



THE SOCIETY FOR THE STUDY OF
NEUROPROTECTION AND
NEUROPLASTICITY



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International Parkinson and
Movement Disorder Society

3rd NATIONAL MOVEMENT DISORDERS TEACHING COURSE

2-4 April 2014 | Hotel Alpin
Poiana Brașov | Romania

FINAL PROGRAM



3rd NATIONAL MOVEMENT DISORDERS TEACHING COURSE

„Non-motor symptoms in Parkinson’s disease”

Course Director:



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Neuroplasticity (SSNN)



Speakers



Faculty

(in alphabetical order)

Angelo Antonini /Italy
Ovidiu Băjenaru /Romania
Sevasti Bostantjopoulou /Greece
Ray Chaudhuri /UK
Cristian Falup-Pecurariu /Romania
Joaquim Ferreira /Portugal
Luigi Ferrini-Strambi /Italy
Spyridon Konitsiotis /Greece
Dafin Mureșanu /Romania
Per Odin /Germany/Sweden
Lăcrămioara Perju-Dumbrava /Romania
Bogdan O. Popescu /Romania
Olivier Rascol /France
Peter Riederer /Germany
Pille Taba /Estonia
Claudia Trenkwalder /Germany
Francesc Valldeoriola /Spain



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CONGRESS VENUE

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Participants Registration Fee Includes:

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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 congress bags may be limited.

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

Congress Language

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

Changes In Program

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



Scientific Program

Scientific Program

APRIL 3rd, 2014

08:00 – 09:00

Registration and general information

09:00 – 09:20

Course and speakers presentation

Session 1

Chairman:

Ovidiu Băjenaru (Romania), Olivier Rascol (France),
DaŃin Mureşanu (Romania)

09:20 – 09:50

Ovidiu Băjenaru (Romania)
Etiopathogenic hypothesis in Parkinson's disease

09:50 – 10:20

Francesc Valldeoriola (Spain)
Diagnostic and treatment of pain in Parkinson's Disease

10:20 – 11:00

Lăcrămioara Perju-Dumbravă (Romania)
Fatigue assessment and treatment in Parkinson's Disease

11:00 – 11:30

Coffee break

Session 2

Chairman:

Joaquim Ferreira (Portugal), Cristian Falup-Pecurariu (Romania)

11:30 – 12:00

DaŃin Mureşanu (Romania)
Brain plasticity and neurorehabilitation in Parkinson's disease

12:00 – 12:30

Olivier Rascol (France)
Clinical trials in non-motor symptoms in Parkinson's disease

12:30 – 13:00

Luigi Ferrini-Strambi (Italy)
Sleep disturbances in Parkinson's disease

13:00 – 14:00

Lunch

Session 3
Chairman:

Oliver Rascol (France), Joaquim Ferreira (Portugal)

14:00 – 14:30

Pille Taba (Estonia)
Impulse control disorders in Parkinson's disease

14:30 – 15:00

Joaquim Ferreira (Portugal)
When to start treatment in Parkinson's disease

15:00 – 15:30

Claudia Trenkwalder (Germany)
Cognitive impairment in Parkinson's disease

15:30 – 16:00

Coffee break

Session 4
Chairman:

Lăcrămioara Perju-Dumbravă (Romania), Francesc Valldeoriola (Spain),
Per Odin (Germany/Sweden)

16:00 – 16:30

Sevasti Bostantjopoulou (Greece)
Anxiety and depression in Parkinson's disease

16:30 – 17:30

Video session

APRIL 4th, 2014

Session 1 Chairman:	Angelo Antonini (Italy), Ray Chaudhuri (UK), Luigi Ferrini-Strambi (Italy)
08:30 – 09:00	Cristian Falup-Pecurariu (Romania) Spectrum of non-motor symptoms in Parkinson's disease
09:00 – 09:30	Ray Chaudhuri (UK) Dopaminergic Pathophysiology and Treatment of Non-motor Symptoms in Parkinson's Disease
09:30 – 10:00	Angelo Antonini (Italy) Parkinson's Disease Management - a Long-term Strategy
10:00 – 10:30	Interactive case presentations
10:30 – 11:00	Coffee Break
Session 2 Chairman:	Pille Taba (Estonia), Claudia Trenkwalder (Germany), Bogdan O. Popescu (Romania)
11:00 – 11:30	Ovidiu Băjenaru (Romania) New standards of care in advanced Parkinson's Disease
11:30 – 12:00	Ray Chaudhuri Challenges and Outcomes in the Management of Non-motor Symptoms in Advanced Parkinson's Disease
12:00 – 12:30	Per Odin (Germany/Sweden) Continuous dopaminergic stimulation therapy: effect on motor symptoms, non-motor symptoms, and quality of life
12:30 – 13:00	Bogdan O. Popescu (Romania) Dopamine agonist treatment in advanced Parkinson's disease
13:00 – 13:30	Angelo Antonini (Italy) Successful Management of CDS Advanced Parkinson's Disease
13:30 – 14:00	Case presentations
14:00 – 15:00	Lunch
Session 3	

Chairman:	Peter Riederer (Germany), Sevasti Bostantjopoulou (Greece), Spyridon Konitsiotis (Greece)
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15:00 – 15:30	Peter Riederer (Germany) Biomarkers in Parkinson's Disease
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15:30 – 16:00	Spyridon Konitsiotis (Greece) Gastrointestinal dysfunction in Parkinson's Disease
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16:00 – 16:30	Video session
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16:30 – 17:00	General discussion. Course evaluation
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Abstracts



SUCCESSFUL MANAGEMENT OF CDS ADVANCED PARKINSON'S DISEASE



**ANGELO
ANTONINI**

Director Parkinson Unit
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Neurology Clinic,
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Long-term L-dopa therapy is associated with the development of motor fluctuations and dyskinesias in the majority of patients with Parkinson's disease (PD). Dyskinesias are likely to result from the interaction between the primary degenerative process and the chronic exposure to pulsatile oral L-dopa therapy.

All studies comparing early L-dopa vs. dopamine agonist early therapy indicate that initiation with agonists is associated with a reduced risk for motor complications – in particular dyskinesias – possibly because agonists longer half-lives provide continuous dopaminergic delivery.

In advanced patients switching from pulsatile to continuous dopaminergic delivery avoids peaks and troughs in L-dopa plasma and may also widen the therapeutic window. Currently this can be accomplished only with subcutaneous apomorphine or duodenal levodopa infusions. Particularly duodenal L-dopa infusion is promising because continuous delivery with an optimized dose can be ensured and peripheral L-dopa can be kept stable within the patient's individual therapeutic window allowing replacement all oral medications.

The levodopa is administered via a permanent catheter implanted into the duodenum by percutaneous endoscopic gastrostomy (PEG) under local anaesthetic. Administration of the drug is controlled by a pump with an adjustable infusion rate allowing fine-tuned titration, individual adaptation of dose and also allows administration of extra doses (if needed).

Current experience indicates that a satisfactory therapeutic window can be achieved and maintained for several months in advanced PD patients. This is associated with improved motor fluctuations and reduced disabling dyskinesia, resulting in significant benefit in quality of life and several non-motor domains.



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PARKINSON'S DISEASE MANAGEMENT - A LONG-TERM STRATEGY

Parkinson's disease is a progressive neurodegenerative disorder triggered by genetic and/or environmental factors. It is now clear that the degenerative process begins many years before motor symptoms become clinically manifest. Current treatment standards include attempts to modify disease progression, prevention and/or delay of levodopa-induced motor complications and management of non-motor symptoms.

Motor complications are likely to result from the interaction between the primary degenerative process and the chronic exposure to pulsatile oral L-dopa therapy.

All studies comparing early L-dopa vs. dopamine agonist early therapy indicate that initiation with agonists is associated with a reduced risk to develop motor complications – in particular dyskinesias – possibly because agonists longer half-lives provide continuous dopaminergic delivery. Rotigotine is a non-ergolinic, lipid-soluble dopamine agonist which is distributed using a silicone-based transdermal patch allowing continuous drug delivery with a linear absorption profile. The advantage is to maintaining a consistent dopaminergic stimulation with steady plasma levels, increased tolerability, greater compliance from a simpler dosing regimen and ease in dose titration. Studies have shown that patients already taking oral agonists may be switched to rotigotine patch overnight, at the same daily dose.

Additional advantages regard the management of non-motor symptoms. Non-motor symptoms occur across all stages of Parkinson's disease, can precede the onset of motor symptoms, and have been identified as having a more significant impact on health-related quality of life than motor symptoms. However, they are often under-recognized, and their treatment remains an unmet need of PD. Dysfunction of dopaminergic and non-dopaminergic systems contribute to the development of non-motor symptoms in PD; symptoms such as depression, anxiety, fatigue, sleep disorders, and pain may have a dopaminergic basis. Indeed application of rotigotine patch showed benefits not only on motor disability but also on sleep disturbances and fatigue which are common problems in PD. In particular the Recover study has shown that rotigotine improves early-morning motor features and reduces the incidence of cramps, with benefit on all sleep quality measures.

The levodopa is administered via a permanent catheter implanted into the duodenum by percutaneous endoscopic gastrostomy (PEG) under local anaesthetic. Administration of the drug is controlled by a pump with an adjustable infusion rate allowing fine-tuned titration, individual adaptation of dose and also allows administration of extra doses (if needed).

Current experience indicates that a satisfactory therapeutic window can be achieved and maintained for several months in advanced PD patients. This is associated with improved motor fluctuations and reduced disabling dyskinesia, resulting in significant benefit in quality of life and several non-motor domains.

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ANXIETY AND DEPRESSION IN PARKINSON'S DISEASE



**SEVASTI
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Depression or anxiety alone or co-existence of depression with anxiety are common psychiatric manifestations in Parkinson's disease (PD) occurring at higher prevalence than in age-matched controls. The estimated prevalence of depression is about 40%-50%, with 17% prevalence of major depression, 22% of minor depression and 13% of dysthymia. Depression in Parkinson's disease is characterized by dysphoria, pessimism, somatic symptoms, loss of initiative and self-esteem. Feelings of guilt or worthlessness and suicidal thoughts are not common. The diagnosis of depression is challenging due to the overlap between physical symptoms of PD and psychological symptoms of depression. Depressive symptoms are related to greater functional impairment, faster disease progression, reduced cognitive performance and poor quality of life. The pathophysiology of depression in PD is complex. Endogenous (neurochemical changes in specific brain areas), genetic, environmental and psychological (reaction to the presence of a debilitating disease) factors are implicated in the development of depression. Treatment options for depression in Parkinson's disease include psychosocial counseling, antidepressants, dopamine agonists, rTMS and electroconvulsive therapy. The prevalence of anxiety in PD ranges between 5% and 60% with generalized anxiety disorder, panic disorder, agoraphobia and social phobia being most common. Anxiety disorders are associated with severe motor symptoms, gait problems, motor complications and poor quality of life. The co-existence of anxiety with depression ranges from 14% to 26%. Anxiety in PD is underdiagnosed since current anxiety scales are non-specific for use in PD. There is no specific pharmacological treatment for anxiety in PD. Anxiolytic drugs and SSRIs have been used but without systemic assessment or large placebo controlled studies.



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CHALLENGES AND OUTCOMES IN THE MANAGEMENT OF NON-MOTOR SYMPTOMS IN ADVANCED PARKINSON'S DISEASE

PD is complicated by the well recognised motor complications but also non motor problems. Non-motor symptoms (NMS) in Parkinson's disease (PD) are common across all stages of PD but often receive little attention as NMS related issues are often overshadowed by the motor symptoms. In spite of their importance there have been only two "holistic" prevalence studies of NMS in PD, the NMSQuest study in 2007 and the PRIAMO study in 2009, both indicating the importance of NMS and its presence across all stages of common PD (Martinez-Martin et al., 2007; Barone et al., 2009). These NMS significantly impair quality of life and may precipitate hospitalization and strongly contribute to the cost of care for PD (Martinez-Martin et al. 2010). NMS create a significant burden for people with PD and caregivers and studies suggest that NMS is a greater determinant of quality of life than motor features. (Martinez-Martin P Et al., 2009). Some NMS such as depression, dementia, dysautonomia and sleep problems, are well established but others such as dysphagia, dribbling of saliva, weight changes, sexual problems and diplopia are poorly recognized and constitute a key unmet need and a clinical challenge. In recent years, a self completed NMS questionnaire and NMS scale has been validated specifically for use in PD and a recent international survey showed that up to 62% of NMS in PD might remain undeclared to health care professionals because patients may be unaware that the symptoms are linked to PD. (Chaudhuri KR, 2010) Imaging with PET have also suggested a dopaminergic basis to some NMS which are thought to be non-dopaminergic in its origin. These include depression and anhedonia, hypothalamic sleep related problems and more recently fatigue (Pavese et al, 2010). As such, targeted dopaminergic therapy may benefit a range of NMS.

Table as follows: A new classification of Non Motor Symptoms in PD.

Todoa A, et al. Pract Neurol 2014;0:1-13. doi:10. 1136/practneurol-2013-000741

- Related to the disease process or pathophysiology
 - Dopaminergic origin
 - Non-dopaminergic origin
- Related to a partial non-motor origin (usually brainstem autonomic impairment with motor end result, such as constipation or diplopia)
- Related to non-motor fluctuations (cognitive, autonomic and sensory subtypes)
 - Fluctuating
 - Constant
- Related to PD drug therapy
 - Specific symptoms (eg, hallucinations, delirium)
 - Syndromes—impulse control disorders, dopamine agonist withdrawal syndrome, Parkinson's hyperpyrexia syndrome (thermoregulatory failure, delirium)
- Possibly genetically determined
 - Dementia in cases with glucocerebrosidase mutation
 - Depression and sleep disorders in cases with leucine-rich repeat kinase-2 mutation



**RAY
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DOPAMINERGIC PATHOPHYSIOLOGY AND TREATMENT OF NON-MOTOR SYMPTOMS IN PARKINSON'S DISEASE

Non-motor symptoms (NMS) of Parkinson's disease (PD) are common across all stages of PD but are often under-recognised, under-declared and therefore, under-treated as management of PD is dominated by motor symptoms.¹ There have been only two "holistic" prevalence studies of NMS in PD, the NMSQuest study in 2007 and the PRIAMO study in 2009, both indicating the importance of NMS and its presence across all stages of common PD.^{2,3} NMS create a significant burden for people with PD and caregivers and studies suggest that NMS is a greater determinant of quality of life than motor features.⁴

A dopaminergic basis to many NMS of PD such as depression, apathy, fatigue, sleep disorders, gastrointestinal and visual problems have been described while non motor off periods remain a major therapeutic challenge. Transdermal rotigotine, Subcutaneous Apomorphine infusion and intrajejunal levodopa infusion are the most potent dopamine agonist used in fluctuating levodopa treated Parkinson's disease (PD) patients usually for management of refractory motor fluctuations. A considerable amount of clinical data suggest that these therapies are also helpful in cognitive and other non motor problems of PD such as sleep disorders, mood problems and non motor fluctuations. Additionally these therapies , particularly use of 24 hr transdermal therapy with rotigotine provide a good basis for continuous drug delivery.

Current and investigational dopaminergic therapies

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Route	Agents	Clinical positioning in PD
Transdermal	Rotigotine	In routine clinical use as monotherapy as well as adjunctive therapy
	Apo-MTD (apomorphine included in a microemulsion and administered by transdermal route) what is MTD ??	One study describes clinical motor or non motor ?efficacy and long action
	Lisuride	Preliminary clinical data on what ? available
	Piribedil	1 clinical study with no significant beneficial effect on what reported
Intranasal	Apomorphine	Not in use due to side effects such as ??
Subcutaneous	Apomorphine	In clinical use usually as injections for predictable motor off and infusion as advanced therapy
	Lisuride	Not in widespread clinical use. Discontinued in UK and USA
	Rotigotine Polyoxazoline Conjugate SER-214	In development and clinical studies in rat models reported



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Clinical grading based on NMSQuest or NMSS now is possible as below:

Box 2 Non-motor Parkinson's disease burden grading using Non-Motor Symptoms Scale (NMSS) and Non-Motor Symptoms Questionnaire (NMSQ) (Kings ISCIII grading)
<p>Kings-ISCIII grading by NMSQuest (can be performed by healthcare professional and based on patient responses using NMSQ)⁴⁶</p> <p>Stage 0 NMSQ—0 no non-motor symptoms</p> <p>Stage 1 NMSQ—1-5 mild</p> <p>Stage 2 NMSQ—6-12 moderate</p> <p>Stage 3 NMSQ—13-20 severe</p> <p>Stage 4 NMSQ—21-30 very severe</p> <p>Grading by NMSS (to be used for clinical and research based studies)⁴⁷</p> <p>Non-motor symptoms burden level 0 NMSS—0 Non-motor symptoms burden level 1 NMSS—1-20 Non-motor symptoms burden level 2 NMSS—21-40 Non-motor symptoms burden level 3 NMSS—41-70 Non-motor symptoms burden level 4 NMSS—>71</p>

We explore possible non motor effects of these therapies in advanced PD patients using the validated non motor symptoms scale for PD (NMSS) which is sensitive to intervention and has robust clinimetrics. The data are based on open label and also multiple cohort comparative studies across all age groups in PD thus avoiding the problem of poor external validity often encountered in randomised controlled studies. In particular, effects on aspects of sleep such as nocturia, nocturnal non motor off periods, pain, dribbling of saliva and mood are reported.

References:

1. Chaudhuri, K.R., Prieto-Juvcynska, C., Naidu, Y., Mitra, T., Frades-payo, B., Tluk, S., et al. Movement Disorders 2010;25:704-709
2. Chaudhuri K.R., Martinez-Martin, P., Schapira, A.H.V., Stocchi, F., Sethi, K., Odin, P. et al. (2006) Movement Disorders 21(7), 916-923.
3. Barone, P., Antonini, A., Colosimo, C., Marconi, R., Morgante, L., Avarello, T.P. et al. (2009) Movement Disorders 24, 1641-1649.
4. Chaudhuri, K.R., Martinez-Martin, P. (2008) European Journal of Neurology 15 (suppl 2), 1-7.



SPECTRUM OF NON-MOTOR SYMPTOMS IN PARKINSON'S DISEASE



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Non-motor symptoms (NMS) in Parkinson's disease (PD) constitute today a challenging topic. It became a rapid growing field of research, due to the fact that are more and more recognized and affects quality of life of these patients.

The spectrum of NMS in PD include: anxiety, depression, cognitive impairment, sleep disorders, sensory symptoms, autonomic dysfunction, sexual dysfunction, gastrointestinal symptoms.

Some of this NMS could precipitate hospitalization. Recent studies suggest that NMS are present even in the moment of PD diagnoses. Moreover, PD patients have some of these symptoms with many years before the PD diagnoses. There are some NMS strong predictors for later development of PD, like olfactory dysfunction, REM sleep behavior disorder, depression, constipation. Longitudinal studies suggest that over time some of these NMS symptoms are worsening. Some NMS could be induced by dopaminergic treatment, like hallucinations, impulse control disorders. NMS are not well recognized in clinical practice.

One important issue is the nondeclaration of NMS to the doctors. Holistic evaluation of NMS shows that there is an agreement between self-reported NMS by the patients and evaluation made by the doctors. Knowing the whole spectrum of NMS could improve the treatment of these patients and ameliorate the quality of life.



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WHEN TO START TREATMENT IN PARKINSON'S DISEASE

The management of Parkinson's disease (PD) implies being able to respond to very pragmatic therapeutic questions like when and how to start treatment. Just by listing the different options (e.g. delay medication treatment, start on a specific medication, start rehabilitation) we realize the need to be able to support with the best experimental data each of the possible therapeutic strategies. For this decision many factors should be considered like the capability to delay disability, the target objectives of the therapeutic intervention (prevent clinical progression, improve parkinsonism, prevent motor complications, maintain compliance) and the best evidence based recommendations for the treatment of early PD.

Although there are no therapeutic interventions, which have demonstrated conclusively to be able to prevent clinical progression, the early symptomatic control of Parkinsonism improves quality of life. However, it is still unknown the long term benefits of such a strategy. Other factors like patients' age, work performance, medication safety profile, and the relevancy of clinical outcomes (delay first occurrence of dyskinesia vs. delay of troublesome motor complications) should also be considered when deciding for individual patients.



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FERREIRA**

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SLEEP DISTURBANCES IN PARKINSON'S DISEASE



**LUIGI
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Parkinson's disease (PD) is classically considered to be a motor system affliction; however, also non-motor alterations, including sleep disorders, are important features of the disease. Sleep disturbances occur in more than 90% of PD patients and include difficulty initiating sleep, frequent night-time awakening and sleep fragmentation, nocturia, restless legs syndrome/periodic limb movements, sleep breathing disorders, rapid eye movements (REM) sleep behavior disorder (RBD), sleep attacks and excessive daytime sleepiness (EDS).

Sleep disturbances in PD can result secondarily to natural disease progression, also as a side effect of the medications used in PD. A recent study showed that newly diagnosed PD patients had minimal differences in subjective or objective sleep disturbance compared to controls apart from increased daytime naps and symptoms such as dream-enacting behaviors of punching or grabbing, suggesting a RBD.

Dopamine agonist use, higher levodopa dose, higher Parkinson's Disease Questionnaire score, non-tremor dominant motor phenotype and COMT met/met genotype have been reported associated with higher levels of EDS. Circadian dysfunction may also underlie EDS in PD. Some authors recently found in PD patients with EDS compared to PD patients without EDS a significantly lower amplitude of the melatonin rhythm and 24-hour melatonin area under the curve.

RBD has been described as a premotor symptom of PD in several prospective, retrospective, and cross-sectional studies. A recent study that evaluated by functional magnetic resonance imaging patients with idiopathic RBD who were at risk for developing PD, PD patients and age-matched controls found an altered nigrostriatal and nigrocortical connectivity in RBD (before onset of obvious motor impairment). Moreover, a significantly faster motor progression in PD patients with RBD and REM sleep without atonia (RWA) than in those with preserved REM sleep atonia has been very recently reported.

While the causal complexity of sleep problems in PD certainly hampers the design of therapeutic studies, multiple general treatment strategies against sleep disorders can however be applied efficiently in PD patients as well. Treatment of sleep disturbances in PD patients is crucial, as what is termed as, "sleep benefit effect" has been shown to improve the symptoms of PD.



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GASTROINTESTINAL DYSFUNCTION IN PARKINSON'S DISEASE



**SPYRIDON
KONITSIOTIS**

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Symptoms of gastrointestinal dysfunction are perhaps the most frequent non motor symptoms in PD and may occur early in the disease process. They include drooling, dysphagia, impaired gastric motility, nausea, and constipation. Drooling or sialorrhea is observed in the majority of patients and is a result of dysphagia, rather than an overproduction of saliva. Dysphagia is usually observed in more advanced disease and may induce aspiration, malnutrition, weight loss, and dehydration, and thus may increase the risk of mortality.

Nausea and vomiting may be related to dopaminergic therapy, but can also result from impaired gastric motility. Since levodopa is absorbed in the duodenum and proximal jejunum, impaired gastric motility is one of the most important reasons for unpredicted and delayed levodopa absorption, and therefore for motor response fluctuations. Constipation, which is experienced early by the majority of patients, may also result from impaired gastrointestinal motility, and can be present many years before the appearance of motor symptoms.

Speech and swallowing therapy may help to teach effective swallowing techniques. Chewing gum can increase swallowing and consequently reduce drooling. Anticholinergics have been used with questionable effectiveness and a study of botulinum toxin demonstrated effectiveness, without worsening dysphagia.

For impaired gastric motility the use of domperidone will improve nausea and levodopa absorption. Dopamine antagonists such as metoclopramide should not be used, as they can worsen PD symptoms.

Prompt application of common therapies such as increased dietary fiber and fluid intake, increased exercise, and stool softeners, are usually enough for managing constipation.



BRAIN PLASTICITY AND NEUROREHABILITATION IN PARKINSON'S DISEASE



**DAFIN F.
MUREȘANU**

Neurological disorders, especially degenerative diseases, represent a leading cause of long term disability all over the world. Many advances have been done in the treatment of these pathologies. The need to identify therapeutic methods, able to limit brain damage or enhance recovery of motor and cognitive functions through neuroprotective and neurorestorative mechanisms, is desirable. There are many animal and human studies trying to elucidate the cellular and molecular mechanisms of plasticity of the nervous system. Neurorecovery is the positive outcome that produces clinically relevant results with immediate functional and late structural effects.

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

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

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CONTINUOUS DOPAMINERGIC STIMULATION THERAPY: EFFECT ON MOTOR SYMPTOMS, NON-MOTOR SYMPTOMS, AND QUALITY OF LIFE



PER ODIN

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The major advantage of continuous dopaminergic stimulation (CDS) therapies is the reduction in 'off' time and dyskinesias, but there is also evidence for improvements in non-motor symptoms (NMS) and quality of life (QoL). Each of the three main CDS therapies (subcutaneous apomorphine [APO] infusion, levodopa/carbidopa intestinal gel [LCIG] infusion and deep brain stimulation [DBS]) has a slightly different profile of effects. The most recent empirical data on CDS therapies will be presented, and their effects on motor symptoms, non-motor symptoms and QoL will be compared. Data that are available on the long-term efficacy of the different advanced therapies will also be compared.

When deciding which CDS therapy to give an individual, many factors come into play. The method of administration, effects on motor symptoms and NMS, side-effects, complications, and patient preference are key considerations. Other factors to be considered include age, comorbidities, contraindications, presence of carer, psychological state and cognitive impairment/dementia. The indications for all CDS therapies are similar, including severe disease, pronounced motor fluctuations and dyskinesias. Contraindications differ slightly between the advanced therapies, such as lack of support/compliance (for APO infusion and LCIG infusion), a tendency for hallucinations (for APO infusion), and contraindications for abdominal surgery (for LCIG infusion). In addition, DBS is less suitable for older patients (aged >70–75 years), when dementia or more pronounced depression/anxiety is present, or if the patient is unsuitable for brain surgery.

Simple algorithms have been developed to guide the selection of appropriate patients and appropriate CDS therapies. New clinical evidence on the relative effects of the CDS therapies may influence treatment selection algorithms to help guide decisions on the most appropriate CDS therapy for the individual patient and this new evidence will be discussed.



FATIGUE ASSESSMENT AND TREATMENT IN PARKINSON'S DISEASE



**LĂCRĂMIOARA
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Fatigue is reported frequently by patients with neurologic disorders. In Parkinson's disease (PD) one third of patients report fatigue as their most disabling symptom. The first step in assessing fatigue is a clear definition and a differentiation between fatigue and fatigability. Fatigue can be considered a subjective sensation, whereas fatigability is rather an objective change in performance.

It is obvious that PD patients can suffer concomitantly from other diseases that can secondarily cause fatigue, therefore one has to determine, when possible if fatigue is primarily due to PD or not.

In order to evaluate the severity of symptoms and to assess the efficiency of interventional methods, objective fatigue rating scales are needed. The scales recommended to be used in clinical studies are: the Functional Assessment of Chronic Illness Therapy-Fatigue (FACIT-F) scale, Parkinson Fatigue Scale (PFS-16), the Multidimensional Fatigue Inventory, the Fatigue Severity Scale (FSS). Each of them has strong points, but also disadvantages when applied.

To date there are treatment options for many of the sleep disturbances in PD patients but fatigue seems to be only scarcely improved by available medication.

Modafinil, Methylphenidate, caffeine, memantine and sodium oxybate were tested as possible treatment options for fatigue in PD. Some studies have shown a reduction of the fatigue measured by the clinical global impression of fatigue in patients treated with modafinil. Just one study succeeded to demonstrate an objective improvement of motor fatigability. Methylphenidate administered three times a day improved fatigue, as shown in one study. Its risk for abuse in patients with dopamine dysregulation syndrome or impulse control disorders has limited its use. Memantine failed in influencing fatigue, as demonstrated in a pilot study. Sodium oxybate could be efficient against fatigue but it has two main disadvantages: it can suppress respiration and has an abuse potential. In conclusion fatigue, as a frequently encountered symptom in PD patients, has to be carefully assessed by means of objective scales and confounding factors should be, if possible eliminated. The medical treatment of fatigue is promising but not yet satisfactory. Further research in the field is needed and possible nonmedical therapies may be added.

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DOPAMINE AGONIST TREATMENT IN ADVANCED PARKINSON'S DISEASE



**BOGDAN O.
POPESCU**

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

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CLINICAL TRIALS FOR NON MOTOR SYMPTOMS IN PARKINSON DISEASE



OLIVIER RASCOL

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Non motor symptoms are common in Parkinson disease (PD). They are numerous, including autonomic dysfunction, pain, cognitive impairment, mood, psychiatric and sleep problems and others, and contributing significantly to patient's impaired quality of life. The treatment of such symptom is therefore essential, but this part of the management of PD is still largely limited to empirical experience supported by poor objective evidence.

There are many reasons for such a situation. Pathophysiological mechanisms are poorly understood and convincing therapeutic targets are lacking. Animal models have been rarely used and preclinical data are scarce. Many non motor symptoms are complex and heterogeneous, like for example sleep problems and pain symptoms. Operational classifications remain controversial, leading to inadequate inclusion criteria and inappropriate population definition. Some non motor symptoms are worsened while others are improved by dopaminergic medications. Many non motor symptoms fluctuate like motor symptoms, but the clinical importance of such fluctuations is still unclear. In many instances, there are no sensitive and well validated clinical scales to measure treatments effects in a reliable manner. The clinical importance of the effects captured by such outcomes is often unknown. Randomized placebo-controlled trials have been rarely the rule in the past.

The rise in interest for non motor symptoms in PD increased the number and the quality of trials for non motor symptoms in the last few years. This is expected to continue in the future, for a better pharmacological and non pharmacological management of PD patients in clinical practice.



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BIOMARKERS IN PARKINSON'S DISEASE

In this educational lecture it is aimed at elucidating the current status of biomarker research in Parkinson's disease (PD). Therefore it is necessary to define the criteria for any kind of biomarker and to discuss critically pitfalls as well as problems in identifying biomarkers. This report concludes that it will be very unlikely to identify one biomarker even for a homogenous subgroup of PD. In contrast a combination of biomarkers is envisaged to identify PD already in a presymptomatic phase.



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IMPULSE CONTROL DISORDERS IN PARKINSON'S DISEASE



PILLE TABA

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Non-motor symptoms, including psychiatric and behavioural disorders, are significant contributors to long-term disability and impairment of quality of life of patients with Parkinson's disease and their carers. Impulse control disorders are characterised by excessive and repetitive activities, with a failure to resist urges or drive to do or think something, that is harmful to the patient or others. Impulse control disorders constitute a group of behavioural disorders including pathological gambling, hypersexuality, compulsive shopping, binge eating, dopamine dysregulation syndrome and punting, that occur in 6-16% of Parkinson's disease patients.

Impulse control disorders are associated with dopamine replacement therapies - dopamine agonists and levodopa at higher doses -, and the risk factors for impulsive control disorders include younger age, male sex, and additionally impulsiveness, substance use or bipolar disorder in history. Impulse control disorders in Parkinson's disease are associated with multiple functional impairments, depressive, obsessive-compulsive and anxiety symptoms, higher novelty seeking and impulsivity. Recognizing of impulse control disorders may be challenging. The self-administered screening instrument the Questionnaire for Impulsive-Compulsive Disorders in Parkinson's Disease (QUIP) has been designed for use in clinical settings for the impulsive control disorders and other compulsive behaviours.

The management of clinically significant impulse control disorders includes pharmacological, surgical and psychological interventions: modification of dopamine replacement therapy, behavioural treatments and caregiver education but the evidence for using of neuroleptics is controversial. There is a lack of specific treatments for the impulse control disorders but early recognition and prevention are of importance.



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COGNITIVE IMPAIRMENT IN PARKINSON'S DISEASE



**CLAUDIA
TRENKWALTER**

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Mild cognitive impairment (MCI) and dementia (Parkinson's Disease dementia, PDD) are particularly common non-motor complications in Parkinson disease (PD). There is considerable variation in the reported prevalences of PDD, with up to 2/3 of patients fulfilling the criteria for dementia in late stage PD, but closely related to increasing age. In cross-sectional investigations of all stages of PD, dementia ranges from 20–44 %, constituting an approximate 3- to 6-fold increased risk for PD patients as compared to non-PD patients. The cumulative incidence of dementia reported in a longitudinal study by Aarsland et al. was 52% after 4 years and nearly 80 % after 8 years. Variations are due to different populations and tools used, including the application of different definition criteria. PDD is preceded or accompanied by other neuropsychiatric symptoms of PD such as hallucinations, psychosis, nocturnal confusion or REM sleep behavior disorder (RBD). Major symptoms of PDD are disturbance of the executive functions, visuospatial orientation, behavioral changes and less prominent memory deficits. When diagnosing PDD in early stages of PD, the differential diagnosis of Lewy Body dementia (LBD) has to be considered. Patients with LBD are characterized by any cognitive decline and hallucinations in the first year of Parkinson motor symptoms. Difficult to decide on a retrospective base, LBD may be misdiagnosed as PDD in later years, but progression of LBD is faster as PDD and prognosis is even worse.

Using established clinical diagnostic standards for dementia the overall rate on routine outpatient neurological care is 28.6%, but using more sensitive neuropsychological measures, rates for cognitive impairment might be up to 2-fold higher. The MMSE revealed strikingly low sensitivity. (Riedel et al 2006)

Recently, diagnosing MCI as the precedent symptomatology for future dementia in PD plays a major role. Therefore a Task Force to define criteria for MCI in PD has been established by the MDS to work out criteria and cut.off values. A uniform definition of PD-MCI is important, because it will help identify (1) the clinical characteristics of the earliest stage of PD cognitive impairment, (2), the best predictors of conversion from PD-MCI to PDD, (3) the effects of PD-MCI on quality of life and day-to-day functioning, the MDS Task Force has delineated diagnostic criteria that include (1) characterization of the clinical syndrome and (2) methods for its diagnosis. 2-level specific criteria are as follows:



Specific guidelines for PD-MCI level I and level II categories
(Litvan et al Mov Disord 2012)

- A. Level I (abbreviated assessment)
- Impairment on a scale of global cognitive abilities validated for use in PDa or
 - Impairment on at least two tests, when a limited battery of neuropsychological tests is performed (i.e., the battery includes less than two tests within each of the five cognitive domains, or less than five cognitive domains are assessed)
- B. Level II (comprehensive assessment)
- Neuropsychological testing that includes two tests within each of the five cognitive domains (i.e., attention and working memory, executive, language, memory, and visuospatial)b
 - Impairment on at least two neuropsychological tests, represented by either two impaired tests in one cognitive domain or one impaired test in two different cognitive domains
 - Impairment on neuropsychological tests may be demonstrated by:
 - Performance approximately 1 to 2 SDs below appropriate norms or
 - Significant decline demonstrated on serial cognitive testing or Significant decline from estimated premorbid levels

Future studies have to identify those PD patients with MCI and their possible progression.

Tools for identifying Dementia in PD have been defined by the MDS and include the following tests: Of the four scales specifically designed for PD, the SCOPA-COG and the PD-CRS have undergone extensive and rigorous validation processes. While the SCOPA-COG mainly assesses “frontal-subcortical” cognitive defects, the PD-CRS also assesses “instrumental-cortical” functions, allowing better characterization of the different patterns of CI that may be present in PD from the earliest stages. The MMP and PANDA scales were designed as brief screening tests for CI and have not yet been subjected to extensive clinimetric evaluations. (Kulisevsky et al 2009).

For the treatment of PDD a recent cochrane Database review concluded the following (Rolinsky et al 2012): „The currently available evidence supports the use of cholinesterase inhibitors in patients with PDD, with a positive impact on global assessment, cognitive function, behavioural disturbance and activities of daily living rating scales. The effect in DLB remains unclear.“

Therefore treatment with either rivastigmine or donepezil should be initiated when PDD is diagnosed. If MCI should be treated with the same substances has to remain open at this stage.



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DIAGNOSTIC AND TREATMENT OF PAIN IN PARKINSON'S DISEASE



**FRANCESC
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Pain is a major complaint in patients with Parkinson's disease (PD). Some people experience pain as an early symptom of Parkinson's, before their disease has even been diagnosed. However, pain in PD often remains undiagnosed and untreated. Therefore, it is crucial to understand that pain can be part of the PD and to learn ways to manage it. Causes of pain include persistent tremor, muscle rigidity, dystonia, musculoskeletal injury, burns and inflammation. Pain caused by dystonia can be diagnosed when there is visible twisting, cramping or posturing of the painful body part. Pain may fluctuate with the medication dosing. Neuropathic pain is less common than nociceptive pain and may present as burning, numbness and tingling, sharp sensations, or electric shock qualities. Fortunately, many options exist for treating pain. Management options for pain in Parkinson's include both the pharmacological and the non-pharmacological. A combination of both may offer the best pain control, and an interdisciplinary model of care can lead to optimal results for pain management. Some treatment options include medications, physical therapy, botulin toxin injections, exercise and stretching. New pharmacological options have been tested recently and may offer some new perspectives to PD patients.



Biographies



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ANGELO ANTONINI

/Italy

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

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. In addition he is actively involved in the use of continuous infusion of levodopa and apomorphine as well as subthalamic nucleus deep brain stimulus (STN-DBS) for the treatment of motor complications in advanced patients.

During his academic career he has received several awards, published more than 250 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 and also treasurer of the Association of Parkinsonism and Related Disorders.



OVIDIU BĂJENARU

/Romania

1983 : M.D. at the Faculty of Medicine of University of Medicine and Pharmacy "Carol Davila" Bucharest
1989 : specialist in neurology, confirmed by the Ministry of Health of Romania
1993 : Ph.D. at the University of Medicine and Pharmacy "Carol Davila" Bucharest
1999 (since) : Professor of Neurology at the University of Medicine and Pharmacy "Carol Davila" Bucharest, Chairman and Head of the Neurology Department of the University Hospital of Emergency Bucharest
2000-2004 : Vice-Dean of the Faculty of Medicine - University of Medicine and Pharmacy "Carol Davila" Bucharest
2001 - 2013 : President of the Romanian Society of Neurology
since 2013 - : Honorary President of the Romanian Society of Neurology
2003 – 2009 : member of the Scientific Committee of ECTRIMS
2004 - 2009 : Member of the Executive Committee of the European Society of Neurology
2008 (since) : Romania official delegate in UEMS – EBN (Board of Neurology)

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

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

Post graduate training :

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

Fields of interest for the scientific research

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



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SEVASTI BOSTANTJOPOULOU

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Sevasti Bostantjopoulou – Kambouroglou is Professor of Neurology and Director of the 3rd University Department of Neurology of the Aristotle University of Thessaloniki.

She received her medical degree from the Aristotle University of Thessaloniki, Greece. After completing her training in neurology she undertook a fellowship for movement disorders at the Center for Parkinson's Disease and other Movement Disorders at Columbia Presbyterian Medical Center, New York, USA.

Her main fields of interest are Parkinson's disease, atypical parkinsonian syndromes and dystonia. She is Chief of the Outpatient Unit for Parkinson's disease and other movement disorders of the 3rd University Department of Neurology, Aristotle University of Thessaloniki. Dr. Bostantjopoulou is the Greek representative in EFNS/MDS-ES Panel on Movement Disorders since 2003. She is the author of 170 papers in peer-reviewed International and Greek Scientific Journals and 130 published abstracts and she is reviewer in several Greek and international medical journals.



RAY CHAUDHURI

/U.K.

Professor K Ray Chaudhuri is Professor in Neurology/Movement Disorders and Consultant Neurologist and at Kings College Hospital and Kings College London, an Academic Health Sciences Centre and also principal investigator at the MRC centre for neurodegeneration research at Kings College, London. He is the medical director of the National Parkinson Foundation International Centre of Excellence at Kings College, London. He sits on the Nervous Systems Committee of UK Department of Health, National Institute of Health Research and served as Chairman of the appointments/liason committee of the Movement Disorders Society (2009-2013) and is currently serving as the member of the scientific programme committee of the MDS (2013-2015).. He is the Chairman of the newly formed MDS non motor study group. He serves on the task force of practice parameter group for PD and RLS and more recently Non Motor Symptoms of Parkinson's, American Academy Neurology. He is the European Editor of Basal Ganglia and is in the editorial board of Parkinsonism and Related Disorders and Journal of Parkinson's Disease..He also represents UK research and development in the National Institute of Health Research (NIHR) as well as at a local level for London South CLRN neurosciences. He serves in the clinical advisory group of Parkinson's UK and is an advisor to the European Parkinson's Disease Association.

Professor K Ray Chaudhuri is the author of 260 papers including reviews, book chapters, co-editor of 4 books on Parkinson's disease and Restless Legs Syndrome and over 250 published peer reviewed abstracts. He is the chief editor of the first comprehensive textbook on non motor aspects of Parkinson's, published by Oxford University Press and recipient of BMA book prize commendation prize. He has contributed extensively to educational radio and television interviews including BBC and CNN, newspaper articles and videos. He has also lectured extensively on PD and restless legs syndrome at international meetings in USA, Japan, continental Europe, South America, South Africa, India and Australia. His major research interests are continuous drug delivery treatment of PD and restless legs syndrome, Parkinsonism in minority ethnic groups and sleep problems in PD. In 2005 he was awarded the DSc degree by the University of London.



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CRISTIAN FALUP-PECURARIU

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

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

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



JOAQUIM FERREIRA

/Portugal

Joaquim Ferreira completed his medical degree and PhD in Neurology at the University of Lisbon. He is Professor of Neurology and Clinical Pharmacology at the Faculty of Medicine, University of Lisbon and Head of the Laboratory of Clinical Pharmacology and Therapeutics.

He became interested in movement disorders during his medical degree and later undertook a Clinical Pharmacology fellowship with Prof. Olivier Rascol in Toulouse, France.

He is currently the Director of the Clinical Research Center of The Lisbon Academic Medical Center. He also serves as chair of the Education Committee of the Movement Disorders Society. His major research interests are neuropharmacology, Parkinson's disease, dystonia and Huntington's disease.



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LUIGI FERRINI-STRAMBI

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Luigi Ferini-Strambi earned his medical and postgraduate degree in Neurology at the State University of Milan, Italy. Since 1990 he is Director of the Sleep Disorders Center, Dept. of Neuroscience, H San Raffaele, Milan, Italy. Professor Ferini-Strambi has published 250 full papers in international Journals, on different topics (sleep medicine, CNS degenerative disorders, neuropharmacology). He is Field Editor of "Sleep Medicine", and frequent reviewer for several journals, including "Neurology", "Sleep", "Journal of Sleep Research", "Journal of Neurology", "Movement Disorders", "JAMA", "Annals of Neurology".

He is member of several international scientific societies. He is the Elect President of the World Association of Sleep Medicine Society.



SPYRIDON KONITSIOTIS

/Greece

Graduated from University of Ioannina Medical School, Greece and then worked in the dept of pharmacology to finish his doctoral thesis on “neuropharmacology and functional anatomy of basal ganglia”. He completed his residency training in neurology at the University Hospital of Ioannina, Greece, and in 1996 started postdoctoral fellowship at the Experimental Therapeutics Branch, National Institute of Neurological Disorders and Stroke, NIH, Maryland, USA under the supervision of Dr. Tom Chase. He participated in clinical research focusing in experimental therapeutics in parkinson’s disease, as well as laboratory research with parkinsonian primates. In 2000 he returned to Greece as lecturer in Neurology, University of Ioannina Medical School where he is currently associate professor of Neurology. He is running the outpatient clinic for movement disorders, is a member of the greek movement disorders group, and has published more than 50 papers both on clinical and laboratory studies. He is lecturing extensively on issues related to treatment of Parkinson’s disease. His main research interests include Parkinson’s disease, restless legs syndrome, imaging in movement disorders and the pathophysiology of levodopa-induced dyskinesias.



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DAFIN F. MUREŞANU
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Muresanu Fior Dăfin, MD, PhD, MBA, FANA, is the President of the Romanian Society of Neurology, Professor of Neurology, Chairman of the Neurosciences Department, University of Medicine and Pharmacy "Iuliu Hatieganu" Cluj-Napoca, member of the Academy of Medical Sciences, Romania. He also acts as the President of the Society for the Study of Neuroprotection and Neuroplasticity. In these roles, he is involved as member of the faculty in international educational programs of European Master (i.e. European Master in Stroke Medicine, University of Krems), organizer and co-organizer of European and international schools and courses (International School of Neurology, European Stroke Organisation Summer School, Danubian Neurological Society Teaching Courses). His activity includes involvement in many clinical studies and research projects, memberships in the executive board of many national and international societies, participations as invited speaker in national and international congresses, a significant portfolio of scientific articles (77 papers indexed on Web of Knowledge- ISI) as well as contributions in monographs and books published by prestigious international publishing houses. In the last 7 years, he was also invited as speaker in over 200 scientific events both national and abroad. Prof. Dr. Muresanu has been honoured with the Faculty of Medicine, University of Medicine and Pharmacy "Iuliu Hatieganu" Cluj-Napoca "Octavian Fodor Award" for the best scientific activity of the year 2010 and the 2009 Romanian Academy of Medical Sciences "Gheorghe Marinescu Award" for advanced contributions in Neuroprotection and Neuroplasticity.



PER ODIN

/Germany/Sweden

Professor Odin finished medical school at the Uppsala University, Sweden 1982, then presented his PhD thesis at the same university in 1987. He became specialist in Neurology 1993 at the Lund University, Sweden and got his first Professorship at the Medical School Hannover, Germany in 1998. Odin is now head of the department of Neurology at the Central Hospital in Bremerhaven, Germany and at the same time Professor of Neurology at the Lund University, Sweden. Odin has focused his interest on Movement Disorders since 1987 and his main research areas concern continuous dopaminergic stimulation, pump therapies for Parkinson's disease (PD), non-motor PD symptoms and cell transplantation in PD. Odin is chair of the Swedish Movement Disorders Society, the Scandinavian Movement Disorder Society and board member of Competence Network Parkinson's disease, Germany.



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LĂCRĂMIOARA PERJU-DUMBRAVA

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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

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Bogdan O. Popescu - born March 8th, 1971 in Bucharest, Romania.

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Academic Education and Appointments

1996	MD, 'Carol Davila' University School of Medicine, Bucharest, Romania
1997 - 2002	Resident in Neurology, University Hospital Bucharest
2000 - 2009	Assistant Professor, 'Carol Davila' University School of Medicine
2001	PhD, 'Carol Davila' University School of Medicine - suma cum laudae
2002 - 2008	Neurologist, University Hospital Bucharest
2004	PhD, Karolinska Institute, Stockholm, Sweden
2005 -	Head of Laboratory of Molecular Medicine, 'Victor Babeş' National Institute of Pathology, Bucharest, Romania
2008-	Senior Neurologist
2009 - 2012	Lecturer, 'Carol Davila' University School of Medicine
2009 -	Senior Researcher, 'Victor Babeş' National Institute of Pathology, Bucharest, Romania
2012 -	Associate Professor, 'Carol Davila' University School of Medicine and Head of Neurology Unit II, Colentina Clinical Hospital

Awards

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

Other current activities

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

Executive editor of Romanian Journal of Neurology

President elect of the Romanian Society of Neurology (2017-2021) and former Secretary General (2001-2013)

Research director of the Society for the Study of Neuroprotection and Neuroplasticity

Director, Victor Babeş' National Institute of Pathology, Bucharest, Romania

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OLIVIER RASCOL

/France

Doctor Olivier Rascol is Professor of Clinical Pharmacology at the Toulouse University Hospital since 1993. He obtained his MD in Neurology (Toulouse, 1985) and his PhD in Neurosciences (Paris, 1992). Dr Rascol is running the Toulouse Clinical Investigation Centre since 1994 and the Toulouse European Space Clinic since 1998. He is also running a Research Group on Motricity in the Research Unit INSERM U825 and is the coordinator of the French Reference Center for Multiple System Atrophy (Atypical Parkinsonism). Dr Rascol is the chair of the national network of the 56 French Clinical Investigation Centers since 2008 and the chair of the NS-Park Neurosciences Network of the French CIC since 2010. From 2011, Dr Rascol is now coordinating the National French Clinical Research Infrastructure Network F-CRIN.

As a neuropharmacologist, Dr Rascol's main fields of interest are Parkinson's disease and movement disorders, drug development for Parkinson's disease and functional neuroimaging. Dr Rascol has been actively involved in the development of several marketed antiparkinsonian medications (ropinirole, rasagiline). He is currently running several research programs for disease progression and symptomatic management of PD (motor signs, dyskinesias and on-off problems, non-motor signs such as pain and sleep problems) with new dopaminergic (dopamine agonists, dopamine reuptake inhibitors, MAO-B inhibitors...) and non-dopaminergic (serotonergic, glutamatergic, adenosine, alpha-adrenergic...) drugs in collaboration with several academic and industry research centres in the US and in Europe. He is acting in this field as an external advisor for French and European scientific organisations, patients' associations, drug agencies and international pharmaceutical companies. He is at the board of the Evidence-Based Medicine MDS Task Force in charge of the continuous assessment of all antiparkinsonian treatments. He is involved in the process of editing French (HAS) and European (EFNS / MDS-ES) guidelines for the treatment of Parkinson's disease. As the chair of the French CIC Network and of the National F-CRIN Clinical Research infrastructure, Dr Rascol has been deeply involved within the last few years in the management and organisation of clinical research in France.

Dr Rascol is member of several French and American neurological and pharmacological societies. He was the Secretary of the international Movement Disorders Society (2006-2009) and is a member of the WFN Research Committee on Parkinsonism and Related Disorders. Dr Rascol is chair of the European Section of the Movement Disorders Society (2013-15). Dr Rascol is working or has worked as associate-editor for the Journal Fundamental and Clinical Pharmacology and is or has been a member of the editorial board of Lancet Neurology, Neurology, the European Journal of Neurology, the Journal of Neural Transmission, Evidence Medicine.

Dr Rascol has published more than 380 articles in International Scientific journals (New England Journal of Medicine, Lancet, Lancet neurology, Annals of Neurology, Neurology, Archives of Neurology, Brain, Movement Disorders...). His H factor is 50. He has also been invited to give more than 250 lectures in various European, North and South American and Asian universities or national and international meetings.



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PETER RIEDERER

/Germany

Date of birth: 21st March 1942, Königsberg, Germany 1960-1968: Studies on Technical Chemistry at the TU Vienna, Austria; 1970: DoctorDegree; 1979: Associate Professor (Univ.-Dozent) TU Vienna; 1983: titl. A.o. Univ.-Prof. TU Vienna, Austria; 1986 – current; from 1971 to 1986 I was Head of ClinicalNeurochemistry at the thereof Ludwig Boltzmann Institute, Lainz Hospital, Vienna,Austria. Since 1986 Univ.-Prof. (University Würzburg); Head, Clinical Neurochemistry;Clinic and Polyclinic of Psychiatry and Psychotherapy, Univ. Wuerzburg, Germany.After my retirement on April 1st 2010 I am guest professor at the later Institute andUniversity.

14 international awards and Honorary Memberships in Scientific Societies; Member of theDeutsche Akademie der Naturforscher Leopoldina (2007); Honorary Member of theHungarian Academy of Sciences (2007); Honorary Dr. degree Int. Univ. Catalunya,Barcelona (2008); Involved in several Current International Joint Projects; Current assistancein several scientific Journals;Board Memberships: President of the (DGBP) German Societyof Biological Psychiatry 1994-1998; President of the (ESCNP) European Society for ClinicalNeuropharmacology (1995/96); President of the (DPG) German Parkinson Society 2000-2004(Vice president until 2000); Organizing Chairman World Congr. Biol Psych. 2001, Berlin.President 16th Int. Symp. Parkinson's Disease 2005, Berlin. President 39th Int. Danube Symp.Neurol Sciences 2007, Würzburg. President 1st Int. ADHD Congress 2007, Würzburg.Membership in 15 scientific societies: Organizer of numerous symposia, workshops,congresses; Publications and lectures: about 950 scientific papers in the field of neurology andpsychiatry, including hand-book articles; 20 books relevant to the field of psychiatry andneurology; Head Brain Bank Center Würzburg and BrainNet EuropeII subgroup

Main scientific interest

My main interests in the past have been research projects in the field of neuropsychiatry,especially introduction of selegiline into the treatment strategies of Parkinson's disease(PD) (1974/5), discovery with my co-workers of amantadines and memantinesmechanisms of action (1989, 1991), discovery of the role of iron in PD (1985 – present),discovery of the loss of respiratory chain activity (complex I) in PD (1989), developmentof the concept of clinical neuroprotection (1983 – present), neurodevelopmental aspectsof neuropsychiatric disorders (2001 – present), etiological aspects of neurodegenerativedisorders.I have a close relation to clinical research programs, human biochemistry/physiology,biochemical, molecular and pharmacological aspects of pathogenesis ofneurodegenerative disorders including post mortem analysis and access to the BrainNetEurope II-initiative. This is reflected by my list of more than 1.000 publications.



PILLE TABA

/Estonia

Dr Pille Taba is an Associate Professor of Neurology, and Head of the Department of Continuing Medical Education at the University of Tartu, Estonia. She has been a member of the Movement Disorder Society since 1995, and has served on the MDS European Executive committee and European Education committee. She is a founding member and President of the Estonian Movement Disorder Society, President of the Estonian Society of Neurologists and Neurosurgeons, and a member of the Scientific Advisory Group of Neurology of the European Medicines Agency.

Dr Pille Taba was graduated from the University of Tartu, Estonia, and received her medical training at the University of Vienna, the Karlstad University Hospital, and the Minneapolis Clinic of Neurology. Her research interests have been focused on toxic parkinsonism, and epidemiology and psychosocial aspects of Parkinson's disease. She was the editor of the Estonian Guidelines for Management of Parkinson's Disease, and an invited speaker at many conferences and educational courses for Estonian and Eastern European neurologists. Pille Taba has developed professional contacts in several countries and has organized conferences and educational meetings.



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CLAUDIA TRENKWALDER

/Germany

Claudia Trenkwalder is Professor of Neurology at the University 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. She was head of the “Movement Disorders and Sleep” research group at the Max Planck Institute of Psychiatry in Munich, before moving to the Department of Clinical Neurophysiology at the University of Göttingen in 2000.

She has published many papers on movement disorders and sleep, especially on the pathophysiology, genetics and treatment of restless legs syndrome. She is currently vice-president of the World Association of Sleep Medicine and member of the Executive Committee of the International RLS Study Group. In addition, she is a member of various Committees of the Movement Disorder Society, and of many national and international medical societies and boards.



FRANCESC VALLDEORIOLA

/Spain

Present position

Consultant in Neurology
Parkinson's Disease and Movement Disorders Unit
Service of Neurology
Institut Clínic de Neurociències
Hospital Clínic i Provincial, Barcelona.
C/ Villarroel, 170, 08036-Barcelona, Spain

Previous professional experience

- 1) Resident in the Neurology Service from the Hospital Clínic of Barcelona.
- 2) Fellowship in the Neurology Service from the Hospital Clínic of Barcelona in the study of "Detection of antineuronal antibodies by immunoblotting in patients with paraneoplastic neurologic diseases".
- 3) PhD fellow in the Service of Neurology of the Hospital Clínic of Barcelona.

Educational Background

M.D. by the University of Barcelona, July, 1988.
Mark: Excellent

DOCTORATE degree by the University of Barcelona, December, 2001, Mark: Excellent "Cum Laude" with the unanimity of the Jury and Doctorate extraordinary award, given by the University of Barcelona in July 2003.

Specialization

-Neurology: Titled by the Spanish Education and Science Ministry, 1993.

Training in other sites

- Centre Hospitalier Universitaire, Grenoble (France). 5/95. Head: Dr. Pierre Pollak.
- Emory University, Atlanta, GA (USA). 11-12/1995. Head: Dr. Malon. DeLong
- Northwestern Hospital, Toronto, Ontario (Canada). 5/97 Head: Dr. A. Lozano



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Memberships

- Catalan Neurological Society
- Spanish Neurological Society
- European Neurological Society
- Movement Disorder Society
- Institut d'Investigacions Biomèdiques "August Pi i Sunyer"
- Brainstem Reflexes Society

Publications

More than 180 publications in indexed journals and books.

More than 200 presentations to Meetings and Congresses.

25 book chapters

More than 200 invited conferences

Organization of International Courses on Deep Brain Stimulation

Participation in expert committees in several fields of movement disorders

Participation in more than 40 clinical trials as associated or principal investigator

Recipient of prizes from the Spanish Neurological Society and the European Neurological Society



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