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# NOTE

## TEMPORAL DISSOCIATION OF THE PREHENSION PATTERN IN PARKINSON'S DISEASE

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**Abstract**—This study assesses the reach to grasp movement of eight Parkinson and eight control subjects. The reach was of either 15, 27.5 or 40 cm. The grasp was either of a small (0.7 cm) or a large diameter (8 cm) dowel. When comparing Parkinson to control subjects, no differences were found in the regulation of movement parameters according to changes in object distance or size. However, for Parkinson's disease patients the onset of the manipulation component was delayed with respect to the onset of the transport component. It is proposed that this reflects a deficit in the simultaneous or sequential implementation of different segments of a complex movement.

## INTRODUCTION

THE PERFORMANCE of everyday motor tasks involves the implementation of movement sequences. An individual will frequently change from a sitting to a standing position and then progress to a walking pattern. Reaching for an object is often succeeded by dispalcement of this object to a different location. The orderly sequencing of each stage of a motor task poses an interesting problem for the nervous system. It can be asked, for example, whether several simple movements are grouped in sequence as a more complex and complete unit or whether each simple movement represents the processing of an individual motor program. Some evidence points to the idea that a sequence of movements is grouped together as a generalized motor program [22]. From studies of professional typists it has thus been found that a word is a unit of organization whereby each letter is keypressed at an invariant ratio of the duration taken to type the word [26].

In contrast to the generalized motor program hypothesis is the theory that individual motor programs are implemented at each step of the whole movement [3]. For example, with a comparison between Parkinson's disease (PD) patients and control subjects, PD patients showed a greater slowing in the performance of sequential movements (such as flexing the elbow and then performing an isometric precision grip) than in the performance of each movement in isolation. It was proposed that this reflected a deficit in the ability to organize a sequence of actions and/or in the ability to switch from one motor program to another [3]. The structuring of some complex actions thus did not support the expression of a single generalized motor program.

The execution of simultaneous as opposed to sequential movements has also been examined [2]. For PD patients, the duration when simultaneously executing two simple movements exceeded the duration of either movement when performed in isolation. This pointed to a deficit in the simultaneous performance of two distinct motor tasks [24].

In the current study, PD patients performed a reach to grasp movement. This well-characterized prehensile movement is thought to manifest the simultaneous activation and temporal coordination of two separate neural channels [14]: (1) a proximal channel whereby a reach (transport) is implemented [4] and (2) a distal channel employing a grasp (manipulation) [18, 21]. The patterning of these two motor programs is dependent on such task requirements as the size of the target object and the distance of the reach [7, 9, 17]. The reach to grasp movement thus provides a natural medium for assessing the simultaneous or sequential activation of two motor programs in subjects with neurological movement disorders.

With reference to PD patients, this study asks: (1) Will changes in the size of the object or in the distance of the

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reach lead to effects upon each component (transport and manipulation) which differ from those reported for non-PD patients?; (2) Will the temporal coordination between the transport and manipulation components show differences across the two groups?; and (3) Can the reach to grasp task be used to assess deficits in the simultaneous activation of two movement components?

## METHOD

*Subjects*

Eight PD patients were examined: five were in stage 1 and three in stage 2 [12]. The prevalent motor dysfunctions were akinesia and bradykinesia. A light resting tremor was present in two patients. The characteristics of these subjects together with the eight age- and gender-matched control subjects are shown in Table 1. The control subjects

Table 1. Characteristics of the subject sample

| Control group | | | Parkinsonian group | | | | |
|---|---|---|---|---|---|---|---|
| No. | Sex | Age | No. | Sex | Age | Hoehn and Yahr scale | Therapy |
| 1 | M | 60 | 1 | M | 60 | 1 | Sinemet |
| 2 | F | 59 | 2 | F | 58 | 1 | Sinemet |
| 3 | M | 71 | 3 | M | 68 | 2 | Sinemet |
| 4 | M | 59 | 4 | M | 61 | 1 | Sinemet |
| 5 | M | 61 | 5 | M | 62 | 1 | nil |
| 6 | M | 57 | 6 | M | 57 | 2 | Sinemet |
| 7 | F | 52 | 7 | F | 52 | 1 | Sinemet |
| 8 | M | 69 | 7 | M | 72 | 2 | Sinemet |

were free from neurological disorders. All subjects gave their consent to participate and were naïve as to the experimental purpose. The PD patients were tested approx. 2 hr after medication. Note that PD patient 5 was not receiving drug therapy.

*Procedure*

The subject was seated in a height-adjustable chair so that the feet and back were supported. For the starting position, the right arm, dominant in all cases, rested on the working table. The shoulder was flexed and internally rotated (approx. 45°), the elbow flexed to 90°, the forearm in mid-pronation and the ulnar border of the hand rested upon a pressure-sensitive switch positioned 15 cm anterior to the thorax. The thumb and index finger were held in a relaxed opposed position. The object to be grasped was a 10 cm high translucent cylindrical dowel made of perspex. It was either of small (0.7 cm) or of large (8 cm) diameter. The dowel was positioned vertically and in the midline at 15, 27.5 or 40 cm from the starting switch. Embedded within the table surface immediately beneath the dowel were computer-controlled light-emitting diodes. When these were activated the dowel was illuminated. Infrared-emitting diodes (IREDS) were securely taped to the skin of the right forearm and hand. The wrist IRED was attached to the dorso-radial aspect of the radial styloid process. One digital IRED was attached to the ulnar side of the thumb-nail; the other to the radial side of the index finger-nail.

The subject was instructed to begin the movement as soon as the dowel became illuminated. This was shortly after (500–2000 msec) a warning tone. He/she was required to reach towards and then grasp and lift the dowel. The dowel was illuminated until grasped. Movement speed was not stipulated except to ask the subject to perform the movement as he/she would normally. For each target/distance combination, the subjects performed 10 practice trials and then a block of 15 experimental trials. Kinematics were recorded only during the 90 experimental trials. To distribute practice effects across condition, the order of blocks was counterbalanced across subjects. All subjects adopted a clear pattern of grasp according to the diameter of the dowel. The small dowel was grasped with a precision grip, characterized by opposition between the index finger and thumb [20]. The large dowel was grasped with a whole-hand prehension, characterized by flexion of all the fingers around the object.

*Movement recording, data processing and analysis*

Trials were recorded and analyzed using the OPTOTRAK 3D system. This consisted of three infrared cameras each containing two charge-coupled device sensors which measured the $x$ (distance in the medio-lateral horizontal plane) and $y$ (distance in the antero-posterior plane) coordinates of the IREDs. Movement recording began from the auditory tone and continued until after the dowel was lifted. The signals from the IREDs were sampled at 250 Hz. The three-dimensional coordinates of each IRED were computed from the two raw-dimensional data from each

camera. The velocity of the wrist IRED was computed following filtering (Butterworth dual pass filter; cut-off frequency 8 Hz). Acceleration data were derived by differentiating the velocity data.

Analysis of the transport component was based on the kinematics of the wrist marker: velocity and acceleration profiles. Onset of this component was taken as the time, shortly after release of the starting switch, at which the wrist IRED exceeded a displacement of 0.3 mm (spatial precision of the OPTOTRAK system). Analysis of the manipulation component was based on the kinematics of the two digital markers: temporal variation of the distance between the thumb and index finger. Onset of this component was taken as the time at which the distance between the thumb and index finger IREDs was more than 0.3 mm greater than the starting position distance. Movement duration was taken as the time from onset of the transport component to the time at which the distance between the digital IREDs indicated that the dowel had been grasped.

For each subject, mean values of each dependent measure were calculated for each *Group* (PD, Control), *Distance* (15, 27.5, 40 cm) and *Type of grasp* (Precision grip, Whole-hand prehension) combination. These data were entered into a factorial analysis of variance whereby *Group* was the between-subjects factor and *Distance* and *Type of grasp* were the within-subjects factors. An alpha level of 0.05 was adopted for all tests of significance. *Post-hoc* contrasts were carried out using the Newman–Keuls procedure.

*Effects of varying the distance of the dowel from the subject*

Means and tests of significance for each of the measured parameters are shown in Table 2. PD patients and control subjects showed similar trends. By changing the distance of the dowel from the subject, both the transport and manipulation components were affected. For the reach of 40 cm several parameters of the transport component showed values which were different from those of the shorter reaches (15 and 27.5 cm). Movement duration was greater and wrist peak velocity was later and of greater amplitude for this longer distance (see Fig. 1). Effects of distance were also evident on the acceleration profile. Peak acceleration was later and of greater amplitude for the 40 cm than for the shorter reaches. Similarly, peak deceleration was later for the larger distance.

For the manipulation component, the time of maximum grip aperture between the thumb and index finger was later for the 40 cm than for the 15 or 27.5 cm reaches.

*Effects of varying the type of grasp*

Means and tests of significance for each of the measured parameters are shown in Table 2. Once again, the PD patients showed the same trends as the control subjects. Kinematics of the transport component varied according to the grasp adopted. Peak velocity was earlier for precision grip than for whole-hand prehension movements. This allowed a greater deceleration time (time from peak velocity to end of movement) for approach of the hand to the smaller than to the larger dowel. Changes to the manipulation component also reflected a greater allocation of approach time for the more precise grip. Maximum grip aperture between index finger and thumb occurred earlier for a precision grip than for a whole-hand prehension movement. As would be expected, and as illustrated in Fig. 2, the amplitude of this maximum aperture was larger for whole-hand prehension (large dowel) than for precision grip (small dowel).

*PD patients: delayed onset of the manipulation component*

As expected, the PD patients showed a generalized slowing of movement. An additional difference between this patient group and the control subjects was found when comparing the onset time of the manipulation component to that of the transport component. For control subjects the onset of manipulation (time at which the index finger and thumb began to open) occurred, on average, 32 msec after the onset of transport (time at which the arm began to reach). In contrast, manipulation began, on average, 82 msec after transport for the PD patients [$F(1, 7) = 12.05$, $P < 0.001$; Fig. 2]. As such a result could be attributed to the slower movement of the PD patients, the onset of manipulation was expressed as a percentage of movement duration. This gave additional confirmation of the delayed onset of manipulation for PD patients. The opening of the thumb and index finger began at 8% of movement duration for PD patients but at 3% for control subjects [$F(1, 7) = 18.45$, $P < 0.001$]. A regression analysis was performed between the onset time of manipulation (absolute and relative values) and movement duration. The finding of no correlations indicated that the later onset of manipulation for PD patients was not due to a relationship between movement duration and manipulation onset.

An interesting feature of this delayed onset of manipulation for PD patients was the difference between precision grip and whole-hand prehension trials. For this patient group, manipulation began 92 msec (on average) after transport when a precision grip (small dowel) trial was performed. For the more gross grasp of whole-hand prehension, manipulation began 70 msec after transport [$F(1, 7) = 25.31$, $P < 0.001$]. The control subjects showed no relationship between the onset time of manipulation and the type of grasp adopted.

## DISCUSSION

The current study assesses the transport and manipulation components of a reach to grasp movement as performed by PD patients and control subjects. Such a prehension task is considered as being subserved by two

Table 2. Effect of varying distance and type of grasp upon the kinematic measures of transport of the arm and upon grip formation

| Distance | 15 cm | | 27 cm | | 40 cm | | |
|---|---|---|---|---|---|---|---|
| Subjects | PD | CONT | PD | CONT | PD | CONT | Test of Significance |
| Movement time (msec) | 1112 (191) | 872 (182) | 1211 (275) | 1018 (246) | 1428 (281) | 1122 (250) | $F(2, 7)=57.25$, $P<0.0001$ |
| Time to peak acceleration (msec) | 301 (24) | 242 (25) | 369 (37) | 267 (22) | 383 (33) | 284 (30) | $F(2, 7)=8.01$, $P<0.005$ |
| Amplitude peak acceleration (mm/sec$^2$) | 1442 (282) | 1793 (351) | 1870 (260) | 2971 (387) | 2341 (362) | 3734 (789) | $F(2, 7)=16.62$, $P<0.001$ |
| Time to peak velocity (msec) | 491 (75) | 364 (74) | 558 (73) | 386 (79) | 635 (123) | 448 (98) | $F(2, 7)=21.16$, $P<0.001$ |
| Amplitude peak velocity (mm/sec) | 258 (112) | 308 (39) | 429 (114) | 568 (121) | 566 (142) | 752 (100) | $F(2, 7)=89.86$, $P<0.0001$ |
| Time to peak deceleration (msec) | 720 (145) | 578 (131) | 825 (180) | 624 (134) | 910 (230) | 661 (108) | $F(2, 7)=12.47$, $P<0.001$ |
| Amplitude peak deceleration (mm/sec$^2$) | 1112 (646) | 1320 (499) | 1358 (536) | 1702 (778) | 1420 (585) | 2459 (973) | $F(2, 7)=11.89$, $P<0.0001$ |
| Time to maximum grip aperture (msec) | 762 (291) | 545 (175) | 907 (346) | 632 (179) | 1075 (372) | 794 (282) | $F(2, 7)=84.16$, $P<0.0001$ |
| Time from peak velocity to the end of movement (msec) | 632 (256) | 509 (172) | 653 (217) | 632 (181) | 793 (181) | 674 (163) | $F(2, 7)=32.95$, $P<0.0001$ |
| % Time to peak deceleration | 65 (12) | 68 (14) | 68 (9) | 72 (7) | 72 (8) | 75 (8) | $F(2, 7)=7.58$, $P<0.005$ |
| % Time to maximum grip aperture | 67 (7) | 62 (13) | 76 (9) | 65 (7) | 76 (10) | 71 (8) | $F(2, 7)=7.66$, $P<0.005$ |

| Type of grasp | PG | | WHP | | |
|---|---|---|---|---|---|
| Subjects | PD | CONT | PD | CONT | Test of Significance |
| Time to peak velocity (msec) | 549 (86) | 387 (93) | 574 (78) | 411 (86) | $F(1, 7)=10.39$, $P<0.001$ |
| Time from peak velocity to the end of movement (msec) | 711 (205) | 643 (202) | 674 (249) | 567 (154) | $F(1, 7)=8.02$, $P<0.005$ |
| Time to maximum grip aperture (msec) | 885 (352) | 628 (218) | 942 (362) | 685 (186) | $F(1, 7)=24.74$, $P<0.001$ |
| Amplitude of grip aperture (mm) | 54 (9) | 55 (2) | 112 (10) | 100 (14) | $F(1, 7)=343.37$, $P<0.0001$ |

S.D. in parentheses. PD: Parkinson patients; CONT: Control subjects; PG: precision grip; WHP: whole-hand prehension.

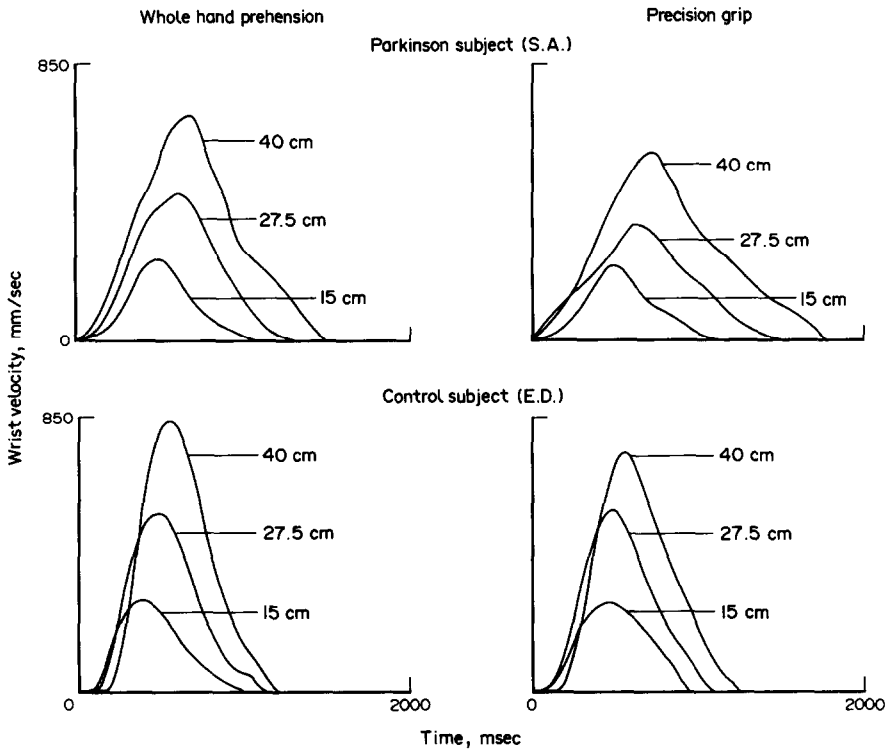


Fig. 1. Wrist velocity profile when reaching to dowels placed at 15, 27.5 and 40 cm. The ordinate shows the velocity as measured from the wrist IRED. The abscissa shows the sampling duration of the trial (2000 msec). Above: a single trial from a PD patient performing a whole-hand prehension (left) and a precision grip (right). Below: a single trial from a Control subject performing a whole-hand prehension (left) and a precision grip (right).

parallel neural channels [14]: one for the more proximal arm movement and one for the distal shaping of the hand [4, 18, 21]. This study thus addresses the effect of impairment of basal ganglia function upon the patterning of each component and upon the coordination between the two neural channels.

*Effects of varying either distance of the object from the subject or the type of grasp*

Analysis of the reach to grasp movement in PD patients indicates a slowed performance but no deficit in the ability to modify the spatio-temporal characteristics of the prehension pattern in response to experimentally imposed changes in either the distance of the object from the subject or the size of the object [8, 19, 27]. For both the patient and the control groups, transport time of the arm and the timing and amplitude of the peaks of arm velocity, acceleration and deceleration all increase with reaching distance. Deceleration time is longer when a precision grip rather than a whole-hand prehension is performed. This latter result supports those of previous studies with non-PD subjects: the approach phase is augmented for movements requiring greater accuracy such as when reaching to grasp more fragile [17] or smaller objects [7, 9]. PD patients also show no abnormalities in the modification of manipulation parameters in relation to reaching distance or to the size of the object to be grasped. As for control subjects, the peak of hand opening for PD patients occurs at an earlier time when reaching to grasp objects which are closer to the subject and/or when reaching to grasp smaller objects. PD patients are thus able to correctly regulate movement parameters. They exhibit no inability to activate the required and appropriate motor programs. In addition, the finding that the time of peak hand opening changes as a function of movement duration indicates that the patterning of one component is related to that of the other. This is also consistent with previous findings for non-PD subjects [7, 9, 13, 14].

In 1980, HALLETT and KHOSHBIN [11] reported that patients with Parkinson's disease were unable to generate the appropriate amount of initial agonist activity in order to generate rapid elbow flexion movements of different amplitudes. The peak of arm velocity did not show an increase for movements of greater amplitude. They

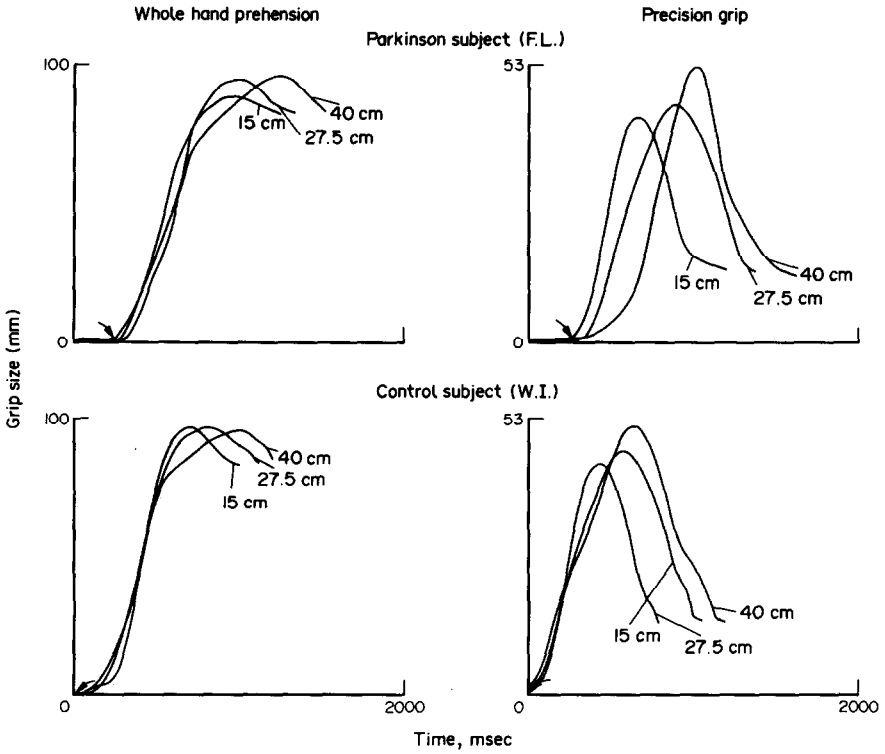


Fig. 2. Grip size profile when reaching to dowels placed at 15, 27.5 and 40 cm. The ordinate shows the distance between the index finger and thumb. The abscissa shows the sampling duration of the trial (2000 msec). Above: a single trial from a PD patient performing a whole-hand prehension (left) and a precision grip (right). Below: a single trial from a Control subject performing a whole-hand prehension (left) and a precision grip (right). Note that for the PD patient (above) the onset of the manipulation component (see arrows) occurs well after the onset of the transport component.

interpreted this as an impairment in the ability to correctly "energize" the agonist muscle. This view [11] of the basal ganglia as a selector and energizer of muscles is not consistent with our findings or with those from previous studies [25]. In this experiment it is found that the velocity of the movement increases with reaching distance. The overall form of the motor program of Parkinson subjects thus appears to be maintained. The selection of muscles and the timing of their activation enables the correct relative timing of all movement parameters of the reach to grasp movement. A suitable number of neuronal sets are mobilized and the temporal arrangement of these sets is maintained.

*Onset delay between transport and manipulation components*

For PD patients, it is the coordination between the two components of the reach and grasp movement which shows abnormalities: the onset of the manipulation component is delayed with respect to the onset of the transport component [8, 27]. This suggests that the reach to grasp movement may be directed by two distinct motor programs which are normally executed almost simultaneously. Three ideas may be advanced to explain this abnormal coordination.

Firstly, given the two neural processing channels of the prehension movement [4, 18, 21], the "abnormality of striopallidal function, caused by nigrostriatal dopamine depletion in Parkinson's disease, results in a deficit in the simultaneous processing of two different motor programs" [2]. The two components of the reach to grasp movement may be under the control of separate but superimposed motor programs. The delay in the near-concurrent activation of the two components of prehension for PD patients could reflect dysfunction of central mechanisms which process the superimposition of two motor programs. Secondly, the delay can be related to an impairment in the performance of sequential movements. This is supported by the finding for the control group that the onset of manipulation is approx. 31 msec later than the onset of transport. This delay may thus be interpreted as

the time to switch from the motor program of the transport component to that of the manipulation component. This is in line with the idea that for the execution of a total motor plan, each of its component programs is delivered in sequence and each acts as a signal to deliver the next [16]. Accordingly, if the basal ganglia are responsible for assisting the switch between motor programs, as has been suggested from clinical evidence [2, 3], shifting from transport to manipulation neural processing channels produces an increase in the time taken by Parkinson subjects to initiate the second movement.

The duration of the delay between the activation of the two components is related to the type of distal program utilized. With the more accurate precision grip task Parkinson subjects show a greater delay than with whole-hand prehension. This adds support to a central neural processing origin for the lag in the activation of the distal motor pattern. Neurophysiological studies indicate that neural channels for precision grip differ from those for the more gross task of whole-hand prehension [18, 21]. The basal ganglia and, in particular, the putamen show a complex pattern of connectivity with cortical regions which have roles in the preparation and execution of complex distal actions [21]. This pattern is referred to as the motor circuit and includes connections from the putamen to frontal areas such as the primary motor cortex, supplementary motor area and the arcuate premotor area [1] and more dorsal connections to the somatosensory cortex and association area [15]. Basal ganglia dysfunction may thus be more evident with the simultaneous or sequential performance of accurate tasks which require more complex neural programming. The level of dissociation between activation of the transport and manipulation components may relate to more complex central processing requirements for the latter component.

Thirdly, a delay in the activation of the distal movement could be attributed to mechanical constraints imposed by the classical symptoms of Parkinsonian rigidity. Muscle stiffness may result in an enhanced mechanical resistance to hand opening which could feasibly lead to a delay of the manipulation component activation. However, it seems unlikely that the mechanical resistance would be greater for a precision grip involving both fewer fingers and a smaller amplitude of movement than whole-hand prehension. In addition, the relative timing of finger aperture was consistently between 60 and 80% of movement time for both PD patients and control subjects suggesting that the movement, once activated, showed no abnormal coordination with the transport component. It is proposed that the delay of component activation more reflects an impairment in the neural organization than of mechanical resistance.

That the basal ganglia are more involved with aspects of complex movement organization is supported by electrophysiological studies. A loose relationship has been found between the activity of basal ganglia neurones and such executed movement parameters as force, direction and velocity [10]. BROTCHIE *et al.* [5, 6] suggest that activity of basal ganglia neurons shows a more definite correlation to cognitive aspects such as contextual setting and task difficulty. Our results point to the role of this circuit for the selection, sequencing and activation of motor programs.

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