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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

dopaminergic medications; (d) any previous neurosurgical intervention, including stereotactic procedures for PD.

2.2. Task and procedure

Tower of Hanoi Puzzle—TOHP: A computerized version of the task was used. Three pegs appeared on the screen, numbered 1–3. Four disks were arranged according to size with the largest disk at the bottom of the extreme left peg (#1). Participants were told that the goal was to move the disks from the left-most peg (#1) to the right-most peg (#3) in a minimum number of steps and that they had to keep the following rules: only one disk could be moved at a time; no disk could be placed on a smaller one; the middle peg had to be used. The optimal solution for four disks requires 15 moves. Time and the number of moves required to solve the puzzle were recorded for offline analysis.

Participants were tested individually. Training (testing) was held over four sessions ((A)–(D)) and participants were required to solve the TOHP consecutively 18 times in each session. The first three sessions were separated by 24–48 h (short delay, A-B, B-C), the 3rd and 4th sessions by 4 to 8 months (long delay, (C)–(D). The length of the sessions varied among participants as a function of the number of moves and time to solution. Thus, the earlier sessions were about 1 h long and later sessions were about half an hour long.

3. Results

3.1. Learning and short term delay—The initial three learning sessions (A, B & C)

A mixed design MANOVA with repeated measures was used in order to test the effects of Group (PD and Control) as a between-subjects factor and Learning trials (1st to 18th), and Session (A)–(C) as within-subject factors. Two dependent measures were analyzed: (a) the number of moves to solution (Nom); (b) the average time per move (seconds) (Tpm).

3.1.1. Number of moves—Nom

The mean Nom required for solving the TOHP for the PD and Control groups across the learning trials in the three sessions, is presented in Fig. 1. Overall, the groups did not differ in the Nom required to solve the TOHP, $F(1, 30)=2.45$, $p>.05$. Main effects of learning trials and Session were significant, $F(17, 510)=4.07$, $p<.001$ and $F(2, 60)=16.97$, $p<.001$, respectively, with an overall decrease of Nom required for solving the TOHP from the 1st to 18th trial and from the first to the third session. Group by Learning trials was the only statistically significant interaction, $F(17, 510)=1.67$, $p<.05$. As can be seen in Fig. 1, the PD group underwent a larger (steeper learning) reduction in Nom (from 31.56 to 22.27) compared to that of the controls (from 24.31 to 18.67). This finding should be interpreted cautiously because it probably reflects a ceiling effect in the control group. Control participants attained task solution on average in 18.88 Nom by the third session (C).

3.1.2. Time per move—Tpm

The average Tpm required for solving the TOHP across the learning trials in the three sessions is presented in Fig. 2. As with Nom, the groups did not differ significantly in the average Tpm in solving the TOHP, $F(1, 30)=1.28$, $p>.05$. Both groups showed an overall significant decrease in the Tpm required to solve the TOHP from first to 18th trial, $F(17, 510)=34.24$, $p<.001$, within the sessions and from the first to the third session, $F(2, 60)=105.08$, $p<.001$. Learning trials by Session was the only interaction that was significant, $F(34, 1020)=10.80$, $p<.001$. As can be seen in Fig. 2 the within session improvements in Tpm were reduced from the first to the third session as overall performance speed improved in both groups.

3.2. Short term delay—First three learning sessions (A to B & B to C)

The effects of the delay, between-sessions, were indirectly reflected in the analyses reported above, when all 18 learning trials of the session were compared. However, to directly assess the effect of the delay in the two groups, performance on the last trial of a given session was compared to that of the first trial of the following session. This yielded two intervals for comparisons (18th trial of session (A) to 1st trial of session (B) & correspondingly between sessions (B) and (C).

3.2.1. Number of moves—Nom

There was a significant effect of delay, $F(1, 3)=7.35$, $p<.05$ as well as a group effect, $F(1, 30)=7.66$, $p<.001$. Indicating that overall, following the between-sessions delay more moves were required to solve the TOHP by participants of both groups and that the PD group required more moves on average to solve the puzzle, both at the end of sessions (A) and (B) and at the beginning of the subsequent sessions (B) and (C). Nevertheless, none of the interactions were significant and importantly, the interaction of Learning trials and Group was not significant, indicating that the effects of the delay intervals were similar in both groups.

3.2.2. Time per move—Tpm

All main effects, Session and delay were significant, $F(1, 30)=15.74$, $p<.001$ and $F(1, 30)=18.98$, $p<.001$, respectively. As with Nom, Tpm slowed down following the delay intervals. However, overall Tpm was faster in the second session. The interaction between these two factors was marginally significant, $F(1, 30)=3.73$, $p=.063$, indicating that the delay effect in the second session was more moderate than in the first. However, again, the interaction of Learning trials and Group was not significant, indicating that the effects of the delay intervals were similar in both groups.

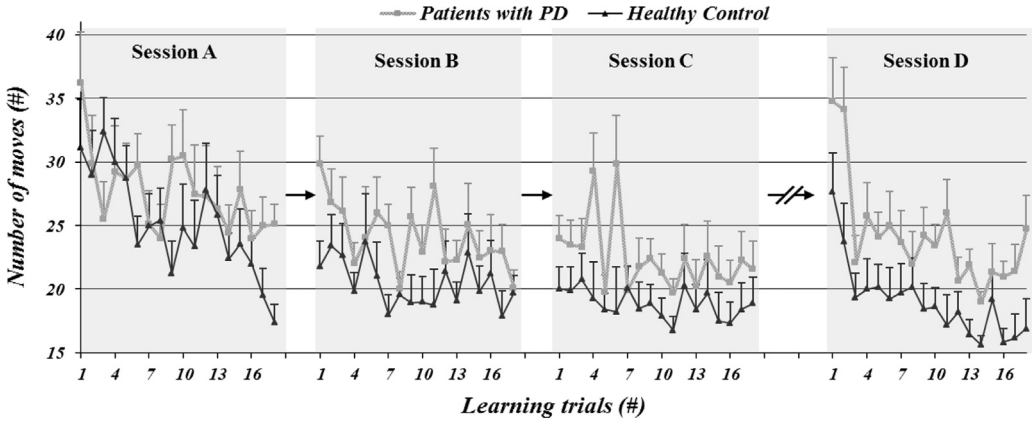

Fig. 1. TOHP (Tower of Hanoi) learning curve showing number of moves. Number of moves for solution of the TOHP, for the PD and control groups, as a function of 18 learning trials by four sessions.

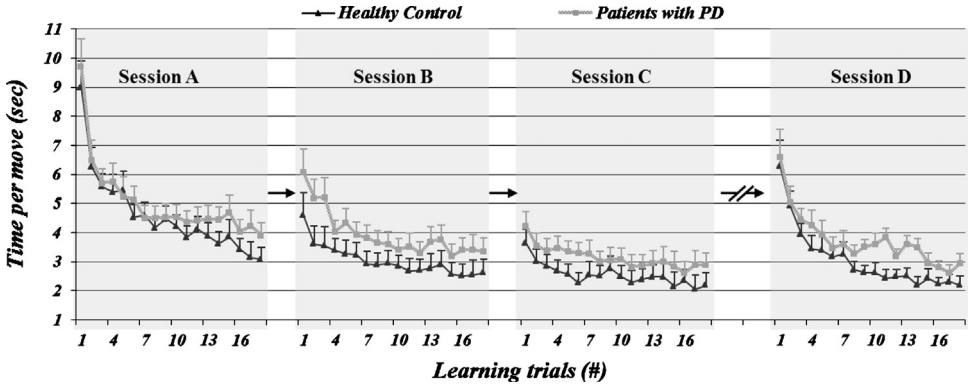

Fig. 2. TOHP (Tower of Hanoi) learning curve showing time per move. Time per move for solution of the TOHP, for the PD and control groups, as a function of 18 learning trials by four sessions.

3.3. Retention over a six months delay—First vs. fourth session (A)–(D)

To assess the long-term retention of the TOHP solution procedure, the performance in the first (A) and the fourth (D) sessions was compared, using a mixed design MANOVA with repeated measures with Learning trials (1–18) and Session (A) and (D) as within-subjects factors and Group (PD and Control) as a between-subjects factor. The number of moves to solution and the average Tpm (seconds) were analyzed separately, as the dependent measures.

3.3.1. Number of moves—Nom

There was an overall significant decrease in the Nom to solution across the learning trials in both sessions $F(17, 306) = 3.97$, $p < .001$ (Fig. 1). However, the Nom required to solve the TOHP in session (D) was significantly reduced compared to session (A), $F(1, 18) = 7.80$, $p < .05$. Thus, both groups demonstrated, to the same extent, retention of the skill learned over a long delay. In both sessions, the PD group required more moves to solve the TOHP, $F(1, 18) = 8.76$, $p < .01$. However, none of the interactions reached significance.

3.3.2. Time per move—Tpm

In both groups, there was a significant decrease in the average Tpm required for solving the TOHP across the learning trials of both sessions (A) and (D) $F(17, 306) = 38.41$, $p < .001$ (Fig. 2). Moreover, in session (A) participants of both groups required on average a significantly longer Tpm compared to session (D), $F(1, 18) = 75.30$, $p < .001$. As can be seen in Fig. 2, the rate of improvement in speed was steeper in session (A) as indicated by the significant Learning by session interaction, $F(17, 306) = 2.91$, $p < .001$. However, there was no significant difference between the groups, $F(1, 18) = 2.00$, $p > .05$, as well as no significant Group interactions, indicating that learning rates and retention over time did not differ significantly between the PD and Controls.

3.4. Retention over a six months delay—Third vs. fourth session (C - D)

In order to assess the long term effect (i.e., 4–8 months) on the learned procedure (i.e., solving the TOHP), performance in the third session (C) was compared to that on the fourth session (D). A mixed design MANOVA with repeated measures was conducted in order to analyze the effects of Group (PD and Control), Learning trials (1st to 18th), and Session (C) and (D). The former is a between-subjects factor and the latter two are within-subjects factors. Two dependent measures, Nom and Tpm, were analyzed.

3.4.1. Number of moves—Nom

The mean Nom required for solving the TOHP for the PD and Control groups across learning trials in the two sessions (C) and (D), is presented in Fig. 1. The group effect was marginally significant, $F(1, 18) = 4.06$, $p = .06$, indicating that overall the PD group required more moves than the control group in order to solve the TOHP. There was an overall significant decrease of Nom required to solve the TOHP from first to 18th trial, $F(17, 306) = 3.47$, $p < .001$. Overall, the Nom required to solve the TOHP in the fourth session was not significantly different from that in the third session, $F(1, 18) = .29$, $p > .05$. The session by Learning Rate was the only interaction that reached significance, $F(17, 306) = 2.40$, $p < .001$. As can be seen in Fig. 1, the learning rate in the fourth session was steeper than that in the third session.

3.4.2. Time per move—Tpm

The average Tpm required for solving the TOHP for the PD and Control groups in the two sessions (C) and (D), is presented in Fig. 2. Unlike the finding with the Nom, overall the groups did not differ significantly in the average Tpm in solving the TOHP, $F(1, 18) = 1.70$, $p > .05$. There was an overall significant decrease in Tpm required for solving the TOHP from first to 18th trial, $F(17, 306) = 28.80$, $p < .001$. Overall, the average Tpm required for solving the TOHP in the third session was significantly lower than that in the fourth session, $F(1, 18) = 37.39$, $p < .001$. The Learning by session interaction was the only interaction that reached significance, $F(17, 306) = 7.35$, $p < .001$. As can be seen in Fig. 2, the learning rate was steeper in the fourth session compared to the third session. The lack of interactions with Group effect indicates that the groups' learning rate and retention over time did not differ significantly.

3.5. Long term delay—Last trial session (C) to first trial session (D)

The delay effect is confounded when the whole session consisting of the 18 learning trials is analyzed. Therefore, comparing the groups' presumably best performance following the three training sessions (trial 18, session (C)), to their first trial of session (D), after a long delay would provide a purer measure of the long delay effect.

3.5.1. Number of moves—Nom

Both main effects reached significance. Overall the PD groups needed more moves to solve the TOHP, $F(1, 18) = 4.65$, $p < .05$. Both groups needed more moves in the session following the long delay (C to D), $F(1, 18) = 14.16$, $p < .005$.

3.5.2. Time per move—Tpm

The Tpm was not significantly different between the groups, $F(1, 18)=0.14$, $p > .05$. Both groups needed more moves in the session following the long delay ((C) to (D)), $F(1, 18)=54.65$, $p < .001$.

4. Discussion

Altogether, the current results do not support the notion of a core deficit, in patients with PD, either in gaining task solution fluency or in generating procedural memory for solving the TOHP. Both speed (time per move) and task solution accuracy (number of moves to solution) improved robustly across the initial training sessions, importantly, with no speed accuracy tradeoff. This pattern of improvement is considered a hallmark of skill acquisition (Shmuelof & Krakauer, 2011; Stelmach, 1996). Moreover, long-term retention, across an interval of six months, was as effective in the patients with PD as in their healthy peers compared with their respective performance in session (C). Also, when performance in the final session (session (D)) was compared to baseline performance (i.e., in session (A)), both groups showed similar, significant gains. Nevertheless, our results show that a difference emerged between the groups in the number of moves to solution and this trend attained statistical significance in sessions (C) and (D) though already apparent in the final trials of session (A). The implications are that although the patients with PD may need more moves to solve the TOHP compared to controls, they preserved the learned skill as effectively as their healthy peers.

As can be seen in Fig. 1, patients with PD set out as equal in performance to their healthy peers but tended to settle on less-than-optimal solutions of the task, i.e., exhibited less efficient task solving as their experience accumulated. We propose that this "less than efficient" performance may reflect more tolerance for unnecessary moves (errors) during the learning process in the patients with PD. The current results are consistent with animal studies of the effects of dopamine depletion on task solution explorations (Doya, 1999; Parush, Tishby, & Bergman, 2011). Thus, our results suggest that patients with PD may have a problem in exploring for optimal task solution rather than in skill acquisition and retention per se.

Our results are consistent with the previously proposed notion that patients with PD may have a problem in attaining an effective task solution (set) (Frith et al., 1986). Similarly, Soliveri et al. (1997) have suggested that patients with PD have no problem in motor, procedural, learning per se but rather mobilize ineffective task solution strategies. One possible deficit that the authors consider is a declarative system failure in their patients with PD; alternatively they suggest that patients may have a problem in moving from a working task solution to a better one. The current results can be considered in the light of these proposals, although the intact performance at the early stages of training is not fully consistent with the approach that patients with PD show a failure of the declarative system. Indeed imaging studies have suggested that patients with PD may rely more on the declarative system in skill acquisition (Doyon, 1997). Moreover, the lack of a learning deficit in such a complex and cognitively demanding task as a four discs TOHP does not support the notion of a problem of setting and using (mobilizing, Soliveri et al. (1997)) appropriate task solution strategies. Nevertheless this task does call for, and in fact requires continuous exploration in order to reach an optimal solution. Studies (e.g., Soliveri et al. (1992)) have also suggested that patients with PD may require more practice to attain optimal task solution. However, in these studies only relatively short practice was afforded and the ability to retain the performance gains over long time intervals (long-term procedural memory) was not tested. The current results suggest that when extensive practice is afforded, patients spend more time on repeating less-than-optimal task solutions, and thus may generate and consolidate into procedural memory relatively ineffective routines.

In the current study participants were trained extensively in three sessions, 24–48 h apart, 18 trials in each session and in an additional session six months later. Such a design is expected to provide sufficient training to enable the setting up (attainment) of a working task solution routine and its mastery (proceduralization), if a potential for this type of cognitive skill learning exists. Our results confirmed this assumption. In some of the previous studies where a procedural memory dysfunction was implicated, the training afforded on the target task was limited, so that it is not clear whether proceduralization was attained; retention was tested only over relatively short intervals (e.g., Saint-Cyr et al. (1988), Schneider (2007), Soliveri et al. (1997), Reber and Squire (1999)). Although our results are much in line with the notion of a 'behavioral persistence deficit' in patients with PD, as proposed by Schneider, to the best of our knowledge the current study is the first to test the effect of extensive training in a complex cognitive task, as well as the retention over short and long delays in PD patients.

In both groups, learning within sessions and between sessions was evident as improvement in accuracy as well as in speed. Furthermore, the retention of the acquired skill did not differ significantly between groups whether tested after short (i.e., 24–48 h) or long delays (i.e., six months). The performance of the two groups differed only in the overall number of moves, but not in the time per move (speed), required to solve the TOHP a measure wherein robust gains were expressed in both groups. As can be seen in Fig. 1, the PD group did learn the task; but unlike the control group, their performance stabilized at a less-than-optimal level by the end of the 3rd and 4th sessions. Thus, the patients had in effect received much practice on longer (less efficient) task solution routines. A similar trend, an overall improvement, was reflected in the measure for speed (see Fig. 2). Although a small gap in speed emerged in the more advanced sessions, the difference in Tpm, between the groups, did not reach significance.

The finding that the PD group apparently stabilized on a less-than-optimal solution can be interpreted as reflecting an impairment in acquisition as well as in the retention of procedural knowledge from session to session (procedural memory). However, our results support an alternative interpretation; the results are consistent with the notion that the patients have a problem in exploring for optimal task solution rather than a deficit in the retention of acquired skill at each session. The ability to retain the gains accrued in training was as robust as the one characterizing the control group.

Visual inspection of the learning curves in Figs. 1 and 2 suggests that a group difference emerges already by the end of the first session (trials 15–18). While in the healthy controls performance in these blocks approaches optimal performance in terms of Nom and continues to improve in terms of speed (Tpm), the patients do not show such improvements in Nom and a small gap in Tpm opens up during the latter part of session (A). We propose that because patients with PD stabilize on a less-than-optimal performance, a gap emerges between their performance and the performance of their healthy peers and is pervasively maintained in subsequent sessions; although both groups show significant improvements in performance. Thus, the relative performance disadvantage of the patients with PD in the 3rd and 4th sessions in terms of Nom, could be due to the consolidation of a less-than-optimal task solution routine. Our results therefore suggest that rather than a problem in learning and the generation of long term memory, PD patients may learn and retain less-than-optimal solutions.

Indirect support for our interpretation comes from some previous studies. Schneider (2007) who tested PD patients with easy and difficult levels of the TOHP found that the patients' difficulties emerged only with the more difficult puzzles. Schneider's interpretation of the results is that unlike controls, the patients have difficulties in persevering, i.e., maintaining mental effort. This interpretation could explain our results as well. That is, the PD patients were able to solve the TOHP, but were less driven to continue searching for a better solution and thus improve their performance by solving the puzzle more efficiently (i.e., with fewer moves). Schneider's study made use of the Tpm measure as well (referring to it as "thinking time per move") and interestingly, as in the present study, when this measure was used the groups did not differ even in the most difficult puzzles, underscoring the fact that the motor impairment per se is not a source of deficit in patients with PD; they are no slower in the initial trials and their fluency robustly increases with practice at a rate not different from that of their matched controls.

The current results and the proposed interpretation are also in line with the theoretical framework suggested by Doya (1999). Doya suggests that while the cerebellum operates as a directional error correction teacher in a model of supervised learning, the BG's operations are a basis of reinforcement learning. BG deficits, therefore, would result in diminished reinforcement in the process of trial and error learning. Diminished reinforcement in turn would entail less-than-optimal solution as observed in the current study. Nevertheless, Willingham, Koroshetz, and Peterson (1996) proposed that while new mapping of visual cues and motor responses may be mediated by the cerebellum the learning of a repeated motor sequence is mediated by the BG. Another proposal (Gabrieli, Stebbins, Singh, Willingham, & Goetz, 1997) suggests that the BG are involved in an open-loop skill learning phase which requires planning and is dependent on delayed feedback. This view of the role of the BG is consistent with the current findings; solving the TOHP requires planning and is dependent on knowledge of results afforded only after subsequent moves, and often only after task completion.

In a recent review, Foerde and Shohamy (2011a) concluded that the BG are involved in response-contingent feedback. Furthermore, based on their study with patients with PD as well as on an fMRI study, Foerde and Shohamy (2011b) suggested that the BG and the hippocampus have complementary roles in learning, processing immediate and delayed feedback, respectively. The present findings do not support this proposal given the delayed feedback in the TOHP. Possibly, the TOHP is not an ideal task to directly test Foerde and Shohamy's proposal because it affords an internal evaluation of the person's own performance rather than external feedback. Our interpretation of the current results as a deficit in optimizing task solution is compatible with the hypothesis proposed by Smith and McDowall (2006) of impaired sequence integration in patients with PD. In their study, using a variant of the SRT task, patients with PD were able to learn spatial and object-response sequences separately, but had difficulties integrating information from these two sequences.

A recent model proposed by Parush et al. (2011) suggests that dopamine has a role in "setting the action policy on a scale of risky to conservative" behaviors (p. 7). The persistence of less-than-optimal task solutions observed in the PD patients, in the current study, could be interpreted in light of this model. Reduced dopamine level in PD patients presumably leads to more conservative and less explorative (and risky) behavior. Thus, when patients reach a working solution they lack the drive to explore more efficient ways of solving the task, thereby increasing the odds for persisting in using a less-than-optimal solution and maintaining it across multiple subsequent task iterations. The fact that Tpm was as effectively shortened in patients and controls, indicates that both groups optimized their performance on their respectively adopted solutions. However, while controls increased fluency on the optimal solution, PD patients, trained and consolidated a less-than-optimal solutions. This was apparent in session (D) (after a six months interval). Both groups showed effective retention (compared to the performance in both sessions (A) and (C), in terms of Tpm and Nom) but the performance of the healthy controls, in terms of Nom, was near optimal by the end of session (D). The patients' performance was less-than-optimal (see Fig. 1).

The finding that the group differences reached statistical significance only at the advanced stages of training is apparently consistent with proposals that PD patients are more impaired in the advanced stages of skill learning, specifically automatization (Doyon et al., 1997; Faglioni et al., 1995, 1997; but see Krebs et al. (2001)). However, the findings that neither speed per move nor the long-term retention were deficient, indicate that automatization per se is preserved. Importantly, the current results suggest the possibility of a qualitative difference in how task solution is attained in individuals with PD compared to their healthy peers rather than in the ability to attain automatization. This interpretation is consistent with recent animal studies on the effects of dopamine depletion in the BG (Doya, 1999; Parush et al., 2011). However, this interpretation should be taken cautiously due to the fact that the patients with PD who participated in the current study were medicated with dopaminergic medications known to impair some cognitive functions while improving others (Cools, Barker, Sahakian, & Robbins, 2001). Therefore, our proposed interpretation of the role of the BG deficits in slowing early task solution choices, rather than reducing the ability to generate procedural memory, should be validated with either newly diagnosed, unmedicated, PD patients or with patients with localized damage to the BG. In addition, future studies should also address younger patients with less severe symptoms. The difference between the groups emerged towards the advanced stages of training. Fatigue cannot account for the current results because the initial sessions were longer than the later sessions; fatigue effects would have been expected to emerge in the longer sessions. In addition, there was a clear improvement in speed within each of the four sessions, in both groups which is inconsistent with fatigue effect.

The fact that the groups did not differ at their early stages of learning indicated that PD patients do not have a difficulty in understanding the task requirements or in problem solving per se. This is in line with previous work showing intact learning and transfer in artificial grammar and dot pattern categorization tasks (Reber & Squire, 1999). There is good evidence that performance of a single trial of the TOHP provides a good measure of executive functions reflecting frontal lobe functioning (Lezak, Howieson, Loring, Hannay, & Fischer, 2004). It would be difficult therefore to attribute the observed deficit in the PD patients in the present study to dysfunction of the frontal lobes. The finding that the initial performance level, of patients with PD, in a tower problem solving task is in line with previous studies (Saint-Cyr et al., 1988) but does not support the proposal of a similarity in the cognitive profile between patients with PD and patients with frontal lobe dysfunctions (Owen, 2004).

A neuroimaging study, using positron emission tomography, has addressed the brain activation associated with the solution of a similar "tower" puzzle, the Tower of London (Beauchamp, Dagher, Panisset, & Doyon, 2008). The authors compared patients with PD to age matched healthy controls. They found that control participants utilized the frontal-striatal system more than the PD patients, who relayed more on hippocampal and right lateral prefrontal cortex activity. Both groups were able to acquire the new skill. These results were interpreted as implicating competing memory systems, the implicit versus the explicit memory, respectively, in the controls and PD patients. However, this interpretation cannot explain our current

findings of robust gains in speed and accuracy as well as in retention, all implicating procedural learning. We propose that the current results as well as the Beauchamp et al. results can be interpreted as supporting the notion that the BG deficits in PD are related to the (impaired) generation and attainment of an optimal solution for the task, but not to the generation per se of procedural memory. Individuals with PD may however subsequently consolidate into skill (procedural) memory a less-than-optimal solution. Further research is required in order to address the question of whether in the protocol used in the current study, individuals with PD over-engage the medio-temporal system, which is likely to be a less efficient system for automatizing solutions for the TOHP. The current finding should be considered in the context of the rehabilitation of patients with PD. Explicit encouragement and training the patients to explore alternative solutions when faced with a problem rather than settling with initial solutions, may contribute to the optimization of learning experiences in patients with PD.

5. Conclusions

Our results show that given extensive training individuals with PD are capable of learning a rather complex cognitive skill task and importantly of retaining it even over long delay periods. Nevertheless, patients may stabilize on a less-than-optimal performance level and generate long-term procedural memory for these less-than-optimal task solutions. We propose that the results are consistent with the hypothesis of an exploration deficit in individuals with PD faced with a novel task, in laboratory settings, in line with the 'behavioral persistence deficit' proposed by Schneider (2007) and the results of animal models of dopamine deficiency (Doya, 1999; Parush et al., 2011).

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