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Direction-specific postural instability in subjects with Parkinson's disease

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Abstract

The purpose of this study was to determine whether and why subjects with Parkinson's disease (PD) have greater instability in response to specific directions of perturbations than do age-matched control subjects and how instability is affected by stance width. This study compared postural responses to 8 directions of surface translations in PD subjects and age-matched control subjects while standing in a narrow and wide stance. PD subjects were tested in their practical OFF state. A postural stability margin was quantified as the difference between peak center of pressure (CoP) and peak center of mass (CoM) displacement in response to surface translations. The control subjects maintained a consistent stability margin across directions of translations and for both narrow and wide stance by modifying rate of rise of CoP responses. PD subjects had smaller than normal postural stability margins in all directions, but, especially for backwards sway in both stance widths and for lateral sway in narrow stance width. The reduced stability margin in PD subjects was due to a slower rise and smaller peak of CoP in the PD subjects than in control subjects. Lateral postural stability was compromised in PD subjects by lack of trunk flexibility and backwards postural stability was compromised by lack of knee flexion, resulting in excessive displacements of the body CoM. Stability margins in PD subjects were related to their response on the pull test in the Unified Parkinson's Disease Rating Scale. Thus, PD patients have directionally specific postural instability due to an ineffective stiffening response and inability to modify their postural responses for changing postural demands related to direction of perturbation and initial stance posture. These results suggest that the basal ganglia, in addition to regulating muscle tone and energizing postural muscle activation, also are critical for adapting postural response patterns for specific biomechanical conditions.

Keywords: Parkinson's disease; Posture; Balance; Equilibrium

Introduction

Although a backward pull at the shoulders is used to identify postural instability in patients with Parkinson's Disease (PD), it is not clear that they are more unstable in the backward direction than in forward or lateral directions (Allum et al., 2002; Greenspan et al., 1998). Assessing dynamic postural stability across different directions of perturbations could help determine whether and why there may be a directional preponderance of falls in PD (Grimbergen et al., 2004).

Based on biomechanical principles, it is generally assumed that standing humans are most unstable in response to backward body displacements since it is more difficult to exert dorsiflexion, than plantarflexion, torque about the ankles (Winter et al., 1996). However, our previous studies of multidirectional surface translations in young healthy subjects showed that the nervous system may compensate for biomechanical constraints by increasing the magnitude of muscle activation and surface reactive forces triggered in response to backward body displacements (Henry et al., 1998b, 2001). These previous studies also showed that healthy young subjects immediately increase the magnitude

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of their proximal postural responses to lateral displacement when the postural demands are increased by standing in narrow stance. These, and other studies, suggest that the healthy nervous system takes into account initial biomechanical conditions to flexibly modify postural responses to maintain stability (Macpherson et al., 1988; Nashner and Cordo, 1981).

Unlike healthy subjects, PD subjects have difficulty modifying the magnitude and patterns of postural responses for changes in postural demand. The importance of the basal ganglia for set-dependent adaptation of postural movement patterns for changes in conditions has been shown previously for changes in arm support, changes from standing to sitting posture, surface rotation versus translation, and instructions to PD and control subjects (Chong et al., 1999, 2000; Diener et al., 1987; Horak et al., 1992; Schiepatti and Nardone, 1991). Recently, we found that PD subjects also do not modify the magnitude of their muscle activity or resulting direction and magnitude of horizontal reactive forces when changing stance width (Dimitrova et al., 2004a,b). The effects of this postural inflexibility on the biomechanics of postural stability in different conditions are unknown.

The purpose of this study was to determine whether and why PD subjects have greater instability in response to perturbations in specific directions than do age-matched controls and how this direction-specific instability is affected by stance width. We compared how PD subjects and age-matched control subjects adapt their kinetic and kinematic postural responses to different directions of surface perturbations and to different stance widths.

A better understanding of postural deficits in PD subjects may provide insight into the role of the basal ganglia in postural control. For example, if the basal ganglia are particularly important for axial control of trunk (axial) coordination, we would expect PD subjects to have more deficits in control of lateral, postural stability because lateral stability results primarily from hip and trunk control, whereas AP stability results primarily from ankle control (Henry et al., 1998b, 2001; Winter et al., 1996). Furthermore, we would also expect larger postural deficits in narrow stance because responses to lateral surface translations involve primarily weighting and unweighting of the legs with very little trunk movement when the legs are far apart, whereas postural responses require large lateral flexion of the trunk when the legs are close together (Henry et al., 2001; Winter et al., 1996). Biomechanical constraints related to the shape, size, and strength of the foot, limitations of joint motion, and stiffness and flexibility of body parts necessarily interact with neural constraints imposed by basal ganglia pathology to affect postural stability differently across perturbation directions.

To restore a falling body to stable equilibrium, the center-of-foot pressure (CoP) must move in front of the falling center-of-mass (CoM) to return the CoM safely within the base of foot support (Winter et al., 1990; Yang et al., 1990). The CoP is the location of the net reactive forces at the surface (Horak and Macpherson, 1996). In standing humans, the difference between the peak CoP and peak CoM has been used as a quantitative measure of the "functional stability margin" (Winter et al., 1996), which is a functional measure of dynamic stability. The closer that the surface projection of the CoM approaches the CoP, the more likely is an individual to loose equilibrium and to fall or stumble (Yang et al., 1990).

In response to forward body sway induced by backward surface translations, our previous study showed that the difference between the peak CoP and the peak CoM in PD subjects was significantly smaller than that in age-matched control subjects (Horak et al., 1996a). This reduced stability in PD subjects occurred despite slower velocity of CoM displacements due to increased passive stiffness. These results suggest that postural bradykinesia, quantified as a smaller and slower motion of the CoP, may be a primary deficit limiting forward postural stability in patients with PD. However, it is not known whether this type of postural bradykinesia has a similar effect on stability for all directions of disequilibrium in PD subjects.

Our previous studies of PD subjects' responses to multidirectional perturbations are consistent with the hypothesis that the basal ganglia are important for optimizing the pattern and magnitude of postural muscle synergies according to changes in the direction of perturbation or the size of the support base (Henry et al., 1998b, 2001). For each direction of postural sway, different sets of muscles are recruited to return the body to equilibrium (Henry et al., 1998b; Macpherson, 1998; Steiger et al., 1996; Weinrich et al., 1988). We found that PD subjects co-activate postural muscles in response to surface translations, resulting in abnormal direction of forces under each foot (Dimitrova et al., 2004a,b). Although the EMG deficits in PD subjects were not specific for particular directions of perturbations, it is unknown whether this co-contraction affects some directions of instability more than others.

The current study investigated the postural stability and kinematic effects of this disordered muscle activation and horizontal surface forces during multidirectional postural perturbations to better understand the pathophysiology underlying postural instability in PD. It compared CoM, CoP, and kinematic hip and knee joint changes in response to 8 directions of surface translations and it investigated the ability of both subject groups to modify postural responses to maintain stability when their support base changed from wide to narrow stance.

Methods

Subjects

Seven healthy, elderly control and 7 patients with idiopathic PD (Hughes et al., 1992) were a subset of subjects included in studies of EMG responses and surface