5th MOVEMENT DISORDERS TEACHING COURSE

1-2 April 2016 | Hotel Alpin
Poiana Brasov | Romania

Welcome Address

It is a great pleasure to invite you to the 5th Movement Disorders Teaching Course in Poiana Brasov. Our event is designed for medical doctors involved in treating movement disorders patients. Senior physicians, residents, as well as students may find this education of value.

The Organizing Committee wishes you to have fruitful idea exchanges, interesting and interactive discussions.

This comprehensive two days course will approach various topics in the field of movement disorders. Video sessions will exemplify the phenomenology and will highlight the key learning message. Interaction with the audience and debate are strongly encouraged. The speakers at this course were selected based on their scientific experience, outstanding clinical reputation and excellent teaching skills.

We very much hope you will find our program of great interest and warmly wish you to have an enjoyable time in Poiana Braşov.

Course Director
CRISTIAN FALUP-PECURARIU

Co Chair Scientific Committee
OVIDIU BAJENARU

Co Chair Scientific Committee
DAFIN F. MURESANU

5th MOVEMENT DISORDERS TEACHING COURSE

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Course Director:

CRISTIAN FALUP-PECURARIU

Faculty of Medicine, Transilvania University
Head of the Department of Neurology,
County Emergency Clinical Hospital,
Brasov, Romania

Co Chair Scientific Committee:

OVIDIU BAJENARU

Honorary President of the Romanian Society of Neurology
University of Medicine and Pharmacy "Carol Davila" Bucharest
Director of the Department of Neurology, Neurosurgery and Psychiatry
Chairman and Head of Dept. Neurology - University Emergency Hospital Bucharest

Co Chair Scientific Committee:

DAFIN F. MURESANU

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

President of the Romanian Society of Neurology

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

Chairman "RoNeuro" Institute for Neurological Research and Diagnosis

Speakers

5th MOVEMENT DISORDERS TEACHING COURSE

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Faculty
(in alphabetical order)

Angelo Antonini /Italy
Ovidiu Bajenaru /Romania
Paolo Barone /Italy
Claudio Bassetti /Switzerland
Roberta Biundo /Italy
Cristian Falup-Pecurariu /Romania
Sharon Hassin-Baer /Israel
Peter Jenner /UK
Poul Jennum /Denmark
Jaime Kulisevsky /Spain
Monica Kurtis /Spain
Maria Marti /Spain
Pablo Martinez-Martin /Spain
Dieter Meier /USA
Dafin F. Muresanu /Romania
Alexander Münchau /Germany
Lacramioara Perju-Dumbrava /Romania
Bogdan O. Popescu /Romania
Peter Riederer /Germany
Fabrizio Stocchi /Italy
Claudia Trenkwalder /Germany

Scientific Program

Scientific Program

DAY 1 – 1st APRIL 2016

MORNING SESSION

08:45 - 09:00	Welcome address
Session 1 Chairman:	Ovidiu Bajenaru (Romania), Dafin F. Muresanu (Romania), Poul Jennum (Denmark)
09:00 - 09:30	Prodromal Parkinson - does it exist? Claudia Trenkwalder/Germany
09:30 - 10:00	Progressive supranuclear palsy Sharon Hassin-Baer/Israel
10:00 - 10:30	Social and welfare burden of movement disorders Poul Jennum/Denmark
10:30 - 11:00	Freezing of gait and falls in Parkinson's Disease Cristian Falup-Pecurariu/Romania
11:00 – 11:30	Coffee-break
Session 2 Chairman:	Roberta Biundo (Italy), Fabrizio Stocchi (Italy), Pieter Riederer (Germany)
11:30 - 12:00	Treatment impact on quality of life in advanced Parkinson's disease patients Dafin F. Muresanu/Romania
12:00 – 12:30	Pain associated with Parkinson Disease – clinical approach and pathophysiology Ovidiu Bajenaru/Romania
12:30 – 13:00	Advanced Parkinson disease: future outlook and new therapies Angelo Antonini/Italy
13:00 – 13:30	Gastroparesis and other gastrointestinal problems in PD: diagnosis and clinical management Fabrizio Stocchi/Italy
13:30 - 14:30	Lunch

DAY 1 – 1st APRIL 2016

AFTERNOON SESSION

Session 3 Chairman:	Claudia Trenkwalder (Germany), Angelo Antonini (Italy), Bogdan Popescu (Romania)
14:30 – 15:00	Non-dopaminergic approaches to treating Parkinson's disease Peter Jenner/UK
15:00 - 15:30	Complex sleep related movement disorders and their differential diagnosis Claudio Bassetti/Switzerland
15:30 - 16:00	Dystonia Maria Marti/Spain
16:00 - 16:30	Neuropsychological assessment in advanced Parkinson's disease Roberta Biundo/Italy
16:30 - 17:00	Coffee break
Session 4 Chairman:	Maria Marti (Spain), Sharon Hassin-Baer (Israel)
17:00 - 18:30	Video session Phenomenology of hypokinetic movement disorders

Scientific Program

DAY 2 – 2nd APRIL 2016

MORNING SESSION

Session 5 Chairman:	Monica Kurtis (Spain), Pablo Martinez-Martin (Spain), Paolo Barone (Italy)
09:00 - 09:30	Patient-reported outcomes: why, which, and how interpret their results? Pablo Martinez-Martin/Spain
09:30 - 10:00	Atypical Parkinsonisms and "Other" Atypical Parkinsonisms Monica Kurtis/Spain
10:00 - 10:30	Neurochemistry and connectivity of proprioceptive systems Peter Riederer/Germany
10:30 - 11:00	Dopaminergic continue stimulation in early and late Parkinson's disease - clinical cases presentation Bogdan O. Popescu/Romania
11:00 – 11:30	Coffee-break
Session 6 Chairman:	Claudio Bassetti (Switzerland), Jaime Kulisevsky (Spain), Cristian Falup-Pecurariu (Romania)
11:30 - 12:00	Natural history of non-motor symptoms in Parkinson's disease Paolo Barone /Italy
12:00 – 12:30	Disease-Modifying Therapies in Parkinson's Disease and other Synucleinopathies Dieter Meier/USA
12:30 – 13:00	Reevaluation of the therapeutic scope in early Parkinson's Disease Jaime Kulisevsky/Spain
13:00 – 14:00	Lunch

DAY 2 – 2nd APRIL 2016

AFTERNOON SESSION

Session 7 Chairman:	Peter Jenner (UK), Dieter Meier (USA)
14:00 - 14:30	The spectrum of tic disorders Alexander Münchau/Germany
14:30 - 15:00	Cardiovascular autonomic dysfunction in Parkinson s disease patients Lacramioara Perju-Dumbrava/Romania
Session 8 Chairman:	Lacramioara Perju-Dumbrava (Romania), Alexander Münchau (Germany)
15:00 - 15:30	Interactive clinical cases discussions: “Patients with hypokinetic movement disorders”
15:30 - 16:30	Interactive video cases: “Patients with hyperkinetic movement disorders”
16:30 - 17:00	Video session: “Complex movement disorders patients”
17:00 – 17:30	Final discussions

Continuing Medical Education Accreditation Points (CME)

The '5th Movement Disorders Teaching Course' is accredited by the European Accreditation Council for Continuing Medical Education (EACCME) to provide the following CME activity for medical specialists. The EACCME is an institution of the European Union of Medical Specialists (UEMS), www.uems.net

The '5th Movement Disorders Teaching Course' is designated for a maximum of (or 'for up to') 12 hours of European external CME credits. Each medical specialist should claim only those hours of credit that he/she actually spent in the educational activity.

Through an agreement between the European Union of Medical Specialists and the American Medical Association, physicians may convert EACCME credits to an equivalent number of AMA PRA Category 1 Credits™. Information on the process to convert EACCME credit to AMA credit can be found at www.ama-assn.org/go/internationalcme

Live educational activities, occurring outside of Canada, recognized by the UEMS-EACCME for ECMEC credits are deemed to be Accredited Group Learning Activities (Section 1) as defined by the Maintenance of Certification Program of The Royal College of Physicians and Surgeons of Canada.

EACCME credits

Each medical specialist should claim only those hours of credit that he/she actually spent in the educational activity. The EACCME credit system is based on 1 ECMEC per hour with a maximum of 3 ECMECs for half a day and 6 ECMECs for a full-day event.

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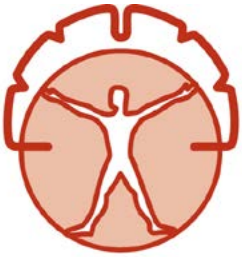

International Parkinson and
Movement Disorder Society

Abstracts

ADVANCED PARKINSON DISEASE: FUTURE OUTLOOK AND NEW THERAPIES

ANGELO ANTONINI

Director Parkinson Unit IRCCS San Camillo Venice and 1st Neurology Clinic, University Hospital of Padua, Italy

The concept of advanced stage of Parkinson's disease (PD) is now broader than in the past, encompassing various types and degrees of disability. It poses multiple management issues, because, despite levodopa still being unequaled in the symptomatic treatment of PD, its clinical effect tends to diminish with disease progression. Conventional therapies and routes of administration are slowly making way for new, innovative approaches, either invasive, such as the duodenal administration of the levodopa/carbidopa intestinal gel, the apomorphine pump, or non-invasive, such as transdermal, nasal, sublingual or pulmonic routes, all intending to optimize delivery. Moreover, additional drugs are required to adequately manage the full spectrum of emerging manifestations, characteristic of the advanced stage making an individualized integrated approach recommended.

Advanced PD raises multiple management issues, as the patient carries the burden of the motor complications and non-motor symptoms making medication adjustments quite complex. Adequate control and optimal quality of life are often difficult to achieve and an integrate understanding of the pathophysiological mechanisms underlying the symptoms and of the multiple drug interactions is mandatory. While this fine balance can be generally achieved to various extents in experienced tertiary centers, mortality remains roughly unchanged, supporting thus the need for further research and therapeutic options that can truly modify PD progression and its milestones.

PAIN ASSOCIATED WITH PARKINSON DISEASE – CLINICAL APPROACH AND PATHOPHYSIOLOGY

OVIDIU BAJENARU

Director of the Department of Neurology, Neurosurgery and

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

Chairman and Head of Dept. Neurology - University Emergency Hospital, Bucharest, Romania

Pain in PD is a non-motor symptom with an average prevalence of approximately 40%, associated with reduced health-related quality of life. It is often described both in early (rated as the most bothersome nonmotor symptom, ranked after the three motor symptoms - slowness, tremor, and stiffness) and advanced stages of PD (perceived as the sixth most troublesome symptom). In this disease more clinical and pathophysiological types of pain could be seen (musculoskeletal, neuropathic – with radicular and central variants, associated with dystonia, akathisia and other origins). It is often seen as a non-motor manifestation in "off" periods. Essentialy, in PD pain may be classified in two physiopathological types: nociceptive pain (directly related to motor symptoms) and neuropathic pain (resulting from an abnormal nociceptive information process). The neuropathic pain in PD could be related mainly to the impairment of the medial spinoreticulothalamic pathway of pain perception (subserving the autonomic, affective and cognitive dimensions of pain) which is a multisynaptic system of slow conducting fibers that projects to the medullary core and mesencephalon, implying many nuclei in the CNS (which often have α-synuclein pathology in different Braak stages in this disease); this pathway projects through intralaminar and medial thalamic nuclei to the insula, parietal operculum, anterior cingulate cortex, amygdala and hippocampus. There is indirect evidence of abnormal somatoensory processing within the basal ganglia and their interconnections; basal ganglia probably perform an important gating role for nociceptive information within the striatum and limbic system before it reaches consciousness. Some clinical and neuroimaging studies have reported lowered pain thresholds and abnormal activations of nociceptive areas in PD patients and, in addition L-dopa administration reduced pain sensitivity by raising subjective and objective pain thresholds and decreasing nociceptive brain areas hyperactivations. Recently (2015), different possible clinical profile subtypes within Parkinson's disease have been identified, according to the cases reported in the literature; among them, a Park pain subtype has also been identified, but it does not encompass all the clinical features of pain often described in the disease, but only the lower limb pain subtype. The Park pain subtype has a higher risk to develop disproportionate pain during the progression of PD compared to the motor severity of disease. A new pain classification for PD has recently been published, as a validated pain scale for PD has allowed exclusion of secondary pain in PD as far as possible (King's PD Pain Scale). The therapeutical approach of pain in PD is not unique, but it is related to the identification of the clinical and mainly pathophysiological pain subtype: manipulation of dopaminergic drugs, STN-DBS (but possible also GPi-DBS, as described in a small study), botulinum toxin, conventional analgesics, opiates, tricyclic antidepressants, some atypical neuroleptics (including clozapine) may be helpful; peripheral nerve blockade does not abolish the pain, supporting the notion that parkinsonian pain originates in the central nervous system.

NATURAL HISTORY OF NON-MOTOR SYMPTOMS IN PARKINSON'S DISEASE

PAOLO BARONE

Professor of Neurology and Chief of the Neurodegenerative Disease Centre at the University of Salerno,

Italy

Non-motor symptoms (NMS) have been recently recognized to be integral to Parkinson's disease (PD) at different stages of the disease: before the classical motor syndrome develops, at onset and throughout the course of the disease.

The non-motor symptoms associated with elevated risk of PD are constipation; depression, olfactory dysfunction and rapid-eye-movement sleep behaviour disorder. During the course of the disease, depression has been associated with poor quality of life while cognitive dysfunction and hallucinations predict the development of dementia in latest stages.

All non-motor symptoms are largely in agreement with the proposed pathophysiological progression of PD. Some of these non-motor symptoms are associated with dopaminergic striatal deficit, and therefore may be improved with dopaminergic therapy. Others are associated with several neurotransmitter systems and require multidimensional treatment strategies.

Despite of both the high frequency and clinical relevance, non-motor symptoms are often missed, misdiagnosed, and left untreated. Furthermore, side effects of antiparkinsonian drugs may induce similar symptoms increasing the difficulty to understand the pathophysiology of non-motor symptoms in PD.

Only recently, evidence of a natural history of non-motor symptoms has suggested the possibility of subtyping PD patients according to the spectrum of motor and non-motor features occurring at the onset and during the course the disease.

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COMPLEX SLEEP-RELATED MOVEMENT DISORDERS

CLAUDIO BASSETTI

Professor of Neurology, University of Bern

Director of the Neurology Department at the University Hospital, Bern, Switzerland

NREM and REM sleep are associated with profound neurophysiological and neurochemical changes in the brain, which lead to changes in the control of motor functions. This explains why movement disorders and disturbances of motor control sometimes appear only or preferentially during sleep.

The first part of the presentation will focus on epidemiological, clinical and etio-pathophysiological features of complex sleep-related movement disorders such as sleepwalking, REM sleep behavior disorder and their main differential diagnoses (e.g. sleep related hypermotor epilepsy).

The second part will discuss the relevance of these disorders related to their 1) association with sleep-associated injuries and violence; 2) effects on sleep (e.g. sleep fragmentation/insomnia) and wakefulness (e.g. sleepiness); 3) their diagnostic implications as first/main manifestation of an underlying brain disorders.

The third part will illustrate the diagnostic and treatment pitfalls of these disorders based on single case presentations.

NEUROPSYCHOLOGICAL ASSESSMENT IN ADVANCED PARKINSON'S DISEASE

ROBERTA BIUNDO

Neurospychologist, San Camillo Hospital Foundation, I.R.C.C.S, Venice, Italy

Parkinson's disease (PD) is a complicated, multi-dimentional disorder characterized by motor and non-motor features including cognitive dysfunctions. One of the major challenge when attempt to understand the nature and evolution of the cognitive deficits in PD is the marked heterogeneity amongst PD patients. Dementia prevalence is about the 80% in patients with 15-20 years of disease duration affecting quality of life for both patients and careers. However only the 25% of PD with dementia (PDD) is recognized during clinical practice. There is an urgent need, therefore, to identify preclinical PDD markers to implement therapeutic and supportive strategies at a stage of the disease were they are more likely to have greatest efficacy. Evidence suggests that mild cognitive impairment (MCI) in PD represents a risk factor for dementia. The attempt to standardize cognitive criteria for MCI did not help in the identification of a preclinical cognitive pattern, mainly due to methodological ambiguities (lack of an ideal neuropsychological battery and valid cut-off norms) that make the frequency of MCI diagnosis heterogeneous (Biundo et al 2013a). An adequate approach would be to first frame the cognitive problems and explore if there is insight with respect to the interference in daily life by an unstructured interview. One can then perform a neuropsychological evaluation to explore performance in tests thought to be more sensitive to predict cognitive decline (Biundo et al 2014) and follow-up sessions to monitor the possible cognitive decline. It is vital that any detailed PD cognitive assessment is undertaken by a qualified clinical neuropsychologist.

Reference

Biundo R, Weis L, Pilleri M, Facchini S, Formento-Dojot P, Vallelunga A, Antonini A. Diagnostic and screening power of neuropsychological testing in detecting mild cognitive impairment in Parkinson's disease.J Neural Transm (Vienna). 2013a Apr;120(4):627-33. doi: 10.1007/s00702-013-1004-2. Epub 2013 Mar 13.

Biundo R, Weis L, Facchini S, Formento-Dojot P, Vallelunga A, Pilleri M, Antonini A. Cognitive profiling of Parkinson disease patients with mild cognitive impairment and dementia. Parkinsonism Relat Disord. 2014 Apr;20(4):394-9. doi: 10.1016/j.parkreldis.2014.01.009. Epub 2014 Jan 22

FREEZING OF GAIT AND FALLS IN PARKINSON'S DISEASE

CRISTIAN FALUP-PECURARIU

Faculty of Medicine, Transilvania University, Department of Neurology, County Emergency Clinical Hospital, Brasov, Romania

Gait abnormalities represent one of the most frequent features of patients with Parkinson's disease. Some distinctive characteristics of walking disturbances include freezing of gait, festination, trembling-in-place, akinesia, balance impairments and falls. Freezing of gait, found in more than half of the patients with a disease duration longer than 5 years, has several provoking factors, like gait initiation, turning, approaching narrow spaces or stressful situations.

In general there are correlations with severity of the disease, akinetic rigid phenotype, cognitive impairment, insufficient response to levodopa.

There are subjective methods to assess freezing of gait (e.g. scales and questionnaires) and objective methods, like video recording and lower limb accelerometry. Falls can be found in 38-68% of patients with Parkinson's disease, being associated with increased age, cognitive impairment, severity and duration of the disease and orthostatic hypotension. Consequences of falls have an important impact on quality of life and can be related to an increased mortality risk.

The presentation will cover some key points of defining, physical examination, assessment, treatment and rehabilitation of these two prominent characteristics of gait disorders in Parkinson's disease.

PROGRESSIVE SUPRANUCLEAR PALSY

SHARON HASSIN-BAER

Director, Movement Disorders Institute

Senior neurologist in Sagol Neuroscience Center and Department of Neurology,

Chaim Sheba Medical Center, Tel-Hashomer, Ramat Gan Israel

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

Progressive supranuclear palsy (PSP) is the second most common neurodegenerative form of parkinsonism after PD. It typically manifests in the seventh decade of life and is clinically characterized by variable combinations of gait impairment, early postural instability, axial rigidity, bradykinesia, ataxia, slow vertical saccades progressing to supranuclear ophthalmoplegia, pseudobulbar palsy and frontal executive dysfunction. PSP is one of a diverse group of neurologic conditions, commonly referred to as tauopathies, that are clinically, morphologically and biochemically heterogeneous neurodegenerative diseases characterized by the deposition of abnormal tau protein in the brain, including also Alzheimer's disease, corticobasal degeneration, frontotemporal dementia and others. The neuropathological phenotypes are distinguished based on the involvement of different anatomical areas, cell types and presence of distinct isoforms of tau in the pathological deposits. In PSP the main affected areas are brainstem, diencephalon and basal ganglia leading to multiple neurotransmitter abnormalities, involving the dopaminergic, GABAergic, cholinergic, and serotoninergic systems. Clinical diagnostic criteria with good specificity but low sensitivity for the early stages as well as a clinical rating scale sensitive to progression are available. While the classical PSP subtype termed Richardson's syndrome is the most common one, there are other clinical phenotypes that are less well recognized and they will be presented. The H1 haplotype surrounding MAPT, the gene that codes for microtubule associated protein tau significantly increases risk for PSP and a large, multicenter genome-wide association study (GWAS) confirmed its role as a major risk factor for PSP.

While diagnosis remains clinical with some support of MRI, cerebrospinal fluid measures are showing promise as early-stage screening tools.

Interventional clinical studies in PSP have focused previously mainly on symptomatic treatment with drugs targeting dopaminergic, GABA-ergic or cholinergic neurotransmission; Advances in the understanding of the molecular basis of PSP have paved the way for well-designed disease-modification trials targeting tau-related mechanisms, mitochondrial dysfunction and restorative approaches.

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NON-DOPAMINERGIC APPROACHES TO TREATING PARKINSON'S DISEASE

PETER JENNER

Emeritus Professor, Neurodegenerative Diseases Research Group, Institute of Pharmaceutical Sciences, Faculty of Life Sciences and Medicine, King's College London, UK

The treatment of Parkinson's disease (PD) is centred on the use of dopaminergic medications to treat motor symptoms of the disorder. However, the pathology and biochemistry of PD is more extensive offering the opportunity to manipulate other neuronal receptor populations both within the basal ganglia and in other brain regions. This approach appears very logical, it would move therapy away for dopaminergic therapies and it could improve the treatment of both motor and non-motor symptoms of PD. However, so far, there has been limited investigation and limited success.

Drugs altering cholinergic and glutamatergic function (anticholinergics, amantadine) are commonly employed for treating motor symptoms but side effects limit their use. Other approaches to the treatment of motor symptoms (and notably dyskinesia) have included drugs altering noradrenergic, serotoninergic and histaminergic receptors and compounds acting on opioid, cannabinoid and adenosine receptors. These approaches have appeared effective in experimental models of PD but the translation to clinical effect in man has been poor.

Non-motor symptoms of PD only show a modest improvement to dopaminergic medication as they largely reflect non-dopaminergic pathology in brain. There has been little effort in developing drugs specifically for non-motor symptoms of PD and this is a reflection of the poor understanding of their pathological and biochemical basis and the failure to use appropriate animal models to unravel the pharmacological approaches needed.

The current state of the art initially appears bleak but in fact, it should be viewed as an opportunity that needs to be exploited as it offers the potential for improving knowledge on the relationship between pathology and symptomatology in PD and for novel pharmacological approaches to the symptomatic treatment of the illness.

SOCIAL AND WELFARE BURDEN OF MOVEMENT DISORDERS

POUL JENNUM

Professor,
consultant in clinical neurophysiology and neurology.
Danish Center for Sleep Medicine,
Department of Clinical Neurophysiology,
Rigshospitalet,
University of Copenhagen, Denmark

Parkinson's Disease (PD), atypical Parkinson's Diseases (AP) and related neurodegenerative disorders (ND) are common and highly debilitating and progressive disorders with significant personal, familiar and societal consequences. The disorders affects the population in late midlife with an increase with advancing age. Pre-diagnostic studies have shown that the diseases are associated with significant pre-motor morbidities years prior to the involvement of motor symptoms. The diseases causes significant health, social and societal consequences involving not only the patients but also the spouses and the families. Consequently the diseases causes significant welfare costs in term of direct and indirect costs and additional community burden and caregiver costs. Although progress have been observed in the medical and general management of disease, the associated comorbidities and mortality are significant, and currently there is no major medical intervention which has major impact on disease consequence. Current research suggest that if disease course should be modified this calls for 1) early disease identification and 2) effective disease modification interventions should be identified.

Due to impact of ND, there is an unmet need for focus on these disorders, diagnoses and management.

REEVALUATION OF THE THERAPEUTIC SCOPE IN EARLY PARKINSON'S DISEASE

JAIME KULISEVSKY

Associate Professor of Neurology at the Autonomous University of Barcelona, Research Professor at the Open University of Catalonia and Director of the Movement Disorders Unit of the Sant Pau Hospital in Barcelona, Spain

In the absence of neuroprotective treatments, the goal of medical management of Parkinson disease is to provide control of signs and symptoms for as long as possible while minimizing adverse effects.

Motor signs of the disease –the ones which usually lead to the diagnosis of PD- respond to drugs that enhance intracerebral dopamine concentrations or stimulate dopamine receptors. Levodopa, dopamine agonists and monoamine oxidase type B inhibitors, remain the pillars of treatment for motor symptoms both in early phases and along the course of the disease.

Evidence has shown that the rate of progression in early PD is faster than in later disease and studies demonstrate that a patient's quality of life deteriorates quickly if treatment is not instituted at or shortly after diagnosis.

Thus, once the diagnosis is made, there is no reason to delay the introduction of symptomatic treatment. Moreover, the rate of progression of PD appears fastest around the time of diagnosis and then decreases with time. Earlier intervention may lead to better long-term motor benefit and has also been shown to maintain PD-related quality of life compared to postponing treatment.

Interestingly, it has been hypothesized that the rapid progression may be caused by the decline of compensatory mechanisms, which maintain normal motor function while the number of dopamine neurones has fallen to 50% of normal. The maintenance of compensatory mechanisms would demand an extra-effort that might be harmful to the remaining neurones with a loss of functional ability that cannot be regained later in the disease course.

Treatment should be tailored to the individual patient. Bradykinesia and rigidity, and less so tremor, consistently respond to treatment with dopaminergic agonists or levodopa. The response of these symptoms to monoamine oxidase type B inhibitors in de-novo patients is less reliable. There is scant information on the use of amantadine in early phases of the disease. Although anti- cholinergic drugs, such as trihexyphenidyl, or clozapine might be of some benefit in selected patients with prominent tremor, its use is controversial.

Convincing evidence supports the notion that initiating treatment with dopamine agonists are associated with a lower incidence of motor fluctuations and dyskinesia than levodopa, especially in younger patients. Still, in all comparative trials of levodopa versus dopamine agonists, levodopa appeared superior to dopamine agonists in the degree of improvement of motor symptoms. As it occurs with levodopa, the advantages of dopamine agonists should be balanced against its particular profile of adverse events, individualizing the prescription in each new patient. MAO-B inhibitors may be effective in early PD and are not associated with significant motor complications; however, there is insufficient evidence to support a neuroprotective effect and experience tell us that the timeframe of effective monotherapy with these agents is relatively short. There is no evidence to support the use of COMT inhibitors added to levodopa in early PD.

Advances in genetics, neuroimaging, and a better knowledge of the signs and symptoms of the premotor period has allowed the identification of subjects at high risk of presenting 'motor PD' in the next future. Some of these subjects presents metabolic abnormalities of the basal ganglia dopamine metabolism. Further research is needed to demonstrate whether immediate dopaminergic treatment in these individuals has a beneficial effect with an acceptable adverse reaction profile, and whether immediate treatment is cost-effective.

Nevertheless, Symptomatic anti-Parkinson disease medications usually provide good control of motor signs of Parkinson disease for 4-6 years. After this, disability often progresses despite best medical management, and many patients develop long-term motor complications, including fluctuations and dyskinesias. Additional causes of disability in late disease include postural instability (balance difficulty) and dementia. Thus, symptomatic therapy for late disease requires different strategies.

The advantages of early initiation of treatment should be counterbalanced with the fact that treating patients earlier may lead to more standard dopaminergic adverse reactions (i.e., nausea, vomiting, and hypotension) and impulse control disorders (e.g., pathological gambling, compulsive shopping, and hypersexuality). Earlier treatment may also expedite motor complications (i.e., dyskinesias and motor fluc- tuations). We have no information on whether the benefits of immediate treatment are worth any increase in adverse reactions, although rasagiline showed comparable side effects with placebo in both TEMPO and ADAGIO.
Further research is needed to demmonstrate whether immediate treatment has a beneficial effect on quality of life with an acceptable adverse reaction profile, and whether immediate treatment is cost-effective.
Further research with large clinical trials of at least 5 years duration is needed to demmonstrate positive effects on patient quality of life outcome measures (e.g., PDQ 39) and health economics evaluations (cost utility and cost effectiveness).

Long-Term Monitoring
Patients with Parkinson disease must have regular follow-up care to ensure adequate treatment of motor and behavioral abnormalities. Once patients are stable on a medication regimen, provide follow-up care at least every 3-6 months, and periodically adjust medication dosages as necessary. Patients also need to be monitored for adverse events, including somnolence, sudden-onset sleep, impulse control disorders, and psychosis. In addition, patients should be evaluated and treated for emergence of clinically relevant nonmotor symptoms, including dementia, psychosis, sleep disorders, and mood disorders.

Drug-induced adverse reactions need to be regarded when deciding on the initial treatment for Parkinson's disease. Dopamine agonists and levodopa are both associated with nausea, daytime somnolence, and oedema, but these side-effects tend to be more frequent with dopamine agonists. Impulse control disorders, including pathological gambling, hypersexuality, binge eating, and compulsive spending, occur much more often with dopamine agonists. Dopamine agonists should therefore be avoided in patients with a history of addiction, obsessive-compulsive disorder, or impulsive personality
because these patients are at high risk for developing impulse control disorders. Dopamine agonists are also more commonly associated with hallucinations and are therefore usually not prescribed for elderly patients, especially those with cognitive impairment. Levodopa provides the greatest symptomatic benefit, but long-term use is associated with motor complications (dyskinesia and motor fluctuations; panel 3). To delay the onset of these complications, a levodopa-sparing initial therapy with a monoamine oxidase type B inhibitor or dopamine agonist can be considered. However, the findings of an open-label randomised trial of treatment of newly diagnosed patients with Parkinson's disease showed no major long-term benefit of a levodopa-sparing strategy, although younger onset patients (age <60 years), who are at greater risk of developing dyskinesia than older onset patients, were not well represented in this study.

A number of clinical questions dealing with the treatment of early PD will be addressed, suchs as: how do the first-line pharmacological treatments change the UPDRS motor score and affect the probability of motor fluctuations and dyskinesia?

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ATYPICAL PARKINSONISMS AND "OTHER" ATYPICAL PARKINSONISMS

MONICA M. KURTIS

Director Movement Disorders Unit of the Neurology Division of the Hospital Ruber Internacional, Madrid, Spain

Parkinsonism is defined by the presence of bradykinesia in combination with rest tremor, or rigidity, or both. Once this syndromic diagnosis is established and secondary causes are excluded, the clinician faces the challenge of making an etiological diagnosis. In the early stages of parkinsonism this task is challenging, even for movement disorder experts. The most frequent forms of neurodegenerative parkinsonism, after PD, are mutiple system atrophy (MSA), Progressive supranuclear palsy (PSP), and cortico basal degeneration (CBD). This video-based session will review the semiological characteristics of these conditions, emphasizing diagnostic pearls and "red flags", and briefly summarize currently available treatments. The differential diagnosis of these entities, including recently described genetic parkinsonian conditions, will also be discussed. Finally, an update on helpful ancillary tests that may aid in the diagnosis will be covered, including novel neuroimaging techniques, MIBG, PET and cranial ultrasound.

DYSTONIA

MARIA JOSE MARTI

Professor of Neurology, Consultant in Neurology and Director of the Parkinson's disease and Movement Disorders Unit of the Service of Neurology. Hospital Clinic i Universitari of Barcelona, Spain

Dystonia is a heterogeneous movement disorder, characterized by sustained or intermittent muscle contractions causing abnormal, often repetitive, movements, postures or both. Classification of dystonia is based on two main axes: clinical features including age at onset, body distribution, temporal pattern and associated features (additional movement disorders or neurological features) and etiology, which includes nervous system pathology and inheritance. By advances of genetics, including contemporary genomic technologies, several gene mutations causing dystonia, have been discovered in recent years, leading to a growing understanding of their molecular underpinnings. The clinical evaluation of a patient with dystonia, beginning with classification of the phenomenology of the movement disorder, followed by formulation of the dystonia syndrome, which finally leads to a diagnosis and investigations of the various underlying etiologies which can be due to inherited, heredodegenerative, secondary and idiopathic causes. Lacking a curative therapy, the treatment of dystonia is directed toward improvement in symptoms. Treatment options include pharmacological treatment (anticholinergics, dopaminergics and antidopaminergic drugs, baclofen, benzodiazepines), intramuscular botulinum toxin injections and peripheral and central (DBS) surgical procedures. If a treatable cause is identified etiology-based specific treatment should be given.

PATIENT-REPORTED OUTCOMES: WHY, WHICH, AND HOW INTERPRET THEIR RESULTS?

PABLO MARTINEZ-MARTIN

Neurologist and Scientist Researcher of the Spanish Public Boards of Research at the National Center of Epidemiology, Carlos III Institute of Health and Consortium for Biomedical Research in Neurodegenerative Diseases, Madrid, Spain

Patient Reported Outcome is a "patient's report of a health condition and its treatment" (FDA and PROs Harmonization Group, 2003). Patient's judgment (rather than clinician's evaluation) is central to the concept of PRO, a kind of measures that provide information about how the patients perceive their condition and the effect of the treatment. This information supplements the professional assessments and is mainly relevant in chronic or life-threatening conditions.

The term PRO includes measures aimed at evaluating Health-related quality of life, Perceived health status, Functional status, Satisfaction with treatment, Satisfaction with care, Well-being, and Preferences. These instruments may be classified as generic or specific for a particular situation (age, symptom, disease). Their content, availability, psychometric attributes, and use frequency must be taken into account to select the most suitable for the objectives of their application in clinical practice and research (Mov Disord 2010; 25: 2704-2716; Mov Disord 2011, 26: 2371-2380).

There are proposed standards for the development and evaluation of health measures, but also there are uncertainties about how to test and evaluate some measurement properties. Responsiveness and, subsequently, interpretation of results (e.g., after an intervention) lack of consensus with regard to which of the many methods available, usually not coincident in their results, is most appropriate. Frequently, in study reports is amazing the scarcity of data to inform appropriately on the importance of the change. An approach presenting simple data (e.g., % of change, effect size, proportion of patients improved/harmed, NNT, Minimally Important Change) can help to fill this gap.

DISEASE-MODIFYING THERAPIES IN PARKINSON'S DISEASE AND OTHER SYNUCLEINOPATHIES

DIETER MEIER

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Parkinson's Disease (PD) is a progressive neurodegenerative disease. The clinical manifestation can include motor and non-motor symptoms (eg. tremor, rigor, bradykinesia, postural instability and sleep disturbances, autonomic dysregulation impairment of cognition, alteration of affect). The neuropathological hallmark of PD and other synucleinopathies is the accumulation of alpha-synuclein (ASYN) containing cytosolic inclusions, called Lewy-bodies

Once correctly diagnosed, the symptomatic treatment of early Parkinson's Disease (PD) mainly based on dopaminergic therapies appears to be relatively straightforward . However, with progressing disease, and also possibly as a result of previous therapies, symptoms of later stages of PD become increasingly difficult to treat.

Several therapies have been proposed to not only alleviate the symptoms of PD but (also) to slow or halt the course of the disease progression. but to date, such putative 'Disease Modifying' approaches have not been borne out in rigourous clinical trials. This may be in part because: symptomatic effects have confounded the disease-modifying actions ; sensitive, validated biomarkers to measure disease progression are lacking and; the term disease-modifying has been mis-applied to symptomatic treatments for promotional purposes.

To develop a truely disease-modifying therapeutic, it will be necessary to stop the progression of the underlying pathophysiology. A tremendous amount of research has been devoted to understanding the role of misfolding and aggregation of the synaptic protein alpha synuclein (ASYN). Both human genetic studies and experiments in animals provide compelling links between the dysregulation of ASYN and PD. As with other misfolded proteins, it is likely that in PD the microscopically detectable Lewy bodies containg ASYN polymers are final deposits while earlier stages of aggregates are involved in the pathogenic process. Recent studies further suggest that membrane-embedded oligomers of ASYN may be a particularly toxic form of ASYN, resulting in disruption of synaptic function, loss of cell membrane integrity and ultimately, in neuronal degeneration.

Treatments that prevent the formation and accumulation of these toxic membrane-embedded oligomeric aggregates of ASYN may prevent further decay or even restore synaptic function in impaired systems and slow the rate of degeneration, thus providing a therapeutic benefit for patients. Treatment approaches that target the misfolding and aggregation process are currently being explored in early clinical studies with both antibodies and small molecules.

Another, therapeutic principle is to enhance the clearance of these protein aggregates by rectifying defects in a dysregulated clearance mechanism . Approaches aimed at enhancing clearance are still at the animal-testing stage but hold out promise because they may prove to be effective even after the disease has progressed to its later stages.

In summary, while the physiological role of ASYN is currently not fully understood, it is clear that the accumulation of misfolded forms of ASYN contributes to the pathology of PD and that preventing the formation of toxic oligomers and enhancing cellular clearance mechanisms may be a viable therapeutic approaches to halt or slow disease progression.

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

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Chairman "RoNeuro" Institute for Neurological Research and Diagnosis

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

THE SPECTRUM OF TIC DISORDERS

ALEXANDER MÜNCHAU

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Tics are sudden, rapid, recurrent and usually non-rhythmic movements or vocalizations that resemble caricatures of normal motor acts occurring in the wrong context or are exaggerated. In children, tics are by far the most common movement disorder. Transient tics can probably be considered part of the normal motor development. Tics can be simple or complex. They are typically preceded by premonitory sensations and fluctuate over time. They are suppressible for a considerable period of time to re-occur later, often with increasing intensity. Tics increase in severity during stress, tension, boredom or tiredness, they are less pronounced with intense concentration or when attention is diverted to other motor processes. Tics have to be distinguished from stereotypies, myoclonus and chorea. They can be classified according to the aetiology as primary or secondary tics. Primary tics are much more common than tics associated with neurodegenerative disorders or other causes. Primary tic disorders are classified according to complexity and duration. Transient tics are defined as lasting less than a year and chronic tics more than a year. According to DSM-V criteria, Gilles de la Tourette syndrome (GTS), by far the most common tic disorder, is characterized by the presence of multiple motor and phonic tics with onset before the age of 18 and a duration of more than one year. GTS often starts in pre-school age with a mean onset age of six years, increasing severity around the age of ten / eleven years and then remission in more than 50% of cases before the age of 18. In addition to tics, GTS is frequently associated with echo-, pali, and coprophenomena. Typical comorbidities include attention deficit hyperactivity and obsessive-compulsive disorder. Tics can be induced or worsened by drugs (e.g. cocaine). Tics are sometimes part of the clinical presentation of Huntington disease and are very typical in Neuroacanthocytosis. However, these diseases usually do not manifest in childhood. Complex tics have been described in pantothenate kinase associated neurodegeneration (PKAN) and a number of X- chromosomal disorders. Tics are also common in autism.

CARDIOVASCULAR AUTONOMIC DYSFUNCTION IN PARKINSON'S DISEASE

LĂCRĂMIOARA PERJU-DUMBRAVĂ

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Besides the well-known motor symptoms, Parkinson's disease (PD) patients show a large series of non-motor issues, including cardiovascular autonomic dysfunction (CAD). CAD occurs in almost all PD patients, and is reported even in the very early stages. They may represent a combination of peripheral sympathetic denervation (as documented with cardiac neuroimaging), and central mechanisms.

CAD main contributing factors include: loss of cardiac sympathetic noradrenergic nerves, extra-cardiac noradrenergic denervation, and arterial baroreflex failure. Antiparkinson medication can be also a potentially contributing factor. Clinically symptoms of CAD are complex including orthostatic hypotension, blood pressure lability, impaired heart rate variability, postprandial hypotension, supine hypertension, fatigue and exercise intolerance. Orthostatic hypotension occurs in 30-60% of all patients with PD, with or without symptoms. It increases the probability of falls and is an independent risk factor for mortality.

CAD should be commonly evaluated by physical examination (Heart rate variability, Valsalva ratio, Blood Pressure responses to Valsalva Maneuver, Head-up tilt table testing) imaging techniques (cardiac uptake of MIBG and 6-[18F] fluorodopamine) and plasma norepinephrine concentration. Cardiovascular imaging techniques showed decreased cardiac uptake already in the early stages of PD, even though routine clinical autonomic tests failed to detect any abnormality. The most significant and constant features of CAD in PD is the loss of noradrenergic myocardial innervations, validated by progressive loss of septal radioactivity.

Identifying and assessing the severity, the distribution and the rate of change of CAD may be useful to distinguish PD parkinsonism from other degenerative non-PD parkinsonism.

DOPAMINERGIC CONTINUE STIMULATION IN EARLY AND LATE PARKINSON'S DISEASE - CLINICAL CASES PRESENTATION

BOGDAN O. POPESCU

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Laboratory of Neuroscience and Experimental Myology, 'Victor Babes' National institute of Pathology, Bucharest, Romania

I will present here 3 clinical cases for which I will discuss the clinical details and the therapeutical decisions. The first one is a typical early case treated from the beginning with a dopamine agonist, the second one is noticeable due to a test of double advanced therapy (deep brain stimulation and intestinal levodopa/carbidopa gel) and the last one is a long period intestinal gel treatment case. The right moment for treatment initiation or change will be discussed.

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NEUROCHEMISTRY AND CONNECTIVITY OF PROPRIOCEPTIVE SYSTEMS

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While proprioceptive systems are useful targets in clinical and therapeutic strategies, their underlying neurochemical principles are less known. Neurotransmitters involved in proprioceptive actions are the classic ones, namely serotonin, catecholamines, acetylcholine, GABA and glutamate. However, also proteins/peptides as well as transcription factors play an important role in signal transduction processes. There is an interaction of those neurotransmitters within the afferent and efferent routes of transmission between spinal cord targets and the fibre systems of muscles. Within the peripheral nervous system afferent fibres make contacts to spinal interneurons at the site of the posterior horn. Those interneurons contact efferent fibres at the site of the anterior horn. Interneurons seem to be primarily gabaergic, occasionally modulated by catecholamines, while the major transmitter in the afferent and efferent fibres seems to be serotonin in addition to proteins/peptides (eg. ephrin-B2, Etv, neurotrophins) and transcription factors like Runx1, Retsignalling, plexin A1, Brn3a/Pou4f1 and others). There is coupling of afferent and efferent systems with acetylcholine, a major neurotransmitter of muscles. Muscle spindle sensory feedback is required for proprioception. Chemical relevant examples for the action of proprioceptive sensory neurons are given for models of amyotrophic lateral sclerosis, postural control in the elderly and gravity-related sensory information. The interaction of information flow between muscles, spinal cord and brain will be highlightened.

GASTROINTESTINAL DYSFUNCTION

FABRIZIO STOCCHI

Professor of Neurology, Consultant in Neurology and Director of the Parkinson's disease and Movement disorders research centre, University and Institute for Research and Medical Care IRCCS San Raffaele Rome, Italy

Gastrointestinal dysfunctions are recognized as symptoms of Parkinson's disease (PD) and constipation could even precede the motor symptoms. Studies suggest gastrointestinal symptoms are one of the most common non-motor symptoms in PD patients. These symptoms and dysfunction not only are very distressing for the patients but interfere with the treatment. The prevalence of gastrointestinal symptoms was recently evaluated in a questionnaire based study on 120 parkinsonian patients and matching controls. Gastrointestinal dysfunctions were significantly more prevalent in PD patients; the most frequent complaints were dry mouth, drooling, dysfagia, constipation and defecatory dysfunction.

The mobility and activities of daily living of parkinsonian patients depend on the blood level of levodopa. Levodopa (L-dihydroxyphenilalanine) is a large neutral amino acid (LNAA) and is absorbed only from the small bowel (mostly in the duodenum, with some absorption in the jejunum and ileum) which contains LNAA transporters. These drugs have a short half-life, therefore any factor which limits or delay levodopa absorption results in a reappearance of parkinsonian symptoms. Gastroparesis or delayed gastric emptying, can occur in Parkinson's disease can produce a variety of symptoms such as early satiety, abnormal discomfort with bloating, nausea, vomiting, weight loss malnutrition and most importantly interfere with levodopa absorption causing delayed-ON and NO-ON phenomena.

New data indicate that Alfasynuclein may affect gastrointestinal tract at the very early stage of PD giving the idea of potential early biomarker but also new idea about the pathogenesis of the disease.

Constipation is a very common and distressing symptom of PD, its pathophysiology and treatment will be discussed.

PRODROMAL PARKINSON – DOES IT EXIST?

CLAUDIA TRENKWALDER

Professor of Movement Disorders, University Medical Center of Goettingen, and Medical Director of the Paracelsus Elena Klinik, Center of Parkinsonism and Movement Disorders, Kassel, Germany

The diagnosis of Parkinson's disease (PD) continues to be established only by the presence of two of the three cardinal motor features: hypokinesia, tremor and rigidity, according to UK Brain Bank Criteria. While in recent years our knowledge about the complexity of both the variety of motor symptoms in PD, and its non-motor features has increased, how these symptoms develop over time provides an insight into the mechanisms of PD itself. Currently, it seems that motor symptoms are not the first but the last symptoms to emerge. Recently, there is a initiative of Parkinson experts worldwide, to look for pre-motor symptoms before definite motor symptoms evolve, and one possibility to denominate this stage is "prodromal Parkinson". The apparition of the first signs in the gastrointestinal system, in sleep-wake regulation, and in the autonomic nervous system, changes in pain perception and psychological changes have already altered a future PD patient's quality of life. When motor symptoms start, more than 50% of dopaminergic neurons are already degenerated, and long before the affection of these midbrain areas α-synuclein positive pathology occurs in the lower brainstem and olfactory bulb.

Some lessons about the premotor stage of the disease can be learned from the Honolulu-Asia Aging Study and now several other US and European cohort studies investigating prospectively the evolving prodromal phase of Parkinson. Part of the prodromal phase is:

(1) Impaired olfaction, (2) autonomic disturbances i.e.constipation and orthostatic hypotension, (3) cognitive alterations such as slow reaction time and impaired executive function, (4) sleep disorders such as excessive daytime sleepiness, insomnia and most specifically REM-sleep behavior disorder (RBD).

There is now also direct evidence that hyposmia, rapid eye movement (REM) sleep behavior disorder (RBD), constipation and depression can be present in the premotor period of PD. These observations were made years ago in clinical cohorts that developed idiopathic RBD, and now, with a more than 18-year follow-up more than 80% of the subjects of the cohort have developed a neurodegenerative disease. But even subtle motor features may precede the cardinal motor signs of PD. Despite controversial discussions in the literature, some publications report an increased prevalence of postural tremor preceding rest tremor in early PD by many years. As slowness of movements and general bradykinesia may go along with advancing age, it is extremely difficult to disentangle motor slowness due to ageing from subtle slowness of movements preceding PD, as the majority of PD patients are older than 60 years.

Although many of the above described features can be part of a premotor parkinson phase, unfortunatley none of these parameters could be shown in each single PD patient, some PD patients even miss many of these non-motor features or develop them later in the disease. Therefore the term “Prodromal Parkinson” has still to be elucidated by larger studies to delineate the most specific pattern of premotor signs.

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Biographies

ANGELO ANTONINI

/Italy

Angelo Antonini, MD, PhD is director of the Parkinson Unit at the Institute of Neurology, IRCCS San Camillo Hospital in Venice and Professor at the University of Padua.

He earned his medical degree from the Università degli Studi di Roma 'La Sapienza', Rome. In November 1990 he completed his neurology training with honors and then undertook a visiting fellowship at the PET Department Paul Scherrer Institute, Villigen, Switzerland before starting his PhD in neuroradiology under the supervision of Professor Klaus Leenders. In February 1995 he was promoted to Associate Professor of Neurology at the New York University and worked at the Neuroimaging laboratory of the North Shore University Hospital, NY directed by David Eidelberg. In November 1997 he moved to the Parkinson Institute in Milan where coordinated Clinical Research at the Department of Neuroscience until March 2009.

His research focuses on pharmacology of dopaminergic medications, neuroimaging as well as cognitive and behavioral aspects of Parkinson's disease, multiple system atrophy, PSP and other movement disorders. In addition he is actively involved in the use of continuous infusion of levodopa and apomorphine as well as deep brain stimulus (DBS) for the treatment of motor complications in advanced Parkinson e dystonia patients.

During his academic career he has received several awards, published more than 300 peer-reviewed manuscripts and several book chapters. He serves as reviewer for the main neurology journals. He is the Chair of the European Education Committee of the Movement Disorders Society, Treasurer of the Association of Parkinsonism and Related Disorders and Secretary of the Italian Parkinson and Movement Disorders Association LIMPE.

OVIDIU BAJENARU

/Romania

1983	: M.D. at the Faculty of Medecine of University of Medecine and Pharmacy "Carol Davila" Bucharest
1983-1985	: post graduate hospital stagium in University Hospital of Emergency Bucharest
1985- 1989	: resident of neurology
1985	: assistant professor – University of Medicine and Pharmacy "Carol Davila" Bucharest- Department of Neurology of the University Hospital of Emergency Bucharest
1989	: specialist in neurology, confirmed by the Ministery of Health of Romania
1993	: Ph.D. at the University of Medecine and Pharmacy "Carol Davila" Bucharest - senior lecturer of neurology - Head of Department and Medical Chief (University Hospital of Emergency, Bucharest
1994 - 1999	: Associate Professor of Neurology
1999 (since)	: Professor of Neurology at the University of Medicine and Pharmacy " Carol Davila" Bucharest and Chairman of the Neurology Department of the University Hospital of Emergency Bucharest
2006:	: Doctor Honoris Causa - University „Ovidius" – Constanta (Romania)
2011	: Director of Department of Clinical Neurosciences - University of Medicine and Pharmacy " Carol Davila" Bucharest
2013 (since)	: Corresponding member of the Romanian Academy of Medical Sciences

Other professional activities :

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

Post graduate training :

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

Fields of interest for the scientific research

- dementia and neurodegenerative diseases (in particular Parkinson's disease)
- multiple sclerosis

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

Other activities:

- coordinator of the Continuous Medical Education (EMC) national program of the Romanian Society of Neurology for neurologists in Romania
- coordinator and author of the Guidelines for diagnosis and treatment of neurological diseases (agreed by the College of Medecins of Romania)→main author of the national guidelines for Parkinson's disease,

Multiple Sclerosis and Dementia

- coordinator of the National Program of the National House of Insurance and Ministery of Health, for treatment of patients with neurological diseases (2000 - 2015)
- coordinator of the first medical team in Romania for DBS in Parkinson's disease.
- chief-editor of Romanian Journal of Neurology (the official journal of the Romanian Society of Neurology)

Scientific affiliation :

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

2005, 2006, 2010, 2011: awarded by the Prize of Excelence in Neurology for the scientific activity in Romania (decided by a National Jury organized by the Health Chamber of the Romanian Parliament)
2008: awarded by the Romanian Society of Internal Medicine for the best scientific activity in a related medical speciality
2014: awarded by the International Brain Foundation and Romanian Academy of Medical Sciences, for excellency in the development of management of patients with multiple sclerosis in Romania
Investigator in an International Program of Research for genetic factors in stroke patients; Country Principal Investigator – in more than 30 international, multicentric clinical trials;
Principal Investigator of the research site – in more than 30 international and national multicentic trials

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PAOLO BARONE

/Italy

Paolo Barone is professor of Neurology and Chief of the Neurodegenerative Disease Centre at the University of Salerno, Italy. He earned the degree in Medicine in 1980 and PhD in Neuroscience in 1988.

He is the author of over 300 published peer reviewed papers and book chapters. His research interests include: neuropharmacology of the dopamine system; clinical and genetics of movement disorders, especially related to Parkinson's disease (PD) and parkinsonisms. Recently, he has coordinated research on non-motor symptoms in PD and on cognitive function in neurodegenerative disorders with special focus on the relationships between cognition and behavioural symptoms including depression, ICD and hallucinations.

Prof Barone has lectured extensively at international meetings in Europe, Japan, Middle East and in United States. He also serves as a member of the International Executive Committee of the Movement Disorders Society and is past-president of the Italian Society for Movement Disorders.

CLAUDIO BASSETTI

/Switzerland

Prof. Claudio Bassetti was born and raised in Ticino (Southern Switzerland), is married and father of three boys. He speaks six languages and enjoys history, literature, music, sports, and travelling.

Claudio Bassetti received his MD degree form the University of Basel in 1984. He trained in neurology in Bern and Lausanne (FMH certification in 1992). He performed two research fellowships in basic neurophysiology (Basel, 1985-1986) and sleep medicine (Ann Arbor-Michigan, USA, 1995-6). In 1997 he became associate professor at the University of Bern. In 2000 he was appointed professor of neurology at the university of Zurich and director of the neurological outpatient clinics of the university hospital. In 2009 he founded the Neurocenter of Southern Switzerland in Lugano which he directed until 2012. Since 2012 he is full professor of neurology at the University of Bern and director of the neurology department at the University hospital.

Claudio Bassetti has authored eight books and over 350 scientific publications. His research interests include sleep, stroke, and movement disorders with a strong translational (human and animal/experimental) approach. He served as president of the European Neurological Society (ENS, 2013-4), European Sleep Research Society (ESRS, 2008-12) and Swiss Neurological Society (SNG, 2008-12). He was the founder and first president of the Swiss Federation of Clinical Neurosocieties (SFCNS, 2009-2013).

Currently he is board member of the Swiss Academy of Medical Sciences (SAMW) and of the scientific committee of the European Academy of Neurology (EAN).

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ROBERTA BIUNDO

/Italy

Roberta Biundo is currently working as a neurospychologist at the "San Camillo Hospital Foundation", I.R.C.C.S, Venice, Italy. She was awarded a Ph.D. in Neuroscience, EU Marie Curie Network, at the University of Hull (UK) exploring preclinical indicators of abnormal cognitive decline in ageing (2010). Previously, she was awarded a Marie Curie fellowship as research assistant at the University of Hull, (UK) (2007) where she investigated deterioration of language skills in patients with neurodegenerative brain diseases using experimental neuropsychological assessment, structural neuroimaging techniques as well as computerized assessments. She was awarded a fellowship to The Vivian Smith Advanced Studies Summer Institute of the Neuropspychological Society at Xilocastro (Greece) (2006) and obtained a master in Neuropsychology Across the Lifespan. She graduated with honors in Experimental Psychology at the University of Trieste (Italy) (2004) and underwent psychology training in Cognitive Psychology and Behavior analysis completing her psychologist training in Trieste (2005). Her main research focus is investigation of the interplay between cognitive-behavioral deficits, genetic variability, and neuronal substrates associated with Parkinson's disease, atypical parkinsonisms and Huntington's disease using neuroimaging techniques (e.g., MRI, fMRI, and cortical thickness). She also indentified indicators of neurorehabilitation treatment efficacy (cognitive training plus t-DCS) that may have prognostic value at the stage of mild cognitive impairment. She is involved in different multicenter study groups: 1) Multicenters longitudinal cohort-study in Lewy-body dementia (DLB): she leads the feasibility of cognitive test in detecting cognitive decline over time; 2)Validation of the Movement Disorders Society criteria for mild cognitive impairment in Parkinson's disease (PD-MCI); 3)Retrospective and longitudinal cohort studies in Multisystem Atrophy (MSA): Develop strategies for definition of dementia in MSA patients. She is currently working at developing strategies for defining cognitive high risk profile in Parkinson dementia and Dementia with Lewy Bodies. She also keen to develop valid global cognitive scales to detect high risk profile for Parkinson dementia. Moreover she actively working in better clarifying the dementia profile in MSA patients. Finally she is working at developing guidelines and detailed protocol recommendations for cognitive tests that may best detect cognitive decline over time in different movement disorders.

CRISTIAN FALUP-PECURARIU

/Romania

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

He hold a 1 year fellowship of the European Neurological Society in movement disorders and sleep medicine at Hospital Clinic, University of Barcelona, Spain.

During his career Cristian Falup-Pecurariu was President of the European Association of Young Neurologists and Trainees (EAYNT), EAYNT Liasion Officer with World Federation of Neurological Society, co-representative of Europe on the International Working Group for Young Neurologists and Trainees (World Federation of Neurology). He was also Secretary of the EFNS/MDS-ES Panel on Movement Disorders, member of the Educational Committee of MDS-ES and currently is member of the MDS Leadership Task Force and European Academy of Neurology Scientific Panel Movement Disorders. He is member of EUROPAR (European Parkinson's Group) and International Parkinson and Movement Disorders Society Non motor study group.

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

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

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SHARON HASSIN-BAER

/Israel

Dr. Sharon Hassin-Baer received her medical degree from the University of Tel-Aviv, Sackler Faculty of Medicine, and completed her residency in Neurology ate Sheba Medical Center, Tel Hashomer, Israel. She then trained in Movement Disorders with Professor Nir Giladi at Tel-Aviv Sourasky Medical Center, following which she returned to Sheba and opened the Parkinson's disease and Movement disorders clinic. In January 2014 the clinic had turned into an institute and she was nominated to direct it.

Dr. Hassin-Baer is a clinician and researcher whose practice and research focus on Parkinson's disease and other movement disorders, including ataxia, tremor, as well as on other rare neurogenetic disorders.

Dr Hassin-Baer is a member of the Movement Disorder Society (in which she is a member of the Membership and Public Relations Committee) and of the Israeli Neurological association (in which she is a member of the scientific committee and the head of the health basket committee). Dr Hassin-Baer is strongly associated with the Israel Parkinson Association for which she is serves on the medical advisory board.

Dr. Hassin-Baer has authored or co-authored over 60 articles, abstracts, book chapters and other materials on Parkinson's disease, deep brain stimulation, genetics of movement disorders and related issues, and has lectured widely on these topics all over Israel. She and her staff have presented the research findings in scientific meetings around the world. Her articles have been published in leading medical research journals in the field of neurology, including the Movement Disorders journal, Parkinsonism and Related Disorders, JAMA Neurology, Neurogenetics, Journal of Neurology, Neurology, Annals of Neurology and more.

Throughout her career Dr Hassin-Baer has held many teaching positions, beginning during her residence years in Nursing school, and ever since her specialization in Tel-Aviv University Medical School and school of physiotherapy in which she heads both undergraduate Neurology course and Movement Disorders Masters course.
Dr Hassin-Baer has been a principal investigator in several international studies of treatments for Parkinson's disease and other MD.

Her research has been supported by the generous donations of the The Bharier Medical Fund, Gonda Foundation, the Saia Foundation (Tel-Aviv University), the Ministry of Science and industry and by A MAGNET program supported by the Office of the chief Scientist (OCS) at the Ministry of the economy, Israel.

PETER JENNER

/UK

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

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POUL JENNUM

/Denmark

Professor, chief consultant in clinical neurophysiology and neurology. MD, DMedSc. Danish Center for Sleep Medicine, Department of Clinical Neurophysiology, Rigshospitalet, University of Copenhagen, Denmark
Board Certified in clinical neurophysiology and neurology. ESRS accredited Sleep Medicine specialist.
Medical education in Copenhagen University hospital, University of Minnesota, USA and Institute of Neurology, Montreal, Canada.

>240 international scientific peer reviewed articles, 2 textbook and 6 chapters in textbooks. 3 Patents. >600 international presentation and invited lectures.
Supervisor for numerous bachelor, master, phd-studerent. Evaluator for several PhD thesis.
Chairman of several subcommities regarding medical and guideline programmes, e.g. information technologies Committee, Copenhagen County (2000-2006), the 2006-7 chairman of the region's information technologies committee. Professional assessor Medicines Management related EU application and Professional assessor in EMEA. Chairman and national reference program for sleep apnea, The National Health Board (2006) and 2012 (revision). Chairman of Danish Sleep medicine society (1998-2012), Nordic sleep research Society (2002-4, 2010-2), Executive committee, ESRS (2006-12), WASM board member 2013-, EFNS scientific sleep committee chairman (2006-, ENS +2013), ESRS scientific board member (2014), AASM member (1990-).
Several international research collaborators.

Main scientific focus: sleep medicine research, early neurodegenerative markers in the ageing population. Societal burden of sleep and brain diseases.

JAIME KULISEVSKY

/Spain

Dr. Kulisevsky is Associate Professor of Neurology at the Autonomous University of Barcelona, Research Professor at the Open University of Catalonia and Director of the Movement Disorders Unit of the Sant Pau Hospital in Barcelona, Spain. He is also the Director of the Research Institute of this Hospital.

He conducts clinical research in Parkinson's disease and other movement disorders. His main research interest has been the cognitive and behavioral consequences of basal ganglia dysfunction in Movement Disorders and the impact of antiparkinsonian treatment on cognition and behavior in Parkinson's disease.

He has been member of the International Movement Disorders Society Task Force for Developing Rating Scales in Parkinson's Disease (Subcomitee for Cognitive Evaluation) and of the Task Force for Mild cognitive Impairment in Parkinson's Disease. He acts as the Spanish Coordinator of the European Huntington`s Disease Network and the ENROLL study (CHDI).

He has been awarded with the Research Prize of the Spanish Society of Neurology, has been Principal Investigator of several public research grants and industry-sponsored studies, as well as Principal Investigator of the Spanish Biomedical Network Research Centre for Neurodegenerative Diseases (CIBERNED-Instituto de Salud Carlos III).

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MONICA KURTIS

/Spain

Mónica M. Kurtis studied biology at Princeton University (USA) and biochemistry at the University of Edinburgh (Scotland) where she received her BSc. She completed her medical degree at the University of Navarre (Spain) and trained in neurology at the Hospital Clínico San Carlos in Madrid (Spain). She undertook a clinical fellowship in Movement Disorders and Clinical Motor Physiology at Columbia Presbyterian Medical Center (USA).

She is currently a clinical neurologist and directs the Movement Disorders Unit of the Neurology Division of the Hospital Ruber Internacional located in Madrid, Spain. She has published extensively in peer-reviewed international journals and is the author of various book chapters. She is a reviewer for Movement Disorders among other journals. She collaborates with national patient associations involved with Parkinson disease, dystonia and Tourette as a medical advisor. Her main fields of interest include Parkinson's disease (particularly non motor symptoms and quality of life), atypical parkinsonisms, tremor, dystonia and patient/care giver education.

MARIA MARTI

/Spain

Maria Jose Marti MD, PhD, is Professor of Neurology, Consultant in Neurology and Director of the Parkinson's disease and Movement Disorders Unit of the Service of Neurology. Hospital Clinic i Universitari of Barcelona. Director of the "Neurodegenerative disease: Clinical and Experimental Research Group" Institut d'Investigacions Biomediques Agusti Pi I Sunyer (IDIBAPS).

She is also the coordinator of the Center of Rare Disease with Movement Disorders , Hospital Clinic-Hospital Sant Joan de Deu , Barcelona. Her research activities have centered mainly on dystonia and clinical, radiological and biochemical markers of Parkinson's disease and atypical parkinsonism. She is the author of more than 150 papers in peer-reviewed scientific journals and reviewer in several international medical journals

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PABLO MARTINEZ-MARTIN

/Spain

Neurologist and Scientist Researcher of the Spanish Public Boards of Research at the National Center of Epidemiology, Carlos III Institute of Health (ISCIII) and Consortium for Biomedical Research in Neurodegenerative Diseases (CIBERNED – ISCIII), Madrid, Spain, since 2006.

He has been Scientific Director of the Research Unit for Alzheimer's Disease at the Alzheimer Center Reina Sofia Foundation (2006-2014); Head of the Section of Neuroepidemiology at the National Center of Epidemiology, ISCIII, Madrid, Spain (2001-2006); and Head of the Department of Neurology in the Hospital de Getafe (Madrid) (1991-2001) and Central Red Cross Hospital (Madrid) (1988-1991). Associated Professor of Neurology, Department of Health Sciences, School of Medicine, Complutense University, Madrid (1989-1993).

He has received 14 awards in Neurosciences and Aging. Member of several international Study Groups, Steering Committees for research, and Task Forces of the Movement Disorder Society and the European Federation of Neurological Societies. Chair of the Committee on Rating Scales Development of the International Parkinson and Movement Disorder Society since 2013.

Main research lines of Dr. Martinez-Martin are: Assessment instruments (rating scales, questionnaires), Patient-Reported Outcomes, particularly health-related quality of life; Outcomes research; Neurodegenerative diseases, particularly Parkinson's and Alzheimer's diseases; Dementia.

He has published over 375 articles in scientific journals, 99 chapters of book and 17 books as editor or co-editor. He has given 173 lectures as Invited Professor in teaching institutions, 176 talks in scientific events, and presented more than 415 reports to congresses. Chair and Convenor of 55 Scientific meetings and Teaching courses.

DIETER MEIER
/USA

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 aiming at changing the course of neurodegenerative diseases.

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 to the registration of e.g. Actilyse for stroke, Pramipexol and Apomorphine in Parkinson's Disease (PD), and the development of several earlier approaches.

Most recently, his team of scientists developed several small molecules interacting with αSynuclein. One of these molecules was partnered with a large pharmaceutical company, and is jointly being developed in the clinic with the target to modify the course of PD.

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 Neurotraumatolgy (AMN) Congresses, Congresses of European Society for Clinical Neuropharmacology, European Congresses of Neurorehabilitation). His activity includes involvement in many national and international clinical studies and research projects, over 300 scientific participations as "invited speaker" in national and international scientific events, a significant portfolio of scientific articles (120 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.

ALEXANDER MÜNCHAU

/Germany

Prof. A. Münchau studied medicine in Hamburg and Berlin, Germany. After a formative final year clinical attachment with Dr. John Patten in Guildford, UK, he was trained in Clinical Neurology and Clinical Neurophysiology in the Neurology Department of St. Georg Hospital in Hamburg. With support from the Jung Stiftung für Wissenschaft und Forschung (Hamburg) and the Tourette Syndrome Association (USA) he spent 3 years at the National Hospital for Neurology and Neurosurgery, Queen Square, London under the supervision of David Marsden, Niall Quinn, Kailash Bhatia, Mary Robertson and Michael Trimble sub-specialising in Movement Disorders and Neuropsychiatry. During that period, he carried out experimental neurophysiology research at the Institute of Neurology, predominantly in the Human Movement and Balance Unit, in the groups of John Rothwell, Adolfo Bronstein and Michael Gresty. From 2001 to 2013 A. Münchau was working in the Neurology Department of Hamburg University Hospital where he became Consultant in 2003 and Deputy Head of Department in 2005. Supported by the Volkswagenstiftung, the Deutsche Forschungsgemeinschaft (DFG; German Research Council) and European research support he set up a Movement Disorders and Motor Systems Neuroscience group with a special focus on paediatric movement disorders and the pathophysiology of paediatric and adult movement disorders. In 2013, he became head of the newly founded interdisciplinary Department of Paediatric and Adult Movement Disorders and Neuropsychiatry in the Institute of Neurogenetics at Lübeck University. A. Münchau is speaker of the Lübeck Center for Rare Diseases, head of the Habilitation Committee of the University of Lübeck and chairman of N.E.MO., a charity for the support of clinical care and research of paediatric movement disorders. He is also founding member of the “Agentur für Überschüsse” (Agency for Surplus), a Neuroscience / Theatre science / Philosophy group addressing the implications and repercussions of movements deviating from set rules and norms in science, society and the arts.

LACRAMIOARA PERJU-DUMBRAVA

/Romania

Lăcrămioara Perju-Dumbravă, MD, PhD is Professor of Neurology within the Neurosciences Department, Faculty of Medicine, University of Medicine and Pharmacy "Iuliu Hatieganu" Cluj-Napoca, Chairman of the First Neurology University Clinic, Cluj-Napoca, Romania. Her academic status includes her position as member of the Board of the Faculty of Medicine and of the University's Senate, as well as Doctorate coordinator in the field of MEDICINE. Her prestigious activity includes: publishing of 3 monographs, co-authorship in other 7 speciality books, 168 scientific papers published in medical journals, chairman and speaker at annual national congresses and conferences, international conferences and membership in editing committees and professional societies, involvement in several clinical studies, her expertise being sought by national medical councils and committees.

BOGDAN O. POPESCU

/Romania

Bogdan O. Popescu graduated the School of Medicine at "Carol Davila" University of Medicine and Pharmacy, Bucharest, in 1996. From 1997 to 2012, he worked as neurologist and Assistant Professor at the University Hospital Bucharest.

Since 2015 he is full Professor of Neurology at "Carol Davila" University, School of Medicine, at "Colentina" Clinical Hospital and President of the Scientific Council at "Victor Babeş" National Institute of Pathology in Bucharest.

He graduated two PhDs, in Bucharest, in 2001, and in Stockholm, Sweden (Karolinska Institute) in 2004, with theses regarding apoptosis and cell signaling in neurodegeneration, respectively.

His scientific contribution refers mainly to mechanisms of neurodegenerative diseases (Alzheimer's and Parkinson's).

He authored over 40 papers in ISI high impact factor journals, being cited more than 1000 times in international publications and having a Hirsch index of 17.

He is member of EAN, MDS, ESO, Romanian Society of Neurology, Society for the Study of Neuroprotection and Neuroplasticity and National Society of Neuroscience.

He was honored with the "Victor Babeș" award for medical research by the Romanian Academy in 2007. During 2001-2013 period he served as General Secretary of the Romanian Society of Neurology. He is the Executive Editor of the Romanian Journal of Neurology since 2001.

He is the Elected President of Romanian Society of Neurology, starting with 2017.

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PETER RIEDERER

/Germany

since 2010	Senior Professor at the University of Würzburg, Medical School, Germany
1986 - 2010	University-Professor (University Würzburg); Head, Clinical Neurochemistry, Department of Psychiatry, Psychosomatics and Psychotherapy at the University of Würzburg, Medical School, Germany
1983	titl. a. o. University-Professor (TU Vienna)
1979	Associate Professor (University-Dozent) TU Vienna
1971 - 1986	Head, Clinical Neurochemistry, Ludwig Boltzmann Institute (LBI) for Neurochemistry (1971 - 1975) and LBI Clinical Neurobiology (1976 - 1986), Lainz-Hospital, Vienna, Austria
1970	Doctor techn. Degree
1969 - 1971	Assistant Professor

Honors and awarded memberships (selection)

2013	Honorary Member, Austrian Society for Parkinson's Disease
2012	Edit. Board Member, International Association of Neurorestoretology (IANR)
2011	WFN - Association of Parkinson Disease Related Disorder- Lifetime Award
2008	Honorary Dr. degree International University Catalunya, Barcelona, Spain
2007	Honorary Member of the Hungarian Academy of Sciences; Member of the Deutsche Akademie der Naturforscher Leopoldina; Honorary President of the German Society for Parkinson's Disease
2006	Honorary membership of the German Society of Biological Psychiatry
2005	Honorary membership of the Austrian Alzheimer Society
2004	Most cited chemist in the field of medicine
1991	AGNP - Award, Award for psychopharmagological research
1986	Senator Dr. Franz Burda-Award

Project coordination, membership in collaborative research projects (selection)

current	International joint project in the field of clinical and experimental studies on Parkinson's disease and dementia of Alzheimer type with: M.B.H. Youdim (Haifa), T. Nagatsu (Aichi), M. Naoi (Gifu), W. Maruyama (Aichi), Z. Lackovic, M. Salkovic (Zagreb) and E. Grünblatt (Zürich)
2004 - 2011	DAAD-Stability Pact Project : Establishing the role of diabetes type II as risk factor for Alzheimer's disease (with S. Hoyer, M. Salkovic, E. Sofic, E. Grünblatt)
2002 - 2012	Brain Net Europe II: Standardization of human post-mortem brain studies at an European level (Europen FP 7 project)
2002 - 2008	BMBF Kompetenznetz HIV/AIDS
2002 - 2008	DFG-project "Benzodiazepines"
2000 - 2012	VITA - Project (Vienna Transdanube Aging Study): A prospective longitudinal aging study to elaborate risk factors for AD
1999 - 2012	Head of the Brain Bank Center (BBC) Würzburg of the National Brain-Net, Germany
1991-1998	BMBF Schwerpunkt "Parkinson"

More than 1.100 publications in the field of Neuroscience

FABRIZIO STOCCHI

/Italy

Fabrizio Stocchi, MD, PhD, is Professor of Neurology, Consultant in Neurology and Director of the Parkinson's disease and Movement disorders research centre and director of the drug development research centre at the University and Institute for Research and Medical Care IRCCS San Raffaele Rome. He is also Scientific advisor of the Institute for Parkinson's Disease Research in Vicenza. Professor Stocchi was awarded his MD from the University of L'Aquila and his PhD from the University of Catania.

Professor Stocchi's research activities have centred on neuropharmacology in the field of movement disorders and neurodegenerative diseases. He has published many books and papers on the genetics, clinical diagnosis, characterisation and treatment of Parkinson's disease, as well as in preclinical research into the disease. He is an active member of 11 societies, including the Movement Disorders Society, the WFN society where is member of the extrapiramidal committee, the European Clinical Neuropharmacology Society and the European Federation Neurological Society.

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CLAUDIA TRENKWALDER

/Germany

Claudia Trenkwalder, MD, Neurologist, is Professor of Movement Disorders at the University Medical Center of Goettingen, Germany, and Medical Director of the Paracelsus Elena Klinik, Center of Parkinsonism and Movement Disorders, in Kassel, Germany since 2003. She started her clinical education in neurology and movement disorders at the University Hospital of the Ludwig Maximilian University in Munich in 1988 and was head of the "Movement Disorders and Sleep" research group at the Max Planck Institute of Psychiatry in Munich from 1993-2000, before moving to the Department of Clinical Neurophysiology at the University of Göttingen. Her main research interests are non-motor symptoms and therapy in Parkinson disease, and movement disorders in sleep (REM sleep behavior disorder and restless legs syndrome). She is involved in designing and performing clinical trials in Parkinson disease and other movement disorders since 1990, many of them as PI. She has published more than 330 peer reviewed papers on Parkinson disease, movement disorders and sleep. She is currently Secretary of the International Parkinson and Movement Disorder Society, was President of the World Association of Sleep Medicine and is member of many national and international medical societies and boards.

Organizers

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COURSE VENUE

HOTEL ALPIN

500001 Poiana Brasov, Brasov, Romania
http://www.hotelalpin.ro

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Course Registration Desk

All course materials and documentation will be available at the SSNN booth. The course staff will be pleased to help you with all enquiries regarding registration, course 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.

Registration Fees

Specialists: 200 EUR
Residents (SNR Members): 100 EUR
the proof of membership on 2016 will be requested at registration

The fee includes

- Course booklet
- Coffee breaks
- 2 lunches
- 3 dinners

Participants Registration Fee Includes:

Admission to all scientific sessions during the course.
Course materials (delegate bag, final program and abstract book etc.)
Admission to Lunches and Coffee Breaks

On-Site Registration

On-site registration will be processed on a first-come, first-served basis.
Priority will be given to pre-registered delegates.
Depending on the number of on-site registered delegates, availability of course bags may be limited.

Name Badges

Participants are kindly requested to wear their name badge at all times during the course.
The badge allows admission to the scientific sessions, coffee breaks and lunches.

Course Language

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

Changes In Program

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

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