J Neurophysiol 98: 2144–2156, 2007.
First published July 25, 2007; doi:10.1152/jn.01360.2006.

Muscle Synergies Characterizing Human Postural Responses

Gelsy Torres-Oviedo and Lena H. Ting
The Wallace H. Coulter Department of Biomedical Engineering, at Georgia Tech and Emory University, Atlanta, Georgia

Submitted 28 December 2006; accepted in final form 18 July 2007

Torres-Oviedo G, Ting LH. Muscle synergies characterizing human postural responses. *J Neurophysiol* 98: 2144–2156, 2007. First published July 25, 2007; doi:10.1152/jn.01360.2006. Postural control is a natural behavior that requires the spatial and temporal coordination of multiple muscles. Complex muscle activation patterns characterizing postural responses suggest the need for independent muscle control. However, our previous work shows that postural responses in cats can be robustly reproduced by the activation of a few muscle synergies. We now investigate whether a similar neural strategy is used for human postural control. We hypothesized that a few muscle synergies could account for the intertrial variability in automatic postural responses from different perturbation directions, as well as different postural strategies. Postural responses to multidirectional support-surface translations in 16 muscles of the lower back and leg were analyzed in nine healthy subjects. Six or fewer muscle synergies were required to reproduce the postural responses of each subject. The composition and temporal activation of several muscle synergies identified across all subjects were consistent with the previously identified "ankle" and "hip" strategies in human postural responses. Moreover, intertrial variability in muscle activation patterns was successfully reproduced by modulating the activity of the various muscle synergies. This suggests that trial-to-trial variations in the activation of individual muscles are correlated and, moreover, represent variations in the amplitude of descending neural commands that activate individual muscle synergies. Finally, composition and temporal activation of most of the muscle synergies were similar across subjects. These results suggest that muscle synergies represent a general neural strategy underlying muscle coordination in postural tasks.

INTRODUCTION

Several studies have demonstrated that muscle synergies, or M-modes (Krishnamoorthy et al. 2003, 2004, 2007), defined as low-dimensional modules formed by muscles activated in synchrony (Cappellini et al. 2006; Cheung et al. 2005; Ivanenko et al. 2003, 2004, 2005; Ting and Macpherson 2005; Torres-Oviedo et al. 2006) or with fixed time delays (d'Avella et al. 2003, 2006) may be used by the nervous system as building blocks for constructing motor output patterns during both locomotor and postural tasks. In this study we define a *muscle synergy* as a group of muscles activated in synchrony with fixed relative gains; thus a synergy represents a muscle activation pattern with consistent spatial characteristics. We investigated whether a few muscle synergies could reproduce the coordinated spatiotemporal muscle activation patterns observed during human postural responses.

In both humans and cats, muscle activation patterns in response to multidirectional balance perturbations vary as a function of perturbation direction, suggesting independent muscle activation (Allum et al. 2003; Carpenter et al. 1999; Henry et al. 1998, 2001; Macpherson 1988). However, in cats, this directional tuning of responses can be reproduced by the activation of a general set of muscle synergies across a wide range of postural tasks (Ting and Macpherson 2005; Torres-Oviedo et al. 2006). Principles of sensorimotor integration during postural control are quite similar in humans and cats, despite differences in stance configuration and biomechanics (Dunbar et al. 1986; Horak and Macpherson 1996). Therefore we investigated whether human postural responses to multidirectional perturbations could also be explained by the activation of a limited set of muscle synergies.

In contrast to cats, humans can use several postural strategies to maintain balance, resulting in considerable intertrial variations of electromyographic (EMG) responses to identical balance perturbations (Horak and Nashner 1986). Factors that affect the choice of strategy include prior experience, habituation, expectation, and fear (Carpenter et al. 2006; Keshner et al. 1987; Woollacott and Shumway-Cook 2002). The two most extreme human postural responses are the "ankle" and "hip" strategies, in which muscle activation patterns have very different spatiotemporal characteristics (Horak and Macpherson 1996; Horak et al. 1997). These strategies have recently been shown to be independent modes of movement (Alexandrov et al. 1998, 2001a,b, 2005; Creath et al. 2005; Massion et al. 2004), suggesting that they could result from the activation of independent muscle synergies. The "ankle" and "hip" strategies can also be concurrently activated, creating a continuum of possible postural responses representing different mixtures of the two strategies (Creath et al. 2005; Horak and Macpherson 1996). In this study, we tested whether muscle synergy analysis could be used to quantify the variable contribution of each strategy to each given postural response.

These postural strategies also induce considerable variations in muscle onset latency in human postural responses. For example, "ankle strategy" responses are characterized by shorter onset latencies than "hip strategy" responses (Horak and Macpherson 1996). Previous studies have overcome this issue by characterizing average muscle activity during the postural response over large time windows (Henry et al. 1998, 2001) or time windows that vary with individual muscle onset (Carpenter et al. 1999). In the current study, we analyzed multiple time windows during the automatic postural response (APR) to explicitly examine whether the temporal variation in muscle onset latencies could be accounted for by differential temporal activation of muscle synergies.

The spatiotemporal features of EMG patterns representing different postural strategies have been traditionally character-

Address for reprint requests and other correspondence: L. H. Ting, The Wallace H. Coulter Department of Biomedical Engineering, at Georgia Tech and Emory University, 313 Ferst Drive, Atlanta, GA 30322-0535 (E-mail: lting@emory.edu).

The costs of publication of this article were defrayed in part by the payment of page charges. The article must therefore be hereby marked "*advertisement*" in accordance with 18 U.S.C. Section 1734 solely to indicate this fact.

ized by analyzing the average across multiple trials that exhibit consistent postural responses (Carpenter et al. 1999; Gruneberg et al. 2005; Henry et al. 1998; Horak and Macpherson 1996; Horak and Nashner 1986; Nashner 1977). This approach, however, has the disadvantage of relying on the repeatability of postural responses. In such studies, intertrial variations must be minimized to avoid confounded averaged responses. This might present a problem especially in clinical research where fewer trials and less-consistent postural responses might be collected, inducing large intertrial variations in EMG patterns. Recent factorization algorithms have been able to identify consistent muscle activation patterns in nonaveraged trials during frog scratching, swimming, and jumping (Cheung et al. 2005; Tresch et al. 1999). We investigated whether a similar factorization analysis could enable us to identify spatiotemporal characteristics of human postural strategies in a data set containing high intertrial variability. This would provide a powerful diagnostic tool because the analysis would not require repeatability in postural responses to characterize muscle coordination of the motor behavior in question.

We demonstrated that a small set of muscle synergies can robustly account for a wide range of muscle activation patterns during human postural responses when subjects stood in a "normal" stance configuration. We were able to reproduce directional tuning of EMG patterns, temporal differences in muscle onset latencies, and intertrial variations in postural strategies using a few muscle synergies. Muscle synergy analysis effectively decomposed "mixed" responses in individual trials into contributions from different postural strategies, demonstrating that consistency in postural responses is not required to identify robust muscle synergies. We further demonstrated similarities in muscle synergy patterns across subjects. Taken together, our findings suggest that the identified muscle synergies represent modules of motor output that can be recruited in variable proportions during postural responses.

METHODS

Experimental setup

Nine healthy subjects, four females and five males (ages 19–27 yr), were tested following an experimental protocol approved by the Georgia Tech and Emory University Institutional Review Boards. Subjects stood on a platform that was made to translate in a set of 12 directions evenly distributed in the horizontal plane (Fig. 1*B*). Ramp-and-hold perturbations [total displacement: 12.4 cm; peak velocity: 35 cm/s; peak accelerations 490 cm/s^2 (0.5 g)] were presented.

Because we were interested in examining the richest possible data set (see DISCUSSION), we used an experimental paradigm that has been shown to elicit a wide range of postural strategies (Horak 1996; Horak and Nashner 1986). In this paradigm, the "normal stance" (medial–lateral stance width of 19 cm) trials were randomly presented among trials of different stance configurations. Over the course of experimental sessions held on two consecutive days, subjects received ten replicates of each perturbation direction in normal stance, which were interspersed with perturbations in other stance configurations. The electrode positions on the subject's body were marked to ensure similar electrode placement in both experimental sessions. We used the randomization of the stance conditions to induce a more variable set of postural responses in the normal stance condition. In the present study variability in postural responses for the normal stance condition were analyzed; the variability in postural responses for the other stance conditions is beyond the scope of the present study.

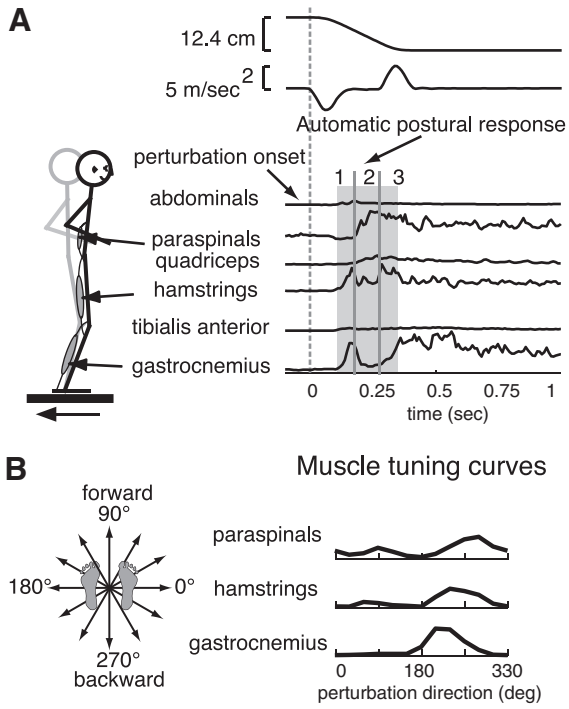

FIG. 1. Example of postural responses to backward perturbation of the support surface. *A*: balance perturbations were induced by a ramp-and-hold motion of the support surface. Electromyographic (EMG) responses are directionally specific and typically occur with a 100-ms onset latency after platform motion (vertical dashed line). Mean EMG activity in 3 time bins of 75 ms (EMG_{APR1}, EMG_{APR2}, EMG_{APR3}) during the automatic postural response (APR) period were computed for each perturbation (shaded areas). *B*: coordinate system for support surface translations in 12 evenly spaced directions in the horizontal plane. Muscle tuning curves represent the magnitude of the APR response in a single time bin over all perturbation directions.

EMG activity was recorded from 16 leg and lower-back muscles of the subject's right side including: rectus abdominalis (REAB), tensor fascia lata (TFL), biceps femoris long head (BFLH), tibialis anterior (TA), semitendinosus (SEMT), semimembranosus (SEMB), rectus femoris (RFEM), peroneus (PERO), medial gastrocnemius (MGAS), lateral gastrocnemius (LGAS), erector spinae (ERSP), external oblique (EXOB), gluteus medius (GMED), vastus lateralis (VLAT), vastus medialis (VMED), and soleus (SOL). Raw EMG data were filtered and processed off-line using a set of custom MATLAB routines. Raw EMG data were high-pass filtered at 35 Hz, de-meaned, rectified, and low-pass filtered at 40 Hz.

Data processing

To account for temporal variations in EMG activity, four time periods ("bins") were analyzed: quiet standing during a 280-ms background period (BK) that ended 170 ms before the perturbation, and three 75-ms time bins beginning 100 ms (APR1), 175 ms (APR2), and 250 ms (APR3) after perturbation onset (Fig. 1).

These time bins were chosen based on previous studies characterizing the different temporal features of muscle activity during the time course of the postural response (Diener et al. 1988). Mean muscle activity during these four time bins was computed for each of the 16 muscles in each trial. From these, we generated a vector of data for each of the 16 muscles that included 4 time bins $\times$ 12 directions $\times$ 10 trials = 480 data points. EMGs were normalized to their respective maximum response amplitude during background and APR period across all perturbation directions so that all values of each muscle were between 0 and 1. Then, each muscle data vector, which consisted of EMG_{BK}, EMG_{APR1}, EMG_{APR2}, and EMG_{APR3} across all pertur-

bation directions, was normalized to have unit variance to ensure the activity in all muscles was equally weighted.

Extraction of muscle synergies

Using nonnegative matrix factorization (Cheung et al. 2005; Lee and Seung 2001; Torres-Oviedo et al. 2006; Tresch et al. 2006), we extracted muscle synergies from the EMG data matrix. This linear decomposition technique assumes that each muscle activation pattern **M** (e.g., Fig. 6*C*, EMG_{APR2} and EMG_{APR3}), evoked by a perturbation at a given time period, is composed of a linear combination of a few (N_{syn}) muscle synergies $\mathbf{W_i}$, each activated by synergy activation coefficient c_i. Thus the net muscle activation pattern vector **M** takes the form

$$\mathbf{M} = c_i\mathbf{W}_i + c_2\mathbf{W}_2 + \cdots + c_n\mathbf{W}_n$$

$\mathbf{W_i}$ is a vector that specifies the spatial pattern of muscle activity defined by muscle synergy i. Each element of $\mathbf{W_i}$ represents a muscle whose relative contribution to the muscle synergy takes a value between 0 and 1. These values forming a muscle synergy are constant over all trials and the entire muscle synergy is modulated by a single, scalar, nonnegative, activation coefficient c_i. The activation coefficient c_i represents the purported neural command to the muscle synergy that determines the relative contribution of the muscle synergy $\mathbf{W_i}$ to the overall muscle activation pattern **M**. For each synergy i, the set of activations c_i across all perturbation directions during quiet stance and during the three APR periods is the vector $\mathbf{C}_i$. The $\mathbf{C}_i$ components during the three APR periods represent the tuning curves that describe how the activation of the muscle synergy $\mathbf{W}_i$ changes as a function of perturbation direction and time. Because of the nonnegative assumption of the algorithm, each muscle synergy characterizes additive features of the analyzed data (Tresch et al. 2006). However, this limits our ability to identify correlated inhibition patterns. Although relative inhibition of an entire muscle synergy can be identified, as manifested by a decrease in the activation of a muscle synergy, inhibition patterns different from the excitatory patterns expressed in the muscle synergies cannot be identified.

In all our subjects we iterated the analysis by varying N_{syn} between 1 and 10 and then selected the least number of synergies that could adequately reconstruct background and APR responses of each muscle in all the trials, as determined by >75% variability accounted for (VAF) in each muscle data vector. VAF is defined as 100 × the coefficient of determination from the uncentered Pearson correlation coefficient (Torres-Oviedo et al. 2006; Zar 1999). This criterion ensured that each muscle tuning curve would be well reconstructed, so that the critical spatiotemporal features of each muscle activation pattern were well accounted for by the muscle synergies. In general, by satisfying this local criterion, the total VAF in the data set was well over 90%.

For cross-validation purposes we also extracted N_{syn} muscle synergies from 60% of the trials at each perturbation direction (training trials) and used them to reconstruct the muscle responses in the remaining trials (testing trials). For each subject, we also extracted a set of N_{syn} muscle synergies from the averaged response for each direction as in previous studies (Ting and Macpherson 2005; Torres-Oviedo et al. 2006) to determine whether the predictive power of muscle synergies increased when intertrial variability was considered.

Muscle synergies extracted from all trials of each subject were ordered based on muscle composition and synergy activation profiles rather than on percentage of contribution to the total data variability (as in other factorization methods such as principal component analysis). We performed a *functional sorting* because subjects might use muscle synergies differently, causing comparable muscle synergies to have large differences in contribution to the total data variability.

To perform the functional sorting we computed averaged synergy activation coefficients across all trials ($\bar{C}$) for each muscle synergy (W) of each subject. Then an initial sorting was performed by grouping muscle synergies based on the similarity of W and/or $\bar{C}$ values ($r^2 > 0.55$) to that of an arbitrary reference subject. From this initial sorting, an averaged set of W and $\bar{C}$ values across subjects was computed. Then, using an iterative process, only muscle synergies that were similar to either averaged W or $\bar{C}$ values, or both ($r^2 > 0.55$), were kept in the group. The averaged set of W and $\bar{C}$ vectors across subjects were updated every time a muscle synergy was discriminated from a group. The r^2 values obtained served not only as a sorting parameter, but also as a measure to evaluate the generality of muscle synergies across subjects. Therefore within each group, the r^2 values were used to identify similarities across subjects in both W and $\bar{C}$, or only W, or only $\bar{C}$.

Subsequent to the functional sorting, we performed a two-way ANOVA analysis with a Tukey–Kramer post hoc test on the peak of the averaged activations of each muscle synergy from all time bins in all subjects to determine the time bin in which mean peak activation of each muscle synergy occurred.

RESULTS

For each subject, a few muscle synergies were found to reproduce spatiotemporal muscle activation patterns recorded during quiet stance and during responses to multidirectional balance perturbations when subject stood in a "normal" postural configuration. Trial-to-trial variations in muscle activations were accounted for by trial-to-trial variations in synergy activation coefficients that represent neural commands to the various muscle synergies. Finally, all subjects exhibited muscle synergies that were similar in terms of muscle composition and spatiotemporal activation pattern.

A few muscle synergies can reproduce EMG patterns that vary with time and perturbation direction

Differences in postural responses elicited across all muscles were observed in the spatial, temporal, and intertrial variability of the data. Each muscle was activated in response to a range of perturbation directions. This directional sensitivity is represented by muscle tuning curves (Fig. 2, *gray traces*), which were unique to each muscle. However, some similarities in muscle tuning curves were observed. Additionally, the onset latencies in the EMG responses varied across muscles. For example, all proximal muscles except for erector spinae, external oblique, and gluteus medius were inactive during APR1 and were highly activated during later time bins (Fig. 2). In addition, the directional tuning of the responses of more proximal muscles, such as vastus lateralis, tensor fascia latae, and rectus abdominalis, changed during the three time bins of the APR (Fig. 2). Finally, intertrial variations of muscle activations were also observed in muscle responses to each perturbation direction (Fig. 2, black dots). These characteristics of postural responses were consistent with results from previous studies using similar paradigms (Carpenter et al. 1999; Gruneberg et al. 2005; Henry et al. 1998; Horak and Nashner 1986)

Six or fewer muscle synergies accounted for 92 ± 2% of the total data variability in the nine subjects. The mean total VAF in the training data sets was 92 ± 2% and in the test data set was 90 ± 2%. Background and APR responses of every muscle in all trials were well reconstructed, as determined by VAF > 75% in all muscles. In all subjects, the number of muscle synergies that could reproduce the postural responses

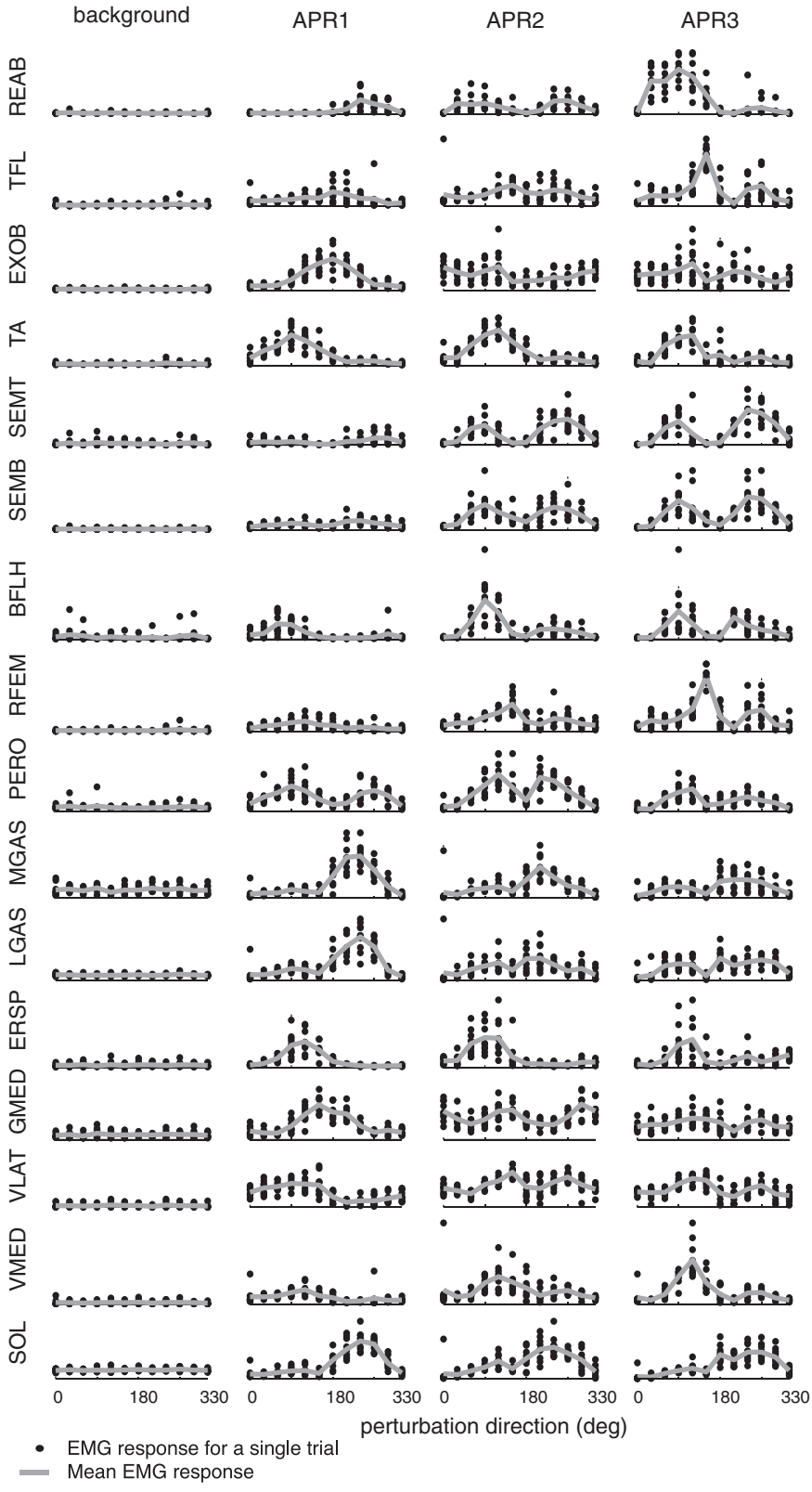

FIG. 2. Muscle tuning curves for all 16 muscles in all 4 time bins in a representative subject. Directional tuning is observed in the activation of all muscles. Differences in muscle onset latencies and changes in directional tuning over time can be observed by comparing responses over the 3 time bins (APR1, APR2, and APR3). *Gray traces* indicate the mean response and black dots represent responses in each trial. Intertrial variations of muscle activations are observed by the vertical spread of data points.

varied between four and six. The number of synergies chosen for each subject was corroborated by the fact that adding more synergies contributed evenly to the VAF of all muscles, suggesting that the extra muscle synergies reconstructed only random variations in the data.

An adequate reconstruction of muscle activation patterns measured over all trials, including all perturbation directions and all time bins, was obtained by the linear combination of a few muscle synergies. Examples of the reconstruction of averaged muscle responses in a sample subject are presented in

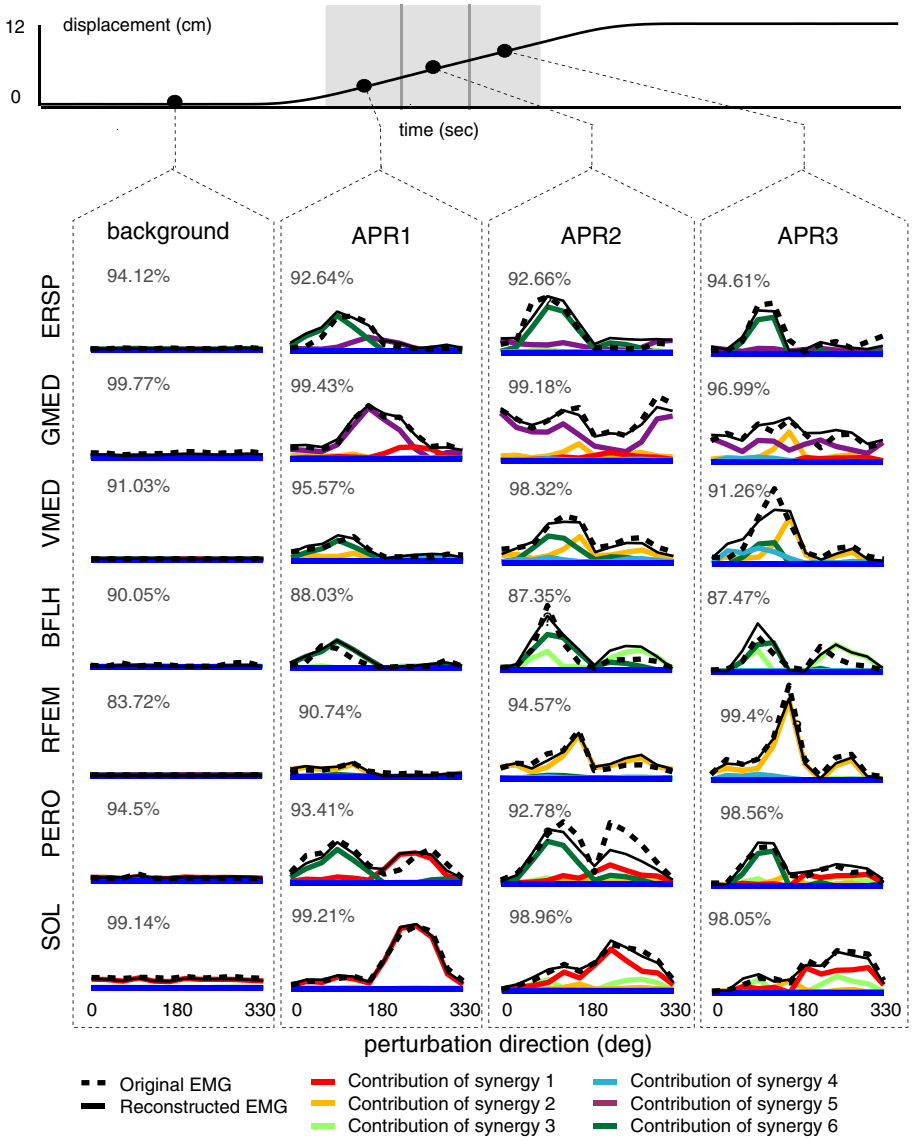

FIG. 3. Mean muscle tuning curves reconstructed using muscle synergies in a representative subject. Original data are shown by the dashed black lines and the reconstructed data by the solid black line. Contribution from each muscle synergy to the reconstruction is shown by the corresponding colored lines, which are added to form the reconstructed muscle tuning curve. Contribution of each muscle synergy is found by multiplying muscle synergy vector W_i by the averaged synergy activation coefficients (C_i). Variability accounted for (VAF) by the reconstruction is displayed in each subpanel.

Fig. 3. The directional tuning of individual muscles across time bins was reproduced by changes in the activation of muscle synergies contributing to the muscle's activity (Fig. 3). The net activation of some muscles such as rectus femoris was accounted for by a single muscle synergy during all time periods, whereas the activation of other muscles such as peroneus was accounted for by the activation of two different muscle synergies. This is similar to reconstructions of averaged muscle tuning curves previously presented in cats (Ting and Macpherson 2005; Torres-Oviedo et al. 2006).

Each muscle synergy (W_i) specifies the activation of several muscles across the body (Fig. 4, *left column*), and each muscle synergy was activated during specific perturbation directions and time bins, as specified by C_i (Fig. 4, *right columns*). Muscle synergies were not strictly grouped by anatomical classification but appear to be grouped by function. For example, in muscle synergies of a representative subject (Fig. 4), W_1 activated the gastrocnemii, peroneus, and soleus, and was active in backward (270°) perturbations, consistent with the "ankle" strategy. W_2 was active in forward perturbations and activated the tibialis anterior, but also includes a number of extensors that were presumably activated to prevent knee and hip joint flexion during the "ankle" strategy to forward perturbations. These two muscle synergies were highly activated during the early time bins of the APR. Synergies W_{3-4} involved trunk and proximal muscles and were active in later time bins (APR_2 and APR_3). W_5 was formed by abductor gluteus medius and lateral trunk muscle external oblique and it was primarily active in medial–lateral (180 and 0°) perturbations. W_6 was composed of biceps femoris, a knee flexor and hip extensor, ankle dorsiflexor tibialis anterior and ankle evertor peroneus, as well as antigravity muscles during upright stance, erector spinae and soleus. The activation and muscle composition of this muscle synergy might be explained by the particular behavior of these subjects who bent their knees in response to perturbations. Only one muscle synergy, W_1, which included soleus, was active during the background period to provide antigravity support (Fig. 4, red muscle synergy). This muscle synergy was observed in all of the subjects (Fig. 7). The independent activation of muscle syner-

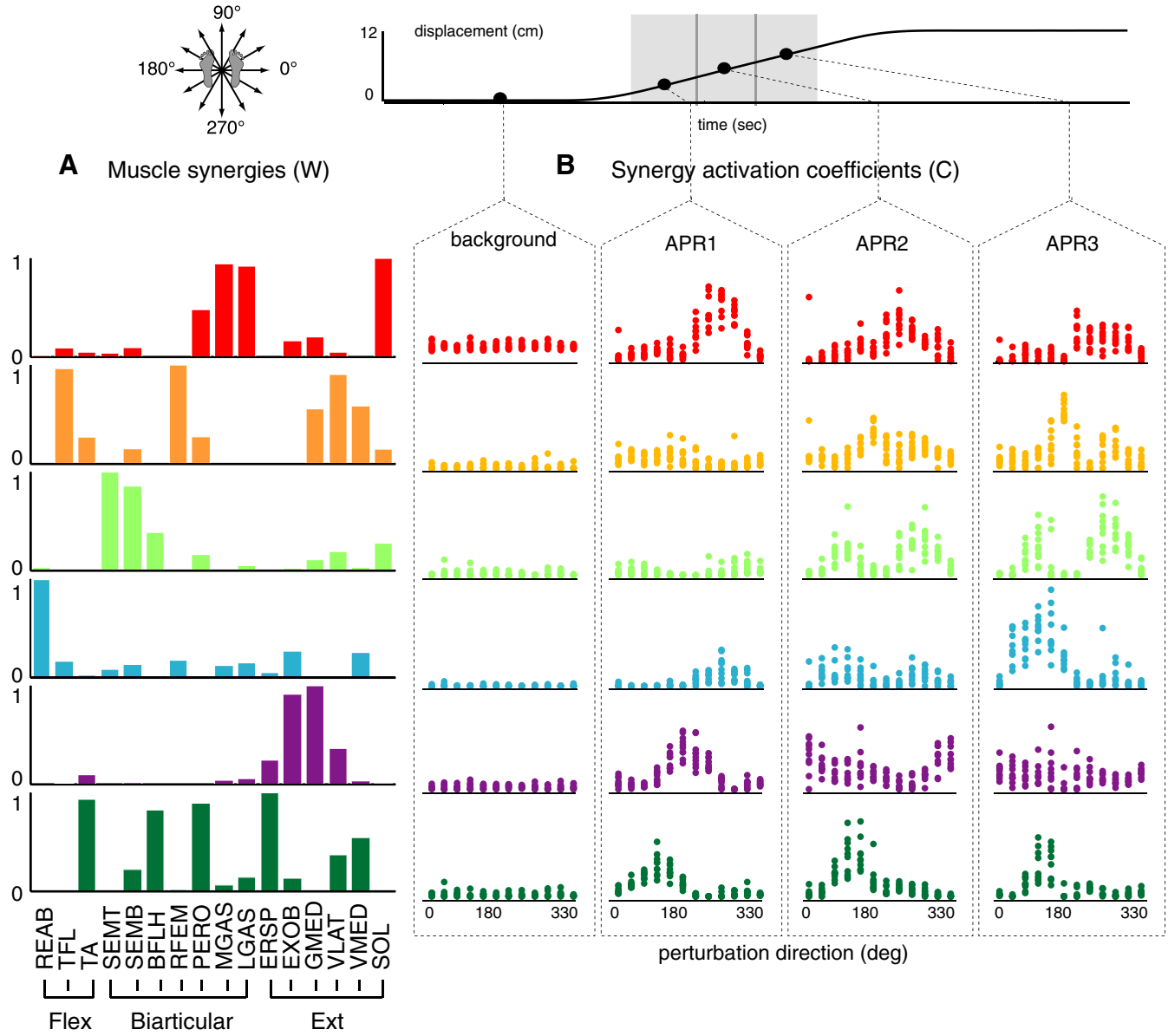

FIG. 4. Muscle synergy vectors and synergy activation coefficients for a representative subject. *A*: muscle synergy vectors, W_i, extracted from EMG data during quiet stance and 3 APR time bins. Each bar represents the relative level of activation of each muscle within the synergy (see METHODS for muscle abbreviations). *B*: activation coefficients, C_i, for each of the 6 synergies during each time bin in multiple perturbation directions. Each dot represents the activity of the muscle synergy in a single trial. Directional tuning of muscle synergies over the 3 time bins can be observed. For example W_1 is mainly active during the initial period of the APR in backward directions (180–270°), whereas W_4 is active during the later time bin of the APR in forward directions (0–90°).

gies enabled them to reproduce changes in EMGs with both time and perturbation direction.

Temporal patterns of activation, specifically the time bins in which maximal activation occurred, varied across muscle synergies (Fig. 5). The mean peak activation coefficients of the "ankle" strategy synergy, W_1 (Fig. 5, $\overline{C}_1$), was significantly larger during APR1 than the other time bins. The mean peak activity of W_5, which was a medial–lateral "hip" strategy, was highest during the early time bins APR1 and APR2 (Fig. 5, $\overline{C}_5$). For $W_{2–3}$ the mean peak activity was greater in APR2 and APR3 than that in the other time bins (Fig. 5, $\overline{C}_2$ and $\overline{C}_3$), whereas the mean peak activity of W_4, a "hip" strategy synergy, was significantly higher during APR3 (Fig. 5, $\overline{C}_3$). Interestingly the activation of muscle synergy 6 ($\overline{C}_6$), "knee" strategy synergy, was the only muscle synergy whose mean peak activity was higher in APR2 compared with all other time bins.

Intertrial variations in EMG patterns are accounted for by muscle synergy activations

EMG activity varied from trial to trial in each muscle, although the patterns of variation were not random or independent across muscles and could be explained by intertrial differences in muscle synergy activation levels. Overall, muscle synergies were directionally tuned, being activated for only a specific range of perturbation directions.

However, the exact level of muscle synergy activation varied from trial to trial (Fig. 4*B*), which affected the level of activity in all muscles within the muscle synergy. For example,

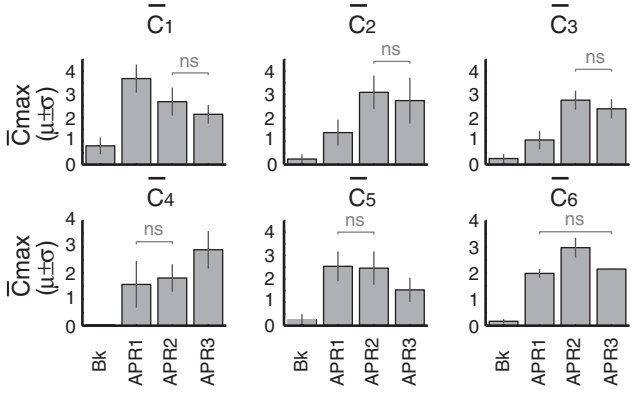

FIG. 5. Mean peak activation of muscle synergies across subjects in all 4 time bins. Each muscle synergy activations level is statistically different in magnitude ($P < 0.05$) from the others in the subpanel, except where specified by the designation *ns*. Mean peak activation of muscle synergy 1, an "ankle" strategy synergy, is largest in APR1, whereas mean peak activation of muscle synergy 4, a "hip" strategy synergy is larger in APR3. "Knee" strategy synergy W_6 has its peak in APR2.

the magnitude of responses in the peroneus muscle to ten forward-left (120°) perturbations varied trial by trial (Fig. 6*A*). In trial 7, peroneus was highly activated (Fig. 6*A*, closed black arrow), whereas in trial 8 peroneus activity was reduced (Fig. 6*A*, open black arrow). This difference was not simply a random variation in muscle activity. The relatively high activity of peroneus in trial 7 could be accounted for by an increase in the activation of muscle synergy W_6. All of the muscles activated by W_6, including peroneus (Fig. 6*A*, closed black arrow), were highly activated in trial 7 (Fig. 6*C*, *left*, dark green bars). In contrast, W_6 activity was lower in trial 8, and all muscles activated by it were reduced (Fig. 6*C*, *right*, dark green bars; peroneus indicated by open black arrow). Similarly, in the magnitude of the gluteus medius response varied across the ten rightward balance perturbations (0°) (Fig. 6*B*). The magnitude of the gluteus medius response could be attributed to the activation of two different muscle synergies (Fig. 6*D*, W_2, yellow; W_5, purple), which affected the overall pattern of activation across all muscles involved in those muscle synergies.

Prior balance perturbation conditions may have affected the responses evoked in subsequent trials. Immediately before trial 7, the subject underwent the same rightward perturbation (0°). In contrast, immediately before trial 9, the subject responded to a perturbation direction in the opposite direction (180°). Thus the contribution of W_5 might have been reduced in trial 7 due to habituation and increased in trial 9 due to the difference in direction between the prior and subsequent perturbation. This observation was consistent with previous studies demonstrating that postural responses are highly influenced by prior trial conditions (Horak and Nashner 1986).

Muscle synergies extracted from averaged data across directions, rather than from all of the trials, had diminished predictive power in reconstructing intertrial variations in EMGs. When muscle synergies from averaged data were used for the reconstruction of muscle activation patterns the mean VAF decreased by 16 ± 5% in the training data set and by 17 ± 6% in the testing data set. Increasing the number of muscle synergies extracted from averaged data did not improve the reconstruction of intertrial variations in EMG patterns. If the variations in EMG patterns were due to random variability, then we would expect a comparable VAF, irrespective of whether averaged data or individual trials were analyzed. However, the analysis of individual trials increased the predictive power of muscle synergies, further suggesting that intertrial variations in EMG patterns were due to variations in the contributions of the muscle synergies to each individual trial.

Similarities in muscle synergies across subjects

Muscle synergy composition and recruitment were similar across subjects. W_1 through W_5 were found in eight of nine subjects (Fig. 7*A*; $0.55 > r^2 > 0.97$), and their muscle synergy activation coefficients were consistent across all subjects (Fig. 7*B*; $0.57 > r^2 > 0.96$). Thus, most muscle synergies categorized in a group were similar in both W and C values, indicating similar muscle synergy patterns as well as directional tuning.

We also identified differences between synergy vectors and activation coefficients within each group. For some groups, there were subjects with muscle synergy patterns similar to those of others in the group, but they were activated with different directional tuning (Fig. 7*A*, open gray boxes). For example, all subjects had a similar W_3 muscle synergy pattern (Fig. 7*A*, green bars), but in four of the subjects, the directional tuning was different (Fig. 7*B*, gray vs. green tuning curves). In contrast, in other groups there were subjects with muscle synergy patterns different from those of the rest of the group, such as W_5 in subjects 1 and 5 (Fig. 7*A*, purple bars with gray shaded box), but they were activated with similar directional tuning (Fig. 7*B*, *purple traces*). Therefore, W_5 in these subjects stabilized the body to the same range of medial–lateral perturbations as the rest of the group. Although W_5 in these subjects had different muscle composition, it appeared to have the same function of performing a medial–lateral "hip" strategy. Therefore these muscle synergies may represent different muscle activation strategies for achieving the same task.

Our analysis also revealed certain subject-specific muscle synergies, such as W_6 in subjects 1 through 4. This muscle synergy was mainly activated in response to forward perturbations, except for subject 4 in which this same synergy was active during background period, when the subject was standing before perturbation, and in response to forward (90°) and backward-left (240°) support surface motions (Fig. 7*B*, *gray trace* in W_6 row).

DISCUSSION

In summary, a few muscle synergies account for the spatial, temporal, and postural strategy variability in human postural responses. In each subject, spatiotemporal characteristics of muscle activation patterns were reproduced by the independent modulation of a few muscle synergies. We were able to quantify the contributions of different postural strategies to "mixed" responses from individual trials. Moreover, repeatability in postural responses was not needed to identify robust muscle synergies. Thus the factorization analysis performed here represents a powerful diagnostic tool that assesses relevant EMG spatiotemporal features in data sets containing high intertrial variations. Further, the consistency of muscle synergy composition across subjects and the similarity in muscle synergy activation patterns across subjects suggest a robust muscle

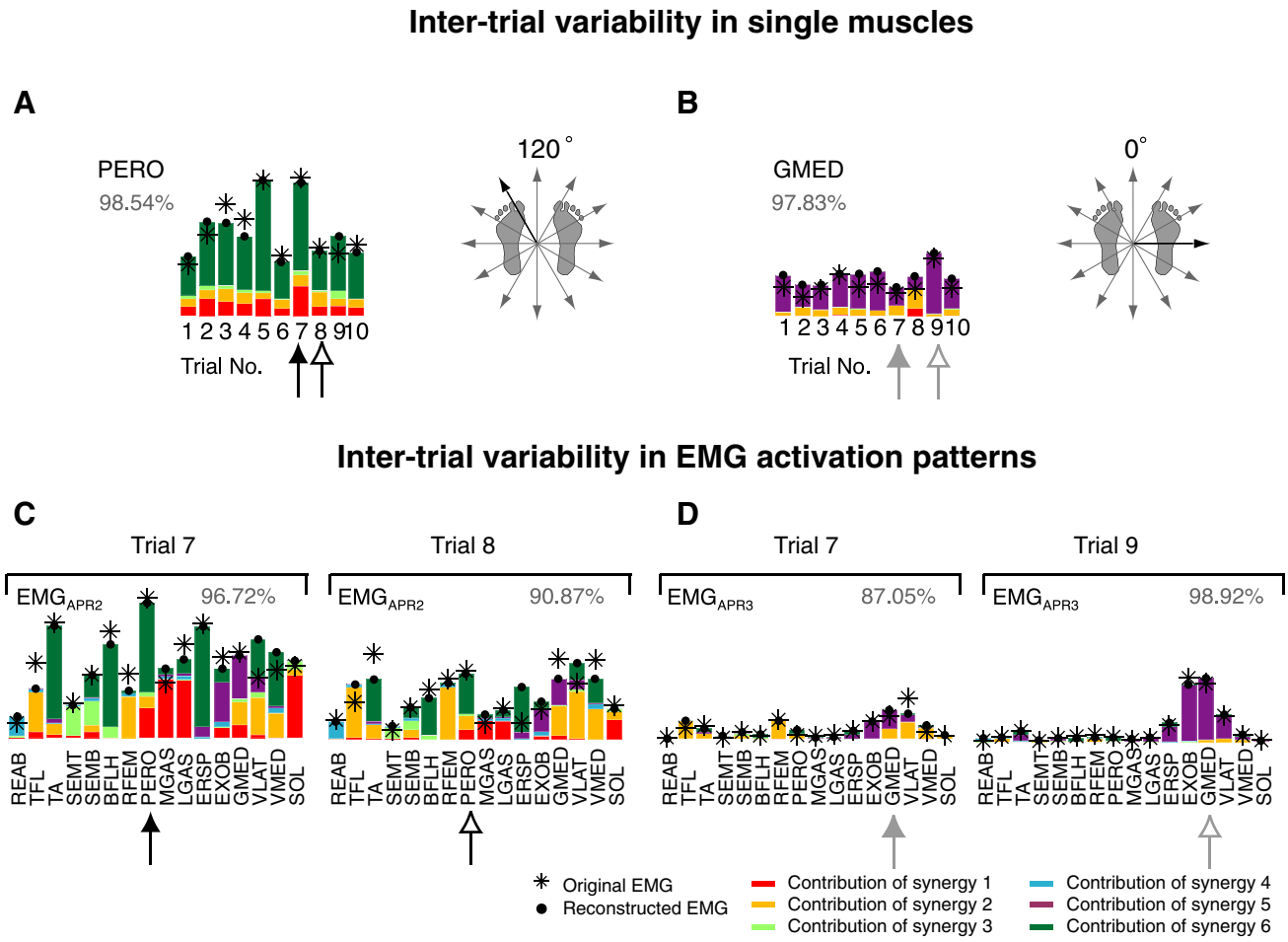

FIG. 6. Intertrial variations in the postural responses of 2 muscles. *A*: peroneus (PERO) responses in APR2 to 10 randomly interspersed trials in the forward-left (120°) perturbation direction. The magnitude of the colored bars represents the contribution of each synergy to the activation of PERO in these 10 trials. Recorded data are indicated by black stars and the reconstructed data by solid black dots. Percentage values indicate the variability accounted for by the muscle synergies (VAF). Magnitude of PERO was high in trial 7, and about half as much in trial 8. *B*: gluteus medius (GMED) responses to 10 to rightward perturbations (0°). *C*: muscle activation levels for all muscles corresponding to trials 7 and 8 shown in *A* in APR2 (EMG_{APR2}). The high activity of peroneus in trial 7 is accounted for by the high activation of muscle synergy W_6 in trial 7, which increases the response magnitude of all of the muscles activated by the W_6 (dark green bars) including peroneus (close black arrow). In trial 8, W_6 activity was relatively low, and all muscles activated by it, including peroneus (open black arrow) was reduced. Therefore trial-to-trial variations in PERO result from the variations in muscle synergies that activate multiple muscles. *D*: muscle activation levels for all muscles corresponding to those in trials 7 and 9 shown in *B*. Magnitude of the gluteus medius response (arrows) could be attributed to the activation of 2 different muscle synergies (W_2, yellow; W_5, purple), which affected the overall pattern of activation across all muscles involved in those muscle synergies.

synergy organization underlying neural control of human balance.

Spatial variability

Our analysis quantitatively identified muscle synergies that produce muscle activation patterns associated with the "ankle" and "hip" strategies previously described in human balance control literature (Horak and Macpherson 1996; Horak et al. 1997). For example, the motor patterns represented by W_{1-2} are consistent with the spatial muscle activation patterns characteristic of the "ankle" strategy. Similarly, W_{3-4} represent motor patterns consistent with the "hip" strategy, where proximal muscles have larger activations than those of ankle muscles, causing a fast movement of the center of mass (CoM) (Henry et al. 1998; Horak and Macpherson 1996). Interestingly, subjects 1 through 4 used a "knee" strategy synergy—W_6—that was not found in other subjects. Knee flexion as a balance strategy in addition to the "ankle" and "hip" strategies has been previously reported in balance studies to support surface rotations (Allum et al. 2003; Gruneberg et al. 2004). The superposition of muscle synergies can generate the more complex motor patterns that have been described as the combination of these two strategies in both the sagittal (Horak and Nashner 1986) and frontal planes (Carpenter et al. 1999; Gruneberg et al. 2005).

Maintaining balance is a multisegmental task that requires interjoint coordination. In multiple studies Alexandrov and colleagues showed that "ankle" and "hip" strategies each define patterns of torque that are coupled across the body to produce coordinated postural responses (Alexandrov et al. 1998, 2001a,b, 2005). In our "ankle" muscle synergies, several proximal muscles are also activated, probably to prevent motion in the hip and knee joints caused by interaction torques (Zajac 2002; Zajac and Gordon 1989). This is particularly true

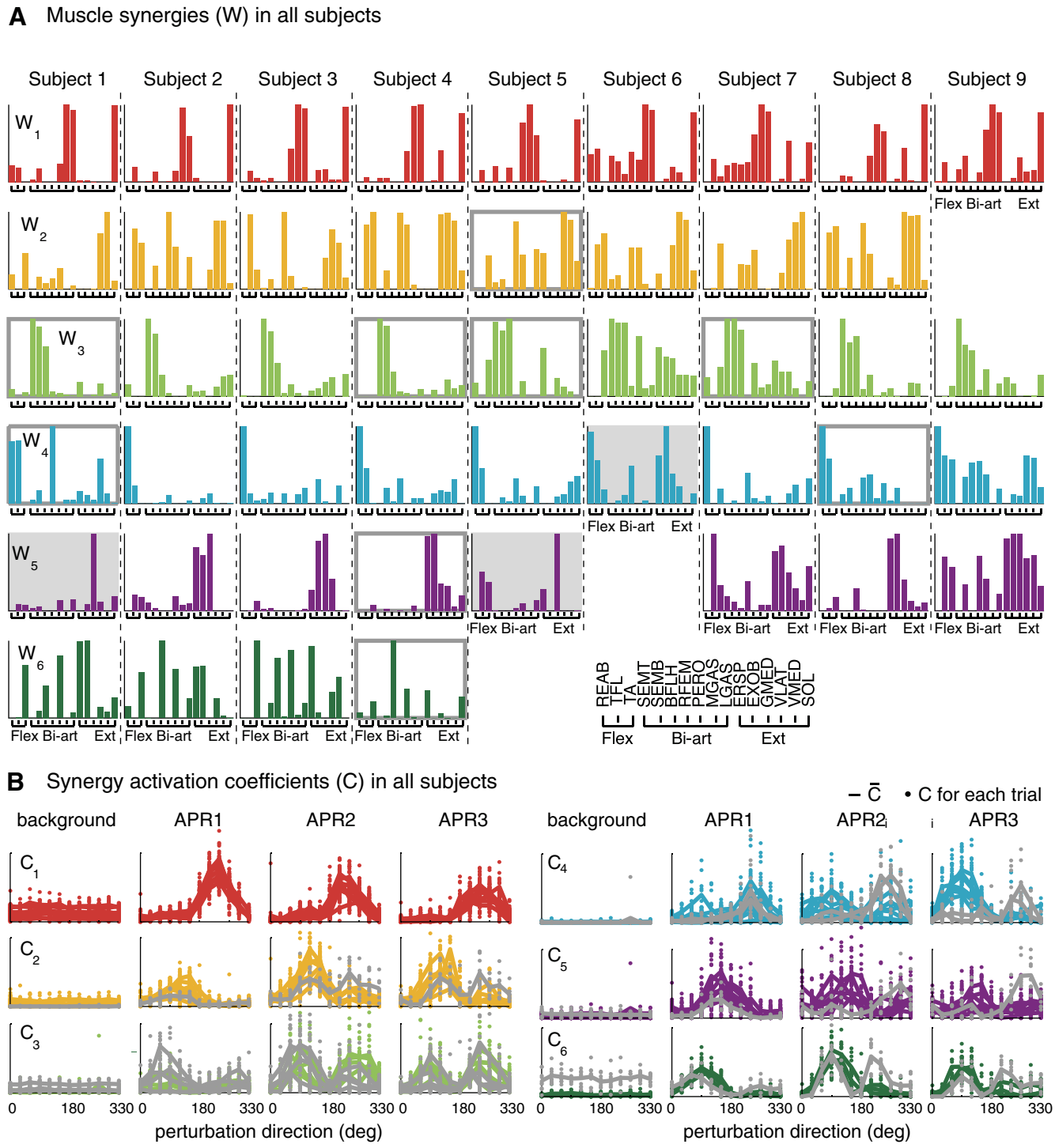

FIG. 7. Muscle synergies and mean synergy activation coefficients for all subjects. Muscle synergies were sorted across subjects and grouped by their similarity. *A*: muscle synergies groups across all subjects. *B*: directional tuning of muscle synergies in each group. Muscle synergies composition of W_{1-5} were similar across subjects ($0.55 > r^2 > 0.97$). However, in 4 subjects, the muscle synergy pattern differed from the group (muscle synergies on gray background); these were grouped based on their muscle tuning curves, which were similar to the rest of the group. These muscle synergies were activated for the same range of balance perturbations as the rest of the group, but had different muscle composition, suggesting that they may be "goal-equivalent" muscle synergies that perform a similar task with different muscles. We also identified muscle synergies that were similar in muscle composition but were activated for different range of perturbation directions when compared with other subjects (muscle synergies in gray outline). *Gray traces* in *B* are the tuning curves of muscle synergies with gray outline in *A*. These muscle synergies were similar in muscle composition across subjects but different in spatiotemporal activation pattern.

in response to forward perturbations where no mechanical limits of joint range can be utilized (cf. backward perturbations that tend to extend the knee) (Horak and Macpherson 1996; Horak and Nashner 1986). Further testing using musculoskeletal models is needed to determine the influence of biomechanics in muscle synergy organization.

Temporal variability

The variations in onset latencies in muscle activity could be accounted for by the differential activation of muscle synergies over time: that is, in response to anterior–posterior perturbations, muscle synergies formed by distal ankle muscles were activated first and muscle synergies formed by proximal leg and trunk muscles were activated later. These results are consistent with previous studies reporting distal to proximal muscle responses to anterior–posterior balance perturbations (Horak and Macpherson 1996). Moreover, during medial–lateral perturbations W_5, mainly formed by proximal muscles and few distal muscles, was the only muscle synergy activated. In medial–lateral balance perturbations, proximal muscles responded with the same early latency as the distal ankle muscles, a temporal organization that is consistent with previous studies (Carpenter et al. 1999; Gruneberg et al. 2005; Henry et al. 1998). Using just three time bins, we were able to characterize basic temporal features in synergy activations that were consistent with previous studies describing temporal features of individual muscle activations. These time bins characterized the primary temporal phases of the postural response. If we were to use a finer temporal resolution, we anticipate that we would observe a more accurate timing profile of synergy activations showing the transitions between postural strategies, but the basic synergy organization and conclusion of the study would remain unchanged.

Our results suggest that the neural commands activating the various muscle synergies have different and independent time courses. Other studies have addressed temporal differences in muscle activation during locomotor behaviors or fast reaching movements by identifying fixed time delays associated with each muscle synergy (d'Avella et al. 2003, 2006), suggesting feedforward muscle synergy activation. Although this may be appropriate for rhythmic locomotor behaviors or ballistic movements, muscle activation in postural responses to balance perturbations is modulated by sensory feedback due to the perturbation (Kuo 1995, 2005; Park et al. 2004; Peterka 2002). Thus in our analysis we assumed that each muscle synergy was a time-invariant muscle activation pattern and the entire muscle synergy could be modulated by time-dependent feedback signals. This is consistent with the effect of sensory feedback on synergy activation coefficients revealed during locomotion behaviors of intact and deafferented frogs (Cheung et al. 2005).

Intertrial variability

Intertrial variations in muscle activation patterns reflect differences in postural strategies used in each trial. Although the nervous system's goal in the studied motor behavior might be to maintain the body CoM within the base of support, which is the area under and between the feet, multiple factors such as environmental context and mental state influence the postural strategy selected (Carpenter et al. 2006; Keshner et al. 1987; Woollacott and Shumway-Cook 2002). For example, adaptation studies show that postural responses to support surface translations on a stable surface are affected by the immediate prior experience of performing the same task on an unstable surface (Horak 1996; Horak and Nashner 1986). In this work we demonstrate quantitatively that intertrial variability results from the variable activation of muscle synergies associated with the various postural strategies.

The question remains of whether systematic variations in muscle synergy activations exist, representing a manifold in muscle synergy space (Krishnamoorthy et al. 2007) that defines the continuum of feasible postural response patterns to the same perturbation. Although we were unable to find any consistent relationships across subjects, there was some evidence of structure in the coordination of muscle synergy activations. In all subjects, we identified correlations ($P < 0.05$) between the activation of at least two muscle synergies in response to either forward (90°) perturbations or backward (270°) perturbations. For example, in subject 3, there was a reciprocal relationship between activation of the "ankle strategy" synergy W_2, versus both the "knee strategy" synergy W_6 and the "hip strategy" synergy W_5 (Fig. 8*A*). That is, the more the ankle strategy was activated, the less the knee and hip strategies were activated. Thus for this subject, a planar relationship exists between these three muscle synergy activations, defining the spectrum of muscle activation pattern responses seen in response to this specific perturbation direction. However, the activation of muscle synergies may lie in a more complex multidimensional space because these correlations disappear when examined over all perturbation directions. Further analysis such as uncontrolled manifold analysis (UCM) (Scholz and Schoner 1999) could be performed to explicitly determine whether variations in muscle synergy contributions lead to a consistent control of CoM displacements to maintain upright balance. This insightful approach has been previously used to study postural tasks such as standing balance on load release (Krishnamoorthy et al. 2003, 2004) or arm postural tasks (Krishnamoorthy et al. 2007). For example, Krishnamoorthy et al. demonstrated that the flexible combination of muscle synergies (M-modes) is organized to stabilize the arm to produce a specific endpoint force (Krishnamoorthy et al. 2007).

Prior studies have had difficulty dealing with intertrial variability because most responses represent compound "ankle" and "hip" strategies, defined as "mixed" strategies (Horak and Macpherson 1996; Horak and Nashner 1986). In our experiments we induced high intertrial variability by randomly interspersing trials with different perturbation directions and stance configurations. We were able to decompose muscle activation patterns in each individual trial or response into explicit contributions of each postural strategy, as represented by a specific muscle synergy. Thus our analysis represents a powerful method that could be used for clinical research to assess important spatiotemporal features of muscle coordination needed to perform a task in a data set containing high intertrial variations. The robustness of muscle synergies across multiple trials suggests that muscle synergies encode goal-directed patterns of motor output that are modulated by higher centers to produce the appropriate postural response based on the particular postural strategy and postural task (Dietz 1992; Horak 1996).

Muscle synergy robustness across subjects

Similarity in muscle synergies may reflect consistency in neural circuitry or biomechanical constraints across subjects. All subjects used a consistent low-dimensional set of muscle synergies over two consecutive days and most muscle synergy patterns were similar across multiple subjects. These results

suggest muscle synergies might be encoded in the nervous system as indicated by studies in kicks and frog locomotor behaviors revealing muscle synergies encoded in the frog spinal cord (Hart and Giszter 2004; Saltiel et al. 2001, 2005). In addition, the biomechanics of the body might also influence the consistency of muscle synergies. For example, the biomechanics of the human hand has been shown to constrain the variability in muscle activation patterns when producing voluntary finger endpoint forces (Valero-Cuevas 2000; Valero-Cuevas et al. 1998).

Differences in muscle synergy composition and activation may reflect subject-specific movement patterns and demonstrate the musculoskeletal redundancy in achieving the task of keeping the center of mass over the base of support during a postural perturbation. W_5 values in all subjects were activated for the same directions of balance perturbation but their muscle composition varied across subjects (Fig. 6; muscle synergies on gray background). Similar intersubject differences in muscle synergies have been shown in upper-arm movements where two different strategies are used to produce the same movement (Sabatini 2002).

Although differences in anatomy may contribute to differences in muscle synergies, it is likely that prior training and motor skill also influenced subject-specific movement patterns. Learning a motor skill may influence the performance of another motor skill (Schmidt and Lee 2005) and this generalization depends on the context in which our limbs are normally used (Krakauer et al. 2006). Also, new synergies and new contributions of each synergy to net motor output can be formed when individuals are trained to perform different motor behaviors (Mussa-Ivaldi and Bizzi 2000). Our analysis might by useful for quantifying changes in muscle synergy organization with rehabilitation, or to compare and contrast strategies used by different subjects.

Muscle synergy generality

In the current study, we demonstrate that more than one synergy can be used to stabilize the center of mass for a given perturbation direction. Similarly, multiple synergies have been identified in the frog for performing leg extension (Saltiel et al. 1998). Therefore while constraining the possible motor output patterns for a particular movement, the use of muscle synergies does not uniquely specify the response pattern used for a given postural perturbation because multiple muscle synergies can be combined in different proportions.

That the muscle synergies identified could account for variations within a single postural task demonstrates that muscle synergies may indeed be modules used for controlling task-level variables, such as center of mass motion. Several studies have shown that the activation of muscle synergies correlates to the control of task-level variables such as endpoint force (Ting and Macpherson 2005; Torres-Oviedo et al. 2006), or center of pressure displacement (Krishnamoorthy et al. 2003) in postural tasks, and endpoint foot kinematics during locomotion (Ivanenko et al. 2003). It remains to be seen whether the muscle synergies identified here are general across a wide range of stance configurations (Henry et al. 2001), or for

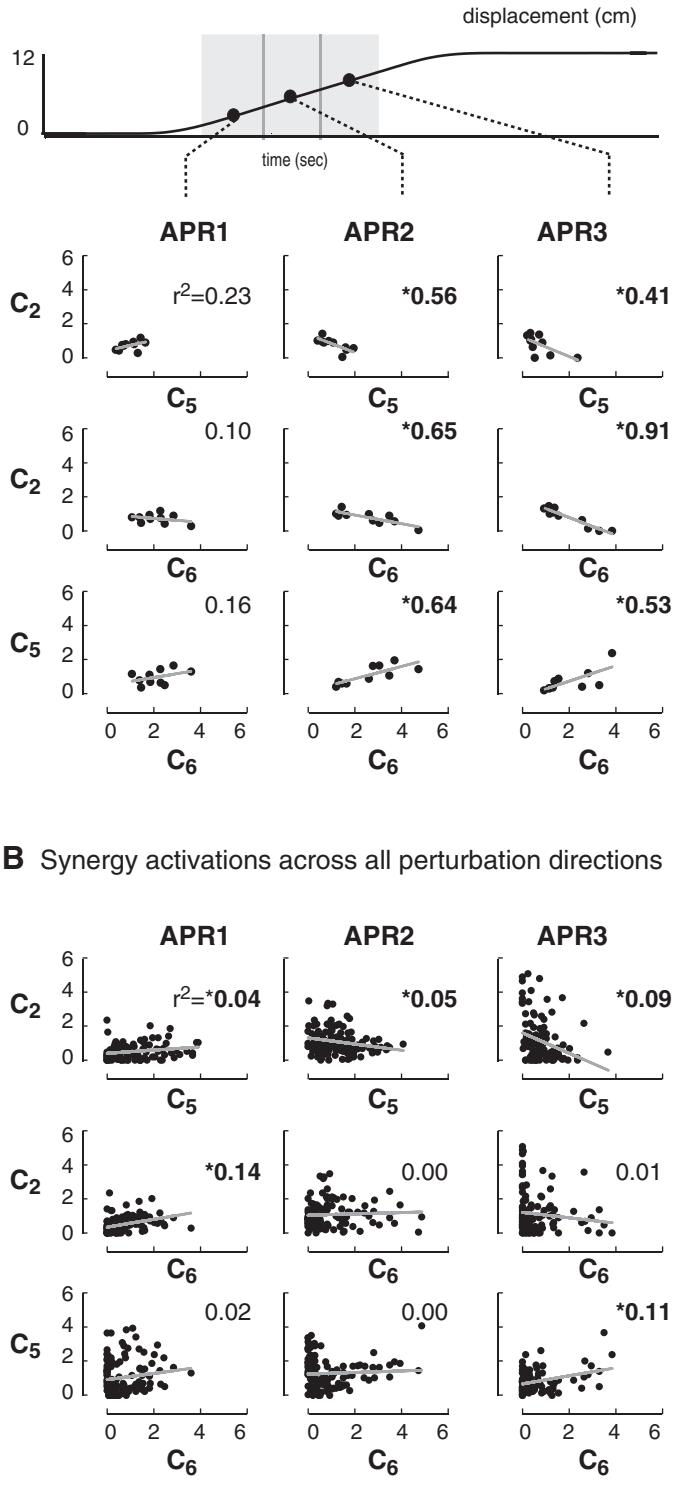

FIG. 8. Example of muscle synergy coordination across trials. *A*: linear regressions between muscle synergy activations in response to all trials of a forward perturbations in subject 3. R^2 values are presented on each plot and statistically significant values ($P < 0.05$) are indicated by an asterisk and boldface. Negative correlations were found between the "ankle strategy" synergy, W_2, and both the "hip strategy" synergy, W_5, and "knee strategy" synergy, W_6. *B*: linear regressions between muscle synergy activations in response to all perturbation directions in subject 3. Strict correlations observed in forward perturbations largely disappear when all perturbations are considered. This suggests that the relationships between the muscle synergies may change as a function of perturbation direction.

different types of postural responses such as taking a compensatory step (McIlroy and Maki 1993, 1999). However, prior work demonstrating the generality of muscle synergies across different postural and locomotor tasks in animals (d'Avella and Bizzi 2005; Torres-Oviedo et al. 2006) suggests that the muscle synergies identified probably represent general motor output patterns for movement. The existence of such modules of motor output are consistent with the fact that neural firing in the motor cortex (Georgopoulos et al. 1982) and spinal cord (Poppele and Bosco 2003) appear to encode task-level variables. This is further supported by the modular behaviors evoked by stimulation of the premotor cortex (Graziano 2006) and the spinal cord (Lemay and Grill 2004; Saltiel et al. 2001). Our results provide evidence for the hypothesis that muscle coordination can be produced by the activation of a few muscle synergies that represent tailored modules controlling specific task-level variables. Moreover, the contributions of each muscle synergy may be modulated by descending influences on postural strategy such as prior experience or anticipation, as well as regulated through sensory feedback to perform motor behaviors.

ACKNOWLEDGMENTS

We thank J. R. Warren and T. Otchy for excellent technical support developing the experimental apparatus.

GRANTS

This work was supported by National Institutes of Health Grants HD-46922 and NS-053822.

REFERENCES

Alexandrov A, Frolov A, Massion J. Axial synergies during human upper trunk bending. *Exp Brain Res* 118: 210–220, 1998.

Alexandrov AV, Frolov AA, Horak FB, Carlson-Kuhta P, Park S. Feedback equilibrium control during human standing. *Biol Cybern* 93: 309–322, 2005.

Alexandrov AV, Frolov AA, Massion J. Biomechanical analysis of movement strategies in human forward trunk bending. I. Modeling. *Biol Cybern* 84: 425–434, 2001a.

Alexandrov AV, Frolov AA, Massion J. Biomechanical analysis of movement strategies in human forward trunk bending. II. Experimental study. *Biol Cybern* 84: 435–443, 2001b.

Allum JH, Carpenter MG, Honegger F. Directional aspects of balance corrections in man. *IEEE Eng Med Biol Mag* 22: 37–47, 2003.

Cappellini G, Ivanenko YP, Poppele RE, Lacquaniti F. Motor patterns in human walking and running. *J Neurophysiol* 95: 3426–3437, 2006.

Carpenter MG, Adkin AL, Brawley LR, Frank JS. Postural, physiological and psychological reactions to challenging balance: does age make a difference? *Age Ageing* 35: 298–303, 2006.

Carpenter MG, Allum JHJ, Honegger F. Directional sensitivity of stretch reflexes and balance corrections for normal subjects in the roll and pitch planes. *Exp Brain Res* 129: 93–113, 1999.

Cheung VCK, d'Avella A, Tresch MC, Bizzi E. Central and sensory contributions to the activation and organization of muscle synergies during natural motor behaviors. *J Neurosci* 25: 6419–6434, 2005.

Creath R, Kiemel T, Horak F, Peterka R, Jeka J. A unified view of quiet and perturbed stance: simultaneous co-existing excitable modes. *Neurosci Lett* 377: 75–80, 2005.

d'Avella A, Bizzi E. Shared and specific muscle synergies in natural motor behaviors. *Proc Natl Acad Sci USA* 102: 3076–3081, 2005.

d'Avella A, Portone A, Fernandez L, Lacquaniti F. Control of fast-reaching movements by muscle synergy combinations. *J Neurosci* 26: 7791–7810, 2006.

d'Avella A, Saltiel P, Bizzi E. Combinations of muscle synergies in the construction of a natural motor behavior. *Nat Neurosci* 6: 300–308, 2003.

Diener HC, Horak FB, Nashner LM. Influence of stimulus parameters on human postural responses. *J Neurophysiol* 59: 1888–1905, 1988.

Dietz V. Human neuronal control of automatic functional movements: interaction between central programs and afferent input. *Physiol Rev* 72: 33–69, 1992.

Dunbar DC, Horak FB, Macpherson JM, Rushmer DS. Neural control of quadrupedal and bipedal stance: implications for the evolution of erect posture. *Am J Phys Anthropol* 69: 93–105, 1986.

Georgopoulos AP, Kalaska JF, Caminiti R, Massey JT. On the relations between the direction of two-dimensional arm movements and cell discharge in primate motor cortex. *J Neurosci* 2: 1527–1537, 1982.

Graziano M. The organization of behavioral repertoire in motor cortex. *Annu Rev Neurosci* 29: 105–134, 2006.

Gruneberg C, Bloem BR, Honegger F, Allum JH. The influence of artificially increased hip and trunk stiffness on balance control in man. *Exp Brain Res* 157: 472–485, 2004.

Gruneberg C, Duysens J, Honegger F, Allum JH. Spatio-temporal separation of roll and pitch balance-correcting commands in humans. *J Neurophysiol* 94: 3143–3158, 2005.

Hart CB, Giszter SF. Modular premotor drives and unit bursts as primitives for frog motor behaviors. *J Neurosci* 24: 5269–5282, 2004.

Henry SM, Fung J, Horak FB. EMG responses to maintain stance during multidirectional surface translations. *J Neurophysiol* 80: 1939–1950, 1998.

Henry SM, Fung J, Horak FB. Effect of stance width on multidirectional postural responses. *J Neurophysiol* 85: 559–570, 2001.

Horak FB. Adaptation of automatic postural responses. In: *The Acquisition of Motor Behavior in Vertebrates*, edited by Bloedel JR, Ebner TJ, Wise SP. Cambridge, MA: The MIT Press, 1996, p. 57–85.

Horak FB, Henry SM, Shumway-Cook A. Postural perturbations: new insights for treatment of balance disorders. *Phys Ther* 77: 517–533, 1997.

Horak FB, Macpherson JM. Postural orientation and equilibrium. In: *Handbook of Physiology. Exercise: Regulation and Integration of Multiple Systems. Neural Control of Movement*. Bethesda, MD: Am. Physiol. Soc, 1996, sect. 12, p. 255–292.

Horak FB, Nashner LM. Central programming of postural movements: adaptation to altered support-surface configurations. *J Neurophysiol* 55: 1369–1381, 1986.

Ivanenko YP, Cappellini G, Dominici N, Poppele RE, Lacquaniti F. Coordination of locomotion with voluntary movements in humans. *J Neurosci* 25: 7238–7253, 2005.

Ivanenko YP, Grasso R, Zago M, Molinari M, Scivoletto G, Castellano V, Macellari V, Lacquaniti F. Temporal components of the motor patterns expressed by the human spinal cord reflect foot kinematics. *J Neurophysiol* 90: 3555–3565, 2003.

Ivanenko YP, Poppele RE, Lacquaniti E. Five basic muscle activation patterns account for muscle activity during human locomotion. *J Physiol* 556: 267–282, 2004.

Keshner EA, Allum JH, Pfaltz CR. Postural coactivation and adaptation in the sway stabilizing responses of normals and patients with bilateral vestibular deficit. *Exp Brain Res* 69: 77–92, 1987.

Krakauer JW, Mazzoni P, Ghazizadeh A, Ravindran R, Shadmehr R. Generalization of motor learning depends on the history of prior action. *PLoS Biol* 4: e316, 2006.

Krishnamoorthy V, Latash ML, Scholz JP, Zatsiorsky VM. Muscle synergies during shifts of the center of pressure by standing persons. *Exp Brain Res* 152: 281–292, 2003.

Krishnamoorthy V, Latash ML, Scholz JP, Zatsiorsky VM. Muscle modes during shifts of the center of pressure by standing persons: effect of instability and additional support. *Exp Brain Res* 157: 18–31, 2004.

Krishnamoorthy V, Scholz JP, Latash ML. The use of flexible arm muscle synergies to perform an isometric stabilization task. *Clin Neurophysiol* 118: 525–537, 2007.

Kuo AD. An optimal control model for analyzing human postural balance. *IEEE Trans Biomed Eng* 42: 87–101, 1995.

Kuo AD. An optimal state estimation model of sensory integration in human postural balance. *J Neural Eng* 2: S235–S249, 2005.

Lee DD, Seung HS. Algorithms for non-negative matrix factorization. *Adv Neural Info Proc Syst* 13: 556–562, 2001.

Lemay MA, Grill WM. Modularity of motor output evoked by intraspinal microstimulation in cats. *J Neurophysiol* 91: 502–514, 2004.

Macpherson JM. Strategies that simplify the control of quadrupedal stance. II. Electromyographic activity. *J Neurophysiol* 60: 218–231, 1988.

Massion J, Alexandrov A, Frolov A. Why and how are posture and movement coordinated? In: *Progress in Brain Research: Brain Mechanisms for*

the Integration of Posture and Movement, edited by Mori S, Stuart DG, Wiesendanger M. Amsterdam: Elsevier Science, 2004, p. 13–27.

McIlroy WE, Maki BE. Changes in early "automatic" postural responses associated with the prior-planning and execution of a compensatory step. *Brain Res* 631: 203–211, 1993.

McIlroy WE, Maki BE. The control of lateral stability during rapid stepping reactions evoked by antero-posterior perturbation: does anticipatory control play a role? *Gait Posture* 9: 190–198, 1999.

Mussa-Ivaldi FA, Bizzi E. Motor learning through the combination of primitives. *Philos Trans R Soc Lond B Biol Sci* 355: 1755–1769, 2000.

Nashner LM. Fixed patterns of rapid postural responses among leg muscles during stance. *Exp Brain Res* 30: 13–24, 1977.

Park S, Horak FB, Kuo AD. Postural feedback responses scale with biomechanical constraints in human standing. *Exp Brain Res* 154: 417–427, 2004.

Peterka RJ. Sensorimotor integration in human postural control. *J Neurophysiol* 88: 1097–1118, 2002.

Poppele R, Bosco G. Sophisticated spinal contributions to motor control. *Trends Neurosci* 26: 269–276, 2003.

Sabatini AM. Identification of neuromuscular synergies in natural upper-arm movements. *Biol Cybern* 86: 253–262, 2002.

Saltiel P, Tresch MC, Bizzi E. Spinal cord modular organization and rhythm generation: an NMDA iontophoretic study in the frog. *J Neurophysiol* 80: 2323–2339, 1998.

Saltiel P, Wyler-Duda K, d'Avella A, Ajemian RJ, Bizzi E. Localization and connectivity in spinal interneuronal networks: the adduction-caudal extension-flexion rhythm in the frog. *J Neurophysiol* 94: 2120–2138, 2005.

Saltiel P, Wyler-Duda K, d'Avella A, Tresch MC, Bizzi E. Muscle synergies encoded within the spinal cord: evidence from focal intraspinal NMDA iontophoresis in the frog. *J Neurophysiol* 85: 605–619, 2001.

Schmidt RA, Lee TD. Retention and transfer. In: *Motor Control and Learning: A Behavioral Emphasis.* Champaign, IL: Human Kinetics, 2005.

Scholz JP, Schoner G. The uncontrolled manifold concept: identifying control variables for a functional task. *Exp Brain Res* 126: 289–306, 1999.

Ting LH, Macpherson JM. A limited set of muscle synergies for force control during a postural task. *J Neurophysiol* 93: 609–613, 2005.

Torres-Oviedo G, Macpherson JM, Ting LH. Muscle synergy organization is robust across a variety of postural perturbations. *J Neurophysiol* 96: 1530–1546, 2006.

Tresch MC, Cheung VC, d'Avella A. Matrix factorization algorithms for the identification of muscle synergies: evaluation on simulated and experimental data sets. *J Neurophysiol* 95: 2199–2212, 2006.

Tresch MC, Saltiel P, Bizzi E. The construction of movement by the spinal cord. *Nat Neurosci* 2: 162–167, 1999.

Valero-Cuevas FJ. Predictive modulation of muscle coordination pattern magnitude scales fingertip force magnitude over the voluntary range. *J Neurophysiol* 83: 1469–1479, 2000.

Valero-Cuevas FJ, Zajac FE, Burgar CG. Large index-fingertip forces are produced by subject-independent patterns of muscle excitation. *J Biomech* 31: 693–703, 1998.

Woollacott MH, Shumway-Cook A. Attention and the control of posture and gait: a review of an emerging area of research. *Gait Posture* 16: 1–14, 2002.

Zajac FE. Understanding muscle coordination of the human leg with dynamical simulations. *J Biomech* 35: 1011–1018, 2002.

Zajac FE, Gordon ME. Determining muscle's force and action in multi-articular movement. *Exerc Sport Sci Rev* 17: 187–230, 1989.

Zar JH. *Biostatistical Analysis*. Upper Saddle River, NJ: Prentice-Hall, 1999.