

NOTE

A KINEMATIC STUDY OF THE REACH TO GRASP MOVEMENT IN A SUBJECT WITH HEMIPARKINSON'S DISEASE

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(Received 30 July 1992; accepted 2 February 1993)

Abstract—The kinematic organization of a reach to grasp movement in a left hemiParkinson subject is compared to that of a control subject. Subjects used the right and left limbs to reach 15, 27 or 40 cm for the grasp of cylinders of 0.7 or 8 cm diameter. In general, the kinematics of the affected limb of the hemiParkinson subject differed from that of the unaffected limb. However, for both arms the hemiParkinson subject showed a delay in the onset of the manipulation component. The subtle dysfunction in the activation of near-simultaneous or sequential movements is thus bilateral, despite unilateral clinical symptomatology.

INTRODUCTION

DURING the early stages of Parkinson's disease the clinical signs and symptoms are often more evident unilaterally with eventual progression to bilateral involvement [19]. However, in a limited number of cases the hemiParkinsonism remains [26, 33].

Few studies have quantified the differences in motor impairment between the two limbs of a hemiParkinson subject. In an early experiment, WILSON [36] demonstrated that when a hemiParkinson subject voluntarily contracted the quadriceps, the affected side showed a delay in initiation and a slow and irregular performance when compared to the unaffected side. Studies which have assessed reaction time have shown that for the affected side this time is greater but not qualitatively different from that of the unaffected side [30, 34, 37]. WING *et al.* [37] consistently found that the affected limb was slower than the unaffected limb in such tasks as writing and aimed tapping.

The present study assesses the kinematic organization of the reach to grasp movement of a single hemiParkinson subject. It aims to give a detailed comparison of the performance of the affected side to that of the asymptomatic contralateral upper limb. Such a kinematic analysis is believed to reveal deficits in movement performance which may not be apparent with clinical testing [20, 22].

METHOD

Subjects

The hemiParkinson subject was a 71-year-old right-handed gentleman who had been diagnosed as having Parkinson's disease 8 years before the study. He initially developed a right hand and leg tremor. Four years later carbidopa/levodopa (Sinemet) was commenced and led to an improvement of clinical signs. A year later, a head CT was unremarkable. A further year later selegiline (Eldepryl) was begun but no change in symptoms was reported. At the time of the current study the subject was at Stage I [19] and complained of intermittent right arm and leg resting tremor, slight micrographia and occasional nighttime drooling. Medication included Sinemet 25/100 three times

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daily, Eldepryl 5 mg twice daily and Vitamin E 1500 units daily. Physical examination revealed an intermittent, low amplitude, 5 Hz resting tremor of the right arm and leg. The right arm and neck showed some cogwheel rigidity. The agility of the right fingers and foot was diminished. Gait was normal except for a moderate decrease in right armswing. No left-sided signs or symptoms were present.

The control subject was a 70-year-old right-handed gentleman who was free from neurological and skeletomotor abnormalities. Both subjects volunteered to participate in the study and were naive as to the experimental design or purpose.

Apparatus and procedure

The subject was seated in a height adjustable chair so that the thorax pressed gently against the edge of the table. The starting position of the reaching arm was as follows: shoulder flexion ($5\text{--}10^\circ$) and internal rotation ($30\text{--}45^\circ$), elbow flexion (90°), forearm semipronation, slight wrist extension and opposition between the thumb and index finger. The ulnar border of the hand rested upon a midline switch positioned 15 cm anterior to the thorax.

The target to be grasped was a 10 cm high, perspex, translucent cylinder of either small (0.7 cm) or large diameter (8 cm) which was vertically positioned in the midline 15, 27 or 40 cm from the starting switch. Computer activation of light emitting diodes beneath the cylinder (within table surface) led to its illumination. Infrared emitting diodes (IREDS) were attached to the forearm and hand of the reaching limb: the wrist IRED to the dorso-radial aspect of the radial styloid process and the digital IREDs to the radial side of the thumb nail and to the ulnar side of the index finger nail.

A tone served as the warning signal before each trial. The interval between the tone and illumination of the cylinder was randomized in order to avoid expectancy effects (500–2000 msec). Upon illumination of the cylinder, the subject was required to reach for, grasp the cylinder and then lift it slightly above the working surface. Movement speed was not stipulated except to ask subjects to perform at a speed they would use normally. The cylinder was illuminated until grasped. For each subject and for each hand, the trials tested were as follows: small cylinder at each distance and large cylinder at each distance. For each cylinder/distance/hand combination, the subjects performed 10 practice trials and then a block of 10 recorded trials. Both subjects used a precision grip, characterized by opposition between the index finger and thumb [28], to grasp the small cylinder and whole hand prehension, characterized by flexion of all the fingers around the object [31], to grasp the large cylinder (see Refs [7] and [8]). The order of blocks was the same for both subjects. To avoid fatigue and lack of concentration the subject rested for half an hour between the recording of each hand. Both subjects were tested at the same time of day with the assessment of the hemiParkinson subject beginning 1 hr after the morning medication. The hemiParkinson subject showed no change of clinical symptoms after the rest period and showed no resting tremor at the time of experimentation.

Movement recording and data processing

Recording and analysis was with the OPTOTRAK 3D system. This consisted of three infrared cameras each with charge-coupled sensor devices which measured the x (medio-lateral distance) and y (antero-posterior distance) coordinates of the IREDs. The three-dimensional coordinates were computed by using this row two-dimensional data from each camera. 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: trajectory, velocity, and acceleration profiles. Analysis of the manipulation component was based on the kinematics of the digital markers: temporal variation of the distance between thumb and index finger. The spatial precision of the OPTOTRAK system was 0.3 mm. Accuracy in different regions of the experimental work space was determined [17]. Each component was considered to begin and end in those frames in which the displacements of the IREDs were consistently larger and smaller, respectively, than 0.3 mm. 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 dowel (the lifting part of each trial was not assessed). Data were filtered using a Butterworth dual pass filter (cut-off frequency: 10 Hz).

For each subject, mean values of each dependent measure were calculated for each *Subject* (hemiParkinson, control), *Distance* (15, 27, 40 cm), *Type of grasp* (precision grip, whole hand prehension) and *Hand* (right, left) combination. These data were entered into a factorial analysis of variance where *Subject* and *Hand* were between-subject factors and *Distance* and *Type of grasp* were within-subject factors. An alpha level of 0.05 was adopted for all tests of significance. *Post-hoc* contrasts were performed 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 temporal kinematic parameters were expressed as a percentage of movement time. This allowed a comparison between the two hands and across the two subjects and also bypassed the overall slowness of movement in Parkinsonism which will not be discussed in this study.

RESULTS AND DISCUSSION

Significant results are shown in Tables 1 and 2. The kinematics profiles of a typical trial from each subject are illustrated in Fig. 1.

Table 1. Results obtained when comparing across hands for each subject (affected vs unaffected for the hemiParkinson subject and non-dominant vs dominant for the Control subject). Each value gives the mean and S.D. (parentheses) of 10 trials. Data has been collapsed according to cylinder size and distance. % indicates that the value is expressed as a percentage of movement time

	HemiParkinson subject		Control subject		Interaction: Subject by Hand
	Affected	Unaffected	Non-dominant	Dominant	
Reaction time (msec)	451 (104)	373 (48)	361 (43)	350 (43)	$[F(1, 22) = 10.05, P < 0.001]$
Movement time (msec)	1323 (123)	1245 (117)	1073 (70)	1052 (78)	$[F(1, 22) = 5.89, P < 0.05]$
Onset of Manipulation Component (%)	6 (0.4)	6 (0.3)	2 (0.1)	2 (0.5)	$[F(1, 22) = 3.50, P < 0.05]$
Time to maximum peak aperture (%)	60 (6)	63 (8)	58 (6)	67 (8)	$[F(1, 22) = 24.41, P < 0.0001]$
Amplitude of peak acceleration (mm/sec ²)	2020 (399)	2291 (396)	3148 (769)	3376 (814)	$F(1, 22) = 15.85, P < 0.001]$
Amplitude of peak velocity (m/sec)	499 (48)	529 (91)	678 (76)	728 (62)	$[F(1, 22) = 50.36, P < 0.0001]$
Amplitude of peak deceleration (mm/sec ²)	1507 (191)	1563 (157)	2528 (299)	2766 (283)	$[F(1, 22) = 13.53, P < 0.001]$

One characteristic of the hemiParkinson subject would appear to reflect abnormal functioning of the basal ganglia. The onset of the manipulation component was considerably later (6% of movement time) than the onset of the transport component (Fig. 1 and Table 1; see also Ref. [39]). This contrasted to the results found for the control subject where onset of the manipulation component was at the same time or slightly later than that of the transport component. The increased delay of onset for the manipulation component of the hemiParkinson subject was not confined to the affected side but was also observed for the unaffected limb. The dysfunction is thus more global than is shown by the unilateral clinical signs. These findings are in accordance with evidence that the pathology tends to be bilateral even when it is markedly asymmetric [26]. They are also compatible with the delayed onset of the manipulation component which has been reported for Parkinson subjects with bilateral clinical signs [9, 11, 39].

The later onset of the manipulation component supports previous suggestions of an abnormality in the ability to perform near-simultaneous or sequential movements [4, 5, 18, 32, 35]. For example, BENECKE *et al.* [4, 5] found that Parkinson subjects showed greater processing times for the simultaneous or sequential performance of such tasks as elbow flexion and finger/thumb opposition. The transport and manipulation components of the task in the current study are believed to be subserved by different neural channels [6, 27, 31]. In the non-Parkinson subject these components are activated almost simultaneously: on average the onset of manipulation is 0–50 msec after that of transport [9, 11]. The delay of the hemiParkinson subject may thus be interpreted as a dysfunction of near-simultaneous or sequential component activation.

The delayed onset of manipulation may naturally result from a larger movement duration. One method of testing this would be to ask both subjects to perform the movement within a set time or to ask the control subject to move more slowly. However, the imposition of a time limit could force unnatural movements and thus the expression of unusual kinematic results. A relationship between the delayed onset and movement duration should be confirmed by regression analysis. However, no correlation was found between these two parameters. It is also of note that the delay for the unaffected limb, where movement duration was less, was the same percentage of movement duration as that for the affected limb.

An alternative explanation for this delay could be the effects of mechanical constraints imposed by the classical symptom of Parkinsonian rigidity. Muscle stiffness may result in an enhanced resistance to hand opening which could feasibly lead to a delay of the manipulation component activation. Two findings argue against this view. Firstly the unaffected hand showed the delay despite having no clinical signs of rigidity. Secondly, the relative timing of the peak grip aperture was consistently between 58 and 68% of movement time for both the hemiParkinson and control subjects suggesting that the movement, once activated, showed no abnormal coordination of the manipulation component with the transport component [21].

It is thus proposed that the delay of manipulation component activation reflects an impairment of central nervous system processing. The motor circuit consists of multiple cortico-striato-nigro-thalamocortical circuits arranged in a parallel and topographical manner [2, 3, 23, 24, 29]. Given this topography it is hypothesized that motor circuit

Table 2. Effects found according to grasp type (A) and distance of the cylinder (B). Each value gives the mean and S.D. (parentheses) of 10 trials. For (A) data has been collapsed according to distance. For (B) data has been collapsed according to grasp type. % indicates that the value is expressed as a percentage of movement time. WHP refers to whole hand prehension. PG refers to precision grip

	HemiParkinson subject				Control subject		Interaction
	Affected hand		Unaffected hand		Dominant hand		
(A) Effects according to type of grasp	WHP	PG	WHP	PG	WHP	PG	Subject by Hand by Grasp
Time to maximum grip aperture (%)	63 (5)	64 (6)	60 (6)	57 (3)	57 (4)	57 (5)	$F(1, 22) = 18.23, P < 0.001$
Amplitude of maximum grip aperture (mm)	101 (3)	47 (5)	104 (7)	48 (4)	102 (4)	39 (4)	$F(1, 22) = 10.80, P < 0.01$
(B) Effects according to distance	27 cm	40 cm	27 cm	40 cm	27 cm	40 cm	Subject by Hand by Distance
Time to peak deceleration (%)	52 (7)	53 (6)	50 (6)	60 (14)	57 (5)	58 (7)	$F(1, 22) = 6.44, P < 0.01$
					27 cm	40 cm	
					56 (4)	61 (5)	

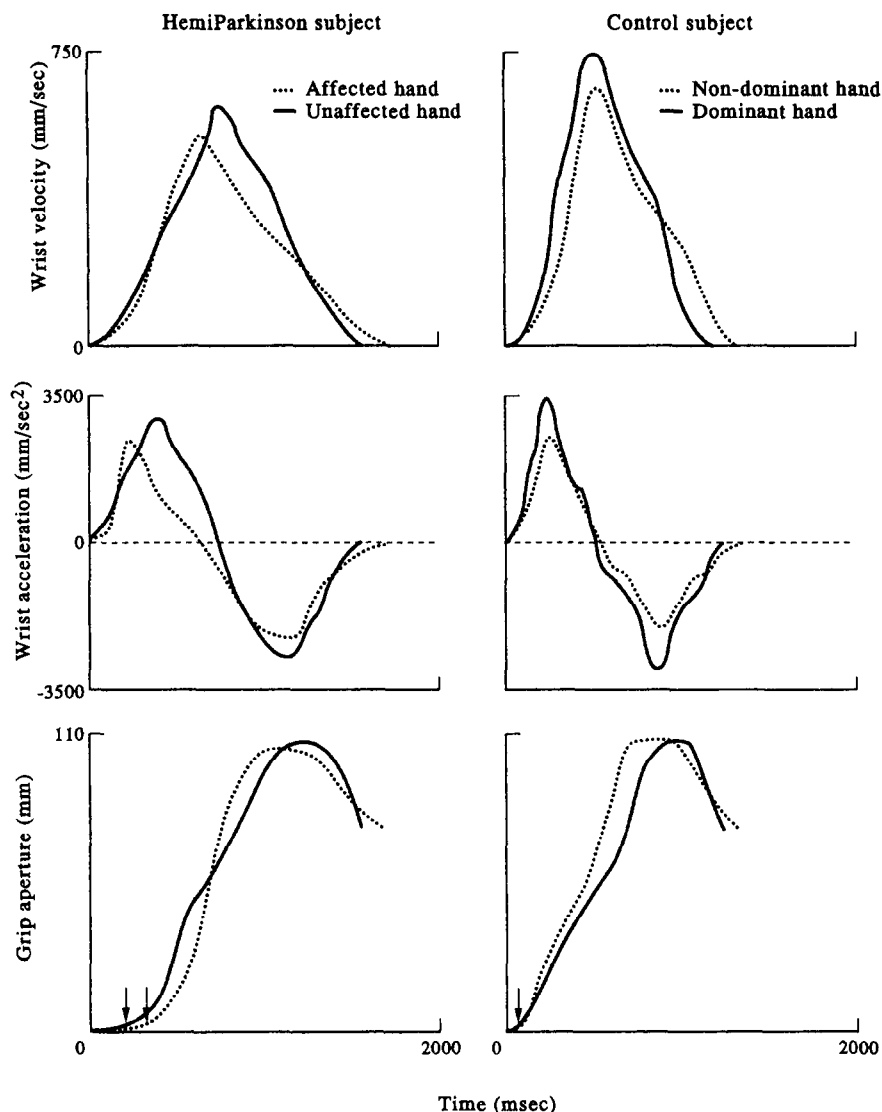


Fig. 1. Kinematic profiles obtained from a single reach to grasp trial (large cylinder, 40 cm distance) by the hemiParkinson subject (left) and the control subject (right). Above: Wrist Velocity. Middle: Wrist Acceleration. Below: Grip Aperture. The vertical arrows indicate the onset of the manipulation component. Time 0 refers to the onset of the transport component.

channels activated for transport are distinct from but run in parallel with those activated for manipulation. CHEVALIER and DENIAU [12] suggest that activation of the striatum promotes inhibition of inhibitory neurones which project to the thalamus. By confining this effect to a channel of neurones, such as those responsible for a movement component, they propose that a pattern of "readiness" is set in premotor networks. These cortical centres can then be "further actuated for the execution of movement" (p. 285). With dopamine depletion the striatal influence is lost, the result being an increased inhibition of the thalamo-cortical pathway [13] and thus less cortical preactivation. The idea of a lack of responsiveness could be used to explain why the manipulation component shows a delay of activation. With inadequate cortical preparation, rapid activation of a complex movement component becomes more difficult.

Apart from the bilateral finding of a delayed manipulation component onset, other measures reflected the unilateral symptomatology. Both reaction time and movement time were longer for the affected than for the unaffected limb of the hemiParkinson subject. The impaired limb was thus slower in the initiation and performance of movements [34]. It is also worth noting that reaction time for the unaffected limb was no longer than that for either limb of the control subject. This concurs with the findings of YOKOCHI *et al.* [38] who found that for subjects with right motor signs, reaction time was longer for the affected than for the unaffected limb whereas for subjects with left signs, reaction time was increased for both limbs.

With collapse of the data according to cylinder size and distance, both the manipulation and the transport components showed different kinematic profiles for the affected limb. For the manipulation component, the peak of grip aperture of the affected hand of the hemiParkinson subject was earlier than that of the unaffected hand. For the transport component, the amplitudes of peak wrist velocity and of peak wrist acceleration and deceleration were lower for the affected than for the unaffected hand.

The difference of kinematic profiles between the affected and non-affected limb of the hemiParkinson subject continued for the comparison of different reaching distances. Peak wrist deceleration of the affected hand was no different across the three distances. This was in contrast to the result found for the unaffected hand of the hemiParkinson subject whereby peak wrist deceleration was earlier for shorter distances.

A comparison of the kinematic organization according to the type of grasp adopted showed that the amplitude of grip aperture, as expected, was greater for whole hand prehension than for precision grip. In addition, this peak of aperture was earlier for the latter grasp but only for the unaffected hand of the hemiParkinson subject. The affected side did not show this anticipation of grip aperture for precision grip.

The hemiParkinson subject reported that although initially right-handed, he now used the left upper limb for most activities of daily living. The kinematic results reflected this shift in hand use. The limb which was predominantly used shows a kinematic organization which differs from the contralateral affected limb (see also Ref. [16]). For the preferred limb, the earlier settings of peak deceleration for shorter reaching distances and of peak grip aperture for precision grip reflect the ability of central neural structures to organize the kinematic arrangement of each component (transport and manipulation) according to the requirements of the end-task [7, 9, 15, 25]. In contrast, the affected limb showed little kinematic change according to target size or to target distance. Overall, however, this limb shows a pattern which could be interpreted as more cautious or tentative. This is firstly evidenced by the slower speed of movement for this limb. Secondly, peak grip aperture was earlier for this than for the contralateral limb and thus a greater amount of time was allocated for final closure of the hand about the cylinder. In addition, the earlier peak of acceleration for the affected as opposed to the unaffected limb of the hemiParkinson subject, indicated that a longer deceleration time was allocated for final approach. It is suggested that the central processing for the reach to grasp movement of this hemiParkinson subject had been modified to promote an optimal level of functioning. This adaptation resulted in a switch of preferred hand use to the unaffected side (see also Ref. [10]).

In conclusion, the use of kinematic analysis reveals a subtle bilateral deficit in the neural organization of the reach to grasp movement of a hemiParkinson subject. This dysfunction can be described as a problem with the near-simultaneous or sequential activation of two movement components. Other temporal and kinematic measures support the unilateral clinical signs and symptoms. These findings indicate that impairments in programming goal-directed prehension movements are not only related to damage of cortical areas [20, 22] but also to damage of the deep nuclei and the pathways running through them [1, 14].

Acknowledgements—This research is supported by a grant from the NIH (NS 17421) to G.E.S. The authors wish to thank Dr Marina Scarpa for reviewing the manuscript and Mr Carl Waterman for implementation of the computer programs for data analysis. The two subjects are thanked for agreeing to participate in this experiment.

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