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A deficit in optimizing task solution but robust and well-retained speed and accuracy gains in complex skill acquisition in Parkinson's disease: Multi-session training on the Tower of Hanoi Puzzle

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ABSTRACT

Introduction: There are inconsistent results in the research literature relating to whether a procedural memory dysfunction exists as a core deficit in Parkinson's disease (PD). To address this issue, we examined the acquisition and long-term retention of a cognitive skill in patients with moderately severe PD. To this end, we used a computerized version of the Tower of Hanoi Puzzle.

Methods: Sixteen patients with PD (11 males, age 60.9 ± 10.26 years, education 13.8 ± 3.5 years, disease duration 8.6 ± 4.7 years, UPDRS III "On" score 16 ± 5.3) were compared with 20 healthy individuals matched for age, gender, education and MMSE scores. The patients were assessed while taking their anti-Parkinsonian medication. All participants underwent three consecutive practice sessions, 24–48 h apart, and a retention-test session six months later. A computerized version of the Tower of Hanoi Puzzle, with four disks, was used for training. Participants completed the task 18 times in each session. Number of moves (Nom) to solution, and time per move (Tpm), were used as measures of acquisition and retention of the learned skill.

Results: Robust learning, a significant reduction in Nom and a concurrent decrease in Tpm, were found across all three training sessions, in both groups. Moreover, both patients and controls showed significant savings for both measures at six months post-training. However, while their Tpm was no slower than that of controls, patients with PD required more Nom (in 3rd and 4th sessions) and tended to stabilize on less-than-optimal solutions.

Conclusions: The results do not support the notion of a core deficit in gaining speed (fluency) or generating procedural memory in PD. However, PD patients settled on less-than-optimal solutions of the task, i.e., less efficient task solving process. The results are consistent with animal studies of the effects of dopamine depletion on task exploration. Thus, patients with PD may have a problem in exploring for optimal task solution rather than in skill acquisition and retention per se.

1. Introduction

There is a commonly accepted notion that the basal ganglia (BG) are involved in the regulation of at least some aspects of procedural knowledge and more specifically, the generation of long-lasting procedural memory (Abbruzzese, Trompetto, & Marinelli, 2009; Foerde & Shohamy, 2011a, b). An influential theoretical framework for the role of BG in procedural learning has been put forward by Saint-Cyr and Taylor (1992). The basic proposal was that the striatum is transiently involved during the early stage of procedural learning mobilizing new procedures and selecting among known procedures i.e., a procedural memory buffer. Others however, have suggested that the role of the BG is in later stages of skill acquisition, consolidation and proceduralization (Doyon et al., 1997). Support for the notion of the BG as

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critical for procedural learning and memory comes from results obtained from patients suffering from degenerative diseases involving the BG such as Parkinson's disease (PD). Other studies, addressing the hypothesis that procedural memory deficits are related to BG dysfunction in PD, provided mixed results and alternative interpretations for the deficit were offered (e.g., Soliveri, Brown, Jahanshahi, Caraceni, and Marsden (1997)) One of the most frequently used tasks in this context is the Serial Reaction Time (SRT) in which an implicit motor sequence is learned (Nissen & Bullemer, 1987). A meta-analysis of studies that tested PD patients with the SRT task, concluded that PD patients were impaired compared to controls (Siegert, Taylor, Weatherall, & Abernethy, 2006). Vakil, Kahan, Huberman, and Osimani (2000) have shown that patients with focal lesions of the BG were also impaired on motor and non-motor versions of the SRT task. These results were interpreted as supporting the involvement of the BG in motor as well as non-motor sequence learning. Patients with PD were found to be impaired in the acquisition of skill when other tasks were used, including complex tracking (Frith, Bloxham, & Carpenter, 1986), the Tower of Hanoi Puzzle (TOHP) (Daum et al., 1995), and the Tower of Toronto (a simplified version of the TOHP) (Saint-Cyr, Taylor, & Lang, 1988). PD patients were also reported to be impaired in a probabilistic learning task (weather prediction) (Knowlton, Mangels, & Squire, 1996). However, apparently conflicting results were reported even within the same study (e.g., Harrington, Haaland, Yeo, and Marder (1990), Soliveri et al. (1997)). In the Harrington et al.'s study, PD patients were impaired in a motor skill learning task (rotary pursuit) but not in learning a visuo-perceptual mirror reading task. Some studies do not support the hypothesis of procedural memory deficit in patients with PD, at the very least the performance differences found were difficult to interpret as indicating procedural learning deficits per se (Frith et al., 1986; Soliveri et al., 1997; Soliveri, Brown, Jahanshahi, & Marsden, 1992). For example, Reber and Squire (1999) reported normal learning rates in an artificial grammar learning task in PD patients.

Attempts to resolve the inconsistencies between research findings with regard to the BG hypothesis of procedural learning focused on the heterogeneity of the PD patients studied (Heindel, Salomon, Shults, Walicke, & Butters, 1989; Vakil & Herishanu-Naaman, 1998) or the heterogeneity of the tasks used to test procedural learning. Daum et al. (1995) have shown that patients with PD had difficulties in learning the TOHP, but not a perceptual task (mirror reading). The authors proposed that, impairment in the acquisition of the more cognitively demanding task (TOHP) is consistent with the dysfunction of the fronto-striatal circuitry in PD patients.

Skill learning is a multi-phase process with the process developing over many practice sessions (Anderson, 1987; Karni et al., 1998). All models agree that 'control' processes are engaged in the early phases, while later phases reflect increasingly more 'automatic' processes and as such are mediated by different brain regions (Chein & Schneider, 2005). Several studies sought to address the question of which phase of skill acquisition patients with PD find most difficult; aiming to characterize the phases of learning mediated by the BG. Doyon et al. (1997) have shown that both patients with PD and patients cerebellar lesions failed to attain 'automatization' in a visuomotor skill learning task in relatively advanced stages of task acquisition. Similarly, studies of prose learning and word list memorization and paired associates learning have suggested that PD patients have difficulties in attaining 'automaticity' (Faglioni, Botti, Scarpa, Ferrari, & Saetti, 1997; Faglioni, Scarpa, Botti, & Ferrari, 1995). In contrast, Krebs, Hogan, Hening, Adamovich, and Poizner (2001) found that patients' impairment was most pronounced in an early phase of training a novel motor task.

Most of the evidence for and against the notion of a skill learning deficit in PD comes from studies which addressed only limited, early, phases of skill acquisition. Performance gains attained within a given training interval do not necessarily suffice to trigger procedural memory consolidation processes and are not synonymous with attainment of the automaticity (fluency with high accuracy) which characterizes skilled performance (Anderson, 1987; Chein & Schneider, 2005; Hauptmann, Reinhart, Brandt, & Karni, 2005; Karni et al., 1998). There is good evidence indicating that the transition from one phase of skill acquisition to the next is highly constrained by the structure of the training experience: specifically, factors such as the number of task iterations afforded within a training instance, time and time in sleep after the training experience and the affordance of multiple training instances (Hauptmann et al., 2005; Korman, Raz, Flash, & Karni, 2003; Korman et al., 2007). Expert performance requires multi-session training (e.g., Korman et al. (2003); and see Chein and Schneider (2005)).

The current study was designed to address the question of what phase, if any, in the acquisition and retention of a cognitive skill is deficient in PD. Data pertaining to this issue would not only be of paramount importance to our understanding of the underlying cognitive deficits in PD, but also in advancing our understanding of the role of the BG in skill learning and the retention of procedural knowledge. To this end participants were trained extensively on the TOHP (i.e., 18 consecutive trials) for three sessions, 24 to 48 h apart, and in an additional session six months later. The TOHP was chosen because it is a well established model task for studying cognitive problem solving (Anderson, Albert, & Fincham, 2005). Cognitive problem solving tasks are considered to be more sensitive for detecting skill learning impairments in PD patients (Saint-Cyr et al., 1988), particularly when the puzzle is not straightforward to solve (Schneider, 2007). Previous studies have shown that PD patients are not impaired in solving Tower puzzles (training effects were not tested) (Alberoni, Della Sala, Pasetti, & Spinnler, 1988; Morris et al., 1988). We hypothesized that a complex tower puzzle would require extensive training before fluency in task solution is attained.

2. Methods

2.1. Participants

Consecutive patients with PD were recruited from the Parkinson Disease and Movement Disorders Clinic at Sheba Medical center. The diagnosis of idiopathic PD was made by a neurologist specializing in movement disorders, on the basis of (a) the presence of at least two of the three cardinal symptoms (bradykinesia, rigidity and resting tremor) and (b) good response to chronic dopamine replacement therapy. Exclusion criteria included (a) diagnosis of dementia on the basis of clinical examination or a Mini-Mental State Examination score (MMSE) of 24 or less (Folstein, Folstein, & McHugh, 1975); (b) history or current evidence of other neurological and/or psychiatric disorders (including head trauma, substance abuse, and major depression); (c) use of active central nervous system therapies other than nocturnal sedatives and dopaminergic medications; (d) any prior neurosurgical intervention, including stereotactic procedures for PD. The study was approved by the local ethics committee, and all participants gave their informed consent prior to inclusion. Sixteen patients (11 males), mean age 60.87, $SD = 10.26$ (42–77 years); formal education 13.77 (8–22) years, diagnosed with idiopathic PD (disease duration: 8.6 ± 4.7 years) were recruited. All patients were on medical treatment with L-dopa formulations with a L-dopa equivalent dose of 705 ± 421.9 mg/day (Tomlinson et al., 2010). The patients were classified as moderate PD according to the Unified Parkinson's Disease Rating Scale (UPDRS, Fahn, Elton, & UPDRS Program Members, 1987) part III, obtained in the on-medication state (16 ± 5.3).

The control group consisted of 20 healthy volunteers (11 males), mean age 61.5, $SD = 10.12$ (40–75 years); formal education 13.5 (8–22) years. The two groups were matched for age, gender and education. Both groups scored similarly on the MMSE (28.4 ± 1.1 and 28.8 ± 0.9, PD and controls, respectively).

Exclusion criteria included (a) diagnosis of dementia on the basis of clinical examination or a Mini-Mental State Examination score of 24 or less (Folstein et al., 1975); (b) history or current evidence of other neurological and/or psychiatric disorders; (c) use of CNS medications other than nocturnal sedatives and