbusch

since 2003 Secretary General World Federation for NeuroRehabilitation (WFNR)

since 10/2004 Vice president of the German Society for Neurological Rehabilitation

since 2005 Panel-Chairman Neurorehabilitation for European Federation Neurological Societies (EFNS)
AMOS D. 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 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 at 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.
IOANA MINDRUTA
ROMANIA

Neurologist, with competence in electrophysiology and special interest in epileptology, mainly presurgical exploration for epilepsy surgery. PhD thesis on “Sleep studies in epileptic syndromes”.

Current position - University Emergency Hospital in Bucharest in the Neurology Department - Epilepsy and Sleep Monitoring Unit, Coordinator in the National Programs for Pharmacoresistant Epilepsy and Rare Disorders.

Academic affiliation - lecturer in neurology at the University of Medicine and Pharmacy “Carol Davila” of Bucharest. Vicepresident of Romanian Association for Clinical Electrodiagnosis (ASNER) since 2009 and member in the board of Romanian Society of Neurology since 2013.
Dieter H. Meier is the CEO of Neuropore Therapies Inc., a San Diego based company, dedicated to the research and development of drugs interacting with misfolded proteins.

As a board certified neurologist, Dieter H. Meier was engaged over the last 20 years in a number of industry positions, mostly in drug development and general management. His contributions to the drug development at various stages let e.g. to the registration of Pramipexol, Apomorphine and the development of several earlier approaches. Most recently his team of scientists developed several small molecules interacting with α-Synuclein; one of these molecules was partnered with a large pharmaceutical company and is entering clinical development.
DAFIN F. MURESANU
ROMANIA

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. Professor Dafin F. Muresanu 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. Muresanu 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 Neurotraumatology (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 200 scientific participations in the last 7 years as “invited speaker” in national and international scientific events, a significant portfolio of scientific articles (113 papers indexed on Web of Science-ISI, H-index: 14) as well as contributions in monographs and books published by prestigious international publishing houses. Prof. Dr. Dafin F. Muresanu 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 of Medical Sciences “Gheorghe Marinescu Award” for advanced contributions in Neuroprotection and Neuroplasticity.
Cristina Aura Panea has graduated the University of Medicine and Pharmacy “Carol Davila” Bucharest in 1986. She has started the neurology specialty and her university teaching career in the Neurology Department of the University Emergency Hospital of Bucharest in 1991 and has obtained her PhD in Medical Sciences in 2000. Starting with 2003, she is Associated Professor and the Head of the Neurology Department of Elias Emergency University Hospital.

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

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

He is Head of the Department of Neurology, County Emergency Clinic Hospital from Brașov, and is Lecturer of Neurology at the Transilvania University from Brașov.

During his career Cristian Falup-Pecurariu was President of the European Association of Young Neurologists and Trainees (EAYNT), EAYNT Liaison Officer with World Federation of Neurological Society, co-representative of Europe on the International Working Group for Young Neurologists and Trainees (World Federation of Neurology), Secretary of the EFNS/MDS-ES Panel on Movement Disorders and currently is member of the Educational Committee of MDS-ES and MDS Leadership Task Force.

His research focuses on non-motor aspects of Parkinson’s diseases and restless legs syndrome.
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 Babeş’ National Institute of Pathology, Bucharest, Romania
2008- Senior Neurologist
2009 - 2012 Lecturer, ‘Carol Davila’ University School of Medicine
2009 - Senior Researcher, ‘Victor Babeş’ National Institute of Pathology, Bucharest, Romania
2012 - Associate Professor, ‘Carol Davila’ University School of Medicine and Head of Neurology Unit II, Colentina Clinical Hospital

Awards
1999 Beaufour-Ipsen prize for the best research study in neurology
2000 Young histochemist award - International Society of Histochemistry and Cytochemistry
2004 Diploma of scientific merit – ‘Victor Babeş’ National Institute of Pathology
2007 Romanian Academy award for medical research
2010 ‘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 Babeş’ National Institute of Pathology, Bucharest, Romania
Selected publications

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.
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 Padova, Padova, Italy (1997); Assistant Director, Molecular Neurobiology Research, SmithKline Beecham Pharmaceuticals, New Frontiers Science Park, Harlow, United Kingdom (1998-2001); Senior Team Leader, Migraine and Stroke Research, Neurology & GI Centre of Excellence for Drug Discovery, GlaxoSmithKline R & D Limited, Harlow, United Kingdom (2002-2003); Senior Team Leader, Neuro Cell Sciences/Neurodegeneration Research, Neurology & GI Centre of Excellence for Drug Discovery, GlaxoSmithKline R & D Limited, Harlow, United Kingdom (2004-2007); Senior Team Leader, Target Validation Dept (Cognition and Pain), Centre of Excellence for Drug Discovery, GlaxoSmithKline R&D Limited, Harlow, United Kingdom (2008); Adjunct Professor, Department of Pharmacology and Anesthesiology, University of Padua, Faculty of Medicine, Padua, Italy (2009-present).

PROFESSIONAL MEMBERSHIPS: Sigma XI (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, Nature Scientific Reports (Neuroscience); Councilor, International Association of Neurorestoratology

REVIEW PANELS: The Wellcome Trust (UK), Biotechnology and Biological Sciences Research Council (BBSRC) (UK), Austrian Science Fund (ad hoc review panel to evaluate interdisciplinary doctoral programmes in neuroscience)

RESEARCH INTERESTS: Molecular biology and cellular mechanisms of cell death in CNS aging, neurodegenerative disorders and neuroinflammation, astrocyte-microglia interactions, oligodendrocyte biology and diseases of demyelination. 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 290 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)

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

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

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

In the end of my half page, I am looking forward to future goals: development of basic research in MS in Romania, a National MS Registry, better drugs, a better education for patients and doctors, a better me...
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