Muscle Synergies Characterizing Human Postural Responses

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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-
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. 1B). Ramp-and-hold perturbations [total displacement: 12.4 cm; peak velocity: 35 cm/s; peak accelerations 490 cm/s² (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.

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 × 12 directions × 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-

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 abdominis (REAB), tensor fascia lata (TFL), biceps femoris long head (BF LH), 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.

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vation 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 \( \mathbf{M} \) (e.g., Fig. 6C, EMGAPR1, and EMGAPR2), 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 \( \mathbf{M} \) takes the form

\[
\mathbf{M} = c_1 \mathbf{W}_1 + c_2 \mathbf{W}_2 + \cdots + c_n \mathbf{W}_n
\]

\( \mathbf{W}_i \) 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 \( \mathbf{M} \). 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 non-negative 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 \( 100 \times \) 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 glutus 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 abdominis, 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 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
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. 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.
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 (Wi) 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 Ci (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), W1 activated the gastrocnemii, peroneus, and soleus, and was active in backward (270°) perturbations, consistent with the “ankle” strategy. W2 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 W3–4 involved trunk and proximal muscles and were active in later time bins (APR2 and APR3). W5 was formed by abductor gluteus medius and lateral trunk muscle external oblique and it was primarily active in medial–lateral (180 and 0°) perturbations. W6 was composed of biceps femoris, a knee flexor and hip extensor, ankle dorsiflexor tibialis anterior and ankle extensor 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, W1, 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-
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 syn-
ergies (Fig. 5). The mean peak activation coefficients of the
“ankle” strategy synergy, W₁ (Fig. 5, C₁), was significantly
larger during APR1 than the other time bins. The mean peak
activity of W₅, which was a medial–lateral “hip” strategy, was
highest during the early time bins APR1 and APR2 (Fig. 5,
C₅). For W₂–₃ the mean peak activity was greater in APR2
and APR3 than that in the other time bins (Fig. 5, C₂ and C₃),
whereas the mean peak activity of W₄, a “hip” strat-
ecy synergy, was significantly higher during APR3 (Fig. 5, C₃).

Interestingly the activation of muscle synergy 6 (C₆),
“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 indepen-
dent across muscles and could be explained by intertrial dif-
fences 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 var-
ied from trial to trial (Fig. 4B), which affected the level of
activity in all muscles within the muscle synergy. For example,
the magnitude of responses in the peroneus muscle to ten forward-left (120°) perturbations varied trial by trial (Fig. 6A). In trial 7, peroneus was highly activated (Fig. 6A, closed black arrow), whereas in trial 8 peroneus activity was reduced (Fig. 6A, 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 W6. All of the muscles activated by W6, including peroneus (Fig. 6A, closed black arrow), were highly activated in trial 7 (Fig. 6C, left, dark green bars). In contrast, W6 activity was lower in trial 8, and all muscles activated by it were reduced (Fig. 6C, 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. 6B). The magnitude of the gluteus medius response could be attributed to the activation of two different muscle synergies (Fig. 6D, W5, yellow; W6, 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 W5 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. W1 through W6 were found in eight of nine subjects (Fig. 7A; 0.55 > r² > 0.97), and their muscle synergy activation coefficients were consistent across all subjects (Fig. 7B; 0.57 > r² > 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. 7A, open gray boxes). For example, all subjects had a similar W3 muscle synergy pattern (Fig. 7A, green bars), but in four of the subjects, the directional tuning was different (Fig. 7B, gray vs. green tuning curves). In contrast, other groups there were subjects with muscle synergy patterns different from those of the rest of the group, such as W5 in subjects 1 and 5 (Fig. 7A, purple bars with gray shaded box), but they were activated with similar directional tuning (Fig. 7B, purple traces). Therefore, W5 in these subjects stabilized the body to the same range of medial–lateral perturbations as the rest of the group. Although W5 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 W6 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. 7B, gray trace in W6 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
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 W1–2 are consistent with the spatial muscle activation patterns characteristic of the “ankle” strategy. Similarly, W3–4 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—W6—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. 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 (EMGAPR2). The high activity of peroneus in trial 7 is accounted for by the high activation of muscle synergy W6 in trial 7, which increases the response magnitude of all of the muscles activated by the W6 (dark green bars) including peroneus (close black arrow). In trial 8, W6 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 (W2, yellow; W5, purple), which affected the overall pattern of activation across all muscles involved in those muscle synergies.
Muscle synergies (W) in all subjects

Synergy activation coefficients (C) in all subjects

Subject 1 Subject 2 Subject 3 Subject 4 Subject 5 Subject 6 Subject 7 Subject 8 Subject 9

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 $W_S$, 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 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. 8A). 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. W5 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 be 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

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**FIG. 8.** Example of muscle synergy coordination across trials. A: Linear regressions between muscle synergy activations in response to all trials of a forward perturbation 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.

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