Neural Control of Posture During Small Magnitude Perturbations: Effects of Aging and Localized Muscle Fatigue

Bradley S. Davidson*, *Member, IEEE*, Michael L. Madigan, Steve C. Southward, and Maury A. Nussbaum

***Abstract*—This study investigated the effects of aging and localized muscle fatigue on the neural control of upright stance during small postural perturbations. Sixteen young (aged 18–24 years) and 16 older (aged 55–74 years) participants were exposed to small magnitude, anteriorly-directed postural perturbations before and after fatiguing exercises (lumbar extensors and ankle plantar flexors). A single degree of freedom model of the human body was used to simulate recovery kinematics following the perturbations. Central to the model was a simulated neural controller that multiplied time-delayed kinematics by invariant feedback gains. Feedback gains and time delay were optimized for each participant based on measured kinematics, and a novel delay margin analysis was performed to assess system robustness. A 10.9% longer effective time delay ($p = 0.010$) was found among the older group, who also showed a greater reliance upon velocity feedback information (31.1% higher differential gain, $p = 0.001$) to control upright stance. Based on delay margins, older participants adopted a more robust control scheme to accommodate the small perturbations, potentially compensating for longer time delays or degraded sensory feedback. No fatigue-induced changes in neural controller gains, time delay, or delay margin were found in either age group, indicating that integration of this feedback information was not altered by muscle fatigue. The sensitivity of this approach to changes with fatigue may have been limited by model simplifications.**

***Index Terms*—Delay effects, fatigue, posture, stability.**

I. INTRODUCTION

AGE-RELATED increases in center of pressure (COP) measures during quiet standing are well documented [1]–[5]. These increases could result from age-related degradation of afferent information from the somatosensory [6], [7] and vestibular [8]–[10] systems. Increases in COP measures could also result from inaccurate or imprecise muscle forces resulting from excitation–contraction uncoupling in older adults [11], [12]. Localized muscle fatigue (LMF) has also been shown to increase COP measures during quiet standing [13]–[16]. Several of these reports have speculated that these changes result from LMF-induced inaccuracies in proprioceptive feedback from the fatigued muscles [13], [15], [17]–[19].

An alternative explanation for the age and LMF-related increases in postural sway is an alteration in how sensory feedback is integrated to generate motor commands or neural control. In support of this, several studies have reported increased COP-based measures of postural sway following LMF in muscles that do not have a critical role in maintaining upright posture, such as the neck extensors [20], [21] and shoulder flexors [16]. Additionally, deficits in ankle proprioceptive acuity have been reported with LMF of the lumbar extensors [22], and deficits in shoulder proprioceptive acuity with LMF of the contralateral shoulder [23]. These results suggest a possible alteration in central processing of proprioceptive signals with LMF [24]. Methods to directly quantify the neural controller of posture are unavailable, so indirect approaches must be used that involve modeling the system.

A variety of approaches have been used to identify the neural controller of upright stance using a variety of methods (for a comparison and review, please see Van der Kooij *et al.* [25]). In particular, researchers have relied upon feedback-controlled models of standing posture to gain insight into the neural controller by simulating quiet stance [26]–[30] or a response to a postural perturbation [31]–[34]. When used in combination with experimental data, these models can be used for system identification to obtain neural controller parameters [28], [29], [35]–[37]. Maurer and Peterka [28], for example, employed this approach to parameterize proportional, integral, and differential gains during unperturbed quiet stance in young and older adults. The system was driven by a stochastic input and produced sagittal plane COP trajectories having the same characteristics as experimentally recorded data. To our knowledge, this study was the first to use such a system identification to investigate the changes in the neural controller with aging (or any other factor).

The purpose of this study was to assess the effects of aging and LMF on the neural control of upright stance during postural perturbations. A system identification approach, similar to that described by [28], was used to parameterize a mathematical model of the postural control system that included invariant proportional and differential neural controller gains and time

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delay in the sensory feedback. Perturbations were used instead of quiet stance as it was assumed that this would facilitate identification of the effects of aging and LMF [25]. In addition, by increasing the kinematic range with perturbations, the relatively small stochastic kinematics associated with quiet stance may be neglected [38], [39]. A novel delay margin analysis was performed on the identified systems to assess changes in system robustness to time delay [40].

II. MATERIALS AND METHODS

A. Participants

Thirty-two physically active participants were recruited from the local community including 16 young (19.4 ± 1.4 years old, 71.4 ± 11.1 kg, 174.8 ± 8.3 cm) and 16 older adults (62.2 ± 5.1 years old, 74.0 ± 10.9 kg, 167.8 ± 8.9 cm), with an equal number of males and females in each age group. All participants were screened for self-reported musculoskeletal disorders and medications that could influence balance. In addition, the older participants were required to pass a medical exam to exclude those with neurological, cardiac, respiratory, vestibular, or musculoskeletal disorders, or any falls within the past year. The experiment was approved by the Virginia Tech Institutional Review Board, and all participants provided informed consent prior to participation.

B. Experimental Protocol

Each participant visited the laboratory for two experimental sessions, separated by approximately one week. In each session, kinematic responses to a series of small magnitude postural perturbations were assessed both before and after fatiguing exercises. The ankle plantar flexor muscles were fatigued during one session, and the lumbar extensor muscles were fatigued during the other. The order that muscles were fatigued was counterbalanced across participants.

During testing, participants stood with their eyes closed, feet together, and hands clasped behind their back, and were instructed to "stand in a relaxed manner". Ballistic pendulums were used to administer anteriorly-directed (AD) and posteriorly-directed (PD) force perturbations to the upper trunk in the midsagittal plane. Heights of contact were at the inferior margin of the scapula and just below the jugular notch for the AD and PD perturbations, respectively. Pendulums were moved away from the participants to a set distance and then released. Perturbation magnitude was quantified using the horizontal linear momentum of the pendulum just before impact. All perturbation magnitudes were small enough that a stepping response was not necessary to maintain upright stance. Both AD and PD perturbations were applied in a random order to reduce anticipation effects, but only results from AD perturbations are modeled since observations of the PD perturbations revealed movement at the hip in most participants. Hearing protection earmuffs were worn to minimize auditory cues to an impending perturbation.

The experiment began with ten AD and ten PD perturbations of small-moderate magnitude (10 N·s AD, 7 N·s PD) administered in random order. These initial perturbations, also used in separate investigation of perturbations with much larger magnitude, were intended to habituate the participants and allow any performance adaptation to occur prior to the data collection. After a 1-min break, five AD and five PD small magnitude perturbations (6 N·s AD and 5 N·s PD, respectively) were administered in a random order. These perturbations were randomly timed to decrease anticipation and delivered such that all perturbations were administered in approximately 1 min. These perturbations, along with those collected after the fatiguing exercise (see the following), were used to investigate the effects of fatigue.

Next, either the lumbar extensors or ankle plantar flexors were fatigued to 70% of their unfatigued isometric maximum voluntary contraction (MVC) moment. Dynamic exercises were performed on a System 3 Isokinetic Dynamometer (Biodex Medical Systems, Shirley, NY) for lumbar extensor fatigue and a seated calf-raise machine (New York Barbell Corporation, Elmira, NY) for plantar flexor fatigue. In each configuration, the participants performed the movement against a constant torque equal to 40% of their maximum isometric torque. Lumbar extensor contractions were performed from 90° trunk flexion to an upright position, and the ankle plantar flexor contractions were performed from the neutral position to maximum plantar flexion. The targeted 70% MVC fatigue level was accomplished by systematically adjusting the number of repetitions performed each minute, using intermittent MVCs, so that MVC decreased 30% at a near-linear rate over 14 min [13], [41]. Four minutes following the fatiguing exercises, ten small magnitude perturbations were administered in the same manner as before the exercises.

Body kinematics were monitored during all perturbations using reflective markers placed bilaterally over the acromion and lateral malleolus (Vicon Motion Analysis System, Lake Forest, CA). Marker positions were sampled at 100 Hz and low-pass filtered (5 Hz cutoff, 6th order zero-phase lag Butterworth) during postprocessing. Sagittal plane body segment orientation (θ) was defined by the angle from vertical of a line connecting each ankle/shoulder pair and averaging across sides. Body segment velocity ($\dot{\theta}$) was calculated using center finite divided difference [42]. Maximum body segment orientation ($\theta_{\max}$) and body segment velocity ($\dot{\theta}_{\max}$), each relative to the value at time of pendulum contact, were calculated to assess kinematic changes with fatigue and aging.

A load cell (Cooper Instruments and Systems, Warrenton, VA) was attached inline to the pendulum to monitor perturbation force and was recorded at 1000 Hz. These forces were used to determine perturbation onset time, and also as input to the dynamic simulations described next. Onset time was defined as the sample in which the load cell force increased above a threshold defined as two standard deviations above the baseline mean.

C. Model Development

A two-dimensional, time-delayed, proportionally and differentially controlled dynamic model of human upright stance was created to simulate the recorded data and used to identify parameters based upon the experimental data from each participant. The model consisted of a single body segment (Fig. 1) based

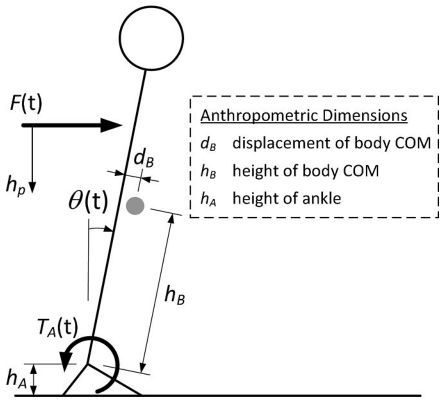

Fig. 1. Free body diagram of the human model (i.e., the plant). $F(t)$ represents the experimentally recorded pendulum force applied to the participant, $T_A(t)$ is the resulting ankle torque produced by the neural controller, $\theta(t)$ is body angle relative to vertical, and h_p is the height of the pendulum upon contact with the participant. (COM = center of mass).

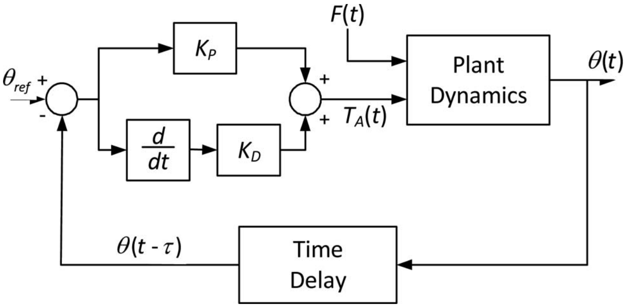

Fig. 2. Schematic of the dynamic model that includes the plant and neural controller (note: passive ankle stiffness and damping are included within the plant dynamics).

on subject-specific anthropometry [43], [44], included passive ankle stiffness and damping, and an active ankle torque established by the neural controller (Fig. 2). Input to the model was the experimentally recorded pendulum force, $F(t)$. Active ankle torque $T_A(t)$ was based on time-invariant proportional (K_P) and differential (K_D) feedback gains operating on the system state delayed by τ_d seconds. An undefined reference angle $\theta_{\rm ref}$ representing the desired equilibrium point was included for each perturbation to account for the naturally occurring slight forward lean during upright stance.

The equations of motion for this system in linear, first-order form are

$$\begin{aligned}\begin{Bmatrix}\dot\theta(t)\\ \ddot\theta(t)\end{Bmatrix} &= \begin{bmatrix}0 & 1\\ \dfrac{-K+m_B g h_B}{J_B} & \dfrac{-B}{J_B}\end{bmatrix}\begin{Bmatrix}\theta(t)\\ \dot\theta(t)\end{Bmatrix}\\ &+\begin{bmatrix}0 & 0\\ \dfrac{-K_P}{J_B} & \dfrac{-K_D}{J_B}\end{bmatrix}\begin{Bmatrix}\theta(t-\tau_d)\\ \dot\theta(t-\tau_d)\end{Bmatrix}\\ &+\begin{Bmatrix}0\\ \dfrac{K_P\theta_{\rm ref}+m_B g d_B}{J_B}\end{Bmatrix}+\begin{Bmatrix}0\\ \dfrac{h_p-h_A}{J_B}\end{Bmatrix}F(t)\end{aligned} \tag{1}$$

TABLE I
PARAMETER SEEDS FOR NELDER-MEAD OPTIMIZATION

Parameter	Starting Value	Initial Step Size
K_P (N·m/rad)	500	200
K_D (N·m·sec/rad)	200	100
τ_d (msec)	170	30
θ_{ref}(1…5) (rad)	0.05	0.1

where $\theta(t)$, $\dot\theta(t)$, and $\ddot\theta(t)$ are the time-dependent angle, angular velocity, and angular acceleration of the body segment relative to vertical; g is gravitational acceleration; h_B, d_B, and h_A describe the anthropometrics of the participant (Fig. 1); m_B and J_B are the mass and moment of inertia of the body segment about the ankle; h_p is the height above the ground at which the pendulum makes contact; K is the passive ankle stiffness, taken from [45]; and B is the passive ankle damping, determined from the empirical formula given in [46] as:

$$B = 0.76\sqrt{4J_B K}. \tag{2}$$

Using body orientation (θ) from the five AD perturbations from each individual, eight model parameters were estimated for each experimental combination of fatigued muscle group (ankle plantar flexor, lumbar extensor) and fatigue level (unfatigued, fatigued). Specifically, these parameters were the system time delay τ_d, proportional gain K_P, differential gain K_D, and five trivial reference angles $\theta_{\rm ref}$ (1…5), one for each of the perturbations. Parameter values were obtained in this underdetermined situation by minimizing a scalar cost function representing the difference between simulated and measured values of θ:

$$V = \sum_{n=1}^{\rm pert}\left(\sum_{m=1}^{\rm sample}(\hat\theta_{mn}-\theta_{mn})^2\right) \tag{3}$$

where the summation is obtained over the m samples in each of the n perturbations, and the hat indicates simulated values. A Nelder–Mead search [47], [48] was used to perform the optimization [28]. Since the Nelder–Mead algorithm is a local nonlinear search, an appropriate choice of the initial parameters is crucial in order to maximize the likelihood of global optimal values. Accordingly, each simulation began with values for neural controller gains and time delay similar to those reported in [28] and small reference angles (Table I). Simulations began at the time of perturbation onset and lasted for 1.7 s. This simulation length was chosen by determining the longest simulation time with stabilized model parameters and prior to noticeable loss of model performance, as indicated by a decrease in r^2 (Fig. 3). All simulations were performed in Fortran90 (Intel Fortran Compiler 9.1, San Jose, CA).

Robustness of the identified control system (i.e., eight estimated parameter values from each individual and condition) was quantified using an absolute delay margin, or the maximum time delay the system could withstand before becoming unstable—poles of the system becoming positive. Absolute delay margin ($\bar\tau_{\rm abs}$) was determined using a frequency-sweeping method, first proposed by [40] and further described in the Appendix. Robustness to time delay must also be considered in light of the existing

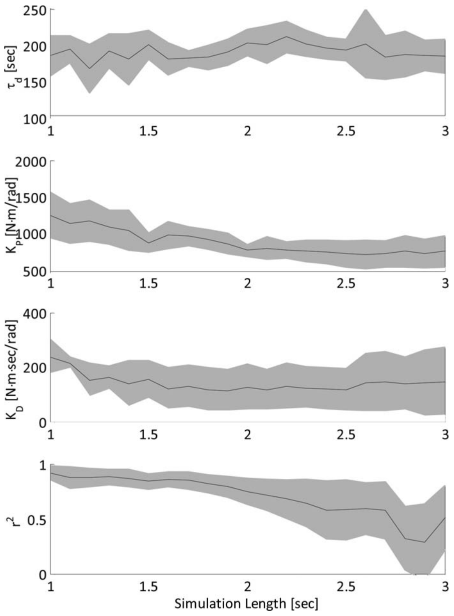

Fig. 3. Mean ± 95% confidence intervals of time-delay (τ_d), neural controller parameters (K_P, K_D), and coefficient of determination (r^2) as a function of simulation length. Selection criteria included the best combination of the following: 1) high r^2-value, 2) low variance in each metric, and 3) mean and variance were stable within the region around the simulation length. Data from four randomly chosen subjects were used to select a simulation length of 1.7 s for this investigation.

TABLE II
SUMMARY STATISTICAL RESULTS FOR ALL VARIABLES

Dependent Measure	Interactions		Main Effects		
	Fatigue x Age	Fatigue x Muscle	Fatigue	Age	Muscle
Kinematics					
$\theta_{\max}$ (deg)	*p*=0.774	*p*=0.496	*p*=0.560	*p*=0.376	*p*=0.103
$\dot{\theta}_{\max}$ (deg/sec)	0.019*	0.336	0.216	0.573	0.336
Model Pars.					
K_P (N·m/rad)	0.291	0.511	0.910	0.953	0.756
K_D (N·m·sec/rad)	0.471	0.416	0.951	0.001*	0.578
τ_d (msec)	0.784	0.955	0.753	0.010*	0.108
Delay Margins					
$\bar{\tau}_{abs}$ (msec)	0.823	0.830	0.815	0.001*	0.635
$\bar{\tau}_{rel}$ (msec)	0.763	0.992	0.817	0.001*	0.933

*Indicates significant age effect ($p \leq 0.05$).

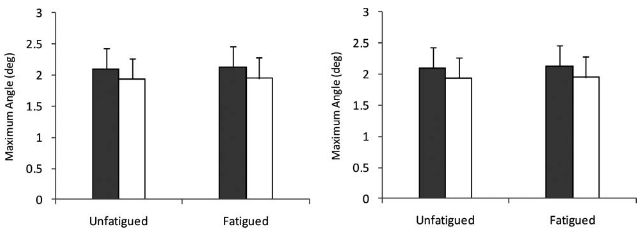

Fig. 4. Least square means (with standard deviation) of maximum angular kinematics for each group and condition. * Indicates significant difference ($p \leq$ 0.05).

time delay present in the postural control system. Therefore, a relative delay margin ($\bar{\tau}_{\mathrm{rel}}$) was also calculated using the time delay identified from the prior optimization

$$\bar{\tau}_{\mathrm{rel}} = \bar{\tau}_{\mathrm{abs}} - \tau_d. \tag{4}$$

Sensitivity of $\theta(t)$ to variations in the identified parameters was assessed by performing additional simulations while varying K_P, K_D, and τ_d between 60% and 140% of the optimized values. This was done for values representing the mean across young and older groups. Because characteristics of a linear system are mathematically bounded relative to changes in the neural controller gains and time delay, qualitative changes in the simulations would be similar across participants regardless of anthropometry. In addition, sensitivity of the absolute delay margin to variations of the neural controller gains K_P and K_D was assessed by simultaneously varying these over a range of 60%–140%.

D. Statistical Assessment

Goodness of fit between simulated body position and the experimental body position was calculated for the series of five perturbations by the coefficient of determination (r^2). A mixed-factor analysis of variance (ANOVA) was used assess the effects of age, muscle group, and LMF on the kinematic variables and identified parameters from the model. Only interactions of LMF with age and LMF with muscle group were included as higher order effects in the statistical model. Dependent variables were the maximum body segment orientation ($\theta_{\max}$), maximum body segment velocity ($\dot{\theta}_{\max}$), optimized neural controller gains (K_P, K_D), time delay (τ_d), and delay margins ($\bar{\tau}_{\mathrm{abs}}, \bar{\tau}_{\mathrm{rel}}$). Since variability of the perturbation magnitude systematically influences body kinematics [49], pendulum momentum at the time of contact was included as a covariate in the statistical model for the kinematic variables. Three of the dependent variables ($K_D, \bar{\tau}_{\mathrm{abs}}$, and $\bar{\tau}_{\mathrm{rel}}$) required a square root transformation to achieve normality. Where significant interactions were found, pairwise comparisons were performed using a Tukey HSD analysis. Effects were considered significant when $p \leq 0.05$.

III. RESULTS

Maximum body angle was not affected by age, muscle group, fatigue, or any two-way interactions of these independent variables (Table II). Maximum angular velocity demonstrated a two-way interaction of age and fatigue ($p = 0.019$). Pairwise comparisons revealed greater maximum velocities in the young group compared to the older group, but no changes with fatigue in either age group (Table II, Fig. 4). Maximum angular velocity in the young group was 0.29°/s (2.9%) and 0.59°/s (6.0%) higher than the older group in unfatigued and fatigued conditions, respectively.

The optimized dynamic model approximated the experimental data with $r^2 = 0.82 \pm 0.09$ and range of 0.55–0.96 (Fig. 5). Compared to the young adults, older adults exhibited 31.0 N·m·s/rad higher differential gain ($p = 0.001$, 31.1%) and 23 ms longer time delay ($p = 0.010$, 10.9%). Older adults also had a 75 ms higher absolute delay margin ($p = 0.001$, 18.5%) and a 52 ms larger relative delay margin ($p = 0.001$, 26.7%).

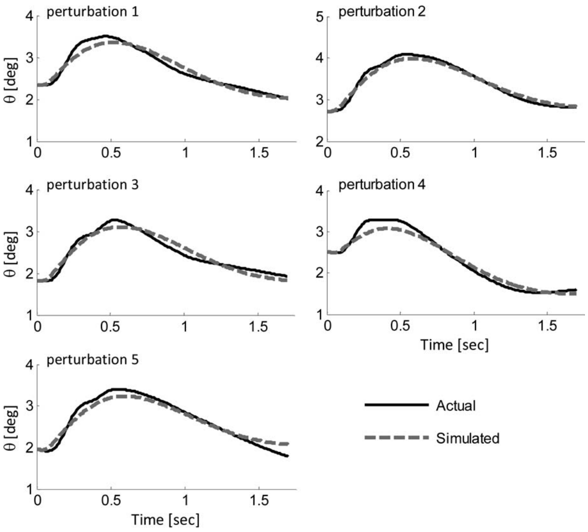

Fig. 5. Representative time histories of experimental and simulated body angle from a single subject during five perturbations. Identified simulation parameters for this series were $\tau_d = 200$ ms, $K_P = 1033.4$ N·m/rad, $K_D = 152.1$ N·m·s/rad, $\theta_{\mathrm{ref}}(1) = 0.014$ rad, $\theta_{\mathrm{ref}}(2) = 0.019$ rad, $\theta_{\mathrm{ref}}(3) = 0.017$ rad, $\theta_{\mathrm{ref}}(4) = 0.016$ rad, $\theta_{\mathrm{ref}}(5) = 0.019$ rad, and $r^2 = 0.91$.

TABLE III
MEAN ± SD OF IDENTIFIED MODEL PARAMETERS AND DELAY MARGINS

Dependent Measure	Young	Older	Unfatigued	Fatigued
Model parameters				
K_P (N·m/rad)	963.9±203.2	864.6±197.3	911.1±223.4	916.6±186.9
K_D (N·m·sec/rad)	98.3±103.5	129.3±82.1*	111.8±93.4	116.1±95.8
τ_d (msec)	188±16	211±21 *	199±21	200±23
Delay margins				
$\bar{\tau}_{abs}$ (msec)	331±98	406±120 *	337±128	365±102
$\bar{\tau}_{rel}$ (msec)	143±98	195±116 *	174±122	165±98

*Indicates significant age effect ($p \leq 0.05$).

No main effects or two-way interactions involving fatigue or muscle group were present (Tables II and III).

Varying neural controller gains and time delay around values representing the mean across young and older groups revealed distinct changes in the simulated response to a postural perturbation (Fig. 6). The angular response retained an underdamped shape with variations in τ_d and K_D. Magnitudes of the displacements around the equilibrium point were generally proportional to changes in τ_d and inversely proportional to changes in K_D. Conversely, varying K_P did change the shape of the response with an increase producing a smaller maximum angular displacement with underdamped characteristics. Decreasing K_P caused an increase in the peak displacement and overdamped characteristics. Simultaneously varying K_P and K_D revealed that $\bar{\tau}_{\mathrm{abs}}$ increased with increasing K_D and decreased with increasing K_P (Fig. 7).

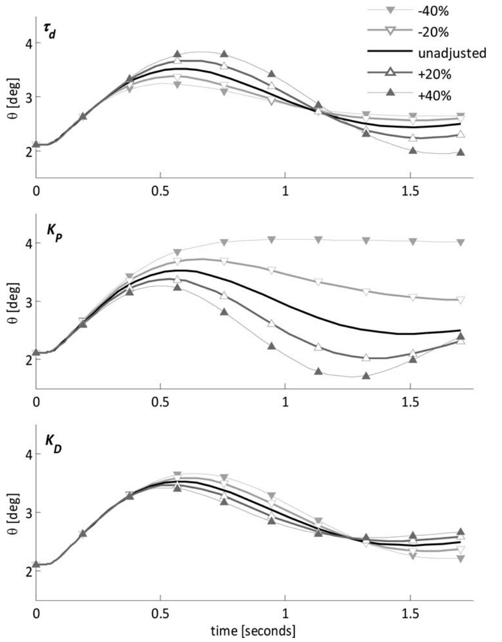

Fig. 6. Effects of varying model parameters on kinematic response to perturbation. The traces are trajectories as the time-delay and neural controller gains are varied from mean −40% to mean +40% in 20% increments. The shape of the trajectory was most sensitive to change in K_P and least sensitive to changes in K_D.

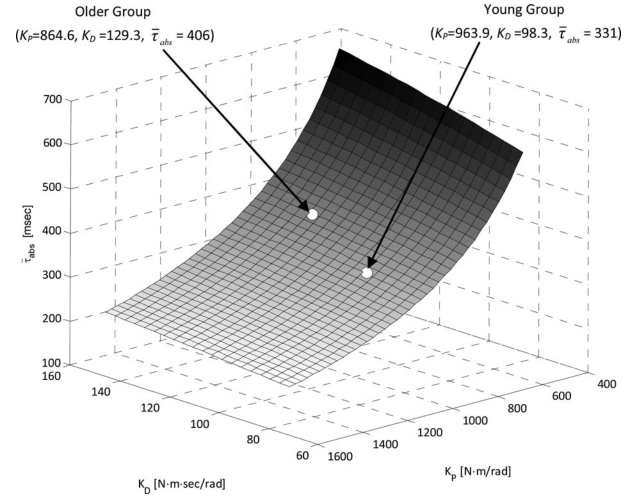

Fig. 7. Combined effects of neural controller gains (proportional gain K_P and differential gain K_D) on the absolute delay margin ($\bar{\tau}_{\mathrm{abs}}$). Here, $\bar{\tau}_{\mathrm{abs}}$ is an indicator of robustness of the system where higher values indicate greater robustness. The interaction of K_D and K_P demonstrated that $\bar{\tau}_{\mathrm{abs}}$ increased as K_D increased, and $\bar{\tau}_{\mathrm{abs}}$ decreased as K_P increased. In general, $\bar{\tau}_{\mathrm{abs}}$ was more sensitive to changes in K_P than K_D.

IV. DISCUSSION

The purpose of this study was to investigate changes with aging and LMF in the neural control of posture in the context of mild perturbations to upright stance using empirical and theoretical methodology. Maximum kinematic variables were calculated from a series of small magnitude postural perturbations, and parameters of a feedback-controlled dynamic model of the human body were optimized to match the kinematic trajectories. Analysis of the kinematics revealed no changes with muscle group, LMF, or age in maximum body angle; however, the young group had 2.9%–5.9% larger maximum velocity than the older group. Parameters derived from the model demonstrated several differences related to age, including a 10.9% longer time delay and 31.1% higher differential gain in the older group. In addition, the older group had 18.5% and 26.7% larger absolute and relative delay margins, respectively, indicating that older adults exhibited an overall more robust control scheme than young adults for small magnitude perturbations. When considering these margins of stability, it should be noted that these values do not represent the stability of the system, but are indicators of robustness (or sensitivity) toward becoming unstable. No changes in neural controller gains, time delay, or delay margins were found with LMF, suggesting LMF did not alter the central processing of position and velocity information that led to corrective motor commands.

The optimized proportional and differential gains exhibited a similar pattern to previously published values [28] determined during quiet stance, in that $K_P > K_D$. Actual values in the current investigation (young group: $K_P = 963.9$, $K_D = 98.3$) were higher than those reported in the model representation [28] that included intrinsic ankle stiffness and damping (young group: $K_P = 573.0$, $K_D = 19.5$). This could simply be a result of differences in the neural control of quiet stance and postural perturbations, or perhaps due to the slight forward lean used in this model. Values of the identified time delay (young group: $\tau_d = 188$ ms; older group: $\tau_d = 211$ ms) were consistent with previous estimates that physiological time delay is >150 ms [50], and were slightly higher than those values identified during quiet stance (young group: $\tau_d = 177$ ms; older group: $\tau_d = 163$ ms) [28]. The higher differential gain found here among older adults is similar to the trend reported by [28] using quiet stance, but the longer time delay in older adults found here differed from the shorter time delay reported in the same study.

A longer time delay in the older group is consistent with the documented decrease in nerve conduction velocity with increasing age [51]–[53]. The identified time delay here can be considered as an effective time delay, rather than an actual physiological value, that represents feedback loops of shorter and longer delays such as short-latency reflexes, long-latency reflexes, and central feedback loops. This effect may reflect a shift in control strategy concomitant with changes in other neural controller variables (discussed as follows). We must also consider that an increase in the effective time delay essentially reduces the quality of the feedback information in the simulations. Since age-related losses in proprioceptive [6], [7] and vestibular acuity [8]–[10] were not explicitly modeled, these deficits may have emerged as an increase in effective time delay.

Differential gain was significantly larger in the older group. This is consistent with the kinematic findings, specifically that body segment velocity was significantly smaller in the older group. Masani *et al.* [54], [55] recently suggested that the neural controller largely relies on velocity feedback during quiet stance. Since velocity feedback contains implicit information about subsequent positions of the body, velocity-based motor commands can be considered "anticipatory" in nature and effectively eliminate the necessity for a feed-forward mechanism to compensate for physiological time delay [54], [55]. In addition, Jeka *et al.* [56] have proposed that afferent velocity information is more accurate than afferent position information. Accordingly, one could surmise that older participants adopt a control scheme with an increased anticipatory element, and also depend more on kinematic information that is more accurate by using a high differential gain in the neural controller.

Relative delay margin, which takes into account the identified time delay, was higher in the older participants despite having a higher time-delay than the young group. This additional robustness (resulting from the higher differential gain) is indicative of a more conservative neural control scheme employed by the older group. It is common in older adults to employ a more conservative or cautious approach in other movements such as during gait [57], [58]. Reasons for adopting a more conservative control scheme may be to compensate for age-related deficits in sensory acuity [6]–[10], impaired force production [11], [12], or as an additional safeguard against a substantial change in recovery strategy. It should be noted that this more robust neural control scheme and these results cannot be extrapolated to larger perturbations, more likely to be associated with falls in older adults. Using small perturbations in this investigation allowed accurate simulation using a single body segment with a fixed foot, and the delay margins are unique to these dynamics. To assess robustness during larger perturbations that elicit a "hip strategy" [59], [60] or stepping response [49], additional degrees of freedom and more complex feedback control must be used.

Sensitivity analysis of the absolute delay margin with respect to changes in neural controller gains revealed a decrease in absolute delay margin with increasing proportional gain and an increase in absolute delay margin with increasing differential gain (Fig. 7). The proportional gain had a greater effect on the absolute delay margin over the range of values investigated. Although lowering the proportional gain resulted in a greater delay margin, it introduced a larger anterior displacement (Fig. 6) and slowed the response such that it may not be physiologically accurate. In contrast, changes in differential gain had a smaller effect on body angle following a perturbation, but still a noticeable effect on the absolute delay margin. These observations are notable because they imply different roles for each of the neutral controller gains in this system. In this scenario, the proportional gain would be primarily responsible for regulating body angle, and the differential gain would be increased to a level necessary to ensure an appropriate margin of stability. Although an increase in differential gain would result in a greater absolute delay margin in this system, an excessive increase would reduce the speed of the system response such that it may not be physiologically accurate.

Neither ankle plantar flexor nor lumbar extensor fatigue elicited changes in kinematics, neural controller parameters, or delay margins. Coupled with the concomitant results of increased differential gain and decreased maximum velocity, these findings suggest that a simple dynamical system may possess the ability to simulate a complex neural control system under certain conditions. However, the question of whether LMF alters central processing in the neural controller to cause changes in kinematic measures remains unanswered in this investigation. By extending this approach to larger magnitude postural perturbations, where kinematic and kinetic effects of fatigue are apparent [49], [61], additional information about the neural control system in the presence of fatigue may be uncovered.

Several limitations and/or assumptions in our model must be mentioned. First, the system dynamics were composed of rigid bodies, linearized, and based on a gross simplification of the neural controller. Although the simulations yielded satisfactory fits to the experimental data, physiological systems such as this are highly nonlinear in both dynamics and control structure [62]. The rigid body assumption precluded the assessment of impact dynamics during the momentum transfer from the pendulum to the participant. We chose to linearize the system because few analytical tools currently exist to address these nonlinearities while also permitting a meaningful interpretation of the results. Before a full analysis was undertaken, simulation results were compared using linear and nonlinear plant dynamics for four randomly chosen participants. Negligible differences in kinematics and feedback parameters were found between these realizations in each participant. Second, the model incorporated only a single degree of freedom based on observations of participants employing the so-called "ankle strategy" [59], [63]. However, recent investigations [64]–[66] present evidence of hip coordination during control of quiet stance involves hip movement suggest that perturbed stance situations such as in this experiment may also merit additional degrees of freedom. Third, passive stiffness of the ankle joint has been shown to be modulated by muscle contractions (damping is not affected) [67]. Therefore, the time-invariant characterization presented here may have affected the accuracy of the simulations. Finally, we assumed that the neural control systems in both young and older adults are identical in structure. Considering evidence indicating aging-specific changes in restructuring of motoneurons and skeletal muscle [68], similar restructuring may occur on a more fundamental level in the central nervous system that directly affects the structure of motor control processing.

In summary, changes in the human neural controller with aging and LMF were investigated using a feedback-controlled dynamic model that accurately simulated experimentally collected responses to small postural perturbations. The results revealed a longer effective time delay and an increased reliance on velocity feedback in the neural controller in the older group. Calculated delay margins indicated that the older participants adopt a more robust control scheme, potentially to compensate for higher time delays or poor sensory feedback information. It is possible that these findings may be generalized to a broader population and may be validated when simulating other paradigms of postural control. Two overlooked aspects of postural control in previous formulation and analysis of upright stance simulations were addressed—forward lean and effects of time delay on system stability. Both additions, which are pertinent to evaluating strategies of the postural control system, can be applied to other models of upright stance and are recommended to future dynamic realizations by other investigators.

APPENDIX

The linear time-delayed dynamics can be written in the following state-space form:

$$\dot{\mathbf{x}}(t) = \mathbf{A}_0\mathbf{x}(t) + \mathbf{A}_1\mathbf{x}(t-\tau) + \mathbf{g} + \mathbf{b}F(t) \tag{A1}$$

where $\mathbf{x}(t)$ is the state vector $[\,\theta(t) \quad \dot{\theta}(t)\,]^T$, $\mathbf{A}_0$ is the nondelayed state matrix, $\mathbf{A}_1$ is the delayed state matrix, $\mathbf{g}$ is a constant acceleration vector, $\mathbf{b}$ is the input vector, and τ is an arbitrary positive-valued time delay.

Linear stability is characterized only by the eigenvalues of the matrices $\mathbf{A}_0$ and $\mathbf{A}_1$, which map the state and the delayed state, respectively, to the time-differentiated state. Thus, we can eliminate the forcing term $\mathbf{b}F(t)$ and constant term $\mathbf{g}$ from the equation.

Transforming the reduced equation into the Laplace domain gives

$$\mathscr{L}[\dot{\mathbf{x}}(t) = \mathbf{A}_0\mathbf{x}(t) + \mathbf{A}_1\mathbf{x}(t-\tau)] \tag{A2}$$

$$sX(s) = \mathbf{A}_0X(s) + \mathbf{A}_1X(s)e^{s\tau} \tag{A3}$$

where s is the Laplace variable, $X(s)$ is the Laplace transformed $\mathbf{x}(t)$, and $e^{s\tau}$ is the time delay multiplier. If the system eigenvalues are located in the left half of the Laplace plane (Re(s) < 0), the system is considered to be stable. Assuming that the non-time-delayed system is stable, there may exist a time delay $\bar{\tau}$ that will cause a system eigenvalue to cross into the right half of the plane (Re(s) > 0). Thus, by sweeping the frequency domain ($s = j\omega \;\forall\; \omega > 0$), the time delay that causes an eigenvalue to cross into the right-half plane is found. Constraining the Laplace variable to the imaginary axis yields

$$j\omega X(j\omega) = \mathbf{A}_0X(j\omega) + \mathbf{A}_1X(j\omega)e^{j\omega\tau}. \tag{A4}$$

The terms are rearranged to obtain

$$(j\omega\mathbf{I} - \mathbf{A}_0)X(j\omega) = \lambda\mathbf{A}_1X(j\omega), \quad \lambda = e^{j\omega\tau} \tag{A5}$$

which is the generalized eigenvalue problem where j is the imaginary variable, ω is the frequency, and $\mathbf{I}$ is a 2×2 identity matrix. Solutions to this problem only exist at a frequency ω, and time delay τ, where the magnitude of λ is equal to the unit modulus according to Euler's formula

$$|\lambda| = \sqrt{\cos^2(\omega\tau) + \sin^2(\omega\tau)} = 1. \tag{A6}$$

The solution τ, when simultaneously solving (A5) and (A6), is the absolute delay margin, denoted as $\bar{\tau}_{\mathrm{abs}}$.

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REFERENCES

[1] R. W. Baloh, T. D. Fife, L. Zwerling, T. Socotch, K. Jacobson, T. Bell, and K. Beykirch, "Comparison of static and dynamic posturography in young and older normal people," *J. Amer. Geriatr. Soc.*, vol. 42, pp. 405–412, May 1994.

[2] R. A. Du Pasquier, Y. Blanc, M. Sinnreich, T. Landis, P. Burkhard, and F. J. Vingerhoets, "The effect of aging on postural stability: A cross sectional and longitudinal study," *Neurophysiol. Clin.*, vol. 33, pp. 213–218, Nov. 2003.

[3] T. Fujita, S. Nakamura, M. Ohue, Y. Fujii, A. Miyauchi, Y. Takagi, and H. Tsugeno, "Effect of age on body sway assessed by computerized posturography," *J. Bone Miner. Metab.*, vol. 23, pp. 152–156, 2005.

[4] H. Nakamura, T. Tsuchida, and Y. Mano, "The assessment of posture control in the elderly using the displacement of the center of pressure

after forward platform translation," *J. Electromyogr. Kinesiol.*, vol. 11, pp. 395–403, Dec. 2001.

[5] T. E. Prieto, J. B. Myklebust, R. G. Hoffmann, E. G. Lovett, and B. M. Myklebust, "Measures of postural steadiness: Differences between healthy young and elderly adults," *IEEE Trans. Biomed. Eng.*, vol. 43, no. 9, pp. 956–966, Sep. 1996.

[6] J. W. McChesney and M. H. Woollacott, "The effect of age-related declines in proprioception and total knee replacement on postural control," *J. Gerontol. A Biol. Sci. Med. Sci.*, vol. 55, pp. M658–M666, Nov. 2000.

[7] S. W. Shaffer and A. L. Harrison, "Aging of the somatosensory system: A translational perspective," *Phys. Ther.*, vol. 87, pp. 193–207, Feb. 2007.

[8] R. W. Baloh, J. Enrietto, K. M. Jacobson, and A. Lin, "Age-related changes in vestibular function: A longitudinal study," *Ann. N. Y. Acad. Sci.*, vol. 942, pp. 210–219, Oct. 2001.

[9] I. Lopez, V. Honrubia, and R. W. Baloh, "Aging and the human vestibular nucleus," *J. Vestib. Res.*, vol. 7, pp. 77–85, Jan./Feb. 1997.

[10] J. J. Park, Y. Tang, I. Lopez, and A. Ishiyama, "Age-related change in the number of neurons in the human vestibular ganglion," *J. Comp. Neurol.*, vol. 431, pp. 437–443, Mar. 2001.

[11] O. Delbono, "Neural control of aging skeletal muscle," *Aging Cell*, vol. 2, pp. 21–29, Feb. 2003.

[12] Z. M. Wang, M. L. Messi, and O. Delbono, "L-Type Ca(2+) channel charge movement and intracellular Ca(2+) in skeletal muscle fibers from aging mice," *Biophys. J.*, vol. 78, pp. 1947–1954, Apr. 2000.

[13] B. S. Davidson, M. L. Madigan, and M. A. Nussbaum, "Effects of lumbar extensor fatigue and fatigue rate on postural sway," *Eur. J. Appl. Physiol.*, vol. 93, pp. 183–189, Oct. 2004.

[14] P. A. Gribble and J. Hertel, "Effect of hip and ankle muscle fatigue on unipedal postural control," *J. Electromyogr. Kinesiol.*, vol. 14, pp. 641–646, 2004.

[15] T. M. Lundin, J. W. Feuerbach, and M. D. Grabiner, "Effect of plantar flexor and dorsiflexor fatigue on unilateral postural control," *J. Appl. Biomech.*, vol. 9, pp. 191–201, 1993.

[16] M. A. Nussbaum, "Postural stability is compromised by fatiguing overhead work," *AIHA J*, vol. 64, pp. 56–61, Jan./Feb. 2003.

[17] O. Caron, "Effects of local fatigue of the lower limbs on postural control and postural stability in standing posture," *Neurosci. Lett.*, vol. 340, pp. 83–86, Apr. 2003.

[18] N. Vuillerme, V. Nougier, and J. M. Prieur, "Can vision compensate for a lower limbs muscular fatigue for controlling posture in humans?" *Neurosci. Lett.*, vol. 308, pp. 103–106, Aug. 2001.

[19] N. Vuillerme, F. Danion, N. Forestier, and V. Nougier, "Postural sway under muscle vibration and muscle fatigue in humans," *Neurosci. Lett.*, vol. 333, pp. 131–135, Nov. 2002.

[20] G. Gosselin, H. Rassoulian, and I. Brown, "Effects of neck extensor muscles fatigue on balance," *Clin. Biomech.*, vol. 19, pp. 473–479, Jul. 2004.

[21] M. Schieppati, A. Nardone, and M. Schmid, "Neck muscle fatigue affects postural control in man," *Neuroscience*, vol. 121, pp. 277–285, 2003.

[22] K. M. Pline, M. L. Madigan, M. A. Nussbaum, and R. W. Grange, "Lumbar extensor fatigue and circumferential ankle pressure impair ankle joint motion sense," *Neurosci. Lett.*, vol. 390, pp. 9–14, Dec. 2005.

[23] M. H. Sharpe and T. S. Miles, "Position sense at the elbow after fatiguing contractions," *Exp. Brain Res.*, vol. 94, pp. 179–182, 1993.

[24] S. C. Gandevia, "Spinal and supraspinal factors in human muscle fatigue," *Physiol. Rev.*, vol. 81, pp. 1725–1789, Oct. 2001.

[25] H. van der Kooij, E. van Asseldonk, and F. C. van der Helm, "Comparison of different methods to identify and quantify balance control," *J. Neurosci. Methods*, vol. 145, pp. 175–203, Jun. 2005.

[26] A. Ishida, S. Imai, and Y. Fukuoka, "Analysis of the posture control system under fixed and sway-referenced support conditions," *IEEE Trans. Biomed. Eng.*, vol. 44, no. 5, pp. 331–336, May 1997.

[27] A. D. Kuo, "An optimal state estimation model of sensory integration in human postural balance," *J. Neural Eng.*, vol. 2, pp. S235–S249, Sep. 2005.

[28] C. Maurer and R. J. Peterka, "A new interpretation of spontaneous sway measures based on a simple model of human postural control," *J. Neurophysiol.*, vol. 93, pp. 189–200, Jan. 2005.

[29] R. J. Peterka, "Sensorimotor integration in human postural control," *J. Neurophysiol.*, vol. 88, pp. 1097–1118, Sep. 2002.

[30] X. Qu, M. A. Nussbaum, and M. L. Madigan, "A balance control model of quiet upright stance based on an optimal control strategy," *J. Biomech.*, vol. 40, no. 16, pp. 3590–3597, 2007.

[31] A. V. Alexandrov, A. A. Frolov, F. B. Horak, P. Carlson-Kuhta, and S. Park, "Feedback equilibrium control during human standing," *Biol. Cybern.*, vol. 93, pp. 309–322, Nov. 2005.

[32] S. Jo and S. G. Massaquoi, "A model of cerebellum stabilized and scheduled hybrid long-loop control of upright balance," *Biol. Cybern.*, vol. 91, pp. 188–202, Sep. 2004.

[33] A. D. Kuo, "An optimal control model for analyzing human postural balance," *IEEE Trans. Biomed. Eng.*, vol. 42, no. 1, pp. 87–101, Jan. 1995.

[34] S. Park, F. B. Horak, and A. D. Kuo, "Postural feedback responses scale with biomechanical constraints in human standing," *Exp. Brain Res.*, vol. 154, pp. 417–427, Feb. 2004.

[35] R. J. Peterka, "Simple model of sensory interaction in human postural control," in *Multisensory Control of Posture*, T. Mergner and F. Hlavacka, Eds. New York: Plenum Press, 1995, pp. 281–288.

[36] R. J. Peterka and P. J. Loughlin, "Dynamic regulation of sensorimotor integration in human postural control," *J. Neurophysiol.*, vol. 91, pp. 410–423, Jan. 2004.

[37] R. Johansson, M. Magnusson, and M. Akesson, "Identification of human postural dynamics," *IEEE Trans. Biomed. Eng.*, vol. 35, no. 10, pp. 858–869, Oct. 1988.

[38] M. A. Riley, S. Mitra, T. A. Stroffregan, and M. T. Turvey, "Influences of body lean and vision on unperturbed postural sway," *Motor Control*, vol. 1, pp. 229–246, 1997.

[39] P. Rougier, "Influence of visual feedback on successive control mechanisms in upright quiet stance in humans assessed by fractional Brownian motion modelling," *Neurosci. Lett.*, vol. 266, pp. 157–160, May 1999.

[40] K. Gu, V. L. Kharitonov, and J. Chen, *Stability of Time-Delay Systems*. Boston: Birkhäuser, 2003.

[41] M. L. Madigan, B. S. Davidson, and M. A. Nussbaum, "Postural sway and joint kinematics during quiet standing are affected by lumbar extensor fatigue," *Hum. Mov. Sci.*, vol. 25, pp. 788–789, Dec. 2006.

[42] S. C. Chapra and R. P. Canale, *Numerical Methods for Engineers*, 4th ed. New York: McGraw-Hill, 2001.

[43] P. de Leva, "Adjustments to Zatsiorsky-Seluyanov's segment inertia parameters," *J. Biomech.*, vol. 29, pp. 1223–1230, Sep. 1996.

[44] M. J. Pavol, T. M. Owings, and M. D. Grabiner, "Body segment inertial parameter estimation for the general population of older adults," *J. Biomech.*, vol. 35, pp. 707–712, May 2002.

[45] A. L. Hof, "In vivo measurement of the series elasticity release curve of human triceps surae muscle," *J. Biomech.*, vol. 31, pp. 793–800, Sep. 1998.

[46] I. D. Loram, S. M. Kelly, and M. Lakie, "Human balancing of an inverted pendulum: Is sway size controlled by ankle impedance?" *J. Physiol.*, vol. 532, pp. 879–891, 2001.

[47] J. C. Lagarias, J. A. Reeds, M. A. Wright, and P. E. Wright, "Convergence properties of the Nelder-Mead simplex method in low dimensions," *SIAM J. Optim.*, vol. 9, pp. 112–147, 1998.

[48] O. Nelles, *Nonlinear System Identification*. Berlin: Springer, 2005.

[49] B. S. Davidson, M. L. Madigan, M. A. Nussbaum, and L. A. Wojcik, "Effects of localized muscle fatigue on recovery from a postural perturbation without stepping," *Gait Posture*, vol. 29, pp. 552–557, Jun. 2009.

[50] P. Cordo, L. Carlton, L. Bevan, M. Carlton, and G. K. Kerr, "Proprioceptive coordination of movement sequences: role of velocity and position information," *J. Neurophysiol.*, vol. 71, pp. 1848–1861, May 1994.

[51] R. A. Mackenzie and L. H. Phillips, II, "Changes in peripheral and central nerve conduction with aging," *Clin. Exp. Neurol.*, vol. 18, pp. 109–116, 1981.

[52] N. A. Shaw, "Age-dependent changes in central somatosensory conduction time," *Clin. Electroencephalogr.*, vol. 23, pp. 105–110, Apr. 1992.

[53] H. C. Tong, R. A. Werner, and A. Franzblau, "Effect of aging on sensory nerve conduction study parameters," *Muscle Nerve*, vol. 29, pp. 716–720, May 2004.

[54] K. Masani, M. R. Popovic, K. Nakazawa, M. Kouzaki, and D. Nozaki, "Importance of body sway velocity information in controlling ankle extensor activities during quiet stance," *J. Neurophysiol.*, vol. 90, pp. 3774–3782, Dec. 2003.

[55] K. Masani, A. H. Vette, and M. R. Popovic, "Controlling balance during quiet standing: proportional and derivative controller generates preceding motor command to body sway position observed in experiments," *Gait Posture*, vol. 23, pp. 164–172, Feb. 2006.

[56] J. Jeka, T. Kiemel, R. Creath, F. Horak, and R. Peterka, "Controlling human upright posture: Velocity information is more accurate than position or acceleration," *J. Neurophysiol.*, vol. 92, pp. 2368–2379, Oct. 2004.

[57] H. B. Menz, S. R. Lord, and R. C. Fitzpatrick, "Age-related differences in walking stability," *Age Ageing*, vol. 32, pp. 137–142, Mar. 2003.

[58] T. Herman, N. Giladi, T. Gurevich, and J. M. Hausdorff, "Gait instability and fractal dynamics of older adults with a 'cautious' gait: Why do certain

older adults walk fearfully?” *Gait Posture*, vol. 21, pp. 178–185, Feb. 2005.

[59] F. B. Horak and L. M. Nashner, “Central programming of postural movements: Adaptation to altered support-surface configurations,” *J. Neurophysiol.*, vol. 55, pp. 1369–1381, Jul. 1986.

[60] L. M. Nashner and M. Woollacott, “The organization of rapid postural adjustments of standing humans: An experimental-conceptual model,” in *Posture and Movement*, R. E. Talbott and D. R. Humphrey, Eds. New York: Raven Press, 1979, pp. 243–257.

[61] E. L. Wilson, M. L. Madigan, B. S. Davidson, and M. A. Nussbaum, “Postural strategy changes with fatigue of the lumbar extensor muscles,” *Gait Posture*, vol. 23, pp. 348–354, Apr. 2006.

[62] D. T. Westwick and R. E. Kearney, *Identification of Nonlinear Physiological Systems*. Piscataway, NJ: IEEE Press, 2003.

[63] L. M. Nashner and G. McCollum, “The organization of human postural movements: A formal basis and experimental synthesis,” *Behav. Brain Sci.*, vol. 8, pp. 135–172, 1985.

[64] B. G. Bardy, “Postural coordination dynamics in standing humans,” in *Coordination Dynamics: Issues and Trends*, V. K. Jirsa and J. A. S. Kelso, Eds. New York: Springer Verlag, 2002, pp. 103–121.

[65] R. Creath, T. Kiemel, F. Horak, R. Peterka, and J. Jeka, “A unified view of quiet and perturbed stance: Simultaneous co-existing excitable modes,” *Neurosci. Lett.*, vol. 377, pp. 75–80, Mar. 29, 2005.

[66] O. Oullier, L. Marin, T. A. Stoffregan, R. J. Bootsma, and B. G. Bardy, “Variability in postural coordination dynamics,” in *Movment System Variability*, K. Davids, S. Bennett, and K. M. Newell, Eds. Champaign, IL: HUman Kinetics, 2006, pp. 25–47.

[67] P. L. Weiss, I. W. Hunter, and R. E. Kearney, “Human ankle joint stiffness over the full range of muscle activation levels,” *J. Biomech.*, vol. 21, pp. 539–544, 1988.

[68] E. Edstrom, M. Altun, E. Bergman, H. Johnson, S. Kullberg, V. Ramirez-Leon, and B. Ulfhake, “Factors contributing to neuromuscular impairment and sarcopenia during aging,” *Physiol. Behav.*, vol. 92, pp. 129–35, Sep. 10, 2007.

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