Postural Feedback Scaling Deficits in Parkinson’s Disease

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Kim S, Horak FB, Carlson-Kuhta P, Park S. Postural feedback scaling deficits in Parkinson’s disease. J Neurophysiol 102: 2910–2920, 2009. First published September 9, 2009; doi:10.1152/jn.00206.2009. Many differences in postural responses have been associated with age and Parkinson’s disease (PD), but until now there has been no quantitative model to explain these differences. We developed a feedback control model of body dynamics that could reproduce the postural responses of young subjects, elderly subjects, and subjects with PD, and we investigated whether the postural impairments of subjects with PD can be described as an abnormal scaling of postural feedback gain. Feedback gains quantify how the nervous system generates compensatory joint torques based on kinematic responses. Seven subjects in each group experienced forward postural perturbations to seven different backward support surface translations ranging from 3- to 15-cm amplitudes and with a constant duration of 275 ms. Ground reaction forces and joint kinematics were measured to obtain joint torques from inverse dynamics. A full-state feedback controller with a two-segment body dynamic model was used to simulate joint kinematics and kinetics in response to perturbations. Results showed that all three subject groups gradually scaled postural feedback gains as a function of perturbation amplitudes, and the scaling started even before the maximum allowable ankle torque was reached. This result implies that the nervous system takes body dynamics into account and adjusts postural feedback gains to accommodate biomechanical constraints. PD subjects showed significantly smaller than normal ankle feedback gain with low scaling and larger hip feedback gain, which led to an early violation of the flat-foot constraint and unusually small (bradykinetic) postural responses. Our postural feedback control model quantitatively described the postural abnormality of the patients with PD as abnormal feedback gains and reduced ability to modify postural feedback gain with changes in postural challenge.

INTRODUCTION

Inflexible, weak postural responses have been reported in patients with Parkinson’s disease (PD) (Frank et al. 2000; Morrison et al. 2008; Termoz et al. 2008). However, postural response measurements are often limited in providing insight into the neural mechanisms responsible for postural abnormalities. For example, in contrast to the common belief that larger postural sway is positively correlated with abnormalities. For example, in contrast to the common belief that larger postural sway is positively correlated with postural instability (Sheldon 1963), fall-prone patients with PD often show smaller center of pressure (CoP) displacements and smaller and slower displacements of the body center of mass (CoM) than those of the healthy elderly (Frank et al. 2000; Horak et al. 1992, 2005; Termoz et al. 2008). Thus univariate measures such as displacements of CoP and CoM alone may not serve as the most sensitive measures of postural performance nor explain systematic dysfunction of postural control. Empirical metrics have limitations that might be addressed with a feedback control model that explicitly attempts to capture the systematic behavior of the nervous system and not just the observed outcomes.

By characterizing kinematic, kinetic, and electromyographic (EMG) patterns of postural coordination across a decreasing base of support or increasing magnitudes of surface perturbations, we and others have shown that healthy subjects use a flexible transition from an “ankle strategy” to a “hip strategy” to maintain equilibrium (Horak and Nashner 1986; Horak et al. 1992; Laessoe and Voigt 2008; Runge et al. 1999; Termoz et al. 2008). In contrast, patients with PD demonstrate a type of postural inflexibility by activating the ankle and hip strategies simultaneously, regardless of their normal postural latencies (Bisdorff et al. 1999; Horak and Macpherson 1996; Horak et al. 1992; Rothwell et al. 1983). Inflexibility of Parkinsonian postural responses is also apparent in the lack of modification of postural muscle synergies normally associated with changes in support conditions (Chong et al. 2000; Horak et al. 1992). While there have been qualitative hypotheses that the postural inflexibility of patients with PD is related to dysfunction of “set switching” by the basal ganglia and neural systems networked to them (Brown et al. 2006; Santaniello et al. 2007; Visser and Bloem 2005), there have been few quantitative models of this behavioral inflexibility.

Postural stabilization of upright stance is usually modeled as a single-segment, linear feedback control system that predicts ankle joint torques based on changes in ankle joint kinematics (Johansson et al. 1988; Masani et al. 2006; Peterka 2000). In a feedback control model incorporating a multi-segmental biomechanical model, multivariate feedback gains quantify the proportional weight of sensory feedback among several joint torques to describe multi-joint kinematics (Barin 1989; Kuo 1995; Ozbay 1999; Park et al. 2004). For example, a high gain of ankle angle feedback suggests that the nervous system increases the relative weight of ankle joint feedback for generating compensatory ankle joint torques. Recently, Park and colleagues demonstrated that normal postural adjustments, associated with a continuum of kinematic strategies from the ankle to hip strategy, can be described in terms of a continuous feedback control system with feedback gain scaling as a function of perturbation magnitudes (Park 2002; Park et al. 2004). Specifically, the nervous system appears to gradually scale feedback gains to accommodate biomechanical constraints for upright stance. Therefore the failure of proper gain selection would induce inappropriate postural strategy changes and violate constraints of upright stance, such as...
lifting the heels off the ground, which would result in postural instability.

In this study, we propose a quantitative model to explain abnormal postural responses of patients with PD. We hypothesize that postural control impairment of subjects with PD can be quantified as an abnormal scaling of postural feedback gain with increased postural challenges. The results show that multivariate linear feedback model simulations can reproduce postural responses in young, elderly, and PD subject groups for a wide range of surface perturbations. Continuous feedback gain scaling was observed for all subject groups, implying that the nervous system automatically adjusts motor output to accommodate changes in biomechanical constraints. Abnormal postural responses of subjects with PD were consistent with smaller than normal ankle feedback gain, larger than normal hip feedback gains, and an inflexible selection of feedback gain as the perturbation conditions change.

METHODS

To quantify the differences in postural control among subject groups, we used a multisegmental biomechanical model incorporating linear feedback control (Fig. 1). Joint kinematics and ground reaction forces were measured in response to backward surface translations of a wide range of magnitudes. Hip and ankle joint torques were then calculated from inverse dynamics and optimization was used to identify the feedback gains for the model simulation that best reproduced our measured joint motions and torques.

Subjects

Seven healthy young (mean age: 24 ± 3 yr) and seven healthy elderly (63 ± 7 yr) subjects and seven age-matched patients with PD (64 ± 9 yr) participated in this study. The young subjects’ data were a subset of previously collected data (n = 13) from a postural feedback scaling study to backward translation (Park et al. 2004). The healthy subjects reported no history of a balance disorder. All PD subjects had medication (On levodopa state) prior to the test and were evaluated to have moderate (23.8 ± 10.2) Parkinsonism according to the Total Motor Score of the Unified Parkinson’s Disease Rating Scale (UPDRS, Part IV) (Table 1). All subjects signed the informed consent form approved by the IRB of Oregon Health and Science University prior to the test.

Protocols

Subjects experienced backward surface perturbations, resulting in forward body CoM displacements with respect to the base of support. Seven translation magnitudes ranged from 3 to 15 cm over 275 ms, such that both the amplitude and velocity of translations increased simultaneously. The seven displacement amplitudes were 3, 4.5, 6, 7.5, 9, 12, and 15 cm for trials of healthy subjects and were 3, 4.5, 6, 7.5, 9, 10.5, and 12 cm for trials of PD patients. The magnitudes of perturbations were designed to induce significant postural strategy changes from the ankle strategy to the hip strategy. Subjects were instructed to stand upright with their arms crossed over their chests and to recover to their initial upright posture in response to the perturbation without stepping or lifting their heels, if possible. The platform was controlled by an electrohydraulic system and programmed to generate five sets of seven randomly ordered ramp translations. Prior to the data collection, subjects rehearsed several trials including the largest translation. To prevent possible falling injury during trials, subjects wore a safety harness system.

Measurement

Ground reaction force and kinematic data were recorded for 10 s, including 2 s of quiet stance prior to the onset of each translation. Kinematic data were measured by eight reflective markers located at the neck (C7), shoulder (acromion), hip (greater trochanter), knee (lateral femoral condyle), ankle (lateral malleolus), toe, heel, and the platform surface, using the optical Motion Analysis system (Santa Rosa, CA) at a sampling rate of 60 Hz. Ground reaction forces and moments were recorded by a custom force plate at a sampling rate of 480 Hz.

Data analysis

The postural feedback control model is shown in Fig. 1. The model consists of a two-segment, inverted pendulum biomechanical model that represents upper and lower body dynamics and a linear feedback controller that describes the CNS’s control of compensatory joint torques (Fig. 1B). Inherently unstable upright posture is stabilized by joint torques, which are proportional to joint angle deviations. The characteristics of a feedback-controlled system are determined by the feedback gains, the coefficient matrix proportion between the joint torques and joint kinematics. For example, the joints with greater feedback gain produce greater joint torques, which results in faster recovery to upright posture but at the cost of greater control effort. We identified the feedback gains that best reproduced the empirical data using optimization.

To formulate the feedback control model, we defined the state vector, x, of joint kinematics referenced to upright posture as $x = [\theta_{\text{ank}}, \theta_{\text{hip}}, \omega_{\text{ank}}, \omega_{\text{hip}}]^T$ where $\theta_{\text{ank}}, \theta_{\text{hip}}$ are ankle and hip joint angle, and $\omega_{\text{ank}}, \omega_{\text{hip}}$ are ankle and hip angular velocity, respectively.

The state space form of the feedback-controlled postural system was obtained as $\dot{x} = Ax + Bu + w$, where $A$ and $B$ are the state matrices obtained in terms of inertial parameters of the body segments and $w$ describes a vector of external perturbation by support transla-
Feedback control input \( u \) (i.e., joint torque \( T \)), was obtained as the form of

\[
u = T = -Kx \]

where \( T_{\text{ank}} \) and \( T_{\text{hip}} \) are ankle joint and hip joint torque, respectively, and \( K \) is a \( 2 \times 4 \) feedback gain matrix of multiple gain components. Then, the closed-loop system has the form of \( \dot{x} = (A - BK)x + w \). We simulated the closed-loop system over time using perturbation data, \( w \), and empirical initial condition, \( x_0 \), defined as joint deviation from the subject’s preferred upright stance, and found feedback gain, \( K \), that makes the closed-loop model simulation the best match for empirical joint motion and torques.

The feedback control gain, \( K \), represents the nervous system’s control law, which produces compensatory joint torques to maintain stance posture. Specifically, larger ankle gains indicate that the nervous system weights ankle joint displacement more heavily than other joint displacements in generating compensatory joint torques for balance recovery, as suggested by the ankle strategy (Kuo and Zajac 1993; Runge et al. 1999). In contrast, a smaller ankle gain, in combination with larger hip gain, indicates that the major contribution to compensatory joint torques is from the hip joint angle feedback rather than the ankle joint, as suggested by the hip strategy (Kuo and Zajac 1993; Runge et al. 1999). Therefore a gradually scaled feedback gain as a function of perturbation magnitude describes the continuous postural strategy change from ankle to hip (Horak et al. 1992).

Multiple gain parameters represent the coupled contribution of other joint kinematics to generate a specific joint torque. For convenience, we defined “ankle gain” and “hip gain” as the gain components that correspond to an ankle joint angle feedback to an ankle joint torque \( (k_{11}) \) and hip joint angle feedback to the hip joint torque \( (k_{22}) \), respectively. Details of the feedback gain scaling and the optimization procedure used to obtain feedback gain that best matches the data were presented in the previously published paper (Park et al. 2004).

It is noteworthy that the feedback control gain obtained from a linear model optimization is different from a linear regression gain (Alexandrov et al. 2005; Barin 1989; Roux et al. 2002; Winter et al. 1998) that is also written in the form of Eq. 1. Because regression gains simply relate two output variables, a linear system simulation with regression gain generally does not guarantee stability of the system or the reproducibility of the joint kinematics and kinetics data (Fig. 2). On the other hand, the model simulation with feedback gain reproduces the data reasonably well and guarantees system stability (Park et al. 2004).

Differences in peak joint angles and peak joint torques corresponding to each perturbation magnitude were tested with the Mann-Whitney \( U \) test. Feedback gains and gain scaling (slope of gain across perturbation magnitudes) were also tested with Mann-Whitney \( U \) with a level of significance of \( P < 0.05 \).

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

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age</th>
<th>Gender</th>
<th>Height, cm</th>
<th>Weight, kg</th>
<th>Duration of PD, yr</th>
<th>Total Motor Score</th>
<th>Total Dyskinesia Score</th>
<th>Side Most Affected</th>
<th>H&amp;Y Score</th>
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<tbody>
<tr>
<td>Sub01</td>
<td>53</td>
<td>M</td>
<td>178</td>
<td>71</td>
<td>9</td>
<td>13</td>
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<td>R</td>
<td>2</td>
<td>93</td>
</tr>
<tr>
<td>Sub02</td>
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<td>M</td>
<td>188</td>
<td>90</td>
<td>3</td>
<td>33</td>
<td>0</td>
<td>R</td>
<td>2</td>
<td>91</td>
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<tr>
<td>Sub03</td>
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<td>F</td>
<td>165</td>
<td>81</td>
<td>5</td>
<td>24.5</td>
<td>0</td>
<td>R</td>
<td>2</td>
<td>96</td>
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<tr>
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<td>M</td>
<td>180</td>
<td>90</td>
<td>11</td>
<td>39</td>
<td>0</td>
<td>R</td>
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<td>95</td>
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<tr>
<td>Sub05</td>
<td>67</td>
<td>M</td>
<td>185</td>
<td>89</td>
<td>1</td>
<td>19.5</td>
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<td>R</td>
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<td>96</td>
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<tr>
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<td>1</td>
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<td>0</td>
<td>R</td>
<td>2</td>
<td>83</td>
</tr>
</tbody>
</table>

*Activities-Specific Balance Confidence scale; PD, Parkinson’s disease.

### RESULTS

While three of seven young subjects could maintain balance even during the largest (15 cm) perturbation that was designed to induce the violation of flat-foot constraints, none of the elderly subjects could recover upright posture due to significant heel-offs at the maximum (15 cm) perturbation. The largest perturbation that most of the elderly and PD subjects were able to recover to the upright posture was 12 and 9 cm, respectively. Two of seven PD subjects showed repeated heel-offs, even for the smallest perturbation (~4.5 cm), and could not complete the study for the perturbations equal or larger than 6 cm, so their data were not considered throughout the analysis.

Joint kinematics and joint torques

The magnitude of joint kinematics and joint torque data were gradually scaled with perturbation magnitudes for all subject groups with different degrees of scaling (Fig. 3). Nonuniform scaling of joint torque trajectories, to avoid violating maximum ankle torque without lifting heels, was observed in the young and the elderly. In contrast, subjects with PD showed less flexibly scaled torque trajectories. Joint angle trajectories were similarly scaled in the young and the elderly groups, but hip joint angles and corresponding joint torques were significantly smaller in subjects with PD (Figs. 3 and 4).

Figure 4 shows peak joint angles and peak joint torques (normalized by subject’s weight multiplied by height) for each subject group. Compared with the young group, the elderly group had significantly smaller ankle joint motions accompanied by smaller ankle joint torques \( (P < 0.05) \), and slightly larger hip joint motion and greater hip joint torques that missed statistical significance \( (P = 0.29) \). Compared with the elderly, the age-matched PD subjects showed significantly reduced hip joint motion in response to the intermediate magnitude of perturbations \( (P < 0.05) \) and reduced hip joint torques but only for 9 cm perturbations \( (P < 0.05) \). The maximum allowable ankle joint torque is defined as the peak ankle joint torque possible without violating the flat-foot constraint. The normalized maximum allowable ankle torques of the elderly \( (0.057 \pm 0.006) \), was significantly smaller than that of the young group \( (0.073 \pm 0.01) \; (P < 0.05) \). The initial position of the CoP from the center of rotation during upright posture of the elderly \( (6.64 \pm 1.50) \) showed significant forward leaning compared with the young \( (1.27 \pm 1.46) \; (P < 0.05) \).
Performance of linear feedback control model simulation

A simple linear feedback control model with time-invariant, optimized feedback gain could reproduce the data of all of the young ($R^2 = 0.84 \pm 0.02$), the elderly ($R^2 = 0.84 \pm 0.04$), and PD subjects ($R^2 = 0.80 \pm 0.04$) reasonably well (Fig. 5) but with significantly better performance ($P < 0.05$) for the young and the elderly. This model could match the data slightly better for the trials subjected to the perturbations of intermediate magnitude than those of the smallest and/or largest perturbations because the former generates very small magnitude of responses, whereas the latter often induces the heel lift, making the two-segment inverted pendulum model less accurate in representing flat-foot, upright posture.

Multiple feedback gains and gain scaling

A feedback control gain ($K$), the proportionality coefficient between the joint torques and kinematics written as Eq. 1, is a $2 \times 4$ gain matrix of multiple gain components (Fig. 6). Gradual scaling of feedback gains as a function of perturbation magnitudes were observed in all three subject groups. Although multiple gains represent cross-joint contributions to generate individual joint torques, the corresponding joint kinematic feedback generally has the greatest contribution to that joint torque (Table 2). The ankle gain ($k_{11}$) and the hip gain ($k_{22}$) contribute more than the other six gain parameters, to the postural strategy change and to postural stability (Park et al. 2004).
The PD group showed significantly different ankle and hip gain scaling compared with the age-matched elderly. The magnitude of ankle gains in the PD group were significantly smaller ($P < 0.05$) and scaled with a smaller slope ($P < 0.05$), whereas hip gain values were significantly larger ($P < 0.05$) compared with those of the elderly. Angular velocity feedback hip gains ($k_{14}, k_{24}$) of the PD group were also statistically larger ($P < 0.05$) than the elderly. There was no significant difference in hip gain scaling, either in its slope ($P = 0.29$) or its intercept ($P = 0.34$), between the young and the elderly groups. The average ankle gain values of the elderly were consistently smaller than those of the young throughout the perturbation magnitudes, but did not attain statistical significance ($P = 0.41$).

**DISCUSSION**

Although many studies have characterized the postural responses of patients with PD, most of the measures were limited to empirical observations without quantitative models of postural abnormalities (Beckley et al. 1993; Horak et al. 1996). We developed a feedback-controlled model of two-segment body dynamics and investigated whether the balance impairments of subjects with PD can be described as an abnormal scaling of postural feedback gain. The results showed that multivariate linear feedback model simulations reproduced postural responses for a wide range of surface perturbations. Continuous feedback gain scaling was observed, implying that the nervous system gradually adjusts postural response strategies from an ankle to a hip strategy to accommodate biomechanical constraints. Subjects with PD showed abnormal hip and ankle gains and inflexible adjustment of feedback gains as perturbations became more challenging.

**Postural feedback response scales to accommodate biomechanical constraints**

Kinematic and kinetic responses of all subject groups showed gradual scaling as a function of perturbation magnitude. This scaling of feedback gain allowed subjects to maintain balance without violating biomechanical constraints on the maximum allowable ankle torque for upright stance with feet flat on the floor.
To recover to an upright posture from larger perturbation magnitudes, larger magnitudes of compensatory joint torques are required, and the maximum allowable ankle torque constraint becomes more challenging to obey. Therefore instead of the peak ankle joint torques uniformly increasing with increased magnitudes of perturbation, they were gradually suppressed with increased perturbation magnitude (Fig. 3, D–F). The gradual scaling started even before the constraint on heel lift was reached, implying that the nervous system represents the biomechanical constraints in a continuous manner rather than at a particular biomechanical threshold (Park et al. 2004).

Postural adjustments in responses to increased perturbation magnitudes were quantified by the scaling of the feedback control gain (Fig. 6). All subject groups reduced ankle gain \( k_{11} \) with increasing postural challenge in order to not violate flat-foot constraints, at the expense of reduced system stability. To compensate for the decreased stability and reduced feedback contribution from ankle joint angle feedback \( k_{11} \) to ankle joint torque, hip joint angle feedback gains \( k_{12}, k_{22} \) increased, which describes the transition of feedback control from an ankle sway to hip sway (Alexandrov et al. 2005; Park et al. 2004). The continuous scaling of feedback gains with perturbation magnitude illustrates the gradual transition of postural strategy from ankle to hip, which has been observed in many of previous studies (Horak and Nashner 1986; Kuo and Zajac 1993; Park et al. 2004; Runge et al. 1999; Van Ooteghem et al. 2008).

Abnormal gains with inappropriate scaling by PD subjects

Postural instability associated with PD is not due to excessive postural sway in response to perturbations. Compared with the age-matched elderly, PD subjects showed significantly smaller postural displacements and smaller hip joint torques (Figs. 3 and 4), which is consistent with our previous studies (Dimitrova et al. 2004; Horak et al. 1992, 2005). Previous studies showed normal EMG latencies and scaling of postural responses to translation velocity but impaired scaling to larger perturbation amplitudes in subjects with PD (Horak et al. 1992; Jacobs and Horak 2006; Marsden 1984; Schieppati and Nardone 1991). This study suggests that abnormal postural feedback gain scaling can explain the postural incoordination of subjects with PD.

The PD patients showed significantly different gain and gain scaling behavior from the healthy elderly. The PD subjects showed much smaller ankle gain \( k_{11} \) with low ankle gain scaling and a larger hip gain \( k_{22} \) with slightly greater hip gain scaling. The small ankle gain \( k_{11} \) of the PD group significantly reduced compensatory ankle joint torque and the rate of change of torque as observed in previous studies (Dimitrova et al. 2004; Horak et al. 1996). However, in response to larger perturbations, low ankle gains but inappropriately low scaling of ankle gain \( k_{11} \); Fig. 6) induce greater ankle joint torques than biomechanically allowable, which in turn leads to the early onset of heel lifts or stepping (e.g., in response to ∼9-cm translations for PD subjects and ∼12 cm for control subjects). These results are consistent with the use of premature compensatory stepping responses to shoulder pulls in patients with PD (Bloem et al. 2001; Jacobs et al. 2006).

Subjects with PD also have significantly larger hip feedback gains \( k_{12}, k_{22} \) than age-matched control subjects, leading to stiffer hip joints so that overall postural sway resembles an inverted pendulum with significantly smaller hip joint motion, as observed previously (Frank et al. 2000; Horak et al. 1992; Termoz et al. 2008). Exceptionally high hip stiffness is con-
consistent with our direct measurements of hip postural tone during free stance, which were even larger than increased trunk or neck axial tone in subjects with PD (Wright et al. 2007). Therefore unusually small postural responses of subjects with PD can be ascribed to increased hip joint feedback gain, while the early violation of the flat-foot constraint (lifting of the heels and stepping) is caused by the inadequate gain scaling.

Using a linear feedback control model, we could quantitatively describe CNS postural control in a multivariate manner that includes postural coordination of a multisegamental system. Although we had a small sample size of subjects with PD, the same model described postural control in young, elderly, and PD subject groups. Our subjects with PD showed the same postural abnormalities as previously reported, such as postural inflexibility and bradykinetic postural responses (Bloem et al. 1995; Horak et al. 1992; Mancini et al. 2008). In this study, however, changes in the perturbed postural responses of subjects with PD were also differentiated from those of the elderly by impaired feedback gain scaling. For example, the small reactive torques of the PD subjects in response to the forward body sway were also observed in previous studies (Horak et al. 1996). The responses are simulated here by a different control plan of the CNS to secure a greater angle torque margin away from the allowable torque stability limits by assigning less weight on sensory feedback from the ankle joints, i.e., smaller ankle gain \( k_{11} \). Reduced ankle gain may also be due to decreased use of proprioceptive feedback in PD, as observed by impaired kinesthesia (Jacobs and Horak 2006). In addition, inflexible gain scaling of the PD group, as opposed to the continuous gain scaling as a function of postural challenges observed in the controls, implies that a nervous system with neurodegenerative basal ganglia disease has difficulty in flexibly adjusting reliance on sensory feedback or flexibly modifying compensatory motor commands (Chong et al. 2000; Horak et al. 1992). Moreover, abnormalities in feedback gains appeared to be consistent for all PD subjects with relatively small inter-subject variation compared with intergroup differences (Fig. 6). This finding implies that a greater sample size would increase the statistical significance, but would not change the major conclusion of the current study, namely that PD subjects exhibit an inappropriate gain scaling behavior.

**Multivariate gain scaling does not change with aging**

Differences in kinematic and kinetic peak values between the young and the elderly, despite the similar gain scaling, illustrate the limitations of using univariate measures to characterize postural control. Compared with the young subjects, elderly subjects showed significantly smaller ankle joint motion and ankle joint torques but slightly larger hip joint motion (Figs. 3 and 4). Although feedback gain distributions of the two groups were not significantly different \( (P > 0.05; \text{ Fig. 6}) \), variations exist in gain parameters such that the elderly group had smaller ankle feedback gains \( (k_{11}, k_{21}) \) and larger hip gain \( (k_{12}) \) and ankle velocity gain \( (k_{13}) \) to ankle torque. In addition, the elderly had significantly greater \( (P < 0.05) \) body mass \( (90.02 \pm 20.43 \text{ kg}) \) with larger upper body mass \( (63.42 \pm 15.84 \text{ kg}) \), compared with the younger subjects \( (59.38 \pm 10.29 \text{ kg of total mass and 39.84 \pm 7.7 kg of upper body mass, respectively}) \). To examine how the different body parameters

**FIG. 5.** Representative time trajectories of ankle (black) and hip (gray) joint angle \((A–C)\) and corresponding normalized joint torque \((D–E)\) of the empirical data (dashed line) and model simulation (solid line) of the young \((A \text{ and } D)\), the elderly \((B \text{ and } E)\), and the PD subjects \((C \text{ and } F)\). \( R^2 \) values comparing data with simulations for each group were 0.86, 0.88, and 0.79, respectively.
and gains affect the observed kinematic differences between the two groups, a model simulation was performed (Fig. 7). Smaller ankle feedback gains ($k_{11}$, $k_{21}$) along with the larger hip gain ($k_{12}$) of the elderly suppressed the ankle motion but enhanced the hip motion significantly. Increased upper body mass amplified the magnitude of inertia forces, which in turn, increased the magnitude of hip joint kinematics. These simulation results suggest that the differences in the measurements of joint motion and torques between the young and elderly groups may be attributed to altered system parameters such as feedback gains and body mass distributions and do not necessarily indicate changes in postural strategy.

Another difference between the young and the elderly groups is the significantly smaller maximum allowable ankle joint torque in the elderly (Fig. 3, D and E), which may be due to the tendency of initial forward leaning at their preferred upright posture as observed in previous studies (Alexander 1994; Horak and Moore 1993; Maki and McIlroy 1996; Woodhull-McNeal 1992). Because the two-segment inverted pendulum biomechanical model was normalized with respect to each subject’s preferred upright posture, an initial forward leaning posture resulted in a reduced functional base of support (King et al. 1994), i.e., the magnitude of maximum allowable ankle torque. The difference in normalized ground reaction torque offset by the initial forward leaning posture of the elderly was 0.022, which approximately matches the difference found in the normalized maximum ankle torque between the young and the elderly (Fig. 3, D and E).

Physiological interpretation of feedback gains

The current postural feedback control model quantifies the postural control of each subject group using gain scaling.

TABLE 2. Averaged correlation between joint torque and individual feedback component of joint kinematics

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<thead>
<tr>
<th></th>
<th>$T_{\text{ank}}$ &amp; $k_{11}\theta_{\text{ank}}$</th>
<th>$T_{\text{ank}}$ &amp; $k_{12}\theta_{\text{ank}}$</th>
<th>$T_{\text{ank}}$ &amp; $k_{13}\theta_{\text{ank}}$</th>
<th>$T_{\text{ank}}$ &amp; $k_{14}\theta_{\text{hip}}$</th>
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<tr>
<td>Young</td>
<td>0.88 ± 0.10</td>
<td>0.25 ± 0.21</td>
<td>-0.11 ± 0.08</td>
<td>0.48 ± 0.22</td>
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<tr>
<td>Elderly</td>
<td>0.78 ± 0.16</td>
<td>0.15 ± 0.15</td>
<td>-0.08 ± 0.11</td>
<td>0.63 ± 0.17</td>
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<tr>
<td>PD</td>
<td>0.91 ± 0.08</td>
<td>0.17 ± 0.23</td>
<td>-0.13 ± 0.16</td>
<td>0.66 ± 0.09</td>
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<tr>
<th></th>
<th>$T_{\text{hip}}$ &amp; $k_{11}\theta_{\text{hip}}$</th>
<th>$T_{\text{hip}}$ &amp; $k_{21}\theta_{\text{hip}}$</th>
<th>$T_{\text{hip}}$ &amp; $k_{13}\theta_{\text{hip}}$</th>
<th>$T_{\text{hip}}$ &amp; $k_{24}\theta_{\text{hip}}$</th>
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<tr>
<td>Young</td>
<td>0.33 ± 0.30</td>
<td>0.77 ± 0.24</td>
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<td>0.46 ± 0.17</td>
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</tbody>
</table>
Appropriate selection of feedback gain stabilized the inherently unstable dynamics of a two-segment inverted pendulum biomechanical model, and the model simulation reproduced the empirical data of joint motion and torques. Feedback gains quantified how the nervous system generated compensatory joint torques in terms of joint kinematics so that the gradual scaling of feedback gains implies continuous change of the postural feedback control strategy. Gain parameters could be interpreted as actively controlled joint stiffness and damping. However, unlike mechanical spring/damper components that apply reactive torques to each limb segment in a symmetrical manner, the gain matrix of the full-state feedback generally does not hold a symmetry constraint, implying that the contribution of joint kinematic feedback to joint torque is not reciprocal.

With the limitations from the noninvasive, behavioral study in verifying the relationship of the actual sensory afferents and their corresponding motor command, the current control model of neural postural feedback mechanism postulates that the multisensory information could represent joint kinematics information and the motor outputs would aim to coordinate multiple muscles to generate compensatory joint torques, as quantitatively demonstrated by the previous studies (Jo and Massaquoi 2004; Welch and Ting 2008). The physiological representation of feedback gains, therefore could be the neural mechanism that associates sensory estimates of body states with motor programs, such as long-loop gain of postural control by the CNS (Jo and Massaquoi 2004; Park et al. 2004; Welch and Ting 2008). Although the current study has limitations in specifying which parts of the CNS are involved in the feedback gain scaling, failure of appropriate postural scaling has been previously observed in patients with PD (Bakker et al. 2006; Beckley et al. 1993; Horak and Diener 1994; Horak et al. 1996), suggesting that the basal ganglia and the associated postural centers of the midbrain and brain stem are involved in postural feedback gain scaling. Previous investigations of postural abnormalities in patients with PD (Horak et al. 1992, 1996; Jacobs and Horak 2006; Marsden 1984) have reported normal postural response latencies but abnormal inflexible reactive joint torques, consistent with the low ankle gain, high hip gain, and inappropriate feedback gain scaling in our study. A recent study also shows higher background postural tone in axial muscles in subjects with PD than controls (Wright et al. 2007). These results are consistent with a role of the basal ganglia in scaling background postural tone and scaling of automatic postural responses.

**Model limitations**

Inappropriate gain scaling explains the postural inflexibility of subjects with PD, but the current model is limited in representing the mechanism for the abnormal gain scaling. From the observation that postural feedback gain scales to accommodate anticipated biomechanical constraints as the size of the perturbation changes (Park et al. 2004), we suggest a global postural objective that quickly returns the body center of mass to its initial equilibrium position without heel lift (Horak and Kuo 2000; Kuo and Zajac 1993). A high priority for this objective would account for the gradual reduction in ankle torque and the increase in hip torque and gradually increasing trunk extension as the magnitude of perturbation increases (Horak and Kuo 2000). It is unclear whether the scaled feedback responses are generated from sensory feedback or are preset and triggered by the perturbation or its anticipation using a feedforward control (Horak and Macpherson 1996; Pavol and Pai 2002; Ruget et al. 2008; Santos and Aruin 2008).

Another limitation of the current study is the task-specific design of the experiment. Subjects were explicitly instructed to recover their upright posture without violating the flat-foot constraint so the same two-segment model of body dynamics could be used across a range of perturbations. However, subjects would actually rather step than stay flat-footed, when they encounter a large unexpected perturbation (Burleigh and Horak 1996; McIlroy and Maki 1993), and the violation of the flat-foot biomechanical constraint, followed by the initiation of a stepping response, was not modeled. In fact, the task goal of upright stance recovery could be modeled as a penalty matrix across several objectives such as stabilizing the body CoM and...
observing explicit and implicit biomechanical constraints, resulting in kinematic and kinetic postural strategy changes (Kuo 1995, 2005; Qu et al. 2007).

A third limitation of this study is that our model does not include nonlinear, temporal aspects of postural physiology, such as the dynamics of muscle mechanics (Lan 2002; Micheau et al. 2003), short-latency responses from reflexes (Ravaioi et al. 2005; Schweigert and Mengner 2008; Van Der Kooij et al. 1999), or long-loop feedback delays (Alexandrov et al. 2005; Peterka 2002; Welch and Ting 2008). If a model includes more components such as time-varying gains and/or time delays, the model simulation would likely fit the data even better at the expense of a large increase in the number of model parameters. Because our simple model fits the data rather well, however, it provides insight into postural neural control mechanisms. The only model parameter identified in our current feedback control model is the time-invariant, feedback control gain (K) that governs body dynamics throughout the response. The implementation of additional model parameters would not change the general conclusion of gain scaling. For example, with the implementation of a time delay that could be mathematically approximated by phase delay (Ozbay 1999), the closed-loop poles of the feedback control system would be identified in a more conservative manner to secure the stability margin, but the overall gain scaling characteristics would not change.

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