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RESEARCH ARTICLE

Rhythmic movement in Parkinson's disease: effects of visual feedback and medication state

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Abstract Previous studies examining discrete movements of Parkinson's disease (PD) patients have found that in addition to performing movements that were slower than those of control participants, they exhibit specific deficits in movement coordination and in sensorimotor integration required to accurately guide movements. With medication, movement speed was normalized, but the coordinative aspects of movement were not. This led to the hypothesis that dopaminergic medication more readily compensates for intensive aspects of movement (such as speed), than for coordinative aspects (such as coordination of different limb segments) (Schettino et al., Exp Brain Res 168:186–202, 2006). We tested this hypothesis on rhythmic, continuous movements of the forearm. In our task, target peak speed and amplitude, availability of visual feedback, and medication state (on/off) were varied. We found, consistent with the discrete-movement results, that peak speed (intensive aspect) was normalized by medication, while accuracy, which required coordination of speed and amplitude modulation (coordinative aspect), was not normalized by dopaminergic treatment. However, our findings that amplitude, an intensive aspect of movement, was also not normalized by medication, suggests that a simple pathway gain increase does not act to remediate all intensive aspects of movement to the same extent. While it normalized movement peak speed, it did not normalize movement amplitude. Furthermore, we found that when visual feedback was not available, all participants (PD and controls) made faster movements. The effects of dopaminergic medication and availability of visual feedback on movement speed were additive. The finding that movement speed uniformly increased both in the PD and the control groups suggests that visual feedback may be necessary for calibration of peak speed, otherwise underestimated by the motor control system.

Keywords Parkinson's disease · Sensorimotor integration · Basal ganglia · Visual feedback · Motor control · Bradykinesia

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Introduction

Parkinson's disease (PD) is a progressive neurodegenerative disease, often characterized by tremor, slowness of

movement, and rigidity (Levy-Tzedek et al. 2007). An increased dependence on visual feedback for movement guidance (Flowers 1976), accompanied by a deficit in the individuals' sense of proprioception (Schneider et al. 1986; Klockgether and Dichgans 1994; Klockgether et al. 1995; Jobst et al. 1997; Abbruzzese and Berardelli 2003; Jacobs and Horak 2006), or in the ability to integrate proprioceptive information successfully (Tatton et al. 1984; Inzelberg and Korczyn 1996; Almeida et al. 2005) has been described. It has been proposed that the deficits in PD either in proprioception or in sensorimotor integration lead to increased reliance on vision (Adamovich et al. 2001; Schettino et al. 2006), which, in turn, accentuates slowness of movement (Sheridan and Flowers 1990; Flash et al. 1992; Ghilardi et al. 2000). Medication has been effective in treating some aspects of the disease (e.g., motor symptoms) better than others (e.g., cognitive decline) (Rowe et al. 2008).

Poizner et al. (2000) studied reach-to-grasp movements in PD, under different feedback conditions (full vision, vision of target and not of moving arm, and no vision of either target or moving arm), with and without dopaminergic medication. We found that, relative to age-matched controls, PD participants off medication were slower in all feedback conditions. We suggested that this was the result of a deficit of an *intensive* nature, meaning a mis-scaling of the gain occurred, such that the output is either too large (e.g., too fast, or an overshoot) or too small (e.g., too slow, or an undershoot). The authors further suggested that such *intensive* deficits can be corrected by manipulation of the gain. This is in contrast to what we defined as *coordinative* deficits, which include difficulties in the integration of different sensory modalities (e.g., proprioception with vision), the utilization of sensory input in generation of motor output, and the coordination of multi-part movements, such as reach and grasp. These *coordinative* deficits, in turn, may be less likely to be amenable to standard medication therapy, as they are likely to depend on specific, time-dependent neural activity, which cannot be readily restored by increasing the gain of the corresponding neural pathway. Consistent with this hypothesis, Schettino et al. (2006) found that dopaminergic therapy significantly increased movement speed, an intensive component, but failed to improve coordination of hand and arm movements during the reach.

We tested whether these results, which were obtained for a discrete reaching task, hold in the case of a continuous rhythmic task. Rhythmic and discrete movements traditionally have been viewed as distinct types of movements governed by different mechanisms (Hogan and Sternad 2007). Schaal et al. 2004 and Yu et al. 2007 argue that rhythmic and discrete movements may be controlled by fundamentally different brain regions and may require different computational processes. Mink and Thach (1991) showed that the cell discharge pattern in basal ganglia nuclei (globus pallidus) but not in cerebellar nuclei (dentate nucleus) differ between rhythmic and discrete movements. Moreover, it has been suggested that the generation of repetitive (Almeida et al. 2005) or sequential (Agostino et al. 1992) movements are specifically impaired in PD. As such, it is not obvious that results obtained in the discrete case would apply in the rhythmic case as well. Specifically, we tested the following hypotheses: (1) intensive parameters (such as speed and amplitude) will be improved by medication; (2) complex parameters (such as coordination of movement speed and amplitude to fit within an enclosed shape on a phase plane, see Methods section) will not be improved with medication, but (3) there will be a benefit from the availability of visual feedback. This last prediction is based on the large literature showing that vision for patients with Parkinson's disease facilitates task performance and accuracy, improving both movement initiation and movement trajectories (Flowers 1976, 1978; Sheridan and Flowers 1990; Flash et al. 1992; Georgiou et al. 1993; Klockgether and Dichgans 1994; Jackson et al. 1995), and even the coordination of multiple effectors (Poizner et al. 2000; Schettino et al. 2006).

In order to cover as broad a range of movement amplitudes and speeds when testing these hypotheses, we tested participants on three target amplitude/peak speed combinations, spanning the gamut from small-amplitude, high-speed movements to large-amplitude, low-speed movements.

The experimental paradigm was designed such that timing cues were not explicit, but rather, timing was an emergent property of the task. Timing was implicitly dictated by a closed shape on a phase-plane display (where velocity is plotted vs. position). The phase plane afforded a way to display target amplitude and frequency of movement to participants without giving them any explicit timing cues (e.g., via a metronome), thereby minimizing requirements for processing explicit timing information. The goal in choosing this display was to minimize the number of sensory modalities the participants need to integrate for successful generation of the movement.

Methods

Participants

Ten PD participants were tested when they followed their normal medication regimen ("PD ON"; UPDRS[1] score

[1] UPDRS is a four-part rating scale. Part III of the UPDRS, whose score is reported here, is designed to assess severity of the motor symptoms in patients with PD. Each item is scored from 0 (normal) to 4 (maximal severity), for a maximum (worst) score of 108.

Table 1 PD participants' clinical characterization

N	Age	Gender	Disease duration (years)	OFF MEDS		ON MEDS		Medications
				UPDRS	H and Y	UPDRS	H and Y	
1	75	M	9	31.5	2	24.5	2	LevR; Rop
2	78	M	4	45	2	33	2	Lev
3	84	F	5	42.5	2	32.5	2	Lev; Rop; Ras
4	65	F	11	45	2	28.5	2	LevR; Lev; Pr
5	66	M	7	36	2	26	2	
6	75	M	8	28	2	20	2	Pr; Azilect
7	79	M	4	44.5	3	37	2	
8	49	M	8	51.5	3	31.5	3	Lev; LevR; Sel; Ent; Rot
9	73	M	8	38	2	33.5	2	Lev; Pr; Sel; Am
10	76	M	5	34.5	2	24	2	Lev; Pr; Am

UPDRS score is out of 108; *H* and *Y* Hoehn and Yahr score, Medication codes: *LevR* Carbidopa/levodopa sustained release, *Lev* Carbidopa/levodopa (regular formulation), *Pr* Pramipexole, *Sel* Selegiline, *Ent* Entacapone, *Rop* Ropinirole, *Ras* Rasagiline, *Am* Amantadine, *Rot* Rotigotine patch

Fig. 1 The experimental setup. The participant performs 1-dimensional horizontal flexion/extension movements with the forearm to control a cursor displayed on a phase plane, where velocity is plotted versus position

29.1 ± 5.3, mean ± SD), as well as after an overnight withdrawal from dopaminergic treatment ("PD OFF"; UPDRS score 39.7 ± 7.3; age: 72.1 ± 9.9 years; 8 men, 2 women, see Table 1). Half were tested first ON and then OFF medication, and the order was reversed for the other half. ON and OFF testing was not necessarily performed on the same day. Thirteen healthy age-matched control participants without any known neurological disorders or tremor were also tested (71.3 ± 5.9 years; 7 women, 6 men). All participants used their dominant hand and gave their informed consent to participate.

Protocol

Details of the experimental apparatus were provided elsewhere (Levy-Tzedek et al. 2010). Briefly, participants were presented with the phase-plane trace of their one-dimensional horizontal forearm motion about the elbow (flexion/extension; see Fig. 1) and were instructed to keep the trace within a doughnut-shaped region on the phase plane (Doeringer and Hogan 1998; Levy-Tzedek et al. 2010, 2011); the horizontal axis displayed angular position and the vertical axis displayed angular velocity. No explicit timing cues were given. Timing on this task was emergent, as the frequency of the movement was determined by the combination of the amplitude and the speed of the movement (instructed by the phase-plane display). The protocol consisted of 3 blocks of 20 trials each, each lasting 20 s. The three blocks were differentiated by the shape of their target regions; the shape displayed was either (i) a tall thin region (fast, small-amplitude movement), (ii) a circular region, or (iii) a wide region (slow, large amplitude; see Fig. 2). Shape parameters were chosen to span a wide range of kinematic values. Approximately half of the participants were tested first on the fast block, and about half were tested first on the slow one; the second block of trials always consisted of the medium-speed condition. Prior to testing in each block, participants were allowed to practice the movement until they felt comfortable with the task, which usually consisted of four 40-s practice trials. The requirement to perform accurate movements whose trace remains between the two target ellipses on the screen was stressed. Of the 20 trials, 5 of them were no-vision (NV) trials; during these trials, participants could see the target region, but not the trace corresponding to their own forearm motion. In those trials, which always followed trials with visual feedback (V), participants were asked to continue to try and move within the guidelines even though the movement trace feedback was not provided and maintain accuracy. In those trials, as in the trials with visual feedback, no explicit timing cues were

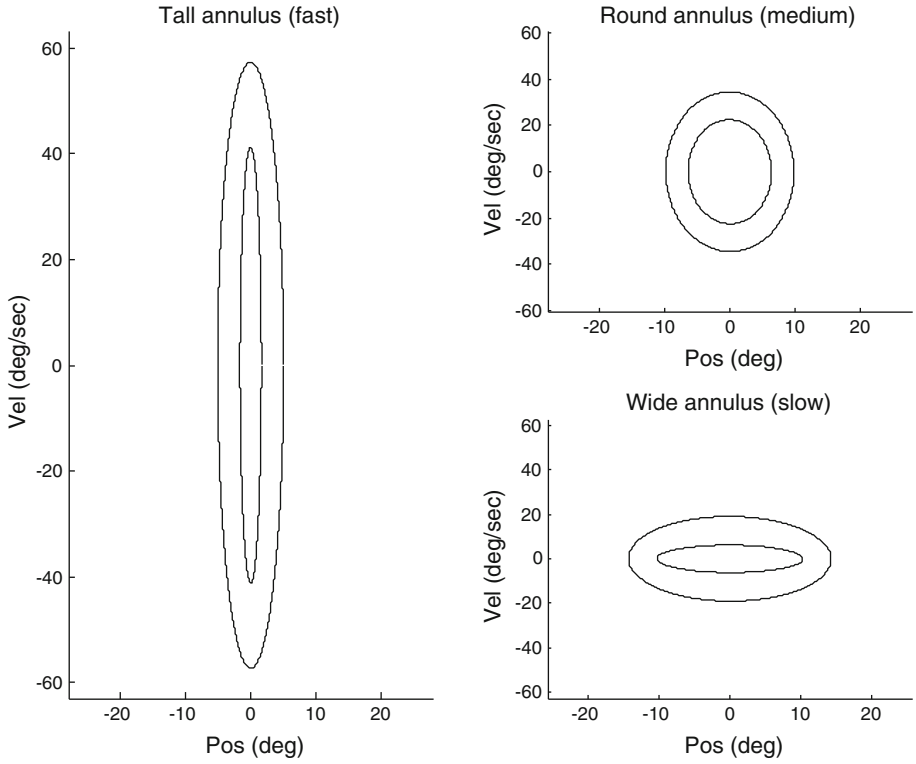

Fig. 2 The three variations in the visual display presented to participants (here, with axes labels). Central frequency and amplitude values from the left panel, clockwise: 2.3 Hz and 6.7°; 0.55 Hz and 16.3°; 0.16 Hz and 24.6°, respectively

provided. A cover was placed above the apparatus, such that during the experiment, the participants' forearm was not visible.

The protocol was approved by the Institutional Review Boards of the Massachusetts Institute of Technology and of the University of California, San Diego.

Data analysis

On occasion, participants produced movements whose center point was shifted compared with their initial center point (especially in the NV trials). To reduce the effect of this drift, any linear trend was removed from the position data. Position and velocity were filtered using a first-order Butterworth filter (cutoff 20 Hz).

Movement amplitude was calculated by taking the average, within each 20-s trial, of the extent of the forearm's angular excursion (about the elbow, in degrees) in each half cycle (flexion/extension or extension/flexion).

Peak speed was calculated by taking the average, within each trial, of the maximum absolute angular velocity in each half cycle.

The average frequency of movement in each trial was estimated by calculating the reciprocal of twice the average peak-trough horizontal distance in the position-versus-time record.

An accuracy score was assigned to each 20-s trial, which represented the percent of the total trial time that was spent inside the target zone on the phase plane.

Statistical analysis

Unless otherwise noted, a 3 (slow/medium/fast) × 2 (vision/no-vision) × 3 (PD ON/PD OFF/controls) ANOVA was used to test the differences among the blocks, feedback conditions, and groups. Post hoc comparisons (Tukey's HSD test) were used to decompose main effects.

Results

Movement traces from the fast block of two PD OFF participants and one control participant are depicted in Fig. 3; as can be seen in panels A, C and E (V trials), both PD participants never reached the minimum required peak speed,[2] and the healthy control was able to, on average, reach peak speeds above the minimum required. Panels B, D, and F (NV trials) demonstrate that all three participants performed faster movements when visual feedback was not available. Panels A and B show traces from a PD OFF participant that, upon removal of visual feedback, was able to perform movement that fit within the target annulus, and panels C and D show traces from a PD OFF participant that, upon removal of visual feedback, was able to perform movement that were even larger than the target annulus,

[2] The minimum required peak speed was denoted on the screen by the vertical extent of the inner ellipse above (and below) the vertical midpoint.

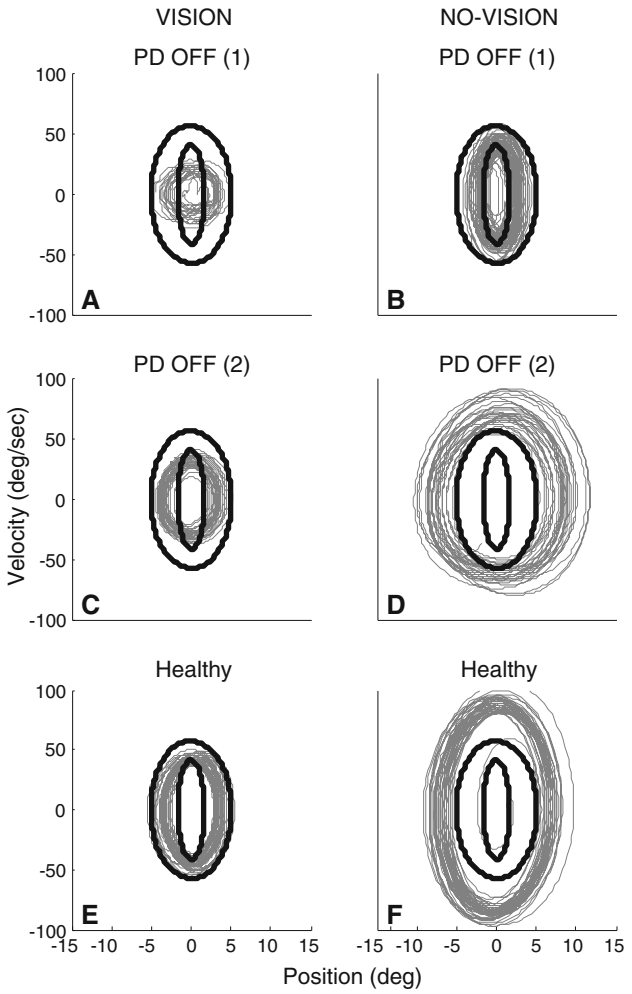

Fig. 3 Phase-plane traces (*in gray*) of two PD OFF participants and one control participant from one vision and one no-vision trial in the "fast" *block*. Participants were asked to move such that their movement traces remain between the two *black ellipses*

both in terms of amplitude and of speed. While the latter is not representative of the group's average, these data from individual participants demonstrate how striking a difference can be observed between the V and the NV conditions.

Tables 2 and 3 list the peak speed and amplitude values for PD OFF, PD ON, and control participants, with and without visual feedback, for the three experimental blocks, as well as the required ranges.

Vision versus no-vision

Participants performed faster movements when no visual feedback was available. On average, in the NV trials, compared with the V trials, the peak angular speed was 21–26% higher in the fast block, 4–20% in the medium block, and 6–24% higher in the slow block. There were significant differences ($P < 0.0001$) between the V and the NV

Table 2 Peak speed values (mean ± SE), in deg/s

	Slow	Medium	Fast
Allowed range	6.3–18.9	22.4–34.4	41.3–57.3
PD OFF			
V	15.4 ± 1.0	27.4 ± 2.0	36.2 ± 2.5
NV	17.3 ± 1.3	32.1 ± 4.7	45.6 ± 4.1
PD ON			
V	17.7 ± 1.1	33.5 ± 2.3	39.6 ± 2.5
NV	21.9 ± 1.6	40.2 ± 3.8	49.1 ± 4.2
Controls			
V	17.7 ± 1.1	35.0 ± 0.9	44.0 ± 1.6
NV	18.7 ± 1.9	36.4 ± 2.1	53.4 ± 3.1

Table 3 Amplitude values (mean ± SE), in deg

	Slow	Medium	Fast
Allowed range	20.6–28.6	12.9–19.8	3.3–10.2
PD OFF			
V	21.9 ± 0.9	10.8 ± 1.1	6.9 ± 0.9
NV	20.4 ± 2.8	13.7 ± 2.6	8.3 ± 1.2
PD ON			
V	22.4 ± 0.4	12.6 ± 1.0	7.4 ± 0.8
NV	21.7 ± 3.2	14.8 ± 2.3	9.0 ± 1.1
Controls			
V	23.0 ± 0.3	14.5 ± 0.4	8.0 ± 0.4
NV	24.3 ± 1.4	17.0 ± 1.3	10.3 ± 0.7

conditions in peak speed, with NV peak speed consistently higher than V peak speed (averaged across groups, in deg/s, mean±SE, 40.3 ± 0.4 vs. 49.7 ± 1.1 in the fast block, 32.2 ± 0.3 vs. 36.2 ± 1.0 in the medium block, and 17.0 ± 0.2 vs. 19.3 ± 0.5 in the V vs. the NV conditions, respectively; for per-group averages, see Fig. 4 and Table 2). A significant positive correlation was found between the UPDRS-ON scores and the percent increase in peak speed in the PD ON condition ($r^2 = 0.59$, $P < 0.01$). No parallel significant correlation was found in the PD OFF condition.

Overall, movements on the NV trials were of larger amplitude than those on the V trials ($P < 0.0001$). However, the differences were not uniform across groups and blocks. The control group performed larger-amplitude movements on NV trials in all three blocks. PD participants both ON and OFF medication performed movements that were on average larger on NV trials in the fast and medium block, but smaller on NV trials in the slow block (for per-group averages, see Fig. 5 and Table 3). To test whether this difference between the groups affected the significance of the results, we additionally performed a 2-way ANOVA

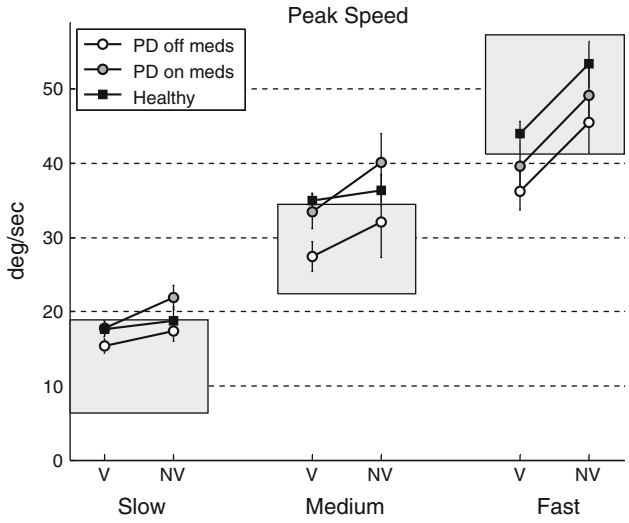

Fig. 4 Peak speed (mean ± SE) of the participants in the PD OFF (*white circles*) and the PD ON (*gray circles*) states, as well as of the control participants (*black squares*). The shaded areas denote the range of allowed peak speeds

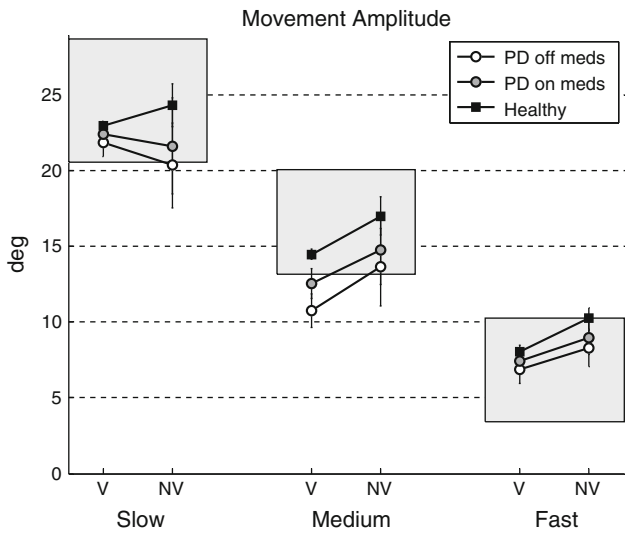

Fig. 5 Movement amplitude (mean ± SE) of the participants in the PD OFF (*white circles*) and the PD ON (*gray circles*) states, as well as of the control participants (*black squares*). The shaded areas denote the range of allowed amplitudes

(block x feedback condition) on the control data and a 3-way ANOVA on the PD group's data (block x feedback condition x medication state). Both tests indicated a significantly larger amplitude on the NV compared with the V trials ($P < 0.0001$ and $P < 0.002$, respectively). The latter test also showed PDs made significantly ($P < 0.002$) larger-amplitude movements with medication, compared to without.

Accuracy scores on the NV trials were significantly lower than those on the V trials ($P < 0.0001$; see Fig. 6), despite a requirement to maintain accuracy on these trials. This result is not surprising, given the need to rely on memory and the less-accurate sense of proprioception (Chaput and Proteau 1996) in the NV trials. It is surprising, however, that participants did not slow down their movements in the NV trials to allow more time for processing of the proprioceptive sensory information (Chaput and Proteau 1996). While we observed an overall drop in accuracy with an increase in speed, when comparing the NV to the V trials, a simple speed-accuracy tradeoff does not explain the result, as evidenced by the U-shaped accuracy function. When comparing the fast to the medium block, we found that movement at a higher rate was more accurate. We previously described the non-monotonic relationship between speed and accuracy on this task in healthy individuals. We suggested that this increase indicates that movements in the fast block are inherently different from those in the medium and the slow blocks, and in fact, these constitute two separate movement types. This conclusion was supported by a series of further analyses (for a detailed account, see Levy-Tzedek et al. 2010). As is evidenced in Fig. 6, the increase in accuracy in the fast block compared with the medium block is preserved in the PD group, whether ON or OFF medication.

We previously reported, for healthy participants, a tendency to perform movements at a slightly lower frequency in the NV compared with the V condition (Levy-Tzedek et al. 2010). Here, too, we find either no change (in the slow block) or a slight decrease in frequency (medium and fast blocks) in the control group (see Fig. 7). Tested separately from the PD group, which showed a slight but significant ($P < 0.0001$) *increase* in frequency in the NV condition, using a 2-way ANOVA (block x feedback condition), reveals a small significant difference between the V and the NV conditions, with $P < 0.04$.

Effects of PD and medication

A main effect of participant group and condition was found for the peak speed of movement ($P < 0.0001$). Consistent with several earlier reports (e.g., Kelly et al. 2002; Schettino et al. 2006), post hoc analysis revealed that PD OFF participants made significantly slower movements than controls. With medication, their movements were significantly faster than off medication, and not significantly different than controls (see Fig. 4 and Table 2).

Amplitude of movement was significantly different ($P < 0.0001$) between control participants, PD OFF, and PD ON. Post hoc analysis revealed that PD OFF made significantly smaller movements than both control participants and PD ON. While medication brought about a significant increase in movement amplitude, it was not enough to normalize the movement, and PD ON participants made move-

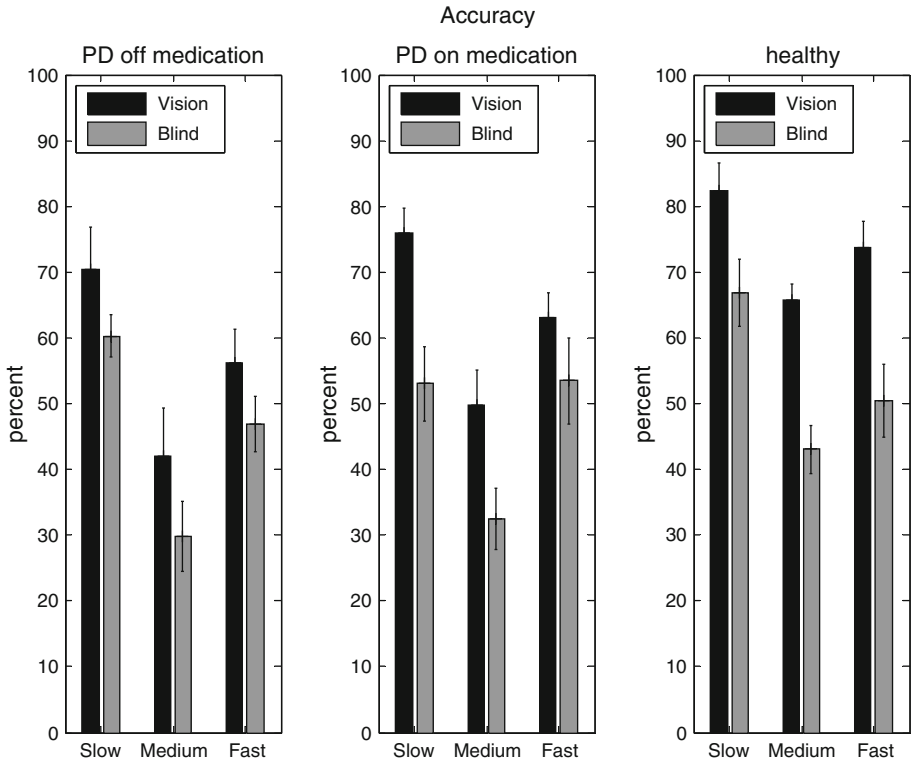

Fig. 6 Accuracy scores (mean ± SE) of the participants in the PD OFF (*left panel*) and the PD ON (*middle panel*) states, as well as of the control participants (*right panel*), with and without visual feedback

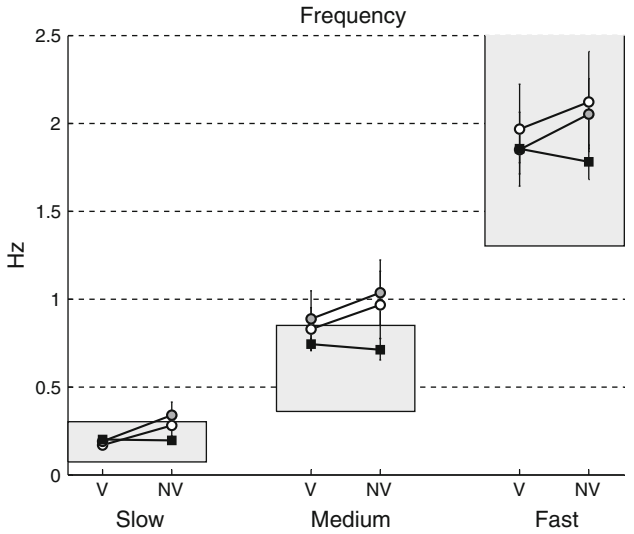

Fig. 7 Movement frequency (mean ± SE) of the participants in the PD OFF (*white circles*) and the PD ON (*gray circles*) states, as well as of the control participants (*black squares*). The shaded areas denote the range of allowed frequencies (the upper end of the range is not shown for the "fast" *block* (5.5 Hz))

ments that had a significantly lower amplitude than those of control participants (see Fig. 5 and Table 3).

Movement accuracy was significantly different ($P < 0.0001$) between control participants, PD OFF, and PD ON. Post hoc analysis showed that all three were significantly different from each other, with PD OFF making the least accurate movements and control participants making the most accurate movements. While medication significantly improved accuracy, it was not enough to normalize the movement, and PD ON participants made movements that had a significantly lower accuracy score than those of control participants (see Fig. 6).

A main effect of group was found in terms of movement frequency ($P < 0.0001$), with the control group performing the task at slightly but significantly lower frequencies that the PD participants, either on or off medication. Medication did not have a significant effect on frequency.

Variability in peak speed and in amplitude across different cycles within a trial was compared among the three groups using the Levene test. No significant difference was found in either metric, for both the V and the NV trials ($P > 0.15$).

Discussion

Basic findings

We tested whether rhythmic, continuous movements of the forearm in PD patients were modulated by vision and dopaminergic therapy in a manner similar to what has been found for discrete movements. We found, consistent with the discrete-movement results, that peak speed (an intensive aspect of movement) was normalized by medication,

while accuracy, which required coordination of speed and amplitude modulation (a coordinative aspect), was not. However, we also found that amplitude, also an intensive aspect of movement, was not normalized by medication. Furthermore, when visual feedback was not available, all participants (PD and controls) made faster movements. Finally, the effects of dopaminergic medication and availability of visual feedback on movement speed were additive.

Effect of visual feedback

We have shown that when performing a continuous rhythmic task, both PD participants and healthy controls performed movements with higher peak speed when visual feedback was not available, compared to when it was available. This uniform result across groups (PD/controls) and medication states (on/off) suggests that visual feedback may be necessary for calibration of peak speed, otherwise underestimated by the motor control system. Difficulty in the control of movement speed in this task has been previously documented in young controls (Doeringer and Hogan 1998).

Studies inspecting movement speed in discrete reaching (Flash et al. 1992; Ghilardi et al. 2000) or reaching and grasping (Schettino et al. 2006) movements produced mixed results. Some found no significant difference between the peak speed of movement with visual feedback compared to movement with no visual feedback (Flash et al. 1992; Schettino et al. 2006). Ghilardi et al. (2000) found that early-stage PD patients off medication (but not control participants) make faster planar reaching movements when no visual feedback is available. The authors, however, did not find a parallel significant increase in the healthy control group.

Effect of dopamine replacement therapy

Dopamine replacement therapy significantly increased movement speed in the PD participants, indeed normalizing it: Overall, movement speed of the PD participants on medication did not differ significantly from that of controls. In contrast, although movement accuracy significantly improved with medication, it was not normalized. It was previously reported that dopamine replacement therapy does not have uniform effects on movement (Schettino et al. 2006; Tunik et al. 2007), but that it ameliorates what have been termed intensive aspects of movement, such as speed, better than coordinative aspects, such as coordination of different body parts. The present results extend these findings to the coordination of two movement attributes (velocity and position) in a one degree of freedom rhythmic movement. Thus, dopamine repletion seems to be able to modulate the gain of specific parameters, such as speed, much more readily than reversing deficits in coordinative parameters. These latter parameters may depend upon precise, highly differentiated patterns of neuronal firing that are not restored simply by increasing dopaminergic tone.

The findings we report here in terms of peak movement speed and accuracy fit well with Schettino et al.'s (2006) hypothesis that dopaminergic treatment in PD may more readily compensate for intensive, rather than coordinative deficits. However, the fact that medication did not normalize movement amplitude suggests that dopaminergic treatment does not act to remediate all intensive deficits to the same extent. While it normalizes peak speed, it significantly improves, but does not normalize, movement amplitude.

Bradykinesia, or slowness of movement, is one of the cardinal manifestations of Parkinson's disease (PD). It has been suggested that with bradykinesia, the problem lies with inappropriate *scaling* of the dynamic muscle force to the required movement parameters (Berardelli et al. 1986; Fellows et al. 1998), perhaps as a result of incorrect perception of the necessary motor effort in order to achieve a desired motor outcome (Demirci et al. 1997). Individuals with PD were found to underscale their movements more when they had to compare movement extent with visual information, than when comparing it with proprioceptive feedback (Demirci et al. 1997). In other words, the ability to respond *appropriately* to visual input may be impaired, rather than the ability to generate the necessary motor commands. Indeed, the performance of the PD group (both on and off medication) in the current experiment at peak speeds below those of the control group and below the minimum required by the task in the fast block when visual feedback was available (see Fig. 4) is not the result of a physical limitation in the ability to generate the necessary muscle force, as demonstrated by their faster movements when visual feedback was removed. This is despite reports of physiological studies, where (1) individuals with PD produced smaller-amplitude multiple agonist bursts, unlike the stereotypical triphasic EMG pattern found in studies of healthy participants (Hallett and Khoshbin 1980), and (2) when individuals with PD perform large-amplitude movements, these bursts do not increase in duration as do those of healthy individuals (Pfann et al. 2001). In other words, slowness of movement in PD is not due to a saturation in the mechanism that produces the burst (cf. Berardelli et al. 1986). Indeed, studies have documented the ability of bradykinetic individuals with PD to make faster movements when required to make larger-amplitude movements (Berardelli et al. 1986), when they are verbally encouraged to make faster movements (Hallett and Khoshbin 1980) and when asked to focus on the sensory awareness of "movement bigness" (Farley and Koshland 2005). It was also demonstrated that individuals with PD were able to perform

a faster gait pattern when visual cues were added to their immediate environment (Morris et al. 1996). Attentional strategies, where participants are encouraged to focus on the sensory awareness of making larger movements, demonstrated an increase in velocity of gait (Morris et al. 1996; Farley and Koshland 2005) and wrist movements (Farley and Koshland 2005). Perhaps the most striking example of all for the retained ability of individuals with PD to perform movements at normal speeds is the phenomenon termed "paradoxic kinesia," or the temporary ability of individuals with PD, suffering from bradykinesia, to perform movements free of bradykinetic characteristics in the context of urgent or externally driven situations (Siegert et al. 2002). It is conceivable that there are multiple sources of movement slowness, and whereas some may be overcome—for example, by withdrawal of visual feedback—others cannot.

The fact that movement speed increased in both PD participants and healthy controls suggests that the increase in speed observed in the PD group is not a "return to normal" performance. Rather, it appears to be a parallel process, where movement speed increases regardless of whether bradykinesia is present, and if it is present, it acts to reduce its effects. In other words, the PD participants appear to exhibit a normal mechanism (increase in speed upon withdrawal of visual feedback) overlaid on top of an abnormal mechanism (slowness of movement).

A plausible explanation for why an increase in movement speed upon withdrawal of vision is not often observed when individuals with PD perform discrete movements[3] (Flash et al. 1992; Schettino et al. 2006)[4] may be that unlike a discrete movement, a rhythmic movement allows individuals to operate at resonance, leading to a large oscillation amplitude, and therefore speed, for minimal forcing input from the neuromuscular system, thereby minimizing the individual's energy expenditure (Raftery et al. 2008) and allowing for stable and reproducible movements (Hatsopoulos and Warren 1996). Why, then, do we not observe this behavior in the vision trials? It is presumed that proprioceptive information influences potential neural oscillators such that the timing of preferred oscillatory movements is not simply dictated by the central nervous system, but is constrained by the dynamics of the musculoskeletal system (Hatsopoulos and Warren 1996). Since, in the vision trials, visual feedback is available in addition to proprioception, it may act to constrain the movement to be within the task's requirements. A deficit in the ability to integrate visual and proprioceptive feedback may result in rhythmic movements that at once are not performed at resonance and that do not match task requirements in terms of speed and/or amplitude. These results support a role of the basal ganglia in visuomotor integration.

It should be noted that the negative effect of vision that we found on movement speed is not the result of visual impairments in the PD participants. While it is the case that PD patients show certain types of visual impairments, including reduced acuity, impairments in color vision and pupil reactivity (Silva et al. 2005; Cardoso et al. 2010) as well as visual hallucinations (Holroyd and Wooten 2006), such deficits could not account for the pattern of results we obtained. First of all, our visual displays were clearly visible, being well above any visual threshold. Second, our PD participants were screened for having visual hallucinations, and no participant reported having any. Finally, any primary visual or visual-perceptual deficit would have affected performance in all of our Vision conditions, yet, this was not the case: The PD participants only performed below the required movement speed in one of the three blocks (fast block).

It should be noted that there have been several reports indicating that visual cues assist PD participants in overcoming difficulties in initiating movement and help them to maintain a more rhythmic gait pattern, associated with a lower risk of falling (Morris et al. 1996). At the same time, visual feedback may also elicit the "freezing" phenomenon, characteristic of PD (Demirci et al. 1997; Almeida et al. 2003). Morris et al. (1996) found that while visual cues were helpful in normalizing gait, they were not necessary, as focusing the participants' attention on the task produced similar results.

It is clear that individuals with mild to moderate PD do not lose their ability to perform faster movements, and proper training may assist them in achieving higher movement speeds, at least in the short term (Morris et al. 1996; Platz et al. 1998). Future experiments should test how ubiquitous this result is, in the context of everyday movements.

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[3] Note there may be a difference between the performance of more natural discrete reaching movements and that of the rhythmic task at hand.

[4] Cf. Ghilardi et al. (2000).

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