Parkinson’s Disease (PD) is named after James Parkinson (1755–1824), who first described the symptoms and called the syndrome shaking palsy (Parkinson, 1817). The syndrome is characterized by involuntary resting tremors, muscle rigidity and slowness of movement. (As the latter phenomenon can be misinterpreted as muscle paralysis, the disease is therefore sometimes also called paralysis agitans). These problems are caused by a dopaminergic depletion in the substantia nigra, resulting in poorly functioning basal ganglia. Often the motor symptoms are dominant in PD patients, but problems with cognition, mood and other systems also occur. Only recently, language functioning problems have been reported. In processing long complex sentences, for example, PD patients are slower and less accurate than healthy control subjects (Grossman et al., 1992; Lieberman et al., 1990) and they show a different activation pattern with less activation in the striatal region (Grossman et al., 2003). Other language problems that have been mentioned in PD are: verb inflection deficits (Longworth et al., 2005; Ullman et al., 1997); verb generation deficits (Crescintini et al., 2008; Péran et al., 2003), impaired semantic priming (Castner et al., 2007; Copland, 2003); difficulty understanding metaphoric meanings (Monetta and Pell, 2007).

Most of these problems have also been reported in patients with agrammatic Broca’s aphasia. Broca patients evidently suffer from a linguistic deficit: their performance is influenced by grammatical variables. For example, English and Dutch speaking Broca patients have problems understanding reversible passive sentences such as (1) (Bastiaanse and Van Zonneveld, 2006; Grodzinsky, 1995). Also, it is more difficult to produce a sentence with an unaccusative verb – a verb in which the theme is subject instead of object - as ‘the glass’ in (2) – than to produce a sentence with a similar verb in a transitive construction (3) (Bastiaanse and Van Zonneveld, 2005; Lee and Thompson, 2004).

(1) the cat is chased by the dog
(2) the glass breaks
(3) the woman breaks the glass

Recently, Bastiaanse et al. (2009) showed that these deficits can only be explained in terms of grammatical complexity; neither the frequency of lexical items nor the frequency of syntactic construction plays a role.

Broca patients often have brain lesions in (the vicinity) of Broca’s area. Their lesions may include or even be restricted to (subcortico-cortico connections with) the basal ganglia. The question that arises is whether the underlying causes of the language problems in Broca’s aphasia and PD are similar, that is, whether the language deficits in PD are caused by a specific underlying linguistic problem, as in Broca’s aphasia, or by other cognitive deficits that are known to accompany PD. For example, both a properly functioning verbal working memory and adequate linguistic processing are required to understand long and complex sentences (4).
Cognitive functions and PD

As said above, PD is caused by dopaminergic depletion in the substantia nigra, resulting in poorly functioning basal ganglia, which causes the typical motor phenomena and a number of cognitive problems that are presumably located in the frontal lobes. The two introductory papers of this special issue discuss the individual and interactive aspects of neurophysiology and cognition in PD (Bartels and Leenders; Koerts et al., respectively).

Patterns of memory deficits, both in working memory and in procedural memory, sequencing problems (not only motor sequencing) and disorders in executive functions are associated with PD. With regard to the latter, problems with inhibition, switching and attention have been mentioned. Each of these cognitive functions plays a role in language processing and, theoretically, each of these may be related to the language problems in PD. As stated above, problems with processing long and complex sentences like (4) might be due to a linguistic deficit similar to Broca’s aphasia and/or to verbal memory problems. However, considering that PD patients have a more general sequencing problem, the problems with these sentences and sentences in general can also be due to the fact that language is a recursive system, which requires sequencing by definition. In fact, according to Lieberman (2007) the basal ganglia are a ‘sequencing engine’ and therefore play a crucial role in sentence processing: this ‘engine’ can generate an indefinite number of sentences by applying a finite number of rules to a finite number of words, and when it does not function properly, language problems are expected.

Though it has been established that PD patients are impaired in several cognitive functions and have a number of language problems, further research must explore the interaction between their language problems on the one hand and their impairments due to sequencing, verbal memory and executive functions on the other. Unfortunately PD has been relatively overlooked in adult language disorder studies; the majority has focused on patients with aphasia. Moreover, in aphasia studies the presence of additional cognitive disorders has not been well controlled, and only seldom is the correlation between language performance and other cognitive functioning a topic of discussion. Language, as a means of communication, is a higher cortical function and, of course, it is intensely interacting with other cognitive functions.

Studying language problems in combination with impaired cognitive processes (verbal memory, sequencing, set switching etc.) in PD provides a window into the relationship between language processing and the other cognitive functions.

Several explanations in terms of cognitive impairments have been given for the language problems in PD: reduced capacity of verbal working memory (Caplan and Waters, 1999; Hochstadt et al., 2006), executive resource limitation (Grossman et al., 2003), sequencing problems (Lieberman et al., 1990), reduced semantic priming (Kischka et al., 1996), set switching (Péran et al., 2003). Thus there is disagreement over the causes of the language impairment in PD.

The goal of the present special issue is to sharpen the distinction between language and other cognitive functions in PD. This is done by presenting a series of recent studies on linguistic processing in PD. In order to put these research papers in a framework, the first two papers give an overview of the neurophysiological (Bartels and Leenders) and cognitive (Koerts et al.) deficits in PD. In these papers it is explained how dopamine depletion leads to malfunctioning of the basal ganglia. The basal ganglia are connected to the frontal cortices and are supposed to mediate specific cognitive functions of the frontal cortex. Impaired functioning of the basal ganglia thus explains why verbal working memory, sequencing and executive functioning – cognitive functions of the frontal lobes – are affected and other cognitive functions located elsewhere in the brain, such as information storage and procedural memory, are relatively intact.

The research papers have been ordered thematically along two dimensions. The first part focuses verb production and semantic processing. These studies show that PD patients perform poorer than age-matched control subjects and qualitatively differently from Broca patients. Their performance is not influenced by linguistic factors, such as linguistic complexity or regularity of past tense, but rather by non-linguistic variables such as sentence length. However, their scores on the language tests are significantly correlated to non-linguistic cognitive functions: verbal working memory and set switching/inhibition abilities (Colman et al.). PD patients’ semantic priming patterns are different from normal (Copland et al.), and the difference increases dramatically when they do not take the drug provided to suppress the symptoms of PD (levadopa) (Angwin et al.). An functional magnetic resonance imaging (fMRI) study reports that these action verb generation problems are related to frontal-motor dysfunctioning (Péran et al., 2003).

All language functions that have been tested in these PD studies are also impaired in Broca’s aphasia. However, the performance of PD patients can be directly related to their impaired cognitive functioning. These studies all show that the dopamine deficiency in the basal ganglia has an effect on cortico-striatal-cortico information exchange, which results in poor performance on language tasks that involve not only executive, but also motor functions stored in the frontal cortices.

The second part of this issue is dedicated to studies that use offline and online [event related potentials (ERP), eye
tracking] methods to test sentence and text comprehension in PD. Impaired comprehension is shown to be related to frontal functions such as ‘theory of mind’ for processing metaphores (Monetta et al.), and sequencing (Kotz and Schmidt-Kassow) and inhibition problems for sentence comprehension (Hochstadt). The final study by Dominey and Innui presents a model that relates language and other cognitive functions to the function of the striatum.

The overall conclusion that can be drawn from the studies is that PD patients encounter problems understanding and producing language. These problems in PD are less severe than in Broca’s aphasia, although there is a great overlap in symptoms: impaired sentence comprehension, poor verb production, diminished semantic activation. Nevertheless, the difficulties are not qualitatively similar. Broca patients and patients with PD have problems with verb production and generation, but with different aspects of the verb. Both have problems with long and linguistically complex sentences; in Broca’s aphasia this is caused by the fact that linguistic processing as such is affected, whereas in PD impaired cognitive functions, that are required to perform the task, seem to be decisive. For example, several studies show that set switching and inhibition influence performance, and these functions are required in the language tests that assess different variables. The patients have to switch between linguistically simple and complex sentences (Hochstadt) or between sentence in the present and past tense (Colman et al.). It is this switching that causes the problems in PD, as shown by Hochstadt’s eye tracking study, rather than linguistic complexity. In other words, the problems that PD patients face when presented with language tasks are not linguistic in nature, but caused by various impaired cognitive functions, such as verbal working memory and set switching.

The studies also show the role of the basal ganglia in language processing. The connections between the frontal lobes and the basal ganglia mediate both automatic and controlled processing (see Koerts et al.), which are both required to understand and produce language. There is no consensus yet as to the exact contribution of the distinguished cognitive functions, such as ‘verbal working memory’, ‘motor functioning’, ‘inhibition’, ‘set switching’. Whether these functions can or should be distinguished, and/or whether the tests that are used to assess these functions are really measuring what they propose to measure, is still debated. However, it is certain by now that PD results in impaired functioning of language, without a linguistic deficit per se: patients perform poorly in comprehending long and complex sentences and understanding metaphors, lies and other ‘second order beliefs’. Also, production of verbs and semantic priming is impaired. This results, naturally, in diminished verbal functioning in daily life, although there is no linguistic deficit. This has important clinical implications: although the cognitive problems result in a language problem, treatment should be different than in aphasia therapy. The focus of PD therapy should not be to train linguistic functions, but rather to address the cognitive functions that are the cause of deficits in verbal working memory, switching, inhibition and sequencing.

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**REFERENCES**


