



Kinematic analysis of the reach to grasp movement in Parkinson's and Huntington's disease subjects

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Abstract—This experiment investigates the kinematic characteristics of the reach to grasp movement of Parkinson's and Huntington's disease subjects under two different experimental conditions. In the first condition subjects were required to perform the movement at a normal speed, while in the second condition they were required to perform the movement as fast as possible. Results showed that the kinematic parameterization of movement in Parkinson's disease subjects did not differ from that of age-matched control subjects for both the normal and the fast condition. However, the performances of Huntington's disease subjects appeared to be different when compared to the other two groups. Differences were mainly related to Huntington's disease patients' inability to properly define the temporal features of the movements. © 1998 Published by Elsevier Science Ltd. All rights reserved.

Key Words: humans; movement disorders; Parkinson's disease; Huntington's disease; reach to grasp; kinematic analysis.

Introduction

Parkinson's disease (PD) and Huntington's disease (HD) are two well known pathologies resulting from lesions of the basal ganglia. Even though these diseases are caused by the impairment of the same anatomical structure, they depend on the selective involvement of two different neural circuits: the dopaminergic nigrostriatal system in PD and the cholinergic intrinsic and gabaergic output system in HD. Clinical observations show that these two disorders are also characterized by opposite clinical features: PD often determines brady/hypokinesia, rigidity and tremor, while the most relevant motor disturbances in HD are hyperkinesia and the presence of chorea [8, 17].

A number of studies have investigated the kinematics of the reach to grasp movement. According to the 'channel' hypothesis proposed by Jeannerod [10, 11], prehension movements are subserved by two functionally independent channels, the transport component and the

manipulation component. The 'transport' component extracts information regarding the spatial location of the object and allows for the transformation of this information into commands that are appropriate for bringing the hand towards the object. The 'manipulation' component extracts information regarding the size and shape of the object thus allowing the implementation of the distal movement pattern necessary to grasp the object.

The main feature of the reach to grasp movement of PD subjects, is that, in spite of a longer movement time, they show no deficits in the ability to modify the spatiotemporal characteristics of the prehension pattern (for a review see [5]). This is in response to experimentally imposed changes in either the distance of the object from the subject or the size of the object. Moreover, the results of perturbation studies showed that PD subjects have little dysfunction in the ability to appropriately respond to perturbation of object size and object location [5].

While there is a growing body of data regarding prehension in PD [5, 9], no attempt has been made until now to characterize from a kinematic point of view the features of the reach to grasp movement in HD subjects. Phillips *et al.*'s study [14] was the only one which dealt with the kinematics of HD subjects. In their experiment HD subjects and their age-matched controls were asked to write a letter four times in a linked cursive script,

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and the kinematics of this sequential movement were analyzed. From the results, it appeared that movement duration progressively increased during the HD subjects' performance, and this increase was associated with the accelerative phase of the movement.

The aim of the present study is to investigate the kinematic parameterization of the reach to grasp movement in PD and HD patients, in order to understand whether the selective neurophysiological impairment at the basis of those diseases leads to differential disturbances in the programming and execution of actions. PD, HD, and control (C) subjects were asked to perform a reach to grasp movement in two different conditions: in the first condition they were required to perform the movement at a normal speed (normal condition), in the second condition they were required to perform the movement as fast as possible (fast condition). It has been demonstrated that a modification of the reaching component, such as a speed increase, also influences the grasping component [18]. Thus, it is believed that this experimental manipulation can reveal deficits in the subjects' ability to respond to changes in task requirements and in the ability to coordinate the two separate components of the reach to grasp movement.

Method

Participants

Eighteen subjects volunteered to participate in this experiment. They were divided into three groups, six Parkinson's disease subjects, six Huntington's disease subjects, and six control subjects. The mean age of the control subjects was 53.3 years. The clinical data of the patients are shown in Table 1. None of the HD subjects had the rigid form of HD. Furthermore, they were all tested for the specific mutation which causes the disease and were found to have expanded (ranging from 43 to 46) CAG repeats in the IT15 gene in chromosome 4p [16].

All subjects were right-handed according to the Edinburgh Inventory [13], reported normal or corrected-to-normal vision, and were ignorant as to the purpose of the experiment. Each participant attended one experimental session of approximately 30 min duration.

Apparatus and materials

The experiment was conducted under normal room-lit conditions. The participant was seated in front of the table working surface (1×1 m). Prior to each trial, the participant placed the right hand on the table in the mid-sagittal plane, 15 cm from the thorax. The index finger and thumb were held gently opposed, and the ulnar border of the hand rested upon a mark which indicated the starting position for each trial. A glass (8 cm height \times 6.5 cm diameter) was placed 30 cm from the starting position on the subject's mid-sagittal plane.

Movements were recorded with the ELITE system [7]. Reflective passive markers (0.3 cm diameter) were attached to the following points of the reaching limb: (a) wrist-radial aspect of the distal styloid process of the radius; (b) index finger-radial side of the nail; (c) thumb-ulnar side of the nail.

Procedure

Subjects were seated at the table with their right hand on the starting position. After hearing a starting signal subjects had to reach and grasp the glass placed on the table and lift it up. The acquisition phase began before the starting signal and finished as soon as the glass was lifted. Acquisition continued until the required number of trials was recorded. In this experiment two different experimental conditions were tested. In the first condition subjects were required to perform the movement at a normal speed (normal condition), in the second condition they were required to perform the movement as fast as possible (fast condition). The order of conditions was counterbalanced across participants.

Data processing

The ELIGRASP (BTS, 1994) software package was used to assess the data. The transport component was assessed by

Table 1. Clinical data of the six Parkinson's disease (PD) and of the six Huntington's disease (HD) patients

Patient	Group	Sex	Age	Duration (years)	Functional assessment	Medication
1	PD	M	77	0.2	13/60	L-Dopa, benserazid, selegiline
2	PD	F	42	5	11/60	L-Dopa, benserazid, selegiline, biperiden
3	PD	F	61	2	3/5*	L-Dopa, benserazid
4	PD	M	71	16	19/60	L-Dopa, carbidopa, pergolid-mesylate
5	PD	F	47	6	20/60	L-Dopa, benserazid, bromocriptine
6	PD	M	57	5	17/60	L-Dopa, benserazid, selegiline, bornaprin
7	HD	M	53	1	12/64	Haloperidol 0.4 mg b.i.d.
8	HD	F	52	1	14/64	none
9	HD	F	66	12	28/64	Haloperidol 0.5 mg t.i.d.
10	HD	F	53	6	24/64	none
11	HD	M	45	4	26/64	Haloperidol 0.4 mg t.i.d.
12	HD	M	56	4	27/64	Clorpromazine 10 mg t.i.d.

The Functional Assessment score was computed according to the UPDRS (Motor Section) for Parkinson's disease patients and by means of the UHDRS (Motor Section) for Huntington's disease patients. *Patient no. 3 was only evaluated according to the Hoehn and Yahr Scale.

analyzing the velocity and acceleration profiles of the wrist marker. The manipulation component was assessed by analyzing each of the finger markers, and the distance between these two markers. The beginning of the movement was taken as the time when the wrist marker overtook 1 mm/s. According to the functioning of the Eligrasp software, each trial was separately analyzed by the experimenter, who could select the position on a graph which corresponded to the beginning of the movement, and then look for the exact value of velocity > 1 mm/s. In this way, data analysis was reliably limited to the movement of interest, and any small movement of the wrist due to tremor or chorea was excluded.

The end of the movement was taken as the time when the velocity of the wrist marker reached its minimum, after the subject's fingers had firmly grasped the target and before the glass was lifted. The period following this, whereby the glass was lifted, was not assessed. Thus, movement duration was calculated as the time elapsing between these two temporal landmarks. Another dependent variable was 'delay', that represents the delay with which the manipulation component begins with respect to the transport component. In particular, delay was calculated by subtracting manipulation time (i.e. from the moment when the distance between the markers on the thumb and the index finger overtook 0.1 mm to the end of the movement) from total movement duration.

For the transport component, other dependent variables were deceleration time, the times to peak velocity, peak acceleration, peak deceleration of the wrist marker, and the amplitudes of these peaks (the amplitude of peak velocity, the amplitude of peak acceleration, and the amplitude of peak deceleration, respectively); for the manipulation component, manipulation time, the time to peak grip aperture, and the amplitude of the aperture. In order to compare kinematic temporal data of each condition and group, each temporal value was also calculated as a percentage of movement duration (relative values).

Results

The mean values for each parameter have been analyzed with an analysis of variance (ANOVA; $\alpha = .05$) with the following factors, group (PD, HD, C) and condition (normal or fast). Given that the aim of this study is to compare the kinematic parameterization of the movement in different groups and in different conditions only the relative values, that allow a better comparison of temporal events, will be reported in the text (only time to peak velocity has been expressed in absolute terms). For other values and statistics referring to the difference between groups please refer to Table 2. *Post hoc* comparisons were performed using the Newman-Keuls procedure.

Movement time. For all groups, movement time was longer in the normal than in the fast condition: 1163 vs 791 ms; $F(1,15) = 70.7$, $P < 0.0001$. For this parameter also the interaction group \times condition was significant: $F(2,15) = 4.18$, $P = 0.036$. In particular, it was significantly different in the two conditions for PD subjects (1199 vs 705 ms, $P = 0.0002$) and for control subjects (1115 vs 687 ms, $P = 0.0004$), while it strongly approached significance for HD subjects (1175 vs 980, $P = 0.0554$). Furthermore, *post hoc* comparison revealed the absence of differences between groups in the normal

condition (PD = 1199, HD = 1175, C = 1115, $P > 0.05$; see Fig. 1).

Delay. Even if it tended to be longer for PD, this difference did not reach significance (PD = 14%, HD = 11%, C = 11%).

Deceleration time. The time from peak velocity to the end of the movement in the fast condition was shorter than in the normal condition (59% vs 55%; $F(1,15) = 7.47$, $P = 0.015$). Also the main effect of group was significant: the mean deceleration time for PD and control subjects was the same (55%), while it was greater for HD subjects (62%).

Time to peak velocity. The time from the beginning of movement to the maximum velocity was shorter when movement was executed in the normal condition: 471 vs 339 ms; $F(1,15) = 61.79$, $P < 0.01$.

Time to peak acceleration. The time from the beginning of movement to peak acceleration was shorter for the HD group than for PD and controls: PD = 30%, HD = 24%, C = 29%.

Time to peak deceleration. As for time to peak acceleration, time to peak deceleration was anticipated in HD patients (51%) with respect to PD (63%) and controls (61%). Also the main effect of condition was significant, the peak being reached earlier in the normal condition (55% vs 62%; $F(1,15) = 10.47$, $P = 0.006$).

Amplitude of peak velocity. This parameter was significantly smaller in the normal than in the fast condition: 553 vs 815 mm/s; $F(1,15) = 76.86$, $P = 0.0000$. Also the interaction group \times condition was significant: $F(2,15) = 4.64$, $P = 0.027$. In particular, in each group, the amplitude of peak velocity was greater in the fast condition.

Amplitude of peak acceleration. The interaction group \times condition was significant ($F(2,15) = 4$, $P = 0.04$). In particular, for both PD and controls the amplitude of peak acceleration was greater in the fast condition (PD: 2429 vs 6793 mm/s², $P = 0.0008$; C: 2541 vs 5995 mm/s², $P = 0.003$), while for HD there was no difference between the two conditions (3423 vs 4708 mm/s², $P = 0.125$).

Amplitude of peak deceleration. The only significant finding was that the amplitude of the peak was smaller in the normal condition than in the fast condition: 2157 vs 4478 mm/s²; $F(1,15) = 41.6$, $P = 0.00001$.

Manipulation time. This parameter did not vary significantly across groups, neither in absolute (PD = 825 ms; HD = 969 ms; C = 797 ms; $F(2,15) = 1.889$, $P = 0.185$) nor in relative (PD = 86%; HD = 89%, C = 89%; $F(2,15) = 0.68$, $P = 0.517$) terms.

Time to peak grip aperture. No significant differences emerged.

Amplitude of peak grip aperture. The maximum fingers aperture increased when movement was performed at fast speed: 100 vs 107 mm, $F(1,15) = 25.2$, $P = 0.0002$.

Discussion

The aim of this study was to investigate the kinematic parameterization of the reach to grasp movement in pat-

Table 2. Mean values of movement duration and kinematic parameters in PD, HD and C

		PD	HD	C	<i>P</i> values
Movement Duration	(ms)	952	1077	901	n.s.
Delay	(ms)	127	108	105	n.s.
	(%)	14	11	11	n.s.
<i>Transport Component</i>					
Deceleration Time	(ms)	536	677	501	n.s.
	(%)	55	62	55	$F(2,15) = 5.49$, $P = 0.016$
Time to peak velocity	(mm/s)	416	400	400	n.s.
	(%)	45	38	45	$F(2,15) = 5.02$, $P = 0.021$
Time to peak acceleration	(ms)	274	256	261	n.s.
	(%)	30	24	29	$F(2,15) = 4.14$, $P = 0.037$
Time to peak deceleration	(ms)	576	535	540	n.s.
	(%)	63	51	61	$F(2,15) = 4.66$, $P = 0.027$
Amplitude peak velocity	(mm/s)	681	676	695	n.s.
Amplitude peak acceleration	(mm/s ²)	4611	4065	4268	n.s.
Amplitude peak deceleration	(mm/s ²)	3467	3110	3377	n.s.
<i>Manipulation Component</i>					
Manipulation Time	(ms)	825	969	797	n.s.
	(%)	86	89	89	n.s.
Time to maximum grip aperture	(ms)	631	616	567	n.s.
	(%)	65	58	63	n.s.
Amplitude grip aperture	(mm)	97	111	102	n.s.

Please note that values refer to the main effect of Group, irrespective of the two experimental conditions (i.e. normal vs fast).

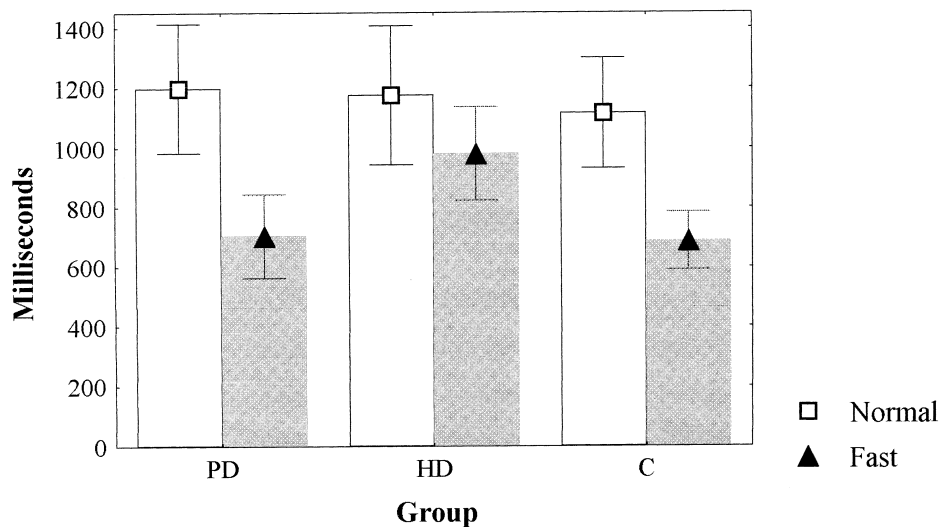


Fig. 1. Movement time for PD, HD and C in the normal and fast conditions. Bars show standard deviation.

ients suffering from Parkinson's and Huntington's disease. The first interesting result was that while the parameterization of movement was very similar for PD patients and control subjects, some differences emerged in the comparison with the HD group.

For example, when looking at movement times in the normal and fast condition in the three groups, HD sub-

jects showed a difficulty in performing the movement at a fast speed. For this group, the time when peak velocity, peak acceleration, and peak deceleration occurred in the two conditions was identical, while for the other two groups, although differences were not statistically significant, all peaks were reached earlier in the normal condition. It is interesting to note that amplitude of peak

velocity was the only parameter that changed between the normal and the fast condition, and it changed for HD in the same way as for PD and control subjects (that is, it was inversely related to movement time). This result seems to confirm the deficit of HD subjects in properly programming movement characteristics, given that there should be no reason to improve amplitude of peak velocity without changing (shortening) the movement time.

Another important finding regarding movement time was the absence of differences between the three groups in the normal condition. Bradykinesia is commonly thought to be a peculiar feature of PD patients [12]. Recently, many researchers demonstrated that bradykinesia can also be considered a fundamental characteristic of the motor deficits of HD [1, 8, 14, 17]. However, the findings of the present study seem to demonstrate that neither PD nor HD subjects are bradykinetic. A possible explanation for this result could be linked to the type of movement performed in the experimental session. The reach to grasp movement requires the implementation of two motor programs, namely the reaching component and the grasp component, that are functionally linked and overlearned [10, 11]. Furthermore, the number of variables to control was low, given that the target object was quite big and no requirements were made as to accuracy. Thus, it could be suggested that PD and HD patients should no longer be considered bradykinetic *tout-court*. As a matter of fact it is likely that bradykinesia reveals itself in a differential way according to the type of movement that has to be executed: that is, it can be observed in the case of complex actions but remains undetected in the case of simple and overlearned actions such as the one described in this study.

Another finding was the absence of differences between groups concerning the 'delay' parameter. This result contrasts with previous literature, where a delay in the activation of the manipulation component with respect to the transport component was found in PD patients [5, 6, 15]. This was significantly greater for PD than for age-matched control subjects. It should be noted, however, that the presence of delay is not constant in PD subjects. For example, Bennett *et al.* [2] reported that not all of the PD patients tested showed a delayed onset of manipulation time, and that, among those who showed it, delay was not present in every trial. These data point to the fact that the PD population shows both a great inter- and intra-subject variability, and it could be suggested that it is this high level of heterogeneity that determines the different results found in the present study [see also 3].

The overall similarity of the reach to grasp for PD and normal subjects, which in turn was significantly different from HD subjects, contrasts with the findings of Brown *et al.* [4]. In a bimanual task they found that the performance of PD, HD and cerebellar subjects was different from the performance of control subjects, while it was not possible to find differences between the groups of patients. The present study, in which a different, more

natural task was used, showed that such differences exist. HD subjects spent significantly more time in the deceleration phase than the other two groups. This longer deceleration time could have benefitted either the transport or the manipulation component. However, given that no difference was found between groups for manipulation time, it could be suggested that the longer amount of time spent by the HD subjects in the deceleration phase was related to the transport component. This result could be interpreted as an error compensating strategy: having a longer deceleration phase, i.e. the part of movement spent in the homing on the target, can allow for a greater number of corrections if movement execution does not run smoothly.

In conclusion, it appears that, although PD and HD both alter the basal ganglia, they affect the motor system in different ways. In PD patients the ability of defining the kinematic parameterization of movements (at least, of the reach to grasp movement) seemed to be preserved, as no differences between patients and normal subjects were found. On the contrary, HD patients showed a disruption of this ability. The differences between PD and HD could depend on the way the nervous system reacts to the motor deficits caused by the disease. In PD the system manages to compensate for a series of motor symptoms such as resting tremor, rigidity and bradykinesia that are quite easily predictable. In the case of HD, instead, motor disturbances such as dyskinesia and choreic movements interfere with voluntary activity in an unpredictable way [8], thus forcing the motor system to adopt a safer parameterization (that is, a longer deceleration phase) which can allow for a greater number of corrections if an involuntary movement occurs during the execution of the action.

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