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RESEARCH ARTICLE

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Relationship between cocontraction, movement kinematics and phasic muscle activity in single-joint arm movement

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Abstract Patterns of muscle coactivation provide a window into mechanisms of limb stabilization. In the present paper we have examined muscle coactivation in single-joint elbow and single-joint shoulder movements and explored its relationship to movement velocity and amplitude, as well as phasic muscle activation patterns. Movements were produced at several speeds and different amplitudes, and muscle activity and movement kinematics were recorded. Tonic levels of electromyographic (EMG) activity following movement provided a measure of muscle cocontraction. It was found that coactivation following movement increased with maximum joint velocity at each of two amplitudes. Phasic EMG activity in agonist and antagonist muscles showed a similar correlation that was observable even during the first 30 ms of muscle activation. All subjects but one showed statistically significant correlations on a trial-by-trial basis between tonic and phasic activity levels, including the phasic activity measure taken at the initiation of movement. Our findings provide direct evidence that muscle coactivation varies with movement velocity. The data also suggest that cocontraction is linked in a simple manner to phasic muscle activity. The similarity in the patterns of tonic and phasic activation suggests that the nervous system may use a simple strategy to adjust coactivation and presumably limb impedance in association with changes in movement speed. Moreover, since the pattern of tonic activity varies with the first 30 ms of phasic activity, the control of cocontraction may be established prior to movement onset.

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Introduction

Muscle cocontraction (or coactivation) is a primary means by which the nervous system stabilizes the position of the limb. Whereas extensive work has been carried out to understand the relationship between movement production and associated kinematics and electromyographic (EMG) patterns (see Latash 1993 and Pfann et al. 1998 for recent summaries), comparatively little is known about the control of cocontraction.

Evidence from behavioral studies suggests that muscle coactivation and movement may be separately controlled. For example, subjects can independently vary the magnitudes of coactivation and reciprocal activity (Yamazaki et al. 1994, 1995), coactivation of antagonist muscles can be modified over a wide range of values while maintaining zero net torque at a joint (DeSerres and Milner 1991; Kearney and Hunter 1990; Milner and Cloutier 1998), and measures of muscle coactivation have been found to progressively decrease in conjunction with motor learning (Osu et al. 1999). At the same time, there is evidence based on measures of joint stiffness to suggest that in naturally occurring behaviors the control of coactivation and movement may be linked. Measures of variables related to coactivation, such as stiffness, have been reported during movements (Bennett 1993; Gomi and Kawato 1996, 1997; Latash and Gottlieb 1991a). Bennett (1993), in particular, has shown that in single-joint elbow movements increases in movement speed are accompanied by increases in stiffness. Similarly, in modeling studies, simulated commands for muscle coactivation must increase monotonically as a function of commands for movement velocity in order to increase speed and stiffness in parallel (Gribble et al. 1998).

The present paper reports a test of the idea that muscle coactivation varies with movement speed and hence that coactivation and movement control are related.

Since muscle coactivation is most readily quantified during static postures and is particularly evident at movement end, we have examined the pattern of muscle coactivation following movement and have related it to movement parameters and patterns of phasic muscle activity. Note that coactivation cannot be easily estimated during movement as a result of the co-occurring influence on muscle activation levels of phasic muscle activity, position dependent afferent input, and other reflexes (see "Discussion").

In the present study, we have measured muscle coactivation following movement in the context of single-joint shoulder and single-joint elbow movements (see Gribble and Ostry 1998 for a related procedure involving multi-joint movement). In order to determine the relationship of cocontraction to movement amplitude and velocity we have sampled a broad range of velocities at different amplitudes. Our analysis focuses on the patterns of tonic muscle activation following movements and the manner in which these are related to movement kinematics and patterns of phasic muscle activity.

Materials and methods

The experimental procedures used in these studies have been approved by the Ethics Committee of the Department of Psychology, McGill University.

Experimental set-up and task

Eight male subjects ranging in age from 27 to 52 years performed single-joint elbow movements and single-joint shoulder movements in a horizontal plane. The forearm was semiprone. Subjects were instructed to move to target locations that were specified by markers on the surface of a glass tabletop. The movement speed was established by providing subjects with a series of audio signals presented at different rates. The upper and lower arms were supported by air-sleds to minimize the effect of friction between the arm and tabletop. Subjects were told to make a single movement from the initial position to the target without corrections. They were told that the movement could be carried out using the shoulder or the elbow alone; however, nothing prevented motion of the other joint (see Gribble and Ostry 1999). Subjects were also instructed to complete the movement in the interval specified by the audio signals.

All combinations of two movement amplitudes (25° and 50°) and three average velocities (250°/s, 125°/s and 83°/s) were tested at each joint. The movements were chosen so that the final joint angles were the same. In the case of single-joint elbow movements, the final joint angles were 50° at the shoulder and 100° at the elbow. In the case of single-joint shoulder movements, the final angles were 70° at the shoulder and 80° at the elbow. Shoulder angles were defined relative to the frontal plane such that larger values corresponded to greater amounts of shoulder adduction. When the shoulder was aligned with the frontal plane, the shoulder angle was 0°. Elbow angles were defined relative to the upper arm. The angle was 0° when the arm was fully extended and increased with elbow flexion. Thus, single-joint elbow movements started from elbow angles of 75° and 50° with the shoulder at 50°. In the case of single-joint shoulder movements, initial shoulder angles were 45° and 20° with the elbow at 80°.

The trials were 5 s each in duration. This enabled data acquisition for a number of seconds both prior to the initiation of movement and following movement end. Elbow and shoulder movements were tested in separate blocks of trials. In each block, the six combinations of movement speed and amplitude (three speeds by two amplitudes) were randomized. Fifteen trials were collected consecutively in each treatment combination. The inter-trial interval was approximately 10 s.

Data collection and analysis

Movement kinematics were recorded at 200 Hz using Optotrak (Northern Digital), an optoelectronic position measurement system. Infra-red-emitting diodes (IREDs) were placed on the upper and lower arms (two on each limb segment). Two additional IREDs were placed on the clavicle near to the sternum to define a vector in the frontal plane. The kinematic data were low-pass filtered at 12 Hz using a second-order Butterworth filter. Elbow and shoulder angles were calculated using the vectors defined by the two points on each segment (the shoulder angle was calculated using the vector in the frontal plane and the vector defining the upper arm).

Electromyographic activity was recorded using Delsys double differential surface electrodes. Activity was measured from eight single- and double-joint shoulder and elbow muscles. The single-joint shoulder muscles were anterior deltoid and pectoralis clavicular head (both shoulder flexors) and posterior deltoid, a shoulder extensor. The double-joint muscles, which act at both the shoulder and the elbow, were biceps short head and triceps long head. The elbow muscles were the elbow flexors biceps long head (a two-joint muscle that acts primarily at the elbow; see Yamaguchi et al. 1997) and brachioradialis (a single-joint elbow flexor) and the single-joint elbow extensor triceps lateral head. A series of test maneuvers involving free movement and isometric force adjustments were carried out to verify the electrode placements.

For all muscles, EMG activity was analog low-pass filtered at 600 Hz and then digitally sampled at 1200 Hz. The resulting signals were band-pass filtered between 30 Hz and 300 Hz and full-wave rectified.

For the purposes of obtaining measures of muscle coactivation, the data were aligned at movement end based on the tangential velocity of the distal IRED on the forearm, using a value of 15% of the peak tangential velocity for alignment (Gribble and Ostry 1998). Measures of muscle coactivation were obtained over a 100-ms window that started 200 ms following movement end. The first 200 ms was not analyzed to avoid any contribution of phasic muscle activity to the measured tonic cocontraction values. During the analysis period there was little movement of the shoulder or elbow. The maximum range of movement during this period averaged 0.35° for the shoulder and 0.37° for the elbow, across subjects. The associated average maximum velocity was 2.15°/s and 2.11°/s for the shoulder and elbow, respectively.

For each trial, a single mean value of tonic EMG activity was calculated for each muscle. In order to permit comparisons of EMG measures between muscles and across subjects, the EMG values were transformed to *z*-scores (see also Gribble and Ostry 1998). *z*-score values for each trial were computed using the tonic EMG level for that trial along with the mean and standard deviation of the tonic activity level over all trials and conditions for a given muscle. The normalization to *z*-scores had the effect of eliminating differences between the mean and standard deviation of tonic EMG among muscles and across subjects.

To verify that the results reported below were not due to the normalization procedure, the analyses of cocontraction level were repeated by normalizing tonic EMG levels to the maximum voluntary cocontraction level, which was recorded separately. The results were qualitatively similar to those reported below.

Coactivation measures were obtained by averaging the *z*-scores of antagonist muscles at each joint. To obtain a measure of coactivation at the shoulder, a weighted average of the *z*-scores of posterior deltoid, anterior deltoid and pectoralis was calculated. In order to represent flexor and extensor muscles equally in the coactivation measure, weights of 0.50, 0.25, and 0.25 respectively were used. Similarly, a weighted average of *z*-scores for biceps long head, brachioradialis and triceps lateral head was used to obtain a

measure of coactivation at the elbow. Again, the contribution of flexors and extensors was weighted equally, using weights of 0.25, 0.25 and 0.50 respectively. A coactivation measure for two-joint muscles was obtained by taking the average *z*-scores for biceps short head and triceps long head. It should be noted that the weighting schemes used here were chosen as a convenient simplification to represent the total activity about a joint. Selected analyses repeated on a per muscle basis show comparable patterns. Thus, other possible weightings – for example, ones in which muscles are represented in proportion to their contribution to total force or torque – give qualitatively similar results.

Trials were eliminated from the analysis if any antagonistic muscle pairs showed reciprocal patterns of activity during the measurement interval. Correlation coefficients were calculated (over the data points in the measurement interval) for all possible combinations of antagonistic muscles at each joint. In cases where any pair of muscles displayed a significant negative correlation ($P<0.01$), the trial was removed. Overall 17% of the data showed negative correlations and were thus removed from the analyses.

Measures of phasic EMG activity were also obtained for each muscle, using interactive computerized routines coded in Matlab. For each trial, the area associated with the first agonist burst was obtained by calculating the integral of the EMG signal between the start and end of the burst. A similar procedure was used to obtain a measure of antagonist activity. In addition, a measure was obtained of the area associated with agonist activity during the first 30 ms following agonist burst onset (see Corcos et al. 1989; Gottlieb et al. 1989).

The EMG values for phasic muscle activity were also transformed to *z*-scores (see above and see also Gribble and Ostry 1998). The calculation of *z*-scores was carried out for each muscle separately. For each trial, the area measure of EMG activity and also the area during the first 30 ms were transformed to *z*-scores. The transformations for each muscle were based on phasic activity measures over all trials and conditions. As in the transformation to *z*-scores carried out for the tonic activity data, this normalization equated the mean and standard deviation of phasic EMG activity among muscles and across subjects.

Results

Tonic EMG and kinematics

The muscle coactivation measures were used to assess the relationship between tonic EMG levels and the kinematics of the preceding movement. Figure 1 shows average kinematic and EMG data for a single-joint shoulder movement from a single subject. The data are for the small amplitude condition at a medium speed; however, they are typical of the data observed in other conditions. Phasic muscle activity patterns can be seen in shoulder and elbow muscles followed by a sustained tonic level at the end of the movement. The vertical bars at the right of the figure indicate the region used for calculation of the coactivation measures.

Figure 2 shows the relationship between tonic EMG levels, movement amplitude and maximum velocity for a single subject. The upper panel gives an example of EMG activity in elbow muscles following single-joint elbow movement. The lower panel shows tonic EMG activity in shoulder muscles following single-joint shoulder movement. Filled circles are for large amplitude movements and open circles are for small amplitude movements.

The figure displays a number of key characteristics of the present data. At each movement amplitude, tonic

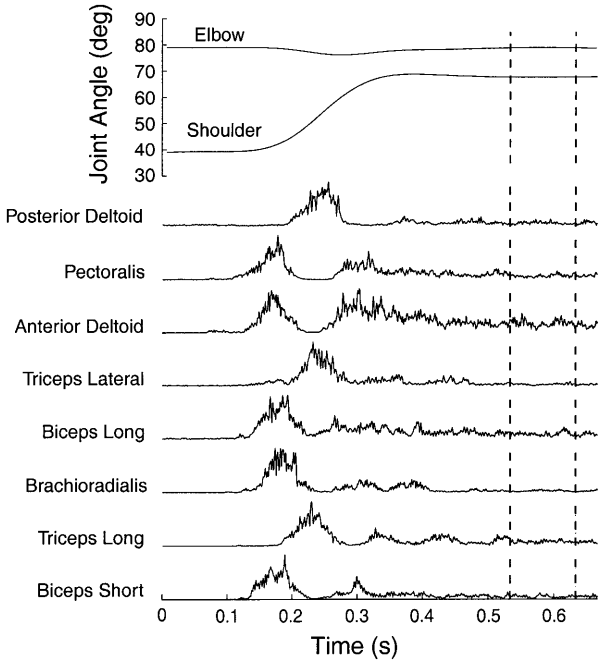

Fig. 1 Mean EMG activity and movement amplitude based on single-joint shoulder flexions for a single subject. Data for the small amplitude, medium speed condition are shown. *Vertical bars at the right* show the region following movement used for calculation of tonic EMG levels

EMG increases with movement velocity. The maximum tonic EMG level observed is typically equal for large and small amplitude movements. However, the range of velocities at the two amplitudes differs. The maximum velocity in the smaller amplitude condition is approximately half that observed in the larger amplitude movements. Thus the velocity at which the maximum EMG is observed is also different for the two amplitudes. In effect, the range of velocities over which the full range of tonic EMG change is observed is compressed for smaller amplitude movements.

It may also be observed that movement amplitude can have as great an effect on tonic EMG levels as movement velocity. Specifically, when the movements are equated for velocity – for example, at a maximum velocity of 300°/s in the case of elbow movements – tonic EMG scores at the two different amplitudes show as great a difference in magnitude as is observed over the range of velocities in each condition. In particular, as movement amplitude increases, tonic EMG levels decrease.

Measures of correlation were computed on a within subject basis between tonic EMG level and movement velocity at each of the amplitudes tested in this study. Of the eight subjects tested, six showed reliable correlations between tonic EMG levels and movement velocity for single-joint elbow muscles in elbow movement, for single-joint shoulder muscles in shoulder movement, and for two-joint muscles in elbow and shoulder movements ($P<0.001$ in all but three cases where $P<0.05$). For the

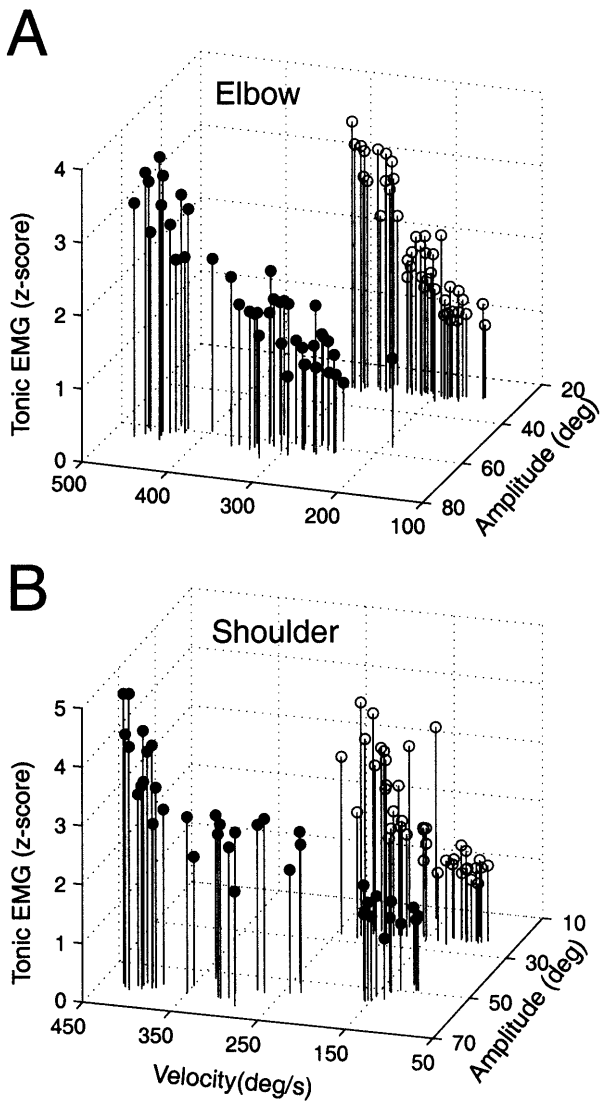

Fig. 2 Tonic EMG activity showing individual trials for single-joint elbow muscles (elbow movement, **A**) and single-joint shoulder muscles (shoulder movement, **B**). EMG activity was normalized to *z*-scores; weighted averages of *z*-scores in antagonist muscles at each joint are shown (see text). *Open circles* indicate small amplitude movements, *filled circles* show large amplitude movement. At both joints, muscle coactivation as reflected by tonic EMG activity increases as a function of movement velocity. For purposes of visualization, a constant (+2) was added to all *z*-scores so that the figure displays only positive values

remaining two subjects, reliable correlations between tonic EMG and velocity were observed for large amplitude movements only ($P<0.001$ in all cases).

Mean values of tonic EMG averaged across subjects are shown in Fig. 3. The figure gives results for single-joint muscles acting at the elbow and double-joint muscles, in the case of elbow movements (upper panels) and single-joint muscles acting at the shoulder and double-joint muscles in shoulder movements (lower panels). A standard error of ±1 is shown for both velocity and EMG measures. It can be seen that the dependence of tonic EMG on movement kinematics is generally similar in shoulder and elbow movements. In all cases, tonic EMG increases with movement velocity. A similar dependence of EMG on velocity is seen at both movement amplitudes.

In conducting statistical analyses, it is important to recognize that the two amplitudes and three velocity conditions tested here do not constitute a fully crossed experimental design in terms of the actual amplitudes and velocities produced by subjects. Whereas subjects were able to produce the desired movement amplitudes with little difficulty, the associated range of movement velocities were amplitude dependent, and not equal in the two amplitude conditions. Maximum velocities ranged from about 100° to 200°/s for smaller amplitude movements and from 200° to 400°/s for larger amplitude movements. The fastest movements at each amplitude were effectively the fastest possible for the subjects in that condition. A fully crossed statistical design is thus precluded by the combination of speeds and amplitudes that subjects are capable of producing in these conditions.

In order to examine the dependence of tonic EMG on both movement amplitude and velocity, statistical analyses were carried out as shown in Fig. 4. Three separate Analyses of Variances (ANOVAs) assessed the dependence of tonic EMG on movement velocity for larger amplitude movements (test 1), the dependence of EMG on velocity for smaller amplitude movements (test 2), and the dependence of EMG on movement amplitude using those cases where velocity was equal but amplitude differed (test 3).

At each movement amplitude, between subjects ANOVAs were carried out to evaluate differences in tonic EMG as a function of movement velocity (tests 1 and 2). Separate tests were conducted for single- and double-joint muscles in the elbow-alone and shoulder-alone movement conditions. In each case, tonic EMG levels were found to vary significantly with velocity ($P<0.01$).

Differences in tonic EMG as a function of movement amplitude were tested by comparing the EMG levels of the fastest small amplitude movements with the slowest large amplitude movements. This enabled us to equate for velocity and to examine the effects of amplitude on EMG (see test 3 in Fig. 4). Separate ANOVAs were conducted for single- and double-joint muscles in both the single-joint elbow and single-joint shoulder manipulations. In all cases, tonic EMG levels varied significantly with movement amplitude ($P<0.01$). For the same movement velocity, as amplitude increased, there was a significant decrease in tonic EMG.

The analyses thus indicate a dependence of tonic EMG following movement on both movement amplitude and velocity. We have assessed in addition the possibility that tonic EMG might be related to a single variable such as acceleration or its higher derivatives. The rationale for these tests was that tonic EMG might reasonably be expected to play a role in stabilizing the limb following movement and thus to vary with factors such as limb acceleration.

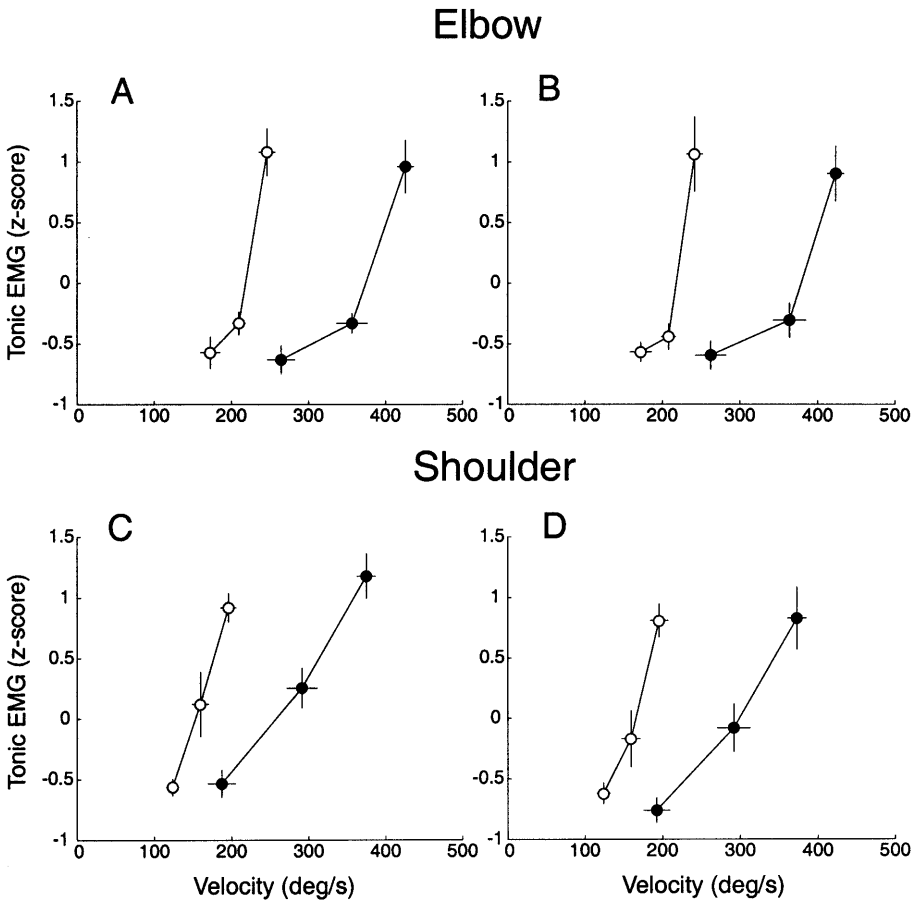

Fig. 3 Tonic EMG activity averaged across subjects in single-joint elbow movements – single-joint elbow muscles **(A)** and double-joint muscles **(B)** and single-joint shoulder movements – single-joint shoulder muscles **(C)** and double-joint muscles **(D)**. *Open circles* are for small amplitude movements, *filled circles* are for large amplitude movements. Tonic EMG can be seen to increase with movement velocity at each amplitude separately for both single- and double-joint muscles at the shoulder and the elbow

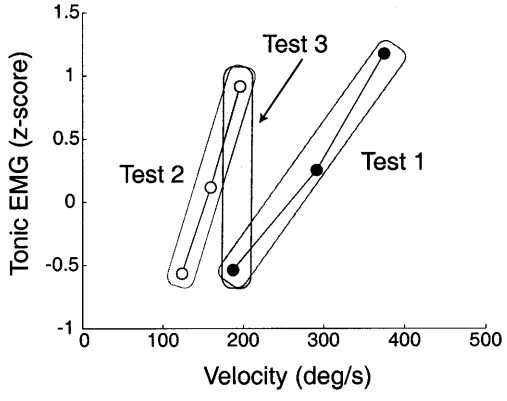

Fig. 4 ANOVA was used to assess the sources of variation in tonic EMG measures. The tests evaluated the dependence of tonic EMG activity on movement velocity for large amplitude movements (test 1), the dependence of tonic EMG on velocity for small amplitude movements (test 2), and the dependence of tonic EMG on movement amplitude, under conditions equated for velocity (test 3, *shaded box*). In this final test, the EMG measures for the fastest small amplitude movements were compared with values for the slowest large amplitude movements

Accordingly, we have examined the relationship between tonic EMG and factors such as peak acceleration, peak deceleration and peak jerk. We have also considered the relationship of tonic EMG to average values of velocity, acceleration and jerk. In all cases, the analyses yielded results of the same general form as that shown in Fig. 3, where differences in tonic EMG due to movement amplitude were found along with differences due to velocity or its higher derivatives. We were unable to identify any single unifying variable that on its own might account for the patterns of tonic EMG seen here.

We have taken a number of precautions to ensure that the patterns reported above reflect tonic activity alone and are not a by-product or carryover of phasic muscle activation. As noted in “Materials and methods,” the data on which coactivation measures were based was taken over an interval of 100 ms beginning at a point in time 200 ms following the end of movement as defined kinematically. We have, in addition, eliminated from the analysis any trials in which a statistically significant negative correlation was observed between the EMG magnitudes of any pair of antagonist muscles. As a further test, we have recalculated the measures of muscle coactivation, in the interval from 500 ms to 600 ms following movement end. The patterns presented in Fig. 3 are essentially unchanged by this procedure. Beyond 600 ms following movement end it was no longer possible to obtain reliable measures of coactivation.

The data on muscle activation following movement were obtained using air-sleds to support the limb. In order to verify that the patterns of coactivation were not a consequence of the use of the air-sleds – for example, as

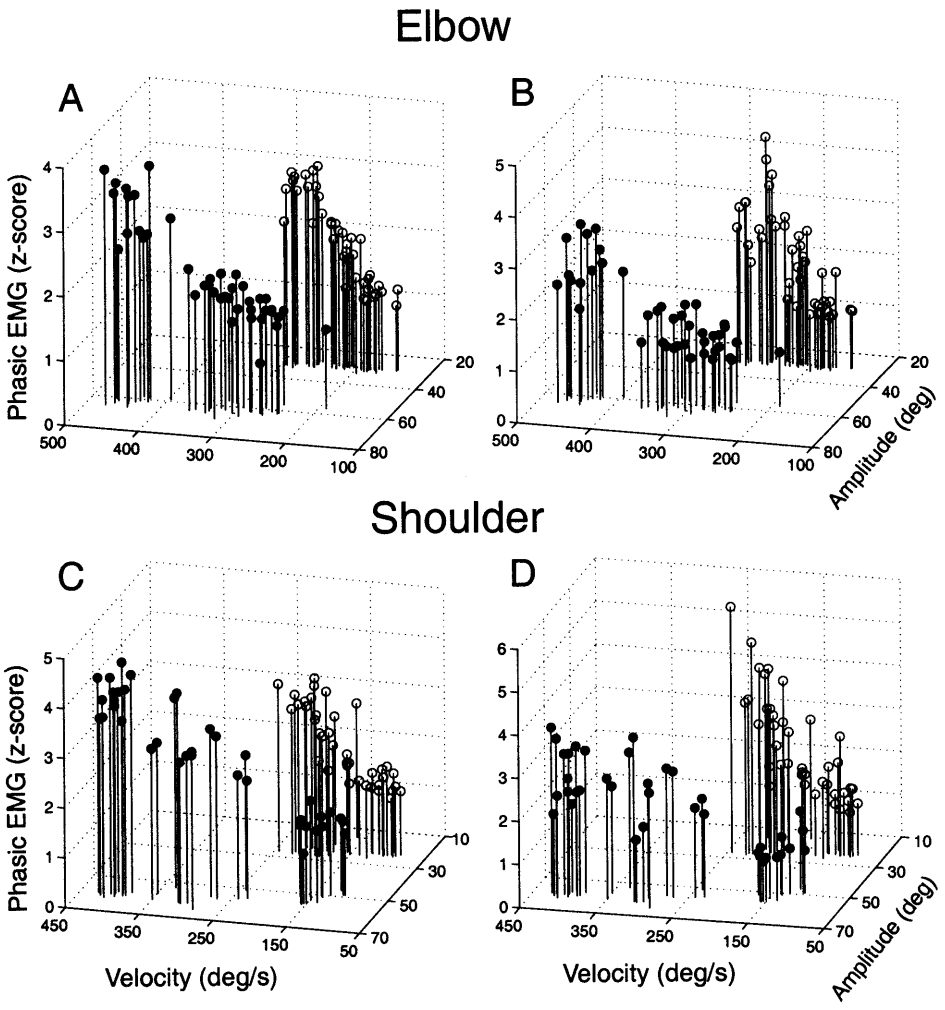

Fig. 5 Phasic EMG activity giving individual trials for single-joint elbow movements **(A, B)** and single-joint shoulder movements **(C, D)**. *Open circles* are for smaller amplitude movements, *the filled circles* are for larger amplitude movements. **A** and **C** give measures of total phasic EMG activity (see "Materials and methods"). **B** and **D** give phasic activity during the initial 30 ms of activation in biceps brachii for elbow movements (*top*) and the clavicular head of pectoralis for shoulder movements (*bottom*). As an aid to visualization, a constant (+2) has been added to all *z*-scores so that all values are positive

a strategy used by subjects to improve accuracy in the low-friction environment – a control study was carried out without air-sleds. Subjects produced single-joint shoulder movements and single-joint elbow movements in a horizontal plane such that the limb did not make contact with the table surface during the movement. Surface EMG activity was measured in shoulder muscles (anterior and posterior deltoid) and elbow muscles (biceps long head and triceps lateral head) respectively. A single movement amplitude (50°) was tested at each of three velocities – the same three average velocities tested above. Three subjects participated in the control study.

The procedure used for data analysis was the same as in the study with air-sleds. As in that study, the EMG values for tonic muscle activity were transformed to *z*-scores, based on the interval from 200 to 300 ms following movement end. ANOVA was used to assess the effects of movement velocity on tonic EMG.

ANOVA indicated that, in the absence of air-sleds, muscle coactivation following movement varied with movement velocity in same manner as described above and illustrated in Figs. 2 and 3. Specifically, coactivation in single-joint shoulder muscles increased significantly with shoulder velocity ($P<0.01$ for all three subjects). Coactivation in single-joint elbow muscles increased significantly with elbow velocity ($P<0.01$ for all subjects).

A direct comparison was not undertaken of the results of the studies conducted with and without airsleds. Differences in the subject sample, the placement of the surface EMG electrodes and the conversion of the data to normalized *z*-scores each precluded a quantitative comparison of the results of these studies.

Phasic EMG

We also examined the relationship between phasic EMG, tonic EMG and movement kinematics. Figure 5 shows a plot of the relationship between phasic EMG levels, movement amplitude and maximum velocity for the same subject shown in Fig. 2. The filled circles are for the larger amplitude movements and the open circles are for the smaller amplitude movements. Panels A and C give measures of total phasic EMG activity (see below) at the elbow and shoulder in single-joint elbow movement and single-joint shoulder movement respectively; panels B and D show phasic activity during the first 30 ms in biceps brachii for single-joint elbow move-

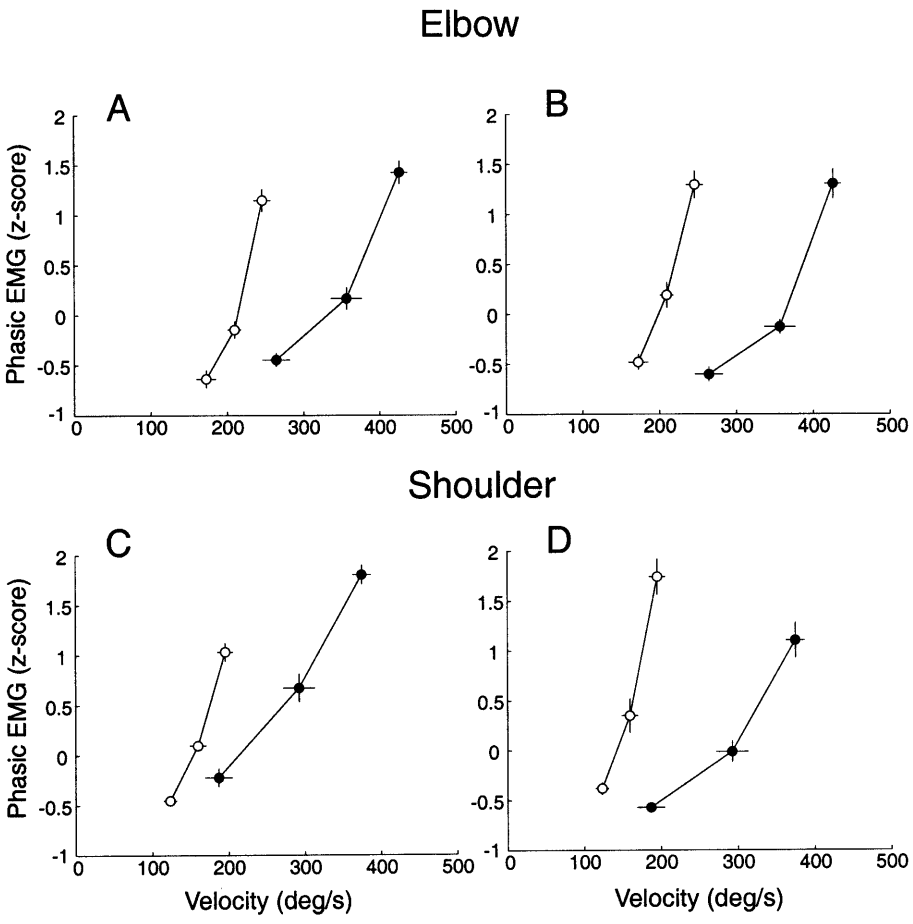

Fig. 6 Patterns of phasic muscle activity in single-joint elbow muscles during elbow movement **(A)** and single-joint shoulder muscles during shoulder movement **(C)**, averaged across subjects. A weighted average of activity in flexor and extensor muscles is shown (see "Materials and methods"). **B** and **D** give the phasic activity during the first 30 ms of activation averaged over subjects for biceps brachii **(B)** in elbow movement and pectoralis **(D)** in shoulder movement, respectively. In all cases, phasic EMG is shown as a function of movement amplitude and velocity

ments (B) and pectoralis (clavicular head) for single-joint shoulder movements (D) (see Corcos et al. 1989; Gottlieb et al. 1989).

In the left-hand panels (A and C), a weighted sum of *z*-scores of agonist and antagonist muscles is presented. The summation procedure is the same as that used for measures of tonic activity. In the right-hand panels (B and D), *z*-scores for individual muscles are shown. We have presented sums (in panels A and C) rather than the magnitudes of agonist or antagonist activity separately because the magnitudes of phasic activity for agonist and antagonist muscles were similar. The sum is used here to provide a composite measure of overall phasic activity.

The relationship shown in panels A and C between phasic EMG activity (total activity), velocity and amplitude is similar to that presented above in Fig. 2 for tonic activity. At each amplitude, there is a systematic increase in phasic EMG with increases in movement velocity. Similarly, the maximum phasic activity level for large and small amplitude movements is comparable. Note that a similar relationship between phasic muscle activity and velocity and movement amplitude has been described previously for single- and multijoint movement (Gribble and Ostry 1999).

We have also examined the activity of agonist muscles during the first 30 ms of activation. Any activity observed during this interval presumably reflects central activation alone and precludes contributions due to voluntary correction and position dependent afferent input and other reflexes. Panels B and D of Fig. 5 give the pattern of phasic activity during this 30-ms interval.

Weighted averages of total phasic muscle activity across subjects are shown in Fig. 6, panels A and C. The values given in the figure were computed in the same way as the values for tonic activity. That is, the *z*-scores for the two flexor muscles and one extensor muscle at each joint were weighted 0.25, 0.25 and 0.50 respectively. Panels B and D show activity from elbow and shoulder agonist muscles during the first 30 ms of activation. The patterns of phasic activity can be seen to resemble those of tonic muscle activity in Fig. 3. In both cases, EMG activity increases monotonically with movement velocity in each movement amplitude condition separately. One difference is that whereas maximum EMG levels tend to be similar for different movement amplitudes in the case of tonic activity, for phasic activity the maximum levels at the shoulder differ.

Statistical tests comparable to those carried out for tonic activity were repeated for the phasic measure to assess the dependence of phasic activity on amplitude and velocity (see Fig. 4). Separate ANOVAs tested the dependence of phasic activity on velocity for larger (test 1) and smaller amplitude movements (test 2) as well as the dependence of phasic activity on amplitude under condi-

tions where velocities were similar but amplitude differed (test 3).

Between subjects ANOVAs were conducted for the elbow-alone and shoulder-alone conditions (mean values are shown in Fig. 6). In all cases, phasic EMG levels increased systematically with movement velocity ($P<0.01$). Similarly, phasic activity differed as a function of movement amplitude under comparable velocity conditions ($P<0.01$ in all cases). As amplitude increased, a reliable decrease in phasic EMG activity was observed. ANOVA was also conducted to assess differences in EMG activity during the first 30 ms of activation. The tests paralleled those carried out for phasic EMG signals but examined the activity patterns of agonist muscles alone. The pattern of results was similar to that obtained for the phasic signal as a whole. EMG levels increased significantly with velocity at both movement amplitudes ($P<0.01$) and differed as a function of amplitude when velocity was constant ($P<0.01$ in all cases).

We also explored the possibility that a single variable, such as peak acceleration or jerk or average values of velocity, acceleration or jerk might account for the pattern of phasic activity observed here. As in the case of tonic activity, phasic measures were systematically related to each of the higher derivatives (peak values and mean values). However, the dependence of this pattern on differences in amplitude was in all cases preserved.

In order to examine the relationship between phasic and tonic muscle activity patterns, Pearson product moment correlation coefficients were calculated between phasic and tonic scores. Figure 7 shows scatter plots of the relationship between total phasic activity and tonic activity measures on a trial-by-trial basis for the same subject as shown in Figs. 2 and 5. As in other figures, weighted averages of activity in agonist and antagonist muscles are presented. The pattern for single-joint elbow muscles is shown in the upper panel. Single-joint shoulder muscles are shown below. Data for larger amplitude movements are represented by filled circles.

It may be seen that tonic and phasic EMG measures are highly correlated ($r=0.89$ at the elbow and $r=0.85$ at the shoulder, $P<0.01$ in both cases). Correlation coefficients were calculated on a per subject basis to examine the relationship between tonic measures and total phasic activity as well as between tonic EMG and phasic activity during the first 30 ms of muscle activity. In the case of the correlation between total phasic activity and tonic activity, values of the correlation coefficient averaged $r=0.85$ across subjects for elbow muscles and $r=0.70$ for shoulder muscles. The correlations between tonic activity and phasic scores during the first 30 ms averaged $r=0.74$ at the elbow and $r=0.63$ at the shoulder. The correlations for all subjects were significant ($P<0.001$) with the exception of the data for one subject whose results were non-significant at the shoulder.

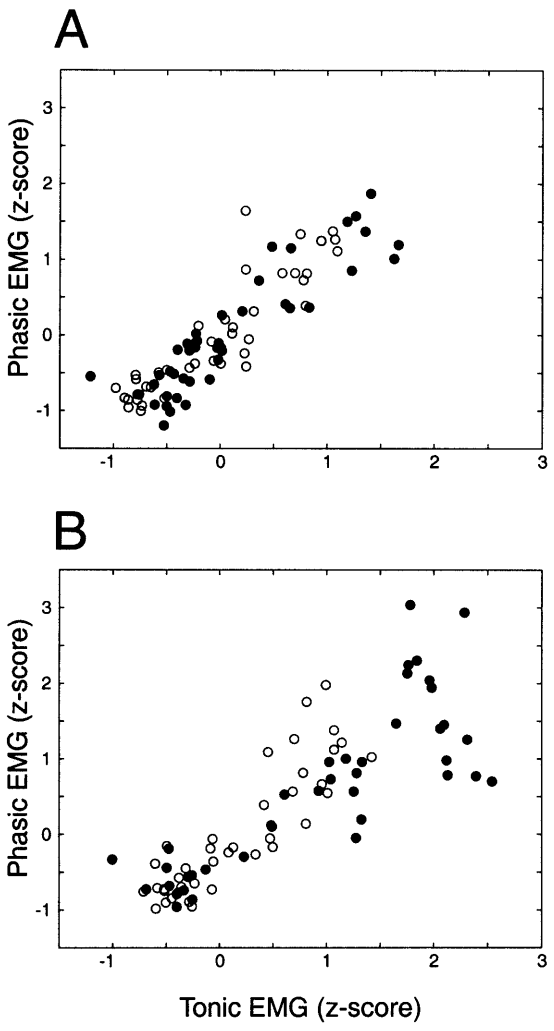

Fig. 7A, B Scatterplot showing the relationship between phasic and tonic activation levels on a trial-by-trial basis for a single subject (same subject as in Figs. 2, 5). EMG values are based upon weighted averages of activity in single-joint antagonist muscles. **A** gives data for single-joint elbow muscles in elbow movement and **B** shows data for single-joint shoulder muscles in shoulder movement

Discussion

A similar pattern relating EMG activity to kinematic variables has been found to characterize muscle coactivation following movement, phasic activity in these same muscles during movement, and phasic muscle activity patterns at the initiation of movement. In each case, EMG magnitudes increased as a function of velocity at two movement amplitudes. The similarity of the phasic and tonic activation patterns suggests that the nervous system may use a relatively simple procedure to modify coactivation in which the signals that determine tonic EMG activity are scaled in relation to those that underlie the phasic signal. In addition, since the patterns of coactivation were found to mirror phasic activity within the first 30 ms of the initial agonist burst, the con-

trol of coactivation may be determined centrally prior to movement onset.

There is evidence of coactivation control from both behavioral and physiological studies. Behavioral evidence includes the ability of subjects to coactivate antagonist muscles while maintaining zero net joint torque (DeSerres and Milner 1991; Kearney and Hunter 1990; Milner et al. 1995; Milner and Cloutier 1998) and to independently vary the magnitudes of coactivation and reciprocal activity (Yamazaki et al. 1994, 1995). The presence of motor units that display "common drive" to antagonist muscles is consistent with the idea of centrally specified coactivation of antagonists (DeLuca and Mambrito 1987; Nielson and Kagamihara 1994). Supporting evidence from electrophysiological studies comes from recordings of single neurons in motor cortex and cerebellar cortex that are active in relation to the coactivation of antagonists but not during reciprocal activation (Frysinger et al. 1984; Humphrey and Reed 1981). In addition, intracortical microstimulation has shown that neurons in motor cortex and red nucleus project to antagonist muscle targets at the periphery (see Fetz et al. 1989 for summary).

These various examples are consistent with the idea that coactivation and movement may be controlled separately. Nevertheless, the present study suggests that, in naturally occurring behaviors, the control of coactivation and movement may be linked. Findings of other recent studies are consistent with this view. Bennett (1993), for example, has provided empirical evidence that joint stiffness during movement increases in proportion to movement speed. In order to replicate these findings in modeling studies, simulated commands for muscle coactivation must increase monotonically as a function of commands for movement velocity (Gribble et al. 1998).

The present findings may appear to be at odds with equilibrium control schemes for movement production (Feldman et al. 1990) and in particular with the postulated independence of central control signals responsible for limb movement and muscle coactivation. It should noted, however, that although the equilibrium point formulation provides for the possibility of independence, it in no way precludes the coordinated use of these commands. Moreover, it should be emphasized that quantitative formulations of the equilibrium point model offer a means to explore the form of control signals and the coordination of commands needed to simulate empirical results (see, for example, Gribble et al. 1998). In this regard, the model can serve as a tool to uncover relationships such as that observed in the present study between movement velocity and muscle coactivation. Indeed, it may be noted that results comparable to those reported in the present study have been described previously in the context of the model (Gribble et al. 1998).

The similarity of patterns of tonic EMG activity following movement to patterns of phasic activity during the initial 30 ms of activation are consistent with the possibility that coactivation is specified prior to movement onset. The initial portion of the first agonist burst is the first detectable event associated with voluntary movement commands and since it occurs before movement onset, it is presumably a reflection of the central input in which influences due to reflexes and voluntary correction are minimized (Corcos et al. 1989; Gottlieb et al. 1989). During this initial period there is a systematic relationship at each movement amplitude between phasic EMG activity and movement velocity. This relationship is mirrored in comparable relationships between total agonist activity and velocity as well as between coactivation following movement and velocity. The similarity between the pattern of activity in the first 30 ms and tonic EMG activity at movement end thus suggests that the tonic signal may be planned in association with the signals that result in the initial agonist burst.

The muscle coactivation levels measured following the fastest movements – which were effectively executed at subjects' maximum speed at those distances – presumably reflect the highest levels of coactivation that naturally accompany voluntary movement. The activation levels were nevertheless far less than the associated levels of phasic activity in the same muscles during the preceding movements. Milner et al. (1995) have similarly shown that activation was less during maximum cocontraction than during reciprocal activation of the same muscles (see also Tyler and Hutton 1986). Milner et al. (1995) suggest that the lower levels of activity during cocontraction may arise due to peripheral mechanisms such as reciprocal inhibition from group Ia afferents or centrally through pathways activated during coactivation.

In order to assess the determinants of coactivation, we have manipulated movement amplitude and average velocity – average velocity was held constant for movements of different amplitude. It should be noted, however, that in so doing subjects produced movements in which equivalent velocities represented different percentages of the peak velocity subjects could achieve over different distances (Gottlieb et al. 1989). Under conditions where movement is less constrained, average velocity, maximum velocity and movement amplitude all covary. Thus, a limitation of the present experiment in terms of its attempt to document the determinants of coactivation and their relation to phasic muscle activity is the difficulty inherent in having measures of amplitude and velocity that are well controlled experimentally but at the same time are typical of naturally occurring movements.

In the present study, we have examined muscle coactivation following movement as a means to explore the relationship between postural stabilization and the control of movement. Direct measures of coactivation during movement would be desirable as would measures of stiffness following movement that could be related to the present measures of coactivation. Measures of stiffness following movement could be obtained in the context of limb perturbation studies. However, direct estimates of coactivation during movement would be difficult to obtain since the neural signals that regulate muscle coacti-

vation co-occur with those associated with phasic muscle activity and activation arising due to reflexes. Gomi and Kawato (1996) have reported measures of stiffness during movement. Stiffness is higher during movement than in statics and the pattern of joint stiffness during movement has a non-monotonic M-shaped pattern that reflects the timing of phasic and tonic muscle activation, afferent inputs and limb geometry (Gribble et al. 1998). Measures such those reported by Gomi and Kawato (1996) in conjunction with a physiologically realistic model of the limb might be a first step in a model-based decomposition of the EMG signal needed to obtain estimates of coactivation during movement.

Tonic EMG levels were assessed using a 100-ms window beginning 200 ms after movement end. An analysis carried out 500 ms after movement end revealed a qualitatively similar pattern of results. Coactivation following movement could be measured to a maximum of about 600 ms under the conditions of this study. It should be noted that data were recorded for approximately 2 s following movement end and thus the present estimate on the duration of cocontraction following movement is not limited by the duration of the recording interval.

Tonic muscle activity beyond movement end presumably contributes to stabilizing the position of the limb. We have suggested that the pattern of coactivation may also be related to a coactivation signal that accompanies movement. However, the evidence for this is indirect. Additional indirect evidence for a link between movement velocity and coactivation during movement is provided by Bennett (1993), who reports that stiffness is higher over the course of faster movements. As we suggest above, the extent to which coactivation measured at movement end reflects coactivation control during movement will likely involve a model based solution.

We have previously investigated muscle coactivation in the context of multijoint movement (Gribble and Ostry 1998). We found that coactivation at the shoulder and elbow varied in proportion to movement amplitude and velocity. A number of additional aspects of coactivation control were described. In particular, it was found that coactivation may be specified separately at the shoulder and elbow – tonic coactivation of shoulder muscles varied in proportion to the amplitude and velocity of shoulder movement but was unrelated to elbow motion, whereas coactivation of elbow and double-joint muscles varied with elbow movement and was not correlated with shoulder motion. An additional determinant of coactivation was the direction of joint rotation. Coactivation was higher when the joints rotated in the same direction (and interaction torques at the shoulder were high) and lower when joints rotated in opposite directions (and interaction torques were low). The present study has focused on coactivation in single-joint movement in order to have better experimental control over movement amplitude and velocity. In future studies it would be desirable to systematically examine movement kinematics and associated phasic and tonic activity in the context of controlled amplitude and velocity variation in multijoint movement.

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