Analysis of the Relationship Between Muscle Tones and Abnormal Postures in a Computational Model

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Abstract— **Patients with Parkinson's disease (PD), a neurodegenerative disorder, exhibit a characteristic posture known as a forward flexed posture. Increased muscle tone is suggested as a possible cause of this abnormal posture. For further analysis, it is necessary to measure muscle tone, but the experimental measurement of muscle tone during standing is challenging. The aim of this study was to examine the hypothesis that "In patients with PD, abnormal postures are those with a small sway at increased muscle tones" using a computational model. The muscle tones of various magnitudes were estimated using the computational model and standing data of patients with PD. The postures with small sway at the estimated muscle tones were then calculated through an optimization method. The postures and sway calculated using the computational model were compared to those of patients with PD. The results showed that the differences in posture and sway between the simulation and experimental results were small at higher muscle tones compared to those considered plausible in healthy subjects by the simulations. This simulation result indicates that the reproduced sway at high muscle tones is similar to that of actual patients with PD and that the reproduced postures with small sway locally at high muscle tones in the simulations are similar to those of patients with PD. The result is consistent with the hypothesis, reinforcing the hypothesis.**

Clinical relevance— **This study implies that improving the increased muscle tone in patients with PD may lead to an improved abnormal posture.**

I. INTRODUCTION

Parkinson's disease (PD) is a common disorder among individuals above 60 years old [1], [2], with an anticipated increase in the number of affected patients [3]. The symptoms of PD include a range of motor impairments and increased muscle tone [4], [5]. Patients with PD also exhibit abnormal postures during standing that are characterized by antecollis and camptocormia, which may lead to dysphagia and pain [6]. The causes of this abnormal posture remain unclear; however, various hypotheses have been proposed, including those related to increased muscle tones [5], [6]. Previous studies showed that lidocaine injections into the external oblique muscles improve these abnormal postures and suggested dystonia of the trunk flexors as a possible cause [7]. For further analysis, it is necessary to measure muscle tone, but the experimental measurement of muscle tone during standing is challenging.

Previous studies used computational models to investigate issues related to postural control in patients with PD. These studies include the changes in the intermittency of control [8] and the effects of postural instability and balance training [9]. However, these studies primarily employed a one degree-of-freedom (DOF) inverted pendulum model as a human body model. Few studies employed computational models to examine the abnormal postures of patients with PD.

The purpose of this study is to examine a hypothesis regarding the relationship between muscle tone and abnormal posture using a computational model. We formulate the hypothesis based on the following assumptions about human standing:

1) Reducing sway represents one of the objectives during standing.
2) The center of mass (COM) velocity serves as an index of sway during standing.

Assumption 1 is based on the fact that the center of the pressure amplitude is significantly smaller than the base of support during standing [10]. Assumption 2 is based on the suggestion of a previous study that humans most accurately obtain the velocity information among the body position, velocity, and acceleration information from sensory input [11].

Based on these assumptions, it is thought that patients with PD aim to reduce their sway at increased muscle tones. Thus, their postures may be those in "In patients with PD, abnormal postures are those with a small sway at increased muscle tones".

We took the approach below to examine our hypothesis. We estimated the muscle tones of patients with PD using their postures and a computational model that could represent the muscle tones [12]. We calculated their muscle tones with four different magnitudes, including that plausible to that of healthy subjects in the previous study [13]. We also calculated the posture with a small sway in the estimated muscle tones using an optimization method based on the provided assumptions. Moreover, we compared the postures and sway obtained from the computational model with the

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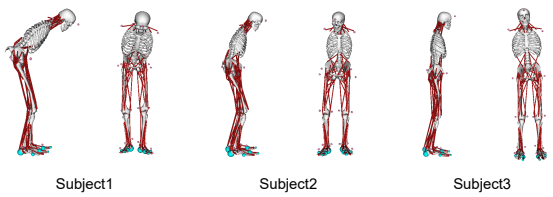

Fig. 1. Scaling results using the data of patients with PD for each subject. The postures of each subject are reproduced in the musculoskeletal model.

actual postures and sway of patients with PD. Our analysis aims to verify whether or not the actual postures of patients with PD, which are referred to as abnormal postures, are those with a small sway at the estimated muscle tones.

II. METHODS

This section describes the computational model used in this work. It also describes the method for muscle tone estimation and calculating a standing posture with a small sway in the estimated muscle tones using the computational model.

A. Problem setting

We focused on the abnormal postures of patients with PD. Therefore, a 5 s static standing task was performed using a computational model. All tasks were performed on the musculoskeletal simulators OpenSim [14], [15] and SCONE (Hyfydy) [16], [17].

B. Musculoskeletal model

We used a musculoskeletal model that can represent abnormal posture. The musculoskeletal model has 21 DOF and 94 muscles based on a previous study [12] and was scaled to each subject using the method described below.

C. Neural controller

A neural controller proposed in the previous study [12] was used to control the musculoskeletal model. This controller consists of feedforward (FF), proprioceptive and vestibular feedback (FB) control, and time delays.

The FF control outputs a constant value independent of the time delays and represents the muscle tones [12]. It outputs to each muscle; hence, the muscle tones can be expressed as a vector with many dimensions as the number of muscles (i.e., 94-dimensional vector). $\|\boldsymbol{u}_{\mathbf{ff}}\|^2$ was used as an index based on the previous study [13] to express the whole-body muscle tone and compare muscle tone magnitude. A large index indicates a high whole-body muscle tone.

The FB control uses proprioceptive and vestibular sense as FB information, and its outputs are the FB information multiplied by the FB gains [12].

The time delays are caused by FB, neurotransmission, and muscle activity. The maximum time delays are 120 ms [12].

D. Posture calculation

This section describes a method for calculating postures with small sway. Especially, it describes a method for scaling the musculoskeletal model using experimental data, muscle tone estimation, and parameter adjustment to the estimated muscle tones.

1) Scaling using the posture data of patients with PD: The musculoskeletal model was scaled using the standing data from actual patients with PD, and abnormal postures were reproduced on the musculoskeletal models. We used 23 marker positions obtained from a motion capture system (6-camera, Oqus 3+/Oqus 5+, Qualisys, Gothenburg, Sweden, 150 Hz) during approximately 4 s of standing by three patients with PD (age: 72.3±6.51 years). These measurements were approved by the Ethics Committee at the National Center of Neurology and Psychiatry.

Inverse kinematics (IK) was performed in OpenSim to reproduce the sway of patients with PD during standing in a musculoskeletal model using the marker data [14], [15].

Hereinafter, the results of the IK reproduction of the marker data are referred to as the experimental results, whereas those reproduced by the neural controller are referred to as the simulation results. We also distinguished the experimental and simulation results using each subject datum as subs 1–3.

2) Muscle tone estimation: The muscle tones of each subject were estimated using the scaled musculoskeletal model by employing the method presented in Section II-D.1. According to the previous study [13], we attempted to make the musculoskeletal model stand without time delays and with only FB control, and calculated muscle tones from the standing results as follows 1. The target posture was that of each subject.

$$u_{\mathrm{ff,i}} = \frac{\int_{t_1}^{t_2} a_i(t)dt}{t_2 - t_1} \tag{1}$$

$u_{\mathrm{ff,i}}$ is the muscle tone of the *i*th muscle; $a_i(t)$ is the muscle activity of the *i*th muscle in *t* seconds; and $t_1,\ t_2$ are 3 s and 5 s based on the previous study [13], respectively.

Various muscle tones can be calculated depending on the FB gains. Muscle tones with arbitrary $\|\boldsymbol{u}_{\mathbf{ff}}\|^2$ were calculated with FB gains adjusted through optimization methods using an evaluation function that evaluates muscle tone magnitude. The optimization method used was the covariance matrix adaptation evolution strategy (CMA-ES) [18].

The magnitude of the actual whole-body muscle tone in patients with PD was difficult to measure. The previous study showed that the simulation result for the muscle tone with an index of $\|\boldsymbol{u}_{\mathbf{ff}}\|^2 = 2$ is plausible to healthy subjects [13]. Therefore, we calculated four types of muscle tones, including that plausible to that of healthy subjects $\|\boldsymbol{u}_{\mathbf{ff}}\|^2 = 2$ ($\|\boldsymbol{u}_{\mathbf{ff}}\|^2 = 2,\ 4,\ 6,\ 8$).

3) Parameter adjustment: The controller parameters were adjusted using an optimization method (i.e., CMA-ES [18]) to calculate the posture that allows standing with a small sway under the condition with time delays in the muscle

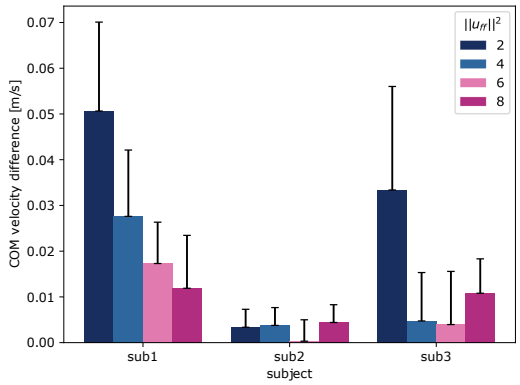

Fig. 2. Differences between the simulation and experimental results for the average COM velocity for each condition. $\|\boldsymbol{u}_{\mathbf{ff}}\|^2$ is an index to express the whole-body muscle tone. The error bars are positive only.

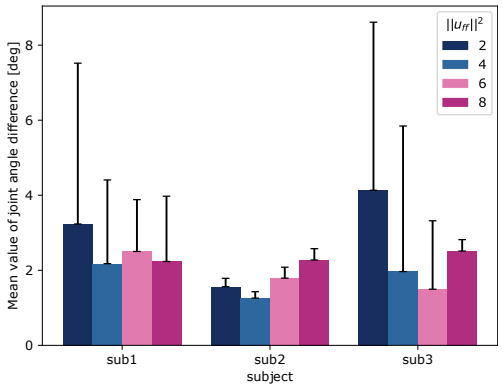

Fig. 3. Mean values of the joint angle differences between the simulation and experimental results for each condition. $\|\boldsymbol{u}_{\mathbf{ff}}\|^2$ is an index to express the whole-body muscle tone. The error bars are positive only.

tones estimated in Section II-D.2. The adjusted parameters were the FB gains and the initial posture (i.e., target posture). Thereby, postures with a small sway in the estimated muscle tones can be calculated. This adjustment can also reproduce standing under the condition with time delays at those calculated postures. A simultaneous parameter adjustment was difficult because of the large number of parameters. These parameters were adjusted by repeating the process in which each parameter was adjusted in turn. The evaluation function evaluated the COM speed. We searched for parameters that reduce the evaluation function value.

$$J = \frac{1}{T_{\mathrm{fall}}}(T_{\mathrm{simu}} - T_{\mathrm{fall}}) + \frac{1}{T_{\mathrm{fall}}}\int (v_{\mathrm{COM,\ x}}(t)^2 + v_{\mathrm{COM,\ y}}(t)^2 + v_{\mathrm{COM,\ z}}(t)^2)dt \tag{2}$$

T_{fall} is the time until the musculoskeletal model falls, T_{simu} is the simulation time (5 seconds), and $v_{\mathrm{COM,x}}(t), v_{\mathrm{COM,y}}(t), v_{\mathrm{COM,z}}(t)$ are the COM velocities x, y and z-axis direction at *t* seconds. The convergence condition was set when the difference in the evaluation function between iterations was less than 1.0×10^{-5}, or the number of iterations exceeded 10000.

E. Evaluation

We compared the postures and the sway obtained in the simulation results with those in the experimental results to verify whether or not the calculated postures with a small sway and the sway of the estimated muscle tones are similar to those of patients with PD. The average difference of all joint angles was used for the posture index, while the COM velocity was employed for the sway index. We verified whether or not the simulation results at each muscle tone were similar to those in the experimental results by calculating the difference in the indices between the simulation and experimental results. Normalization also was performed by dividing each index by the mean value of the simulation results for each subject to compare, considering both indices. We used the distance from the origin in both index plots as the index for the similarity of the simulation results to the experimental results. This index allowed us to compare the simulation results with the experimental ones, considering both the posture and sway indices for each muscle tone. We compared the results of $\|\boldsymbol{u}_{\mathbf{ff}}\|^2 = 2$ to higher muscle tones because the previous study showed that the simulation result for the muscle tone with an index of $\|\boldsymbol{u}_{\mathbf{ff}}\|^2 = 2$ is plausible to healthy subjects [13].

III. RESULTS

All optimizations converged within 10000 generations, one of the convergence conditions. Fig. 1 shows the abnormal posture reproduced in the musculoskeletal model using the data of patients with PD. The musculoskeletal model could represent the flexed postures of patients with PD.

Fig. 2 shows the COM velocity differences between the simulation and experimental results. The differences in the COM velocities were smaller at higher muscle tones than $\|\boldsymbol{u}_{\mathbf{ff}}\|^2 = 2$ (sub1: $\|\boldsymbol{u}_{\mathbf{ff}}\|^2 = 4, 6, 8$, sub2: $\|\boldsymbol{u}_{\mathbf{ff}}\|^2 = 6$, sub3: $\|\boldsymbol{u}_{\mathbf{ff}}\|^2 = 4, 6, 8$).

Fig. 3 shows the mean difference of all the joint angles between simulation and experimental results. The standard deviations of the higher muscle tones are smaller than those of $\|\boldsymbol{u}_{\mathbf{ff}}\|^2 = 2$ in subs 1 and 3.

Fig. 4 depicts the normalized values of these differences. The muscle tones with the smallest distance from the origin were sub1: $\|\boldsymbol{u}_{\mathbf{ff}}\|^2 = 8$, sub2: $\|\boldsymbol{u}_{\mathbf{ff}}\|^2 = 6$, and sub3: $\|\boldsymbol{u}_{\mathbf{ff}}\|^2 = 6$. The distances were sub1: 0.98, sub2: 1.04, and sub3: 0.66.

IV. DISCUSSION

Fig. 2 illustrates that the differences in the COM velocities were small at higher muscle tones than $\|\boldsymbol{u}_{\mathbf{ff}}\|^2 = 2$, which was plausible to healthy subjects in the simulations [13]. The large difference in the COM velocities between the simulation and experimental results implied that the sway was reproduced differently in the computational model from the experimental results. These results demonstrated that the sway of the simulation results at higher muscle tones than $\|\boldsymbol{u}_{\mathbf{ff}}\|^2 = 2$ was similar to that of the experimental results.

Fig. 3 demonstrates that the standard deviations at the high muscle tones were smaller than those at the muscle

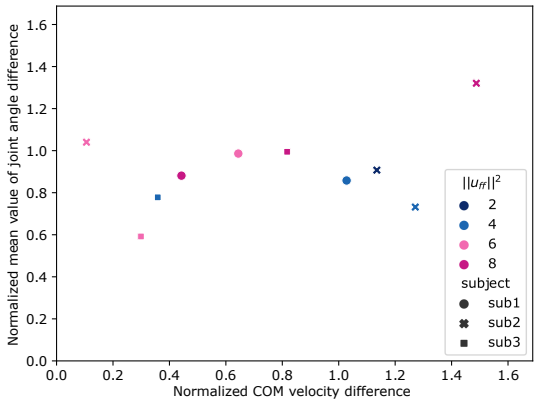

Fig. 4. Normalized average values of the joint angle difference and normalized differences of the average COM velocity between simulation and experimental results for each subject. Normalization was performed by dividing each value by the average value of the simulation results in each condition. The distance from the origin evaluated the difference between the experimental and simulation results for each condition. Each plot's shape and color represent the subjects and the muscle tone index $\|\boldsymbol{u}_{\mathbf{ff}}\|^2$, respectively.

tones where $\|\boldsymbol{u}_{\mathbf{ff}}\|^2 = 2$ in subs 1 and 3. The small standard deviations implied small changes in the posture angle (i.e., less sway at each body). In other words, the standings reproduced at these muscle tones in the simulations were possible, even without the small sway, which was not reflected in the COM velocity.

Regarding both indices, Fig. 4 shows that the muscle tones with the smallest distance from the origin were $\|\boldsymbol{u}_{\mathbf{ff}}\|^2 = 6$ or 8 for each subject. The standings reproduced in these muscle tones in the simulations were the most similar to those in the experimental results compared to those reproduced in the other muscle tones in the simulations. These muscle tones were about twice higher than $\|\boldsymbol{u}_{\mathbf{ff}}\|^2 = 2$, which was plausible to healthy subjects in the simulations [13]. We optimized the parameters, including postures, based on the evaluation function of the sway at the muscle tones of various magnitudes. The result showed that the reproduced postures and sway at higher muscle tones than those were plausible for healthy subjects in simulations [13] were similar to those in the experimental results. The similarity of the sway in the simulation results to that in the experimental results indicates that the muscle tones, where the reproduced sway is similar to that in the experimental results, are plausible for patients with PD compared to other muscle tones. Meanwhile, the similarity of the postures indicated that the postures similar to those of patients with PD (i.e., abnormal postures) are one of the local solutions (postures) with a small sway in the simulations. Therefore, the result that both indices were small at higher muscle tones than $\|\boldsymbol{u}_{\mathbf{ff}}\|^2 = 2$ suggests that these muscle tones in the simulations are plausible to those of patients with PD, and that calculated postures with a small sway locally at these muscle tones in the simulations are similar to actual postures of patients with PD. These simulation results are consistent with our hypothesis.

In conclusion, the computational model results are consistent with our hypothesis. The results also reinforce the hypothesis and imply that the abnormal posture in actual patients with PD may be a static standing posture with a small sway locally in response to increased muscle tones. We aim to explain the abnormal postures and postural control disorders in patients with PD on a computational model by analyzing the relationship between the FF and FB control parameters and abnormal postures.

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