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RESEARCH ARTICLE

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Inability to activate muscles maximally during cocontraction and the effect on joint stiffness

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Abstract In order to determine the maximum joint stiffness that could be produced by cocontraction of wrist flexor and extensor muscles, experiments were conducted in which healthy human subjects stabilized a wrist manipulandum that was made mechanically unstable by using positive position feedback to create a load with the characteristics of a negative spring. To determine a subject's limit of stability, the negative stiffness of the manipulandum was increased by increments until the subject could no longer reliably stabilize the manipulandum in a 1° target window. Static wrist stiffness was measured by applying a 3° ramp-and-hold displacement of the manipulandum, which stretched the wrist flexor muscles. As the load stiffness was made more and more negative, subjects responded by increasing the level of cocontraction of flexor and extensor muscles to increase the stiffness of the wrist. The stiffness measured at a subject's limit of stability was taken as the maximum stiffness that the subject could achieve by cocontraction of wrist flexor and extensor muscles. In almost all cases, this value was as large or larger than that measured when the subject was asked to cocontract maximally to stiffen the wrist in the absence of any load. Static wrist stiffness was also measured when subjects reciprocally activated flexor or extensor muscles to hold the manipulandum in the target window against a load generated by a stretched spring. We found a strong linear correlation between wrist stiffness and flexor torque over the range of torques used in this study (20–80% maximal voluntary contraction). The maximum stiffness achieved by cocontraction of wrist flexor and extensor muscles was less than 50% of the maximum value predicted from the joint stiffness measured during matched reciprocal activation of flexor and extensor muscles. EMG recorded from either wrist flexor or extensor muscles during maximal cocontraction confirmed that this reduced stiffness was due to lower levels of activation during cocontraction of flexor and extensor muscles than during reciprocal contraction.

Key words Cocontraction · Stiffness · Stretch reflex · Mechanical stability · Human

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Introduction

Muscle and joint stiffness play a prominent role in much of the current thinking regarding control of posture and movement by the central nervous system. Stiffness is a component of mechanical impedance, the property used to quantify the resistance of a mechanical system to imposed movement. Knowledge of the mechanical impedance of the musculoskeletal system is important not only for determining the motion of limb segments in response to internally generated muscle forces, but also in determining how the musculoskeletal system will respond to external forces, particularly when those forces are produced by another mechanical system with different impedance characteristics.

Because of the large range over which it can be modulated, joint stiffness can be used to profoundly alter the mechanical impedance of a limb segment. Furthermore, since inertia is fixed and changes in joint viscosity tend to be relatively inconsequential (Hunter and Kearney 1982; Gielen and Houk 1984; Lacquaniti et al. 1993), the only real means of modifying mechanical impedance for a given limb geometry is by changing joint stiffness.

Joint stiffness can be controlled independently of joint torque through antagonist muscle cocontraction. In particular, joint stiffness can be modified over a wide range even when muscles are producing zero resultant

torque at a joint (Kearney and Hunter 1990). An alternative means of independently modulating joint stiffness by changing the length-dependent reflex gain appears to be relatively ineffective (Bennett et al. 1994) and perhaps, therefore, much less frequently used (De Serres and Milner 1991). As Hogan (1984) pointed out, antagonist muscle co-contraction may be a more desirable means of increasing the stiffness of a joint than by increasing its reflex stiffness because of the inherent instability problems associated with delayed reflex feedback.

Antagonist cocontraction is frequently used to increase the mechanical stability of a joint, and a number of studies have shown that the degree of antagonist cocontraction increases in proportion to the degree of mechanical instability inherent in the task (Houtz 1964; Lacquaniti et al. 1982; Akazawa et al. 1983; Hogan 1984; De Serres and Milner 1991; Milner and Cloutier 1993). Smith (1981) has described other situations where cocontraction is vital, in particular, during prehension. Given that antagonist cocontraction is prevalent in normal activities, it is of interest to determine the maximal capacity of the neuromuscular system for cocontraction and to examine what the limiting factors might be. This aim was achieved by rendering a manipulandum mechanically unstable and requiring human subjects to stabilize it.

Materials and methods

Two experiments were conducted, the first with ten normal, healthy subjects (four women and six men), ranging in age from 21 to 38 years, the second with five normal, healthy male subjects, ranging in age from 22 to 40 years, one of whom was left-handed. The experiments conformed to the ethical standards of the 1964 Helsinki Declaration. All subjects gave informed consent to the procedure.

Subjects were asked to perform a task with the right hand to stabilize a load applied to a wrist manipulandum. In certain cases there was no load, while in the others the load was either a negative spring, a spring opposing flexion or a spring opposing extension. The subjects were required to hold the wrist in its neutral position for a variable time period against the load, after which the wrist was displaced in the direction of extension to measure the static wrist stiffness.

Apparatus

A torque motor (PMI U16M4) was used to generate loads under computer control during an initial holding period. Positive position feedback to the motor was used to produce mechanical behavior equivalent to a negative spring, where the negative stiffness of the spring could be varied by changing the feedback gain. A servo-controlled ramp and hold angular displacement in the direction of wrist extension was used to measure the static stiffness of the wrist joint. The maximum torque that could be produced by the motor was 5 Nm. The position and velocity of the motor were measured by means of 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.

In experiment 1, the ramp consisted of a displacement of 3° executed in approximately 25 ms. The displacement was often underdamped because the velocity feedback gain in the position servo loop could not be made sufficiently high to achieve the required damping. From the displacement traces of Figs. 1 and 2 it can be seen that about 100 ms was required for complete damping of the oscillations. To prevent such underdamped oscillation, displacements were made more slowly (3° in 60 ms) in experiment 2. It is apparent from Figs. 5 and 6 that the displacements were better damped with the slower displacements.

Procedure

The subject was seated comfortably in a chair with the right forearm resting on a padded support. The forearm was oriented midway between pronation and supination and immobilized to restrict movement to flexion and extension of the wrist joint. The subject's hand was clamped securely between two curved pads at the palm, also restraining the thumb. These pads were positioned to align the axis of rotation of the wrist over the motor axis.

The subject was required to position a hairline cursor within a 1° target window indicated by bars displayed in the middle of a computer screen. The cursor position on the screen corresponded to the angular position of the wrist. The target zone was adjusted to be at the neutral position of the wrist for each subject. The load was activated simultaneously with the appearance of the target on the screen.

Following the appearance of the target, the subject was required to move the cursor into the target window and maintain a stable position for a random period ranging from 1.5 to 2.0 s. On successful trials this was followed by a small ramp displacement, which extended the wrist. Failure to stabilize the wrist within 15 s of the appearance of the target constituted an unsuccessful trial. Subjects were given four chances to successfully complete a trial.

Experiment 1

The stability limit for each subject was determined by incrementally increasing the positive position feedback gain to the motor until the subject could no longer successfully complete the trial. Five feedback gains between 0.1 Nm/deg and the stability limit were then each presented ten times in random order.

Prior to starting the trials with the negative spring load and again following their completion, subjects were asked to position the wrist in the target window in the absence of any load and to cocontract the wrist flexor and extensor muscles maximally to stiffen the wrist. Most subjects had no previous experience with the negative spring load. Subjects were given no practice with the negative spring load prior to the collection of the experimental data.

Experiment 2

Of the five subjects who participated in experiment 2, one had previously participated in experiment 1. The subjects performed a series of tasks, which included isometric contraction with the manipulandum locked at the target position and contraction against a load while maintaining the wrist in the target window. Two types of loads were used, either the negative spring, described above, or two parallel stiff springs (combined stiffness: 13.1 N/cm) attached to the manipulandum. The springs were stretched to produce large loads opposing either flexion or extension. The tasks are listed below in chronological sequence. Stiffness was measured only in tasks 3–5. Subjects rested for at least 30 s between each trial and were given longer rest periods when necessary to recover from fatigue. All subjects had practiced extensively with the negative spring load on different occasions prior to the experiment.

1. *Determination of maximal voluntary contraction (MVC)*: a maximal sustained contraction of the wrist flexor muscles and then of the wrist extensor muscles (duration 3–5 s). Contractions were isometric with the manipulandum locked.
2. *Pulsatile contraction*: five cocontractions at maximal effort, five maximal isometric contractions to flex the wrist, and five maximal isometric contractions to extend the wrist. The duration of each contraction was approximately 500 ms. The manipulandum was locked for isometric contraction, but not for cocontraction.
3. *Sustained cocontraction*: five cocontractions at maximal effort (duration more than 3 s).
4. *Negative spring load*: from two to four blocks of five trials with a negative spring load in order to determine the limit of stability. The negative stiffness of the load was usually set to −0.3 Nm/deg for the first block and incremented by −0.1 Nm/deg on each successive block until the subject was unable to successfully complete five trials. This value was taken as the limit of stability. This was the same type of load as used in experiment 1, but the protocol differed in that trials involving different feedback gains were presented systematically in blocks, rather than being presented in random order.
5. *Spring load*: one block each of five trials at 20%, 30%, more than 60%, 40%, and 50% of flexor MVC, and one block of five trials at more than 90% of extensor MVC. The actual percentage of MVC for the flexor torque (more than 60%) was chosen to match the extensor torque. For four of the five subjects the extensor load was between 91 and 97% MVC. One subject had difficulty maintaining the 90% level, so an 80% level was used in its place. The range for the flexor torque was 62–80% MVC. To prevent fatigue in these trials, the experimenter held the subject's wrist at the target position prior to the beginning of the trial and then released the wrist as soon as the target and cursor appeared on the screen.

Recording

Electromyographic (EMG) activity was recorded from six muscles contributing to torque at the wrist: flexor carpi radialis (FCR), flexor digitorum superficialis (FDS), flexor carpi ulnaris (FCU), extensor digitorum communis (EDC), extensor carpi radialis longus (ECRL), and extensor carpi ulnaris (ECU). The EMG was recorded using active, bipolar, stainless steel, surface electrodes (Liberty Mutual MYO 111) with a bandpass of 45–550 Hz. The electrode contacts were 3 mm in diameter and spaced 13 mm apart. Before the recording session began, the placement of the electrode over each wrist muscle was determined by observing the EMG activity during brisk test movements. These movements were: ulnar deviation and wrist extension for ECU, ulnar deviation and wrist flexion for FCU, finger flexion for FDS, finger extension for EDC, wrist flexion (fingers relaxed) for FCR, and wrist extension (fingers relaxed) for ECRL. Each electrode was placed so as to maximize the signal during the appropriate movement while minimizing the signal during other movements. The position, velocity and torque of the wrist and the EMG signals were amplified and digitized at 2 kHz.

Analysis

The EMG records from individual trials were rectified off-line and the mean amplitude of the rectified signal was computed over selected time intervals. In the case of isometric tasks, this was a single 200-ms interval, where the summed EMG from the agonist muscles was maximal. For all other tasks, four intervals were used: a 200-ms interval prior to the ramp displacement to compute background EMG and three consecutive intervals beginning 20 ms following the onset of the displacement, at short latency (20–60 ms), intermediate latency (60–100 ms) and long latency (100–150 ms).

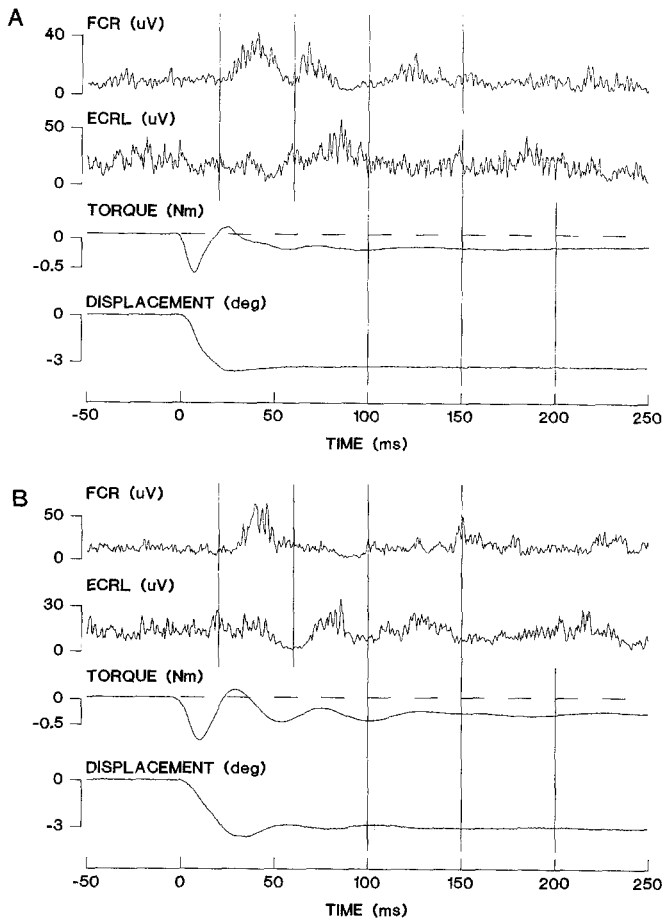


Fig. 1A, B Stabilization task with a negative spring load of −0.1 Nm/deg in experiment 1. All traces are means of ten trials. Traces 1 and 2 are rectified EMG activity recorded from flexor carpi radialis (*FCR*) and extensor carpi radialis longus (*ECRL*) muscles. Trace 3 is wrist torque, which is negative in the flexor direction. Trace 4 is wrist displacement with respect to the neutral position (0°) and is negative in the direction of wrist extension. Intervals for computation of reflex changes in EMG are indicated by *vertical lines* on EMG traces: short latency 20–60 ms; intermediate latency 60–100 ms; long latency 100–150 ms. *Vertical lines* on torque and displacement traces indicate 50-ms intervals used in calculating static wrist stiffness. The subject in **A** has both short- and intermediate-latency reflex responses in FCR, while the subject in **B** has only a short-latency response. Both subjects have an intermediate-latency excitatory reflex response in ECRL

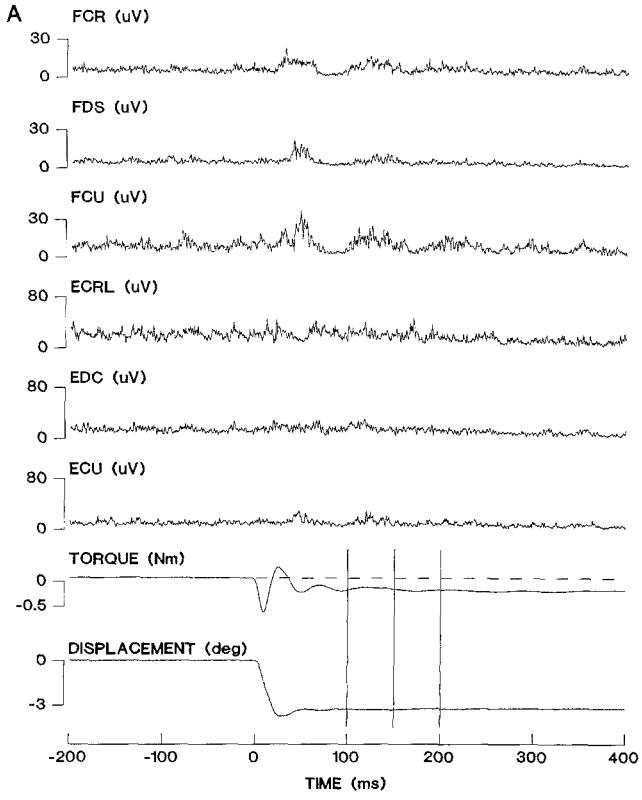
The division into three intervals was based on earlier observations of the time of occurrence and duration of short-, intermediate- and long-latency reflex responses (De Serres and Milner 1991), which corresponded closely to the myotatic, late myotatic and post-myotatic reflex responses of Jaeger et al. (1982). The reflex responses of two subjects are shown in Fig. 1. With such small amplitude stretches there was frequently little or no response in the wrist flexor muscles at the intermediate latency (Fig. 1B). This was consistent with our earlier observations (De Serres and Milner 1991).

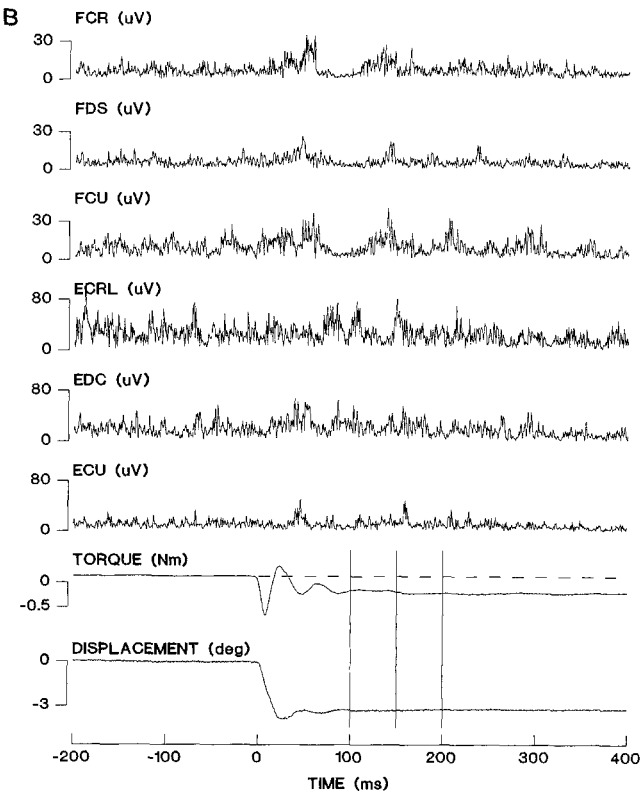
The change in EMG following the displacement was computed for each interval by subtracting mean rectified EMG prior to the displacement (background) from the mean rectified EMG over the reflex interval. This was also expressed as a percentage change by dividing by the background value. The mean change in EMG for the group of subjects was tested for significance using a *t*-test ($P < 0.05$).

The mean torque during stable posture before the displacement was computed and subtracted from the mean torque following the displacement. Static wrist stiffness was then computed by dividing this difference in wrist torque by the actual displacement. This was done for three separate intervals, namely, 60–100 ms, 100–150 ms and 150–200 ms following the onset of the displacement. During the earliest of these intervals there was usually some residual motion of the manipulandum before it stabilized at the final servo position. We quantified the residual motion by computing the variance in manipulandum position over each of the three intervals. The variance during the 60- to100-ms interval was about twice that for the 150- to 200-ms interval, during which the manipulandum position was most stable.

Differences in wrist stiffness or mean rectified EMG between different conditions were computed for individual subjects and the mean difference over all subjects was tested for significance using a *t*-test ($P < 0.05$) for means or paired comparisons. Linear regression analysis was carried out between mean rectified EMG, wrist stiffness and flexor torque. Slopes were tested for significance using a *t*-test ($P < 0.05$).

Fig. 2A, B Stabilization task with negative spring load for one subject in experiment 1. **A** Mean of ten trials for negative stiffness of −0.10 Nm/deg; **B** Mean of three trials for negative stiffness of −0.16 Nm/deg, the limit of stability for this subject. Traces 1–6 are rectified EMGs recorded from FCR, flexor digitorum superficialis (*FDS*) flexor carpi ulnaris (*FCU*), ECRL, extensor digitorum communis (*EDC*), and extensor carpi ulnaris (*ECU*) muscles, respectively. Traces 7–9 are wrist torque, angular velocity, and displacement, respectively. *Vertical lines* indicate 50-ms intervals used in calculating static wrist stiffness. There is more activity in all muscles in **B** than **A** and a larger increase in torque due to the displacement, i.e., greater wrist stiffness





Results

Experiment 1

Muscle cocontraction

Subjects stiffened the wrist by cocontracting wrist flexor and extensor muscles to counteract the negative stiffness of the load and stabilize the wrist. When the manipulandum was made more unstable by increasing the negative stiffness of the load, subjects responded by increasing the amount of antagonist cocontraction (Fig. 2). This was shown quantitatively by computing the ratio of the mean rectified background EMG recorded when the negative stiffness was set to the subject's limit of stability, to that recorded when it was set to −0.1 Nm/deg. The mean ratio for the ten subjects was significantly greater than 1 for all muscles (FCR 2.49, $P < 0.0005$; FDS 2.82, $P < 0.0025$; FCU 2.73, $P < 0.005$; ECRL 2.00, $P < 0.0005$; EDC 2.19, $P < 0.0005$; ECU 1.87, $P < 0.0025$).

Stiffness

The stiffness calculated over the 150- to 200-ms interval best represented the static stiffness of the wrist, because there was little or no residual motion of the manipulandum during this time (Figs. 1, 2). For this

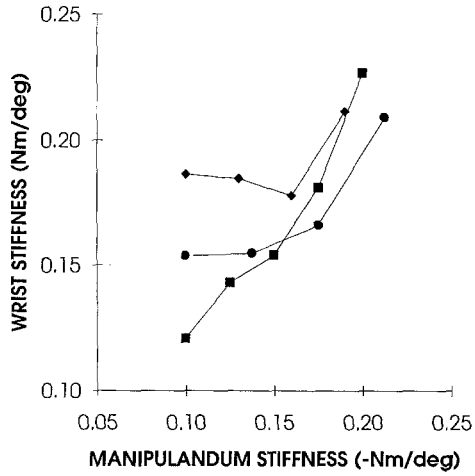


Fig. 3 Changes in wrist stiffness with increasing negative stiffness of the manipulandum. Different symbols represent the responses of three different subjects in experiment 1. The subject represented by the *diamonds* stiffened the wrist much more than necessary for low values of negative stiffness, whereas the subject represented by the *squares* used the minimum stiffness necessary to stabilize the manipulandum

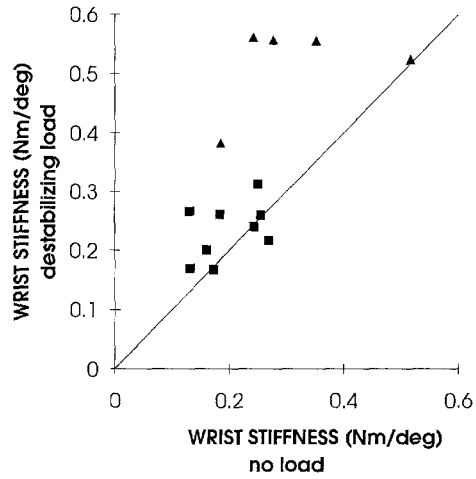


Fig. 4 Maximum wrist stiffness recorded when subjects stabilized the manipulandum under the negative stiffness condition (*destabilizing load*) is plotted against the maximum wrist stiffness recorded when subjects were asked to cocontract wrist muscles maximally in the absence of any load (*no load*). *Squares* represent subjects from experiment 1 and *triangles* represent subjects from experiment 2. The subject who participated in both experiments is represented by a single symbol (*triangle*). The *line* has a slope of 1, indicating points of equal stiffness. With one exception, all points lie on or above the line, indicating greater stiffness under the negative stiffness condition

reason, the comparisons between different conditions were all made using the stiffness values calculated for this interval. Using stiffness values calculated over the 100- to 150-ms interval for the comparisons gave similar results and led to the same conclusions, albeit with stiffness values that were consistently about 10% lower. Stiffness values were calculated for the 60- to 100-ms interval, as well, and found to be about 15–20% lower than those calculated for the 150- to 200-ms interval. However, they could not be considered reliable, due to residual manipulandum motion. Therefore, they were not used in analyzing the effects of increasing the negative spring load.

Stable equilibrium between the wrist and the negative spring load required that the wrist stiffness be greater than or equal to the magnitude of the load stiffness. However, subjects employed different strategies to achieve this result. At one extreme was a strategy in which wrist stiffness was maintained at a very high level for all values of negative load stiffness. In this case, the wrist was considerably stiffer than necessary when the magnitude of the negative stiffness was low. At the other extreme, subjects modulated the wrist stiffness such that it was approximately equal in magnitude to the negative stiffness of the load. Most subjects used a strategy intermediate between the two. The two extremes and an example of the intermediate strategy are shown in Fig. 3.

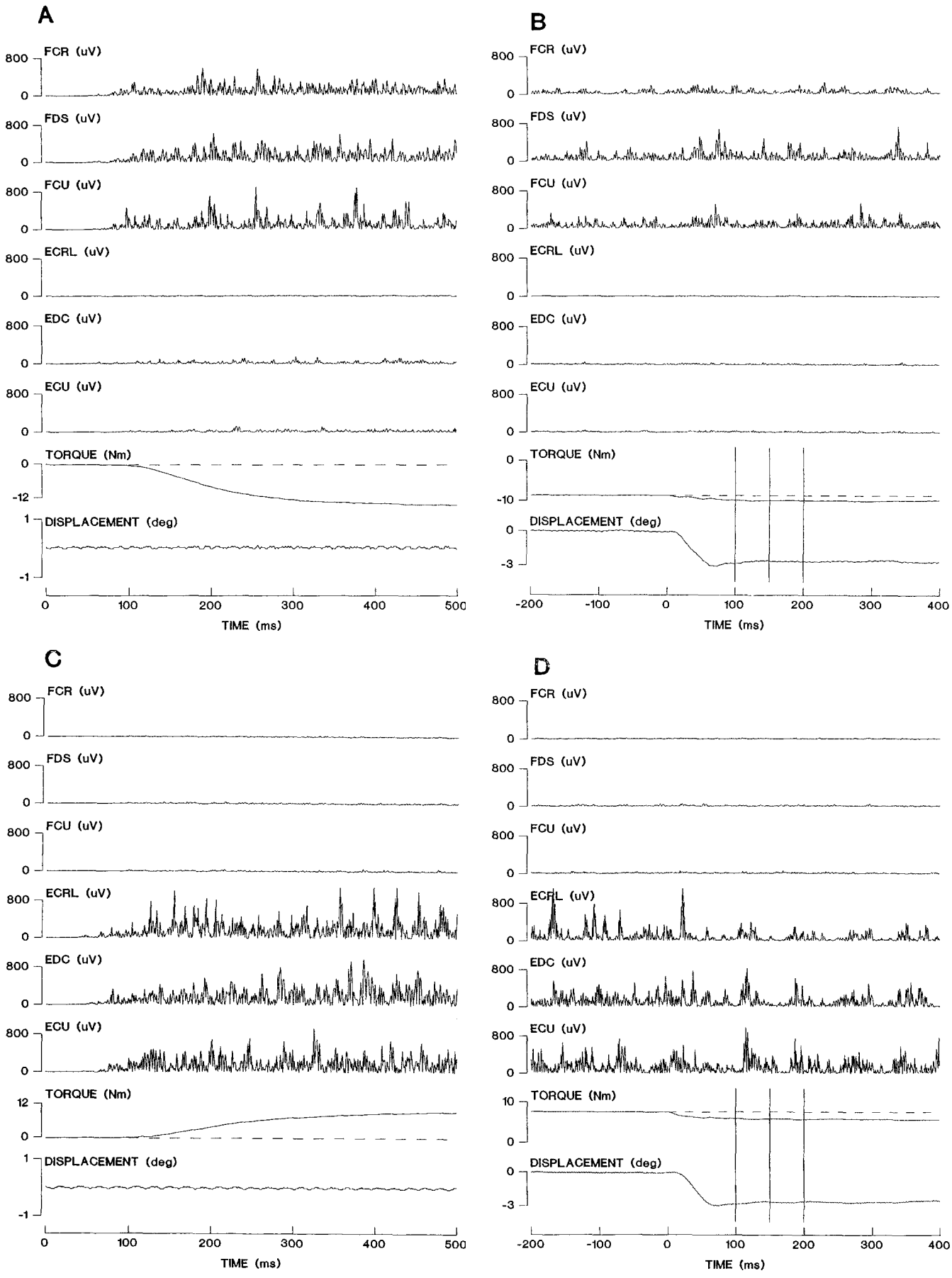
We combined the data from all conditions for each subject and computed the linear regression between the total EMG (sum of the mean rectified background EMG of all muscles) and wrist stiffness. The slope of the regression line was significantly greater than zero for nine of the ten subjects, the mean correlation coefficient being 0.93 (SD = 0.05, n = 9).

In order to determine whether subjects were capable of achieving higher wrist stiffness than during the load-stabilization task without producing a net joint torque, we asked them to stiffen the wrist maximally by voluntarily cocontracting flexor and extensor muscles when no load was applied. We measured the maximum stiffness that they could achieve in this way, both before they began the stabilization trials and after they

Table 1 Experiment 1: Reflex response as percentage change in mean rectified EMG (*NS* negative spring of −0.1 Nm/deg; *FCR* flexor carpi radialis, *FDS* flexor digitorum superficialis, *FCU* flexor carpi ulnaris, *ECRL* extensor carpi radialis longus, *EDC* extensor digitorum communis, *ECU* extensor carpi ulnaris)

Muscle	Load	Short latency (20–60 ms)	Intermediate latency (60–100 ms)	Long latency (100–150 ms)	n
FCR	NS	262	84	117	10
FDS	NS	175	124	14*	10
FCU	NS	170	83	106	10
ECRL	NS	−10*	84	4*	10
EDC	NS	−3*	81	22*	10
ECU	NS	−9*	74	12*	10

*Not significantly different from zero


A
FCR (uV)
FDS (uV)
FCU (uV)
ECRL (uV)
EDC (uV)
ECU (uV)
800
0
TORQUE (Nm)
0
-12
DISPLACEMENT (deg)
1
-1
0
100
200
300
400
500
TIME (ms)
B
FCR (uV)
FDS (uV)
FCU (uV)
ECRL (uV)
EDC (uV)
ECU (uV)
TORQUE (Nm)
0
-10
DISPLACEMENT (deg)
0
-3
-200
-100
0
100
200
300
400
TIME (ms)
C
FCR (uV)
FDS (uV)
FCU (uV)
ECRL (uV)
EDC (uV)
ECU (uV)
800
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TORQUE (Nm)
12
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DISPLACEMENT (deg)
1
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400
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TIME (ms)
D
FCR (uV)
FDS (uV)
FCU (uV)
ECRL (uV)
EDC (uV)
ECU (uV)
800
0
TORQUE (Nm)
10
0
DISPLACEMENT (deg)
0
-3
-200
-100
0
100
200
300
400
TIME (ms)

Fig. 5A–D Maximum isometric flexion (**A**); contraction against a flexor spring load of approximately −8 Nm (62% of the subject's isometric maximum) (**B**); maximum isometric extension (**C**); and contraction against an extensor spring load of approximately 8 Nm (97% of the subject's isometric maximum) (**D**). Records are single trials from one subject in experiment 2. Traces as in Fig. 2

had completed them. In comparing this value with the maximum stiffness achieved during the stabilization trials, we found that the latter was usually higher. As can be seen in Fig. 4, almost all of the data points for the subjects of experiment 1 lie on or above the line of slope 1.

Reflex responses

The reflex responses produced by the wrist displacement were quantified by computing the change in mean rectified EMG as a percentage of the background EMG prior to the displacement. The percentage change was computed for three different time intervals: 20–60 ms, 60–100 ms, and 100–150 ms after displacement onset (Fig. 1). Looking first at FCR and FCU, we found that there was a statistically significant increase in activity with respect to background at all latencies. This was highest at the shortest latency, declined substantially at intermediate latency, then increased somewhat at the longest latency. Mean values of the percentage change in mean rectified EMG for the ten subjects are listed in Table1.

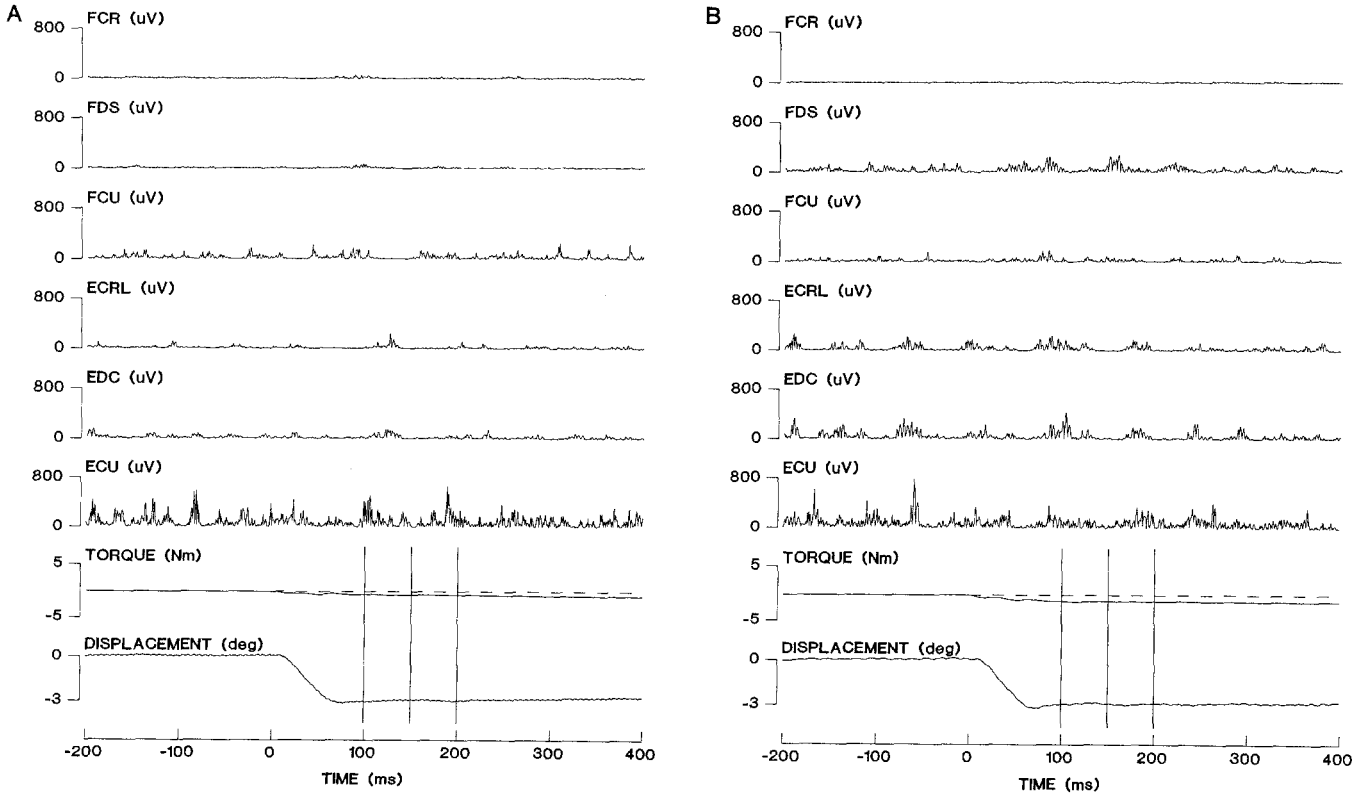
The responses in the finger flexor, FDS, were slightly different from FCR and FCU in that the activity did not increase at intermediate latency, but declined toward the background level like the wrist extensors. In all three extensors, we found a small decrease in activity with respect to background at short latency, although it was not statistically significant. This was followed by a statistically significant increase in activity at intermediate latency and a return to the background level at the longest latency (Table 1). The reflex responses observed at the limit of stability (not listed) were essentially the same as those observed for the negative spring load of −0.1Nm/deg, although reduced in magnitude. However, only in the case of the FDS was the reduction in activity significant ($P < 0.05$).

Experiment 2

Stiffness

The wrist stiffness measured in experiment 1, where subjects performed maximal cocontraction of wrist flexor and extensor muscles, was seemingly much lower than we would have predicted based on simultaneous

Fig. 6A, B Maximal cocontraction with no load (**A**); maximal cocontraction while stabilizing a negative spring load of −0.4 Nm/deg (**B**). Records are single trials from the same subject as in Fig. 5; all traces plotted to the same scale as Fig. 5



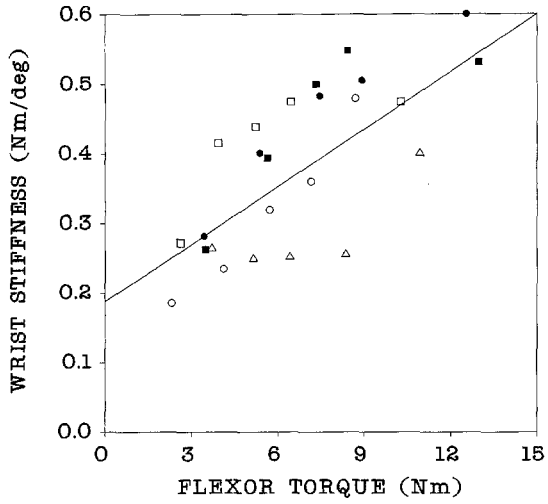


Fig. 7 Linear regression for static wrist stiffness as a function of wrist flexor torque for combined data from all five subjects in experiment 2 (slope 0.0294; correlation coefficient 0.73). Stiffness was measured over the interval 150–200 ms following onset of wrist displacement. Different symbols represent data from different subjects

sustained MVC of wrist extensor muscles and matching contraction of wrist flexor muscles. We, therefore, designed a second experiment to investigate this further. In experiment 2 we made a direct comparison between the wrist stiffness generated by near-maximal voluntary activation of wrist extensor muscles, the stiffness generated by a matching contraction of the wrist flexor muscles, and the stiffness generated by maximal cocontraction of wrist extensor and flexor muscles. Subjects first performed maximal cocontractions and then sustained contractions against a stiff spring load opposing either extensor or flexor muscles. The wrist stiffness was measured by applying a 3° displacement, which stretched the flexor muscles and shortened the extensor muscles (Figs. 5 and 6).

As noted above, the interval 150–200 ms following the onset of wrist displacement was determined to be the most appropriate interval for the calculation of static wrist stiffness. Using the 100- to 150-ms interval gave similar results, although the stiffness values were typically 5–10% higher under all conditions. Stiffness values for the 60- to 100-ms interval were about 25% lower than those for the 150- to 200-ms interval. The lower values are due in part to the fact that the final servo position was not always reached within 60 ms. Therefore, they were not used in the analysis.

In a mechanical system consisting of linear springs arranged in parallel, the total stiffness is equal to the sum of the stiffnesses of the parallel springs. For small amplitude displacements, human joints behave mechanically like second-order linear systems, where antagonist muscles are arranged in parallel opposition (Kearney and Hunter 1990). Thus, the maximum static stiffness of the wrist, achievable by balanced (zero resultant torque) cocontraction of wrist flexor and extensor muscles, would be expected to equal the sum of the extensor stiffness and the flexor stiffness.

The mean flexor stiffness was 0.50 Nm/deg and the extensor stiffness was 0.79 Nm/deg, giving a maximum combined stiffness of 1.29 Nm/deg. In contrast, the stiffness during cocontraction at the limit of stability was 0.56 Nm/deg, while the stiffness when stiffening the wrist without load was only 0.34 Nm/deg. Thus, the stiffness actually achieved by cocontraction was significantly less than the predicted achievable value ($P < 0.001$). It can be noted, as well, that the subjects of experiment 2 (included in Fig. 4) performed similarly to those of experiment 1 in that the maximum wrist stiffness achieved by cocontraction was greater when stabilizing the negative spring load than when stiffening the wrist without load ($P < 0.05$).

There was a strong linear correlation between wrist stiffness and flexor torque. The combined data from the five subjects is plotted in Fig. 7. The slope of the regression line is 0.0294 and the correlation coefficient is 0.73. There was evidence of saturation of wrist stiffness at high levels of flexor torque for two subjects (represented by the open and closed squares).

Table 2 Experiment 2: Reflex response as percentage change in mean rectified EMG (*NS* negative spring; *FS* flexor spring, *ES* extensor spring)

Muscle	Load	Short latency (20–60 ms)	Intermediate latency (60–100 ms)	Long latency (100–150 ms)	*n*
FCR	NS	61	234	20*	5
FDS	NS	61	187	47	5
FCU	NS	45	185	35*	5
ECRL	NS	−7*	18*	6*	5
EDC	NS	−6*	25	13*	5
ECU	NS	−5*	28*	23*	5
FCR	FS	27*	60	38	5
FDS	FS	54	182	45	5
FCU	FS	39	91	21	5
ECRL	ES	−9*	14*	3*	5
EDC	ES	−1*	21*	12*	5
ECU	ES	−8*	34	5*	5

*Not significantly different from zero

Muscle cocontraction

In experiment 1 we noted a strong correlation between total EMG and static wrist stiffness. This correlation held, as well, in experiment 2, where the different conditions produced a wider range of muscle activation levels. We combined the data from all conditions and computed the linear regression between total EMG (sum of the background EMG of all muscles) and wrist stiffness. The mean correlation coefficient was 0.82 (SD = 0.11, $n = 5$). Based on this correlation, we expected to find less muscle activation in both flexor and extensor muscles when cocontracting maximally than when contracting reciprocally against the largest spring loads (Figs. 5, 6). This was confirmed by comparing background EMG, normalized with respect to its value when the muscle was contracting against the largest spring load. The mean rectified EMG was greater when a muscle was contracting reciprocally than during cocontraction (FCR $P < 0.0005$; FDS $P < 0.05$; FCU $P < 0.005$; ECRL $P < 0.0025$; EDC $0.05 < P < 0.1$; ECU $P < 0.0025$).

One potential explanation for the inability to maximally activate antagonistic muscle groups during cocontraction is central fatigue. It might not be possible to sustain maximal neural drive to a large number of muscles for any length of time. To test whether the duration of the contraction was a contributing factor, we asked subjects to perform brief maximal isometric contractions. We found that the flexor EMG was higher during brief reciprocal contractions than during brief cocontractions ($P < 0.005$). This was fully expected, because the flexor muscles, being stronger than the extensors, could not be maximally activated without producing movement. However, the extensor EMG was also higher during brief reciprocal contractions than during cocontractions (ECRL $P < 0.0125$; EDC $P < 0.05$; ECU $P > 0.3$). Consequently, central fatigue is unlikely to have played a significant role in the failure to achieve maximal muscle activation during cocontraction.

In adding flexor and extensor stiffness to obtain total wrist stiffness, we made the implicit assumption that sustained contractions against spring loads were purely reciprocal. However, this may not have always been the case. In some subjects (not shown) we observed surprisingly high levels of activity when muscles were functioning as antagonists. This may have been due, in large part, to the need to balance finger flexor and extensor torques and torques producing radial and ulnar deviation of the wrist. The mean rectified EMG when these muscles were acting as antagonists was, on average, about 20% of that recorded when the same muscles were functioning as agonists. Similar observations regarding cocontraction have been made during isometric contractions of elbow flexor muscles (Hébert et al. 1991) and thumb flexor muscles (Capaday et al. 1994). It should be pointed out, though, that we were recording with surface electrodes and part of the antagonist EMG may have simply been cross-talk from the strongly activated agonists.

We found a strong linear correlation between the sum of the mean rectified background EMG of the three wrist flexor muscles and the wrist flexor torque. The mean correlation coefficient was 0.98 (SD = 0.01, $n = 5$). Because the extensor muscles appeared to be coactivated with the flexor muscles during flexor efforts against the spring load, we also performed linear regression between the sum of the mean rectified background EMG of the three extensor muscles and the wrist flexor torque. Again, we found a strong linear correlation with a mean correlation coefficient of 0.92 (SD = 0.06, $n = 5$).

Reflex responses

As in experiment 1, reflex responses were quantified by computing the change in mean rectified EMG as a percentage of the background EMG prior to the displacement. When subjects were cocontracting at their limit of stability, the displacement, which stretched flexor muscles and shortened extensors, produced a short-latency increase in the activity of flexor muscles and a decrease in the activity of extensors (Table 2). Although the increase in flexor activity was significant, it was not as large a change as in experiment 1. However, the increase in activity of flexor muscles at intermediate latency was much larger, while at the longest latency it was considerably smaller (except for FDS), compared with experiment 1. The increase in activity of extensor muscles at intermediate latency was somewhat smaller than in experiment 1, but similar at the longest latency.

We quantified the reflex responses observed in flexor muscles, when exerting a flexor effort against a spring load, and those observed in extensor muscles, when exerting an extensor effort against a spring load, in the same way. The percentage change for the three response intervals are listed in Table 2 for the largest spring loads used in this study. The magnitude of the changes in activity are similar to those observed during cocontraction.

We also examined the reflex activity (percentage change with respect to background, as above) of flexor muscles in relation to the size of the spring load. The increase in flexor activity at short and intermediate latency was greatest for the smallest load (20% MVC) and tended to decline progressively as the load increased, the decline being considerably greater for the short-latency than the intermediate-latency response. In contrast, the change in activation at the longest latency was relatively unaffected by the size of the load.

We tried to determine whether the difference between predicted maximum stiffness and maximum stiffness measured during cocontraction might be due to a

difference in reflex gains. To test this possibility we determined the flexor spring load for each subject, where the background EMG of a particular flexor muscle most closely matched that during maximal cocontraction. We then performed a paired-comparisons test between the mean rectified EMG under the two conditions for each of the three reflex intervals. For none of the three flexor muscles was there a significant difference between the reflex activity when contracting against a spring load and when cocontracting maximally for any of the three reflex intervals ($P > 0.1$). This suggests that there was no significant difference in the reflex gain at any latency.

Discussion

The task of stabilizing the manipulandum when it was mechanically unstable due to negative stiffness required a subject to stiffen the wrist sufficiently that the sum of the positive wrist stiffness and negative stiffness of the load was greater than zero. This was achieved by cocontraction of wrist flexor and extensor muscles. However, as the negative stiffness of the load was increased, a limit was reached beyond which the subject could no longer stabilize the manipulandum by means of cocontraction. Beyond this point, the subject could no longer maintain the manipulandum in the target window for the required time. The maximum wrist stiffness that was achieved by cocontraction was found to be substantially less than predicted on the basis of MVC of wrist extensor muscles. This was not due to a smaller reflex contribution to static wrist stiffness, but rather to the inability to activate muscles to the same level during cocontraction as during reciprocal activation.

Maximum wrist stiffness

Assuming that the static stiffness of flexor and extensor muscles could be summed linearly in determining total wrist stiffness, and that flexor and extensor muscles could be activated maximally during cocontraction, we calculated that the wrist stiffness should have been more than double what we measured when subjects were maximally cocontracting these muscles. It is unlikely that the summation of flexor and extensor stiffness was non-linear, since the static mechanical behavior of human joints is quite linear in response to small-amplitude displacements of the type used in the present study (Kearney and Hunter 1990). The predicted maximum stiffness was based on measurements of wrist stiffness while subjects reciprocally activated either flexor or extensor muscles. As a first approximation we attributed all of the measured stiffness exclusively to one muscle group or the other. We did not take into account that both muscle groups could be contributing to the stiffness even during reciprocal activation. This assumption is obviously incorrect, since the passive stiffness of the antagonist muscles was always present. In adding the stiffness of flexor and extensor muscles, we counted the passive stiffness twice. In an earlier study with 12 subjects (De Serres and Milner 1991) we found the mean value of the passive wrist stiffness to be 0.06–0.07 Nm/deg. If we subtract 0.07 Nm/deg from the summed stiffness we are left with 1.22 Nm/deg, still more than double the stiffness measured during maximal cocontraction.

Active stiffness contributed by antagonist muscles during reciprocal activation could also have accounted for the greater stiffness when summing contributions from flexor and extensor muscles. We did measure a significant amount of EMG activity in antagonist muscles – approximately 20% of that recorded when they were functioning as agonists in the same task. Some of the antagonist muscle activity may have been due to cross-talk from the agonist muscles whose activation levels were very high, since surface EMG recording is particularly susceptible to this type of cross-talk (Koh and Grabiner 1993). However, the electrodes that we used are relatively immune to cross-talk when recording from trim subjects, as in experiment 2 (de la Barrera and Milner 1994). Consequently, we will assume that none of the antagonist EMG could be accounted for by cross-talk. As we showed, in the case of wrist flexor muscles, that the mean rectified EMG was proportional to wrist stiffness, we can conclude, in the worst case, that antagonist muscles contributed 20% of the active stiffness measured when contracting against the largest spring loads. If we assume again that the passive stiffness is 0.07 Nm/deg, the total active stiffness would be 1.15 Nm/deg. Reducing this by 20% and adding back the passive stiffness gives 0.99 Nm/deg, still much greater than 0.56 Nm/deg, the stiffness measured during maximal cocontraction.

Reflex contributions to wrist stiffness

The stiffness that we measured comprised both stiffness due to intrinstic muscle mechanics and stiffness due to reflex feedback. Our protocol did not allow us to separate the contributions from the two sources. However, there are several observations which are pertinent to addressing this issue.

Short-latency response

The peak in the short-latency reflex response occurred 35–40 ms after the onset of wrist displacement. Given that the twitch tension rise time is in the order of 60 ms (Riek and Bawa 1992) one would expect the peak torque produced by the short-latency response to occur approximately 100 ms after stretch onset. This may

have contributed to the higher stiffness values which we measured during the interval 100–150 ms following the onset of wrist displacement compared with the 60–100 ms interval. Observations of the delay between EMG onset and onset of joint acceleration suggest that joint torque develops within 10 ms of electrical activation even in muscles with long tendons (Milner 1986). Consequently, the short-latency reflex would have been contributing to the wrist torque within 30–35 ms after the onset of the wrist displacement, so the difference in stiffness between the two intervals is probably an underestimate of the reflex contribution. Movement of the manipulandum during the 60- to 100-ms interval is unlikely to have introduced a significant error, since it tended to be relatively slow and the effects of viscosity and inertia would have offset each other while the manipulandum was decelerating.

Intermediate-latency response

In experiment 2, the wrist stiffness for the 100- to 150-ms interval was as high or higher than that for the 150- to 200-ms interval, whereas in experiment 1 it was lower. This corresponds with the greater increase in reflex EMG seen in the interval 60–100 ms following the displacement in experiment 2 compared with experiment 1 and suggests that the intermediate-latency reflex response also contributed noticeably to the static wrist stiffness. We observed little or no decline in wrist torque between 100 and 200 ms following the onset of the displacement.

The reflex and mechanical responses were consistent across tasks involving reciprocal contraction and cocontraction of wrist muscles. There was no evidence that the reflex contribution to static stiffness was reduced by cocontraction. This conclusion is also supported by the results of Carter et al. (1993), which suggest that during cocontraction of antagonistic muscles there can be greater than linear summation of the reflex component.

In both experiments we observed intermediate latency reflex excitation of wrist extensor muscles which were caused to shorten by the manipulandum displacement. This excitation occurred both during reciprocal activation of wrist extensor muscles and during cocontraction of wrist flexor and extensor muscles. In the case of cocontraction, it sometimes occurred even in the absence of an intermediate-latency response in the wrist flexor muscles (Fig. 1B). Reflex cocontraction of wrist muscles at a similar latency has been reported in other stabilization tasks of this nature (Lacquaniti et al. 1991; Goodin and Aminoff 1992; Chequer et al. 1994). Although such reflex cocontraction would not serve to return the wrist to its original position, it would increase the wrist stiffness subsequent to perturbation.

Influence of mechanical parameters on reflex responses

The differences in the percentage increase in reflex activation of wrist flexor muscles between experiments 1 and 2 are probably consequences of the lower stretch velocity and higher wrist stiffness achieved by subjects in experiment 2 (Fig. 4). Lee and Tatton (1982) have shown that high-velocity, short-duration stretch produces a considerable reflex response of wrist flexor muscles at short latency, but relatively little response at intermediate latency. On the other hand, longer-duration stretch of the same amplitude leads to a reduced short-latency response and an increased intermediate-latency response, as we observed. The higher muscle stiffness of the subjects in experiment 2 would have had a similar effect to the reduced stretch velocity, since a greater proportion of the applied stretch would have been taken up by the tendons in experiment 2 compared with experiment 1. In addition, it is likely that the subjects in experiment 2 were able to achieve a greater percentage of maximum muscle activation during cocontraction than the subjects in experiment 1, accounting for their greater wrist stiffness (Fig. 4). This would also have contributed to a smaller percentage increase in reflex activity at shorter latency, based on our observation that the response became proportionately smaller as the torque produced by flexor muscles increased.

Inhibition of muscle activation during cocontraction

It was evident from comparison of forceful flexor or extensor contractions with maximal cocontraction that, whether the contraction was sustained or brief, the EMG was always less during cocontraction. This and the observation that stiffness was lower than predicted during maximal cocontraction suggest that at least one of the two antagonist muscle groups was significantly inhibited during cocontraction. Several other studies have provided similar evidence of reduced muscle activation during cocontraction. Tyler and Hutton (1986) showed that the activity of biceps and triceps muscles was lower during cocontraction than during maximal isometric contraction. Jongen et al. (1989) showed that subpopulations of motor units within the biceps muscle behaved differently during flexion and cocontraction tasks, one of which was apparently inhibited during cocontraction of the triceps muscle. Kearney and Hunter (1990) refer to a preliminary study in which they found that subjects had difficulty in achieving high levels of muscle coactivation while producing zero net torque at the ankle.

The physiological mechanism responsible for limiting the level of muscle activation during cocontraction may involve postsynaptic inhibition mediated by interneurons or propriospinal neurons in the spinal

cord or it may originate from the voluntary commands issued by the motor centers in the brain.

The most likely source of postsynaptic inhibition would be reciprocal inhibition from group Ia afferents. There are at least two ways that this could occur. First, there is evidence in humans and monkeys that electrical or magnetic stimulation of the motor cortex, which evokes activity in upper or lower limb muscles, facilitates reciprocal inhibition of the antagonist muscles (Jankowska et al. 1976; Rothwell et al. 1984; Iles and Pisini 1992; Nielsen et al. 1993). This suggests that cortical commands, sent to simultaneously activate antagonistic muscle groups (cocontraction), could produce reciprocal inhibition that would reduce their potential maximum activation.

A second possibility is that the Ia afferents originating from muscle spindles are strongly activated during isometric contraction. Under the quasi-isometric conditions of our experiments this could only have arisen if there had been significant fusimotor drive to the muscle spindles, i.e., strong α–γ coactivation, but, as Hagbarth et al. (1975) have demonstrated, large increases in spindle discharge do occur when the receptor-bearing muscle is activated while being held isometric.

Alternatively, the inhibition may originate from within the motor centers of the brain. Humphrey and Reed (1983) identified two different classes of task-related cells in the precentral motor cortex of the monkey, related either to reciprocal activation or cocontraction of wrist flexor and extensor muscles. Subsequently, De Luca and Mambrito (1987) provided evidence of common central drive to flexor and extensor muscles of the thumb during cocontraction. This suggests the possibility that descending pathways activated during cocontraction project to only a portion of the flexor and extensor motoneuron pools.

Although it is clear that some form of inhibition, most likely reciprocal in nature, limits the amount that antagonisitc muscle groups can be coactivated, we cannot distinguish between the possible alternatives. The inhibition may be of peripheral origin, central origin, or a combination of the two.

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