

The reaching movements of patients with Parkinson's disease under self-determined maximal speed and visually cued conditions

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Summary

Two-dimensional kinematic analysis was performed of the reaching movements that six subjects with Parkinson's disease and six healthy subjects produced under self-determined maximal speed and visually cued conditions. Subjects were required to reach as fast as possible to grasp a ball (i) that was fixed stationary in the centre of a designated contact zone on an inclined ramp (self-determined maximal speed condition), or (ii) that rolled rapidly from left to right down the incline and into the contact zone (visually cued condition). Parkinson's disease subjects displayed bradykinesia when performing maximal speed reaches to the stationary ball, but not when they reached for the moving ball. In response to the external driving stimulus of the moving ball, Parkinson's disease subjects showed the ability to exceed their self-determined maximal speed of reaching and still maintain a movement

accuracy that was comparable to that of healthy subjects. Thus, the bradykinesia of Parkinson's disease subjects did not seem to be the result of a basic deficit in their force production capacity or to be a compensatory mechanism for poor movement accuracy. Instead, bradykinesia appeared to result from the inability of Parkinson's disease subjects to maximize their movement speed when required to internally drive their motor output. The occasional failure of Parkinson's disease subjects to successfully grasp the moving ball suggested errors of coincident anticipation and impairments in grasp performance rather than limitations in the speed or accuracy of their reaches. These results are discussed in relation to the notion that the motor circuits of the basal ganglia play an important role in the modulation of internally regulated movements.

Keywords: Parkinson's disease; basal ganglia; bradykinesia; reaching; visual cues

Abbreviation: RMSE = root mean square error

Introduction

One of the most prevalent motor signs of Parkinson's disease is a slowness of movement referred to as bradykinesia. Individuals with Parkinson's disease have been reported to exhibit bradykinesia when attempting to perform large, ballistic movements (Flowers, 1975, 1976; Hallett and Khoshbin, 1980), when performing movements that require high degrees of accuracy (Sanes, 1985; Teasdale and Stelmach, 1988; Montgomery and Nuessen, 1990; Sheridan and Flowers, 1990) and when tracking moving targets (Flowers, 1978*a, b*; Hufschmidt and Lucking, 1995). Although several proposals have been offered as explanations for why Parkinson's disease subjects move slowly, consensus

on a single, unifying mechanism for bradykinesia has not been achieved.

The hypothesis that bradykinesia results from a depressed magnitude of force production has been derived from observations that Parkinson's disease subjects display low levels and inefficient patterns of motor unit activity when they attempt to move quickly (Hallett and Khoshbin, 1980; Godaux *et al.*, 1992; Glendinning and Enoka, 1994) or perform isometric motor tasks (Wiesendanger, 1978; Stelmach *et al.*, 1989; Corcos *et al.*, 1996). However, a number of studies have shown that bradykinesia also arises from the failure of Parkinson's disease subjects to optimally

modulate their motor output in relation to the spatial and temporal constraints of motor tasks, rather than simply from a basic deficit in the ability to generate high levels of agonist muscle activity (Berardelli *et al.*, 1986; Teasdale *et al.*, 1990). Sheridan and Flowers (1990) suggested that individuals with Parkinson's disease decrease movement speed, shorten movement amplitude and increase their use of visual feedback as compensatory mechanisms for a motor system with inherently high variability of motor output. This proposal provides a second hypothesis for bradykinesia: that individuals with Parkinson's disease adopt a behavioural strategy of moving slowly in order to maintain their accuracy. The notion that bradykinesia results from an excessive trade-off of movement speed for accuracy is supported by reports that Parkinson's disease subjects show disproportionately high compromises in movement speed when they perform motor tasks that contain strict accuracy constraints (Sanes, 1985; Montgomery and Nuessen, 1990). Parkinson's disease subjects appear to have the ability to perform large, fast movements, but only at an unusually high cost to their movement accuracy (Sheridan and Flowers, 1990; Phillips *et al.*, 1994).

The bradykinesia of Parkinson's disease subjects cannot be fully explained as a compensatory mechanism for poor movement accuracy. Bradykinesia has been shown to persist when the spatial accuracy constraints of tasks are removed (Sheridan *et al.*, 1987; Teasdale *et al.*, 1990). In addition, recent studies have revealed that Parkinson's disease subjects display movement anomalies during the performance of motor tasks at slow, preferred speeds as well as at fast speeds (Isenberg and Conrad, 1994; Phillips *et al.*, 1994). Irregularities appear not only in the deceleration phase of movements, which would be expected if bradykinesia were being used to compensate for poor movement accuracy, but also in the initial acceleration phase of movements. Thus, in addition to a behavioural strategy for improving movement accuracy, bradykinesia may result from a basic deficit that individuals with Parkinson's disease have in their ability to internally organize their motor output to optimize their performance of motor tasks.

Flowers (1975, 1976) proposed that individuals with Parkinson's disease were unable to use internal references of the spatial and temporal constraints of motor tasks to generate accurate movements based upon anticipatory or feedforward means of control. He showed that Parkinson's disease subjects did not use fast, ballistic movements to track a target that moved along a predictable, jagged path. Instead, the subjects tracked the target through slower, feedback-mediated movements that were visually guided (Flowers, 1978*a, b*). Subsequent studies have shown that although Parkinson's disease subjects are able to formulate predictive motor strategies (Bloxxham *et al.*, 1984; Day *et al.*, 1984; Schnider *et al.*, 1995), they do not generate and co-ordinate fast, accurate movements within sequential or repetitive movement tasks (Benecke *et al.*, 1986, 1987*a, b*; Castiello *et al.*, 1993;

Agostino *et al.*, 1994; Bennett *et al.*, 1995; Hufschmidt and Lucking, 1995).

The hypothesis tested in the present study is that individuals with Parkinson's disease display bradykinesia because they are unable to internally drive or optimally modulate their motor output when the speed of their movements is self-regulated. This hypothesis is supported by evidence that Parkinson's disease subjects are able to perform repetitive and sequential motor tasks faster in the presence of external temporal cues than when the maximal speed of their movements is self-determined (Cooke *et al.*, 1978; Freeman *et al.*, 1993; Georgiou *et al.*, 1993; Morris *et al.*, 1994, 1995; Thaut *et al.*, 1996). For example, Morris *et al.* (1994, 1995) and Thaut *et al.* (1996) demonstrated that visual and auditory temporal cues could be used to increase the cadence and gait velocity of bradykinetic Parkinson's disease subjects. Georgiou *et al.* (1993) showed that Parkinson's disease subjects were able to use auditory timing cues to increase the speed at which a previously learned upper limb movement sequence was performed.

The effect of visual and auditory temporal cues on the movement speed of Parkinson's disease subjects has been reported only for sequential and repetitive movement tasks that have required subjects to perform a series of pointing, tracking or stepping movements in rapid succession. External temporal cues have been used to test the ability of Parkinson's disease subjects to increase their movement speed, but have not been used to test the maximal speeds that these subjects are capable of producing. Freeman *et al.* (1993) and Georgiou *et al.* (1993) showed that Parkinson's disease subjects performing repetitive or sequential movements in the presence of auditory timing cues did not move as fast as healthy adults. However, experimental tasks which require the alternation or switching of movements in rapid succession may not provide as accurate a measure of the maximal speed capabilities of Parkinson's disease subjects as single motion motor tasks (Bloxxham *et al.*, 1984; Benecke *et al.*, 1987*a, b*).

Previous studies that have reported that Parkinson's disease subjects are not able to perform unidirectional, maximal speed movements as fast as healthy adults (Hallett and Khoshbin, 1980; Berardelli *et al.*, 1986; Montgomery and Nuessen, 1990; Flash *et al.*, 1992; Godaux *et al.*, 1992; Muller and Stelmach, 1992) have not tested subjects in the presence of external temporal cues. Subjects have been forced to self-determine and internally drive their maximal speed movements while moving to a fixed target (Hallett and Khoshbin, 1980; Berardelli *et al.*, 1986; Montgomery and Nuessen, 1990; Flash *et al.*, 1992) or while reaching to grasp a stationary object (Muller and Stelmach, 1992). The purpose of this study was to determine whether Parkinson's disease subjects, when provided with a visuotemporal stimulus, are able to exceed their self-determined maximal speed of reaching without compromising their movement accuracy. If individuals with Parkinson's disease exhibit bradykinesia because they are unable to drive their motor output internally, the introduction of an external temporal stimulus would be

expected to promote faster movements. Any increases in movement speed that result should be accompanied by losses in movement accuracy if bradykinesia is associated with a speed/accuracy trade-off.

In this study we analysed the movement behaviour of healthy and Parkinson's disease subjects under two task conditions. Subjects were first instructed to reach as fast as possible to grasp a stationary ball that was fixed within a contact zone. This condition required subjects to use their own internal drive to generate a self-determined, maximal speed reach. In the second condition, subjects were instructed to grasp the ball as it rapidly rolled through the contact zone. The moving ball provided a visuotemporal stimulus that required subjects to increase their reaching speed if they were to successfully grasp the ball. Comparing the reaching behaviour of subjects under these two conditions allowed us to determine whether Parkinson's disease subjects were able to move faster when their movement speed was externally rather than internally driven.

Method

Subjects

Six adults diagnosed with stage 3 (Hoehn and Yahr, 1967) Parkinson's disease and six healthy control subjects with no history of neurological disorder volunteered for the study. Each group comprised four males and two females between the ages of 64 and 74 years. All subjects were right-hand dominant, had vision corrected to 20/20, and had a level of cognition sufficient to understand the requirements of the experimental task. Each of the Parkinson's disease subjects was on a daily regimen of Sinemet[®], which was continued during this study. Parkinson's disease subjects did not show any evidence of resting tremor or dyskinesia during the testing period. Prior to their participation, all subjects were fully informed about the nature and procedures of the experiment and gave their consent to be tested. The study was approved by the review board of Columbia Presbyterian Hospitals, Columbia University, New York City, USA.

Task and apparatus

The experimental task required seated subjects to reach and grasp a ball (6.8 cm in diameter) as fast as possible under two conditions. In the first condition, the ball was placed and remained stationary in a specified contact zone (10 cm in length) that was located in the middle of an inclined track (233×2.5 cm). In the second condition, the ball rolled from behind a barrier, left to right down the inclined track and through the contact zone. The designation of a fixed contact zone ensured that subjects grasped the ball from the same track location under both task conditions.

The track was positioned parallel to the frontal plane of subjects at a distance equal to 80% of arm length. Pilot work indicated that setting the track at a 20° angle of inclination

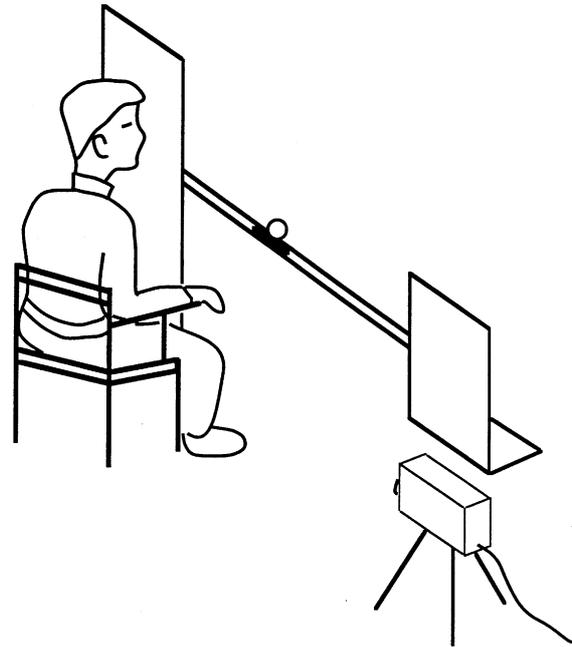


Fig. 1 Experimental setup. Subjects reached as fast as possible to grasp a ball that was either fixed on an inclined ramp in the centre of a 10 cm contact zone (blackened area) or rolled from behind a barrier, left to right down the ramp and into the contact zone.

produced a moving ball velocity that challenged the maximal reaching speed of Parkinson's disease subjects, but still made ball grasp possible. At this angle, the ball moved at a velocity of 130 cm/s as it first appeared from behind the barrier, accelerating to 210 cm/s by the time it entered the contact zone. The track length from the edge of the barrier to the contact zone was 71 cm, which provided a moving ball viewing time of 400 ms. A track height was used that placed the contact zone approximately at the mid-trunk level of the subject. The position of the track in the frontal plane was positioned such that the contact zone was located directly in front of the right upper limb. This position allowed subjects to perform the reaching task within a comfortable reaching workspace, but confined their reaches to the parasagittal plane. A video camera (Panasonic 500SX) positioned to the right of subjects at a distance of 2 m was used to record reaching movements (Fig. 1).

Procedure

Subjects sat in a chair with their right arm on the armrest. The shoulder was positioned at 0° of flexion, the elbow at 90° of flexion, and the forearm in full pronation. A 1.5×1.5 cm reflective marker was placed on the ulnar styloid for the collection of wrist trajectory data. Subjects familiarized themselves with the task by performing six practice trials, using a self-selected, preferred reaching speed to grasp a stationary ball from the contact zone. Subjects were then tested for six trials under each of three task conditions: (i) reach and grasp the stationary ball as fast as possible; (ii)

reach and grasp the moving ball as it passed through the contact zone; and (iii) repeat the reach and grasp of the stationary ball as fast as possible. Data from the second stationary ball condition were used to assess whether there were carryover effects from the moving ball condition to subsequent reaches to a stationary ball. In the moving ball condition, the velocity of the ball and the brief time that the ball was available for grasp inherently required a great deal of attention from subjects. Under stationary ball conditions, subjects were verbally cued after every other trial to reach as fast as possible, to ensure that their attention was similarly focused on the performance of fast, accurate movements. To guard against the possibility that reaching to a moving ball would bias the baseline measures of reaching to a stationary ball, the moving ball condition was presented to subjects in the second block of reaching trials. A 20–30 s rest was provided between trials, resulting in a total testing time of ~30 min.

Data processing and analysis

The wrist marker was manually digitized at a rate of 60 Hz using Kinematic Analysis software (Schliehauf, Scarsdale, NY, USA). The digitized wrist position data were processed through a 4th order dual-pass Butterworth digital filter with a low-pass cutoff frequency of 8 Hz, and used to compute wrist paths and velocities.

The onset of reach was defined as the time when the forward wrist movement of subjects exceeded a criterion velocity of 2.5 cm/s for three successive samples. Reach termination was the time at which the metacarpal/phalangeal joints of the hand crossed the frontal plane of the contact zone, which coincided with the time of ball contact. Because the main focus of this study was the effect of a visuotemporal cue on the maximal speed reaching movements of Parkinson's disease subjects, the kinematics of ball grasp was not analysed.

Based on the findings of pilot work, it was expected that Parkinson's disease subjects would occasionally perform a reach during the moving ball trials that would be initiated prior to the appearance of the ball or would be initiated too late for ball contact to occur. Therefore, all subjects performed six trials under each of the task conditions to ensure that data from at least five correctly executed trials were collected. To avoid uneven sample sizes, when a subject performed all six trials of a task condition appropriately, movement analysis was limited to the five trials which showed the greatest similarity in velocity profile.

The moving ball velocity that was used in this study was intended to challenge the maximal reaching speed of Parkinson's disease subjects, but not surpass it. It was anticipated that Parkinson's disease subjects would perform a number of reaching trials in which the moving ball was contacted but not successfully grasped. The frequencies of ball contact with and without successful grasp were recorded,

to determine whether a correlation existed between the success or failure of ball grasp and the kinematics of reaching.

The movement times of subjects were determined from the video tape and wrist velocity data. The kinematic variables of peak velocity and time to peak velocity were calculated from the wrist velocity data, and were used to analyse the acceleration phase of reaches. Analysis of the deceleration phase of reaches included examination of the contact velocity of the wrist at reach termination and the percentage of movement time spent in deceleration. The mean for each of these kinematic variables was computed for the five trials of subjects within each task condition. The pattern of group means across trials and task conditions were analysed using a 2 (group) × 3 (task condition) × 5 (trials) repeated measures analysis of variance mixed factorial design. Tukey's Honestly Significant Difference multiple comparison test was used for *post hoc* testing of differences between groups and task conditions ($P < 0.01$).

The wrist paths of subjects were plotted for descriptive analysis. The x and y coordinates of the wrist marker at ball contact were recorded and used to assess the spatial accuracy of reaching. The mean x and y coordinates of the wrist were computed for each subject for each task condition. The differences between the x and y coordinates for each reaching trial and the mean x and y coordinates were used to calculate the root mean square reaching error (RMSE) of each subject within each task condition. Group means of RMSE were analysed using a 2 (group) × 3 (task condition) repeated measures analysis of variance mixed factorial design.

Results

Reaching speed

The wrist tangential velocity profiles of Parkinson's disease and healthy subjects were invariably unimodal and bell-shaped from movement initiation to ball contact. As illustrated by the representative wrist velocity profiles presented in Fig. 2, all subjects produced single, ballistic reaching movements, regardless of whether the speed of their movement was self-determined or externally cued.

Differences were observed between the reaching behaviour of Parkinson's disease and healthy subjects across task conditions. As shown in Fig. 3A–C, Parkinson's disease subjects reached more slowly than healthy subjects when the ball was stationary, but not when the ball was in motion. Group differences varied across task conditions, resulting in significant group × task condition interaction effects for peak velocity [$F(2,20) = 5.66, P = 0.011$], time to peak velocity [$F(2,20) = 8.20, P < 0.01$] and movement time [$F(2,20) = 5.96, P < 0.01$]. For the percentage of time spent in deceleration and contact velocity, no interaction effects were found; only task condition was statistically significant [$F(2,20) = 6.25, P < 0.01$ and $F(2,20) = 9.66, P < 0.01$, respectively]. Thus, the slower movements of Parkinson's disease subjects under the stationary ball conditions appeared

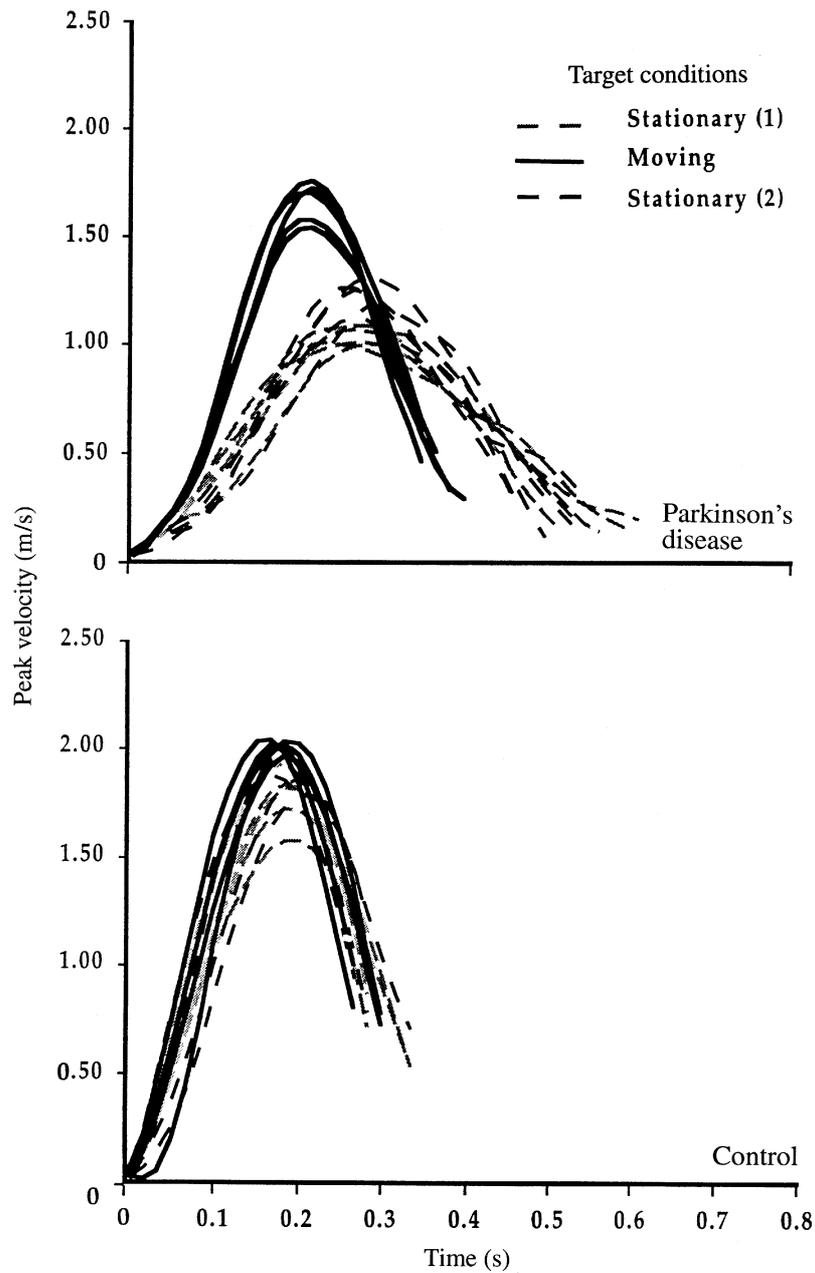


Fig. 2 Wrist velocity profiles of one representative Parkinson's disease subject (PD3) and one healthy control subject (C1) reaching under the three conditions. Parkinson's disease subjects reached faster to the moving than to the stationary ball. Healthy controls showed no difference in reaching speed between the conditions.

to be related to impairments in the acceleration phase of reaching rather than to changes in movement deceleration. The absence of main effects or interaction effects between trial order and the kinematic variables that were analysed demonstrated that the pattern of reaching that each group demonstrated was not affected by practice. Comparisons of group means confirmed that under the stationary ball conditions Parkinson's disease subjects reached with a lower peak velocity, a longer time to peak velocity and a longer movement time than healthy subjects.

Under the moving ball condition there were no significant

differences between Parkinson's disease and healthy subjects on any kinematic measures. Despite exhibiting bradykinesia under stationary ball conditions, Parkinson's disease subjects were able to increase their peak velocity, shorten their time to peak velocity and shorten their movement time in response to the moving ball, and match the reaching speed of healthy subjects. The peak velocity of Parkinson's disease subjects increased by 61% under the moving ball condition, time to peak velocity decreased by 25% and movement time decreased by 39%. In contrast, no differences were shown in the time to peak velocity or movement time of healthy

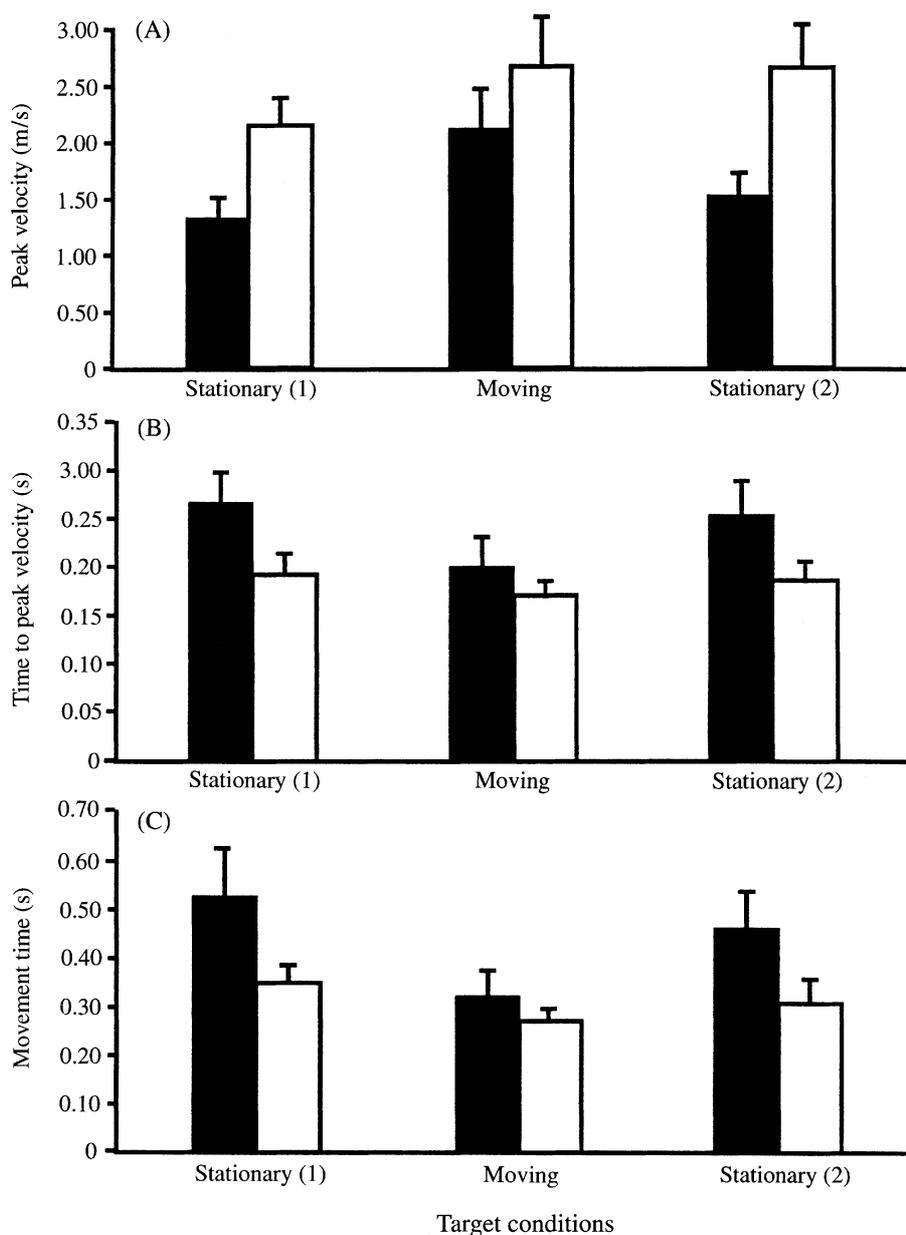


Fig. 3 Group means and standard deviations of the peak velocity (A), time to peak velocity (B) and movement time (C) of Parkinson's disease subjects (filled bars) and healthy controls (open bars) under each of the three task conditions. See text for discussion.

subjects between stationary and moving ball conditions. Healthy subjects did, however, show an increase in peak velocity from the initial stationary ball condition to the moving ball condition. When returning to the performance of maximal speed reaching to a stationary ball, the peak velocity of healthy subjects remained higher than in their initial attempts, and not statistically different from their reaches to the moving ball. In contrast, Parkinson's disease subjects did not carry over the increased speed set of the moving ball condition to the second set of stationary ball reaches. When they returned to self-paced, maximal speed reaching in a stationary ball condition their reaches significantly slowed, with lower peak velocity, longer time

to peak velocity and longer movement time. These findings provided evidence that Parkinson's disease subjects were not able to reach as fast when they were forced to drive their motor output internally as when they reached in response to an external temporal stimulus. Furthermore, the effects that resulted from reaching in response to the visuotemporal stimulus of the moving ball did not transfer to internally driven movements.

Reaching accuracy

Under both stationary and moving ball conditions, all subjects used a variety of wrist paths and wrist orientations to grasp

the ball within the contact zone. Subjects tended to maintain the wrist in full pronation and grasp the ball with an overhand approach under stationary ball conditions. When the ball was in motion, subjects supinated their wrists and used a side approach to grasp the ball. Because supination changed wrist marker orientation, the wrist trajectory and final wrist position of subjects appeared to be slightly lower in the parasagittal plane for moving than for stationary ball reaches. This change in wrist orientation and the tendency of subjects to contact the moving ball in a range of locations within the contact zone was reflected by a slightly higher variability of the x and y coordinates of the wrist at ball contact in the moving ball condition than under stationary ball conditions ($P = 0.062$). The wrist paths of a representative Parkinson's disease subject and a healthy control subject are illustrated in Fig. 4.

The spatial accuracy of the reaches of Parkinson's disease subjects was not shown to be different from the accuracy of healthy controls. No group differences were found in the RMSE data of the x and y coordinates of the wrist at ball contact, and group \times task condition interaction effects were not present. Thus, the increased reaching speed shown by Parkinson's disease subjects in the moving ball condition was not associated with an excessive compromise in the spatial accuracy of their reaches.

If the criterion for accurate reaching is defined by successful ball grasp, then the reaching accuracy of Parkinson's disease subjects might appear to have been impaired under the moving ball condition. Healthy subjects successfully grasped the ball in each task condition. In contrast, although Parkinson's disease subjects successfully performed ball grasp under stationary ball conditions, they contacted but frequently failed to grasp the ball when it was moving. Of the five moving ball trials analysed for each subject, four Parkinson's disease subjects had two misses, one subject had one miss and one subject had misses on every trial. The wrist trajectory data presented in Fig. 5A and B illustrates, however, that the failure of Parkinson's disease subjects to successfully grasp the moving ball was not the result of aberrant wrist paths or notably different positions of the wrist at ball contact. The RMSE data of the x and y coordinates of the wrist during ball contact for missed attempts were not greater than those that Parkinson's disease subjects showed during successful grasps. Similarly, no relationships were found between failure of ball grasp and any of the kinematic variables that were analysed. It appeared that the failure of Parkinson's disease subjects to successfully grasp the ball was related to errors in the timing of their reaches in relation to the moving ball, and possibly from impairments in the mechanics of grasp, rather than to decreased moving speed or a compromise in reaching accuracy.

Discussion

The bradykinesia that Parkinson's disease subjects displayed when they performed self-determined, maximal speed reaching movements to a stationary ball was not present

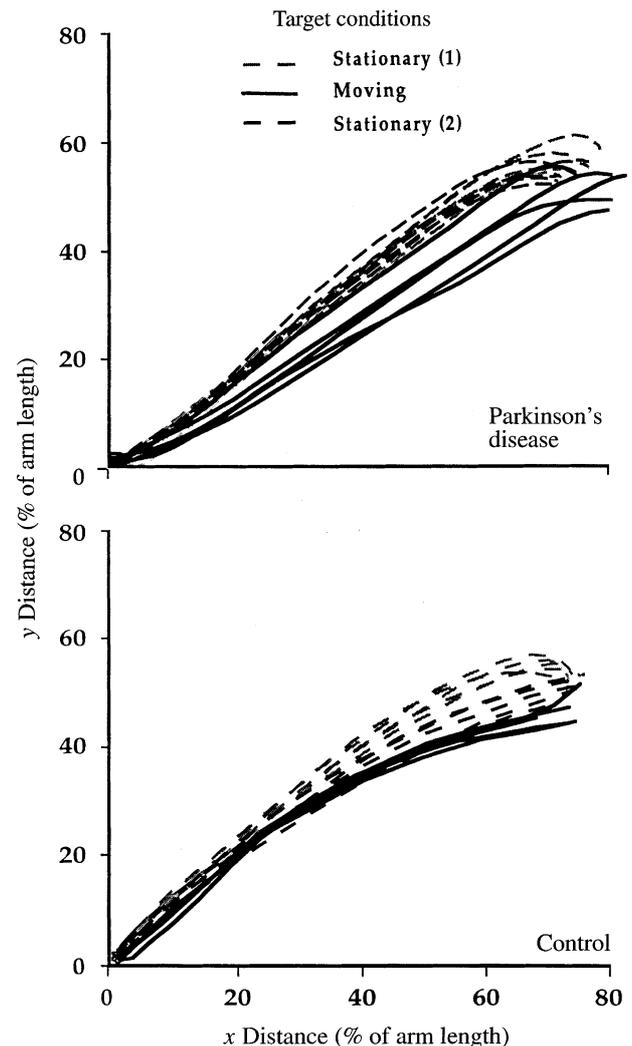


Fig. 4 Wrist paths of one representative Parkinson's disease subject (PD3) and one healthy control (C1) subject under each of the three task conditions. All subjects showed a dispersion of wrist paths around the contact zone which reflected not only the variability of the flight of the wrist to the contact zone but also the different wrist orientations that were used to grasp the ball. The wrist paths of Parkinson's disease subjects and their wrist positions at ball contact were comparable to those of healthy controls.

when they moved to intercept a moving ball. In the presence of a visuotemporal stimulus, Parkinson's disease subjects were able to generate reaching velocities that exceeded their self-regulated maximal speed and that matched the speed of healthy subjects. Furthermore, Parkinson's disease subjects increased their reaching speed without a notable compromise in movement accuracy. These findings dispute the notions that bradykinesia results merely from a basic deficit in force production capacity, or that individuals with Parkinson's disease must excessively decrease their movement speed to maintain the spatial accuracy of their movements. We offer our data as evidence that individuals with Parkinson's disease display bradykinesia during the performance of self-regulated motor tasks because they have a basic deficit in their ability

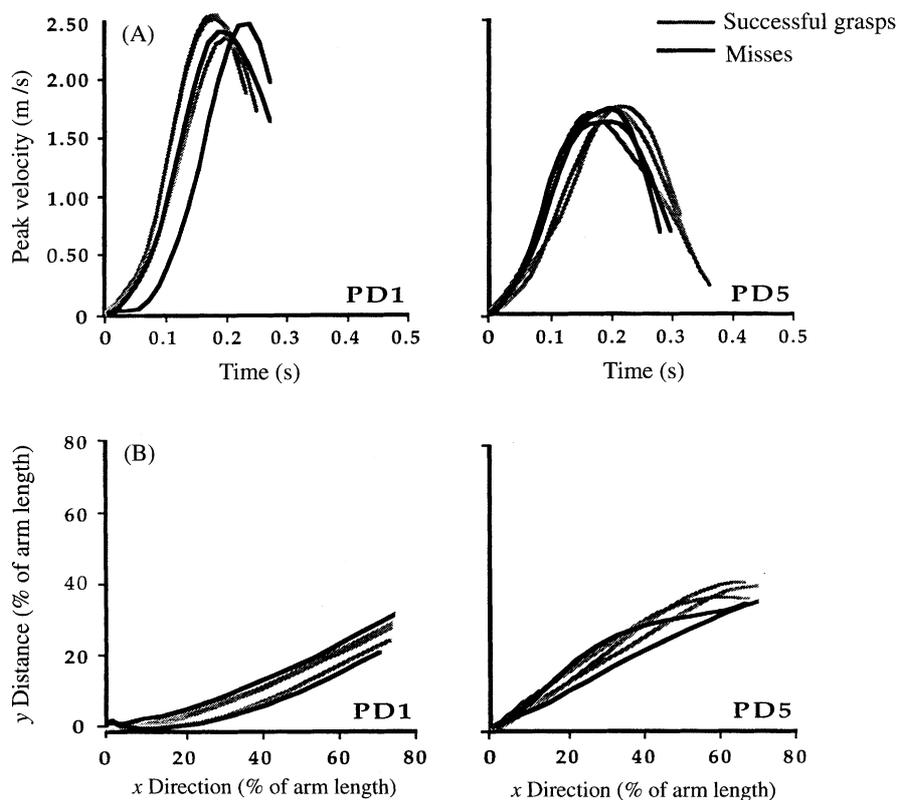


Fig. 5 Successful grasps and misses of Parkinson's disease subjects. **(A)** Wrist velocity profiles of two representative Parkinson's disease subjects (PD1, PD5). **(B)** Wrist paths of the same two Parkinson's disease subjects. The successful grasps and misses of Parkinson's disease subjects were not distinguished by differences in the kinematics of reaching.

to drive their motor output internally in the absence of external temporal cues.

If bradykinesia was directly related to impairments in force production, a change in task conditions should not have affected the maximal speed movements of Parkinson's disease subjects. However, under the moving ball condition Parkinson's disease subjects increased the peak velocity and acceleration of their movements and decreased their movement time to values that were comparable to those of healthy subjects. The lack of a distinct relationship between deceleration time, contact velocity and movement time indicated that Parkinson's disease subjects moved faster by increasing the force and acceleration of their movements rather than by altering their movement deceleration. Parkinson's disease subjects were able to produce a wide range of movement speeds but appeared to be particularly sensitive to the conditions under which their motor behaviour was tested.

These findings do not imply that individuals with Parkinson's disease have the full capacity to move as fast as healthy adults. The fastest reaching speed required of subjects in this study was defined by the time available to grasp the moving ball. Although a ball velocity was used that challenged the maximal reaching speed of Parkinson's disease subjects, it may not have forced healthy subjects to move at their own maximal reaching speed. This suggestion is corroborated by

the fact that healthy subjects produced reaching speeds under the stationary ball conditions that were comparable to the reaching speeds required for successful grasp of the moving ball. Unfortunately, the present experimental design did not allow us to test whether healthy subjects could have moved at speeds that Parkinson's disease subjects were unable to match. It is conceivable that individuals with Parkinson's disease have lower limits of maximal movement speed than healthy individuals regardless of task conditions. Nonetheless, none of the Parkinson's disease subjects in this study appeared to display a depressed capacity of force production when their movements were externally driven.

Our results suggest that the bradykinesia of individuals with Parkinson's disease does not simply represent an excessive trade-off of movement speed for movement accuracy. Parkinson's disease subjects demonstrated the ability to exceed their self-determined maximal speed of reaching under the moving ball condition and still intercept the ball in the contact zone. Although one Parkinson's disease subject repeatedly failed to grasp the moving ball, the remaining five subjects successfully performed ball grasp at least 50% of the time. Although the inability of Parkinson's disease subjects to grasp the moving ball consistently might be interpreted as a deficit in their movement accuracy, the reaching kinematics, wrist paths and terminal wrist positions of Parkinson's disease subjects were relatively similar across

the moving ball trials, and were comparable to those of healthy subjects. Thus, the failure of Parkinson's disease subjects to grasp the moving ball did not appear to be the result of decreased movement speed or poor movement accuracy. Failure of ball grasp may have been the result of errors in the timing of reaches, impaired co-ordination of reach and grasp or inefficient patterns of prehension. The moving ball condition may have driven the reaching speed of Parkinson's disease subjects, but not the timing or speed of their grasp. We were not able to assess the grasp kinematics of subjects within the present experimental design, but evidence that Parkinson's disease subjects show delays in hand opening and insufficient grip aperture in reaching tasks has been reported elsewhere (Muller and Stelmach, 1992; Castiello *et al.*, 1993; Scarpa and Castiello, 1994; Bennett *et al.*, 1995). At the present time, additional study is under way to assess the reach and grasp kinematics of Parkinson's disease subjects during the performance of self-determined maximal speed and externally cued prehension tasks, with a particular focus on the analysis of the coordination of reach and grasp during the acquisition of rapidly moving objects.

Our finding that Parkinson's disease subjects were able to increase their reaching speed in the moving ball condition without compromising their reaching accuracy may have reflected the relatively liberal spatial accuracy constraint that was defined within the task by the 10 cm contact zone. Previous studies investigating the accuracy of the maximal speed movements of Parkinson's disease subjects have used spatial targets ranging from single points and lines to circular targets as large as 3.8 cm in diameter (Flowers, 1976; Montgomery and Nuessen, 1990; Sheridan and Flowers, 1990; Flash *et al.*, 1992). The width of the contact zone that was used in this study was felt to be necessary in order for subjects to have a reasonable chance of grasping the moving ball within a spatially defined area. Subjects were able to contact the moving ball across a range of points within the contact zone, which may have decreased the sensitivity of our measure of accuracy. However, the similarities that Parkinson's disease subjects and healthy controls showed in wrist paths and in the dispersion of x and y coordinates of the wrist at ball contact suggested that any changes in reaching accuracy that occurred between subjects or task conditions were relatively small.

The observation that Parkinson's disease subjects were bradykinetic during self-regulated maximal speed reaching but not when they reached in response to a visuotemporal cue illustrated that their movement behaviour was strongly tied to the constraints of the task being performed. We offer three possibilities for why Parkinson's disease subjects reached faster under the moving ball than under the stationary ball condition. First, the slower reaching speeds generated by Parkinson's disease subjects under self-determined maximal speed conditions could have reflected a behavioural strategy. Parkinson's disease subjects may have placed a higher priority on successful ball grasp than on movement speed when their movements were self-paced. The moving ball condition

directly forced subjects to move fast if they were to catch the ball successfully. Although Parkinson's disease subjects were consistently able to contact the ball when it was in motion, their attempts to grasp the moving ball occasionally failed. Thus, when given the opportunity to reach more slowly under the stationary ball conditions Parkinson's disease subjects may have intentionally returned to a reaching speed that guaranteed task success 100% of the time. The maximal speed movements of individuals with Parkinson's disease may not simply be a function of the spatial constraints of motor tasks, but may also reflect how these individuals prioritize the speed and accuracy components of the task they are performing.

It might be argued that Parkinson's disease subjects were able to reach faster under the moving ball condition because they increased their allocation of attentional resources to the reaching task when the ball was in motion, compensating for the disruption of basal ganglion motor function through the use of attentional cortical control mechanisms. Parkinson's disease subjects have been reported to show improvements in motor performance when they strongly focus their attention on the execution of a functional task (Morris *et al.*, 1995). However, the design and findings of our study allow us to reject this notion. Prior to every other trial of the stationary ball conditions, subjects were verbally reminded and encouraged to reach as fast as possible. Although verbally cueing subjects in this manner did not guarantee that their attention would be as high in the stationary ball conditions as it was in the moving target condition, subjects were certainly urged to focus their attention on the reaching task when attempting to generate their maximal speed movements. Because this study allowed subjects to initiate their stationary ball reaches at their own discretion, the slow reaches of Parkinson's disease subjects cannot be attributed to a lack of preparation time. It is reasonable to assume that if greater attentional resources were allocated to reaching in the moving ball condition an increase in attention would have occurred after the first trial of the moving ball condition. Only then would subjects have realized that they needed to increase their attention to the task in order to successfully grasp the ball. If Parkinson's disease subjects had raised their attention in response to the moving ball, we would have expected them to maintain this higher level of attention, at least through the initial trials of the stationary ball condition that followed. Thus, differences in attention should have been evident from interaction effects between group, task condition and trials. No evidence of these interaction effects were found.

A second explanation for the fast reaching behaviour of Parkinson's disease subjects under the moving ball condition is that Parkinson's disease subjects may be able to use external temporal cues to organize the timing and speed of their movements. Previous evidence that external temporal cues can be used to increase the movement speed of Parkinson's disease subjects has been reported by Teasdale *et al.* (1990), who showed that Parkinson's disease subjects were able to exceed the speed of their self-determined

maximal speed movements by 10% when they were trained with verbal temporal feedback. Whereas Teasdale showed that the speed of Parkinson's disease subjects could be indirectly influenced by providing temporal feedback after individual task trials, external cues appear to have a greater effect when they are contained within the task itself. This notion is supported by reports that Parkinson's disease subjects are sensitive to visual and auditory temporal cues within repetitive and sequential motor tasks (Cooke *et al.*, 1978; Freeman *et al.*, 1993; Georgiou *et al.*, 1993; Morris *et al.*, 1994, 1995; Thaut *et al.*, 1996). Although these earlier studies showed that Parkinson's disease subjects were able to increase their movement speed moderately in response to external cues, the Parkinson's disease subjects of the present study were able to increase their peak velocity of reaching by an average of 61% and decrease their movement time by an average of 39% in the presence of a visuotemporal stimulus. Thus, the effect that external sensory stimuli have on the motor behaviour of individuals with Parkinson's disease appears to depend to some degree on the type of task that is being performed and the manner in which temporal cues are provided.

Our findings suggest that bradykinesia reflects a deficit in internal motor drive: the inability of individuals with Parkinson's disease to efficiently modulate their motor output and maximize their movement speed during self-regulated motor tasks. The inability of Parkinson's disease subjects to formulate an internal representation of intended movement dynamics has previously been suggested to be a major impairment of Parkinson's disease subjects (Flowers, 1976; Day *et al.*, 1984; Flash *et al.*, 1992). External temporal constraints may provide individuals with Parkinson's disease a means of organizing the timing and speed of their movements that compensates for their loss of internal cueing mechanisms.

The notion that individuals with Parkinson's disease are unable to optimize the temporal parameters of their movements is supported by recent neurophysiological evidence that the basal ganglia normally serve the role of an internal motor generator (Roland *et al.*, 1980a, b, 1982; Deecke, 1987; Jones, 1987; Brotchie *et al.*, 1991a, b; Ianssek *et al.*, 1995). The basal ganglia appear to be involved in the establishment of appropriate motor sets for preplanned movements, and in the effective timing or switching of motor sets when motor tasks require the performance of repetitive or sequential movements. The basal ganglia have been shown to modulate the activity of the supplementary motor area and lateral premotor areas prior to the initiation of ballistic movements (Tanji and Kurata, 1982; Mink and Thach, 1987, 1991a, b), internally generated movements (Mushiake *et al.*, 1991), and well-learned or highly predictable motor acts (Schultz and Romo, 1992). A number of studies have reported that the activation of these areas prior to the performance of self-initiated or internally driven movements is much lower for Parkinson's disease subjects than for healthy adults (Jahanshahi *et al.*, 1995; Touge *et al.*, 1995). In addition to

a decrease in premovement activity, Parkinson's disease subjects have been noted to show prolonged phasic activity of these areas following the individual movements of sequential motor tasks (Cunnington *et al.*, 1995). The disruption of activity of the direct and indirect motor circuits of the basal ganglia which occurs as a result of Parkinson's disease (Albin *et al.*, 1989; Alexander and Crutcher, 1990; Marsden and Obeso, 1994; Ianssek *et al.*, 1995) appears to lead to inappropriate activation and modulation of cortical motor areas, which compromises the production of internally generated and self-regulated movements.

A third explanation for the increased reaching speed of Parkinson's disease subjects is specifically related to the visual cue that subjects were provided in the moving ball condition. The visuotemporal stimulus of the moving ball may have allowed Parkinson's disease subjects to generate fast movements through the activation of cortical and subcortical motor circuits that are less dependent upon basal ganglia function. Zeki *et al.* (1991) have identified cortical areas in humans that respond selectively to rapid visual motion cues. Glickstein and Stein (1991) have suggested that bradykinetic Parkinson's disease subjects may be able to perform fast movements when visual motion cues activate motion-detection cells in cortical visual areas that stimulate cerebellar-cortical motor circuits. A shift in movement organization from basal ganglion motor circuits, which are typically associated with internally driven movements, to cerebellar motor circuits that can be externally driven may underlie the ability of Parkinson's disease subjects to perform faster movements in response to visuotemporal stimuli than under self-regulated conditions.

In conclusion, the results of our study provide evidence that Parkinson's disease subjects who display bradykinesia during the performance of self-regulated maximal speed movements are able to generate fast reaching speeds in response to a visuotemporal stimulus. The increased reaching speed Parkinson's disease subjects showed did not appear to compromise their movement accuracy, although it may have affected their ability to time and coordinate the grasp component of reaching. Our findings suggest that the bradykinesia of individuals with Parkinson's disease is not merely the result of a basic deficit in force production capacity or a compensation mechanism for poor movement accuracy. Bradykinesia appears to reflect a basic deficit in the inability of these individuals to internally drive their motor output and maximize their movement speed during the performance of self-regulated motor tasks. Further study is required to determine whether it is the presence of an external temporal cue or the visual stimulus of a moving object that allows individuals with Parkinson's disease to exceed their self-determined maximal speed of moving when they perform motor tasks in association with visuotemporal cues.

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