Electromyographic Responses to an Unexpected Load in Fast Voluntary Movements: Descending Regulation of Segmental Reflexes

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1School of Kinesiology, University of Illinois at Chicago, Chicago, Illinois 60608; 2Neuromuscular Research Center, Boston University, Boston, Massachusetts 02215; 3Department of Epidemiology and Biostatistics, Norman J. Arnold School of Public Health, University of South Carolina, Columbia, South Carolina 29208; 4Department of Neurological Sciences, Rush-Presbyterian St. Luke’s Medical Center, Chicago, Illinois 60612; 5Departments of Bioengineering, Physical Therapy, and Psychology, University of Illinois at Chicago, Chicago, Illinois 60612

Received 19 September 2001; accepted in final form 18 April 2002

Shapiro, Mark B., Gerald L. Gottlieb, Charity G. Moore, and Daniel M. Corcos. Electromyographic responses to an unexpected load in fast voluntary movements: descending regulation of segmental reflexes. J Neurophysiol 88: 1059–1063, 2002; 10.1152/jn.00776.2001. This study examined the effects of unexpected loading on muscle activation during fast goal-oriented movements. We tested the hypothesis that the electromyographic (EMG) response to an unexpected load occurs at a short latency after the difference between the expected and the unexpected movement velocity exceeds a fixed threshold. Subjects performed two movement tasks as follows: 1) 30° fast elbow flexion movement with an inertial load added by a torque motor; and 2) 50° fast elbow flexion movement with no added load. These movement tasks were chosen to have similar timing parameters, such as movement time, time-to-peak velocity, and duration of the first agonist burst, while the magnitudes of the angular displacement, velocity, and acceleration were different. In task 1, in random trials a viscous load was substituted for the inertial load at movement onset. In task 2, the same viscous load was added in random trials. The earliest consistent response to the unexpected load was detected in the agonist (biceps) EMG at the same time, about 200 ms from the EMG onset, in both tasks. However, the velocity errors were different in the two tasks and no velocity error threshold dependency could be found. Therefore we reject the hypothesis that the timing of the EMG response to an unexpected load is related to a velocity error threshold. Instead, we suggest that the timing of the EMG response is primarily determined by descending regulation of segmental reflex gain.

INTRODUCTION

Muscle activation patterns during movement depend on the load (Gottlieb 1996). When moving a known load, it is possible for subjects to use an internal model that allows the motor system to generate control signals based on the expected dynamic requirements of the impending movement (Flanagan and Wing 1997; Shadmehr and Mussa-Ivaldi 1994). In everyday life, however, the parameters of a load are rarely known exactly and it is generally believed that muscle activation emerges as a combination of predictive central control and sensory feedback (Latash 1994; Sainburg et al. 1999) but there is considerable controversy as to how sensory feedback is used.

An unexpected load alters a movement’s trajectory and the muscles’ activation patterns. What determines the timing of the changes in muscle activation? Hallett and colleagues (1975) observed that the first agonist and antagonist bursts were not modifiable. Thus they concluded that corrections involved the descending responses from higher centers and could not be accounted for by purely segmental pathways.

Another hypothesis is that the timing of electromyographic (EMG) responses is related to movement velocity. Smeets and colleagues (1990, 1991) suggested that the segmental stretch reflex can produce responses 25–40 ms after the velocity differences between expected and unexpected movements exceed a threshold value. The present experiment tests the threshold dependency of the timing of EMG responses.

METHODS

Subjects

Eight neurologically normal volunteers aged 22 to 44 years old performed fast point-to-point, single-joint elbow flexion movements in a horizontal plane. All subjects gave informed consent according to a University-approved protocol.

Apparatus

The apparatus included a motor-driven manipulandum that consisted of a metal bar freely rotating in a horizontal plane around a pivot. The seated subject abucted the shoulder 90° and rested the forearm on the bar with the elbow joint aligned with the pivot. A computer monitor in front of the subject showed markers indicating the initial and target positions, and a cursor indicating the current joint angle.

The torque motor simulated an additional inertial or viscous load by generating resistive torque proportional to the angular acceleration or velocity of the bar. Torque was measured by a transducer inserted between the pivot and motor shaft. Additional details may be found in Gottlieb (1996). Surface EMGs in biceps, brachioradialis, and long and lateral heads of triceps were measured using a Bagnoli system (Delsys). EMG signals were digitally full-wave rectified and low-pass filtered with a dual-pass Butterworth filter with 50-Hz cutoff.

Experimental protocol

Subjects aligned the cursor with the initial position marker and made fast elbow flexions to the target in response to a “GO” beep.
waited for the “END” beep, and moved back to the initial position. The computer turned a load on simultaneously with the GO beep and turned the load off with the END beep. The instruction was “move as fast as possible, try to hit the target but do not correct if you miss it.”

Subjects performed two different movement tasks. The task order was randomized across the subjects. Task 1 was a fast, 30° flexion movement with a 0.18 kgm² inertial load added to the manipulandum by the torque motor. Task 2 was a fast 50° flexion movement with no added load. These movement tasks were chosen to have similar timing parameters, such as movement time, time-to-peak velocity, and duration of the first agonist burst, while the magnitudes of the angular displacement, velocity, and acceleration were different.

In each task, subjects made 30–50 practice trials and then made a series of 60 movements. In 12 randomly chosen trials, the motor applied an unexpected viscous load of 3.6 Nms/rad. In task 1 the inertial load was unexpectedly replaced with the viscous load, while in task 2 the viscous load was simply added. Thus a combination of the unexpected viscous load and manipulandum inertia of 0.1 kgm² was the same in both tasks. The unexpected load altered the trajectory and produced a “velocity error,” defined as the difference between the velocities of movements with the expected and unexpected loads. The velocity error is negative when the limb moves faster than expected and positive when the limb moves slower than expected. This experimental design produced unexpectedly loaded movements that should have very different velocity errors in the two tasks.

Data analysis

In the analysis of 48 trials against the expected load in each condition, we excluded atypical trials which included between two and three of the shortest, longest, slowest, and fastest trials, usually 8–12 trials total. All 12 trials against the unexpected load were accepted. The data were aligned on the biceps (agonist) EMG onset. For the purpose of illustration, the data were averaged.

The timing of the EMG response to the unexpected load was determined as follows. Every 2 ms, the EMG values from the individual trials against the expected (n = 36–40) and unexpected loads (n = 12) were compared using Satterthwaite’s modified t-test (Armitage and Berry 1994). The test was repeated every 2 ms for t = 0–0.6 s to generate a P-value time series (Fig. 1E). The EMGs in movements with expected and unexpected loads were considered statistically different if P fell below 0.05 and remained there for ≥10 ms. In addition, the sign of the difference in the EMG was checked against the sign of the velocity error 30 ms prior to the onset of this EMG difference. For example, a reflex decrease in the agonist activity should result from a negative velocity error. Therefore although a small, short decrease in the biceps EMG in task 1 at about 0.19 s (Fig. 1, D and E, left) was statistically significant, it was not considered an EMG response to the unexpected load because the velocity error had been positive for about 30 ms (Fig. 1B, left, and Fig. 2A).

In the remainder of the article, all time values are reported with respect to the biceps EMG response to the unexpected load unless explicitly stated otherwise.

RESULTS

When the unexpected viscous load is applied in task 1, the resistive torque is proportional to velocity and initially lags behind the torque proportional to acceleration that is expected by the subject (Fig. 1C, left). This causes the limb to initially move faster than expected (Fig. 1B, left) until after 0.16 s when the unexpectedly loaded movement slows considerably. In contrast, in task 2 the unexpectedly loaded movement is slower than expected (Fig. 1B, right) because the resistive torque is larger for the entire distance (Fig. 1C, right). As predicted, the two velocity errors are quite different (Fig. 2A).

The initial relative acceleration of the limb in task 1, which is reflected in the negative velocity error (starting at 0.07 s in Fig. 2A, also seen in Fig. 1B, left), should lead to reflex

FIG. 1. Representative movements with expected (solid lines) and unexpected (dotted lines) loads: angle (A), velocity (B), applied motor torque (C), and muscle EMG (D) for 1 subject in the 2 tasks. The antagonist (long head of triceps) EMG traces in D are inverted. E: results of Satterthwaite’s modified t-test for biceps; horizontal line P = 0.05 indicates the threshold for statistical significance. Averaged data for 1 subject are shown. The arrows mark the time (0.220 and 0.212 s) of the biceps EMG response in 2 tasks.

FIG. 2. A: velocity errors for 1 subject (same subject as in Fig. 1) calculated as the difference between the averaged expected and the unexpected velocities. The arrows mark the time of the biceps EMG response in 2 tasks. B: velocity errors for 7 subjects. C: the same velocity errors realigned on the time of the biceps EMG response for each subject. Time 0 in A and B indicates the biceps EMG onset, while time 0 and arrow in C indicate the onset of response in biceps EMG. Horizontal lines indicate a threshold value 0.6 rad/s of the velocity error (Smeets et al. 1990). No threshold dependence of the timing of EMG response can be seen at 0.6 rad/s or other levels of the velocity error.
inhibition of the agonist muscle and facilitation of the antagonist. Such a decrease in biceps activity occurred in only one of eight subjects at about 0.12 s (not shown). It was followed by an increase in the biceps EMG in response to the load-induced relative slowing of the limb at 0.2 s. In the other seven subjects, the biceps EMG showed no statistically significant decrease in response to the initial relative acceleration of the limb (Fig. 1D, left). In these subjects, the earliest change in the biceps EMG was an increase in response to the load-induced relative slowing of the limb at about 0.2 s.

Similar to the biceps, in task 1 all eight subjects showed an increase in the brachioradialis (agonist) EMG and a decrease in the triceps (antagonist) EMG in response to the load-induced relative slowing of the limb at about 0.2 s (Table 1). In addition, some subjects showed an early decrease in the brachioradialis EMG (3 subjects) and/or increase in the EMG in the long head of triceps (2 subjects) and/or lateral head of triceps (3 subjects) in response to the initial relative acceleration of the limb. These responses occurred in 0.1- to 0.14-s interval, were small in magnitude, short in duration, and could not be related to a threshold of the velocity error.

In the case of task 2, the limb moved slower than expected from the movement onset (Fig. 1B, right) and the velocity error was always positive (Fig. 2A). Statistically significant differences between expected and unexpected EMGs were not found until about 0.2 s in all subjects and all muscles (Table 1, and Fig. 1, D and E, right).

We analyzed the velocity error traces based on the earliest response in the biceps EMG. We excluded the data of one subject in whom the earliest biceps EMG response in task 1 was a decrease at 0.12 s, while in the other seven subjects the earliest biceps EMG response was an increase at 0.2 s. For consistency, we excluded the data for this subject from the statistical analysis of the timing of the EMG responses in all muscles. In the analysis of the timing of the EMG response, we accepted the time of the EMG response to the relative slowing of the limb at about 0.2 s that was consistent across the subjects and across the muscles. We also repeated the analyses with the data from all eight subjects included, as well as with the time of the earliest EMG response accepted in all cases. The results were the same as presented below.

### DISCUSSION

We suggest that the change in muscle EMG observed at 0.2 s in both tasks was mediated by segmental reflexes and was not a voluntary or long-latency response. Consider task 1 in which the velocity error initially became negative. A voluntary or long-latency response would be a decrease in the biceps EMG at about 0.2 s. The observed increase in the biceps EMG at about 0.2 s, however (Fig. 1C, left), must have been a response to a positive velocity error. The velocity error changed sign at about 0.15 s (Fig. 2A) so that the biceps EMG response occurred within 50 ms after the velocity error became positive. This short time indicates that the observed change in the biceps EMG was due to segmental reflexes. In task 2, the velocity error was positive from the movement onset but there was no EMG response until 0.2 s. Velocity error appeared starting at

### Statistical analysis

Statistical analysis revealed that the times of the EMG response in the biceps and both heads of triceps did not differ in the two tasks (Table 1), although the time courses of the velocity errors were markedly dissimilar (Fig. 2, A and B). The velocity error traces for all subjects (Fig. 2B) crossed a threshold value of +0.6 rad/s (Smeets et al. 1990) within a wide interval of 0.115–0.185 s. If the crossing of this threshold is the trigger for the EMG response in the flexor muscles, then the velocity errors aligned on the biceps EMG response should intersect that threshold 30–40 ms earlier. These realigned velocity errors are shown in Fig. 2C. They do not intersect at any threshold. The values of velocity errors 30 ms prior to the time of the biceps EMG response were significantly different in the two tasks (Table 1).

The expected movements in tasks 1 and 2 had significantly different peak accelerations and peak velocities (Table 1). However, the times to peak acceleration and times to peak velocity did not differ. Consistent with the commonly observed correlation between the duration of the first agonist burst and the time to peak velocity (Cooke and Brown 1994), we infer that there was also no difference in the duration of the first agonist burst in the two tasks. In addition, we visually inspected the averaged agonist EMG bursts in the two conditions and could identify no differences in duration.

### Table 1. Kinematic parameters and times of the EMG response in the two tasks

<table>
<thead>
<tr>
<th></th>
<th>Task 1</th>
<th>Task 2</th>
<th>Difference Between the Tasks</th>
<th>Paired t-Test P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Time of the EMG response in movements with unexpected loads</strong></td>
<td></td>
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<tr>
<td>Biceps</td>
<td>0.209 ± 0.023</td>
<td>0.191 ± 0.023</td>
<td>0.019 ± 0.033</td>
<td>0.193</td>
</tr>
<tr>
<td>Brachioradialis</td>
<td>0.227 ± 0.014</td>
<td>0.209 ± 0.016</td>
<td>0.018 ± 0.04*</td>
<td>0.014*</td>
</tr>
<tr>
<td>Triceps long head†</td>
<td>0.205 ± 0.022</td>
<td>0.190 ± 0.015</td>
<td>0.015 ± 0.031</td>
<td>0.293</td>
</tr>
<tr>
<td>Triceps lateral head</td>
<td>0.200 ± 0.017</td>
<td>0.186 ± 0.019</td>
<td>0.014 ± 0.022</td>
<td>0.140</td>
</tr>
<tr>
<td><strong>Kinematic parameters in movements with expected loads</strong></td>
<td></td>
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<tr>
<td>Time to peak acceleration</td>
<td>0.114 ± 0.018</td>
<td>0.126 ± 0.019</td>
<td>0.017 ± 0.016</td>
<td>0.1061</td>
</tr>
<tr>
<td>Time to peak velocity</td>
<td>0.192 ± 0.025</td>
<td>0.196 ± 0.028</td>
<td>0.004 ± 0.016</td>
<td>0.543</td>
</tr>
<tr>
<td>Peak acceleration (deg/s²)</td>
<td>2613 ± 756</td>
<td>4742 ± 1753</td>
<td>2129 ± 1109*</td>
<td>0.0023*</td>
</tr>
<tr>
<td>Peak velocity (deg/s)</td>
<td>225 ± 26</td>
<td>405 ± 83</td>
<td>180 ± 59*</td>
<td>0.0002*</td>
</tr>
<tr>
<td>Velocity error (deg/s)†</td>
<td>48 ± 50</td>
<td>121 ± 69</td>
<td>73 ± 59*</td>
<td>0.0172*</td>
</tr>
</tbody>
</table>

Values are mean ± SD in s for 7 subjects, unless noted otherwise. EMG, electromyographic. * Statistically significant difference. † Values at 30 ms prior to the biceps EMG response. ‡ n = 6, no EMG response in 1 subject in task 2.
about 0.07–0.09 s in both tasks (Fig. 2B), and segmental reflex responses to the initial negative velocity error in task 1 and positive velocity error in task 2 were expected to occur at about 0.12 s. The question therefore is: why were segmental reflex responses absent during the initial part of unexpectedly loaded movements in both tasks?

It has been suggested that segmental reflex response may appear late in unexpectedly loaded movements because it is triggered when the velocity error crosses a threshold (Smeets et al. 1990). However, our results show that the EMG response was not related to the crossing of a fixed threshold by the velocity error. One of the reasons we reached a conclusion different from that of Smeets and colleagues might be that in our experiment the movements spanned larger distances and the time to peak velocity was about 50–100 ms longer (cf. Fig. 2 in Smeets et al. 1990). It is therefore possible that the threshold dependency of EMG responses cannot be generalized across different movement conditions. Another possibility is that if the planned movements are short so that the EMG responses occur relatively early, then the spread of the re-aligned velocity error traces turns out to be narrow enough to give a misleading appearance of a velocity threshold.

A possible explanation for the initial absence of segmental reflex response is that, although the segmental reflex response is driven by velocity error, reflex sensitivity during movement is centrally regulated. The idea that segmental stretch reflexes should act at short latency when a stationary limb is perturbed has an unassailable physiological basis. However, when mechanical perturbations are superimposed on voluntary movement, most investigations have found a strongly reduced reflex sensitivity during a large part of the movement (Gottlieb and Agarwal 1980; Gottlieb et al. 1970; Hallett et al. 1975; Soechting et al. 1981). Our results are compatible with most previously published studies involving unexpected loads in that the EMG responses always occurred close to the end or after the first agonist burst (e.g., Day and Marsden 1982; Gottlieb 1996; Hallett et al. 1975; Latash 1994; Lee et al. 1986; Marsden et al. 1976; Sanes 1986). We hypothesize that in our experiment, for the biceps EMG to remain unmodulated about 0.2 s despite large changes in movement kinematics, the segmental reflex response must have been suppressed. The question is: what determines its onset time?

We suggest that the time interval during which segmental reflex responses are suppressed is determined by the central command which both activates the muscles and regulates reflex excitability. The features of this command determine the timing of the agonist and antagonist bursts and the duration of reflex inhibition. The timing parameters of the central commands in the two tasks were similar, as indicated by the fact that the times-to-peak velocity did not differ (Table 1). In our experiment, movement tasks 1 and 2 were qualitatively similar (i.e., fast point-to-point elbow flexion movements against an inertial load), and we believe that the control processes during these movements were similar. We conclude that, despite the variations in the expected and unexpected movement kinematics, the time of onset of EMG responses to the velocity error did not differ in the two tasks because the intervals of centrally modulated segmental reflex inhibition were the same.

We acknowledge the valuable comments of Dr. David Vaillancourt. This study was supported in part by National Institutes of Health Grants F32 HD-08596, RO1-AR-33189, and RO1-NS-28127 and RO1-NS-40902.

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