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Effects of focal basal ganglia lesions on timing and force control

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Abstract

Studies of basal ganglia dysfunction in humans have generally involved patients with degenerative disorders, notably Parkinson's disease. In many instances, the performance of these patients is compared to that of patients with focal lesions of other brain structures such as the cerebellum. In the present report, we studied the performance of patients with focal basal ganglia lesions on three fundamental motor tasks. The patients all had suffered unilateral damage in the striatum and were tested in the chronic state. The first task required the participants to tap with their index finger as fast as possible; this test provided a simple assessment of motor competence. Compared to controls, the maximum tapping rate was lower for the patients when tapping with their contralesional limb, although the deficit was not severe. The second and third tasks were designed to assess timing and force control, two functions that have been associated with basal ganglia function. The patients performed similar to controls on both tasks and showed no evidence of impairment when using their contralesional limb compared to their ipsilesional limb. The results indicate that unilateral basal ganglia lesions tend to produce minor motor problems in force control, and fail to support the hypothesized role of the basal ganglia in timing.

1. Introduction

The functional contribution of the basal ganglia has been the subject of extensive research. Traditionally, the basal ganglia have been considered part of the motor pathways, although more recent theories have pointed to potential roles of this structure in learning and cognition (e.g., Doya, 2000; Krebs, Hogan, Hening, Adamovich, & Poizner, 2001; Sommer, Grafman, Clark, & Hallett, 1999). Other researchers have taken an information processing approach in exploring basal ganglia function, conceptualizing its role to be a type of gate that may inhibit unwanted motor plans (Marsden & Obeso, 1994; Mink, 1996) or correlate information from various cortical structures in an efficient manner (Boraud, Bezard, Bioulac, & Gross, 2002).

Much of this work has been motivated, at least in part, by consideration of the profound deficits observed in humans with Parkinson's disease. Given the prominent motor problems experienced by these patients, a considerable effort has been devoted to identify the functional contribution of the basal ganglia to various movement parameters. This line of research has catalogued many of the movement impairments associated with Parkinson's disease, including deficits in the temporal control of movements (Harrington, Haaland, & Hermanowicz, 1998; O'Boyle, Freeman, & Cody, 1996), and force control (Sheridan, Flowers, & Hurrell, 1987; Wing, 1988).

Parkinson's disease has been an essential model system for studying basal ganglia function. One concern with this approach, however, is that the effects of this degenerative disease are not limited to the basal ganglia. Alteration in brain function is observed outside of the basal ganglia, most notably in the frontal cortex. The cortical abnormalities may be a direct consequence of

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the disease process: the dopamine loss in the striatum is mirrored by a reduction in dopaminergic projections to the frontal cortex, although the extent of this reduction is markedly less than in the striatum (Piccini, Pavese, & Brooks, 2003; Scatton, Javoy-Agid, Rouquier, Dubois, & Agid, 1983). In addition, changes in cortical function may be an indirect consequence of alterations in basal ganglia function (Owen, Doyon, Dagher, Sadikot, & Evans, 1998). Given this, the motor deficits in Parkinson's patients may reflect abnormal function of not only the basal ganglia, but in other neural systems as well. This concern is, of course, inherent in all studies involving the use of brain lesions. It is difficult to localize function through the analysis of dysfunction, or to account for changes in neural structure that result in compensation of neurological injury. Nonetheless, degenerative diseases are especially problematic, given their systemic nature.

In the current paper, we employed a different patient population to assess the effects of basal ganglia damage, individuals who have suffered a stroke centered in the basal ganglia. In addition to determining how the performance of these patients compares to that of Parkinson patients, the focal lesion group allowed us to make a within-subject comparison between performance with the contra- and ipsilesional hand. This form of comparison has proven useful in the study of other motor disorders (e.g., Ivry, Keele, & Diener, 1988), but is not usually appropriate for Parkinson's patients given that their symptoms are generally bilateral. In this study we report the observation of patients with focal lesions of the basal ganglia on tests of movement speed, movement timing, and force control. Before turning to the Methods, we provide a brief review of lesion-based work in animals examining the role of the basal ganglia on these types of tasks, as well as previous studies involving human patients with focal basal ganglia lesions.

1.1. Basal ganglia modulation of force production

A variety of methods have been used to study the role of the basal ganglia in force control. The study of controlled lesions in animals has provided some insight into the movement parameters associated with basal ganglia function. Jeyasingham and colleagues reported hyperactive grip strength and impaired reaching in rats that had received nigrostriatal lesions unilaterally, and bilateral reaching deficits in rats receiving unilateral lesions to the striatum (Jeyasingham, Baird, Meldrum, & Dunnett, 2001). Similar deficits have also been observed in primates with experimental lesions of basal ganglia output pathways. Abolition of the globus pallidus nuclei produced slowed movement times in animals trained to reach to targets. However, reaction times appeared to be normal, suggesting deficits in the acceleration phase of reaching movements (Horak & Anderson, 1984). Similarly, when monkeys were trained to reach and grasp an object, local lesions created by muscimol application to the globus pallidus produced slower movement times but only in extension of the elbow and not in flexion (see also Inase, Buford, & Anderson, 1996; Wenger, Musch, & Mink, 1999), suggesting deficits in the modulation of movement amplitude.

This work is complemented by studies of humans with Parkinson's disease (PD). A prominent feature of this disorder is bradykinesia, or movement slowness. Similar to experimental research conducted with animals, the study of movement times has implicated the basal ganglia system in the regulating the production of force. Analysis of the movement trajectories produced by PD patients, have revealed that slower movement times stem from abnormalities in the acceleration/deceleration phase of movement (Platz, Brown, & Marsden, 1998). The force control problem in PD patients, however, is not simply a matter of a reduction in the ability to produce maximum force. PD patients also are impaired on tasks requiring a decrease, or modulation in force (Wing, 1988).

EMG studies have also pointed to a problem in scaling muscular activity to match movement amplitude (Hallett & Khoshbin, 1980). When trying to achieve larger movements, PD patients tend to generate a series of agonist bursts of a stereotypic size, rather than increase the size of the burst. The end result may be accurate, but with abnormal kinematics. Similarly, on an isometric force control task, PD patients were as accurate as controls, yet they exhibited abnormal force–time profiles (Stelmach & Worringham, 1988). This result has led the researchers to conclude that a more accurate description of the deficit of the force regulation in PD patients may be in terms of regulating the force–time profile of an isometric contraction (Ivry & Corcos, 1993).

1.2. Temporal prossessing and the basal ganglia

The role of the basal ganglia in temporal processing has been the subject of considerable study. Much of this work has involved pharmacological and lesion methods with animals, focusing on the question of whether manipulations of dopamine levels alter the rate of an internal clock (see Gibbon, Malapani, Dale, & Gallistel, 1997; Malapani & Rakitin, 2003; Meck, 1983, 1996, 2003; Meck & Benson, 2002). These studies have tended to use a task that may best be characterized as one of time perception as the intervals studies are considerably longer than those required for motor coordination.

Of greater relevance for our present concerns are studies that have examined the performance of PD patients on repetitive movement tasks, focusing on the temporal consistency of such movements. Results from these studies are inconclusive and contradictory. In an experiment involving a group of PD patients performing