Postural Muscle Responses to Multidirectional Translations in Patients With Parkinson’s Disease

Diana Dimitrova, Fay B. Horak, and John G. Nutt

Neurological Sciences Institute and Department of Neurology, Oregon Health and Science University, Beaverton, Oregon 97006-3499

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INTRODUCTION

Postural instability is a hallmark of Parkinson’s disease (PD); patients with PD fall at five times the rate of age-matched elderly subjects (Bloem et al. 1998, 2001; Koller et al. 1988; Nutt and Horak 1996). Although PD subjects fall in many different directions, the mechanisms responsible for postural instability in different fall directions may differ (Nutt and Horak 2002). Our recent studies in the same PD subjects as reported here support the clinical observation that they are especially unstable in response to backward sway and to lateral sway when stance is narrow (Bloem 2002; Dimitrova et al. 2003; Horak et al. 2003; Nutt and Horak 2002). That is, PD subjects’ center of mass (CoM) was displaced further than controls while their center of pressure (CoP) moved less than controls for all directions of perturbation, but especially for backward and lateral directions of dysequilibrium. In addition, surface reactive forces under each foot in response to multidirectional perturbations were hypometric and abnormal in direction for PD subjects, particularly in response to lateral perturbations associated with smaller than normal lateral flexion of the torso (Dimitrova et al. 2003).

It is unknown whether the particular instability to backward postural perturbations is due to specific abnormalities of parkinsonian postural muscle synergies for the backward direction. It is also unknown whether the poor lateral postural stability in narrow, compared with wide, stance can be explained solely by biomechanical constraints or whether abnormal proximal muscle activation critical for narrow, but not wide, stance also contributes. The current study investigates the spatial temporal pattern of postural muscle activation in response to multidirectional surface translation to better understand the neuromuscular basis for direction-specific postural instability in subjects with PD. Previous studies quantifying postural muscle activation in patients with PD have been limited to evaluating postural responses to anterior or posterior (AP) perturbations and to recording only a few muscles (Allum et al. 1988; Dietz et al. 1993; Horak et al. 1996). Examination of the coordination of many postural muscles in response to multidirectional perturbations is needed to understand the physiological mechanisms for postural instability across many fall directions in patients with PD. Studies of postural responses to AP perturbations have shown that, although the latencies of muscle activation in PD subjects are normal or earlier than normal, the force generated by this muscle activation is not normal. The earlier-than-normal activation of antagonists in response to forward body sway from surface translations results in postural cocontraction that increases postural stiffness and further decreases postural corrective forces in PD subjects (Horak et al. 1996). The magnitude of their agonist postural muscle responses may be either smaller (Horak et al. 1996) or larger (Beckley et al. 1991) than normal, depending on whether the postural muscle is stretched or shortened and on immediate prior experience and initial conditions. Thus results from studies of AP perturbations suggest that the basal ganglia are not responsible for creating or triggering automatic postural synergies but are involved in optimizing the magnitude and agonist–antagonist relationships for postural responses.

Analyzing the patterns of postural muscle activation in response to multiple directions of perturbation allows us to determine whether the problems with AP postural synergies generalize across directions for which different muscle types (flexors versus extensors and proximal versus distal) are responsible for returning the body to equilibrium. It also allows us to determine whether the cocontraction of postural muscles observed in PD subjects is due to an inability to generate directionally specific muscle synergy patterns. Can the patterns of postural muscle activation across multiple directions of...
displacement account for the particular instability that PD subjects show in response to backward and lateral sway displacements?

Multidirectional perturbations in healthy young adults have shown that postural muscle activation patterns are directionally specific and appropriately tuned for each direction of perturbation. Individual muscle responses tend to be maximally activated for one direction of perturbation (usually diagonal) and are only active in one quadrant of the postural sway directions with very little, or no, activation of antagonist muscles in the same directions (Henry et al. 1998b; Macpherson 1988). The paraspinal and abdominal muscles are an exception, showing substantial activity to both forward and backward postural sway directions, albeit at different latencies such that paraspinal and abdominal muscles never cocontract (Henry et al. 1998b). Latencies of automatic postural responses triggered by multidirectional surface translations ranged from 100 to 200 ms, which is longer than short-latency muscle stretch reflexes, but shorter than most voluntary muscle activations (Bloem et al. 1998b). A study of postural responses to multidirectional surface rotations in the elderly show that older adults have longer latencies and reduced magnitude of initial muscle responses (Allum et al. 2002).

Because of their inability to quickly change movement patterns, we predict that patients with PD will show less directionally specific postural muscle responses to multidirectional perturbations. This prediction is based on the hypothesis that the basal ganglia is important for quickly optimizing muscle synergy patterns for particular contexts (Chong et al. 1999, 2000; Horak et al. 1992b). Studies have shown that PD subjects have difficulty adapting the magnitude of postural responses to initial conditions. For example, suddenly changing the direction of surface perturbation from a translation to a rotation results in muscle responses more appropriate to the previous, than to the current, direction of perturbation in PD, but not in age-matched control subjects (Chong et al. 2000). Also, unlike healthy subjects who significantly reduced the magnitude of ankle muscle activation in response to surface displacements when they held onto a stable handle or sat on a stool, subjects with PD did not suppress ankle muscle responses in the first trial (Chong et al. 2000; Horak et al. 1992b; Schieppati and Nardone 1991). Only with five to seven repetitions did subjects with PD gradually reduce the unnecessary activity in leg muscles in response to perturbations (Chong et al. 2000). The inability of patients with PD to selectively activate only the most effective muscles for a task has also been shown for voluntary arm movements, suggesting an important role of the basal ganglia in optimizing muscle synergy patterns, regardless of whether for voluntary or postural tasks (Curra et al. 1997; Teulings et al. 1997).

Based on studies that illustrate how subjects with PD have difficulty changing postural response patterns to changes in initial conditions, we hypothesize that patients with PD will also have difficulty changing the size of their postural responses to changes in initial stance width. In young, control subjects, the magnitude of multidirectional postural responses are adapted to initial stance width within a single trial (Henry et al. 2001). When subjects stood with narrow, compared with wide, stance width, the bursts of muscle activation in response to surface translations were significantly larger in all directions, especially in trunk and ankle muscles, with no significant change in latency or muscle selection (Henry et al. 2001). The increased muscle activation in narrow stance was associated with much larger lateral flexion of the trunk, suggesting the addition of active trunk control to compensate for the decreased lateral stability (Henry et al. 2001).

Although narrow stance is more unstable than wide stance, as PD progresses and postural disorders become more severe, patients with late-stage PD tend to stand and to walk with narrower and narrower stance width (Nutt and Horak 1996). Axial rigidity in PD subjects may make it particularly difficult to control lateral postural stability because responses to lateral perturbations require more control from trunk and hip muscles than responses to AP perturbations that are generally controlled with an “ankle strategy” (Henry et al. 1998a; Horak et al. 1989a). An inability to increase the amplitude of postural responses and to control the trunk in narrow stance could be responsible for poor lateral stability and the sideways falls in patients with PD (van Wegen et al. 2001).

If the basal ganglia are important for focusing postural muscle activation and for changing muscle activity when the conditions change, PD subjects should have difficulty developing directionally specific postural muscle synergy patterns that change magnitude with changes in stance posture. Specifically, we investigated the ability of subjects with PD and aged-matched control subjects to adapt the pattern of activation of 16 muscles for narrow and wide stances, in response to eight directions of surface translation. The effects of these muscle activation patterns on surface reactive force patterns and postural stability in these same PD and elderly control subjects are presented in associated papers (Dimitrova et al. 2004; Horak et al., unpublished data). These results have also been presented as abstracts (Dimitrova et al. 2000; Mientjes et al. 2001).

**METHODS**

**Subjects**

All subjects gave informed consent for protocols approved by the Institutional Review Board of Oregon Health and Science University (OHSU). Thirteen age-matched (mean age 64.8 ± 2.0 yr) healthy control subjects (4 females and 9 males) were compared with 13 subjects (mean age 64.2 ± 2.1 yr) with mild to severe Parkinson’s disease (3 females and 10 males), recruited from the Movement Disorders Clinic of OHSU (see Table 1). The control subjects were mobile with no complaints of gait or balance difficulties and had normal perception of vibration and light touch on the feet. Inclusion criteria for the subjects with PD included diagnosis of idiopathic PD with clinical evidence of axial and/or postural problems and minimal tremor [Unified Parkinson’s Disease Rating Scale (UPDRS) tremor scores ranged from 0 to 2]. The subjects with PD had a wide range of postural motor disabilities, as determined by the (UPDRS Part III, Motor Scale) and its postural subcomponents (Item 22, Leg Rigidity; Item 28, Posture; Item 29, Gait; and Item 30, Postural Stability) and by the Hoehn and Yahr scale, both of which were administered just before experimental testing (Fahn and Elton 1987; Hoehn and Yahr 1967). Medical history and screening excluded persons with musculoskeletal psychological and other neurological disorders and those taking medications that could affect postural control, except for PD subjects who withheld their PD medications for this study. All PD subjects were taking carbidopa/levodopa and most were on various adjunctive agents: dopamine agonists, catechol-O-methyltransferase inhibitors, amantadine, and selegiline.
Experimental protocol

Subjects with PD were tested in the OFF state when they had been without antiparkinsonian medications for 12 h overnight. Before they were tested, they rated their parkinsonian condition as 3–5 on a scale from 0 (worse OFF state) to 10 (best ON state).

Subjects stood on a platform moved by hydraulic actuators with a sigmoidal signal (Henry et al. 2001). Subjects were exposed to five sets of trials; each set consisted of eight directions of platform translation, presented randomly. Figure 1 shows the eight directions of body sway imposed by the surface translations (R, right; FR, forward-right; F, forward; FL, forward-left; L, left; BL, backward-right; B, backward; BR, backward-right). Postural responses were tested in two stance conditions: first, in a narrow stance with inside of feet parallel to each other and 4.5 cm between them and then in a wide stance with feet parallel and 26 cm between them. Stance width was blocked to show quickly PD and control subjects adapted the magnitude of their responses when the biomechanical conditions of the task changed. Subjects were encouraged to rest whenever fatigued and at least after every set of eight trials.

Subjects were instructed to focus on an art poster 3 m in front of them with arms hanging at their sides and to keep their balance without moving their feet during the trial. To assure the same AP and lateral weight distribution, the experimenters coached subjects based on weight distribution monitored on an oscilloscope prior to each trial. A ramp-and-hold signal was used to translate the platform 9 cm over 1000 ms with a peak acceleration of 2 m/s². To avoid excessive compensatory stepping or falls in narrow stance, these perturbation characteristics are the same amplitude, but twice the duration and 50% of the peak acceleration, from our previous studies of multidirectional translations in young, healthy adults (Henry et al. 1998a,b, 2001).

Data collection and analysis

The electromyographic activity (EMG) was collected at 480 Hz from right and left: lumbar erector spinae (ESP), rectus abdominis (ABD), tensor fascia latae (TFL), adductor longus (ADD), tibialis anterior (TIB), and soleus (SOL). Surface EMG activity was recorded differentially with electrodes placed 2 cm apart along the length of each muscle belly, with a ground electrode at C7. Control subjects’

### TABLE 1. Characteristics of subjects with Parkinson’s disease

<table>
<thead>
<tr>
<th>Subjects with PD</th>
<th>Age, yr</th>
<th>Duration of PD, yr</th>
<th>Motor UPDRS Score</th>
<th>Posture (0–4)</th>
<th>Postural Stability (0–4)</th>
<th>Gait (0–4)</th>
<th>Leg Rigidity (0–8)</th>
<th>Hoehn and Yahr Stage</th>
<th>No. Falls in Narrow Stance</th>
<th>No. Falls in Wide Stance</th>
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<tr>
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<td>6</td>
<td>4</td>
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<td>69</td>
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Mean ± SE: 64.2 ± 2.1 13.9 ± 3.1 46.3 ± 4.5 1.9 ± 0.3 1.8 ± 0.3 2.2 ± 0.2 4.3 ± 0.8 2.8 ± 0.3 52 Falls 11 Falls

PD, Parkinson’s disease; UPDRS, Unified Parkinson’s Disease Rating Scale; F, forward; LF, left forward; L, left; B, backward; LB, left backward; R, right; RB, right backward.
skin impedance ranged from 5.7 to 9.3 KΩ and PD subjects’ skin impedance ranged from 6.6 to 10.6 KΩ. No significant differences between control and PD group muscle impedances were found.

To minimize difference in EMG magnitude due to amplifier characteristics, the same amplifiers were used for each muscle for each subject, and background amplifier and preamplifier biases were subtracted from each channel. The EMG signals were amplified (5,000–20,000×), band-pass filtered (15–2,500 Hz), full-wave rectified, and integrated at a cut-off frequency of 100 Hz. Subjects were asked to selectively contract each of the 12 muscles before the experiment, and the EMGs were monitored on an oscilloscope to verify signal quality and to insure minimal cross-talk between muscles.

The magnitude of muscle responses to multidirectional perturbations in each trial was determined by integrating the EMG (IEMG) during a fixed time window (70–470 ms) following the onset of surface translation (Fig. 1). This window was initiated at the time of the earliest, medium latency, postural muscle response and included most of the activity responsible for corrective forces during the platform translation (Henry et al. 1998a). Similar results were found for both the first 100 ms and the entire EMG burst (400 ms), so we only report the EMG integral for the entire response responsible for correction equilibrium. The background level of EMG activity was estimated as the mean value over a 500-ms period prior to platform onset and was subtracted from the active postural response. “Muscle tuning curves” across directions were determined for both stance conditions as the subject mean and group mean IEMG magnitudes plotted in polar coordinates against the direction of body sway caused by each platform displacement (Macpherson 1988; Henry et al. 1998b). The EMG latency of muscle responses to platform translation was identified by inspection of each trial for the first burst above the baseline ≥ 50 ms following translation onset.

The main (preferred) direction of muscle activity for each muscle was estimated as a vector sum of the IEMG responses to each direction of body sway induced by the platform translations. Directions of body sway in which each muscle showed <50% of its maximal activity across directions in the control group were considered directions of antagonist activity for that muscle (see STATISTICAL ANALYSIS). To compare changes in EMG activity from wide to narrow stance conditions between patients and control groups, the differences between EMG responses to various directions for each muscle were normalized to the corresponding responses in narrow stance for each subject.

Statistical Analysis

The effects of PD, stance width, and direction of body sway on each muscle’s EMG magnitudes and latencies were analyzed with a three-way ANOVA (two subject groups × two repeated stance widths × eight repeated directions). Bonferroni corrections for multiple comparisons maintained the a priori type I error rate at α 0.05. Mann–Whitney nonparametric tests were used to compare the mean background level of EMG activity between subject groups and between stance conditions as the subject mean and group mean IEMG magnitudes at different directions in which this muscle (see STATISTICAL ANALYSIS). To determine whether the quantified postural measurements are related to the severity of PD, the difference in IEMG between narrow and wide stance, as well as the muscle activity in nonpreferred directions (antagonist), were correlated with the clinical scores from Table 1 using the Spearman rank correlation.

RESULTS

Subjects with PD have similar postural synergies as control subjects

Subjects with PD fell or took a step in 10% of the trials in narrow stance and 2% of the trials in wide stance, especially during backward and lateral sway (Table 1). In contrast, control subjects maintained their balance in all trials. EMG analysis included all trials, with and without falls, because no significant differences in EMG patterns, amplitudes, or latencies were found between fall and nonfalling trials.

The patterns of muscle activation in response to each direction of perturbation were similar in control and PD subjects. In both control subjects and PD subjects, an EMG burst of activity was followed by tonic activity for the duration of the surface translation. Figure 2 shows the mean EMG activity for each subject group in response to a backward-right body sway induced by a forward-left platform translation. For this direction of body sway, both subject groups activated primarily the ankle muscles, R SOL and R TIB, and the proximal muscles, R TFL and R ESP muscles on the side of the body loaded by passive weight shift to the right. Although the R ADD and R ABD muscles were also activated in response to backward-right sway, the corresponding muscles on the unloaded, left side were about twice as active and activated earlier. The other muscles on the unloaded, left side were minimally active to this direction of body sway.

The PD subjects showed polar plot shapes similar to control subjects for all but the ABD muscles. Polar plots of IEMG across eight directions of body sway show that muscles were primarily activated in response to three contiguous directions of translation, except the ESP muscles, which tended to be activated in response to both forward and backward translations (Fig. 3). Unlike controls, PD subjects showed large activation of ADB for all eight directions.

The magnitude of muscle activation was similar in the two groups, except the trunk muscles (ESP and ABD) were larger and TFL was smaller in the PD than in the control group (Figs. 2 and 3). PD subjects’ EMG responses to multidirectional perturbations were larger than responses in control subjects for the trunk muscles in both stance widths (main effect: \(F_{(1,384)} = 38\) for R ESP; \(F_{(1,384)} = 41\) for L ESP, \(F_{(1,384)} = 39\) R ABD; and \(F_{(1,384)} = 15\) for L ABD; \(P < 0.001\)). In contrast to the trunk muscles, the EMG activity of TFL in the PD subjects was generally smaller than that in the control subjects but depended on the direction of body sway (interaction: R TFL \(F_{(7,384)} = 4.7; P < 0.001\); L TFL \(F_{(7,384)} = 5.2; P < 0.001\)). Compared with the control subjects, PD subjects showed smaller TFL responses in the directions of body sway in which this muscle was an agonist (i.e., directions in which it had more than 50% of its maximum activity; R, BR, and FR sway for R TFL; and L, BL, and FL sway for L TFL). Compare the relative size of polar plots for L ESP, L ABD, and L TFL with the same scale for control and PD subjects in Fig. 3.

PD subjects show impaired adaptation to stance width

In both subject groups, changing from wide to narrow stance increased EMG activity significantly (\(P < 0.001\)) for all the muscles tested (Figs. 2 and 3). The relative percentage of change in IEMG from wide to narrow stance is compared for
the control and PD groups in Fig. 4. Notice that some muscles, such as TFL and TIB, increased their IEMG as much as 80–90%, whereas ABD changed only 40% from wide to narrow stance. As expected, the largest change in IEMG due to stance width was always in response to lateral directions of body sway, followed by diagonals. For the SOL and TFL muscles, the largest changes due to stance width were only for their largest lateral activation direction, whereas, for all other muscles, large changes in IEMG due to stance width occurred for both lateral directions (Fig. 4).

In the PD subjects, the relative increase in IEMG from wide to narrow stance was significantly smaller compared with the relative increase in the control subjects for the SOL, TIB, ADD, and ESP muscles (Fig. 4 and Table 2). This reduced ability to change the magnitude of IEMG for changes in initial posture was not likely due to an inability to increase IEMG since PD subjects showed a larger-than-normal magnitude of some muscle activations (see R ESP and R ABD in Fig. 2).

There were no significant interactions between direction and subject groups ($P > 0.7$).

The ability of PD subjects to increase the magnitude of their TIB muscles’ EMG activity from wide to narrow stance was inversely correlated with their Hoehn and Yahr scores (R TIB: $r = -0.45$, $P < 0.05$; L TIB: $r = -0.60$, $P < 0.01$) and also with their total motor UPDRS scores and with their UPDRS posture, gait, and postural stability scores ($P < 0.01$). The other muscles did not consistently show significant correlations with the clinical severity of Parkinsonism.

Both PD and control subjects showed the same direction of maximal muscle activation

The primary direction of each muscle’s activation, defined as a vector sum of the IEMG activity in response to the eight directions of body sway, was not significantly different between subject groups (between groups $P$ values ranged from

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FIG. 2. Control and Parkinson’s disease (PD) group average EMG responses in right-sided (loaded) muscles to right-backward body sway. Responses during narrow stance width are black. Responses to wide stance are gray. Each muscle plot is scaled with reference to its maximum activity across all directions in the narrow stance for both subject groups. Shaded rectangle denotes 70- to 470-ms period of integrated EMG. Platform translation onset is 0 ms and ends at 1000 ms. R ESP, right erector spinae; R ABD, right abdominals; R TFL, right tensor fascia latae; R ADD, right adductor; R TIBI, right tibialis; R SOL, right soleus.
Narrowing the base of support from wide to narrow changed the direction of muscle activation from AP to lateral/diagonal for the SOL, TIB, and ESP muscles in both control and PD subjects. For example, the SOL muscle changed its preferred direction of activation from 29° to 59° in wide stance to 60° in narrow stance.

**TABLE 2.** ANOVA results for change in IEMG from wide to narrow stance

<table>
<thead>
<tr>
<th>Muscles</th>
<th>Control Versus PD Subjects $F_{(1,192)}$</th>
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<tbody>
<tr>
<td>R SOL</td>
<td>8.9*</td>
</tr>
<tr>
<td>L SOL</td>
<td>5.1†</td>
</tr>
<tr>
<td>R TIB</td>
<td>9.7*</td>
</tr>
<tr>
<td>L TIB</td>
<td>4.4†</td>
</tr>
<tr>
<td>R TFL</td>
<td>0.4</td>
</tr>
<tr>
<td>L TFL</td>
<td>2.3</td>
</tr>
<tr>
<td>R ADD</td>
<td>8.0*</td>
</tr>
<tr>
<td>L ADD</td>
<td>4.4†</td>
</tr>
<tr>
<td>R ESP</td>
<td>25.0‡</td>
</tr>
<tr>
<td>L ESP</td>
<td>28.0‡</td>
</tr>
<tr>
<td>R ABD</td>
<td>0.2</td>
</tr>
<tr>
<td>L ABD</td>
<td>0.2</td>
</tr>
</tbody>
</table>

Values are $F_{(1,192)}$. IEMG, integrated electromyographic activity; PD, Parkinson’s disease; SOL, soleus; TIB, tibialis anterior; TFL, tensor fascia latae; ADD, adductor longus; ESP, erector spinae; ABD, rectus abdominis. * $P < 0.01$. † $P < 0.05$. ‡ $P < 0.001$. 

**FIG. 3.** Average control and PD group polar plots of the magnitude of integrated EMG (IEMG) activity for each direction of body sway in left side muscles. IEMG in narrow stance is indicated by black lines, and wide stance by gray lines, with the difference between narrow and wide shaded. All polar plots are scaled to the coordinate system shown in the upper left plot. L ESP, left erector spinae; L ABD, left abdominal; L TFL, left tensor fascia latae; L ADD, left adductor; L TIB, left tibialis; L SOL, left soleus.

**FIG. 4.** Polar plots showing mean difference in IEMG between narrow and wide stance (as a percentage of IEMG in narrow stance) in the control group (black line) and the PD group (gray line) for 8 directions of body sway, as indicated in the upper left plot. Muscle abbreviations are the same as in the legend to Fig. 2.
for distal muscles during AP body sway and for proximal muscles during lateral body sway. For example, TFL was unilaterally activated in the loaded leg in the elderly control subjects, but TFL was often bilaterally activated for many directions of body sway in the PD subjects. Figure 6B illustrates excessive TFL–ADD EMG coactivation in a representative PD subject (Subject 2) from an average of five trials in response to leftward sway. This example demonstrates how excessive coactivation occurs in subjects with PD due to small-

**Muscles are activated in more directions in PD than control subjects**

Compared with the control subjects, the subjects with PD showed more coactivation around both distal and proximal joints and between the same muscles on both sides of the body. Figure 6A shows examples of polar plots comparing activation areas between antagonist muscle pairs in a representative control subject and a representative subject with PD (Subject 5). Notice that the PD subject has a larger overlap of antagonist muscle tuning curves because the muscles were activated similarly in many nonpreferred directions. Such coactivation was observed for different functional antagonist muscles, such as

**FIG. 5.** Direction of IEMG maximum activation for each muscle as a group mean net vector sum for control subjects and subjects with PD in (A) narrow and (B) wide stance. The directions of body sway and muscle abbreviations apply to all 4 plots. Note: Because ESP and ABD muscles have bidirectional activity, the maximum activation is calculated for both forward and backward body sway. The abbreviations are as in the legends to Figs. 2 and 3.

**FIG. 6.** A: sample polar plots of normalized IEMG magnitude illustrating excessive activation in nonpreferred directions of activity in antagonist muscles around the pelvis (L TFL/R TFL), thigh (L TFL/L ADD), and ankle (L SOL/L TIB) in a representative subject with PD (Subject 5), but not in the control subject. Each muscle is scaled to its own maximal activity. Shaded area indicates coactivation of muscles. B: sample average EMG activity from 5 trials, illustrating excessive coactivation of L TFL and L ADD in response to leftward sway in a representative subject with PD (Subject 2) compared with a representative control subject. The analyzed period of IEMG is indicated as in the legend to Fig. 1.
er-than-normal agonist activity as well as to larger-than-normal (and earlier than normal) antagonist activity. 

For the directions that the muscles in the control group responded with <50% of their maximal activity (considered antagonistic muscle activity, see METHODS), the PD subjects showed significantly larger IEMG responses than did the control subjects (Table 3). In narrow stance, PD subjects showed significantly larger antagonist activity in TFL, ADD, and ABD muscles than did control subjects. In wide stance, the same three muscles, as well as the TIB and ESP muscles of PD subjects, showed larger than normal antagonist muscle activity (Table 3). 

The magnitude of PD subjects’ right and left ESP IEMG activity in the nonpreferred directions (i.e., coactivation) was significantly correlated with their Hoehn and Yahr scores ($r = 0.30, P < 0.05$) and with their Motor UPDRS scores ($r = 0.50, P < 0.001$). The other five muscles did not show any significant correlation between the antagonist muscle activity and the severity of the disease.

**Background EMG activity was larger in PD subjects than control subjects**

Subjects with PD showed larger mean background EMG activity than did control subjects bilaterally for TIB, ADD, and ESP in both stance widths ($P < 0.05$). There was a nonsignificant trend for the background EMG levels to increase in narrow, compared with wide, stance, but this trend was significant only in bilateral TIB in PD subjects ($P < 0.01$) and in bilateral SOL in the control subjects ($P < 0.01$).

**EMG response latencies are shorter in PD subjects compared with control subjects**

Overall, subjects with PD showed shorter EMG latencies than the control subjects in both narrow and wide stance for all muscles tested except for ESP (group main effect $P < 0.005$ for R TFL and $P < 0.001$ for the rest of the muscles). The differences in EMG latencies between the subject groups were significantly affected by the direction of body sway for all muscles except TIB and ESP for TFL muscles. Only when they were used as antagonists (i.e., directions of body sway for which muscle activity was <50% of the maximum) did PD subjects activate muscles earlier than did the control subjects. Table 4 presents the directions of body sway in which the subjects with PD showed EMG latencies significantly shorter than did the control subjects. No main effect of stance width and no interaction between the effect of subject group and stance width was found for EMG latencies. For all tested directions of body sway, the ADD and ABD muscles in subjects with PD showed shorter latencies compared with those of the control subjects. Although the latency of ESP was not different between groups across all directions of body sway and stance widths (insignificant main effect), there was a significant three-way interaction among group, direction, and stance width (interaction: for R ESP, $F_{(7,352)} = 2.2, P < 0.05$; for L ESP, $F_{(7,352)} = 2.9, P < 0.01$). Thus, in narrow stance, the EMG onset of ESP appeared earlier in PD subjects than in control subjects when it acted as an antagonist for lateral sway whereas it was activated later when it acted as an agonist. In wide stance, ESP latencies were similar between subject groups (Table 4).

All muscle latencies, except ABD, were affected similarly.

**TABLE 3. Antagonist muscle activity (% of the maximal IEMG) in subject groups**

| Muscles | Right Side | | Left Side | |
|---------|------------|------------|------------|
|         | Control Group, % | PD Group, % | $F$         | Control Group, % | PD Group, % | $F$         |
| Narrow Stance | TFL | 22 | 40 | $F_{(4,120)} = 38*$ | 24 | 31 | $F_{(4,120)} = 81*$ |
|          | ADD | 35 | 54 | $F_{(4,120)} = 21*$ | 37 | 52 | $F_{(4,120)} = 15*$ |
|          | ABD | 48 | 57 | $F_{(4,120)} = 42$ | 45 | 62 | $F_{(4,120)} = 16*$ |
| Wide Stance | TIB | 18 | 26 | $F_{(4,144)} = 18*$ | 16 | 28 | $F_{(4,144)} = 35*$ |
|          | TFL | 34 | 59 | $F_{(4,120)} = 38*$ | 35 | 49 | $F_{(4,120)} = 14*$ |
|          | ADD | 28 | 54 | $F_{(4,120)} = 54*$ | 25 | 43 | $F_{(4,120)} = 37*$ |
|          | ESP | 39 | 50 | $F_{(3,96)} = 62$ | 42 | 55 | $F_{(3,96)} = 91*$ |
|          | ABD | 42 | 54 | $F_{(4,120)} = 87$ | 37 | 55 | $F_{(4,120)} = 20*$ |

Values are averages across the directions that the control group activated muscles less than 50% of their maximum. For abbreviations, see Table 2. Degrees of freedom are 4,120 for TFL, ADD, and ABD; 5,144 for TIB, and 3,96 for ESP. $^*$ $P < 0.001$. † $P < 0.01$. ‡ $P < 0.05$. 

**TABLE 4. EMG latencies in control and PD subjects for directions with significant differences between groups**

<table>
<thead>
<tr>
<th>Muscles</th>
<th>Direction of Sway</th>
<th>Control Subject Latency, ms</th>
<th>Subjects with PD Latency, ms</th>
</tr>
</thead>
<tbody>
<tr>
<td>R SOL</td>
<td>L, LB, R, RB, LB</td>
<td>152 ± 5</td>
<td>138 ± 5</td>
</tr>
<tr>
<td>L SOL</td>
<td>R, RB, LB, LB</td>
<td>154 ± 5</td>
<td>139 ± 4</td>
</tr>
<tr>
<td>R TIB</td>
<td>RF, LF, RF, LF</td>
<td>142 ± 3</td>
<td>122 ± 2</td>
</tr>
<tr>
<td>L TIB</td>
<td>RF, RF, LF, LB</td>
<td>133 ± 3</td>
<td>123 ± 2</td>
</tr>
<tr>
<td>R TFL</td>
<td>RF, LF, F, R, LB</td>
<td>142 ± 2</td>
<td>135 ± 2</td>
</tr>
<tr>
<td>L TFL</td>
<td>RF, LF, F, R, RB</td>
<td>144 ± 2</td>
<td>126 ± 2</td>
</tr>
<tr>
<td>R ADD</td>
<td>All</td>
<td>140 ± 2</td>
<td>116 ± 1</td>
</tr>
<tr>
<td>L ADD</td>
<td>All</td>
<td>138 ± 2</td>
<td>118 ± 1</td>
</tr>
<tr>
<td>R ABD</td>
<td>All</td>
<td>155 ± 2</td>
<td>122 ± 2</td>
</tr>
<tr>
<td>L ABD</td>
<td>All</td>
<td>152 ± 3</td>
<td>116 ± 2</td>
</tr>
<tr>
<td>Narrow Stance</td>
<td>R ESP</td>
<td>139 ± 7</td>
<td>163 ± 8</td>
</tr>
<tr>
<td></td>
<td>L ESP</td>
<td>186 ± 14</td>
<td>159 ± 5</td>
</tr>
<tr>
<td></td>
<td>L</td>
<td>140 ± 6</td>
<td>161 ± 6</td>
</tr>
</tbody>
</table>

Wide Stance

| R ESP   | 139 ± 4                     | 146 ± 5                      |
| L ESP   | 148 ± 9                     | 146 ± 6                      |

Values are means ± SE. The latencies combine values for narrow and wide stance except ESP because no significant main effects of stance width or interaction effects were found. For abbreviations see Tables 1 and 2.
by stance width in control and PD subjects. For example, in both groups, the latency of SOL EMG activity increased from narrow to wide stance when acting as an antagonist. Also, in both subject groups, ESP was activated earlier in wide than narrow stance. However, ABD was correspondingly activated later in wide stance than in narrow stance in control subjects, but not in subjects with PD.

**Summary**

PD subjects select similar postural muscle patterns as age-matched control subjects across all directions of surface perturbations, although the torso muscles were overactive and the hip muscles were underactive. The main deficits in PD subjects were impaired ability to increase EMG responses from wide to narrow stance, cocontraction of antagonists, and larger background EMG.

**Discussion**

**Role of the basal ganglia**

The excessive activation of PD subjects’ antagonist muscles across many perturbation directions and their limited ability to modify postural muscle activity with changes in stance width in our study support the hypothesis that the basal ganglia are important for optimizing muscle activation patterns by quickly switching motor patterns when the task or environment changes.

Previous work on voluntary arm movements in PD subjects as well as behavioral and electrophysiological studies in mammals also supports this hypothesis (Brooks 2001; Krebs et al. 2001; Kropotov and Etlinger 1999). Functional imaging studies in human and nonhuman primates suggests that tonic dopamine release by the basal ganglia is responsible for focusing the patterns of muscular activity used to reach a goal by filtering out unwanted muscle activation, whether the movement is self-initiated or externally cued (Brooks 2001; Juelpner and Weiller 1998; Kropotov and Etlinger 1999). Recording of dopamine neurons in primates suggests that phasic dopamine release in the basal ganglia is responsible for focusing the patterns of muscular activity used to reach a goal by filtering out unwanted muscle activation, whether the movement is self-initiated or externally cued (Brooks 2001; Jueptner and Weiller 1998; Kropotov and Etlinger 1999). Recording of dopamine neurons in primates suggests that phasic dopamine release in the basal ganglia is important for changing motor set in novel situations and studies of learning arm movements in patients with PD support this hypothesis (Brooks 2001; Laforce and Doyon 2002). Our previous studies of AP postural responses in parkinsonian subjects also showed deficits in postural muscle activation patterns and in changing postural set deficits that suggest that the basal ganglia plays similar roles in externally triggered postural motor control (Chong et al. 1999, 2000; Horak et al. 1992b).

Although the current study of postural responses to multidirectional surface translations in PD subjects support these hypotheses, some characteristics of postural responses are relatively unaffected by parkinsonism, perhaps because externally triggered movements can also be controlled via extrastriatal pathways (Hanakawa et al. 1999; Thaler and Passingham 1989). For example, arm movements and voluntary step initiation that exhibit slow initiation and poor force output may exhibit normal initiation and force output when they are triggered by an external stimulus, such as a tone, a somatosensory cue, or a visual cue (e.g., a stripe on the floor) (Burleigh-Jacobs et al. 1997; Jahanshahi et al. 1995). It has been suggested that externally triggered movements utilize cerebellar–premotor cortex pathways that may be intact in subjects with PD and that internally initiated movements require basal ganglia–supplementary motor cortex pathways that are impaired (Jahanshahi et al. 1995). Thus postural responses that are externally triggered from a surface translation may be less affected in PD subjects than self-initiated postural adjustments, such as postural preparation for step initiation or voluntary rising onto toes (Burleigh-Jacobs et al. 1997; Frank et al. 2000).

**Agonist Selection**

Although the PD subjects in this study had significant balance or axial disorders in clinical testing (Table 1), many aspects of their postural muscle synergies were surprisingly normal. The direction of maximum activation for each muscle was not significantly different between elderly control and PD subjects. For example, the age-matched elderly control and PD subjects showed maximum activation of SOL and TIB for diagonal body sway and maximum activation of ADD for the backward body sway direction, orthogonal to this muscle’s line of pull. The directions of maximal muscle activation in the PD and elderly control subjects were also similar to the muscle activation directions from our studies of young control subjects (Henry et al. 1998b).

PD subjects activated the same set of agonist postural muscles at the Shank, thigh, and trunk as the control subjects. Thus the general shape of the IEMG polar tuning curves were similar for the PD and elderly control subject groups. The only difference was that PD subjects had larger relative activity of muscles for directions of body sway in which the muscles acted as antagonists (see Muscle Coactivation). The muscle tuning curve with the largest difference between the PD and control groups was the ABD muscle, which showed bidirectional, instead of unidirectional, tuning curves in the PD subjects. However, young control subjects in a previous study also showed bidirectional ABD muscle activation like the PD subjects in this study (Henry et al. 1998b). The lack of an early ABD muscle activation in response to forward sway in the elderly control subjects of the current study may be due to the slower platform acceleration in this study. The forward sway perturbations may not have been challenging enough to require elderly control subjects to add a “hip strategy” to the “ankle strategy” (Horak et al. 1989a). In contrast, these perturbations were just at the threshold for PD subjects to take a step or fall, especially during narrow stance and for the backward sway direction.

Thus PD did not appear to significantly disrupt the muscles selected to resist each direction of postural perturbation. This result supports the hypothesis that the basal ganglia are not critical for building or storing externally triggered, automatic postural synergies. Muscle selection is probably determined based on the pattern of peripheral somatosensory stimulation that selects and triggers a synergy that is centrally organized at the brain stem level (Horak and Macpherson 1996; Keck et al. 1998; Lee 1984; Macpherson 1991).

**Agonist latencies**

Latencies of agonist muscle synergy activation were also similar between PD and elderly control subjects for all directions of perturbation; that is, for each muscle’s maximum
activation direction and nearby directions. Thus even though bradykinesia responses to an external perturbation are not
responsible for PD subjects’ problems with balance control. Previous studies have shown that elderly subjects have longer postural muscle latencies than younger subjects (Allum et al. 2002) and the latencies of the elderly control subjects in our study were longer than for previous studies of young subjects (Henry et al. 1998b). Previous studies in humans and cats show that the latency of postural responses to surface displacements depends heavily on the conduction velocity of somatosensory pathways from the periphery (Inglis et al. 1994). This study shows that agonist postural response latencies do not depend on intact pathways in the basal ganglia.

**Magnitude of postural synergies**

The sizes of postural muscle responses (IEMGs) were also similar between the PD and elderly control groups across perturbation directions, except for the proximal, axial muscles. The ESP and ABD muscles showed larger than normal activation, whereas the TFL showed smaller than normal activation in subjects with PD. These abnormal proximal muscle response magnitudes suggest that the basal ganglia may play a role in regulating postural trunk gain control or that subjects with PD compensate for other postural deficits by altering the magnitude of these proximal, postural muscles. The abnormal magnitude of proximal muscle activation in PD subjects is consistent with their difficulty in controlling balance laterally and backward in stance because these directions require more control from axial trunk muscles than does forward sway (Henry et al. 1998b).

Although subjects with PD had larger-than-normal background EMG activity, this cannot be responsible for the larger-than-normal medium latency bursts of activity in the trunk muscles because IEMG activity in the bursts was estimated above the background level. Furthermore, Bloem et al. (1993) demonstrated that the magnitude of medium- and long-latency responses to surface perturbations were not dependent on background EMG levels in subjects with PD, unlike short latency muscle reflexes (Bloem et al. 1993). It is unlikely that exaggerated shortening reactions were responsible for the larger-than-normal ESP activity since it was overactive both when it was lengthened with forward body sway as well as when it was shortened with backward body sway. We cannot rule out, however, that the stooped postural alignment of the subjects with PD contributed to the larger-than-normal trunk and smaller-than-normal TFL responses (Bloem et al. 1999). Despite the excessive activity of the trunk muscles and reduced activity of TFL across many directions, the direction of maximum activation of these muscles were normal in the PD subjects, consistent with reliable spatial somatosensory tuning for directional gain control (Henry et al. 1998b; Macpherson 1988).

**Muscle coactivation**

Unlike age-matched control subjects, PD subjects coactivated postural muscles. Coactivation occurred because the antagonist muscles were activated at larger than normal magnitudes and at shorter-than-normal latencies. Except for SOL, PD subjects had significantly larger IEMGs for every recorded muscle when it acted as an antagonist (Table 3). The torso muscles in this study, ESP and ABD, were particularly active in PD subjects, with less directional specificity than in elderly control subjects, consistent with the axial rigidity so commonly observed clinically in subjects with PD (Van Emmerik et al. 1999). The 33–36 ms earlier muscle initiation in PD subjects’ torso muscles resulted in antagonists being activated during the medium-latency, agonist burst that generally lasted 75 ms (Diener et al. 1988). In contrast, in control subjects, trunk antagonists were reciprocally activated with a 46- to 60-ms delay (see Table 4). The result of earlier-than-normal antagonist muscle activation in PD subjects was a nearly simultaneous activation of agonists and antagonists in the trunk for lateral perturbations in both narrow and wide stance.

Antagonist responses to forward body sway have been previously reported to be earlier than normal in PD subjects (Dietz et al. 1993; Horak et al. 1996). It is possible that the shorter antagonist muscle latencies in subjects with PD were partially due to increased tonic background activity in these muscles. It is also possible that the shorter latencies reflect loss of muscle selectivity with basal ganglia disease. The basal ganglia may be responsible for the inhibition of muscles not integral to the movement (Filion 2000). Support for a role of the basal ganglia in inhibiting extraneous muscle activation in response to joint displacement come from studies of a parkinsonian model in monkeys. A single globus pallidus neuron in an 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine–poisoned monkey often was activated in response to movement of more joints and to more directions of joint motion (Filion et al. 1988). This suggests that the basal ganglia are important for filtering out somatosensory noise and producing proprioceptive-specific antagonist muscle activation. Thus, although the basal ganglia may not be important for selection of appropriate agonist muscles for postural synergies, it may be important for inhibiting unneeded or inappropriate muscle activation via a mechanism such as surround-inhibition.

It is also possible that subjects with PD showed excessive coactivation in our study because the perturbation directions were randomly presented and, therefore, required quick changes in strategy selection (Horak et al. 1989a). Subjects with PD have been shown to have difficulty changing postural strategies quickly in the first trial after initial conditions change (Bloem et al. 1995; Chong et al. 1999, 2000; Horak et al. 1992b; Schieppati and Nardone 1991). However, if subjects with PD are repeatedly exposed to the same condition, they gradually alter the magnitude of their postural responses and approach normal magnitudes within several trials. This suggests that subjects with PD retain the ability to alter postural responses based on prior experience, probably using cerebellar pathways (Horak and Diener 1994; Timmann and Horak 1997). Thus, if we had presented subjects with PD with predictable, repeated perturbations in one direction, it is possible that they would have eventually produced more direction-specific postural responses with less coactivation of antagonists.

Coactivation may also serve as a default strategy to defend against potential perturbations from any direction when direction-specific strategies are not immediately available. Coactive stiffening, however, is an ineffective strategy for regaining equilibrium in response to perturbations in multiple directions because coactive stiffening cannot produce directionally spe-
specific surface reactive forces at the ground to return the center of mass to equilibrium (Henry et al. 1998b; Horak and Macpherson 1996). Examination of surface reactive forces under each leg in these subjects revealed abnormal magnitude and directions of forces in PD compared with control subjects (Dimitrova and Horak 2003). In addition to increased coactivation of postural muscle bursts, increased stiffness of inherent passive elastic muscle elements and increased stiffness from increased background tonic activity also play a role in postural stiffness in subjects with PD. Studies have shown reduced initial center of mass velocity, decreased displacement of ankle joints, and increased surface shear forces in the passive period (prior to EMG onset) in response to perturbations in subjects with PD (Dietz et al. 1988; Dimitrova and Horak 2003; Frank et al. 2000). Fear of falling could also contribute to excessive coactivation in postural responses because subjects with PD have been shown to have less confidence in their balance than age-matched controls (Adkin et al. 2003), and control subjects with fear of falling have more coactivation and stiffness in response to surface displacements (Adkin et al. 2000; Maki et al. 1991).

**PD subjects show poor adaptation to changes in stance width**

When stance width was altered, subjects with PD showed significantly less change in IEMG than elderly control subjects in four of six muscle pairs: ESP, ADD, SOL, and TIB (see Fig. 4). The reduced ability of subjects with PD to change the magnitude of EMG with changes in stance width was not due to the saturation of EMG magnitude, since subjects with PD activated some muscles (e.g., ESP and ABD) more than did control subjects.

The reduced ability to alter postural responses with changes in stance width could be due to the impaired ability of subjects with PD to change their central set quickly. PD subjects do not suppress the magnitude of postural responses in the legs as much as do age-matched control subjects when they sit, hold onto a stable support, are exposed to a surface rotation following surface translation, or are instructed to voluntarily reduce the size of their postural response (Bloom et al. 1995; Chong et al. 1999, 2000; Horak et al. 1992; Schieppati and Nardone 1991). However, PD subjects retain the ability to gradually modify postural response magnitude when the same condition is presented sequentially. The wide and narrow stance width in the current study was presented sequentially over many trials (7 repetitions \(\times\) 8 directions = 56 trials in each stance width), which should have allowed PD subjects to use prior experience to modify postural response magnitude appropriately for each stance width. Perhaps we would have observed even more impaired ability to adapt postural response to changes in stance width if we had altered the stance width randomly.

The poor ability of subjects with PD to alter postural synergies for changes in initial stance posture may also be related to their reduced ability to use proprioceptive information to select postural muscle activation patterns based on the specific pattern of displacements (Dinnerstein et al. 1962; Moore 1987; Schneider et al. 1982b). PD subjects have impaired proprioceptive guidance of movement (Demirci et al. 1997; Dinnerstein et al. 1962; Jobst et al. 1997; Khudados et al. 1999; Moore 1987; Schneider et al. 1982a). For example, subjects with PD, unlike control subjects, do not alter voluntary ankle or arm movements when muscle spindle feedback changes in the presence of muscle vibration (Khudados et al. 1999; Richards and Cody 1997). In addition, PD subjects overestimate the extent of movements as if they mismatch proprioceptive feedback and corollary discharge (Moore 1987). The specific pattern of proprioceptive stimulation resulting from joint and muscle displacements help select and shape the pattern of centrally programmed postural synergies (Horak and Macpherson 1996). Our previous study showed that PD subjects could use proprioceptive inputs to scale the magnitude of their initial postural responses but were unable to scale to predictable displacement amplitudes, based on central set from prior experience (Horak et al. 1996). This previous study suggests that the mechanisms for scaling postural burst magnitude based on peripheral sensory information from proprioceptive coding is intact in PD subjects, whereas central mechanisms responsible for scaling postural response patterns to remembered displacement amplitudes are deficient.

**Clinical implications**

There is no evidence from this study that the mechanism for falling in different directions during stance may be due to different underlying physiological mechanisms in PD subjects. Our associated studies of center of body mass control by surface reactive forces show that these PD subjects were significantly less stable in response to backward sway and to lateral sway in narrow stance (Horak et al., unpublished data). Nevertheless, the problems of abnormal magnitude of muscle activation, coactivation of antagonists and inability to change the magnitude of muscle activation with initial stance position, apply to all directions of postural sway. Since proximal muscles were more likely than distal muscles to show coactivation, these proximal muscles may be more critical for control of backward sway and lateral sway in narrow stance than for other directions (Henry et al. 1998b). Perhaps the increased propensity of PD subjects to fall with backward perturbations is due to their tendency to hold their body CoM posteriorly (Schieppati and Nardone 1991), their stooped posture, and the biomechanical difficulty of exerting adequate postural reactive torques with the lever arm of the foot so small behind the ankles. Sideways falls would be a particular problem for PD subjects who stand with narrow stance width because of the smaller than normal activation of hip abductor, TFL, and cocontraction of trunk muscles that limit quick, lateral motion of the body CoM (Henry et al. 1998b). Forward falls in PD subjects are rarer during stance than during gait, because of the large ankle stiffness due to parkinsonian rigidity and large lever of the foot length for exerting stabilizing torques during stance.

Given the severe balance disorders of subjects with PD, it may be surprising that so many aspects of automatic postural response to external perturbations are similar to those of age-matched control subjects. For example, 5 of 13 PD subjects in this study fell in the clinical “pull test” component of the UPDRS, and all but 1 of the remaining 8 PD subjects had retropulsion in response to a backward tug at the shoulders. Based on the clinical tests of balance and gait in the UPDRS, the timely recruitment of appropriate muscle patterns in response to external perturbations would not have been pre-
dicted. However, the clinical pull test did not test our subjects’ ability to use “in-place” postural responses to maintain equilibrium since the tug at the shoulders is strong enough to generate rapid stepping for postural recovery, unlike the perturbations in our study, which were below threshold for generating a step in all but 10% of trials. Nevertheless, the ability to adapt TIB muscle response amplitude to narrow and wide stance significantly and the ability to inhibit ESP as an antagonist to avoid coactivation significantly correlated with several clinical measures of balance in the PD subjects. The reason that these two muscles were the most sensitive indicators of clinical balance disorders may relate to the importance of these two agonists in responding to backward body sway (Henry et al. 1998b). Both the UPDRS and the Hoehn and Yahr tests of balance include the backward pull test of posture (Postural Stability Item 30). The TIB and ESP are the two earliest, and most critical, agonists for correcting equilibrium using a combination of ankle and hip strategies, when the body is displaced backward (Henry et al. 1998b, 2001; Horak et al. 1998a).

CONCLUSIONS

This work provides novel insights into postural instability associated with PD that could not be addressed in previous studies of responses to unidirectional perturbations. First, we show that, contrary to expectations, subjects with moderate to severe PD in the off state have quite normal patterns of postural muscle synergies across many directions. This (negative) result suggests that dopaminergic basal ganglia systems are not critical for this directional-specificity of postural synergies. Second, we show data in support of the hypothesis that the basal ganglia are important for focusing muscle activation by filtering out unwanted muscle activation. Specifically, when muscles were activated as antagonists, they were activated too early, in too many directions, and with too large a magnitude, resulting in excessive coactivation, regardless whether they are across proximal or distal joints or across the two sides of the body. This coactivation is consistent with the hypothesis that the basal ganglia is important for focusing postural muscle activation patterns by inhibiting unnecessary muscle activation (Brooks 2001; Filion 2000). Third, we demonstrate a new task example (narrow versus wide stance width) in support of the hypothesis that the basal ganglia are important either for mapping proprioceptive information about body position or for modifying postural strategies based on this proprioceptive information indicating changes in body position.

Although many of our results are consistent with previous studies of postural responses in response to forward sway perturbations in PD subjects (Beckley et al. 1991; Horak et al. 1992, 1996), this is the first study to show that the same primary constraints (cocontraction due to early and large antagonist activity) also occur for lateral and diagonal and backward sway perturbations. This result is new and surprising given our other studies showing that PD subjects have much worse postural stability and kinematic patterns in response to backward and lateral perturbations (Horak et al., unpublished data). Thus the increased instability of PD subjects to backward, compared with other directions of perturbation, are not due to neurological deficits specific for the backward direction but are due to biomechanical constraints.

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