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1. Introduction

1.1 Cognitive impairment in Parkinson’s disease

Idiopathic Parkinson’s disease (PD) is a neurodegenerative disorder characterized by basal ganglia dysfunction frequently being associated with frontostriatal dysfunction and cognitive impairment. The prevalence of PD increases with age and is estimated at 100-200/100,000 people (Chen et al., 2001; Schrag et al., 2000) worldwide. The clinical hallmarks of PD are akinesia, rigidity and tremor (Douglas et al., 1999; Hughes et al., 1992). In the past PD has been considered as a pure movement disorder, but in recent years the presence of non-motor symptoms in PD has been recognized. Non-motor symptoms include a variety of autonomic dysfunctions such as orthostatic hypotension, postural tachycardia, bladder dysfunction, sleep disturbances, psychiatric symptoms, i.e. depression, hallucinations or psychosis and cognitive impairment. Non-motor symptoms such as pain, depression or sleep disturbances might precede the onset of motor symptoms in PD and are sometimes even more disabling than motor deficits. For many years cognitive impairment and the occurrence of dementia have been considered as not typical for IPD. James Parkinson (Parkinson, 1817) wrote in his essay on the shaking palsy “the senses are not disturbed”. However, there is now enough evidence in the literature that dementia might occur in up to 40% of PD-patients (Emre et al., 2004). PD dementia is the third most common reason for dementia. Dementia in PD has been associated with reduced quality of life, greater sensitivity to medication, higher risk of developing psychosis, shortened survival (Levy, 2002), increased caregivers stress and frequent transfer to nursing homes (Aarsland et al., 2000) compared to PD-patients without dementia. In contrast to dementia mild cognitive impairment might occur early in the course of the disease. Approximately, a quarter of PD-patients without dementia have mild cognitive impairment (PD-MCI) and 20% might have MCI at the time of diagnosis (Aarsland et al., 2011). The cognitive deficits in PD are specific and include executive dysfunction, attentional and visuospatial deficits. Executive functions include control, manipulation, and cognitive flexibility (Funahashi et al., 2001; Lezak, 1995) and is part of working memory (Carpenter et al., 2000). The executive system is thought to
be involved in handling new situations outside the domain of automatic psychological processes (no reproduction of learned schedules or set behaviours). The theoretic model of the executive system has been modified several times over the years. Crucial contributions to the concept of executive functions came from Norman (1980, 2000), Shallice (1982), Baddeley (1986) and Miller & Cohen (2001). In summary, executive functions involve planning and decision making, influence our handling and the processing of information. Furthermore, they are involved in error corrections or troubleshooting, in situations which require new sequences of actions. Components of the executive systems are attention (focusing on relevant information), selective visual attention, inhibition (inhibition of irrelevant information) (Smith & Jonides, 1999), overcoming of strong habitual responses or resisting temptation (Burgess & Shallice, 1996), task and time management, monitoring and coding of information for processing in the working memory, flexibility, set maintenance and set shifting. The executive system can be viewed as a manager enabling the adaptation of the perceptive, cognitive and motor system to new tasks. Some authors have claimed that cognitive control is the primary function of the prefrontal cortex (Miller & Cohens, 2001). Cognitive control is implemented by increasing gain of sensory or motor neurons that are involved in task or goal relevant actions (Miller & Cohen, 2001). Patients with impaired executive functions face many difficulties in everyday life. They have a low attention span, difficulties in problem solving and decision making, in dual tasking, in set shifting, in visuoconstructive tasks, in adaptation to new tasks and even in verbal learning and delayed recall. Thus, PD-patients with impairment of executive functions have difficulties in simultaneously driving a car and searching for a street or in preparing a meal for several people. They also have difficulties in keeping appointments. Relatives report that patients avoid difficult tasks and retreat from social life. Executive dysfunctions also affect the social components and the interaction with other people (Smith & Jonides, 1999). Patients are reported of being more irritable and having difficulties in suppressing inadequate behaviour.

It has been proposed that executive dysfunction underlies all manifestations of cognitive impairment in PD (Lewis et al., 2005) as part of the ‘frontal-executive brain syndrome’ (Godefroy, 2003). In accordance Colman et al. (2009) found that executive dysfunction also underlies the performance of PD-patients on verb production. Pathophysiologically (Leverenz et al., 2009) cognitive impairment in PD might be either associated with catecholaminergic or indolaminergic neurotransmission or with Alzheimer’s disease (AD) related pathology. While the first form manifests mainly with non amnestic features like impaired EF, and might be correlated with Lewy related pathology in limbic and neocortical regions. The second type of CI manifests in amnestic CI and might derive from processes of AD intersecting with PD. 40% of patients develop dementia (Emre et al., 2004).

1.2 Pathophysiology of cognitive impairment in PD
Decline of cognitive performance in PD might result from rupture of nigro-striatum-thalamus cortical circuit interconnecting the striatum to the prefrontal cortex, cholinergic deficits through the differentiation of neurons in the nucleus basalis of Meynert and the pedunculopontine-lateral dorsal tegmental neurons (Calabresi et al., 2006).
In PD the production of dopamine (DA) in the substantia nigra (SN) is decreased. DA is a major neurotransmitter of the basal ganglia, contributing seriously to the development of


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Diagnostics and Rehabilitation of Parkinson's Disease presents the most current information pertaining to news-making topics relating to this disease, including etiology, early biomarkers for the diagnostics, novel methods to evaluate symptoms, research, multidisciplinary rehabilitation, new applications of brain imaging and invasive methods to the study of Parkinson's disease. Researchers have only recently begun to focus on the non-motor symptoms of Parkinson's disease, which are poorly recognized and inadequately treated by clinicians. The non-motor symptoms of Parkinson's disease have a significant impact on patient quality of life and mortality and include cognitive impairments, autonomic, gastrointestinal, and sensory symptoms. In-depth discussion of the use of imaging tools to study disease mechanisms is also provided, with emphasis on the abnormal network organization in parkinsonism. Deep brain stimulation management is a paradigm-shifting therapy for Parkinson's disease, essential tremor, and dystonia. In the recent years, new approaches of early diagnostics, training programmes and treatments have vastly improved the lives of people with Parkinson's disease, substantially reducing symptoms and significantly delaying disability. Written by leading scientists on movement and neurological disorders, this comprehensive book should appeal to a multidisciplinary audience and help people cope with medical, emotional, and practical challenges.

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