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A Feedback Model Reproduces Muscle Activity During Human Postural Responses to Support-Surface Translations

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Welch TD, Ting LH. A feedback model reproduces muscle activity during human postural responses to support-surface translations. *J Neurophysiol* 99: 1032–1038, 2008. First published December 19, 2007; doi:10.1152/jn.01110.2007. Although feedback models have been used to simulate body motions in human postural control, it is not known whether muscle activation patterns generated by the nervous system during postural responses can also be explained by a feedback control process. We investigated whether a simple feedback law could explain temporal patterns of muscle activation in response to support-surface translations in human subjects. Previously, we used a single-link inverted-pendulum model with a delayed feedback controller to reproduce temporal patterns of muscle activity during postural responses in cats. We scaled this model to human dimensions and determined whether it could reproduce human muscle activity during forward and backward support-surface perturbations. Through optimization, we found three feedback gains (on pendulum acceleration, velocity, and displacement) and a common time delay that allowed the model to best match measured electromyographic (EMG) signals. For each muscle and each subject, the entire time courses of EMG signals during postural responses were well reconstructed in muscles throughout the lower body and resembled the solution derived from an optimal control model. In ankle muscles, >75% of the EMG variability was accounted for by model reconstructions. Surprisingly, >67% of the EMG variability was also accounted for in knee, hip, and pelvis muscles, even though motion at these joints was minimal. Although not explicitly required by our optimization, pendulum kinematics were well matched to subject center-of-mass (CoM) kinematics. Together, these results suggest that a common set of feedback signals related to task-level control of CoM motion is used in the temporal formation of muscle activity during postural control.

INTRODUCTION

We recently demonstrated that the entire time course of muscle activity following postural perturbations to standing balance in cats could be reproduced using a simple feedback model of postural control (Lockhart and Ting 2007). A single-link inverted-pendulum model with a delayed feedback controller reproduced the characteristic temporal patterns of muscle activation throughout the cat hindlimb. Temporal patterns of muscle activation were generated through a combination of center-of-mass (CoM) acceleration, velocity, and displacement waveforms. These results suggest that a common set of variables related to the task goal of controlling body CoM motion are used to coordinate the activation of proximal and distal muscles during balance control. The goal of this study was to determine whether the same sensorimotor transformation could also be used to describe the temporal patterns of muscle activation observed in human postural responses.

Typically, feedback models of human postural control have reproduced joint torques and segmental motions of the body, but not muscle activity. Using single- or multilink inverted-pendulum models, they demonstrate that a set of time-invariant feedback gains can explain joint kinematics during either quiet standing or postural responses to perturbations (Alexandrov et al. 2001a; Bortolami et al. 2003; Kiemel et al. 2002; Kuo 1995; Park et al. 2004; Peterka 2000; Runge et al. 1995; van der Kooij et al. 1999). Because feedback loops at each joint are used to generate stabilizing joint torques, these models cannot uniquely specify temporal patterns of muscle activation. Muscles must be explicitly included because the low-pass dynamics of the body introduce redundancy in the temporal domain, whereby different temporal patterns of muscle activation can produce similar kinematic outputs (Gottlieb et al. 1995; Lockhart and Ting 2007).

Evidence suggests that muscle activity during human postural responses is dependent on acceleration, velocity, and displacement signals, as previously demonstrated in cats. In response to support-surface translations, temporal patterns of muscle activity in humans and cats have a similar rapid initial rise followed by a longer, sustained plateau region (Macpherson et al. 1989). In cats, this waveform is due to CoM acceleration, velocity, and displacement feedback (Lockhart and Ting 2007). Consistent with this feedback model, muscle activity in human postural responses have been shown to be modified by perturbation velocity and total excursion (Diener et al. 1988), smoothness of the initial perturbation trajectory or acceleration (Brown et al. 2001; Siegmund et al. 2002; Szturm and Fallang 1998), and the deceleration impulse at the end of the perturbation (Bothner and Jensen 2001; Carpenter et al. 2005; McIlroy and Maki 1994).

We hypothesized that the activity of multiple muscles during human postural responses to perturbation is generated by a common delayed feedback law based on CoM motion. As a first step, we scaled the single inverted-pendulum feedback model used in Lockhart and Ting (2007) to human dimensions (similar to Peterka 2000) and examined whether this model was capable of reconstructing temporal patterns of muscle activation in proximal and distal muscles. We examined forward and backward support-surface perturbations to standing balance that elicited "ankle strategy" responses (Horak and Nashner 1986). We demonstrate that a delayed feedback law on CoM acceleration, velocity, and displacement can reconstruct temporal patterns

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of both muscle activity and CoM kinematics during postural responses to support surface translations.

METHODS

Seven healthy subjects (five male, two female) from the Georgia Institute of Technology student population, ages 19.4 ± 1.4 yr (mean ± SD), participated in the study. The experimental protocol was approved by both the Georgia Institute of Technology and Emory University Internal Review Boards. Subjects stood on two force plates installed on a movable platform that translated in the horizontal plane. Subjects were instructed to cross their arms at chest-level, look straight ahead, and react naturally to the support-surface perturbations. A set of 20 acclimatization perturbations were followed by a set of 170 randomized forward and backward perturbations of varying peak velocity and acceleration. To test the feasibility of our model in this study, we analyzed responses to forward and backward perturbations of 12-cm total excursion, 25-cm/s peak velocity, and 0.3-g peak acceleration. For each subject, five trials from each direction were collected and averaged. A minimum of 5-min seated rest was enforced between each set of 60 perturbations to reduce muscle fatigue.

Platform acceleration and position, and surface EMG from 11 muscles in the legs and trunk were collected at 1,080 Hz, synchronized with body-segment kinematics collected at 120 Hz (Fig. 1*A*). Platform signals were low-pass filtered at 30 Hz (third-order zero-lag Butterworth filter). EMGs were collected from the following muscles on the right side of the body: TA, tibialis anterior; MG, medial gastrocnemius; SOL, soleus; VLAT, vastus lateralis; RFEM, rectus femoris; SEMB, semimembranosus; SEMT, semitendinosus; BFLH, long head of biceps femoris; BFSH, short head of biceps femoris; ES, erector spinae; and RA, rectus abdominis. Raw EMG signals were high-pass filtered at 35 Hz (third-order zero-lag Butterworth filter), demeaned, half-wave rectified, and low-pass filtered at 40 Hz (first-order zero-lag Butterworth filter). EMG signals were then normalized to the maximum EMG observed in each muscle over all conditions for each subject. Body-segment kinematics were derived from a custom bilateral Helen–Hay 25-marker set that included head–arms–trunk (HAT), thigh, and shank-foot segments. Center-of-mass motion was calculated from kinematic data as a weighted sum of segmental masses (Winter 2005).

Reconstruction of EMG using a feedback control model

We determined whether our feedback model could reproduce the time course of EMG signals in each subject. The model consisted of a single-link inverted pendulum, with a point mass m (equivalent to each subject's mass) and length h (equal to the height of each subject's CoM during quiet standing) (Fig. 1*B*). Disturbance torques calculated from experimentally recorded platform accelerations were applied at the ankle to model the effect of support-surface perturbations (Lockhart and Ting 2007; Peterka 2000). Delayed feedback of horizontal CoM trajectories [displacement, $p(t)$; velocity, $v(t)$; and acceleration, $a(t)$] were used to stabilize the inverted pendulum (Fig. 1*B*). EMG reconstructions (EMG_p) were taken as the output of the feedback controller, which was a linear combination of the weighted horizontal CoM kinematic trajectories at a common neural transmission delay (λ)

$$EMG_p = k_p p(t-\lambda) + k_v v(t-\lambda) + k_a a(t-\lambda) \quad (1)$$

Each EMG reconstruction was half-wave rectified and converted to a muscle torque using a first-order muscle model with a 40-ms time constant (He et al. 1991; Lockhart and Ting 2007).

For each muscle in each subject, the feedback gains (k_p, k_v, k_a) and delay (λ) that best matched the EMG reconstruction to the measured EMG signal were found. We used an optimization (MATLAB, *fmincon.m*) to find the values of k_i and λ using the following cost function

$$\min_{K\epsilon G} \left\langle J = E\left\{ \int_0^{t_{end}} [e_m^T \mu_s e_m + \max(\mu_m |e_m|)] dt \right\} + W\dot{e}_x(t_{end}) \right\rangle \quad (2)$$

The first term penalized the error between the reconstructed and measured EMG signal over time as represented by the vector e_m with

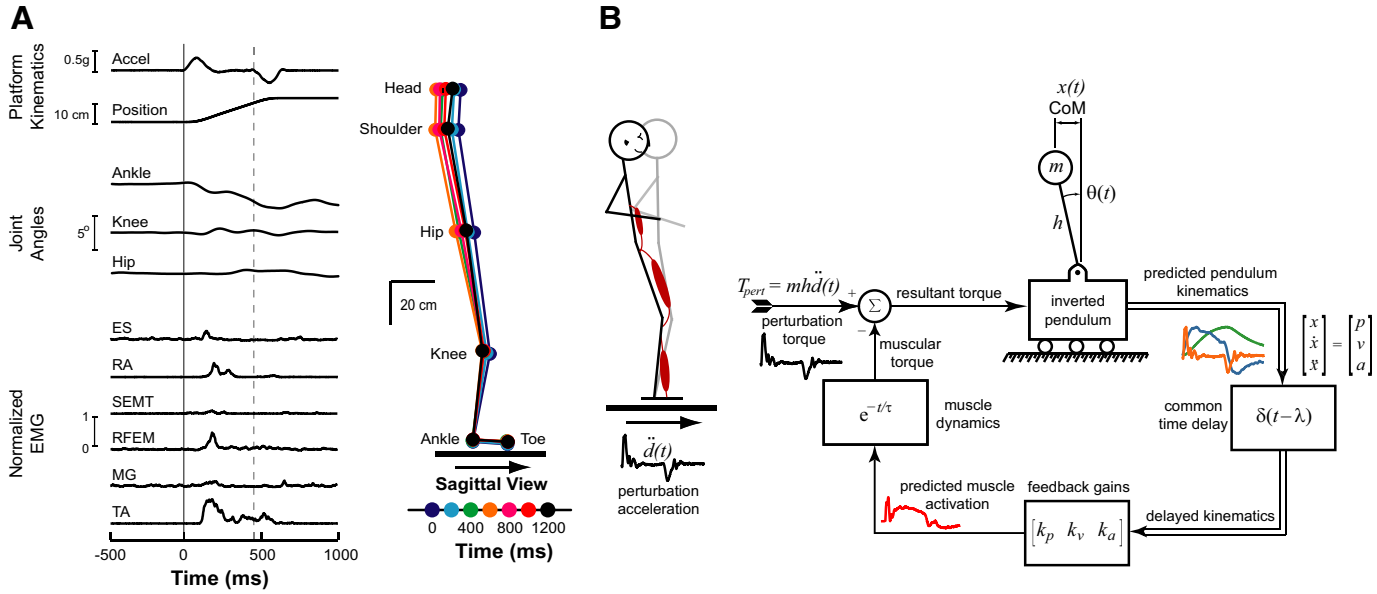

FIG. 1. Example postural response, modeled as an inverted pendulum under delayed feedback control. *A*: in response to a forward support-surface perturbation, the primary joint motion occurred at the ankle joint. Muscles throughout the body were activated in a coordinated fashion to counteract the disturbance (*left*). Vertical lines indicate onset of perturbation acceleration (solid) and deceleration (dashed). The original postural configuration was typically restored within one second of perturbation onset (*right*). *B*: the standing human was modeled as an inverted pendulum that was perturbed with a torque based on recorded platform acceleration [$\ddot{d}(t)$]. To generate the reconstructed electromyographic (EMG) activity, pendulum displacement, velocity, and acceleration (p, v, a) were subject to a common time delay (λ) and feedback gains on each channel (k_p, k_v, k_a). The reconstructed EMG signal was then passed through a first-order muscle model to generate the muscle torque to counteract the perturbation.

weight μ_s. The second term penalized the maximum deviation between the reconstructed and measured EMG signals at any single point in time with weight μ_m. The final term penalized the final state of the inverted pendulum if it differed from that of the experimental subject (i.e., upright configuration) with weight W. Note that this differs from the formulation of Lockhart and Ting (2007) in that only the EMG pattern, and not the CoM kinematics, was matched. Feedback gains were restricted to have values between 0 and 100, and the delay was restricted to values between 60 and 250 ms. We assessed the goodness of fit between reconstructed and measured EMG signals using both the coefficient of determination (r^2) and the uncentered Pearson's coefficient of determination [variability accounted for (VAF)].

Recorded and reconstructed EMG patterns were compared with those predicted by an optimal control model (Lockhart and Ting 2007). Using a controller design similar to that of the linear quadratic regulator (He et al. 1991), this delayed quadratic regulator (DQR) model determined gains for CoM kinematic feedback channels, without a priori knowledge of recorded EMG, through the use of a quadratic cost function and time-delayed feedback. Feedback gains on delayed CoM kinematics (k_i) were optimized using the following cost function

$$\min_{K \epsilon G} \left\{ J = E\left[\int_0^{t_{end}} (x^T Q x + \rho u) \mathrm{d}t + \Omega x(t_{end}) \right] \right\} \quad (3)$$

The first term penalized deviations from zero of the pendulum position, velocity, and acceleration (where $x = [p\ v\ a]^T$) with weights $Q = [0.05\ 50\ 1]$. The second term penalized EMG activation level (u) with weight $\rho = 20$, requiring the minimum possible level of muscle activation to achieve the postural task. The final term penalized final pendulum configurations that were not upright with weight Ω. Because the optimization process consistently selected the minimum allowable feedback delay, this delay was set to 100 ms for all subjects to allow the calculation of an intersubject average of the optimal postural control solution and to facilitate qualitative comparisons with recorded and reconstructed EMG patterns.

RESULTS

Temporal patterns of muscle activity throughout the leg in both forward and backward perturbations were reconstructed by our feedback model in all subjects. Reconstructed EMG activity in ankle muscles TA and MG were well matched to measured EMG activity in forward and backward perturbations, respectively (VAF $>$ 0.75; Fig. 2, *A* and *B*). Notable variations in the temporal patterns of muscle activity were observed across subjects; these variations were accounted for by differences in feedback gains (Fig. 3*D*). Still, ankle muscle activity in all subjects resembled the optimal control solution, although an exact match was not achieved by any subject (cf. Fig. 2, *A* and *B*, DQR prediction; TA: $r^2 = 0.53 \pm 0.16$, VAF $= 0.73 \pm 0.09$; MG: $r^2 = 0.45 \pm 0.13$, VAF $= 0.68 \pm 0.08$). Although the ankle-strategy responses evoked produced little knee or hip motion (Fig. 1*A*), muscle activity in biomechanically relevant proximal muscles was also well described by the feedback model (VAF $>$ 0.67 across all muscles and subjects; Fig. 2*C*). The time course of experimentally recorded CoM kinematic trajectories was similar to the motion of the inverted-pendulum model controlled by the reconstructed EMG pattern (Fig. 3*C*). This was surprising because we explicitly required only the temporal EMG patterns, and not kinematics, to match the experimentally recorded data, suggesting that the kinematics of the body are indeed encoded in the pattern of muscle activation used by the nervous system for postural control.

A decomposition of the reconstructed EMG into contributions from each feedback component demonstrates that the initial burst region was predominated by acceleration feedback, whereas velocity and displacement feedback contribute to the plateau region of muscle activity (Fig. 3*B*). Acceleration feedback from the deceleration of the platform also contributed to the termination of muscle activity (solid gray, Fig. 3*B*). The mechanical dynamics of the pendulum defined the temporal separation of the various feedback contributions; addition of independent delays for each channel had no significant effect on the model reconstructions [TA: $\Delta r^2 = 0.00$ ($P = 0.80$), ΔVAF $= 0.01$ ($P = 0.31$); MG: $\Delta r^2 = 0.05$ ($P = 0.11$), ΔVAF $= 0.02$ ($P = 0.10$)].

Acceleration feedback was required to reconstruct EMG activity using physiological delays. When acceleration feedback was removed, delays shorter than the 55-ms latency of the stretch response during postural perturbations (Diener et al. 1984) were required (intersubject range = 10–60 ms; Fig. 3*D*). Without acceleration feedback, the early EMG activity in the initial burst and plateau regions, including the initial slope of the response, were underpredicted (data not shown). Further, the goodness of fit between reconstructed and recorded EMGs was reduced in TA [$\Delta r^2 = -0.14$ ($P = 7 \times 10^{-4}$); ΔVAF $= -0.07$ ($P = 0.006$)], but not MG [$\Delta r^2 = -0.05$ ($P = 0.42$); ΔVAF $= -0.03$ ($P = 0.13$)]. In both cases, however, the reconstructed EMGs without acceleration feedback were often insufficient to maintain the pendulum in an upright configuration (data not shown).

DISCUSSION

Our results demonstrate that the neural mechanisms generating temporal patterns of muscle activity for postural control in humans can be described by a feedback transformation from body kinematics to EMG. For ankle strategy responses, an inverted-pendulum model of human posture reproduced muscle- and subject-specific muscle activation patterns throughout the lower body using delayed feedback of acceleration, velocity, and displacement of the pendulum. The pendulum motion also matched recorded CoM kinematics, although not explicitly required by the optimization. Our simulation therefore provides a mechanistic model that functionally validated the sensorimotor transformation between CoM motion and muscle activity. These results suggest that a common set of feedback signals related to the task-level control of CoM motion are indeed used in the temporal formation of muscle activity during postural control.

The nervous system may take advantage of the naturally occurring physical relationships between acceleration, velocity, and displacement to provide feedback control of the CoM without need for feedforward control mechanisms. Previous studies have observed a positive, phase-leading correlation between muscle activity during quiet stance and CoM motion, suggesting the use of predictive, feedforward control (Fitzpatrick et al. 1992, 1996; Gatev et al. 1999). The phase-lead characteristics of acceleration feedback may serve to explain this observation in the context of feedback control. In our

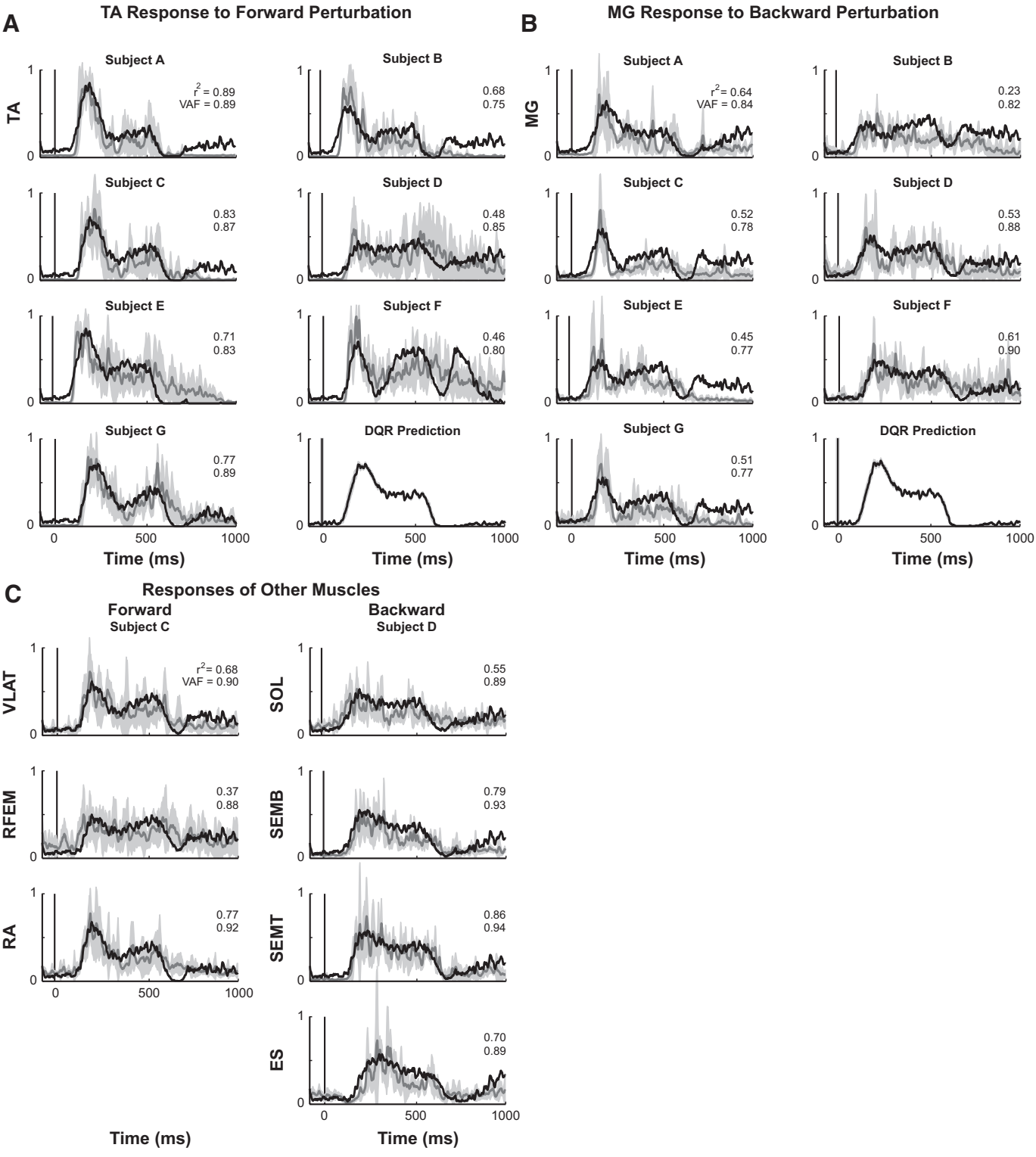

FIG. 2. Averaged time courses of recorded (solid gray) and reconstructed (solid black) EMGs during postural responses. Gray-shaded regions indicate 1SD from the mean recorded EMG for each muscle across five trials. *A*: tibialis anterior (TA) EMG signals in response to forward perturbations across all subjects are presented with the average optimal control solution [delayed quadratic regulator (DQR) prediction]. Significant variations in the temporal patterns of TA EMGs were observed across subjects; however, each response resembled the optimal DQR prediction. The feedback model was able to reproduce these variations with >75% variability accounted for (VAF) by choosing a slightly different set of feedback gains and delay for each subject. *B*: similarly, medial gastrocnemius (MG) EMG signals in response to backward perturbations were reconstructed with >77% VAF across all subjects and resembled the DQR prediction. *C*: additionally, EMG signals from knee, hip, and pelvis muscle that were active during either forward or backward perturbations were also reproduced with >67% VAF across all subjects.

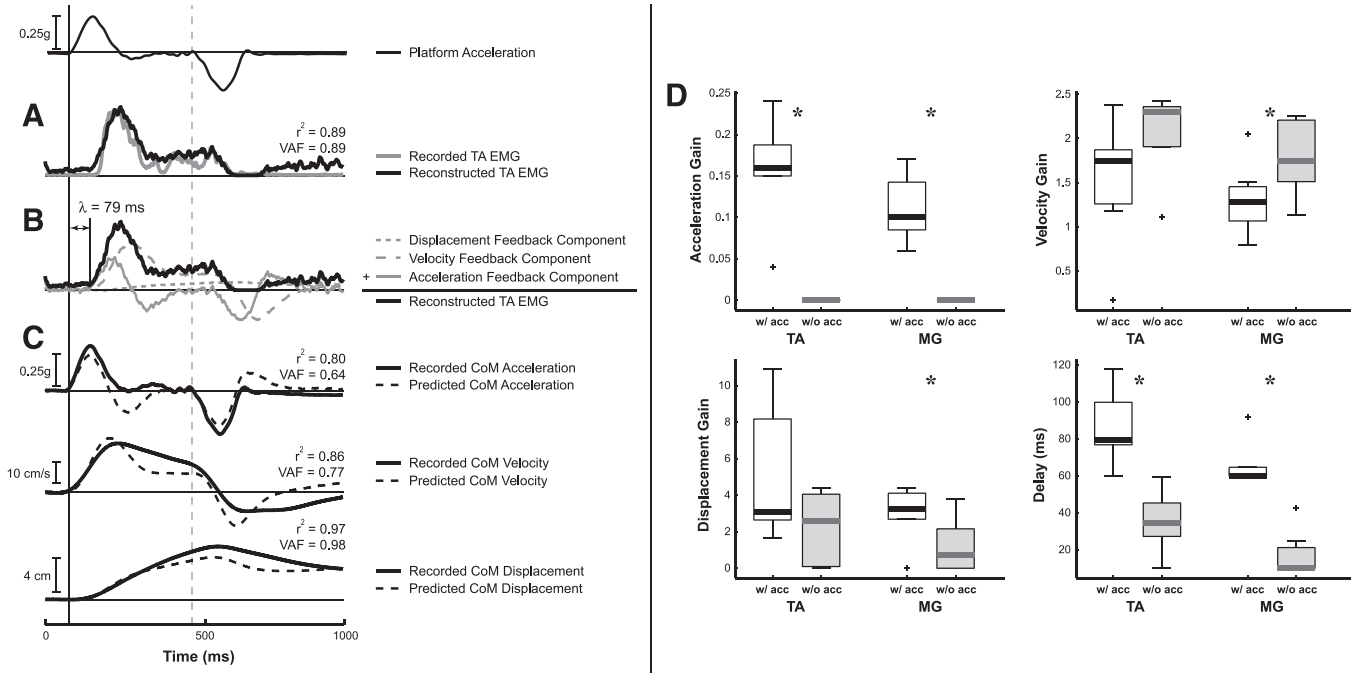

FIG. 3. Contributions of each feedback component to the time course of EMG depend on muscle- and subject-specific feedback gains. *A*: recorded (gray) and reconstructed (black) TA EMG signals for Subject A. Vertical lines indicate onset of perturbation acceleration (solid) and deceleration (dashed). *B*: decomposition of the reconstructed EMG signal (black) into individual feedback components from acceleration feedback (gray line), velocity feedback (gray dashed line), and displacement feedback (gray dotted line). Acceleration feedback contributes to the rapid initial rise in EMG activity. Velocity and displacement feedback contribute to later activity during the plateau region. *C*: recorded (solid line) and predicted (dashed line) center-of-mass (CoM) acceleration, velocity, and displacement trajectories are also similar. This was surprising because our optimization explicitly required only temporal patterns of EMG signals to be matched between the model and experiment. These results suggest that CoM kinematics are indeed used by the nervous system in generating EMG signals. The time course of each feedback component's contribution to the reconstructed EMG is determined by these CoM kinematic trajectories after a delay (λ). The mechanical dynamics of the pendulum thus define the temporal separation of the contributions from each feedback channel, illustrated in *B*. The amplitude of the contributions from each feedback channel depends on the magnitude of the feedback gains, which varies across muscles and subjects. *D*: variations in feedback gains for TA and MG muscles across subjects (white boxes) when acceleration feedback was included. Boxes delimit the middle 50% of the data, with the center indicating the median value (thick black line). Whiskers delimit the full range of the data, excluding outliers (indicated with a +). When acceleration feedback was removed from the model (gray boxes), the remaining model parameters changed (* represents significant difference at $P < 0.05$), resulting in modest or insignificant changes in goodness of fit. However, the range of the delays required to reproduce the EMG signals without acceleration feedback was reduced to durations shorter than the 55-ms short-latency stretch response during postural perturbations, and were therefore not physiological.

model, the contribution of acceleration feedback is fully reflected in the muscular response before significant displacement-related information becomes available. Moreover, the acceleration component of the reconstructed muscular response leads CoM displacement, but occurs after the CoM acceleration induced by the perturbation. The phase lead of acceleration feedback with respect to CoM displacement in our simulations was about 135 ms, consistent with the 100- to 250-ms phase lead observed experimentally for high-frequency postural sway (Fitzpatrick et al. 1992). The early burst of muscle activity during postural responses to perturbation, here shown to arise from acceleration feedback, was previously attributed to a feedforward component (Diener et al. 1988). Consistent with our model, however, the middle portion of the response varies with changes in perturbation velocity, whereas the late response is affected by changes in perturbation displacement (Diener et al. 1988).

Several other studies provide support for acceleration feedback in postural control. Postural responses have been shown to scale with perturbation acceleration in the neck muscles of seated subjects (Siegmund 2004; Siegmund et al. 2002) and in perturbations to arm movements (Soechting and Lacquaniti 1988). In standing posture, muscle onset latency and total ankle moment are also affected by perturbation acceleration (Brown et al. 2001; Siegmund et al. 2002; Szturm and Fallang 1998). Further, the rate of muscle activity onset during perturbations to treadmill walking has also been related to perturbation acceleration (Dietz et al. 1987). Several studies during standing postural responses suggest that the termination of the postural response results from feedback on the deceleration impulse (Bothner and Jensen 2001; Carpenter et al. 2005; McIlroy and Maki 1994). Consistent with this finding, in our model, termination of the postural response can also be attributed to the delayed effects of the deceleration impulse (Fig. 3*A*).

Our study supports the idea that a small set of variables related to task-level goals are used to coordinate multiple muscles throughout the body during postural control and other movements. Activity in muscles crossing the hip, knee, and ankle joints all exhibited temporal patterns that were explained by combinations of the CoM motion as modeled by an inverted pendulum. Although the hip and knee joints did not undergo appreciable joint angle changes (Fig. 1*A*), proximal muscle activity may be necessary to minimize joint motions from interaction torques generated by ankle muscle activity (van Antwerp et al. 2007; Zajac and Gordon 1989). Therefore whenever the ankle muscles are activated, the proximal muscles must also be activated to maintain the postural configuration. We propose that a muscle synergy defining consistent

spatial patterns of multiple muscle activity for ankle-strategy responses (Torres-Oviedo and Ting 2007) may be temporally regulated by feedback signals. The spatiotemporal patterns of muscle activation for postural control could thus be specified by defining a constant set of gains on CoM acceleration, velocity, and displacement for each muscle.

Although we have demonstrated the feasibility of task-level feedback in explaining ankle-strategy responses to support-surface translations, more complex biomechanical models may be necessary to represent the full range of responses—ankle, hip, and mixed strategies—in the postural-control suite (Alexandrov et al. 2001b; Horak and Nashner 1986; Runge et al. 1999). This is especially pertinent for modeling muscular responses to backward translations, as well as to support-surface rotations and upper-body perturbations, where hip-strategy responses produce significant joint motions and muscle activation about the proximal joints (Jo and Massaquoi 2004; Runge et al. 1999). Because the hip-strategy response has a distinct muscle synergy pattern that can be decomposed from a mixed response (Torres-Oviedo and Ting 2007), it is possible that the hip-strategy response is also regulated by a task-level feedback controller that is independent of the ankle-strategy controller.

Comparisons of experimentally recorded EMG with an optimal control solution suggest that the postural responses of our human subjects, although similar to the optimal solution, may not have completely achieved the optimal feedback pattern for responding to support-surface translations during the course of our experiment. In contrast, cats subjected to a similar perturbation protocol exhibited EMG patterns that matched the optimal solution as predicted by the DQR model (Lockhart and Ting 2007). The cats underwent a rigorous training regimen in which they learned to stand on the perturbation platform over the course of several weeks or months (cf. Macpherson et al. 1987). Our human subjects, however, were completely naïve to postural perturbation studies and each completed the experimental protocol in less than 1 h. We hypothesize that, during their training regimen, the cats may have slowly adapted their muscular responses toward the optimal control solution for the task. We therefore predict that, with training, human muscle activity during postural responses may more closely match the optimal feedback pattern predicted by our DQR model. Alternately, it may be possible that each human subject used a different set of optimality criteria, which could be modeled either by varying the weights in the cost function (Qu et al. 2007) or by changing the components of the cost function altogether.

Our feedback model may provide a low-dimensional framework for understanding variability in muscle activation patterns during postural control (Ting 2007). Extensive intersubject variability in temporal patterns of muscle activity may be accounted for by varying only three feedback gains (Fig. 2, *A* and *B*). Rather than performing a point-by-point adjustment of neural activity over time, the CNS may adjust gains to each feedback channel. This differential weighting of feedback channels may explain changes in muscle responses due to habituation and changes in central set (Horak et al. 1989). For example, when the interval between acceleration and deceleration of translation perturbations is short and predictable, subjects anticipate the deceleration timing (Carpenter et al. 2005; McIlroy and Maki 1994). The advance in the timing of response termination might occur due to changes in CoM velocity and displacement feedback gains, which alter the time at which the acceleration feedback triggers the offset of EMG activity.

GRANTS

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