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Wrist muscle activation patterns and stiffness associated with stable and unstable mechanical loads

S.J. De Serres[1] and T.E. Milner[2]

Institut de Génie Biomédical, [1]École Polytechnique de Montréal and [2]Université de Montréal, Montréal, Québec, Canada

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Summary. The objectives of this study were to examine the effects of load mechanical characteristics and agonist-antagonist muscle cocontraction, on the stretch reflex response of wrist flexor muscles, and to measure the associated wrist stiffness. Subjects were required to maintain a constant wrist angle while operating against flexor loads with different stability characteristics (constant, elastic or unstable). We measured the stretch reflex responses and joint stiffness by applying step displacements of 3° and 10°. Subjects used very little cocontraction of wrist flexor and extensor muscles when the load was constant or elastic, but increased cocontraction dramatically when the load was unstable, in order to increase the wrist stiffness. Although the magnitude of stretch reflex responses also increased with cocontraction, this simply reflected the level of tonic flexor muscle activity. We found no evidence to suggest that phasic stretch reflexes contributed significantly to the joint stiffness in this task. Clear differences in flexor muscle synergy were observed in the presence and absence of cocontraction, particularly when comparing the FCR and FCU muscles.

Key words: Wrist – Muscles – Unstable load – Stiffness – Human

Introduction

The elasticity of muscle is perhaps its single most important mechanical property as relates to the maintenance of posture. Muscle responds immediately to an increment or decrement in length with a corresponding increment or decrement in force resulting from the elastic behavior of cross-bridges that constitute its integral structure. This response is supplemented after some delay by a reflex increase or decrease in muscle activation mediated by muscle afferents. As in any mechanical system with a position feedback loop, the apparent stiffness of a muscle could be modified by modifying the efficacy of the reflex.

Estimates of the intrinsic muscle stiffness from experiments with human subjects suggest that it increases in a linear fashion with muscle force throughout the force range (Sinkjaer et al. 1988). When the contribution of the stretch reflex is added, the relation between stiffness and force remains linear at low force levels, although for any given force, the stiffness is proportionately higher than without the reflex (Hunter and Kearney 1982; Cannon and Zahalak 1982; Sinkjaer et al. 1988). However, as the force progresses through its midrange, the total stiffness levels off and may even begin to decline as maximum force is approached (Sinkjaer et al. 1988). A similar behavior has been previously described in the decerebrate cat (Hoffer and Andreassen 1981). This behavior is apparently strictly related to the excitability of the motoneuron pool and simply reflects the level of tonic central drive.

However, the responsiveness of the stretch reflex could also be influenced by a variety of central mechanisms which affect either the γ-motoneuron bias of intrafusal muscle fibers or the synaptic transmission of spindle afferent activity to α-motoneurons and hence the responses to muscle stretch may vary according to task demands. One particular paradigm which has been extensively investigated is that of maintaining an initial posture with the instruction to resist or not to resist a subsequent perturbation (Hammond 1956). Although long latency responses are often augmented by the instruction to resist, it is generally conceded that stretch reflex responses at monosynaptic latency are not subject to modification (Crago et al. 1976; Evarts and Vaughn 1978; Colebatch et al. 1979; Jaeger et al. 1982; Calancie and Bawa 1985). Nevertheless, the monosynaptic reflex might well be modulated independently of tonic central drive in other postural tasks. In the case of the long latency stretch reflex response of finger muscles, Akazawa et al. (1983) and Kanosue et al. (1983) have shown that it can be modified by the stability of the external load. Whether or not the

Offprint requests to: T.E. Milner, Institut de réadaptation de Montréal, Centre de Recherche, 6300 Darlington Avenue, Montréal, Québec H3S 2J4, Canada

monosynaptic reflex undergoes a similar adaptation to the load stability could not be addressed by these experiments since monosynaptic responses were not elicited from the finger muscles. This question could be answered by examining stretch reflex responses in more proximal muscles.

A confounding factor in the experiments of Akazawa et al. (1983) was muscle cocontraction. While reflex responses and stretch-evoked stiffness increased as the instability of the load increased, so did antagonist muscle cocontraction. They showed that both the reflex responsiveness and stretch-evoked stiffness increased with the cocontraction. However, they did not systematically examine whether the reflex responsiveness was simply a function of the tonic agonist muscle activation.

Hogan (1984) examined agonist-antagonist muscle cocontraction in a postural maintenance task. He argued that maintaining posture by reflex feedback would be more energy efficient than muscle cocontraction, but might be less effective because of gain limitations due to transmission delays in the feedback pathway. Based on this assumption, he carried out a theoretical analysis and predicted that cocontraction would be required to stabilize a joint at an unstable equilibrium position in order to counteract small random perturbations. Indeed, he found coactivation of the biceps and triceps muscles when the forearm was placed in such an unstable equilibrium position.

Recently, Al-Falahe and Vallbo (1988), in studying muscle spindle discharge in finger muscles, concluded that reflex responses were quite ineffective in opposing sudden displacements, even when expected, because of their relatively long delay. Rather, agonist-antagonist cocontraction appeared to be a more effective way to counteract disturbances. Furthermore, they suggested that any reflex modifications which they observed, simply paralleled central excitability changes accompanying muscle contraction.

Starting from the assumption that the primary function of stretch reflexes is to compensate for small disturbances, rather than large perturbations, Colebatch and McCloskey (1987) estimated elbow stiffness using very gradual displacement of the limb. Their results indicated that when subjects were required to control position, the joint stiffness was greater than when they were required to control force. They claimed that this stiffness modulation was not simply due to agonist-antagonist cocontraction. However, since they recorded activity from only one muscle, an agonist in the task, the possibility that cocontraction was present cannot be ruled out.

In order to examine the relative importance of agonist-antagonist cocontraction and to resolve the question of whether the monosynaptic stretch reflex is modulated by load stability in a manner that is independent of pre-existing tonic muscle activation (central drive to the motoneuron pool), we have chosen to compare the stretch reflex in wrist flexor muscles under three different stability conditions. Since several muscles contribute to wrist flexion and their synergy may change with agonist-antagonist cocontraction, we monitored the activity of three wrist flexor muscles and one wrist extensor muscle. In addition, we measured the joint stiffness and related it to the level of muscle activation. By using stretches of two different lengths we were also able to compare the behavior of monosynaptic and polysynaptic reflex responses.

Material and methods

This study was conducted with twelve normal subjects (6 male and 6 female, ranging in age from 22–44). Eleven of the subjects were right-handed, the other left-handed. Experiments were carried out on the left hand only. All of the subjects gave their informed consent to the procedure.

Apparatus

A torque motor (PMI U16M4) was used to generate loads under computer control during an initial holding period. This was followed by a ramp angular displacement of either 3° or 10° during which the motor was position servoed using an analog feedback circuit. The duration of the ramp was approximately 25 ms in the case of a 3° displacement and 75 ms in the case of a 10° displacement. The stiffness of the position servo was 1.95 Nm/deg while the maximum torque which could be generated by the motor was 3.4 Nm. Due to the limited torque capability of the motor, the velocity during the ramp was not constant. The peak velocity depended on the size of the angular displacement during the ramp and the bias torque present at the onset of the ramp. In the case of a 3° ramp, the mean peak velocities were 200, 186, 168 and 155 deg/s for bias torques of 0, 0.5, 1.0 and 1.5 Nm, respectively, while for a 10° ramp with the same bias torques the mean peak velocities were 346, 335, 326 and 328 deg/s.

Position and velocity were measured by a potentiometer and tachometer, respectively while the torque was measured by a linear strain gauge mounted on a cylinder, coupling the motor shaft to a wrist manipulandum.

Procedure

The subject was seated comfortably in a chair with the left forearm resting on a padded support. The forearm was oriented midway between pronation and supination and was immobilized so as to restrict movement to flexion and extension of the wrist joint. The subject's hand rested on a platform that was coupled to the shaft of the motor. The hand was clamped securely between two narrow curved pads at the level of the palm, retaining the thumb, but leaving the fingers free. The pads were positioned so as to align the axis of rotation of the wrist over the motor axis.

Prior to each ramp displacement, the subject was required to maintain the manipulandum within a 1° target window for a random period ranging from 1–2 s. The target was adjusted to be at the neutral position of the wrist for each subject. During the holding period, the subject was provided with an oscilloscope display of the wrist position and target position. At the end of the hodling period, the hand was servoed to a new position either 3° or 10° into extension. The display of the wrist position disappeared from the screen during the servo period. The hand was held at the new position by the servo controller for about 700 ms and then gradually released as the torque was ramped slowly back to zero. The subject was instructed not to intervene voluntarily in response to the servoed displacement of the wrist. Trials in which there was evidence of voluntary intervention (variation of more than 0.1 deg in the angular position or of more than 0.1 Nm in the torque) during the interval 350–550 ms after the onset of the displacement were rejected.

During the holding period, the subject opposed one of four bias torques acting in the direction of wrist extension. The bias levels were 0, 0.5, 1.0 and 1.5 Nm. The load torque was either isotonic or was modulated linearly as a function of the angular position of the wrist. Position dependent torque modulation was achieved using either

negative or positive position feedback to the motor in order to produce the characteristics of a spring or an unstable load. For both position dependent loads, the absolute value of the feedback gain was 0.1 Nm/deg. When the feedback gain was negative with a bias torque of 0 Nm, the equilibrium position of the manipulandum was at the target position. When the subject displaced the manipulandum, the motor responded with a torque proportional to the angular displacement, directed toward the equilibrium position. Bias torques greater than 0 Nm were produced by shifting the equilibrium position of the motor. When the feedback was positive, the motor pushed the wrist away from the equilibrium position with a torque proportional to the angular distance from the equilibrium position. Hereafter, the three types of loads will be referred to as constant, elastic and unstable.

A block of 80 trials was executed for each of the three load types. The loads were presented in the same order for all subjects: elastic, constant and unstable. Within each 80 trial block, the bias torques were presented as follows: 1.0 Nm on trials 1–20, 0 Nm on trials 21–40, 1.5 Nm on trials 41–60 and 0.5 Nm on trials 61–80. The 3° and 10° servo displacements were administered in a random order.

Recording

For eleven subjects, EMG was recorded from the flexor carpi radialis (FCR), flexor digitorum superficialis (FDS), flexor carpi ulnaris (FCU) and extensor digitorum communis (EDC) muscles. In the remaining subject the FCU was replaced by the extensor carpi radialis (ECR) muscle. The choice of the EDC as a representative muscle on the extensor side was based on the fact that it showed the largest increase in activation during postural maintenance of the unstable load. It was therefore felt to be the best indicator of agonist-antagonist muscle cocontraction.

The EMG was recorded using active, bipolar, stainless steel, surface electrodes (Liberty Mutual MYO111) with a bandpass of 45–550 Hz. The electrode contacts were 3 mm in diameter and were spaced 13 mm apart. Before the recording session began, the optimal placement of the electrode on each wrist flexor muscle was determined by observing the EMG activity in response to three rapid test movements: 1) wrist flexion with fingers extended (FCR); 2) finger flexion without wrist movement (FDS); 3) wrist adduction with the fingers extended (FCU). Each electrode was placed so as to maximize the signal during the appropriate movement while minimizing the signal during the other two.

Position, velocity, torque and EMG signals were amplified and digitized at 2 kHz then stored on disk for later analysis.

Analysis

The EMG records were rectified and all of the trials with the same parameters (load type, bias torque and displacement amplitude) were then averaged. The mean values of the position and torque and the rms value of the tonic activity in each muscle were calculated for the interval 50–250 ms before the onset of the displacement and for the interval 350–550 ms after the onset of the displacement. The muscle stiffness was calculated as the ratio of the change in mean torque divided by the change in mean position.

A normalization procedure was applied to the rms EMG values of the flexor muscles in order to reduce the variability among subjects. First, the linear regression coefficients for the relation between the tonic EMG prior to wrist displacement and the initial torque were computed for each subject using the data obtained under the constant load condition. There was little or no antagonist muscle contraction in this case, so the relation was highly linear ($r \geq 0.97$ for most subjects: 11/12 for FCR, 8/12 for FDS, 7/11 FCU; the lowest r value was 0.75 for the FCU). The slopes of the tonic EMG versus torque relation were then used to scale all of the subject's EMG data (by dividing the EMG of a particular muscle by the respective slope).

The velocity trace was differentiated using a digital finite impulse response filter to obtain the acceleration. Because the displacement was quite rapid there was a large inertial component in the torque record while the motor was moving. The relative contribution and duration of this inertial torque could be estimated from the acceleration.

The flexor muscles responded to the 3° stretch with a short burst of activity at monosynaptic latency. On occasion there was also a second burst at a longer latency, but it was always clearly separated from the first burst. The short latency response was demarcated by determining the time on either side of the peak where the amplitude of the burst dropped below the rms value of the tonic EMG (calculated during the 50–250 ms interval before the onset of the stretch). The tonic EMG was then subtracted and the integral of the EMG was calculated for the reflex interval. Like the tonic EMG, the integrated reflex EMG was scaled by dividing by the slope of the tonic EMG versus torque relation. In the case of the 10° stretch there were usually two reflex responses, one at short latency and the other at longer latency. Often the two responses merged without a clear separation, although the beginning of the early response and the end of the late response could be easily determined. When this occurred, the division between the two responses was taken as the time at which the short latency response ended under the corresponding load conditions for the 3° stretch. The short latency and long latency reflex responses to the 10° stretch were then separately quantified.

Data from all twelve subjects were pooled for each load type (constant, elastic or unstable) and linear regression coefficients were computed for the following dependent variables as functions of the initial torque: 1) rms EMG before the displacement; 2) rms EMG after the displacement; 3) short latency reflex EMG; 4) long latency reflex EMG. This was done for each flexor muscle, as well as for the EDC in the case of the first two variables. In addition, linear regression coefficients were computed for stiffness as a function of the initial torque. Statistical comparisons of mean values, slopes and intercepts were carried out by applying the Student t-test and testing the null hypothesis.

Results

Reflex responses

When the wrist was displaced in the direction of wrist extension, all three wrist flexor muscles (FCR, FDS and FCU) responded with a brief burst of activity at monosynaptic latency (Fig. 1).

In most cases, a wrist displacement of 10° produced a second burst of activity in the flexor muscles that immediately followed the short latency response. In 6 of the 12 subjects, a long latency burst was also observed occasionally in response to a 3° displacement (Fig. 1). However, its amplitude was relatively small compared to the short latency burst, as well as being quite variable.

The reflex responses in the flexor muscles were followed by a period of inhibition during which activity dropped below the level prior to wrist displacement. A brief period of strong inhibition was also observed in the EDC activity. Following the inhibitory period, the EMG of the wrist flexors returned to a level which was often slightly higher than that observed before the imposed wrist extension. The EDC EMG, on the other hand, remained slightly lower.

Mechanical response to displacement

During the displacement of the wrist, the torque progressed through a transient phase before stabilizing at a new

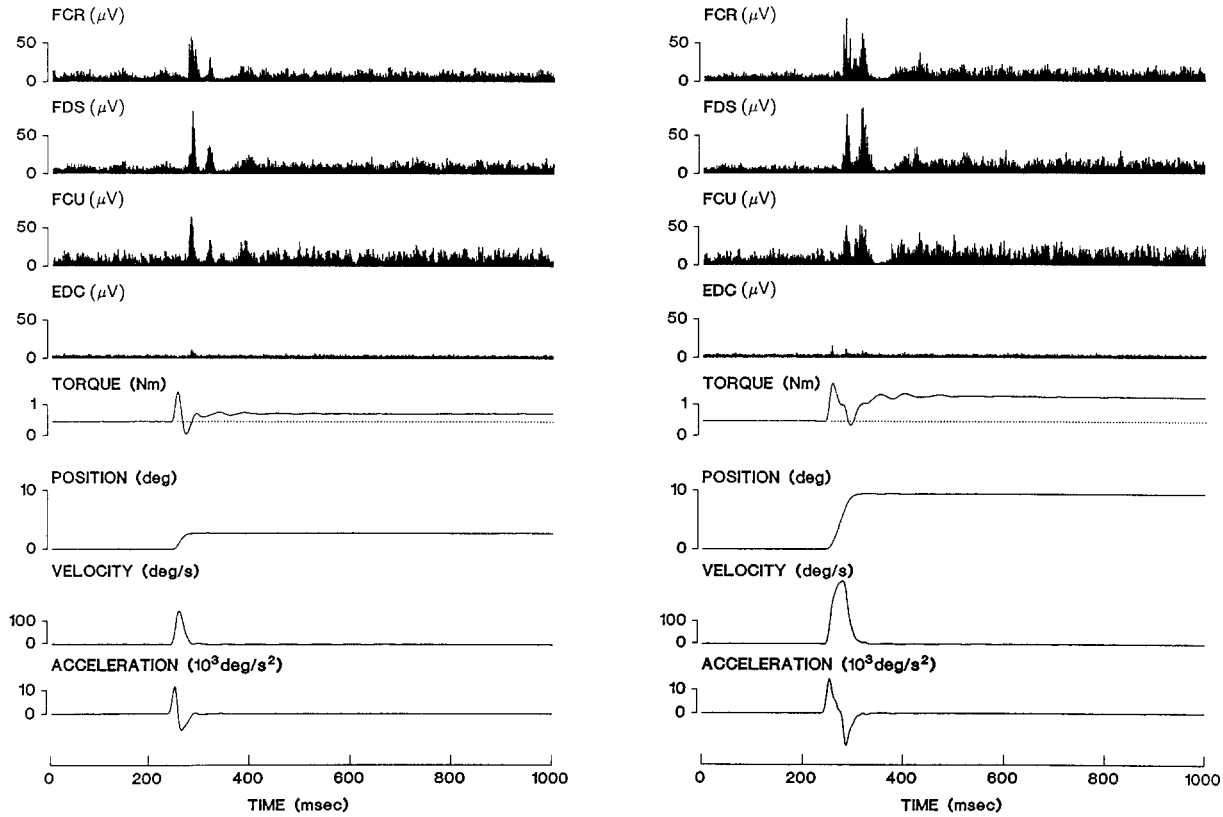

Fig. 1. Reflex responses to displacements of 3° (*left*) and 10° (*right*) with an initial load of 0.5 Nm under the constant load condition. Each record is the average of 10 trials. The top four traces are records of the rectified EMG in the flexor carpi radialis (FCR), flexor digitorum superficialis (FDS), flexor carpi ulnaris (FCU) and extensor digitorum communis (EDC) muscles. The following three traces are the torque, angular position and angular velocity, measured from transducers while the bottom trace is the angular acceleration, computed by digital differentiation of the velocity. The *dashed line* in the torque trace represents the initial torque load

level. If one compares the torque and acceleration profiles during the period of displacement, synchronization of peaks and inflections in the two signals is clearly evident (Fig. 1). This is due to the fact that the moment of inertia of the hand and manipulandum generated a relatively large torque during acceleration and deceleration of the motor. This inertial torque was superimposed upon the elastic torque produced by stretching the wrist flexor muscles. As the wrist stiffness increased with increasing levels of muscle activation for higher initial torques, the relative magnitude of the inertial torque decreased. When the acceleration returned to zero at the end of the displacement, the wrist torque stabilized at a value greater than its value prior to the onset of displacement, due to the wrist stiffness.

Load stability

The muscle activity and mechanical responses observed with the elastic and constant loads presented a similar picture, in marked contrast to the unstable load. Qualitative differences between stable (constant and elastic) and unstable loads will be discussed first, followed by presentation of the quantified results.

When the subject was required to maintain a stable wrist position in the face of an unstable load, there was a significant increase in the initial tonic activity of all muscles, indicating agonist-antagonist cocontraction. This can be seen in Fig. 2 where recorded data from the stable and unstable load conditions are compared. The change in the steady-state torque was greater in the case of the unstable load than the stable load, when the initial torques were equal, indicating higher wrist stiffness.

The flexor reflex responses were considerably larger in the case of the unstable load than the stable load for low initial torques (Fig. 2), but they increased relatively less when the initial torque increased (Fig. 3). Because of the increased wrist extensor muscle activity, the inhibitory period in the EDC was clearly evident in the case of the unstable load.

The effect of increasing the initial torque, maintained by the subject during the holding period prior to wrist displacement can be seen by comparing Figs 2 and 3. The mean level of activity in the flexor muscles increased with initial torque, both during the holding period prior to and following the displacement, for both the stable and unstable load conditions. The reflex responses also increased with the initial torque. The change in steady-state torque was greater when the initial torque was larger, for the same wrist displacement, indicating an increase in wrist stiffness.

Reflex responses increased more dramatically with an increase in initial torque for the stable load than the unstable load. There was also a more dramatic increase in stiffness with initial torque under the stable load than the

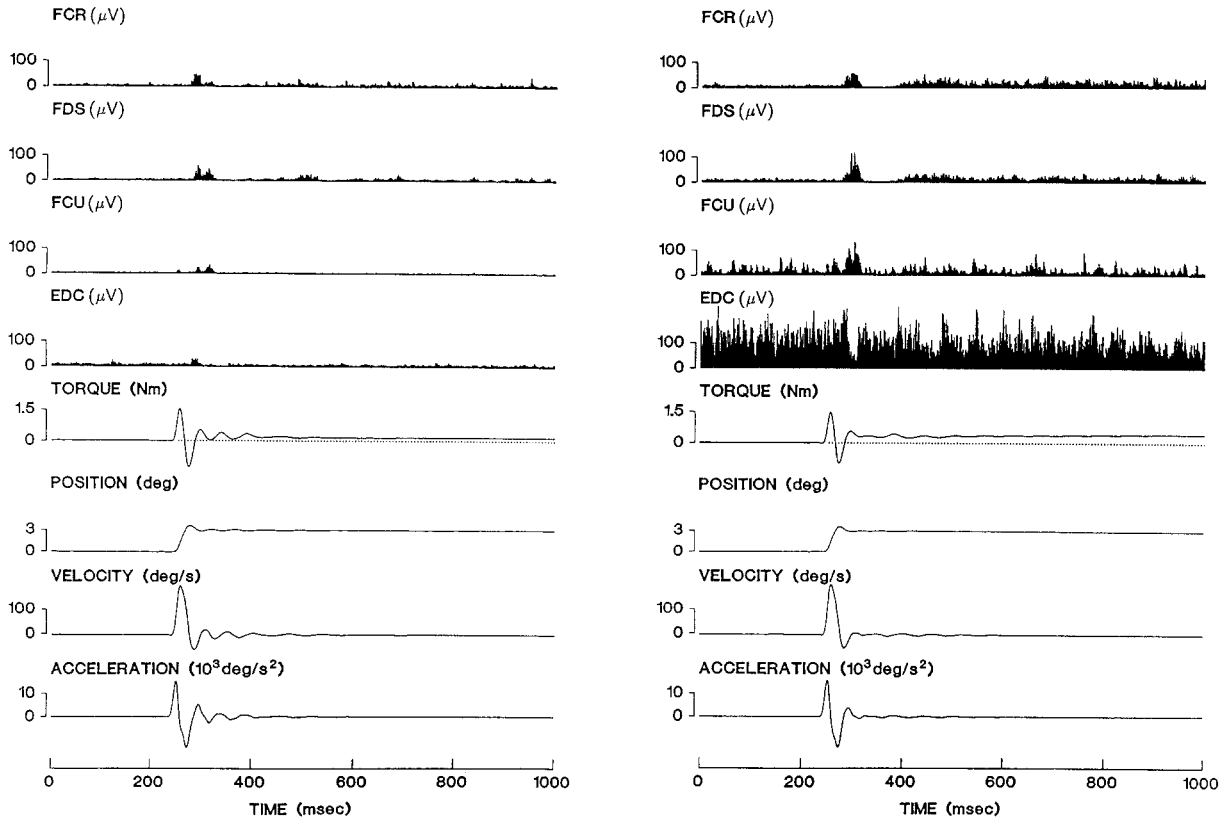

Fig. 2. Reflex responses to displacements of 3° under elastic (*left*) and unstable (*right*) load conditions with an initial load of 0 Nm, represented as in Fig. 1

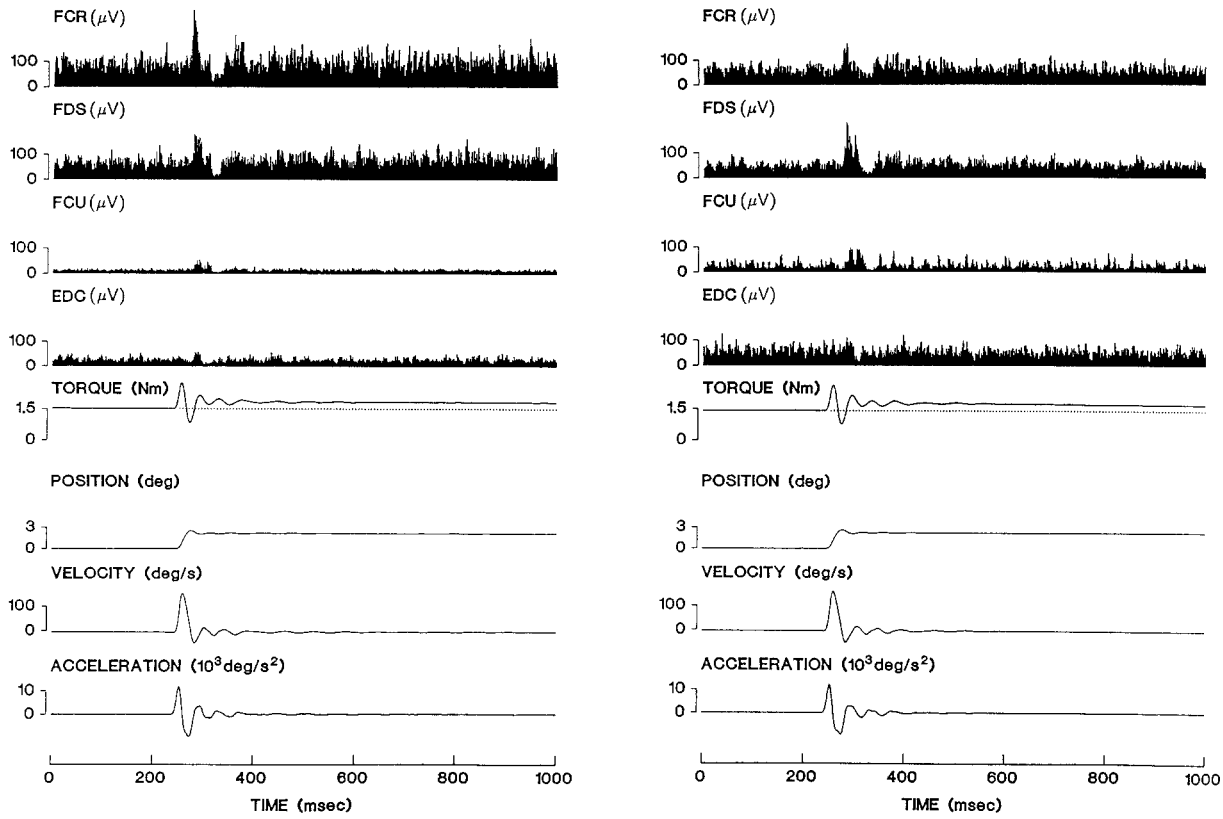

Fig. 3. Reflex responses to displacements of 3° under elastic (*left*) and unstable (*right*) load conditions with an initial load of 1.5 Nm, represented as in Fig. 1

unstable load conditions. In the latter case, there was often very little change in stiffness with changes in the initial torque.

A quantitative comparison of the reflex and mechanical responses for the different load types was made by comparing the slopes and intercepts of the regression lines for EMG and stiffness as functions of the initial torque. In general, the differences between constant and elastic loads were small, whereas the differences between constant and unstable or elastic and unstable loads were often relatively large.

Agonist-antagonist muscle cocontration was considerably greater for the unstable load than either the constant or elastic load, as reflected by upward shifts of the regression lines of tonic EMG plotted against initial torque in Fig. 4 (significant difference in intercepts, $p<0.05$). However, tonic flexor activity increased more slowly with initial torque when the load was unstable (significant difference in the slopes of constant and unstable loads, $p<0.05$). This can be attributed in large part to the fact that the antagonist activity declined as the initial torque increased. Thus, relatively less flexor activity was required to balance the extensor activity. The results for the 10° displacement are not shown, since they differed little from those of the 3° displacement.

The change in tonic flexor EMG and EDC EMG produced by the displacement can be seen on the right in Fig. 4. There was an increment in the tonic EMG for stable loads which tended to rise in a similar manner with initial torque for all three flexor muscles. In contrast, when the load was unstable, the change in tonic flexor EMG clearly differed among the three muscles. The difference between stable and unstable loads became increasingly more pronounced going from FCR to FDS to FCU, as the change in tonic EMG progressed from an increment to a decrement. The displacement also produced a decrement in tonic EDC EMG which, while insignificant for stable loads, became dramatic when the load was unstable.

The principal effect of the unstable load on the short latency reflex response (Fig. 5, left) was an upward displacement of the regression lines, the greatest change taking place in the case of the FCU muscle (significant difference in intercept, $p<0.05$). The short latency reflex excitability in flexor muscles parallels their initial tonic activity (Fig. 4).

The long latency response was analyzed only in the case of the 10° displacement where it was present for all subjects (Fig. 5, right). As in the case of the short latency response, there was a significant upward shift of the regression lines in going from a constant to an unstable load (significant difference in intercepts, $p<0.025$).

The relations between steady-state stiffness and initial torque for the three load types are compared in Fig. 6. Two

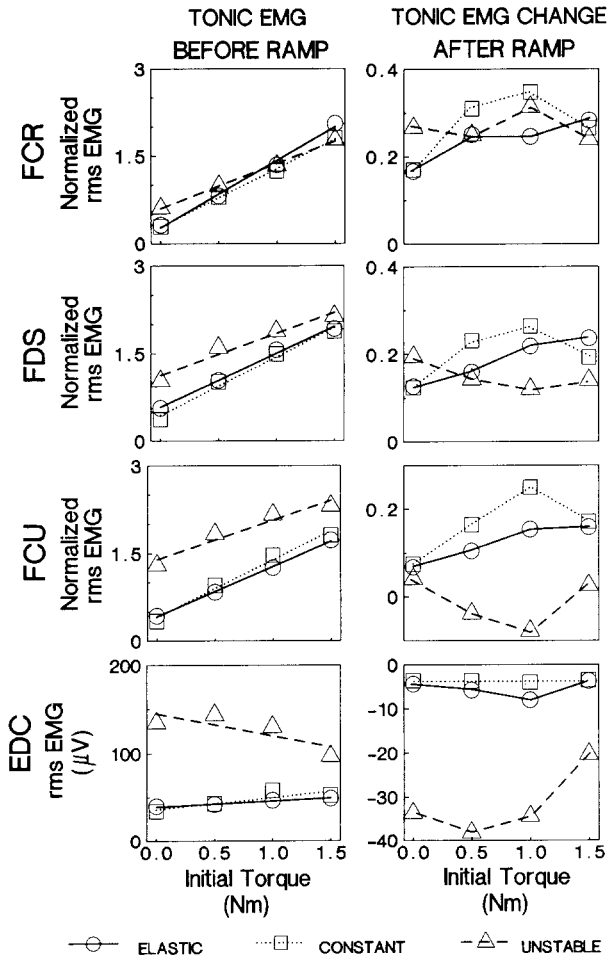

Fig. 4. *Left*: comparison of regression lines for the tonic EMG (rms values) as a function of initial torque, before a 3° displacement, under the three load conditions. The EMG has been normalized in the case of the three wrist flexor muscles, but not in the case of the EDC muscle. *Right*: change in tonic EMG following the wrist displacement (post-displacement EMG minus pre-displacement EMG)

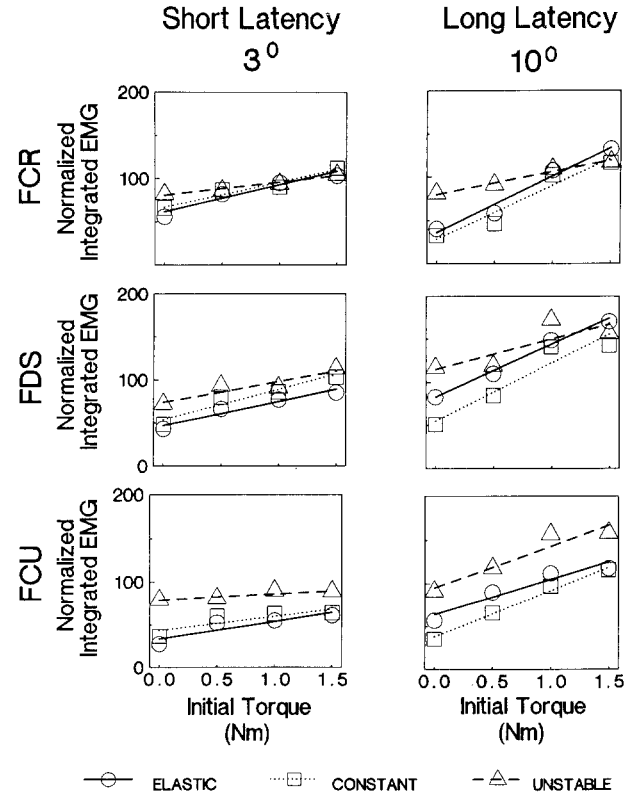

Fig. 5. Comparison of regression lines for the short latency reflex response to a 3° displacement (*left*) and long latency response to a 10° displacement (*right*) of wrist flexor muscles (integrated EMG) as a function of initial torque under the three load conditions

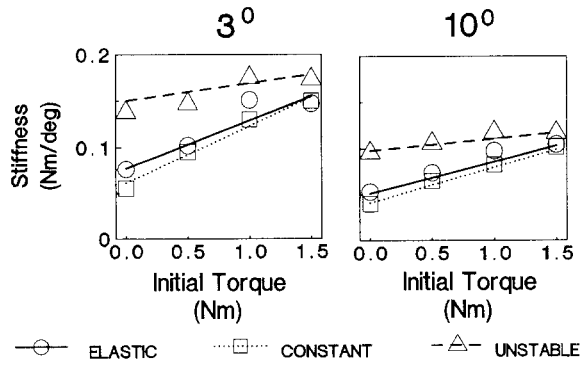

Fig. 6. Comparison of regression lines for wrist stiffness as a function of initial torque for a 3° displacement (*left*) and a 10° displacement (*right*), under the three load conditions

features are immediately evident. First, the stiffness for a 3° displacement under a given load condition was greater than for a 10° displacement. Second, in the case of the unstable load, the stiffness at low initial torques was substantially greater than that of either the constant or elastic load. This observation was substantiated quantitatively by comparing the intercept of the regression lines, i.e. the intercept when the load was unstable was significantly greater than when the load was either constant or elastic ($p < 0.0005$). The slope of the regression line for the unstable load was significantly less than that of either the constant or elastic load ($p < 0.01$).

We found that when the load was unstable, the stiffness was frequently greater for an initial torque of 1.0 Nm than 1.5 Nm. This implied that there was more agonist-antagonist cocontraction in the former case. Since the 1.0 Nm load was always presented for the first 20 trials while the 1.5 Nm load was presented only after 40 trials, we felt that the reduced cocontraction might have been the product of learning, as the subject minimized the wrist stiffness necessary to maintain postural stability.

To test this hypothesis we compared the tonic EDC EMG prior to the displacement for the first, seventh and thirteenth trials with the 1.0 Nm load, using Friedman's method for randomized blocks. We found that there was a drop in the EDC EMG for the seventh and thirteenth trials with respect to the first trial, although the level of significance was only $0.05 < p < 0.1$.

Discussion

The primary objective of this study was to determine the effect of an unstable mechanical load on patterns of agonist-antagonist muscle activation and the resulting joint stiffness at the wrist. Clearly, the position dependent instability used in these experiments was counteracted by significant coactivation of wrist flexor and extensor muscles. However, extensor muscle cocontraction did not parallel the pattern of flexor muscle activation as the initial torque increased. While flexor muscle activity increased in proportion to the initial torque, EDC muscle activity actually declined for the highest initial torque. This resulted in a negative slope for the relation between EDC muscle activity and initial torque.

This behavior can be easily understood if the mechanical interaction of the load and the wrist joint are examined. The unstable load had slope of 0.1 Nm/deg. In order to stabilize this load it was necessary that the local wrist stiffness be greater than 0.1 Nm/deg. This wrist stiffness could be achieved by any combination of flexor and extensor muscle activity that produced sufficient flexor torque to counteract the external load while at the same time generating the necessary stiffness. The local wrist stiffness is the sum of the flexor and extensor stiffness. When the initial torque was low, little flexor activity was required to counteract the external load and consequently considerable extensor activity was needed to achieve the stiffness necessary to stabilize the load. As the initial torque increased, more flexor activity was required to counteract the external load with a consequent increase in the contribution of the flexor muscles to the total wrist stiffness. As a result, the extensor muscle contribution could be reduced. The local wrist stiffness was always greater than 0.1 Nm/deg, as can be judged from the intercept of the regression line relating muscle stiffness to initial torque, which was 0.15 Nm/deg for a 3° wrist displacement. The fact that the intercept was slightly less than 0.1 Nm/deg in the case of a 10° stretch is of little consequence because it is only necessary that the stiffness be greater than 0.1 Nm/deg in a local region around the equilibrium position of the wrist in a postural task of this type. It is, nonetheless, noteworthy that the stiffness was nearly large enough for stability even with a deviation as large as 10° from the equilibrium position.

The lower stiffness with a 10° displacement than with a 3° displacement was predictable from a knowledge of intrinsic muscle properties. The relatively high value of short-range muscle stiffness is a well-documented muscle nonlinearity (Hoffer and Andreassen 1981; Kearney and Hunter 1982). Our results corroborate these previous findings.

The bursts of muscle activity constituting the short and long latency reflex responses seem to have made little contribution to the wrist torque, contrary to the claims of Sinkjaer and Hayashi (1989). We draw this conclusion because we observed no significant fluctuations in the measured torque following wrist displacement that were not synchronized with fluctuations in the acceleration. These fluctuations were therefore due to inertial torque produced by acceleration of the hand and manipulandum. Furthermore, in those cases where the acceleration was relatively flat following wrist displacement, torque fluctuations were very small as compared to the net change in steady-state torque.

In contrast to Akazawa et al. (1983), we found no evidence that the stretch reflex was modulated independently of the level of tonic muscle activation existing prior to the stretch. Since our results are not only similar to theirs, but show even more marked differences in muscle behavior under constant and unstable load conditions, we are inclined to believe that this disagreement is a matter of interpretation of the data. As explained at the outset of the Discussion, we obtained a very consistent picture between the tonic activity in agonist and antagonist muscles and the mechanical response to stretch. Furthermore, we have shown that the reflex behavior parallels the tonic behavior

as the initial torque increases. For example, we see a very different behavior in the tonic activation of the FCR and FCU muscles when going from the constant to the unstable load. While the regression line for the FCR undergoes a rather small clockwise rotation about its central point, the regression line for the FCU is translated upward by a substantial amount with little change in slope. The reflex responses for these two muscles faithfully reproduce their tonic behavior. This is clear from comparison of the corresponding regression lines for the tonic muscle activity and the reflex responses. Akazawa et al. (1983) based most of their conclusions on observations from a single muscle. On the strength of our observations from four muscles, including further evidence to be discussed below, we must conclude that in the context of our experiments, there is no modulation of reflex activity that is independent of motoneuron excitability.

Our finding that the activity of the FCR was little altered by cocontraction of the wrist extensors whereas FCU activity increased markedly, casts some doubt on the conclusion reached by Colebatch and McCloskey (1987) that stretch reflex gain and joint stiffness of the elbow could be modified without a change in agonist muscle activation. Their conclusion was based on the observation that joint stiffness increased without a concomitant increase in biceps EMG when the subject was instructed to maintain a constant position as opposed to a constant force. However, the biceps is only one of five elbow flexors. Had we examined only the FCR, we would have drawn a similar conclusion about the absence agonist-antagonist cocontraction, which clearly would have been in error.

We attribute the increased FCU activity with the unstable load partly to a requirement for adductor torque at the wrist in order to cancel an increasing abductor torque generated by cocontraction of the extensor carpi radialis muscles. We did not routinely record from the ECR muscles, but when we did we found that their activity increased significantly with the unstable load.

Agonist-antagonist muscle cocontraction is potentially quite fatiguing, hence one would expect that subjects would avoid this strategy for maintaining postural stability if there was another option, such as increasing the stretch reflex gain. Our evidence suggests that this was either not possible or it was not a viable alternative. Consequently, subjects used cocontraction, although they apparently kept it at the minimal level required to accomplish the postural task. Two observations support the latter claim. First, subjects reduced the tonic EDC muscle activity as they became more accustomed to the unstable load. Second, they maintained nearly constant stiffness over the entire range of initial torques with the unstable load. This is in sharp contrast to the constant and elastic load conditions, where the stiffness increased more than twofold over the same range of initial torques.

We conclude that subjects used a strategy of augmenting joint stiffness through agonist-antagonist muscle cocontraction because it was the most effective way to provide the necessary postural stability to counteract the unstable load. Hogan (1984) reached a similar conclusion with a different type of load instability. Although the magnitude of stretch reflex responses also increased with cocontraction, this simply reflected the underlying excitability of the motoneuron pool, which is related to the amount of tonic activation of agonist muscles (Al-Falahe and Vallbo 1988). We found no evidence to suggest that the larger reflex bursts, associated with increased levels of tonic activation, contributed significantly to the joint stiffness.

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