POSTURAL CONTROL MODEL OF SPASTICITY IN PERSONS WITH MULTIPLE SCLEROSIS

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INTRODUCTION
Multiple sclerosis (MS) is a neurological disease affecting the myelin sheaths of nerves in the central nervous system. One symptom caused by demyelination and its location is spasticity, a velocity-dependent tightness or resistance to stretch of the muscle. Previous research indicated that spasticity was associated with worse postural sway in persons with MS [1]. However, it is unknown why spasticity should result in greater postural sway. Mathematical models of the postural control system may help to clarify this apparent relationship between spasticity and sway.

Closed-loop inverted single link pendulum models with time delayed proportional-integral-derivative (PID) or proportional-derivative (PD) control in the anterioposterior (AP) direction are traditionally the models of choice for examining the upright standing postural control system (e.g., [2, 3]). Recent work has also used the inverted pendulum model to examine sway in the mediolateral (ML) direction [4]. Inverted pendulum models in the AP plane have shown that parameters, such as stiffness and damping in the neurological controller and physiological noise, increase as postural sway increases due to aging [2, 5].

METHODS
As per previous modeling work [2-4], the inverted pendulum represented the human body. The neural controller was represented by a PID controller with a neutral set point, and proportional, derivative, and integral gains (Kp, Kd, and Ki). Additional control components were included which account for the passive components of body at the ankle (stiffness Kpass and damping Bpass) (Figure 1a). The input noise characteristics, which simulate physiological noise such as breathing, were determined by two gains, KN and τN. KN was the gain of a low pass filter on the input noise, and τN was 1/fc, where fc is the cutoff frequency of the low pass filter. The last parameter was the time delay in the feedback control concerning the transmission and processing time of neurological signals, τd. The body parameters were set to represent a 50th percentile Caucasian adult female who sways only at the ankle joint (mass 62 kg, height of center of mass 0.85 m, moment of inertia in AP (Jb= 61 kg·m²), and ML directions (Jb = 60 kg·m²)) (Figure 1b) [6].

The postural control data of 16 subjects with MS (9 high, 7 low spasticity) and sixteen age- and sex-matched controls (44±3 y, 14 females, 2 males) were used in the current study and were previously presented [1]. There was no significant difference in disability status (EDSS) between spasticity groups.

Five model parameters (Kp, Kd, KN, Kpass, Bpass) were optimized to match participant COP data. Traditional COP measures such as 95% power frequency, sway range, and maximum distance were calculated from the output of the model simulation for each direction, and were then compared to similarly computed measures from the participant COP data [1]. The difference in these parameters was minimized to find the optimum model gains. As in the previous work, the model was divided into three test groups (healthy controls and persons with MS with low and high spasticity). Independent samples t-tests were used to compare the model-created traditional parameters to subject parameters. Univariate ANOVA tests with group as the between-subject factor were used to compare model gains. All statistical analyses were completed with SPSS version 17.0 (SPSS, Inc, Chicago, IL).

RESULTS/DISCUSSION
There were no significant differences between the experimental subject traditional COP parameters and corresponding model simulation parameters for any test group. Therefore the models recreated traditional sway parameters for the two MS and control group as noted previously [1].

The model gains used to achieve the simulated COP trajectories were analyzed (Table 1). In the AP
direction, the low spasticity model had significantly higher $K_p$-AP ($p=0.05$) and lower $K_{\text{pass}}$-AP ($p=0.02$). In the ML direction, analysis revealed that the high spasticity model had a significantly higher $K_N$-ML than the controls model ($p=0.04$).

Table 1: Significant model gains ($p < 0.05$).

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>MS- low spasticity</th>
<th>MS- high spasticity</th>
</tr>
</thead>
<tbody>
<tr>
<td>$K_p$-AP</td>
<td>10.3 ± 1.1</td>
<td>12.1 ± 2.9**</td>
<td>10.8 ± 1.0</td>
</tr>
<tr>
<td>$K_{\text{pass}}$-AP</td>
<td>10.7 ± 0.6</td>
<td>9.4 ± 1.6**</td>
<td>10.4 ± 0.6</td>
</tr>
<tr>
<td>$K_N$-ML</td>
<td>0.5 ± 0.3</td>
<td>2.2 ± 3.5</td>
<td>3.3 ± 3.6*</td>
</tr>
</tbody>
</table>

* Different from control  
** Different from control and MS-high spasticity

Since differences were seen between the control and MS-high spasticity group, as well as between the control and MS-low spasticity group, it is likely that these differences are related to spasticity as well as to MS. Persons with MS and low spasticity potentially use different compensatory mechanisms, than those with high spasticity, in the AP direction to help control instability in their posture caused in part by the neurological signal degradation due to MS. It is possible that a difference in persons with MS and high spasticity and controls is not seen in the AP direction, as persons with high spasticity may be unable to modulate the sway in the AP direction due to higher levels of spasticity, and thus compensate by swaying in the ML direction. One could speculate that the increased $K_N$-ML physiologically represents the degradation of the neural signal transduction in persons with MS [2].

**CONCLUSIONS**

The goal of this study was to determine if the difference in experimental COP measures between controls and persons with MS could be recreated in inverted pendulum models of AP and ML postural sway and which model gains varied to cause the group differences in traditional COP parameters.

These significant differences in gains suggest that persons with MS use different control strategies depending on the level of spasticity than healthy persons to maintain postural control, such as increasing sway in the ML direction, where they have a larger base of support or modulating the stiffness of muscles to better control sway.

Results from this study can be used to inform future research on postural control and the role of spasticity in persons with MS, as well as rehabilitation to regain normal postural control.

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**REFERENCES**