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Motor functions of the basal ganglia

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Summary. A study of movement disorders such as Parkinson's disease and Huntington's disease can provide an indication of the motor functions of the basal ganglia. Basal-ganglia diseases affect voluntary movement and can cause involuntary movement. Deficits are often manifested during the coordination of fine multi-joint movements (e. g., handwriting). The disturbances of motor control (e.g. akinesia, bradykinesia) caused by basal-ganglia disorders are illustrated. Data suggest that the basal ganglia play an important role in the automatic execution of serially ordered complex movements.

Introduction

The disruptions of skilled movement caused by basal-ganglia diseases are commonly called *movement disorders*. Movement disorders are of interest because they dramatize the computational role of the brain in the coordination of movement (Phillips, Muller, & Stelmach, 1989).

Researchers have examined basal-ganglia disease for a variety of reasons (Brown, 1989). There is a need to assess patients' functional status to assist clinical management and diagnosis (see Selby, 1990). Such research is driven by clinical impressions and by paper-and-pencil neuropsychological tests. An examination of movement disorders may also provide an indication as to processes involved in the coordination of movement (Benecke, 1989). This research must invoke current theories of movement coordination. In addition, a consideration of the functional loss caused by well-localized diseases can provide information as to the function of specific brain structures (Marsden, 1990). This research requires an understanding of neurology and neuroanatomy, as well as an understanding of movement coordination.

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The present paper will illustrate some of the disturbances of fine motor control caused by basal-ganglia disease, consider which aspects of movement coordination are affected, and outline current theories of basal-ganglia functioning. We shall focus primarily upon functional losses caused by basal-ganglia disease (Marsden, 1985). In particular we shall examine Parkinson's disease and Huntington's disease: diseases that have been at the cutting edge of clinical research into the biochemistry (Parkinson's disease), and genetics (Huntington's disease) of basal-ganglia dysfunction. While our grasp of the functions of the basal ganglia is only partial, a historical perspective suggests that there has already been considerable progress in understanding diseases of these structures (Barbeau, 1981; Selby, 1990), and we feel that this bodes well for the further elucidation of the role of the basal ganglia in the control of behaviour.

Diseases of the basal ganglia such as Parkinson's disease and Huntington's disease cause disorders of movement that are salient and serious (and embarrassing) for the affected patient (Jankovic, 1987). These disorders demonstrate the importance of computational mechanisms in the smooth execution of movement, and a consideration of functional disturbances caused by these disorders may provide information about basal-ganglia function (Marsden, 1985). As will be outlined, these diseases cause problems in the performance of *voluntary movement*, and cause *involuntary movement* (Folstein, Jensen, Leigh, & Folstein, 1983; Selby, 1990).

Basal-ganglia disease can cause problems of voluntary movement control (Hallett, 1990; Hallett & Khoshbin, 1980; Folstein, 1989). Affected patients may exhibit a number of symptoms. Clinically, such patients have difficulty in maintaining movements (bradykinesia). In experimental paradigms, the slowness and jerkiness of movement produces prolonged movement times that are employed as an index of bradykinesia. In addition, patients may have difficulty in initiating movements (akinesia). This slowness with which patients initiate a response produces prolonged reaction times that are used as an index of akinesia.

Basal-ganglia disease can also cause involuntary movements, which may interfere with voluntary movements (Lohr & Wisniewski, 1987). Depending upon disease and medication status, patients may exhibit different forms of involuntary movement. Clinically patients may show rhythmic involuntary movements (tremor). The Fourier analysis of patients' movements can be used experimentally to document the frequency of such tremor. Patients may also show spontaneous, random, irregular movements (chorea or dyskinesia). Electromyography shows that muscles are activated inappropriately in patients exhibiting such choreiform movements (Hallett, 1983).

While Parkinson's disease and Huntington's disease both affect basal-ganglia function, the diseases in fact affect different nuclei within the basal ganglia. Parkinson's disease causes degeneration of the dopaminergic nigrostriatal pathway which predominantly innervates the putamen. This seems to cause a loss of control of thalamic structures, resulting in tremor (Marsden, 1985; 1990). Huntington's disease causes degeneration initially of the caudate nucleus. This may cause a loss of control of the globus pallidus and subthalamic nuclei, resulting in choreiform movements (Marsden, 1985).

The identification and localization of function is never a simple process. However, a consideration of functional loss (negative signs) can provide an indication of the function of the basal ganglia.

Akinesia and bradykinesia are the more debilitating symptoms of Parkinson's disease (Delwaide & Gonce, 1988) and Huntington's disease (Folstein et al., 1983). A study of akinesia and bradykinesia can potentially provide details as to how movements are prepared and executed. A casual observation of a patient with a movement disorder reveals that basal-ganglia diseases tend to cause a slowness, or jerkiness, of movement. Our first impressions might be that the disruptions of coordination are all due to impairments in the computational control of movement (see Stelmach & Hughes, 1984). This viewpoint, however, is perhaps somewhat simplified. To make inferences as to basal-ganglia function, we need to take into account factors such as the effects of natural ageing processes, any biomechanical differences, and any effects of medication. We then need, wherever possible, to rule out any alternative explanations of functional impairments (Brown & Marsden, 1987).

Converging systematic observations using age-matched controls are essential in an examination of basal-ganglia disease, because Huntington's and Parkinson's disease affect older adults, and there are also age-related declines in the preparation and execution of movement (Goggin & Stelmach, 1990). In addition, one must be cautious when interpreting motor impairments in terms of disturbances in the *computational* control of movement, because Parkinson's disease and Huntington's disease can cause *biomechanical* differences associated with abnormalities of muscle tone (Lohr & Wisniewski, 1987). For instance, a slowness in initiating movement could reflect stiffness, rather than any deficit in preparatory processes. In addition, it is necessary to consider the effects of medication on performance, since the drugs used to treat basal-ganglia disorders can have side effects upon cognitive and motor functioning (Lohr & Wisniewski, 1987).

Akinesia

It was initially thought that akinesia was the result of impaired preparatory processes (Marsden, 1982). However, experiments have shown that patients suffering from Parkinson's disease can prepare their movements more or less normally in some circumstances (Stelmach, Worringham, & Strand, 1986). Indeed, prolonged reaction times are less consistently reported than prolonged movement times, either in patients suffering from Parkinson's disease (Evarts, Teravainen, & Calne, 1981) or from Huntington's disease (Hefter, Homberg, Lange, & Freund, 1987). The absence of clear demonstrations of akinesia in experimental studies of single movements is at odds with clinical experience, and Marsden (1990) has suggested that the basal ganglia must have a role in engaging subsequent movements in a movement sequence.

Patients suffering from Parkinson's disease are less likely to prepare sequences of movements in advance. While studies of normal healthy subjects show that RT increases with increases in the number of submovements in a movement sequence, Stelmach, Worringham, and Strand (1987) found that patients' RT did not increase with the number of finger taps in a response sequence. Studies carried out by Stelmach, Phillips, and Chau (1989), and Harrington and Haaland (1991) suggest that patients have more problems with complex movements. Stelmach et al. (1989) found that patients were able to prepare finger taps in advance when there was a compatible relationship between stimulus and response, but not when the relationship between stimulus and response was incompatible. Similarly, Harrington and Haaland (1991) examined the initiation of sequences of homogenous and heterogeneous hand postures, and found that patients were able to prepare sequences of similar repetitive movements, but were less likely to prepare a sequence of disparate alternating movements. There are some reports that patients suffering from Huntington's disease also have problems in performing sequences of movements (Thompson et al., 1988).

While preparatory processes are fairly well documented in Parkinson's disease, very little is known about the effects of Huntington's disease on preparatory processes. We have examined patients' ability to use advance information to prepare their movements in a sequential button-pressing-movement task, which measured the time spent holding down buttons (an index of preparatory processes), and the time spent moving between buttons (an index of efficiency of movement) (Bradshaw et al., 1992; Jones et al., 1992). Subjects were presented with varying amounts of advance information and the degree of improvement in performance was examined.

In Cue condition A, the next button in the series to be depressed was illuminated by the computer only when the present button was released; that, is, there was no advance information. In Cue condition B, the next button in the series to be depressed was illuminated by the computer when the present button was depressed; that is, one button

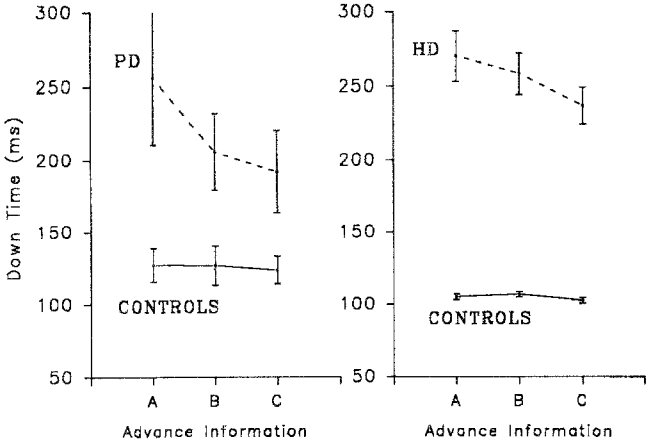

Fig. 1. Down Time for Parkinson's-disease patients (PD) and Huntington's-disease patients (HD) and age-matched controls, at three levels of advance information (A none, B medium, C High)

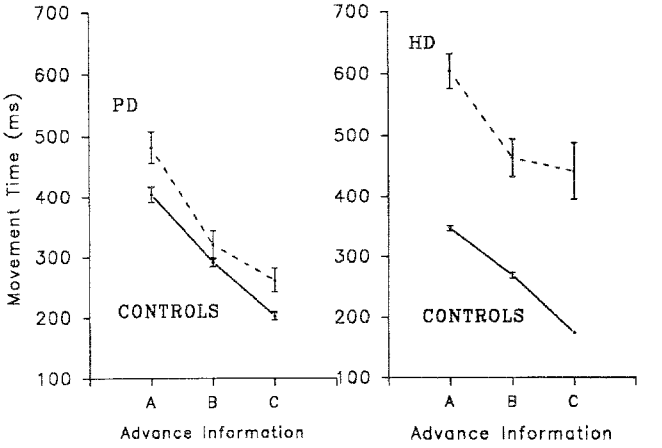

Fig. 2. Movement Time for Parkinson's-disease patients (PD) and Huntington's-disease patients (HD) and age-matched controls, at three levels of advance information (A none, B medium, C High)

was prepared in advance. In Cue condition C, the next button in the series to be depressed was illuminated by the computer when the previous button was released; that is, two buttons were now prepared in advance.

In Bradshaw et al. we examined 18 patients suffering from Huntington's disease (mean age 45.1 years) and an equal number of age-matched controls (mean age 44.7 years). In Jones et al. we used the same procedures and apparatus to examine 13 patients suffering from Parkinson's disease (mean age 66.6 years) and 13 age-matched controls (mean age 66.7 years). Data from the two experiments are presented for comparison in Figures 1 and 2. Means are based upon 160 button presses in each experiment.

Figure 1 presents the interactive effects of Group × Cue condition upon the time spent holding down buttons from studies by Bradshaw et al. and Jones et al. Both Parkinson's-disease patients, $F(2,48) = 6.29$, $p < .05$, and Huntington's–disease patients, $F(4,102) = 3.67$, $p < .05$, had significant difficulty in initiating movements, as compared to healthy controls. Subanalyses showed that patients were slow at initiating movements without external cues (Cue A), indicating in both patient groups that an aspect of the preparatory process is impaired.

Preparatory processes enable the fast smooth execution of movements in healthy subjects. Figure 2 presents the interactive effects of the Group × Cue condition upon the time spent moving between buttons. There were again significant interactions. Both Parkinson's-disease patients, $F(2,48) = 3.68$, $p < .05$, and Huntington's-disease patients, $F(4,102) = 2.63$, $p < .05$, had significant difficulty in maintaining movements, as compared to healthy controls. Subanalyses revealed that patients suffering from Parkinson's disease and from Huntington's disease had problems in using advance information in Cue condition C to produce faster movements.

Patients suffering from Parkinson's disease appear to move faster than patients suffering from Huntington's disease. This is potentially of note since bradykinesia is considered more characteristic of Parkinson's disease than of Huntington's disease. Any differences might reflect the medications involved (dopamine agonists or antagonists). While patients suffering from Parkinson's disease were all being treated with dopamine agonists, only five of the Huntington's patients were taking dopamine antagonists. Obviously, further work is required before conclusions are made about the respective disease processes.

Bradykinesia

Hallett (1990) has suggested that for several reasons the bradykinesia and akinesia of Parkinson's disease are the result of different pathophysiological processes. First, he observed that akinesia is less consistently reported than bradykinesia (Evarts, Teravainen, & Calne, 1981; Hefter et al., 1987). Secondly, Hallett noted that the symptoms respond differently to medication: dopamine improves bradykinesia in patients suffering from Parkinson's disease, but does not improve akinesia as measured by simple reaction time (Bloxham, Dick, & Moore, 1987).

Hallett and Khoshbin (1980) had suggested that patients suffering from Parkinson's disease were bradykinetic because they had difficulty in energizing their muscles. They observed that patients suffering from Parkinson's disease (when compared with normal subjects) required more cycles of agonist and antagonist muscle activity to produce a movement. They hypothesized that patients' initial agonist muscle activation was insufficient, and that patients needed to employ more small bursts of activity to produce the same movement as that of normal subjects, so that patients require more bursts of muscle activity to produce faster or longer movements.

Evidence suggests that bradykinesia is not simply a problem of weakness and difficulty in energizing muscles. Indeed, it is possible to find situations where patients actually produce too much, rather than insufficient, force. For example, Muller and Abbs (1990) examined precision grip in patients suffering from Parkinson's disease. In this study, patients were required to grip an object without dropping it (this requires forces greater than the weight of

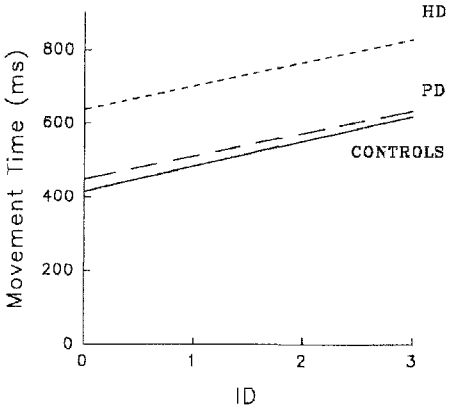

Fig. 3. Fitt's law, with large targets for patients suffering from Parkinson's disease (PD), Huntington's disease (HD), and age-matched controls

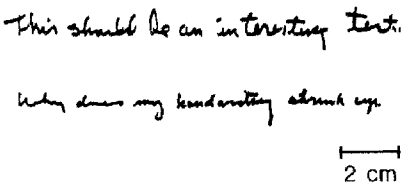

Fig. 4. Sample of handwriting of a patient suffering from Parkinson's disease, showing micrographia

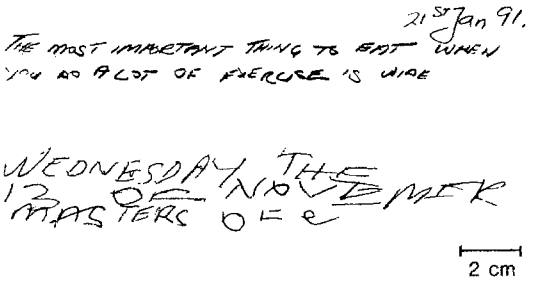

Fig. 5. Samples of handwriting of a patient suffering from Huntington's disease, showing macrographia. Samples are from 21 January and 13 November of the same year

the object). Although the patients suffering from Parkinson's disease are typically thought to produce insufficient force, in this study patients overgripped, producing more force than was required to grip the object.

Instead of a simple problem of force production, it has been suggested that bradykinesia is a function of movement context (Teasdale & Stelmach, 1988), since deficits are more often seen in tasks that have precise accuracy requirements. The effects of movement-precision requirements have been examined by means of Fitts' Law. Sanes (1985) examined the movements of patients suffering from Parkinson's disease, using a stylus to targets of small (1, 2, or 4 mm) or large (10, 20, or 40 mm) widths. Halsband, Homberg, and Lange (1990) used a similar technique to examine movements of patients suffering from Huntington's disease (target widths of 5, 10, and 15 mm). Both Parkinson's- (Sanes, 1985) and Huntington's- (Halsband et al., 1990) disease patients showed greater increases in movement time with increases in the index of difficulty of movement (Fitts' Law) in target-aiming tasks. Sanes (1985) found that increases in movement time were noticeable when precision was important.

To demonstrate the paramount importance of movement precision, we used Fitts' Law in a pilot study, to examine the rate of gain of visual information for movement, in two patients with Parkinson's disease, two patients with Huntington's disease, and age-matched controls. We used larger targets (10 and 20 mm) than those of Sanes (1985) or Halsband et al. (1990), with movement amplitudes of 60 or 120 mm, so as not to place excessive demands upon precision (ID 0.7, 1.7, 2.7). As may be seen in Figure 3, the rates of gain of information are comparable in the three groups, implying that mechanisms for visual-feedback guidance are intact and can compensate for any functional impairment. Deficits would seem to appear in situations in which greater demands are placed upon feedback guidance – that is, where greater precision is required, so that feedback guidance mechanisms can no longer cope.

An understanding of functional disturbance is to some extent complicated by strategic compensations in patient's behaviour, so that they rely upon visual feedback (Flowers, 1976) and intact cerebellar mechanisms (Glickstein & Stein, 1991). Patients seem to manage tasks that can be broken down into small visually guided chunks, but have problems with more automatic movements that must be tightly linked in sequence (e. g. handwriting, walking).

Handwriting is of particular interest, since it is a precision skill that requires a fluent motor output. Such fine, precise, multi-joint movements are sensitive to disorders of movements, and may provide early signs of disease (see McLennan, Nakano, Tyler, & Schwab, 1972; Penney et al., 1990). We have therefore examined a variety of writing and drawing movements in patients suffering from Parkinson's disease and Huntington's disease, using graphics tablets. Some of the changes in handwriting brought on by basal-ganglia disease are illustrated in Figures 4 and 5. Patients suffering from Parkinson's disease can exhibit micrographia (McLennan et al., 1972), while patients suffering from Huntington's disease can exhibit macrographia (Podoll, Caspary, Lange, & Noth, 1988). Figure 4 shows the handwriting of a patient with Parkinson's disease. This patient showed some difficulty in maintaining writing size and speed.

Figure 5 shows two samples of the handwriting of a patient suffering from Huntington's disease. This patient showed a difficulty in initiating and maintaining writing size and speed, which is sensitive to the progression of the disease.

While it is sometimes difficult to determine specific motor impairments in simple discrete movement tasks (Benecke, 1989), it is certainly possible to demonstrate impairments in more ecologically valid tasks such as handwriting. Graphics tablets allow a sensitive recording of the kinematics of patients' movements. Movements were recorded at 200 Hz on a graphics tablet and low-pass filtered at 10 Hz to remove any quantization error (Teulings &

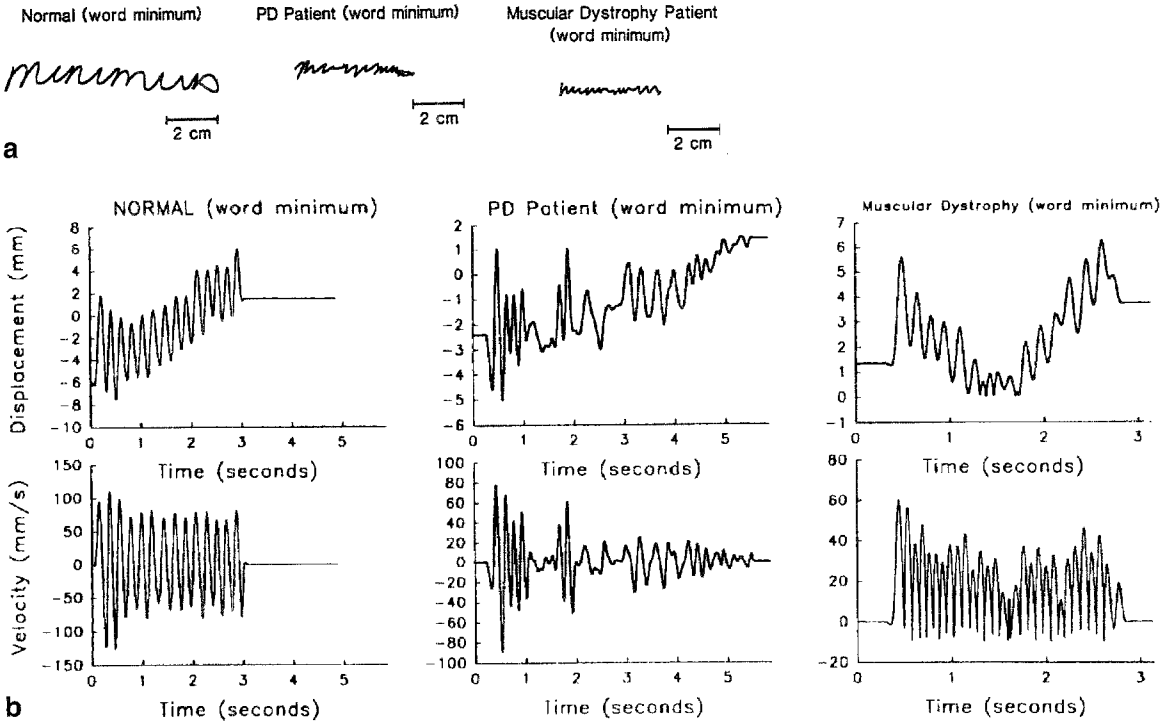

Fig. 6 a. The word "minimum" (without "i" dotted), written by a healthy adult, a Parkinson's-disease patient, and a patient suffering from muscular dystrophy. **Fig. 6 b.** The corresponding Y-displacement and Y-velocity functions over time, for the word "minimum" (without "i" dotted), written by a healthy adult, a Parkinson's-disease patient, and a patient suffering from muscular dystrophy

Maarse, 1984). Velocity and acceleration were calculated with the use of a 9-point central finite difference procedure.

Figure 6a shows the best attempts at writing the word "minimum", by a healthy adult and by a patient suffering from Parkinson's disease who showed severe micrographia. While the healthy subject shows fluent and regular up- and- down writing strokes, the Parkinson's-disease patient shows a difficulty in maintaining size and movement velocity, and in linking chunks of the word together (see Figure 6b).

To illustrate that the motor problems are a function of precision, we examined the same patient suffering from Parkinson's disease, when the patient was producing simple scibbling movements. Figure 7 shows simple scribble movements of about 7.5-cm extent. This patient's movements seem to improve dramatically during unguided scribbling movements. As may be seen, both the age-matched control and the patient suffering from Parkinson's disease can produce scribbles of similar size, velocity, and acceleration. Note, however, the acceleration function of the patient suffering from Parkinson's disease. There is some uncertainty of movement forces inherent in even simple scribble movements. The uncertainty that occurs in the production of simple movements obviously causes difficulty when patients are required to produce complex, precise movements such as handwriting.

We doubt that this is simply a problem of weakness and insufficient force production. Figures 6a and 6b also show the handwriting of a patient whose weakness was caused by peripheral nervous system problems (muscular dystrophy). This patient also shows small handwriting, but does not exhibit the halting and progressive slowing shown in the Parkinson's-disease patient.

It is unlikely that the slowness of movement in patients suffering from Parkinson's disease is simply a product of tremor and rigidity. Bradykinesia has been reported to be independent of tremor and rigidity, clinically (Marsden, 1990) and experimentally (see Phillips, Stelmach, & Teasdale, 1991; Yanagawa, Shindo, & Yanagisawa, 1990). Indeed tremor and rigidity can be substantially improved by thalamotomy without any beneficial effects on movement speed (Selby, 1990). Similarly, bradykinesia in patients suffering from Huntington's disease is unlikely to be a product of chorea. Bradykinesia is observed to vary independently of chorea (Folstein et al., 1983).

The two diseases demonstrate the importance of preparatory processes for the smooth execution of movements. Patients have some difficulty in preparing movements, so that movements are laborious rather than smooth and effortless. The exact nature of the control process(es) disrupted by basal-ganglia disease is not clear. It is apparent that the general form of motor programs is intact. Some of the changes in scale of patients' handwriting would point to deficits in the specification of a movement parameter (Rosenbaum, 1980), such as extent; however, evidence to this effect has not been forthcoming (Stelmach et al., 1986). Instead, data suggest that there are deficits in the preparation of movement sequences (Harrington & Haaland, 1991), possibly affecting the hierarchical-program structure. Rosenbaum (1985), for example, has suggested that a motor program consists of a hierarchical list of associations between motor commands and clock

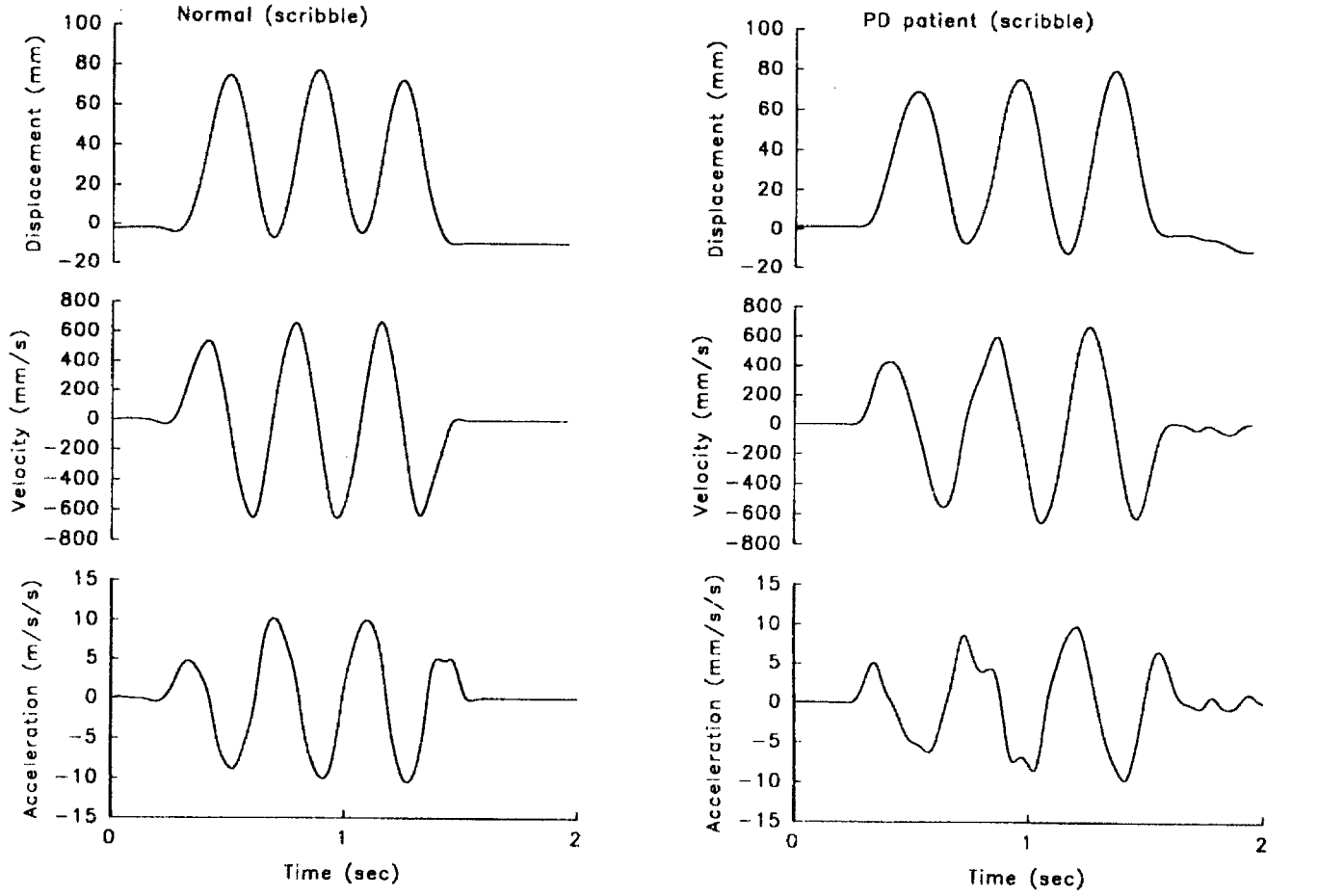

Fig. 7. X displacement, velocity, and acceleration during horizontal scribble (6 strokes) in a patient suffering from Parkinson's disease, and an age-matched control subject

pulses. These preparatory processes are less likely to be required when precision is not important (see Franks & Van Donkelaar, 1990), but become important in complex movement sequences. This would explain some of the slowness and jerkiness seen when patients attempt complex movements such as handwriting, since motor programs serve to link or schedule successive submovements into a coordinated whole (Lashley, 1951), and allow submovements to be automatically performed. This leads us to suggest that the basal ganglia have a role in scheduling submovements, which allows the automatic execution of serially ordered, complex movements.

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