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Perturbation of a Prehension Movement in Parkinson's Disease

Marina Scarpa and *Umberto Castiello

*Neurological Department, University of Modena, Modena, and *Department of Psychology, University of Bologna, Bologna, Italy*

Summary: Movement kinematics of the transport and manipulation components of a double-step prehension task were studied in eight Parkinson patients and eight control subjects. The aims were to (a) assess the effects of a spatial perturbation upon the movement for the two groups and (b) add data to the controversy about the damage/preservation of predictive behaviour in Parkinson patients. The results showed: (a) Both groups are able to preprogram a movement. (b) In both groups, the perturbation results in an anticipation of all kinematic parameters, both of the transport and manipulation components. (c) Parkinson patients, when adopting a predictive behavior, show a delay between the beginning of the two components, and thus activate them in sequence rather than simultaneously. This delay is significantly reduced by the perturbation, indicating that Parkinson patients, when using a responsive behavior, can recouple the two motor components. **Key Words:** Prehension movement—Perturbation—Parkinson's disease.

Several neurophysiological investigations (1,2) on animals have shown that a considerable proportion of cells within the corpus striatum fire when "interesting" objects appear. These neurons are polimodal units. However, as they do not respond to proprioceptive or kinaesthetic input, they are not thought to contribute to the on-line adjustment of a motor program and are therefore excluded from the closed-loop, feedback-based, responsive system of movement control. Other striatal cells, via inhibitory connections with the nucleus centromedialis of the thalamus and cortex, deal with attention. These neurons act like a filter by directing attention to a particular target while inhibiting all the other distracting inputs. They thus play a role in aimed motor behavior.

Flowers (3–5) demonstrated that subjects with disruption of nigrostriatal pathways (Parkinson's disease; PD), have difficulties in making accurate, fast, open-loop ballistic movements during step-tracking tasks. They appear to respond to events rather than anticipate them, and they show an increased dependence on visual information. He proposed that this reflected a deficit of predictive motor behavior.

Similar conclusions have been forwarded by subsequent authors. Cooke et al. (6) used visually and nonvisually guided step-tracking and ramp movements of PD patients and control subjects. In the absence of visual information about arm location, PD subjects were unable to maintain the correct arm position. Once again, they suggested that PD patients were more reliant on visual feedback. Patients' performance of a visual step-tracking task was also assessed by Baroni et al (7). They examined motor behavior of PD patients and control subjects under the following conditions: closed-loop, open-loop, and closed-loop with expected perturbation. The patients' performance was worse than the control subjects' under the two latter conditions but improved after L-DOPA treatment.

Against evidence pointing to a deficit of predic-

Address correspondence and reprint requests to Dr. M. Scarpa at Neurological Department, University of Modena, Via del Pozzo 71, 41100 Modena, Italy.

tive behavior, other investigators have found that PD patients can conditionally adopt an open-loop strategy (e.g., see refs. 8 and 9). However, for activities of daily living, patients more commonly adopt a responsive behavior. One interpretation of this shift to a responsive mode is the increased incidence of error and loss of accuracy that accompanies the use of a predictive behavior by PD patients (10). Alternatively, it has been proposed that PD subjects, though capable of adopting a predictive mode, are unable to do so for the initiation of a new movement (11) or for the switch from one motor program to another (12).

This brief literature review illustrates that there is some controversy as to the precise deficit of predictive motor behavior in PD. As Teasdale and Stelmach (13) recently pointed out, the unique characteristics of each experiment appear to influence the kind of results that are obtained. In the present study, the problem is tackled differently, that is, from a kinematic point of view. A double-step paradigm is applied to a prehension movement. In this paradigm, the nonperturbed, well-trained movement can be said to be performed with a predictive strategy, whereas the perturbed movement relies upon a responsive behavior.

The double-step paradigm has recently become popular in the study of sensorimotor coordination. It has been speculated that perturbing the movement at different times produces different effects (14). For the perturbed trials of the current study, a change of target location was time-locked with the onset of a reaching-grasping movement. This paradigm has already been used in a study of normal subjects by Paulignan et al. (15). In this latter study, the targets were graspable dowels located on a concentric array at a fixed distance from the hand. For these perturbed trials, subjects demonstrated a complex rearrangement of the wrist and finger trajectories, with a relatively small increase of movement duration (100 ms). The initial acceleration of the arm was terminated, and a secondary acceleration took place in order to direct the hand to the final target. The first acceleration peak occurred earlier in perturbed than in nonperturbed trials (on average, 105 instead of 130 ms after movement onset). This thus suggested that it takes ~100 ms for the visuomotor system to react to a change in target location. This duration approximately corresponds to the estimated minimum delay for visual feedback (16).

The aim of the present research is to study the effect of a spatial perturbation upon the kinematic characteristics of a prehension movement, both in normal subjects and PD patients. The nonperturbed condition is assumed to correspond to a predictive behavior—the task being automatically run. The perturbed condition is assumed to correspond to a responsive behavior—visual feedback being necessary to identify the new target location. The findings of abnormal kinematics under the nonperturbed condition, but normal kinematics in the perturbed condition, should thus point to a selective disruption of the open-loop, predictive motor control. Moreover, if a different pattern of acceleration curves is found between the two groups, it could underlie a different response to the change in target location. The ability of each subject group to superimpose or to adjust motor programs can thus be assessed.

METHODS

Subjects

Eight PD patients and eight age- and gender-matched controls served as subjects. All were right-handed. Of the eight patients, four were in stage II and four in stage III of Hoehn and Yahr's scale. None had motor complications or dyskinesias, and the prevalent feature was akinesia. A light tremor was present in two patients. All control subjects were free from neurological disorders. The characteristics of the PD patients, including the Unified Rating Scale for PD (URSPD) score and current therapy, together with those of the control subjects are shown in Table 1. All patients (except if de novo) were examined 1–2 h after drug therapy, that is, near the peak performance. All subjects gave their consent to participate in the two experimental sessions and were naive as to the purpose of the experiment.

Procedure

Each subject was comfortably harnessed within a chair. For the starting position, the ulnar border of hand rested on a marker that was placed 15 cm from the thorax in the mid-sagittal plane. The shoulder was internally rotated and slightly flexed, the elbow flexed, the forearm semipronated, and the index finger and thumb held opposed. The ulnar border of the forearm rested upon a microswitch; this enabled reckon time recording.

The targets were three translucent perspex spheres of 4 cm in diameter. Each contained three

TABLE 1. *Characteristics of the experimental subjects*

Control subjects			Parkinson patients				
No.	Sex	Age (years)	No.	Sex	Age (years)	URSPD-III	Therapy
1	M	49	1	M	68	31	Sinemet (500 mg), Jumex (10 mg)
2	M	66	2	M	82	14	Madopar (375 mg)
3	M	67	3	F	74	51	Madopar (500 mg), Parlodel (20 mg)
4	F	76	4	M	49	28	No therapy
5	M	83	5	M	74	44	Madopar (750 mg), Jumex (10 mg)
6	F	56	6	F	58	35	No therapy
7	F	62	7	M	60	48	Sinemet (250 mg), Parlodel (30 mg)
8	M	67	8	F	66	30	No therapy

light emitting diodes (LEDs). These spheres were placed in the mid-sagittal plane, 15, 27, and 40 cm (D1, D2, D3, respectively) from the starting marker (Fig. 1).

The subject was instructed to start the movement immediately upon illumination of one target. He/she was required to reach and grasp the target, and then take it to the starting position by moving at a speed that allowed an error-free performance. The sphere was illuminated until grasped.

First Session (Condition 1, C1)

For each target, the subject performed a block of 30 training trials followed by 15 experimental trials whereby movement kinematics were recorded. The subject was told which of the targets would be illuminated—no perturbations were introduced. These trials are referred to as blocked trials. To avoid practice effects, the order of blocks was counterbalanced across subjects.

Second Session (Condition 2, C2)

This session was performed on the following day. It consisted of 30 training trials and 60 recorded trials. The go-signal was always illumination of the target located at 15 cm. For 40 trials, illumination continued until the sphere was grasped (control trials). For 20 trials, a visual perturbation was introduced. These perturbed trials were random and interposed among the control trials. For this perturbation, the target at 15 cm was initially illuminated, but as soon as the subject began the reaching movement (release of the starting switch) illumination shifted either to the target at 27 cm (10 trials) or to that at 40 cm (10 trials). The subject was thus unexpectedly required to grasp another target. Illumination of the second sphere continued until it was grasped.

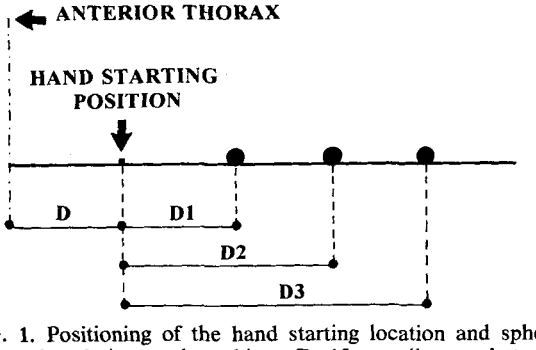

FIG. 1. Positioning of the hand starting location and spheric objects in relation to the subject. D: 15 cm, distance between subject's thorax and hand starting position; D1: 15 cm, distance between hand starting position and the first spheric object; D2: 27 cm, distance between hand starting position and the second spheric object; D3: 40 cm, distance between hand starting position and the third spheric object.

Movement Recording and Data Processing

All experimental trials were recorded and analyzed using the ELITE system (17). This system consisted of two TV cameras (sampling rate, 50 Hz) and a processor for the real time images of the marker shape. The markers were plastic spheres (diameter, 0.5 mm) that were covered with reflecting material (passive markers). The markers were securely taped to the following positions on the right forearm and hand: (a) wrist—dorsoradial aspect of the distal radial styloid process; (b) thumb—ulnar aspect of the thumb nail; (c) index finger—radial aspect of the index finger nail; and (d) dorsal aspect of the first carpometacarpal joint. Coordinates of the markers were reconstructed with an accuracy of 1/2,500 over the field of view and sent to a host computer (PDP 11/53). The computer performed the following operations: (a) three-dimensional reconstruction of the position of the markers; (b) data filtering (FIR filter); and (c) computation and graphic representation of the kinematic parameters.

Analysis of the transport (reach) component was based on the kinematics of the wrist marker: trajec-

tory, tangential velocity, and acceleration. Analysis of the manipulation (grasp) component was based on the kinematics of the three hand markers: temporal variation of the angle between the index finger and thumb.

The two components were considered to have commenced and to have finished in those frames in which the marker displacement was respectively greater and less than 0.4 mm (spatial error of the ELITE system). Data were analyzed only until grasp of the target; the section of the trial from grasp until the target was placed on the starting marker was not assessed.

As will be described in Results, the onset time of the transport component was computed separately from that of the manipulation component. Even though the two components began at different times, they both ended almost simultaneously (<10 ms difference; no consistent pattern according to trial type or subject group).

The time course of the transport movement was divided into three parts: (a) T1: the time from beginning of movement to the first acceleration peak [Paulignan et al. (15) found that this parameter was often modified with the introduction of a perturbation]; (b) T2: the time from the first peak of acceleration to the velocity peak [this parameter gives an indication of the duration of the modification]; (c) T3: the time from the first peak of velocity to the end of movement [this parameter allowed an assessment of the final part of the movement]. These three time intervals are shown in Fig. 2.

The time course of the manipulation movement was also divided into three parts: (a) M1: the time between onset of the transport and manipulation component [this parameter was identified during the experiment and was chosen for further assessment as it showed an unique pattern for PD patients]; (b) M2: the time from the beginning of the movement to the time of maximum aperture between index finger and thumb [this parameter, as found by Paulignan et al. (15) is also often modified in response to perturbation]; (c) M3: the time from onset to completion of the manipulation component. These three time intervals are shown in Fig. 2.

Each parameter (T1, T2, T3; M1, M2, M3) was submitted to an esavariate MANOVA, where Group (G1 = control subjects, G2 = PD patients) was entered as the between-subjects layout. Condition (C1 = session one, blocked trials, C2 = session two, perturbed + control trials) by Distance (D1 = 15 cm, D2 = 27 cm, D3 = 40 cm) were entered as the six dependent variables. The effects of Condition, Distance, and their interaction were tested by using the profile method (18). Multiple comparisons were performed using the Roy and Bose (19) method, where theta values are referred to the generalized beta distribution with 2 degrees of freedom (dof) for the effect and 13 dof for the error.

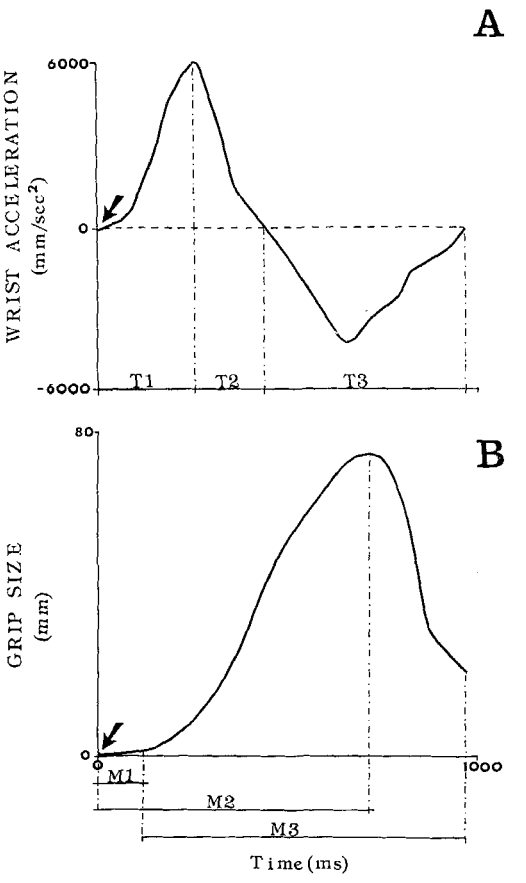

FIG. 2. Measured parameters of the transport (A) and manipulation (B) components. A: T1 = time to peak acceleration. T2 = time from peak acceleration to peak velocity. T3 = time from peak velocity to the end of the movement. B: M1 = onset of manipulation component with respect to onset of the transport component. M2 = time to maximum hand aperture. M3 = total duration of manipulation component. The arrow indicates the onset of the transport component.

RESULTS

Movement Trajectory

The trajectory of arm movement (wrist marker) for the nonperturbed trials was similar for both groups. In the sagittal plane, it showed a bell-shape (downward concavity). This single curve contrasted to the often doubled trajectory curves of the per-

turbed trials. For the control subjects, the wrist trajectory showed this m-shaped curve for 97.5% of trials, whereas for the PD patients it was evident for 83.7% of trials. There was no obvious difference in the characteristics of this doubled curve between the two groups.

A similar pattern was found for the manipulation component. The nonperturbed trials showed an increase of hand aperture to a maximum and then a decrease until the target was grasped. With perturbation, the opening/closing sequence was doubled (control subjects, 97.8% of trials; PD patients, 77.3%).

Transport Component

T1: Time to the First Acceleration Peak

The pattern of this parameter was not different for the PD patients and control subjects (Table 2). Thus, both groups showed significant sources of variability according to Condition ($p < 0.05$), and with the interaction between Condition and Distance ($p = 0.10$, one tailed.) [The one-tailed interpretation of CD comparisons was justified by the results of Paulignan et al.'s (18) study, which allowed the expectation of shorter times to the first acceleration peak in perturbed conditions.]

A comparison between C1 and C2 showed that, within Condition 1, T1 progressively increased with distance. This was not found in Condition 2, where peak acceleration was earlier for the perturbed trials (27 and 40 cm) than for the control trials (15 cm).

These results indicate that a spatial perturbation leads to an earlier tuning of the peak acceleration. This anticipation is not affected by PD.

T2: Time from the First Acceleration Peak to the First Velocity Peak

The results for this measurement were similar to those found for T1. The significant sources of variability were as follows: Group ($p < 0.05$), Condition ($p < 0.01$), the interaction Group by Condition ($p < 0.05$), Distance ($p = 0.10$, one-tailed), and the interaction Condition by Distance ($p < 0.05$). Overall T2, as for T1, was less for Condition 2 (122 ms) than for Condition 1 (145 ms). However, the interaction Group by Condition showed that this difference was only for the PD Group. Both groups showed differences of T2 when comparing C1 to C2 according to reaching distance. Again, as for T1, T2 was less for the perturbed trials to the 27 and 40 cm distances than for the blocked trials. No difference was found when comparing the 15-cm control trials of C2 to the 15-cm blocked trials of C1.

This indicated that peak velocity was earlier for perturbed than for nonperturbed trials. Once again,

TABLE 2. *Means of the data submitted to the esavariate MANOVA*

	Blocked trials			Control	Perturbed	
	C1D1	C1D2	C1D3	C2D1	C2D2	C2D3
Transport component						
G1						
T1	199 (38)	195 (50)	203 (49)	189 (58)	143 (30)	148 (45)
T2	104 (14)	107 (15)	133 (18)	105 (12)	104 (22)	107 (24)
T3	357 (93)	452 (112)	560 (182)	374 (110)	661 (111)	847 (129)
G2						
T1	219 (69)	253 (79)	241 (57)	225 (64)	214 (91)	197 (60)
T2	157 (48)	177 (26)	190 (35)	151 (42)	125 (22)	140 (51)
T3	479 (60)	715 (218)	778 (207)	452 (35)	740 (139)	953 (177)
Manipulation component						
G1						
M1	2 (5)	12 (6)	39 (8)	29 (5)	25 (3)	31 (8)
M2	452 (126)	563 (138)	679 (215)	457 (133)	429 (38)	426 (72)
M3	680 (113)	772 (145)	873 (194)	668 (129)	891 (102)	1068 (110)
G2						
M1	113 (18)	160 (9)	234 (15)	110 (11)	127 (21)	108 (12)
M2	615 (186)	841 (165)	899 (301)	571 (144)	555 (202)	583 (179)
M3	742 (145)	984 (301)	954 (228)	703 (39)	950 (187)	1198 (199)

[a] All data in msec. Values in brackets represent SD.

G1, control subjects; G2, Parkinson patients; C1, nonperturbed condition; C2, perturbed condition; C2D2, control trials; D1, 15 cm; D2, 27 cm; D3, 40 cm; T1, time to the first acceleration peak; T2, time between the first acceleration peak and the first velocity peak; T3, time between the first velocity peak and the end of movement; M1, delay of onset of the manipulation component with respect to the transport component; M2, time to the first maximum aperture of the hand; M3, total grasping time.

such an effect was common to both groups, though greater for PD patients.

Wrist velocity profiles are illustrated in Fig. 3. The lower two rows illustrate the doubling of the transport component for the distance perturbed trials.

The acceleration profiles of Fig. 4 confirm this doubling with perturbation and further illustrate the similar patterning of the transport component with perturbation for both subject groups.

T3: Time from the First Velocity Peak to the End of the Movement

T3 also showed a lengthening with perturbation. The significant sources of variability were as follows: Group ($p < 0.05$), Condition ($p < 0.05$), Distance ($p = 0.000$), and the interaction Condition by Distance ($p < 0.05$). As would be expected with the generalized slowing in PD, T3 for PD subjects was longer than for control subjects. In all other respects, the results were similar for both subject groups. Thus, T3 was longer in Condition 2 than in Condition 1, and, irrespective of Condition, it increased with Distance. The difference according to Condition was once again due to the difference between perturbed and nonperturbed trials, T3 being longer for the first ones.

Therefore, this time interval is longer with PD patients, in perturbed trials, and at longer distances.

Manipulation Component

M1: Onset of the Manipulation Component

Analysis of this measure showed very clear differences for the PD subjects (Table 2). The signifi-

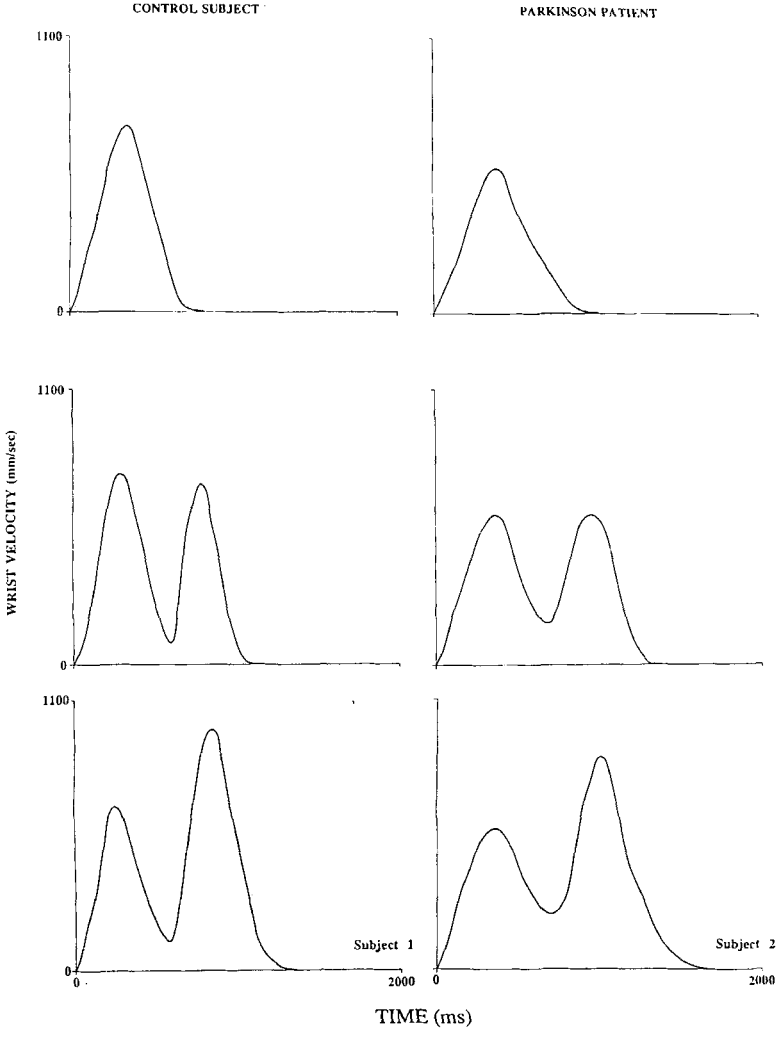

FIG. 3. Wrist velocity profiles (Condition 2) of a single trial for a control subject (left) and a PD patient (right). **Upper row:** Control trial to a sphere placed at 15 cm. **Middle row:** Distance perturbed trial from the sphere at 15 cm to another at 27 cm. **Lower row:** Distance perturbed trial from the sphere at 15 to another at 40 cm. Ordinate: velocity as measured from the wrist marker. Abscissa: trial sampling duration (2,000 ms).

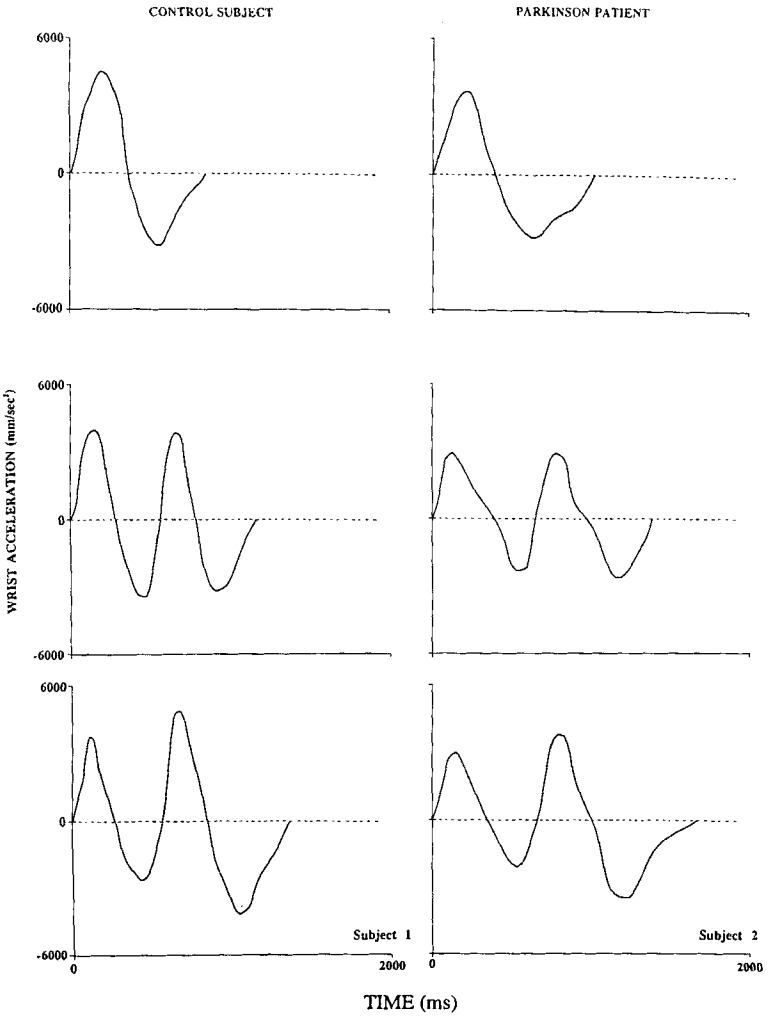

FIG. 4. Wrist acceleration profiles (Condition 2) of a single trial for a control subject (left) and a PD patient (right). **Upper row:** Control trial to a sphere placed at 15 cm. **Middle row:** Distance perturbed trial from the sphere at 15 cm to another at 27 cm. **Lower row:** Distance perturbed trial from the sphere at 15 cm to another at 40 cm. Ordinate: acceleration as measured from the wrist marker. Abscissa: trial sampling duration (2,000 ms).

cant sources of variability were as follows: Group ($p < 0.05$), the interaction Group by Condition ($p < 0.05$), and the interaction Condition by Distance ($p < 0.05$). Overall, the onset of the manipulation component with respect to that of the transport component was much later for the PD patients. Further differences between the two groups were also found for the pattern of this parameter with perturbation. To begin, M1 for the PD subjects was much less for Condition 2 than for Condition 1. Control subjects showed a markedly smaller difference between two conditions. Much of the difference between conditions for the PD group could be attributed to differences between perturbed and nonperturbed trials. This was particularly pronounced for the 40-cm reaching distance: M1 for the perturbed trials was clearly much less than that for the blocked trials ($p < 0.05$).

The delayed onset of the manipulation component was thus unique to the PD subjects. In addition, this delay was greater for nonperturbed than for perturbed trials.

M2: Time to the First Maximum Aperture

In contrast to M1, both groups showed a similar pattern for M2. The significant sources of variability were as follows: Condition ($p = 0.005$), Distance ($p < 0.05$), and the interaction Condition by Distance ($p < 0.001$). The Group effect did not prove significant. M2 was less for Condition 2 than for Condition 1. This difference was also attributable to differences between perturbed and nonperturbed trials ($p < 0.05$). There was little difference in M2 when comparing blocked to control trials.

Perturbation leads to an earlier maximum hand aperture, and such anticipation is not affected by PD. Thus, not only kinematic parameters of the

transport but also those of the manipulation component are brought forward with the unexpected introduction of a different target.

Figure 5 shows examples of the grip size profile with and without perturbation. This shows the late onset of the manipulation component for the nonperturbed trials—a finding unique to the PD subjects.

M3: Total Grasping Time

M3 also showed no difference according to Group. The significant sources of variability were as follows: Distance ($p = 0.000$), and the interaction Condition by Distance ($p < 0.005$). Across both groups, the total grasping time increased with reaching distance and was also greater for the perturbed than for the nonperturbed trials of both subject groups.

Figure 6 shows examples of the blocked trials of Condition 1 as performed by a subject from each group. This serves to illustrate that the patterning of both the transport and the manipulation components is similar for both the PD patient and the control subject. The only clear difference is the later onset of the manipulation component for the PD patient.

DISCUSSION

The aims of the present study were to (a) determine the modifications of a prehension movement in response to a spatial perturbation; (b) elucidate whether these modifications are different for PD pa-

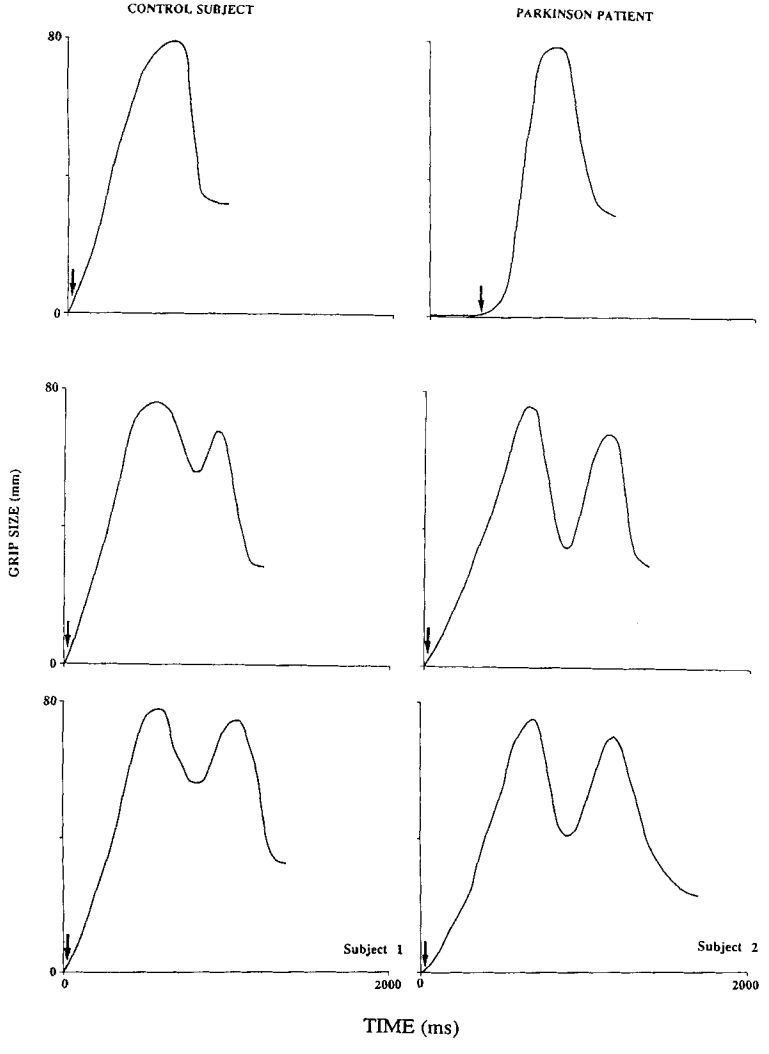

FIG. 5. Grip size profiles (Condition 2) of a single trial for a control subject (left) and a PD patient (right). **Upper row:** Control trial to a sphere placed at 15 cm. **Middle row:** Distance perturbed trial from the sphere at 15 cm to another at 27 cm. **Lower row:** Distance perturbed trial from the sphere at 15 cm to another at 40 cm. Ordinate: distance between the index finger and thumb markers. Abscissa: Trial sampling duration (2,000 ms). Note that for control trials the onset of grip aperture (vertical arrow) is later for the PD patient than for the control subject. The delay is not so obvious for the perturbed trials.

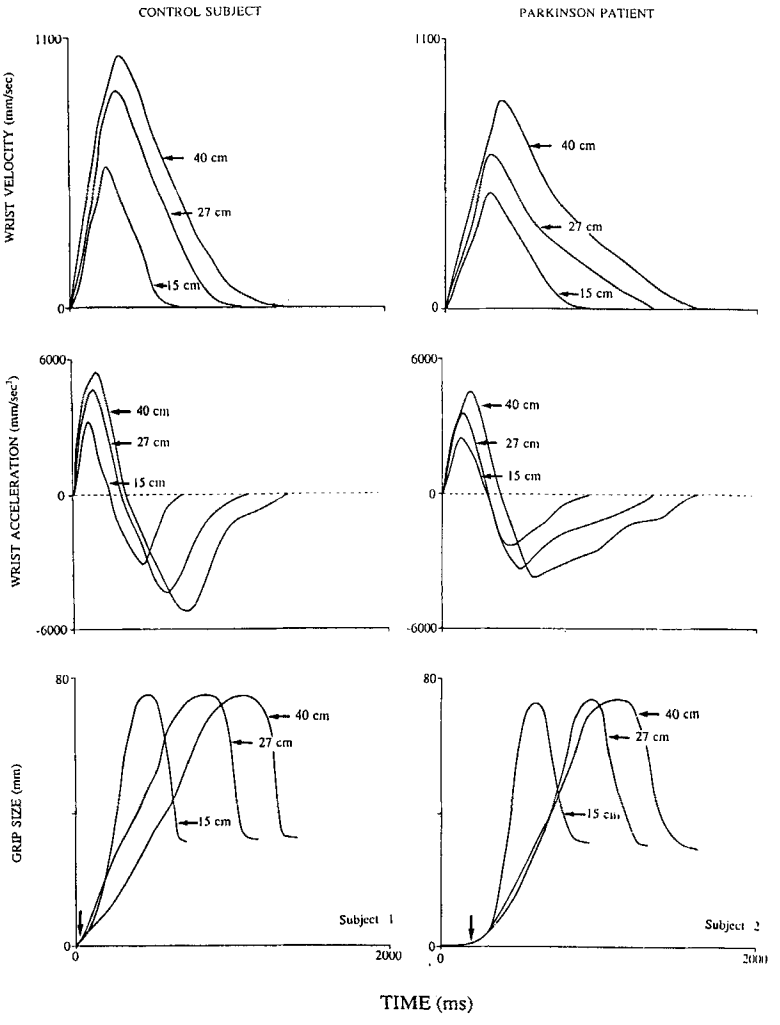

FIG. 6. A comparison of the prehension characteristics of a single trial as performed by a control subject (left) and a PD patient (right) when reaching to spheres placed at 15, 27, and 40 cm (Condition 1). Abscissa: sampling duration of the trial (2,000 ms). **Upper row:** Velocity profiles as measured from the wrist marker. **Middle row:** Acceleration profiles. **Lower row:** Grip size profiles (distance between index finger and thumb). Note that the onset of grip aperture (vertical arrow) is later for the PD patient than for the control subject.

tients and control subjects; and (c) collect empirical evidence, which further assists to clarify the role of the basal ganglia in motor behavior.

Transport Component

For both PD patients and control subjects, the (first) peak of arm acceleration and of velocity occur earlier in perturbed than in control trials: both parameters are anticipated as a result of spatial perturbation.

An alternative explanation for the difference of results between perturbed and nonperturbed movements could relate to different premovement strategies. For example, prior to blocked trials (C1) to the sphere at 27 cm, the subject begins with the intention of grasping this rather than the sphere at 15 cm—as is the case for the perturbed trials of C2. However, against this explanation are the findings from the comparison between control and perturbed trials of C2. In these cases, the subject had the intention of grasping the sphere placed at 15 cm, yet the kinematic parameters of the transport component were also earlier for the perturbed than for the nonperturbed movements.

The results of the present study concur with those of Paulignan et al.'s (15). They studied the effect of angular displacement of a target upon the kinematics of reaching to grasp movements. These authors also found that the initial acceleration peak for perturbed movements occurred ~100 msec after movement onset and was earlier than for nonperturbed movements. They interpreted this finding as an abortion of the first movement due to a rapid, on-going motor reorganization caused by the discrepancy with the pattern of reafferences expected. In the present study, the first detectable change for perturbed movements (i.e., the first acceleration peak) occurred later than in Paulignan et al.'s work

(15) (on average, 145 and 205 ms for control subjects and PD patients, respectively). Differences between the two studies could be attributed to differences of experimental technique. For example, the movements may have been performed at different speeds. In any case, both studies illustrate that changes or corrections to the movement in response to perturbation can be attributed to the effect of visual feedback upon motor output.

When presented with an unexpected visual stimulus, PD patients are clearly able to both call up and execute correct motor programs in order to reach towards and accurately grasp the new target. This finding is in agreement with several studies that have also found that PD does not affect the overall integrity of responsive movement patterns (3–5,7). There is some indication that PD patients begin the correction as early as control subjects but that they require more time to complete the change. The time to peak acceleration was no different across the two groups: (anticipation at distance 27 cm + anticipation at distance 40 cm)/2 was 53 ms for control subjects and 41 ms for PD patients. In contrast, the time between peak acceleration and peak velocity was different according to group. The average value of the anticipation of peak velocity with perturbation was 14 ms for control subjects but 51 ms for PD subjects. This latter group thus show a slowness in completing the adjustment of a motor program. Such a finding is not really surprising given the bradykinesia associated with PD.

Manipulation Component

In both control subjects and PD patients, the perturbation causes a lengthening of the total manipulation time and a less obvious shortening of the time to reach the maximum aperture of the hand angle. This latter finding shows that not only parameters of the transport component but also those of the manipulation component are accelerated by the onset of the new target. This is in agreement with Paulignan et al.'s (15) results. The anticipation of maximum hand aperture, as discussed for the anticipation of the acceleration peak, can thus also be attributed to on-line feedback corrections.

The results of parameter M1 need a more accurate inspection. They show that a peculiar feature of the kinematics of PD patients is the delay between onset of the manipulation component and onset of the transport component. This delay is greater for the nonperturbed condition. However, with perturbation, the delay is less evident and, in fact, is more similar to that of the control subjects.

The delayed pattern can be interpreted as a lack of coordination between the two components, and its correction an attempt at recoupling. Together with the dissociation of the double pattern between the transport and manipulation components of perturbed trials, the delayed onset adds evidence suggesting that the reaching-grasping movement is directed by two distinct motor programs. These programs are usually executed simultaneously but can be desynchronized either by a perturbation of stimulus size (20) or by a specific difficulty in simultaneously executing otherwise correct motor programs. It is well known that PD patients have a marked deficit in making simultaneous movements (21–27). For example, Benecke et al. (25–27) have reported that PD patients show difficulty with the simultaneous performance of two movements (flexion of the elbow and squeezing with the hand) by the same limb and that performance improves after L-Dopa administration. Therefore, if the reaching-grasping movement requires the activation of two motor programs, it can be concluded, in agreement with Benecke et al. (25–27), that PD subjects show a deficit in the execution of two distinct motor programs by the same hemisphere. The basal ganglia might thus play a role not only in movement execution but also in motor programming. Moreover, if "any movement can be fully characterized by how muscles are energized and the time order in which the energized muscles are activated" (28), not only the "energizing" function but also the "timing" function might be ascribed to the basal ganglia; i.e., they would both start the motor programs that specify the sequence of muscle activation, and select the correct muscles and the amplitude of their activation.

CONCLUSION

The main points of the present article can be summarized as follows. PD patients show no deficit in the preprogramming of prehension movements. In addition, they show normal anticipatory changes to the kinematic parameters of the first manipulation and transport movements in response to a spatial perturbation. The main difference between PD patients and control subjects is the delay of onset of manipulation with respect to the transport component. This delay is significantly reduced with the perturbation, indicating that PD patients, when us-

ing a responsive behavior, can recouple the two motor components. It appears, therefore, that PD patients can adopt a predictive behavior in that they can preprogram the movement once they know where the target will be. However, they show a deficit of movement organization whereby the main components are executed sequentially rather than simultaneously.

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