

## **Patients with Parkinson's disease and Spindle Alteration**

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### **Abstract**

Parkinson's disease is a neurodegenerative disorder characterized primarily by motor symptoms, including bradykinesia, rigidity, postural instability, and tremor. Although the disease process in PD is not restricted to a specific brain area, these symptoms are mostly caused by the loss of dopaminergic neurons in the substantia nigra pars compacta resulting in a reduction or depletion of dopamine. Lewy body aggregations of alpha-synuclein in the brain are a central feature of PD pathology. These inclusions typically start in caudal areas of the brain and progress anteriorly, and may take place years prior to involvement of the substantia nigra and associated development of motor symptoms. Specifically, Braak et al.'s PD staging is based on Lewy-body distribution, which rise from the dorsal motor nucleus of the vagus nerve in the medulla and in the olfactory bulb (stage 1) emerging through the subceruleus-ceruleus complex and the magnocellularis reticular nucleus (stage 2), the substantia nigra, the pedunculopontine nucleus and the amygdala (stage 3), the temporal mesocortex (stage 4), and finally reaching the neocortex (stage 5 and 6). Stage 1 and 2 were considered as pre-Parkinsonian states, stage 3 and 4 as Parkinsonian states and 5 and 6 as late-Parkinsonian states. In addition to the motor manifestations that define PD, non-motor symptoms such as sleep problems, depression, dementia and attention deficit, autonomic symptoms as abnormal heart rate variability and gastrointestinal symptoms such as nausea and constipation are all well known in patients with PD. Stating the presence of at least two of the four motor symptoms resting tremor, bradykinesia, rigidity, and postural imbalance typically makes the clinical diagnosis of PD, although it has been indicated that the pathological changes in the striatal dopaminergic system develop several years before the clinical appearance of PD. Further development of the pathology may result in Lewy Body Dementia. Polysomnographic (PSG) EEG data from 15 patients with PD and 15 sex- and age-matched control subjects with no history of movement disorder, dream-enacting behaviour or other previously diagnosed sleep disorders were included in this study. The subjects were all recruited from the Danish Center for Sleep Medicine (DCSM) in the Department of Clinical Neurophysiology, Glostrup University Hospital in Denmark. All patients were evaluated by a movement specialist with a comprehensive medical and medication history and a PSG analysed according to the American Academy of Sleep Medicine (AASM) standard. The diagnostic certainty for PD at Danish neurological departments has been reported to be 82%. None of the PD patients had dementia at inclusion, but one of the patients with PD later developed Multiple System Atrophy (MSA), indicated as the Parkinsonian type (MSA-P) as the patient had predominating PD-like symptoms. Subjects were excluded from the study if they were taking medications known to effect sleep. However, dopaminergic treatments were permitted despite their potential effect on vigilance and SS characteristics. In addition to ethical concerns regarding discontinuing dopaminergic treatment in these subjects, we wanted to avoid deleterious discontinuation effects on the PSG, as well as unpleasant and negative motor effects that could interfere with the study. The quality of each PSG recording was individually examined, and recordings with disconnections or significant amounts of signal artifact were not included.