Postural feedback scaling describes the postural abnormality of Parkinsonian patients

1Seyoung Kim, 2Fay B. Horak, 2Patricia Carlson-Kuhta and 1Sukyung Park
1Department of Mechanical Engineering, KAIST, Daejeon, Korea
2Neurological Sciences Institute, Oregon Health & Science University, Portland, OR, USA
e-mail: sukyungp@kaist.ac.kr, web: http://biomt.kaist.ac.kr

INTRODUCTION

We investigated whether the postural responses of young subjects, elderly subjects, and subjects with Parkinson’s disease (PD) can be described as a continuous feedback control system, and whether the balance impairment of the subjects with PD can be described as abnormal scaling of postural feedback gain. Although various forms of univariate analysis methods have been used to assess postural control of the elderly and patients with PD, results are not consistent. We hypothesize that the balance impairment of subjects with PD can be quantified as abnormal scaling of postural feedback gain with increases in perturbation magnitude. Our results show that multivariate, linear feedback model simulations of postural responses to a range of surface perturbations are consistent with continuous feedback control and that postural kinematic strategy of subjects with PD could be explained by abnormal hip and ankle gains and inflexible selection of feedback gain.

METHODS

Seven healthy young (mean age 24±3 yrs), 7 healthy elderly (63±7 yrs) subjects, and 7 age-matched patients with PD (64±9 yrs) participated. The healthy subjects reported no history of balance disorder. All PD subjects have medication (on levadopa) prior to the test and were evaluated to be moderate (23.8±10.2) Parkinsonism according to the Total Motor Score of the Unified Parkinson Disease Rating Scale (UPDRS).

Seven subjects in each group experienced backward perturbations with 7 different backward translation magnitudes ranging from 3–15 cm with a constant duration of 275msec. The magnitude of perturbation was designed to induce significant postural strategy change from ankle to hip strategy [1,2]. 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.

Postural responses were quantified with ground reaction forces and joint kinematics. A full-state, 2-segment feedback model was used to quantify how the nervous system generates compensatory joint torques (Figure 1). Feedback control gain is written as a form of coefficient that relates joint kinematics and joint torques as follows,

$$
\begin{pmatrix}
\theta_{\text{ank}} \\
\theta_{\text{hip}}
\end{pmatrix} =
\begin{bmatrix}
T_{\text{ank}} \\
T_{\text{hip}}
\end{bmatrix} =
\begin{bmatrix}
k_{11} & k_{12} & k_{13} & k_{14} \\
k_{21} & k_{22} & k_{23} & k_{24}
\end{bmatrix}
\begin{bmatrix}
\theta_{\text{ank}} \\
\theta_{\text{hip}}
\end{bmatrix}
$$

Figure 1: Schematics of postural feedback control model by the central nervous system (CNS). Sensory information of body postural coordination are measured by vision, vestibular organ and muscle spindles, and then sent to the CNS to be processed. Based on the estimate of body kinematics, appropriate control plans are selected and then motor commands are produced as joint torques.
where $T_{\text{ank}}$, $T_{\text{hip}}$ are ankle joint and hip joint torque, $K$ is a 2 by 4 feedback gain matrix of multiple gain components, $\theta_{\text{ank}}$, $\theta_{\text{hip}}$ are ankle and hip joint angle, and $\dot{\theta}_{\text{ank}}$, $\dot{\theta}_{\text{hip}}$ are ankle and hip angular velocity, respectively. Multiple gains represent that 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. Feedback control gain was obtained from an optimization that minimizes the fitting error between the data and the model simulation.

RESULTS AND DISCUSSION

All three subject groups showed gradual scaling of feedback gains as a function of perturbation magnitudes (Figure 2), and the scaling started even before the maximum allowable ankle torque was reached. This implies that the postural control can be interpreted as a feedback process that takes into account body dynamics and biomechanical constraints [2].

While the young and elderly groups showed similar gain parameter distributions across translation magnitudes, PD subjects significantly larger hip joint angle feedback gains ($k_{12}$, $k_{22}$), than young or age-matched controls, leading to stiffer hip joints so that overall postural sway resembles an inverted pendulum and displays significantly smaller hip joint motion. Therefore, unusually small postural responses of the PD subjects can be ascribed to larger than normal hip joint angle feedback gain, while the early violation of the flat-foot constraint could be accounted for by inadequate ankle gain scaling.

The current, postural feedback gain scaling model provides a quantitative understanding of neural control of posture. Unlike conventional gain parameters which usually describe how the postural response magnitude changes across perturbation magnitudes [1], the feedback gain of the current model describes the long-loop gain of the automatic postural control system by the CNS. While the slope of the change of postural responses describes the ability of the CNS to generate the initial motor command trajectory, the feedback gain quantifies how the nervous system makes use of sensory feedback to generate compensatory motor commands [1,2]. In conclusion, our feedback control gain model quantitatively described the postural abnormality of the patients with PD as reduced ability to modify postural feedback gain with changes in perturbation magnitude.

REFERENCES


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