Movement Disorders—Limb Movement and the Basal Ganglia
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The primary concern of this article is to review experimental methods that may lead to a better understanding of the functional role of the basal ganglia in the control of movement. Two models of basal ganglia impairment are considered: Parkinson's disease and Huntington's disease. The review focuses primarily on akinesia and bradykinesia because they are key abnormalities of basal ganglia dysfunction. In general, through electromyography and kinematic analysis of movement, it may be possible to characterize specific movement disorders. Specifically, if damage sustained by the central nervous system is traced to a certain structure, it may provide insight on the extent of involvement and functional role of that structure in the control of movement. Much of the data reviewed suggests that the basal ganglia may play a specific role in the initiation and regulation of force control. [Stelmach GE, Phillips JG. Movement—disorders: limb movement and the basal ganglia. Phys Ther. 1991;71:60-67.]

Key Words: Basal ganglia; Huntington chorea; Kinesiology/ biomechanics, general; Movement control; Parkinson disease.

Movement Disorders and Motor Control

One method of attaining inferences about the role of a brain structure in the control of movement is the association of that structure with impairments of function. Such association may take place through brain imaging or pharmacologically. For example, in Huntington's disease, there is enhanced activity in the dopaminergic system, whereas in Parkinson's disease, there is a loss of dopaminergic cells in the substantia nigra, such that patients respond to dopamine agonists therapy. However, inferences about basal ganglia function are difficult to interpret because the basal ganglia mediate between higher and lower brain structures, receiving, for example, inputs from cortical areas and the substantia nigra and innervating thalamic and midbrain nuclei.

Damage to structures such as the basal ganglia, cerebellum, or frontal cortex may interfere with smooth execution of movement. Deficits in functioning may indicate the role a structure normally plays in the control of movement. Careful consideration, however, must be given to a number of factors before making an attempt at identification of functional loss. Observed disruption of function could result from any of the following: (1) natural age-related decline, (2) deficits in higher cognitive processes (eg, depression, dementia), (3) side effects of drug therapy (eg, dyskinesia, confusion), (4) biomechanical changes (eg, rigidity, body mass), and (5) deficits in the coordination of movement (eg, preparatory processes, feedback guidance). Given the number of possible causes of functional disruption, expertise is required of researchers in the areas of neurology, physiology, psychology, and experimental methodology.

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Assessment of Functional Loss

A number of techniques are currently used in the assessment and identification of functional losses in patients with basal ganglia impairment. Reaction-time paradigms, electromyography (EMG), and kinematic analysis may be used to examine deficits in the control of movement. Such techniques attempt to document how movement in patients deviates from normal movement. The assessment of functional loss, therefore, requires an understanding of normal functioning.

Reaction-time paradigms have been used to assess the preparatory processes in patients with movement disorders. In general terms, reaction-time studies can provide strong indications of whether there are pre-movement abnormalities. In particular, response latencies have been used as an index of difficulty in movement preparation. Researchers reason that disproportionate difficulty in preparing movement parameters should be evident from longer response latencies. Reaction-time paradigms have been used to examine whether patients with movement disorders are slower than control subjects when given an opportunity to plan movements, choose among response alternatives, and use advance information.

Electromyography has been used to examine the voluntary movement of patients with a variety of movement disorders and is typically used to determine whether observed movement deficits are attributable to disordered force control (eg, scaling the magnitude and duration of EMG). Normal ballistic movement is presented by a triphasic (agonist, antagonist, agonist) pattern of muscle activity. Patients with movement disorders often exhibit disruptions of this pattern, which, if studied, may contribute to our understanding of the brain's role in the organization of movement. For example, in comparing similar movements, patients with Parkinson's disease require a number of cycles of agonist-antagonist activity, suggesting that the basal ganglia have a role in facilitating movement. In contrast, patients with cerebellar deficits exhibit problems with the duration of activity of agonist or antagonist muscles, which suggests that parts of the cerebellum have a role in determining the end points of movement.

Electromyography has also been used to describe the involuntary movements of patients with various movement disorders. The involuntary movement accompanying Tourette's syndrome has been shown to have a normal triphasic pattern. The involuntary movement seen in Huntington's disease, however, presents a variety of possible patterns of activity, ranging from tonic activation, to co-contraction, to triphasic patterns of activation. Application of EMG techniques ultimately may be useful in the differential diagnosis of movement disorders.

Kinematic analyses may provide indicators of disruptions in normal motor coordination resulting from deficits in movement preparation and in muscle activation. Kinematic analysis has tended to be descriptive in its application. It permits thorough, microscopic inspection of the spatial and temporal characteristics of a movement in relation to joint segments. Because it is important to establish the precise ways in which the movement trajectories of patients with movement disorders differ from those of healthy subjects, indexes have been derived to describe the efficiency of movement trajectories. An optimum trajectory between initiation and termination of movement has only one cycle of acceleration and deceleration. The movement of patients with Parkinson's disease and cerebellar disorders tends to be irregular and asymmetrical. For example, Stelmach and Worthingham and Teulings and Stelmach have shown that parkinsonian patients do not have precise control over their movement trajectories.

Parkinson's Disease and Basal Ganglia Function

Parkinson's disease significantly impairs a patient's quality of life. Without medication, or during on-off phases of therapy, there are dramatic incapacitating changes in motor control. Parkinson's disease often produces akineti and bradykinetic symptoms, resulting in problems performing discrete movements as well as sequences of rhythm. For example, handwriting tends to be slower and smaller in patients with Parkinson's disease. During rhythmic movement, movements tend to be reduced in size and/or duration, causing a hastening (destination) of movement.

The damage associated with Parkinson's disease tends to be localized to a loss of the dopaminergic cells in the substantia nigra that project to the basal ganglia. This loss disrupts basal ganglia function. Thus, Parkinson's disease provides a model for making inferences about the motor functions of the basal ganglia. The basal ganglia are a collection of subcortical nuclei consisting of the caudate, putamen, external and internal segments of the globus pallidus, subthalamic nucleus, and pars compacta and pars reticulata of the substantia nigra.

Generally, patients with Parkinson's disease initially present unilateral symptoms, but exhibit bilateral symptoms as the disease progresses. Problems with postural reflexes and gait arise at still later stages. The four cardinal symptoms associated with Parkinson's disease are (1) akinesia—a difficulty in initiation of movement, (2) bradykinesia—a slowness in execution of movement, (3) rigidity—a resistance to the passive stretch of muscles, and (4) tremor—a trembling or shaking at rest of about 4 to 5.5 Hz. In addition, they have marked changes in gait and posture.

Marsden has suggested that positive symptoms, such as tremor and rigidity, are due to the loss of the inhibitory influences within the basal ganglia, but reasons that basal ganglia function may best be understood through examination of the loss of function, that is, the negative symptoms of akinesia and bradykinesia. Most of the research effort to date has...
observed deficit in choice reaction time was not disproportionately delayed in parkinsonian patients, despite the fact that they clearly demonstrated that patients can use advance information. Interestingly, Pullman et al. reported that, although patients’ choice reaction times were normal, they increased significantly as current L-dopa levels decreased.

Extending reaction-time paradigms, Stelmach et al. systematically assessed the ability of patients with Parkinson’s disease to prepare components of an arm movement. Patients performed a series of reactions in which precued information provided components of the upcoming movement. The precued information varied between complete and partial information and permitted the patients to prepare movement dimensions such as arm or direction, arm and extent, or arm, direction, and extent. Although patients took longer than healthy control subjects to prepare movements, and ultimately were slower in executing them, no specific movement dimension was disproportionately more difficult than another. Thus, the deficits observed in patients with Parkinson’s disease appear to be deficits in the control of movement.

Akiniesia may be caused by disturbances in the preparation of movement. Reaction time, therefore, should be a good index of such an impairment. In simple reaction-time tests, because there is no response uncertainty, subjects have an opportunity to fully prepare the intended response. In choice reaction-time situations, this is not possible because the response is not known until the imperative stimulus is given. Thus, in contrast to simple reaction time, choice reaction time stresses the subject’s ability to select among response alternatives.

Evarts et al. found in patients with Parkinson’s disease substantial delays in simple reaction time without any observed deficit in choice reaction time and interpreted these data to suggest that patients were unable to benefit from the opportunity to prepare the required response. Stelmach et al. also found that choice reaction time was not disproportionately delayed in parkinsonian patients, despite the fact that they clearly demonstrated that patients can use advance information. Interestingly, Pullman et al. reported that, although patients’ choice reaction times were normal, they increased significantly as current L-dopa levels decreased.

In simple reaction-time tests, patients do not tend to superimpose elementary motor programs in multi-joint movements, but rather complete one movement before starting the next. The lack of ability to superimpose elementary movement components is in line with similar observations by Benecke et al. and Shimizu et al. Akinesia and bradykinesia may result from force-control deficits. Flash has demonstrated recently that parkinsonian patients do not tend to superimpose elementary motor programs in multi-joint movements, but rather complete one movement before starting the next. The lack of ability to superimpose elementary movement components is in line with similar observations by Benecke et al. and Shimizu et al. Akinesia and bradykinesia may result from force-control deficits. Flash has demonstrated recently that parkinsonian patients do not tend to superimpose elementary motor programs in multi-joint movements, but rather complete one movement before starting the next.
contraction of arm muscles in patients with Parkinson's disease. Patients were required to produce forces at 25%, 50%, and 75% of the maximum force they could produce. Although patients were slow, and somewhat variable, in force production, they were as accurate as age-matched controls. Inspection of the forces' curves revealed that they were irregular and asymmetrical. A subsequent experiment considered production of these forces in more detail. Patients with Parkinson's disease were required to produce forces at 15%, 30%, 45%, and 60% of the maximum force they could produce. Although the patients showed accurate force production compared with age-matched controls, they took longer to reach their peak forces and exhibited very irregular time curves. This finding suggests Parkinson's disease does not cause impairment in the intentional control of forces (indeed, patients intentionally compensate for their rate deficits), but in the rate of force development.

Taking a slightly different approach, Flowers found that aiming accuracy of ballistic movement in patients with Parkinson's disease was disrupted when visual guidance was removed. When the target could not be seen, movement speed also appeared to decrease. Judging from these results, it would seem that patients with Parkinson's disease slow their movement to make use of visual feedback. In further support of this view, Inzelberg et al. found that arm movements, with vision, followed a straight path in healthy subjects, they underwent multiple time-consuming corrections in patients with Parkinson's disease. Thus, it appears that parkinsonian patients are highly dependent on visual feedback.

Force-control deficits may also be understood by considering how parkinsonian movement deviates from optimum movement trajectories. A variety of indexes are available to better describe movement trajectories. Stelmach and associates used a measure of smoothness and economy of movement to characterize movement trajectories in patients with Parkinson's disease. An examination of the change of sign in the second derivative of force gave an indication of the number of changes in the rate of force production. Whereas healthy subjects produced near-optimal forces, patients with Parkinson's disease showed considerably more jerkiness in their force production. Sheridan et al. summarized the Parkinson's disease motor problem as difficulty in maintaining a computed force, difficulty in initiating a force, and a difficulty of increased variability in time and space.

Teulings and Stelmach considered the regularity, relative to the variability, of features of parkinsonian patients' handwriting movement trajectories. They used signal-to-noise-ratio (SNR) analyses to indicate the efficiency of the control of aspects of movement trajectories. The SNR expresses the ratio between the amount of modulation attributable to the invariant, programmed component (signal) and the amount of modulation attributable to the impairment (noise). Thus, the SNR is high if a movement pattern is reproduced accurately and low if a pattern is variable because of motor impairment. This experiment explicitly raised the question of whether patients with Parkinson's disease had impairments in force amplitude or force duration. Force amplitude refers to the strength of the force and force duration to the control of force initiation and cessation. It was found in patients with micrographia that handwriting was more impaired in terms of force amplitude than in terms of force duration. This impairment is suggested to be located at the level of motor unit recruitment. Phillips et al. provide a slightly different view, that slowness of handwriting movements in patients with Parkinson's disease may be explained partially by temporal characteristics of muscle-force changes and, to a lesser degree, by low-level force amplitude problems. They also reasoned that the impaired force amplitude may contribute to a deliberate compensatory behavior for movement duration.

Movement sequencing provides another task variable to explore parkinsonian dysfunction. Stelmach et al. showed, as previously reported in this article, that patients with Parkinson's disease have difficulty producing alterations of force within a movement sequence. When patients had to produce a stress tap in a sequence of finger taps, they tended to break the tapping sequence at the point of the stress tap. Regardless of where in the sequence the stress (additional force) occurred, the interpulse interval after the stress tap was disproportionately lengthened. Parkinsonian patients' force pulses were also more irregular than those of healthy subjects and not well sustained. A known sequencing problem in patients with Parkinson's disease is the "hastening phenomenon" reported by Nagasaki and Nakamura. Parkinsonian patients exhibited hastening of movement when tapping at frequencies of 2.5 or 5 Hz. As the predominant frequencies in handwriting are close to 5 Hz, such a hastening effect could cause problems in parkinsonian patients trying to write at normal speeds. Freund found in patients with Parkinson's disease that serial hand movements faster than 2 Hz are possible only if they are synchronized with the tremor. This is similar to the hastening phenomenon and is suggested to lead to disturbances of handwriting.

Benecke et al. and Goldenberg et al. found that sequential movements in patients with Parkinson's disease are executed slower than when executed in isolation. The second movement part especially is slowed, and pauses between the first and second movement parts are increased. Even more difficulty is experienced when patients with Parkinson's disease attempt to superimpose two separate motor programs. This difficulty could relate to two-joint ballistic movements (eg, movements required to draw triangles and squares of different sizes and shapes), in which a larger number of EMG bursts are observed in patients with Parkinson's disease than in control subjects.

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Electromyography demonstrates that muscle activation is not optimal in patients with Parkinson's disease. The issue then is whether the number of bursts (peripheral control) or size of burst (central control) is impaired. For healthy subjects, movements about a single joint are known to be made via a single biphasic or triphasic burst of EMG activity. Movements of longer amplitude have been shown to be produced by EMG patterns of increasing amplitude and duration. Hallett and Khoshbin suggest that movements in patients with Parkinson's disease are slow and irregular because of difficulty in activating muscles, such that more cycles of muscle activity are required to produce a movement. In particular, they suggest that patients with Parkinson's disease require more bursts of muscle activities to produce faster or longer movements. Although this is quite a plausible explanation for the observed slowness and irregularity in movement, it tends to suggest that impairment occurs at a peripheral level, such that the size of bursts of muscle activity cannot be appropriately scaled for a required movement.

The issue of muscle activity has also been addressed by Berardelli and associates and Teasdale and associates. Berardelli et al examined muscle activity in parkinsonian patients' movements of differing extents (wrist movements of 15° and 60°). They found that size of agonist bursts increased in a normal fashion with extent of movement, although the bursts were not scaled to meet task demands. This finding indicates that the size of bursts in muscle activity can increase in patients with Parkinson's disease. Teasdale et al examined parkinsonian patients' muscle activity during production of arm movements of differing durations through an angle of 20 degrees. In addition to moving their arm at a self-determined speed, they were asked to move their arm 10% faster, 30% slower, and 60% slower. The results showed that patients with Parkinson's disease were slower than control subjects and that they could vary their speed as requested. The results also demonstrated that irregularities were present as the movement speed varied and that more bursts of muscle activity were required to produce slower movements rather than faster movements.

Experiments by Teasdale et al and Berardelli et al demonstrated that irregular force production is not a function of the degree of muscle activation. Indeed, the mechanisms for producing longer or slower movements would appear to be intact, with the number of bursts of muscle activity increasing normally with longer or slower movements. Instead, patients exhibit difficulty in scaling the central signals involved in muscle activation.

It may be asked whether observed slowness and irregularity of movements in patients is simply a function of a slower and more tonic pattern of muscle activity. This is not thought to be the case: for example, Teasdale et al observed more bursts of muscle activity when patients moved at speeds similar to those of age-matched controls.

It may also be asked whether observed irregularity of movement is simply a function of tremor. Tremor is not the sole cause of jerkiness in movement of patients with Parkinson's disease. Parkinsonian tremor characteristically occurs at rest, and is minimal in some patients. Phillips et al examined handwriting in patients with minimal tremor. Although their movements were jerky and irregular, the jerkiness was not periodic and did not occur at frequencies associated with tremor (ie, 5-8 Hz). These observations suggest that Parkinson's disease causes an attenuation in the central commands involved in muscle activation and that the basal ganglia have some role in the activation of muscles.

Gait and posture experiments have examined repetitive movement sequences in detail and suggest that the observed changes are partly due to a reduction in the size of parkinsonian movement (ie, hastening, or festination). Knutsson and Martensson have postulated that some of the abnormalities of posture and gait may be the result of reduced preparatory postural adjustments, rigidity, and adaptation to impaired postural reflexes. Support for this view comes from Bazollette et al, who found that patients with Parkinson's disease did not make preparatory postural adjustments before initiating voluntary movement.

Experiments that have assessed long-latency reflexes in patients with Parkinson's disease have found that, although the latency of stretch reflexes is normal in these patients, there are abnormalities in the gain of the longer-latency components. Moreover, it has been noted that these abnormalities are related to poorer balance and gait. It appears that the abnormalities of posture and gait may be the result of parkinsonian rigidity and adaptation to impaired postural reflexes; however, much more research is needed.

**Huntington's Disease and Basal Ganglia Function**

Huntington's disease is a hereditary disorder characterized by a progressive atrophy of the caudate nucleus within the basal ganglia, and then of cortical structures. It is thought to be a glutamate-dependent neurotoxic process, producing substantial reductions in dopaminergic neurons. Results from the loss of specific sets of cholinergic neurons and neurons that synthesize gamma-aminobutyric acid. Although causal mechanisms are as yet unclear, it may be stated that multiple neurotransmitter systems are affected, specifically, the disease is associated with enhanced dopaminergic activity and reduced cholinergic activity.

Because Huntington's disease is a progressive disorder that involves other brain structures, one must be cautious in drawing inferences about basal ganglia function. In addition, both rigidity and hypotonia (enhanced and reduced muscle tone, respectively) are reported, depending on the age of the patient.
Jerkiness and lack of coordination. Whereas Parkinson's disease is associated with enhanced activity in dopaminergic neurotransmitter systems, Huntington's disease responds to treatment with dopamine agonists, aspects of Huntington's disease respond to treatment with dopamine antagonists. Indeed, excessive use of dopamine agonists produces choreiform movements, such as those seen in patients with Huntington's disease, and excessive use of dopamine antagonists produces parkinsonian-like symptoms. These findings suggest that Huntington's disease may also serve as a model for basal ganglia function. In comparison with Parkinson's disease, however, there are fewer studies available. An understanding of excessive movement, related to the dopaminergic system as it operates in chorea, may provide an indication of basal ganglia function.

Choreiform movement exhibits reflexive, ballistic, and tonic patterns of muscle activity and is difficult to characterize electromyographically. Indeed, Hallett reported that patients may show any pattern of muscle activity at any time. Marsden and associates investigated choreiform movement in patients with Huntington's disease. They observed continuous change in activity from one muscle to another, and within individual muscles from one pattern of activity to another. Muscle activation in chorea ranges from very brief bursts of activity (eg, 50–200 milliseconds) to prolonged contractions (eg, 2 seconds). Muscle activity is not correctly sequenced, because co-contractions occur that interfere with voluntary movement.

Kinematic analysis also reveals that choreiform movement lacks a clear pattern. Myers and Falek used an accelerometer to assess resting hand tremor in patients with Huntington's disease and in control subjects. Whereas the control subjects had a dominant tremor frequency, the patients with Huntington's disease showed intermittent bursts of tremor at no consistent frequency.

Akinesia has been reported in Huntington's disease. The symptom of akinesia has been found to be a better predictor of functioning than severity of chorea. Reaction time tends to be longer in patients with Huntington's disease. As in Parkinson's disease, however, findings of prolonged reaction time in patients with Huntington's disease are less reliable than those of prolonged movement time. This may be because involuntary choreiform movement affects the initiation of voluntary movement. For example, Lasker et al found that patients have difficulty suppressing eye saccades in a simple reaction-time task. On the other hand, in the case of Parkinson's disease, it would appear that patients with Huntington's disease tend to use online control during movement execution, rather than preparing them in advance.

Bradykinesia in patients with Huntington's disease has been examined by Hallett et al and Thompson et al. Hallett et al examined muscle activity during ballistic movement in the fingers of patients with Huntington's disease. The normal agonist-antagonist pattern was maintained in these patients, suggesting that the ability to select proper muscle groups was unimpaired. However, patients required longer to attain peak force. Hallett et al reported a linear relationship between contraction time and contraction amplitude in their patients (ie, movement of greater amplitude required longer contraction time).

Thompson et al examined muscle activity during simple and complex movements in the wrists of patients with Huntington's disease. Movement was characterized by slow, prolonged contractions. Central deficits in the activation of movement were indicated, as patients had additional difficulty performing more complex simultaneous or sequential movements (eg, squeezing the hand and flexing the elbow).

The use of reaction-time, EMG, and kinematic analyses are not so prevalent in patients with Huntington's disease. Thus, it is unclear as to how enhanced basal ganglia activity relates to functional movement control. The data that are available suggest that, as in Parkinson's disease, movement control is profoundly impaired. These impairments appear to be partially related for the temporal and spatial aspects of force control.
Conclusion

Although insight into basal ganglia function originally came from clinical settings, more recent insights have come from experimental motor science. Deficits in proper muscle activation observed in patients with Parkinson’s disease and Huntington’s disease support the suggestion that the basal ganglia have a general role in the normal activation of muscle. The available data on these patient populations, however, have not consistently demonstrated movement execution impairments beyond those of bradykinesia. In some studies, selective difficulties have been noted; in other studies, no such selectivity has been found. The data do suggest, however, that basal ganglia impairment does influence the initiation and regulation of force. This should provide a setting for further research.

The spatial and temporal organization of a movement involves precise control over the forces applied and their durations. Deficits in either parameter can lead to faulty movement execution. The recent work by Teulings and Stelmach is promising, as it attempts to separate the force-control components. These studies suggest that, on a relative basis, force amplitude is more impaired than force duration.

The study of movement disorders, when coupled with current issues in normal motor control research, can provide converging evidence about the roles brain structures play in the control of movement. Further research, however, must be directed at understanding multisegmented movements in which the intersegments’ dynamics break down in impaired populations. Movement disorders research may engender new paths of research and new approaches to physical therapy interventions, while challenging contemporary theoretical frameworks. It is hoped that this brief review illustrates some of the current issues in basal ganglia research and promotes an interdisciplinary framework advocating an isomorphism between neurophysiological and cognitive-psychological accounts of motor control.

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