

## NOTE

### PERTURBATION OF THE GRASP COMPONENT OF A PREHENSION MOVEMENT IN A SUBJECT WITH HEMIPARKINSON'S DISEASE

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**Abstract**—The response to perturbation of the manipulation component during prehension was assessed for both hands of a hemiParkinson and of a Control subject. With perturbed trials, the hemiParkinson showed the same pattern as the Control: a reorganization of kinematic parameters and no increase of movement time. However, for both limbs of the hemiParkinson subject there was a transition phase from precision grip to whole hand prehension—this was more pronounced for the affected limb. The manipulation component did not show a delay of activation [2]. Thus the global dysfunction in the performance of sequential movement patterns was related to aspects of task predictability.

## INTRODUCTION

DURING execution of a goal-directed movement a perturbation can unexpectedly be applied. The corrections generated in response to such a disruption provide clues as to the central neural mechanisms of the movement's control [20]. How quickly, for example, are corrective measures generated? What adaptive responses are shown by the movement component which has been perturbed? Do the effects of perturbation extend to other movement components?

In the current study, a perturbation is applied to the reach to grasp action [4, 5, 7, 27, 28]. This movement consists of two parallel components: transport and manipulation [19, 20]. Many studies have elucidated the means by which these two components can be coordinated and synchronized for a functional grasp [4, 5, 15, 19, 20, 23]. Results from previous perturbation experiments [4, 5, 27, 28] demonstrate that arm transport and hand shaping show corrective responses which covary. This supports previous theories that the two components of prehension act in synergy. Perturbation of object location [28] should, in principle, disrupt only the transport component, however grip formation is also affected. Similarly perturbation of object size not only results in a reorganization of the manipulation component but adaptive responses in the transport component [4–6, 27].

The aim of this study is to determine whether reorganization of the transport and manipulation components of the affected limb of a hemiParkinson subject differs from that of the unaffected limb during a perturbed reach to grasp movement [4, 5].

## METHOD

### *Subjects*

The hemiParkinson and control subjects have been described in the preceding note [2]. In brief the hemiParkinson subject was a 71-year-old right-handed gentleman with right-sided resting tremor and rigidity. The control subject was matched for gender, handedness and age.

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### *Apparatus and procedure*

The starting position, placement of the IREDs and the recording system were the same as described in the previous note [2]. Within a dimly lit room, the subject was required to reach, grasp and then lift a target cylinder which was placed 35 cm anterior to the starting switch. A small cylinder (0.7 cm diameter; 10 cm high) stood vertically within the centre of a large cylinder (8 cm diameter; 6 cm high). Three light-emitting diodes (LEDs) were embedded within the table surface in a concentric row beneath these cylinders. Illumination of the small cylinder was with activation of the central LED. Illumination of the large cylinder was with activation of the two lateral LEDs. Subjects were instructed to begin the movement as soon as one cylinder became illuminated. This was shortly after a warning tone as described in the previous note [2]. Following a few practice trials recording began. The right and left hands were tested during different experimental sessions (100 trials each hand). Each trial started with illumination of either the small or the large cylinder. For 80/100 trials either the small or the large cylinder remained illuminated until completion of the movement: precision grip (PG) non-perturbed trials for the small cylinder; whole-hand prehension (WHP) non-perturbed trials for the large cylinder. For 20/100 trials which were randomly interspersed with the non-perturbed trials, movement onset (release of starting switch) triggered a shift of illumination. This was either from the small to the large cylinder (PG–WHP perturbed trials) or from the large to the small cylinder (WHP–PG perturbed trials).

### *Data processing*

Reaction time was computed from the time of initial cylinder illumination until release of the starting switch. Analysis of the transport component was based on the kinematics of the wrist marker. Analysis of the manipulation component was based on the kinematics of the digital markers. The two components were considered to begin and end in those frames in which the displacements of the IREDs were respectively larger and smaller than 0.3 mm (see Ref. [2]). Movement time was thus taken as the time shortly after release of the starting switch to the time at which the digits first contacted the cylinder. Data were filtered using a Butterworth dual pass filter (cut-off frequency 10 Hz).

Data obtained from the PG non-perturbed trials were compared to the PG–WHP perturbed trials. Data from the WHP non-perturbed trials were compared to the WHP–PG perturbed trials. For each subject, mean values of each dependent measure were calculated for each *Subject* (hemiParkinson, control), *Type of trial* (non-perturbed, perturbed) and *Hand* (right, left) combination. These data were entered into a factorial analysis of variance where *Subject* and *Hand* were between-subject factors and *Type of trial* was a within-subject factor. An alpha level of 0.05 was adopted for all tests of significance. *Post-hoc* contrasts were conducted with the Newman–Keuls testing procedure. For all reported results the level of significance for each of these contrasts ranged from  $P < 0.05$  to  $P < 0.01$ . The absolute temporal value of each kinematic parameter was expressed as a percentage of movement time.

## RESULTS AND DISCUSSION

Using either limb the hemiParkinson subject was able to rapidly adjust movement kinematics in response to perturbation of the grasp pattern. Significant results are shown in Tables 1 and 2. For neither limb was movement time greater for perturbed than for non-perturbed trials. Thus despite the requirement for an unexpected change from a precision grip to a whole-hand prehension or for the converse adaptation, the kinematic parameters were rearranged within the originally prescribed movement duration. The same result applied for both limbs of the Control subject. Movement duration thus appeared to be a reference parameter for organization of the movement.

The means of achieving such invariance was with the earlier temporal setting of kinematic landmarks of the transport component for perturbed than for non-perturbed trials. This finding was more evident for the unaffected arm of the hemiParkinson subject: the peaks of wrist acceleration and deceleration were earlier for perturbed than for non-perturbed trials (Fig. 1A). The affected limb of the hemiParkinson subject showed only a trend for peak deceleration to be earlier for perturbed than for non-perturbed trials. Thus for both limbs early modifications shortly after movement onset ensured that the following reorganization resulted in no prolongation of movement duration.

For both subjects, the amplitudes of peak acceleration and peak deceleration were lower for perturbed than for non-perturbed trials (Fig. 1A). The hemi-Parkinson subject was thus able to modify movement parameterization in a manner which was similar to that shown by the Control subject (see also Ref. [22]). This contrasts to a study which found that Parkinson subjects were unable to adjust the velocity of an elbow movement [17]. The difference in results could be attributed to the fact that the movement in the current study does not have the ballistic characteristic of that in the latter study.

Earlier temporal settings for the perturbed trials were also found for the manipulation component. With perturbation from whole-hand prehension to precision grip, the grip aperture showed an initial opening for the large cylinder and then a smooth closure for the small cylinder. The maximum opening was earlier and smaller than that for the non-perturbed trials. This indicated that the manipulation component also showed a reorganization which presumably acted to ensure that the movement was performed without prolongation of time. For the opposite

Table 1. Comparison of the perturbed (Pert) and non-perturbed (Non-Pert) trials for each limb of each subject. The mean and S.D. (parentheses) for each parameter is indicated. %—the value is expressed as a percentage of movement time

	HemiParkinson subject				Control subject				Interaction: Subject by hand by Type of Trial
	Affected		Unaffected		Non-dominant		Dominant		
	Pert	Non-Pert	Pert	Non-Pert	Pert	Non-Pert	Pert	Non-Pert	
Reaction time (msec)	458 (51)	463 (46)	418 (32)	421 (38)	342 (38)	335 (32)	329 (31)	331 (34)	$F(1, 36) = 88.05$ $P < 0.0001$
Movement time (msec)	1148 (111)	1129 (92)	1103 (103)	1089 (107)	988 (91)	1012 (100)	1002 (101)	1021 (100)	$F(1, 36) = 10.59$ $P < 0.001$
Time to peak acceleration (%)	30 (2)	31 (3)	29 (3)	33 (5)	29 (2)	31 (3)	28 (2)	33 (4)	$F(1, 36) = 19.02$ $P < 0.0001$
Time to peak deceleration (%)	70 (7)	73 (7)	64 (6)	75 (8)	59 (4)	61 (7)	58 (6)	63 (6)	$F(1, 36) = 4.23$ $P < 0.05$
Amplitude of peak acceleration (mm/sec <sup>2</sup> )	1522 (153)	1684 (178)	1732 (175)	2060 (200)	1960 (198)	2630 (268)	2230 (225)	3028 (300)	$F(1, 36) = 62.07$ $P < 0.0001$
Amplitude of peak deceleration (mm/sec <sup>2</sup> )	1320 (125)	1480 (140)	1464 (145)	1677 (170)	1720 (170)	2240 (222)	2120 (215)	2840 (300)	$F(1, 36) = 74.62$ $P < 0.001$
Onset of manipulation component (%)	2 (0.1)	3 (0.1)	2 (0.1)	3 (0.1)	2 (0.1)	2 (0.2)	3 (0.2)	2 (0.2)	

Table 2. Comparison of the perturbed (Pert) and non-perturbed (Non-Pert) trials for each limb of each subject. The mean and S.D. (parentheses) for each parameter is indicated. %—the value is expressed as a percentage of movement time

	HemiParkinson subject				Control subject				Interaction; Subject by hand by Type of Trial
	Affected		Unaffected		Non-dominant		Dominant		
	Pert	Non-Pert	Pert	Non-Pert	Pert	Non-Pert	Pert	Non-Pert	
(A) Perturbation from whole-hand prehension to precision grip									
Time to maximum grip aperture (%)	51 (5)	73 (8)	55 (4)	72 (8)	53 (5)	68 (7)	58 (5)	70 (7)	$F(1, 36)=6.67$ $P<0.001$
Amplitude of maximum grip aperture (mm)	74 (2)	113 (14)	50 (5)	98 (3)	74 (8)	96 (2)	68 (7)	107 (3)	$F(1, 36)=5.51$ $P<0.05$
(B) Perturbation from precision grip to whole hand prehension									
Time to first inflection (%)	22 (2)	—	23 (3)	—	—	—	—	—	
Onset of second aperture (%)	38 (4)	—	36 (5)	—	—	—	—	—	
Time to maximum grip aperture (%)	58 (6)	47 (5)	57 (5)	50 (5)	55 (5)	43 (4)	59 (6)	49 (5)	$F(1, 36)=11.14$ $P<0.001$
Amplitude of maximum grip aperture (mm)	104 (9)	40 (4)	103 (8)	38 (3)	108 (9)	40 (4)	105 (8)	38 (3)	$F(1, 36)=46.44$ $P<0.0001$

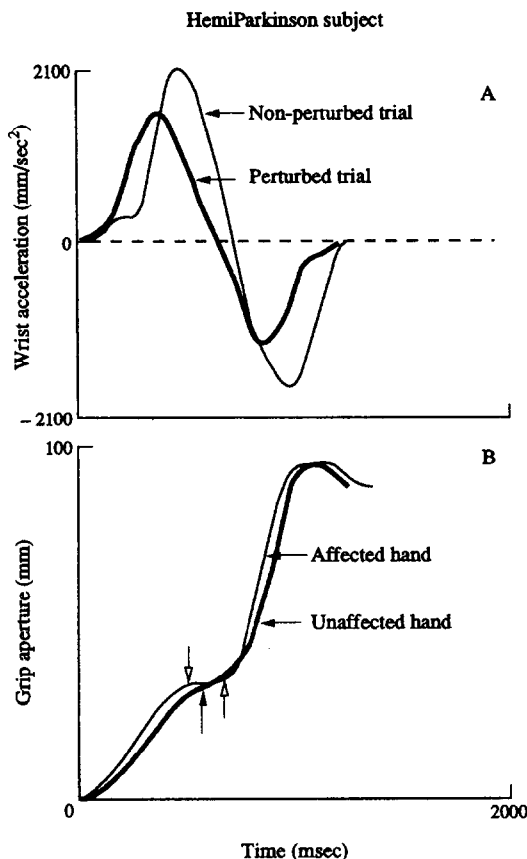


Fig. 1. (A) Acceleration profile for a perturbed (dark line) and a non-perturbed (light line) trial performed with the unaffected limb of the hemiparkinson subject. This demonstrates the earlier timing and smaller amplitudes of the peaks of acceleration and deceleration for the perturbed trials. Note however that movement time is similar for both trials. (B) Grip aperture profile for a single trial performed with the affected (light line) and the unaffected (dark line) hand of the hemiparkinson subject. Open arrows indicate the points of inflection (left) and onset of the second grip aperture (right) for the affected hand. Note the plateau of grip aperture between these points. The closed vertical arrow indicates the point of inflection for the unaffected hand. The onset of the second grip opening could not be determined in this case.

perturbation, differences between the Control and hemiparkinson subject became apparent when looking at the transition period from precision grip to whole-hand prehension. In particular, for the affected hand of the hemiparkinson subject there was a plateau of grip aperture between the point of maximum initial opening for the small cylinder and the point at which the hand began to further open for the large cylinder. This is illustrated in Fig. 1B. The use of a break detection algorithm enabled the determination of the times of these two points (first inflection and onset of second aperture, respectively) although a 1–2% (of movement time) error margin is estimated. Despite this margin, it was clear that the onset of the second aperture was approximately 16% of movement time after the first inflection. The unaffected hand also showed evidence of a transition period between the two grasps however a plateau was not present. Rather, the initial inflection was followed by a slowly increasing opening of aperture before a second inflection which was assumed to represent the onset of the second opening. The times between these two inflections was, on average, 13% of movement time, although in some cases it was not possible to determine the timing of either the first or second inflection (e.g. Fig. 1). In any case an obvious transition from one to the other grasp was not apparent for either hand of the Control subject where only a slight inflection signalled the second opening. The timing of this inflection could not be determined.

The prolonged transition period for the hemiparkinson subject may reflect a dysfunction in the performance of

sequential movement patterns [1, 18, 30]. Precision grip is thought to be subserved by neural channels that differ from those for whole-hand prehension [25, 29]. Thus the longer shift from one to the other grasp could represent difficulties in (a) deactivating the precision-grip channels and (b) activating the whole-hand prehension channels. Evidence for the first supposition comes from the plateau of grip aperture, as if the first grip pattern perseverates. This could suggest that the basal ganglia not only function to set appropriate levels of cortical excitability [9] but also to cancel already activated channels. Further support for such a deactivation role comes from neurophysiological data which demonstrate that a proportion of neurons in the anterior globus pallidus show phasic discharge in relation to the end of a wrist movement [3] (see also Ref. [24]). This mechanism may operate both to terminate sustained activity in cortical regions and to prepare these regions for the upcoming movement.

The transition period could also be indicative of problems with the shifting of attentional focus [8]. For example, the hemiParkinson subject may be slow at enlarging the focus of attention from the small to the large cylinder. If such attentional mechanisms are important for the guidance and generation of limb movements [30] this could explain the prolonged switching period from PG to WHP. In addition, if the deficit of attentional focus was more for shifts from small to large than from large to small, this could explain why a prolonged transition was not evident for the perturbation from WHP to PG.

An alternative explanation for the prolonged transition from PG to WHP but the absence of an obvious transition from WHP to PG, may relate to physical factors. Biomechanically there may be more advantage for closure than for opening [10]. For a task focused upon a grasping action, the biomechanical setting for the flexors (e.g. with wrist extension) would be more favoured. With the requirement for a sudden hand opening, and thus digit extension, the hemiParkinson subject may show more obvious dysfunction. The argument against this is the finding of a prolonged transition even with the unaffected limb where rigidity cannot be implicated.

A further difference between the hemiParkinson and the Control subject was found when comparing the onset time of the manipulation component for the trials of the current experiment to those of the previous note [2]. For both the perturbed and non-perturbed trials of the current note, the manipulation component began very shortly after the transport component (generally 2–3% of the transport movement time; see Table 1). For the hemiParkinson subject in contrast, the manipulation component of the blocked trials in the previous note began at around 6% of transport movement time [2]. This difference between the blocked and the perturbed and non-perturbed trials can be attributed to differences of trial characteristics prior to cylinder illumination. For the blocked trials, the subject was aware of all task requirements prior to cylinder illumination. For the perturbed/non-perturbed trials the subject did not know which cylinder would be illuminated nor whether a perturbation would be applied. Under these latter "uncertain" conditions, the hemiParkinson showed no dysfunction of the ability to activate the manipulation component. This finding is in agreement with previous studies of Parkinson subjects which have described similar differences between experimental tasks which cannot be predicted (e.g. perturbed/non-perturbed trials) and those which can be largely predicted (e.g. blocked trials) [11–14]. The rapid onset of the manipulation component for perturbed and non-perturbed trials suggests that basal ganglia motor circuits have been bypassed. It can be postulated that an alternative system is utilized to trigger and/or set the cortical responsiveness under conditions of unanticipated movement. For example, the lateral system involving the cerebellum and arcuate premotor area could be proposed (see, Ref. [16] for a review). However, this is thought to be a feedback based system. The onset of the manipulation component for perturbed trials was less than 40 msec after that of the transport component. This brief period excludes the possibility that the trigger for activation of manipulation resulted from afferent feedback of the reaching movement. Alternative systems could involve cortico-cortical circuits which influence the cortical regions directly (e.g. anterior cingulate cortex [25]).

In conclusion, the hemiParkinson shows similar mechanisms to the Control subject when confronted with the unexpected requirement for a change of grasp pattern. This perturbed movement is performed within the same duration as a non-perturbed movement and this duration equivalence is primarily achieved by rearrangement of the temporal settings of the kinematic landmarks of each component. The transitional phase from precision grip to whole-hand prehension is prolonged and although more evident for the affected side is also noted for the unaffected limb. This supports the idea that Parkinson's disease subjects have a global deficit in the ability to perform sequential movements. The finding of a normal activation time for the manipulation component with perturbed/non-perturbed trials may indicate that the deficit with activation of sequential movements is related to the level of task predictability.

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