Control of Multijoint Arm Movements in Huntington's Disease

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Multijoint arm movements of individuals with Huntington's disease (HD) were examined using three-dimensional kinematic analysis. Six HD patients with chorea and four healthy subjects performed pointing movements to a 2.5 cm target positioned at three distances in the sagittal plane, two of which required trunk motion. Healthy subjects moved in relatively straight hand paths to the targets. All HD patients produced curved hand paths, in which they brought their hand first upward and then outward to the target. Healthy subjects made single smooth movements, while HD patients made large initial movements followed by multiple submovements as the hand honed in on the target. Lower functioning HD patients had particular difficulty moving to the farthest target, which required the greatest amount of trunk motion. Although the HD patients had longer movement times across all conditions, their initial velocity was often similar to that of the healthy subjects. This suggests that bradykinesia is related to the production of submovements, rather than a deficit in initial force production. The presence of submovements in Huntington's disease might reflect a deficit in controlling deceleration of the limb or an adaptive strategy to maximize accuracy. **Key Words:** Bradykinesia—Submovements—Arm control—Pointing—Basal ganglia—Chorea.

Introduction

Although the most easily recognized motor disorder in Huntington's disease (HD) is involuntary choreic movements, deficits in the control of voluntary movements have been emphasized in recent literature (1,2,3,4). Impairments in voluntary motor control, such as akinesia (difficulty in initiating movements) and bradykinesia (slowness in executing movements), have

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been found to correlate better with functional impairments than chorea (5).

Investigations of voluntary motor control in HD have found several deficits in movement planning and execution. Hefter and colleagues (2) examined isometric finger contractions and reported that more severely impaired HD patients do not scale the rate of rise of force with amplitude of force as do healthy subjects. These authors emphasized impairments in force control as a primary deficit in individuals with HD. Other researchers have focused on deficits in performing sequential or simultaneous movements. Thompson and colleagues (3) reported that individuals with HD have difficulty performing two tasks (elbow flexion and hand squeezing) either simultaneously or sequentially. Bradshaw and colleagues (1) have further suggested that HD patients have difficulties initiating movements in the absence of external visual cues and in using advance information to control sequential movements.

More complex skills, such as handwriting, have been recently studied by Phillips and colleagues (4,6). Kinematic

analysis of handwriting skills in HD patients has revealed that HD patients are slower and more variable in such parameters as stroke length and duration than healthy subjects (4). Handwriting disrupted by choreic movements was poorer on several measurements, including quality and efficiency of handwriting. When the obvious choreic movements were excluded, irregularities in these movement parameters persisted. Phillips and colleagues (4) have suggested that observed irregularities in stroke length and duration of HD patients are related to the construction of movement parameters rather than the production of individual strokes. Thus, bradykinesia in HD could be linked to problems with force efficiency, rather than force development.

Although each of these studies employed different tasks, all reported the presence of bradykinesia in individuals with HD. The underlying basis for bradykinesia has not been extensively studied in HD but has been for Parkinson's disease (PD), another neurologic disorder affecting the basal ganglia. Hallett and Khoshbin (7) suggested that bradykinesia in PD resulted from an inability to generate sufficient forces. Alternatively, Sheridan and Flowers (8) hypothesized that bradykinesia in PD may reflect a strategy employed to handle errors in movement generation rather than a force production problem. Montgomery and Nuessen (9) further suggest that PD patients decrease their movement speed to a greater extent than healthy subjects as a strategy to maximize spatial accuracy.

Conclusions regarding bradykinesia in Parkinson's disease and Huntington's disease are speculative. While current research in HD has identified specific deficits when performing simple one or two joint movements, it has not addressed deficits in the coordination of multijoint, goal-directed movements. The control of multijoint pointing movements has been studied extensively in healthy individuals (10,11,12,13). Kinematic analysis has been utilized to identify invariant features of these movements, which serve as a window into the planning and execution of goal-directed movements in general. For example, relatively straight hand paths and smooth bell-shaped velocity profiles have been observed in pointing movements to targets in the absence of strict accuracy constraints (10). Such movements are thought to be based primarily on feed-forward control, in which one smooth movement can bring the hand directly to the target (14). As accuracy constraints on the movement increase, visual and proprioceptive feedback are utilized more to guide the hand to the target, resulting in a prolongation of the deceleration phase.

More recent research has investigated the coordination of arm and trunk motion in healthy subjects (15). These authors reported that in healthy subjects, trunk motion integrates smoothly into the transport phase of the hand. The trunk acts not only as a postural stabilizer during reaching and pointing, but becomes highly integrated in positioning the hand close to the target. Thompson and colleagues (3) have reported that patients with HD have particular difficulty coordinating more complex actions, such as simultaneous or sequential movements. The tasks used in this study were somewhat unnatural, e.g., squeezing the hand and flexing the elbow. It is not known how individuals with HD coordinate more natural movements, such as multijoint reaching.

Although reaching and pointing movements (with and without trunk motion) typically result in relatively smooth unimodal velocity profiles, deviations from this shape can occur. Movements with high accuracy requirements also tend to produce multimodal velocity profiles (16,17). For example, Milner and Ijaz (17) reported multiple peaks in hand velocity profiles which corresponded to changes in the hand path direction as subjects honed in on the target. These secondary movements were termed *submovements* and occurred soon after peak velocity was reached. The authors suggested that for movements requiring high accuracy, visual feedback is used to monitor movement of the limb and to modify the ongoing movement. This online error correction may be reflected by the presence of submovements in the hand velocity profile and changes in the direction of the hand path.

The presence of submovements has been reported in a limited number of studies in HD and PD. Phillips and colleagues (4) reported submovements in HD patients performing handwriting tasks. These authors primarily attributed the occurrence of submovements to inefficiencies in force production rather than to the interference of choreic movements. Flash and colleagues (18) have observed multiple peaked velocity profiles in individuals with Parkinson's disease. In this study PD patients performed arm pointing movements with a stylus to targets in the absence of high accuracy constraints. While the PD patients had relatively straight hand paths, the authors reported prolonged deceleration phases with alternating periods of acceleration and deceleration as the subjects approached the target. Flash and colleagues (18) concluded that PD patients depended more heavily than healthy individuals on visual information for error-detection feedback.

The aim of the present study was to investigate coordination strategies and identify deficits related to planned movements that require varying degrees of postural coordination in individuals with Huntington's disease. Unrestrained, fast pointing movements were performed to three target distances, two of which required movement of the trunk. Movements that require trunk

motion place increased organizational demands on the system by introducing additional degrees of freedom (19). Three-dimensional kinematic analysis of both hand and joint paths enabled us to examine the complexity of movements common to patients with choreic movements.

Methods

Subjects

Six HD patients and four healthy subjects volunteered for this study. Inclusion criteria for participation were: (1) age 35–60; (2) at least five years since onset of HD symptoms; (3) chorea score of 2–3 on a 5-point scale (20); (4) right-handed; (5) able to follow two-step commands. All subjects in this study were ambulatory for household distances and were able to maintain a sitting position for the duration of the experiment. This study was approved by the Institutional Review Board of JFK Medical Center in Edison, New Jersey. All subjects signed informed consent prior to participation in the study.

The healthy subjects ranged in age from 34 to 58 years, with a mean age of 47.2 years. The HD patients ranged in age from 40 to 57 years, with a mean age of 47.8 years. The functional status of each patient is displayed in Table 1. The diagnosis of HD was confirmed by a neurologist based on family history and clinical signs.

Experimental Apparatus and Paradigm

Subjects were seated in a locked wheelchair without armrests, with their feet resting on a foot plate. The subject's back was resting against a firm back support (50 cm in height), angled at 95° relative to the seat. Subjects performed arm pointing movements to three target locations. A 2.5 cm circular target was placed at shoulder level on a vertical pole directly in front of the subject's right shoulder. Subjects were required to point to the same target placed at one of three distances from the subject's right shoulder: close, 90% of arm length; middle, 115% of arm length; and far, 140% of arm length. Arm length was measured from the acromion process to the distal end of the second finger with the elbow fully extended. The middle and far distances required movement of the trunk to reach the target, whereas the close distance did not.

For the starting position of each trial, the subject's hands were resting on the thighs and the back was resting against the back support of the wheelchair. Subjects were instructed to touch the center of the target with their right index finger, moving as fast as possible. Following three practice trials, each subject performed five to eight successive test trials for each condition. The first three successful test trials in which the subject was in the correct starting position were chosen for analysis. All subjects also performed the same pointing movements sitting without back support. The order was counterbalanced for target distance and back support. Only those trials for which subjects had back support are discussed in this report. Subjects completed all trials for each back support and target distance before changing to a new condition.

Trials were videotaped using standard VHS cameras placed at three locations. Two cameras were placed at 30° to the right and left of the sagittal plane, six feet in front of the subject. The third camera was placed six feet to the right of the subject, perpendicular to the sagittal plane. The sampling rate was 30 Hz. Reflective markers (2 cm in circumference) were placed over the subject's skin or tightly adhered to the subject's clothing for digitizing purposes. These markers were placed at the center of the following joints: the hip, on the greater trochanter and

Table 1. *Descriptive information for six patients with Huntington's disease*

Subject	Barthel Index*	Age (yrs.)	Time since onset (yrs.)	Chorea score**
1	14	57	11	3
2	15	44	4	3
3	16	45	14	3
4	11	46	11	3
5	11	40	21	4
6	13	55	8	3

* Barthel Index score of functional capabilities (1–20 Scale) (21)
** Chorea score obtained from Quantitative Neurological Examination (1–5 scale) (20)

groin; the shoulder, on the greater tuberosity and anterior gleno-humeral joint; the elbow, on the lateral and medial epicondyles; and the wrist, over the ulnar and radial styloids.

Data Analysis

The Ariel Kinematic Analysis System (Ariel Dynamics, Inc., Trabuco Canyon, CA) was used to digitize the markers from the videotapes in order to obtain three-dimensional position data. The raw data were digitally filtered with a second-order low pass Butterworth using a cutoff frequency of 8 Hz.

All temporal and spatial measurements reflecting hand motion were obtained from movement of the wrist. The hand path refers to the series of positions that the hand follows in Cartesian space. The onset of movement was defined as the time when the subject's hand lifted from the starting position and exceeded a tangential velocity of 15 cm/s; termination of movement was defined as the time when the subject's finger first contacted the target. The initial peak velocity was the first peak in the tangential velocity profile of the hand. The following parameters were obtained for each trial: *maximum velocity of the hand* was the greatest tangential velocity achieved at any point during the movement; *movement time* was the time from onset of movement to target contact; *acceleration time* was the time from onset of movement to the initial peak velocity; and *deceleration time* was the time from initial peak velocity to the end of movement. *Percentage of time in deceleration phase (PTDP)* was determined by dividing the deceleration time by the movement time and multiplying by 100. Shoulder and elbow joint displacement data were calculated from position data of the wrist, elbow, shoulder, and hip joints.

For the joint analysis, the *shoulder angle* was defined as the angle between the upper arm (the line from the shoulder marker to the elbow marker) and the horizontal. The *elbow angle* was defined as the angle between the upper arm and forearm (the line from the elbow marker to the wrist marker). The *hip angle* (trunk initial position) was defined as the angle between the trunk segment (the line from the shoulder marker to the hip marker) and the horizontal.

The initial peak in the velocity profile (rather than the maximum velocity) was used in this study to differentiate the acceleration and deceleration phases of the movement. Although the first peak in the velocity profile is not always the largest, it is most likely representative of the initial burst of acceleration based on feed-forward control (14,18).

Tangential velocity profiles of the hand were analyzed for each trial for each subject. The *number of velocity peaks* was determined by counting the peaks in the hand tangential velocity profile for each trial. This was done by decomposing the velocity profiles into multiple submovements (22). The beginning of the first submovement was taken as the onset of movement. The end of each submovement was determined when the velocity decreased to a local minima, followed by an increase in velocity (deceleration followed by reacceleration). The local minima was taken as the ending of the previous submovement and the beginning of the next submovement.

The straightness of hand paths was evaluated by examining the ratio of the actual distance the hand traveled between the start and end of the movement to the shortest distance (a straight line path). A linearity ratio of 1 would indicate a straight line path; values greater than 1 indicate deviation from a straight path.

Statistical Analysis

The statistical analysis model used was a 2 (healthy and HD) x 3 (close, middle, and far target distances) factorial ANOVA with repeated measures on the second factor (23). A p level of 0.05 was judged statistically significant. The following dependent variables were analyzed with this model: movement time, acceleration time, PTDP, maximum velocity, and linearity ratio. The HD patients were further divided into two groups to analyze differences based on Barthel scores, which rate level of functional ability (21). Wilcoxon Rank Sum tests (24) were used to analyze differences between high and low functioning HD patients on movement time, acceleration time, PTDP, maximum velocity, linearity ratio, and number of velocity peaks.

Results

In the first part of this section, we report the spatial characteristics of pointing movements: hand paths, shoulder-elbow coordination, and trunk initial position. In the second part, parameters relative to the time course of the hand movement (movement time and velocity characteristics) are reported. Lastly, the differences within the HD patient group are discussed. Illustrative records from single subjects are presented in each figure. Summary statistics based on all subjects are also reported.
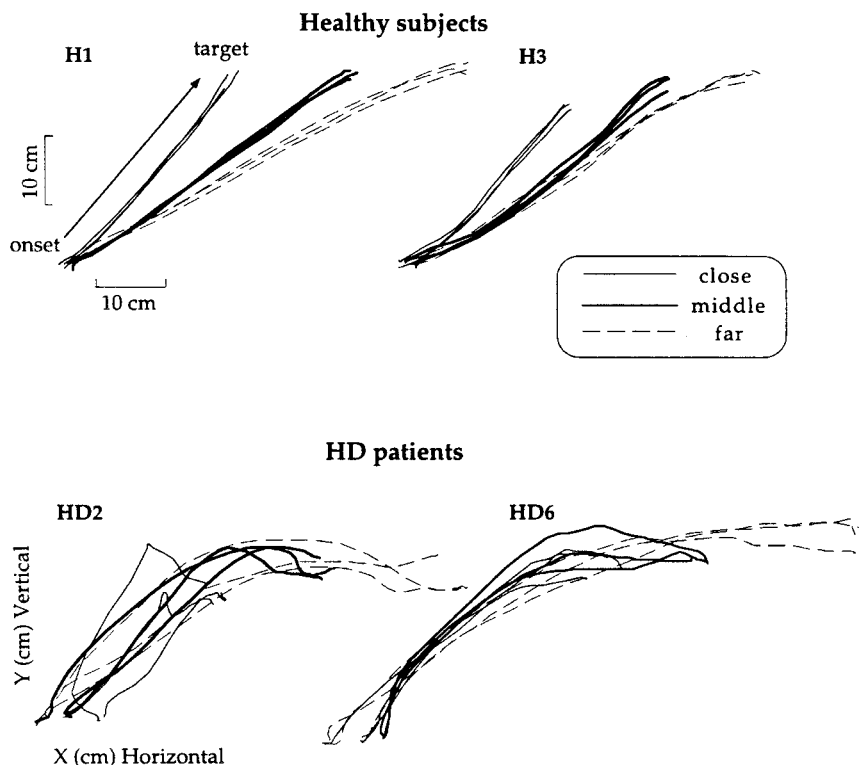
Spatial Characteristics

HAND PATHS

Figure 1 represents hand paths in the sagittal plane for two healthy subjects (H1 and H3) and two HD patients (HD2 and HD6). As expected, healthy subjects moved in relatively straight hand paths to all three target locations. In contrast, HD patients had curvilinear hand paths. This curvilinear pattern was consistent for all three target distances. Figure 2 shows the hand paths of the same subjects and trials as those of Figure 1, viewed from the frontal plane. As compared to the healthy subjects, more deviations in the frontal plane of motion were noted for the HD patients.
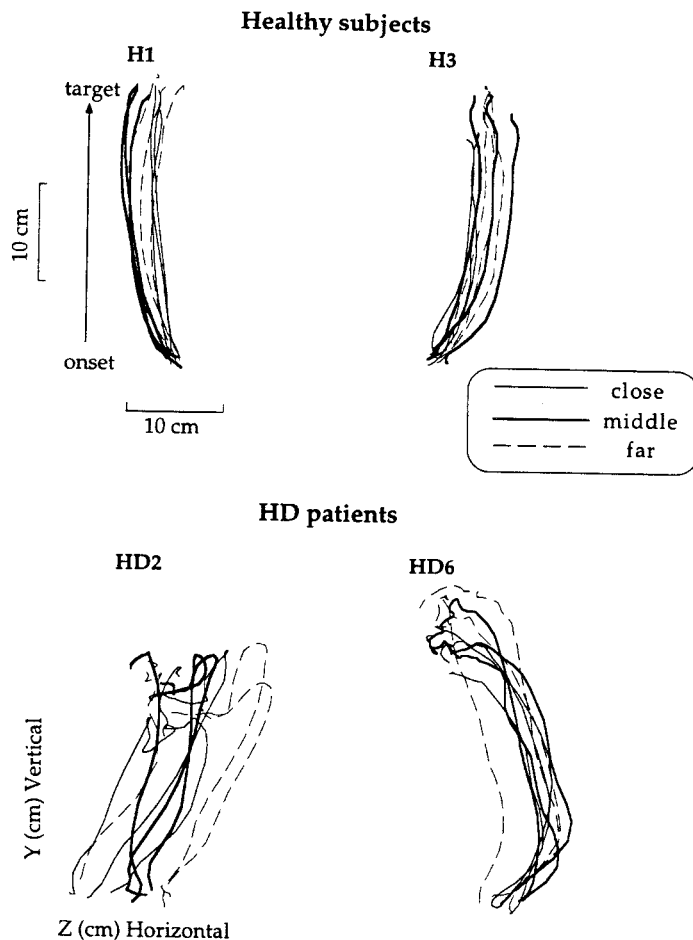
The highly curved hand paths for the HD patients were reflected by significantly greater linearity ratios as compared to the healthy subjects, averaged across target distance (Table 2); $[F(1,7) = 18.82; p < 0.05]$. Hand path curvature was not significantly affected by target distance $(p > 0.05)$, and no interaction effect between the two groups was observed $(p > 0.05)$.

SHOULDER AND ELBOW COORDINATION

We investigated the possibility that the curvilinear paths of the HD patients could result from poor joint coordination. In particular, subjects might have difficulty moving both joints simultaneously. Sequential single joint motions could result in curved hand paths. Figure 3 shows angle-angle plots and illustrates the coordination between the shoulder (y axis) and elbow joints (x axis) for two healthy subjects (H1 and H3) and two HD patients (HD2 and HD4) for all trials. Generally, the healthy subjects displayed simultaneous movements of the shoulder and elbow toward extension. The HD patients had different patterns of coordination. The initial movement of the HD patients often consisted of the shoulder and elbow joints moving toward flexion (Figure 3). Shoulder and elbow joint motions were typically simultaneous toward the end of movement, as illustrated by the diagonal orientation of the plots of patient HD2. Although the joint paths for some patients were quite variable, all HD patients demonstrated the ability to simultaneously move the elbow and shoulder joints.

Figure 1. Hand paths of pointing movements in the XY (sagittal) plane for two healthy subjects (H1 and H3) and two HD patients (HD2 and HD6). Three trials are represented for each of the three target distances that are 90% (close), 115% (middle), and 140% (far) of subject's arm length.

Healthy subjects

H1   **H3**

target

10 cm

onset

10 cm

─────── close
─────── middle
─ ─ ─ ─ far

HD patients

HD2   **HD6**


Y (cm) Vertical

Z (cm) Horizontal

Figure 2. Hand paths in the YZ (frontal) plane of two healthy subjects (H1 and H3) and two HD patients (HD2 and HD6). Three trials for the three different target distances are represented.

Table 2. *Temporal and kinematic parameters for healthy subjects and HD patients for three target distances (means and standard deviation)*

Parameter	Group	Close target	Middle target	Far target
Hand path linearity ratio	H	1.05 ± 0.02	1.06 ± 0.02	1.06 ± 0.01
	HD	1.36 ± 0.14	1.22 ± 0.07	1.24 ± 0.17
Trunk initial position (degrees)	H	94.07 ± 4.34	96.03 ± 2.10	95.80 ± 2.93
	HD	100.15 ± 6.48	96.68 ± 4.29	100.77±7.18
Movement time (sec)	H	0.54 ± 0.02	0.66 ± 0.04	0.77 ± 0.06
	HD	1.08 ± 0.16	1.01 ± 0.19	1.44 ± 0.51
Acceleration time (sec)	H	0.23 ± 0.02	0.25 ± 0.01	0.26 ± 0.05
	HD	0.18 ± 0.07	0.24 ± 0.19	0.20 ± 0.05
Percentage of time in deceleration (PTDP) (%)	H	58.9 ± 2.6	62.2 ± 3.3	63.7 ± 3.5
	HD	83.0 ± 7.8	76.9 ± 17.0	84.9 ± 5.9
Maximum hand velocity (cm/sec)	H	115.4 ± 8.3	138.1 ± 17.3	155.8 ± 35.1
	HD	101.0 ± 12.6	108.1 ± 38.8	125.7 ± 19.8

Note: H refers to healthy subjects; HD refers to Huntington's disease patients.

Healthy subjects
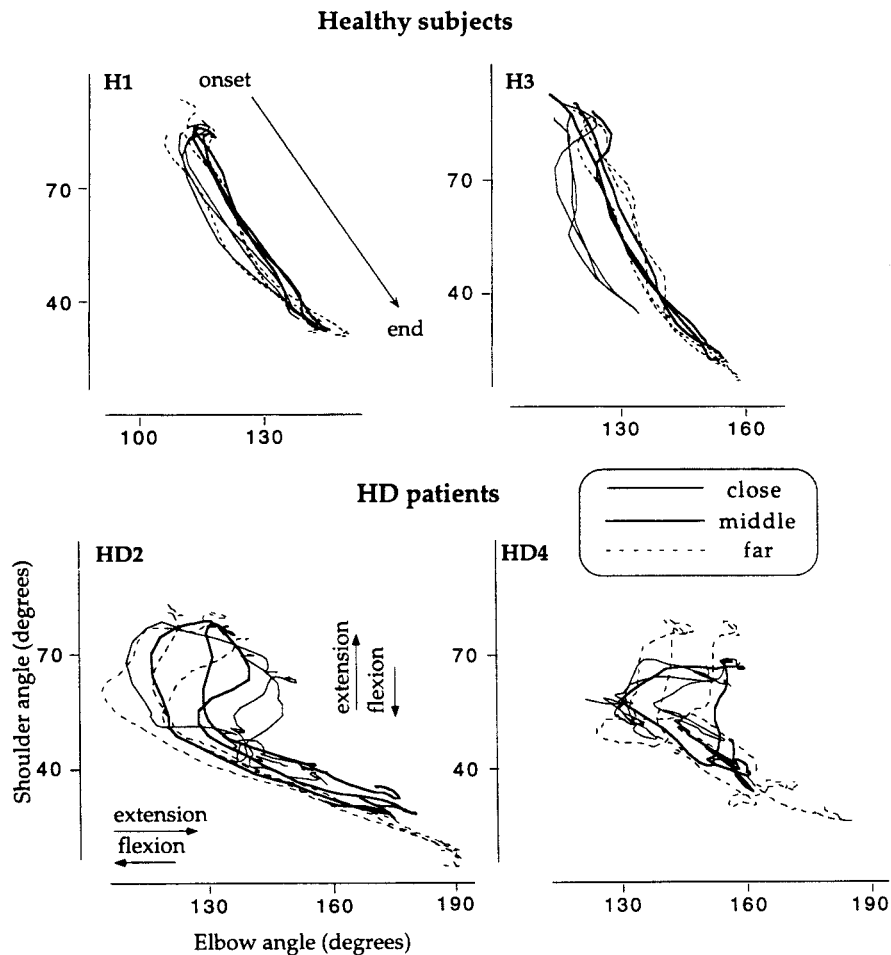
HD patients

Figure 3. Shoulder and elbow angle-angle plots of pointing movements for two healthy subjects (H1 and H3) and two HD patients (HD2 and HD4). The shoulder angle is relative to the horizontal. When the upper arm is parallel to the horizontal plane, the angle is 0°; when the upper arm is parallel to the subject's body, the angle is 90°. An elbow angle of 180° corresponds to full elbow extension.

TRUNK INITIAL POSITION

At the start of each trial, all subjects were positioned in the chair in an upright position, with their back resting against the chair. We observed that some of the HD patients had difficulty maintaining this position and tended to push their hips forward in the chair. This gave the appearance that they were more reclined in the chair, as opposed to the upright position maintained by the healthy subjects. We examined whether the degree of trunk inclination at the start of movement differed for the two groups, as this may affect coordination patterns. Table 2 shows the mean starting hip angles (trunk initial position) for all subjects and patients. No significant difference was found between the starting hip angles of the healthy subjects and HD patients ($p > 0.05$).

Time Course of Hand Movements

MOVEMENT TIME

HD patients took significantly longer to complete the pointing task than healthy subjects across all target distance conditions (Table 2); [$F(1,7) = 24.19$; $p < 0.05$]. The HD patients displayed more variability in their movement times than the healthy subjects, as reflected by greater standard deviation values (Table 2). As target distance increased, movement time increased for both the HD patients and healthy subjects [$F(1,7) = 9.81$; $p < 0.05$]. This effect was not different between the two groups (interaction effect; $p > 0.05$).

HAND TANGENTIAL VELOCITY PROFILES

Figure 4 shows the tangential velocity profiles of the hand for two healthy subjects (H2 and H3) and two HD

Healthy subjects
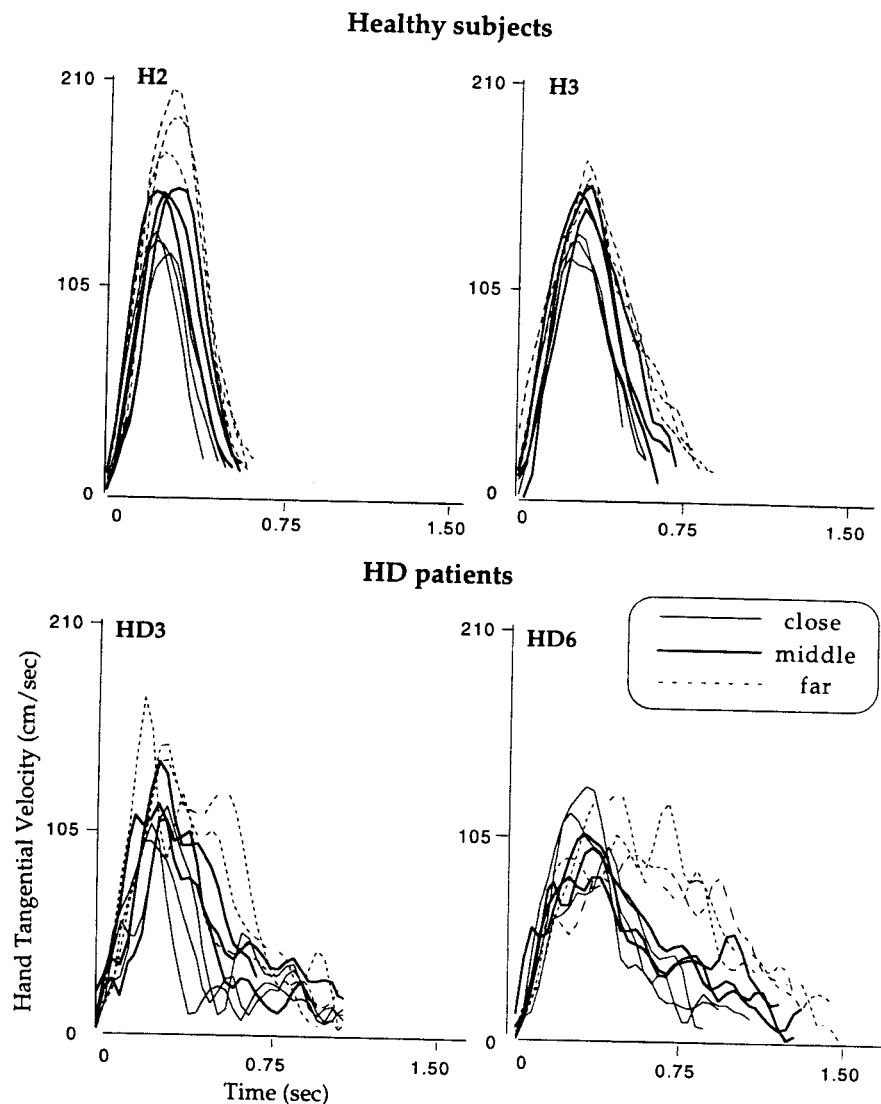
HD patients

Figure 4. Tangential velocity of the hand for pointing movements for two healthy subjects (H2 and H3) and two HD patients (HD3 and HD6). The velocity profiles are aligned with the onset of movement. Three trials are represented for each of the three target distances.
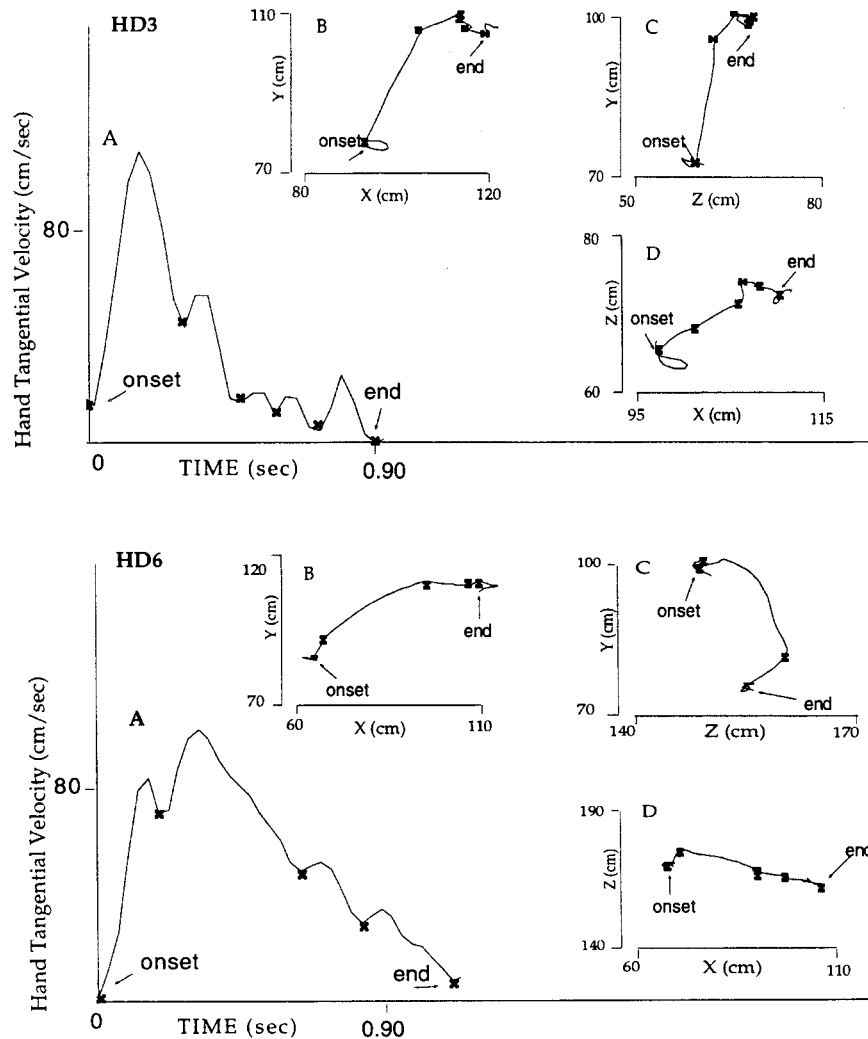
subjects (HD3 and HD6). Hand velocity profiles for healthy subjects were smooth and unimodal. Movements of HD patients tended to be multimodal for all three target distances. Velocity profiles of the HD patients had two general patterns: (1) a relatively smooth displacement of hand movement up to a peak velocity, followed by alternating accelerations and decelerations; and (2) multiple peaks of lower amplitude velocity throughout the movement.

Figure 5 shows a more detailed analysis of the multiple peaks that occurred in the velocity profiles of HD patients. Two representative velocity profiles and corresponding hand paths in three dimensions are illustrated.

The markings (x) along hand paths correspond to local minima in the velocity profile. Reversals in the velocity profile typically corresponded to changes in hand path direction. For most patients, these directional changes brought the hand closer to the target.

The velocity profiles of the HD patients had two general patterns. The top figure (HD3) displays a typical pattern used by some patients, in which there was a single burst of movement followed by subsequent accelerations and decelerations that gradually approached zero velocity. For other patients, as represented in the bottom figure (HD6), the velocity profiles consisted of multiple peaks that occurred throughout the movement.

Figure 5. Hand tangential velocity profiles for two HD patients (HD3 and HD6). The corresponding hand paths are represented in 3D: side view (B), front view (C), and top view (D). Note that inflections, or submovements, in the velocity profile correspond to changes in the direction of the hand path.

Velocity profiles were further analyzed by the time spent in acceleration and deceleration phases of movement. There was no significant difference for acceleration times of HD patients and healthy subjects (p > 0.05). The initial burst of movement toward the target was of a similar duration for the two groups. As a percentage of the overall movement time, there were significant differences between the two groups. The HD patients spent a greater PTDP compared to healthy subjects (Table 2); [F(1,7) = 69.05; p < 0.001]. As seen by the velocity profiles in Figures 4 and 5, HD patients had a prolongation of movement from the initial peak to the end of movement, as compared to healthy subjects. There were no sig-

nificant trends or interaction effects for acceleration time or PTDP as a function of target distance (p > 0.05).

The maximum hand velocities for HD patients were not significantly different from those of healthy subjects (Table 2; p > 0.05). Although the movement time was prolonged, the HD patients were able to achieve maximum velocities that were similar to those of healthy subjects. A significant linear trend between maximum velocity and target distance was observed for both groups [F(1,7) = 5.73; p < 0.05]. Both healthy subjects and HD patients scaled maximum hand velocity with target distance; the farther the target distance, the greater the maximum velocity. This effect was not sig-

nificantly different between the two groups (interaction effect; $p > 0.05$).

Differences Between High and Low Functioning HD Patients

Although HD patients who participated in this study had similar levels of choreic movements, differences could be distinguished based on functional abilities. In order to analyze these differences, the six HD patients were divided into two groups based on functional level according to the Barthel Index (21). Table 3 lists the means ($\pm$ SD) for the temporal and kinematic parameters for higher functioning (HD-high) and lower functioning (HD-low) HD patients.

One important observation in comparing higher and lower functioning patients was that the two groups used different movement strategies as reflected by the hand velocity profiles. As noted earlier, some HD patients produced velocity profiles with an initial burst of movement followed by subsequent accelerations and decelerations that gradually approached zero velocity. This strategy was used most often by HD-high patients (Figure 5, HD3). HD-low patients produced multiple velocity peaks throughout the movement, as represented by patient HD6. HD-low patients utilized both strategies, most frequently using the second strategy for the middle and far conditions.

There was a significant difference between high and low functioning HD patients for the number of velocity peaks for the far distance. HD-low patients had a significantly greater number of velocity peaks compared with HD-high patients ($W = 6.0$; $p < 0.05$). Additionally, the maximum velocities were significantly greater for the HD-high compared to the HD-low patients for the far target ($W = 15.0$; $p < 0.05$). HD-low patients seemed to have had particular difficulty for the far target condition, whereas the HD-high patients did not. This may indicate a greater level of difficulty for the HD-low patients in moving the trunk a farther distance. All other movement parameters were not significantly different between the two groups ($p > 0.05$).

Discussion

Curvilinear Hand Paths

One of the key findings of this study was that the Huntington's disease patients consistently produced curved hand paths, bringing their hands upward and then outward toward the target. This pattern was different from that of the healthy subjects, whose hand paths were relatively straight. Despite the potential influence of choreic movements, all HD patients used the same strategy to accomplish the task. There are several possible explanations for the presence of curvilinear hand paths in the HD patients: (1) joint incoordination; (2) a strategy to reduce postural perturbation; or (3) the need for direct visual monitoring of the limb.

Table 3. *Temporal and kinematic parameters for high and low functioning HD patients for three target distances (means and standard deviations)*

Parameter	Group	Close target	Middle target	Far target
Hand path linearity ratio	HD-High	1.43 ± 0.22	1.21 ± 0.03	1.17 ± 0.07
	HD-Low	1.29 ± 0.14	1.24 ± 0.07	1.32 ± 0.18
Movement time (sec)	HD-High	1.11 ± 0.14	1.02 ± 0.18	1.24 ± 0.30
	HD-Low	1.05 ± 0.16	1.00 ± 0.18	1.63 ± 0.51
Acceleration time (sec)	HD-High	0.16 ± 0.04	0.17 ± 0.12	0.22 ± 0.06
	HD-Low	0.20 ± 0.09	0.31 ± 0.26	0.19 ± 0.05
Percentage of time in deceleration (PTDP) (%)	HD-High	86.15 ± 2.06	84.10 ± 8.72	82.42 ± 4.81
	HD-Low	79.90 ± 10.85	69.74 ± 22.23	87.41 ± 6.80
Maximum hand velocity (cm/sec)	HD-High	114.5 ± 7.5	129.25 ± 30.34	147.98 ± 23.30
	HD-Low	87.47 ± 12.62	86.9 ± 38.76	103.55 ± 19.8
Number of velocity peaks	HD-High	3.67 ± 0.33	3.44 ± 0.77	3.56 ± 0.51
	HD-Low	3.78 ± 0.77	3.33 ± 0.58	6.44 ± 2.80

The curvilinear hand paths could have resulted from the shoulder and elbow moving sequentially. Analysis of joint motion onsets revealed a more sequential pattern of shoulder and elbow motion for the HD patients compared to the healthy subjects. It was observed that most of the HD patients clearly demonstrated the ability to move these joints simultaneously, which was most evident in the last phase of the movement. The curvilinear hand paths probably did not result from an inability to move the joints simultaneously. The sequential joint motions seen in the initial phase of movement were more likely a strategy employed by the HD patients, rather than a deficit in coordination of shoulder and elbow motion.

A second possible explanation for the curved hand paths is that the HD patients may have first brought their arm upward in order to decrease the initial postural perturbation, which would be greater if the arm were brought immediately outward. Initially bringing the hand upward lessens the immediate disturbance to the center of mass compared to a movement directly outward. Patients may have utilized this strategy to maximize their postural stability.

A third explanation is that the HD patients brought their hand upward in line with the target to permit direct visual monitoring of the hand. This approach may have reduced the variability in limb movement to an acceptable level, as Sheridan and Flowers (8) have proposed in Parkinson's disease. By having the hand in direct line of vision, assuming that vision is directed at the target, information about errors may be more easily ascertained.

Presence of Bradykinesia and Submovements

A second major finding of this study was that the HD patients had longer movement times compared to the healthy subjects. One possible explanation for longer duration movements was that the HD patients moved a further distance due to hand path curvature. Movement time was not systematically affected by changes in total distance in either healthy or HD patients. Distance alone could not have accounted for differences in movement time.

The mechanisms underlying bradykinesia in Parkinson's disease have been studied extensively (7,8,25). Hallett and Khoshbin (7) and Teasdale and colleagues (25) have suggested that bradykinesia results from multiple cycles of EMG triphasic activity. Hallett and Khoshbin (7) have reported that PD patients have difficulty generating forces adequate to reach a target in a single burst of movement, as healthy subjects can do. Similarly, Hefter and colleagues (2) have suggested that bradykinesia in Huntington's disease is a result of impaired speed

control mechanisms. These authors reported a marked prolongation in EMG activity before peak force was reached in isometric contraction of finger muscles. They concluded that bradykinesia in HD results from an inability of patients to increase their rate of rise of tension accordingly with increasing amplitude.

In the present study, force production deficits did not seem to underlie bradykinesia in the HD patients. The maximum velocities for the HD patients were similar to those of the healthy subjects. For the HD-high patients, the acceleration phase was generally smooth up to the maximum velocity, indicating that these patients were able to adequately activate the appropriate musculature to advance their limb forward toward the target. Deficits in the HD-high patients are more related to the deceleration phase, in which error correction mechanisms are employed. In contrast, HD-low patients exhibit deficits more similar to those described previously for PD patients (7,25). There were a number of trials for the HD-low patients in which the multiple velocity peaks occurred throughout the movement, rather than being concentrated at the end of the movement. The HD-low patients were unable to generate high velocities, particularly for movements to the far target. Lower functioning HD patients may produce lower velocity movements as a strategy to maximize their postural stability for movements that are particularly challenging due to incorporation of trunk motion.

Sheridan and Flowers (8) have disputed the belief that bradykinesia in PD results from a deficit in force production. These authors reported that PD patients have the ability to move fast, but by doing so their accuracy diminishes significantly. Sheridan and Flowers (8) have suggested that bradykinesia in Parkinson's disease may be the result of a behavioral adaptation to compensate for an inability to accurately direct large movements to a target, rather than a direct effect of basal ganglia dysfunction. HD and PD patients may be forced to increase the duration of their movements so that visual feedback can be utilized.

The presence of multiple velocity peaks, or submovements, have been reported in healthy subjects moving to targets with high accuracy (16,17). Meyer and colleagues (16) proposed a model for arm movements based on a relationship between the accuracy requirements and the prevalence of submovements. These authors proposed that the number of observed submovements can be predicted by the time, width, and distance of the target. They reported that the number of submovements increases inversely with target size. Similarly, Milner and Ijaz (17) found that the submovements corresponded to changes in the direction of the hand paths. This suggests that submovements can be used by healthy subjects for on-line error correction in order to

maximize accuracy as the hand hones in on the target. The presence of multiple peaks in the velocity profiles in the present study seem to be similar to the submovements described by Milner and Ijaz (17) and Meyer and colleagues (16).

In addition to small, error-correcting changes in hand path direction, HD patients produced movements that were highly curved in the sagittal plane. Curvilinear movements are associated with velocity profiles that have more than one peak (10,26). The curvilinear movements produced by HD patients could have contributed to alterations in the velocity profiles. The majority of submovements occurred toward the end of the movement and corresponded to changes in direction toward the end of the movement. Although curvilinear hand paths could have contributed to the multiple peaked velocity profiles for the HD patients, error-correcting movements seemed to contribute most to these changes.

The task in the present study may have required a degree of spatial precision and force control that was relatively higher for the HD patients than for healthy subjects. These constraints may have posed particular difficulties for HD patients. Inspection of hand velocity profiles revealed that the slowness occurred primarily in the deceleration phase, which was punctuated with submovements. HD patients may have used strategies similar to those of healthy subjects under high accuracy constraints. An increase in the time spent in deceleration and the presence of multiple submovements could have been a strategy employed by HD patients to improve their accuracy. These deficits may not be reflective of impairments in force control or an inherent inability of HD patients to move more quickly.

The slower movement times of the HD patients may have been due to difficulty decelerating their hand to a zero velocity. Teasdale and colleagues (25) have reported that while PD patients exhibited bradykinesia, they did not seem to have problems in the appropriate activation of the agonist muscle. These authors concluded that PD patients may use multiple cycles of triphasic activity throughout the movement in order to reduce the need for terminal control via antagonist muscle activity (25). The relaxation time of PD patients following isometric contractions was found to be greater than that of healthy subjects, particularly when patients were off medication (27). Corcos and colleagues (27) suggest that deficits in muscle deactivation may represent a component of bradykinesia. Difficulty in "braking," or decelerating the limb, would be reflected in the deceleration phase of the movement by an increase in time spent in this phase and an irregularity of movement. The prevalence of submovements in the deceleration phase for the HD patients in this study suggests that their problem is related to deceleration of the limb or in honing in on the target.

Deficits in Movements Requiring Trunk Motion

For the high functioning HD patients, movement requiring trunk motion did not appear to be more difficult than movement without trunk motion. HD-high patients were able to achieve similar maximum velocities as healthy subjects under all target distances. The HD-low patients showed particular difficulty with movements to the far distance target. Compared to HD-high patients, HD-low patients had low maximum velocities of the hand and produced a greater number of submovements for the far distance. This suggests that movements requiring a relatively large degree of trunk motion posed particular difficulty for lower functioning HD patients.

Thompson and colleagues (3) studied movements of HD patients performing a simple task of hand squeezing versus a more complex task involving simultaneous or sequential movements of hand squeezing and elbow flexing. While slowness of movement was noted for relatively simple single joint tasks, an increase in movement times was noted for the more complex simultaneous or sequential movements. Incorporation of trunk motion in the present study may have increased the task complexity by increasing the number of degrees of freedom the system must control. Such simultaneous movements requiring coordination of multiple joints may be particularly difficult for lower functioning HD patients (3). This difficulty may have caused the HD-low patients to move more slowly, particularly for the far distance condition.

Study Limitations

Kinematic analysis in Huntington's disease is difficult due to the complex and unpredictable nature of choreic movements. Although kinematic analysis was a particularly useful tool to analyze multijoint arm movements, the sampling rate used in this study was 30 Hz. Although this is just at an acceptable level for movement analysis (28), the sampling frequency limited further analysis of the complex submovements produced by the HD patients. The number of subjects used in this study is also a potential limitation, but all six HD patients had remarkably similar impairments across all target conditions. Although some differences were evident between higher and lower functioning patients, all HD patients produced curved hand paths and had prolonged, multimodal velocity profiles. Further examination of differences between high and low functioning HD patients are

beyond the scope of this study and certainly demand further investigation.

Summary

Presence of Curved Hand Paths and Submovements in HD

The reason for the curvilinear hand paths in HD patients remains unclear. It seems evident that it was not due to an inability to coordinate shoulder and elbow motion simultaneously. Curved hand paths may have been a strategy to minimize postural perturbations or to reduce variability by bringing the hand in direct line of vision with the target. The presence of curved hand paths and multiple submovements in the hand velocity profiles suggests that HD patients may rely more heavily on visual feedback than healthy subjects. The underlying mechanism for bradykinesia and submovements in Huntington's disease remains unknown, but it does not appear to be due to a deficit in force production. Slowing down movement of the limb as the hand approaches the target may be a behavioral adaptation used by HD patients in order to maximize their accuracy or may be a result of impairments in controlling deceleration of the limb. In contrast to higher functioning patients, movements that require incorporation of a relatively large degree of trunk motion are particularly difficult for lower functioning HD patients.

Implications for Role of Basal Ganglia

In basal ganglia diseases such as HD, it seems that there is a greater amount of variability in motor performance. As a result, individuals with HD may adopt a strategy of using ongoing feedback corrections to maximize their accuracy in performing goal-directed movements. As the progression of functional impairments occurs, individuals with HD may be less able to execute these strategies and may be more limited by deficits in control and coordination of posture and movement. The modulating role of the basal ganglia in the planning and execution of limb movements might be closely related to keeping a low level of variability in the motor system (29).

References

1. Bradshaw JL, Phillips JG, Dennis C, et al. Initiation and execution of movement sequences in those suffering from and at-risk of developing Huntington's disease. *J Clin Exp Neuropsychol* 1992;14(2):179–92.
2. Hefter H, Homberg V, Lange HW, Freund HJ. Impairment of rapid movement in Huntington's disease. *Brain* 1987;110:585–612.
3. Thompson PD, Berardelli A, Rothwell JC, et al. The coexistence of bradykinesia and chorea in Huntington's disease and its implications for theories of basal ganglia control of movement. *Brain* 1988;111:223–44.
4. Phillips JG, Bradshaw JL, Chiu E, Bradshaw JA. Characteristics of handwriting of patients with Huntington's disease. *Mov Disord* 1994;9(5):521–30.
5. Girotti F, Carella F, Scigliano G, et al. Effect of neuroleptic treatment on involuntary movements and motor performances in Huntington's disease. *J Neurol Neurosurg Psychiatry* 1984;47(8): 848–52.
6. Phillips JG, Chiu E, Bradshaw JL, Iansek R. Impaired movement sequencing in patients with Huntington's disease: a kinematic analysis. *Neuropsychologia* 1995;33(3):365–69.
7. Hallett M, Khoshbin S. A physiological mechanism of bradykinesia. *Brain* 1980;103(2):301–14.
8. Sheridan MR, Flowers KA. Movement variability and bradykinesia in Parkinson's disease. *Brain* 1990, 113:1149–61.
9. Montgomery EB, Nuessen J. The movement speed/accuracy operator in Parkinson's disease. *Neurology* 1990;40(2):269–72.
10. Abend, W, Bizzi, E, Morasso, P. Human arm trajectory formation. *Brain* 1982;105:331–48.
11. Atkeson CG, Hollerbach JM. Kinematic features of unrestrained vertical arm movements. *J Neurosci* 1985;5(9):2318–30.
12. Kaminski T, Gentile AM. Joint control strategies and hand trajectories in multijoint pointing movements. *J Motor Behavior* 1986;18(3):261–78.
13. Soechting JF, Lacquaniti F. Invariant characteristics of a pointing movement in man. *J Neurosci* 1981;1(17):710–20.
14. Woodworth RS. The accuracy of voluntary movement. *Psychol Rev* 1898;3:1–14.
15. Kaminski TR, Bock C, Gentile AM. The coordination between trunk and arm motion during pointing movements. *Exp Brain Res* 1995;106:457–66.
16. Meyer DE, Smith JEK, Kornblum S, Abrams RA, Wright CE. Speed-accuracy tradeoffs in aimed movements: toward a theory of rapid voluntary action. In: Jeannerod M (ed.). *Attention and performance XIII.* Hillsdale, NJ: Erlbaum, 1990:173–226.
17. Milner TE, Ijaz MM. The effect of accuracy constraints on three-dimensional movement kinematics. *Neuroscience* 1990;35(2): 365–74.
18. Flash T, Inzelberg R, Schechtman E, Korczyn AD. Kinematic analysis of upper limb trajectories in Parkinson's disease. *Exp Neurol* 1992;118(2):215–26.
19. Bernstein N. *The coordination and regulation of movements.* Oxford: Pergamon, 1967.
20. Folstein SE. *Huntington's disease: a disorder of families.* Baltimore: The Johns Hopkins University Press, 1989.
21. Wade DT, Collin C. The Barthel ADL Index: a standard measure of physical disability. *Int Disabil Stud* 1988;10(2):64–67.
22. Jagacinski RJ, Repperger DW, Moran MS, Ward SL, Glass B. Fitts' law and the microstructure of rapid discrete movements. *J Exp Psychology Hum Percept Perform* 1980;6(2):309–20.
23. Judd CM, McClelland GH. *Data analysis: a model-comparison approach.* San Diego: Harcourt Brace Jovanovich, 1989.

24. Howell DC. *Statistical methods for psychology*, 3rd ed. Belmont: Duxbury Press, 1992:611–17.
25. Teasdale N, Phillips J, Stelmach GE. Temporal movement control in patients with Parkinson's disease. *J Neurol Neurosurg Psychiatry* 1990;53(10):862–8.
26. Lacquaniti F, Terzuolo C, Viviani P. The law relating the kinematic and figural aspects of drawing movements. *Acta Psychol* 1983;54(1–3):115–30.
27. Corcos DM, Chen CM, Quinn NP, McAuley J, Rothwell JC. Strength in Parkinson's disease: relationship to rate of force generation and clinical status. *Ann Neurol* 1996;39(1):79–88.
28. Winter, DA (1990). *Biomechanics and motor control of human movement*, 2nd ed. New York: John Wiley & Sons, Inc.
29. Alexander GE, Crutcher MD. Functional architecture of basal ganglia circuits: neural substrates of parallel processing. *Trends Neurosci* 1990;13(7):266–71.