Working Memory Functioning in Medicated Parkinson's Disease Patients and the Effect of Withdrawal of Dopaminergic Medication

Nathalie Fournet
Université de Savoie

Jean Luc Roulin
Université de Savoie

Olivier Moreaud
Centre Hospitalier Universitaire de Grenoble

Bernadette Naegele and Jacques Pellat
Centre Hospitalier Universitaire de Grenoble

Cognitive impairments in Parkinson's disease (PD) could be explained by a central executive (CE) deficit in A. D. Baddeley's (1986) working memory model. To test this hypothesis, verbal, spatial, and double span tasks were given to 12 medicated PD patients and control participants, with varying recall delays. The double span task was assigned to explore the coordinating and integrating function of the CE, and lengthening the recall delay was expected to implicate more attentional resources. PD patients had lower spans relative to controls in all tasks. However, the more specific implication of the CE was difficult to prove. One reason could be that PD patients were on dopaminergic treatment when tested. To control this effect, 12 PD patients on and off medication were studied in a second experiment using the same tasks. PD patients off medication had lower spans only in the double task; this result underlines the role of dopamine on working memory processes.

Many studies have highlighted cognitive deficits in Parkinson's disease (PD; for a review, see R. G. Brown & Marsden, 1988), especially on executive tasks, suggesting a frontal dysfunction (Cools, Van Den Bercken, Horstink, Van Spanendonck, & Berger, 1984; Morris et al., 1988; Owen, Downes, Sahakian, Polkey, & Robbins, 1990). The nature of the psychological processes underlying cognitive impairments in PD and their relationship with dopaminergic systems remains controversial. Many authors have argued that an attentional deficit is a possible explanation. The cognitive component responsible for this attentional deficit could be, according to R. G. Brown and Marsden (1988), the supervision attentional system (Norman & Shallice, 1986) or the central executive (CE) in the working memory model (Baddeley, 1986, 1992). The working memory system refers to a dynamic conception of processing and temporary storage of information. Baddeley's (1986) model is assumed to comprise three components: a limited-capacity CE, supervising cognitive functioning, and coordinating two independent "slave" systems. The phonological loop is involved in the storage and processing of verbal material.

The visuospatial sketchpad (VSSP) performs a similar function for visuospatial material.

Working memory (WM) deficits are found in PD, implying a disturbance of the CE component (R. G. Brown & Marsden 1988, 1991; Dalrymple-Alford, Kalders, Jones, & Watson, 1994; Robertson, Hazlewood, & Rawson, 1996). In previous research (Moreaud, Fournet, Roulin, Naegele, & Pellat, 1997), we assessed the functionality of the slave systems: Medicated PD patients showed normal phonological similarity and word-length effects, attesting for the integrity of the phonological loop. Dalrymple-Alford et al. (1994) tested the idea that PD patients may be impaired at the level of CE processes with a dual-task paradigm developed by Baddeley, Logie, Bressi, Della Sala, and Spinnler (1986) and Baddeley, Bressi, Della Sala, Logie, and Spinnler (1991). Dalrymple-Alford et al.'s participants performed a random pursuit-tracking task (involving the visuospatial component of WM) while recalling forward digit span sequences (involving the verbal component of WM). Initial performance of PD patients and control participants on the component tasks used in this dual-task testing were equated across participants by varying the size of the target square and by using individual participants digit spans. The authors observed a poorer performance for PD patients than for control participants when the two tasks were combined. This deficit could result from difficulties for PD patients in the coordinating and integrating function of the CE component in Baddeley's WM model. However, as the authors pointed out, deficits in the dual-task testing in PD patients could also reflect "problems with the time sharing aspects of CE functions... rather than a simple reflection of poorer coordination of CE processes" (Dalrymple-Alford et al., 1994, p. 365).

This distinction between the coordination and time-sharing functions of the CE suggests that this attentional...
component is more complex and multidimensional than was originally proposed (Baddeley, 1992). Yee, Hunt, and Pellegrino (1991) clarified the distinction between coordination and time sharing. The coordination tasks necessitate the integration of several types of information. The participant must ensure that information from each component task is available at the right time and in comparable format. In Yee et al.’s view, the coordination process of the several component tasks is a task in itself, separate from the information processing for each channel. In the dual tasks, two component tasks compete for the same resource (time-sharing function) but are otherwise independent (the two tasks do not involve the same processes).

In previous research (Fournet, Moreaud, Roulin, Naegele, & Pellat, 1996), we studied the CE resource reduction by means of interference tasks of varying costs, combined with primary span tasks (verbal, spatial, or visual spans). Although the spans were systematically lower in medicated PD patients than in controls, there was no evidence for a CE deficit in this experiment. Indeed, when interference tasks became more demanding, the decrease in PD patients’ spans was no more important than that of the control participants. However, because Dalrymple-Alford et al. (1994) and R. G. Brown & Marsden (1991) described such a deficit in PD patients, it seemed interesting to us to study more precisely the CE functions. We therefore propose an investigation of the coordinating and integrating function of the CE by means of a task involving a real coordination of the subsystems. In this task (called the “double span task”; Loisy & Roulin, 1992), participants have to memorize words localized in a $5 \times 5$ matrix. This information is believed to require the participation of the two slave systems and their coordinated functioning for an efficient recall. If PD patients have difficulties in the coordinating function of the CE, then they should be deficient, relative to control participants, when the information to be maintained has a double nature (verbal and spatial), leading the CE to intervene and coordinate both types of information. In contrast, PD patients should be less deficient when the information to be maintained is single in nature (verbal or spatial) because simple span tasks are more dependent on the subsystems’ functioning. Bradley, Welch, and Dick (1989) and Owen, Iddon, Hodges, Summers, and Robbins (1997) demonstrated that patients with mild to moderate PD were impaired on a test of visuospatial WM, whereas performance on an analogous test of verbal WM was unaffected. From this view, a more pronounced deficit in the spatial span task than in the verbal span task could be observed in our experiment.

To assess the effect of greater demands on the CE, we manipulated the retention interval of information (e.g., the delay between presentation and recall of stimuli) in several span tasks. Our assumption, on the basis of previous results (Fournet et al., 1996), was that maintaining information for a longer time would make greater demands on the central resource pool. If lengthening the delay has a cost on the CE, then this procedure could be a new indicator for a resource reduction in PD.

A second hypothesis is tested in this article and is based on research stressing the role of dopamine on the WM function. Cooper et al. (1992) showed that the withdrawal of dopaminergic medication in PD selectively impaired cognitive performance in a working memory task (the digit-ordering task). Luciana, Depue, Arbizi, and Leon (1992) showed, in a study with normal healthy humans, an effect of dopamine on a visuospatial working memory task: The injection of a D2 dopamine agonist receptor (bromocriptine) improved participants’ performance in this task. Our aim was to determine whether the dopamine depletion altered the CE functioning in PD. If it did, then the withdrawal of dopaminergic medication in PD patients would impair performance in the double span task. Furthermore, the effect of delay would be greater when PD patients are off medication than when on medication, especially in the double span task.

Experiment 1: Is There a Deficit of the Coordinating Function of WM in Medicated PD Patients?

**Method**

**Participants.** Twelve idiopathic PD patients (6 men and 6 women), aged from 40 to 75 years ($M = 65.6$ years, $SD = 8.2$), with good response to dopaminergic medication volunteered to serve as the experimental group. The mean duration of the disease was $9.7$ years ($SD = 5.6$). The severity of the disease was rated by means of the participant’s score on the motor version of the Unified Parkinson’s Disease Rating Scale (UPDRS; Fahn et al., 1987; mean score $= 26.95$, $SD = 13.8$) and by the Hoehn–Yahr scale (Hoehn & Yahr, 1967). Two patients were scored at Hoehn–Yahr Stage 1, 5 patients at Stage 2, 2 patients at Stage 2.5, 2 patients at Stage 3, and 1 patient at Stage 4. At the time of testing, all patients were taking L-dopa with a decarboxylase inhibitor; 9 patients were taking a dopaminergic agonist (bromocriptine or piribedil), and 4 patients were taking selegiline. Patients with clinical evidence of dementia, according to the Diagnostic and Statistical Manual of Mental Disorders (3rd ed.; American Psychiatric Association, 1980) criteria, history of stroke, head injury, or neurosurgery (including thalamotomy) were excluded. Patients under tricyclic antidepressants or antidepressants known to present an anticholinergic activity and benzodiazepines exceeding 10 mg valium per day were also excluded. Twelve control participants (5 men and 7 women) were chosen from a volunteer participant panel and were matched to PD patients for age ($M = 64.5$ years, $SD = 14.5$) and years of education ($M = 11.5$ years, $SD = 4.6$). All participants gave informed consent.

**Procedure.** The aim of the present experiment was to assess the existence of a specific deficit of the CE in PD patients, which might partly account for their reduced spans in WM tasks (Fournet et al. 1996). We used three tasks to test each component of the working memory model: a verbal span task testing the phonological loop, a location span task testing the VSF component, and a double span task involving the CE participation for the coordination of the two subsystems’ functioning. For each span task, the retention interval delay was varied. The initial hypothesis was that maintaining information for a longer time would make greater demands on the central resource pool. If lengthening the delay has a cost on the CE, then this procedure could be a new indicator for a resource reduction in PD.

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100-ms interstimulus interval. Participants had to recall the presented words after a 0-s, 4-s, or 8-s retention interval. A short auditory tone indicated the end of the retention interval. Participants were not instructed to recall the words in their initial presentation order, and there was no time limitation. The sequences of the words were generated randomly, with several constraints: Each series was made up of one-syllable, concrete, nonsimilar words that were of controlled frequency and different semantic categories, randomly chosen from a 20-word list corresponding to the cited criteria. Each word could appear more than once in the whole task but not within a series.

The location span task, assumed to involve the spatial component (VSSP) of the WM model consisted of remembering series of filled cells presented sequentially in a 5 x 5 matrix. Ten series were presented to participants, and the locations of cells changed at each series. One cell remained filled for 1,500 ms and then became black again before the next in the series was filled. Once the filled cells of a series had been presented, the matrix disappeared during a retention interval of varying delay and then reappeared with a simultaneous tone. The participant's task was then to remember the locations of filled cells after a 0-s, 4-s, or 8-s delay interval by pointing out the cells with a pointer. The participants were not instructed to recall the position of the filled cells in the correct order of succession, and no recall time limitation was given. Despite a random selection of filled cells within the 5 x 5 matrix for each series, some configurations were eliminated: The same cell could not be filled twice within a series, and the pattern resulting from the sequentially filled cells was not symmetrical.

The double span task, then, was assumed to engage more resources than simple span task. This task consisted of the successive presentation of words in the cells of a 5 x 5 matrix. Each word (the same words as in the verbal span task were used) appeared in a particular cell of the matrix (the same as in the location span task) for 1,500 ms and then was replaced by the next word presented in another position. An auditory tone indicated the end of the series. Immediately after hearing this tone, the participant had to repeat aloud each word of the series while simultaneously pointing out its location on the blank matrix. No instructions were given about the order of recall, and there was no recall time limitation. The constraint concerning the random generation of the stimuli (i.e., word and position in the matrix) corresponded to the constraints assigned to the stimuli of the verbal and location span tasks. The type of response (corresponding to a verbal recall and a simultaneous pointing to locations) required the coordination of different types of information formats. Thus, the verbal-location link, which cannot be registered by the phonological loop or by the VSSP only, would engage resources of the CE (for storage or for coordination). Performance on this double span task was compared with the performance on simple span tasks, to evaluate the CE efficiency.

An adapted span procedure evaluated participants' span: Within each task, the first series of stimuli was fixed for each participant (three words presented in the verbal task, two locations or localized words in the spatial span and double span task). After each correctly recalled series, the number of stimuli was increased by one in the next series; in the event of failure, the number of stimuli, was reduced by one. Ten series were presented in all. This method made it possible to fluctuate around each participant's optimal performance and to then minimize the number of series needed to estimate his or her span. The mean number of presented stimuli in the last 8 series was considered to reflect the participant's span (the first 2 series being considered as practice trials). Participants were seen once, individually, within a session lasting for about 1 hr, in which they completed the verbal, spatial, and double span tasks with varying retention intervals of information. To begin, the general procedure of the task was verbally explained. Participants were always told how many stimuli to expect before each trial.

**Material.** Matrix and stimuli for all span tasks were presented on a compatible PC 386, with a 14-in. (36-cm) monitor. The use of a stick in pointing locations or localized words, for recall, facilitated the patients' movements, and the experimenter recorded response exactness.

**Results**

Analyses of mean spans were carried out by an analysis of variance (ANOVA) including a between-subjects factor (group) and two within-subjects factors (task and delay). Where significant effects or interactions were found, we conducted further post hoc comparisons to elucidate the nature of the effect. In Figure 1, we report spans for the verbal, spatial, and double span tasks.

We found a significant effect for group, $F(1, 22) = 12.08$, $p < .01$: PD patients were globally deficient in WM tasks. A main effect of task was present, $F(2, 44) = 102.76$, $p < .01$, and spans were ordered as follows: verbal > location > double. There was no Group x Task interaction, $F(2, 44) = 2.39$, ns. There was a main effect of delay, $F(2, 44) = 24.42$, $p < .01$: The longer the delay, the weaker the span. The Group x Delay interaction failed to reach significance, $F(2, 44) = 2.22$, ns: The effect of delay was the same in both PD patients and controls. We observed a significant Task x Delay interaction, $F(4, 88) = 3.26$, $p < .05$. The effect of delay was present only in the spatial task, $F(2, 44) = 24.53$, $p < .01$, and the double task, $F(2, 44) = 10.56$, $p < .01$, but not in the verbal task, $F(2, 44) = 1.63$, ns. There was no three-way interaction (Group x Task x Delay), $F(4, 88) = 0.20$, ns: The Delay x Task interaction was the same in PD patients and controls.

**Discussion**

Our results show that PD patients are significantly impaired in WM tasks, as previously reported (Cooper et al., 1992; R. G. Brown & Marsden, 1991; Dalrymple-Alford et al., 1994). The deficit was present on simple and double span...
tasks. The deficit in the spatial span task was not different from the one on the verbal span task. This result is contradictory to other studies (Bradley et al. 1989; Owen et al. 1997) in which material-specific differences were observed, with PD patients being more impaired on spatial WM tasks than on verbal WM tasks. This difference could be explained by the fact that our tasks were not strictly WM tasks but short-term memory tasks (the participants were only asked to maintain verbal or spatial stimuli without any processing of this information).

There was a span deficit, regardless of the delay (0 s, 4 s, or 8 s) for PD patients, but increasing the retention interval had different effects in the verbal and spatial span tasks. The performance in the verbal task was not sensitive to the manipulation of the retention interval. The phonological loop gets so much practice in daily life that it does not have any (or few) resource demands to function (Baddeley, 1986). The absence of effect of delay in the verbal span task is compatible with results classically observed in the Brown-Peterson paradigm (J. Brown, 1958; Peterson & Peterson, 1959). In a no-interference condition, no decline in performance with verbal information to maintain (three consonants) was observed with increasing delay (0–30 s).

In the verbal span task, with all delays confounded, PD patients showed lower performance than control participants. This deficit did not depend on the phonological loop’s functionality (a previous study showed phonological similarity and word-length effects in medicated PD patients, attesting for a normal functioning of the phonological loop in PD; Moreaud et al., 1997). This deficit in verbal span in PD patients could arise from a poor efficiency of the phonological loop refreshment process (a study of the articulatory rate of PD patients, compared with that of control participants, would be necessary to prove this hypothesis). However, the origin of this slowing of the refreshment process must be clarified.

The spatial task seems more sensitive to the lengthening of retention interval, which is not surprising because the maintenance of visual or spatial stimuli would involve more central attentional resources (Baddeley, 1986; Logie & Marchetti, 1991; Logie, Zucco, & Baddeley, 1990; Parr, 1992). This effect of delay in a VSSP short-term task is compatible with Wilson, Scott, and Power’s results (1987), which showed a memory decay over a 10-s unfilled interval in a pattern span task, and with results obtained by Parr (1992) or Owen et al. (1993) in delayed matching-to-sample tasks. Because this subsystem is more demanding, its efficiency decreases as delay lengthens. In our experiment, control participants showed the same decrease of spans with delay in this spatial task as the PD patients (no Group × Delay interaction in the spatial task), which suggests that the VSSP is functional in PD.

For the double span task, the hypothesis was that coordinating the two subsystems would monopolize the CE resources, so performance in this task may decrease for the two groups when compared with performance in simple span tasks. Furthermore, it was expected that lengthening the retention interval in the double span task would lead to a more marked decrease in PD than in controls (Group × Delay interaction in the double span task). We observed a significant effect of delay in the double span task, but this effect was not more important for PD patients than for controls (no interaction). Lengthening the retention interval did not disturb the coordination of information in working memory but only acted on the maintenance of several pieces of information (spatial or double, that is, verbal and spatial). Medicated PD patients would not have a deficit in the coordinating function because the difference in double spans between PD patients and control participants was not greater than the difference between the two groups in simple spans. This was valid even if the delay was lengthened.

It seems difficult to relate the systematic decrease in PD patients’ performance in all WM tasks (simple or double span tasks) to a reduction in the capacity of attentional resources. Indeed, PD patients are no more impaired than control participants when delay is increased, and they have no specific deficit in the double span task. This absence of a CE deficit in medicated PD patients is contradictory to previous studies (R. G. Brown & Marsden, 1991; Dalrymple-Alford et al., 1994). The reason for this could be that our tasks may not involve the same functions as other tasks evaluating the CE. R. G. Brown and Marsden (1991) and Dalrymple-Alford et al. (1994) used a dual-task paradigm in which the main attentional function is a time-sharing one (cf. Baddeley et al. 1986; 1991). One can interpret this by the fact that the time-sharing function of the CE is deficient in medicated PD patients, but the coordinating function is not. Another possibility would be that the double span task does not evaluate the CE but implicates only the two subsystems. In this respect, it would have been interesting to compare patients’ and controls’ performances on the double task by means of a multiple regression analysis to ascertain how much performance on the double task is accounted for by the verbal and spatial memory spans and how much of the variance is unaccounted for by these two simple factors. However, this analysis would have been interesting only if we had many patients, which is not the case (12 participants for each group).

How, then, can the PD patients’ deficits in all span tasks be explained? Another hypothesis can be put forward: As well as the verbal mechanism, the VSSP refreshment process may also be slowed in PD, thus explaining lower spans of PD patients in the verbal and spatial tasks. This slowing down of the refreshment processes in WM could be secondary to a more general slowing down of information processing in PD (Revonsuo, Portin, Koivikko, Rinne, & Rinne, 1993). Another explanation could be a deficit in the registration (or encoding) processes in immediate memory and a deficit in the response programming steps (Le Bras, Pillon, Damier, & Dubois, 1999).

Another alternative, which does not necessarily contradict the previous ones, is that the supply of dopamine in PD patients could influence performance in WM tasks and thus mask their attentional deficits. There have been relatively few attempts to investigate the role of dopamine on WM processes, and current knowledge is confined almost exclusively to animal neurophysiology in which evidence is accumulating for an involvement of dopamine (among other
neurotransmitter systems; cf. Rupniak & Iversen, 1993, for review). However, in humans, Luciana et al. (1992), Cooper et al. (1992), and Lange et al. (1992) have observed an improvement in WM performance by dopamine supply. To clarify the role of dopaminergic treatments on WM deficits, we studied the effect of withdrawal of dopaminergic medication in PD patients on and off medication in Experiment 2.

Experiment 2: Does the Withdrawal of Dopaminergic Medication Modify WM Performance in PD?

Method

Studying the role of L-dopa would allow us to validate the implication of dopaminergic systems in WM functioning. If WM is dopa sensitive, then medication would improve performance in WM tasks, and withdrawing this dopaminergic medication (off state) would lead to poorer performance than when on medication, especially on the double span task, which directly depends on the CE component.

Participants Twelve idiopathic PD patients, 5 from Experiment 1, volunteered for Experiment 2. All patients responded to the same inclusion and exclusion criteria as described in Experiment 1. All patients were, at that time, on dopaminergic medication and were studied under two conditions, serving as their own controls: on dopaminergic medication (patients received their treatment as usual—on state) and after a withdrawal of dopaminergic medication for about 10 hr (off state). There were 4 men and 8 women, with a mean age of 63.75 years (SD = 8.7). The mean duration of the disease was 9.2 years (SD = 4.6). The severity of the disease was rated by means of the UPDRS motor score and the Hoehn-Yahr Scale when patients were both on and off medication. The mean UPDRS score when PD patients were on medication was 20.29 (SD = 14.9) and was 40.89 (SD = 11.00) when off medication. When on medication, 2 patients were scored at Hoehn-Yahr Stage 0, scoring Stages 1 and 4 when off medication. Two patients were scored at Hoehn-Yahr Stage 1 when on medication and at Stage 3 when off. Three patients were scored at Stage 2 when on medication and at Stage 3 when off; and 2 patients were scored at Stage 2.5 when on medication and at Stages 3 and 4 when off. Finally, 2 patients were scored at Stage 3 when on medication, with their score for when off medication unknown. When off medication, with their score for when off medication unknown. Two patients were scored at Hoehn-Yahr Stage 1 when on medication and at Stage 3 when off. Three patients were scored at Stage 2 when on medication and at Stage 3 when off; and 2 patients were scored at Stage 2.5 when on medication and at Stages 3 and 4 when off. Finally, 2 patients were scored at Stage 3 when on medication, with their score for when off medication unknown. When off medication, with their score for when off medication unknown.

Procedure and material. The material and tasks used in this experiment were the same as in Experiment 1. The three WM tasks (verbal, spatial, and double span tasks) were given to the participants, and a manipulation of the retention interval was made (recall after a 0-s, 4-s, or 8-s delay). These nine experimental conditions were submitted twice to PD patients, once in each of the two medication conditions. The on and off medication conditions were counterbalanced to avoid order effects.

Results

Analyses of mean spans were carried out by an ANOVA that included three within-subjects factors (medication condition, task, and delay). Where significant effects or interactions were found, we conducted further post hoc comparisons to elucidate the nature of the effect. In Figure 2, we report, as in Experiment 1, spans for verbal, spatial, and double tasks.

The ANOVA indicated a nonsignificant effect of medication, F(1, 11) = 1.32, ns: Globally, there was no difference between the on and off medication conditions in PD patients. There was a significant main effect of task, F(2, 22) = 77.52, p < .01, and a significant main effect of delay interval, F(2, 22) = 24.53, p < .01: The longer the delay, the more difficult it became to maintain information, and this was valid along the two medication conditions, F(2, 22) = 1.97, ns. There was a Task × Delay interaction, as in Experiment 1, F(4, 44) = 5.63, p < .01. The effect of delay was different within each task, F(4, 44) = 5.63, p < .01: There was no delay effect on performance in the verbal task, F(2, 22) = 2.72, ns, but there was a significant effect in the spatial task, F(2, 22) = 21.90, p < .01, and in the double task, F(2, 22) = 7.38, p < .01. No difference was observed between the on and off medication conditions with regard to the Delay × Task interaction, F(4, 44) = 0.51, ns. The Task × Medication condition interaction was significant, as we hypothesized, F(4, 44) = 3.16, p < .05: Withdrawing dopaminergic medication had no effect on the verbal task, F(4, 44) = 0.09, ns, and the spatial task, F(4, 44) = 0.01, ns, but had a significant effect on the double span task, F(4, 44) = 11.03, p < .01.

Discussion

To explore the role of dopamine on WM processes, we studied PD patients who were either on or off dopaminergic medication in Experiment 2. Our results show an interaction between medication and tasks: The withdrawal of dopaminergic medication affects WM performance but only on a double span task. Simple span tasks involve the participation of each subsystem (the phonological loop to maintain verbal information and the VSSP to maintain spatial information), and in this condition, no effect of dopaminergic medication was found. However, the double span task, in which the coordination of verbal and spatial information is necessary, is thought to be carried out by means of the CE component and proves to be sensitive to the withdrawal of dopaminergic
medication. As predicted in our hypothesis, PD patients off medication were impaired in this double span task, and the treatment improved their performance. This result underlines the contribution of a central processing component to the WM deficits of PD patients and WM's sensitivity to dopaminergic medication. A number of studies have demonstrated that the lateral frontal cortex is critically involved in certain aspects of WM (Owen et al. 1990, 1997; Petrides & Milner, 1982) and have underlined the implication of the striatofrontal circuits in working memory (Partiot et al., 1996).

The initial hypothesis on the effect of delay was that the greater the demand on the CE was (longer delays), the lower the performance, more specifically on the double span task. If PD patients had poorer attentional resources, this effect would be greater in the off medication than on medication condition. However, results show no differential effect between the on and off conditions when the delay increased. Maintaining information for a longer time did not affect performance of PD patients off medication any more than when they were on medication. This result seems controversial with the previous finding concerning the double span task: If the two effects may be explained by the implication of the same component (the CE), then one would expect the manipulation of dopaminergic medication to have an effect in both cases, which has not been observed. Furthermore, the effect of medication is minimal at the longest delay: Perhaps simply increasing the delay does not equate to an increase in the load of attentional resources.

Conclusion

The first main topic of this study was to assess WM deficits in PD and to specify the impaired component by means of paradigms derived from Baddeley's (1986) WM model. R. G. Brown and Marsden's hypothesis (R. G. Brown & Marsden, 1988, 1991) of a resource regulation in PD is not validated by our experimental results. Lengthening the retention interval did not affect PD patients' spans any more than control participants' ones, regardless of the task. We previously put forward the hypothesis that the CE was functional in medicated PD patients (Fournet et al., 1996), although other authors have found some deficits (Dalrymple-Alford et al., 1994; Owen et al., 1997). The short-term memory deficit of medicated PD patients in Experiment 1 (lower spans in all span tasks) could have arisen from a general slowing in information processing, already described in PD (Revenssuo et al., 1993).

However, from the view of a multidimensional CE, the attentional component seems to have some deficits when there is no medication supply. We suppose that the coordinating and integrating function of the CE, distinct from the time-sharing function, is involved when performing a double span task (the participant has to coordinate the verbal and spatial nature of stimuli). A deficit in PD patients' performance is found in this double span task only when patients are off dopaminergic medication. This result shows that the CE may be affected in several ways and underlines the role of dopamine on WM, especially on the CE component processes. Such a finding confirms the assumption of Cooper et al. (1992), Lange et al. (1992), and Luciana et al. (1992): L-dopa withdrawal in PD selectively impairs cognitive performance in tests sensitive to frontal lobe dysfunction, that is, tests that necessitate CE participation.

It would have been interesting to know the implication of variables such as age, severity of the disease, or severity of the motor deficit in the cognitive deficits of PD patients. However, given that the number of participants was restricted in those experiments, correlations between cognitive test scores and clinical variables (age, UPDRS motor scores or duration of disease) have not been observed. As a conclusion, it is clear that the different paradigms used to study the CE functions in PD lead to discordant results. Thus, the necessity to dissociate these groups of tasks, that is, to specify which function each of them serve, which one is affected in PD, and which one is sensitive to dopaminergic medication, is emerging.

References


