Exp Brain Res (2002) 143:406–416
DOI 10.1007/s00221-002-1001-4

RESEARCH ARTICLE

Theodore E. Milner

Adaptation to destabilizing dynamics by means of muscle cocontraction

Received: 11 December 2000 / Accepted: 7 December 2001 / Published online: 9 February 2002

Abstract Adaptive control of wrist mechanics was investigated by means of destabilizing dynamics created by a torque motor. Subjects performed a 20° movement to a 3° target under the constraint that no motion should occur outside of the target zone once 800 ms had elapsed from movement onset. This constraint served as the minimum acceptable level of postural stability. The ability of subjects to modify their muscle activation patterns in order to successfully achieve this stability was investigated by creating three types of destabilizing dynamics with markedly different features: negative stiffness, negative damping, and square-wave vibration. Subjects performed sets of trials with the first type of destabilizing dynamics and were then required to adapt to the second and third. The adaptive response was quantified in terms of the rms electromyographic (EMG) activity recorded during various phases of the task. Surface EMG activity was recorded from three muscles contributing to wrist flexion and three muscles contributing to wrist extension. With negative stiffness, a significant compensatory increase in cocontraction of wrist flexor and extensor muscles was observed for slow movements, but there was little change in the muscle activity for rapid movements. With negative damping, muscle cocontraction was elevated to stabilize rapid movements, declining only gradually after the target was reached. For slow movements, cocontraction occurred only when negative damping was high. The response to square-wave vibration (10 Hz, ±0.5 Nm), beginning at movement onset, was similar to that of negative damping, in that it resulted in elevated cocontraction. However, because the vibration persisted after the target was reached, there was no subsequent decrease in muscle activity. When the frequency was reduced to 5.5 Hz, but with the same torque impulse, cocontraction increased. This is consistent with greater mechanical instability. In summary, agonist-antagonist cocontraction was adapted to the stability of the task. This generally resulted in less of a change in muscle activity during the movement phase, when the task was performed quickly compared with slowly. On the other hand, the change in muscle activity during stabilization depended more on the nature of the instability than the movement speed.

Keywords Mechanical instability · Wrist · Stiffness · Damping · Vibration · Human

T.E. Milner (✉)
School of Kinesiology, Simon Fraser University, Burnaby, British Columbia, Canada V5A 1S6
e-mail: tmilner@sfu.ca
Tel.: +1-604-2913499, Fax: +1-604-2913040

Introduction

Cocontraction of antagonistic muscle groups is a strategy used to increase the stiffness of a joint (De Serres and Milner 1991; Milner et al. 1995). It also increases damping, albeit to a lesser degree than stiffness (Milner and Cloutier 1998). The overall effect is to increase the mechanical stability of the joint, but at greater metabolic cost. Hogan (1984) has pointed out that increased joint stiffness could be achieved more economically through reflex feedback, but that the inherent delays would threaten mechanical stability. Thus, while cocontraction may not be the most efficient means of achieving mechanical stability, it may often be the most appropriate.

Given that cocontraction is metabolically costly, one would expect it to be used sparingly and that the level of cocontraction would be regulated in proportion to the level of mechanical instability. This does indeed appear to be the case. Parsimonious use of cocontraction occurs during ball catching (Lacquaniti and Maioli 1987), where flexor and extensor muscles at the wrist and elbow are briefly coactived prior to and following impact of the ball with the hand. Proportionate cocontraction has been reported for ankle muscles, while balancing in increasingly unstable postures (Houtz 1964); for elbow muscles, as the line of action of a hand-held weight is varied with respect to the center of joint rotation (Hogan 1984); and for wrist muscles, in response to a position-

dependent load of increasing instability (Milner et al. 1995). Milner and Cloutier (1998) have investigated the ability of subjects to perform goal-directed movements in the presence of negative damping. They have shown that subjects increase cocontraction of flexor and extensor muscles in proportion to the amount of negative damping. They have also shown that cocontraction gradually declined as oscillations about the final position were attenuated.

Cocontraction also appears to decrease as skill improves (Clément and Rézette 1985). Milner and Cloutier (1993) have shown that the amount of cocontraction was reduced following practice, without loss of mechanical stability. Thoroughman and Shadmehr (1999) have introduced an index to quantify the amount of unnecessary activation by deriving the minimum necessary muscle activation from model simulations. They have shown that this unnecessary activation, which they term "wasted" contraction, decreases as subjects become more proficient at moving in a perturbing force field.

In previous studies, we have shown that the amount of cocontraction increases with load instability, that joint stiffness and damping increase with cocontraction, and that the amount of cocontraction decreases with practice (Milner and Cloutier 1993, 1998; Milner et al. 1995). The objective of the present study was to determine how temporal patterns of agonist-antagonist cocontraction are adapted to the nature of the destabilizing dynamics. Specifically, changes in the temporal features of agonist-antagonist cocontraction of wrist muscles were examined when the nature of the destabilizing dynamics was varied. Negative stiffness was used to create a position-dependent instability, and negative damping was used to create a velocity-dependent instability. Square-wave vibration was used to create the type of instability that might be encountered in using reciprocating power tools. This type of instability is not dependent on movement kinematics. Both movement speed and the type of destabilizing dynamics were shown to have a significant effect on the temporal pattern of muscle activation used to stabilize the wrist.

Materials and methods

Eleven normal, healthy subjects (1 woman and 10 men, ranging in age from 16 to 47 years) participated voluntarily in this study. All except one subject were right-handed. The experiments were carried out with the right hand only. All of the subjects gave their informed consent to the procedure, which was approved by the Research Review Committee of the Institut de Réadaptation de Montréal and conformed to the Declaration of Helsinki. Subjects were required to move a wrist manipulandum from an initial position to a final target position at two different speeds.

Apparatus

A torque motor (PMI U16M4) applied torque to the wrist manipulandum under computer control. Although the motor could produce up to 5 Nm of torque, the loads used in this study did not exceed 1 Nm, which represented less than 15% of the subjects' maximum voluntary torque. The position and velocity of the motor 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.

The torque motor had negligible viscosity, i.e., there was no measurable velocity-dependent torque when the motor was rotated at different speeds. There was, however, a small amount of friction (0.05 Nm), which was constant at all speeds. The friction torque is sometimes evident as a slight positive offset in torque records during and following movement. The torque motor was used to generate three different types of mechanical instability. A position-dependent instability was generated by means of positive position feedback to the torque motor (negative stiffness). A velocity-dependent instability was generated by means of positive velocity feedback (negative damping). An instability, which was independent of movement kinematics, was generated by means of a square-wave command (vibration) to the motor. All three types of instability had the common feature that no net wrist torque was required at the target position, although stiffness and/or damping were necessary for stable equilibrium. In the case of the position-dependent instability, there was a load throughout the movement equal to the position feedback gain (negative stiffness) multiplied by the distance to the target. This torque pushed the wrist away from the target and dropped to zero in a linear fashion as the target was approached. In the case of vibration, the objective was to produce a fast muscle stretch and thereby elicit a strong monosynaptic reflex response. Consequently, square-wave pulses were used, rather than sinusoidal oscillation, because of their greater effectiveness as a stimulus to primary muscle spindle afferents.

Values of feedback gain for negative stiffness and damping were selected to be within the limits of loads which could be stabilized, based on earlier studies of wrist movement (Milner and Cloutier 1998; Milner et al. 1995), with the constraint that the maximum command to the motor should not exceed 1 Nm. The stability limits for male subjects were previously determined to be approximately $-14\ \mathrm{Nm \cdot rad^{-1}}$ and $-0.11\ \mathrm{Nm \cdot s \cdot rad^{-1}}$ for negative stiffness and negative damping, respectively. For comparison, stiffness of the relaxed wrist is about $3\ \mathrm{Nm \cdot rad^{-1}}$ (De Serres and Milner 1991), while damping is 0.02–0.03 $\mathrm{Nm \cdot s \cdot rad^{-1}}$ (Gielen and Houk 1984). Vibration frequencies were chosen to focus on two aspects of the neuromuscular system which can affect stability. A frequency of 5.5 Hz was chosen to approximate the natural frequency of the wrist under the no-load condition, while a frequency of 10 Hz was chosen to produce a destabilizing response from the monosynaptic stretch reflex. Vibration amplitudes were chosen to produce the same torque impulse at both frequencies.

Protocol

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 moved the manipulandum by applying force to two curved pads, which were securely clamped around the thumb and palm. These pads were positioned to align the axis of rotation of the wrist over the motor axis. The subject did not grip the manipulandum, but could cocontract the finger flexor and extensor muscles, which crossed the wrist, to increase wrist stiffness.

The subject was required to move a hairline cursor from an initial zone on the right side of a computer screen to a target zone on the left side by flexing the wrist. The initial zone was 1° wide, the target zone was 3° wide, and the center-to-center separation of the zones was 20°. The cursor position on the screen corresponded to the angular position of the wrist.

Prior to initiating a movement, the subject held the manipulandum within the initial zone for 1 s. Subjects were instructed to move the wrist to the target position, then to reduce any oscillations about the target position as quickly as possible, so as to

Table 1 Movement parameters: speed and load combinations

Condition	Speed	Load
1	Fast	No Load
2	Slow	No Load
3	Fast	Negative stiffness –1.15 Nm·rad^{-1}
4	Slow	Negative stiffness –1.15 Nm·rad^{-1}
5	Fast	Negative stiffness –2.29 Nm·rad^{-1}
6	Slow	Negative stiffness –2.29 Nm·rad^{-1}
7	Fast	Negative damping –0.0573 Nm·s·rad^{-1}
8	Slow	Negative damping –0.0573 Nm·s·rad^{-1}
9	Fast	Negative damping –0.0859 Nm·s·rad^{-1}
10	Slow	Negative damping –0.0859 Nm·s·rad^{-1}
11	Fast	Vibration 10 Hz
12	Slow	Vibration 10 Hz
13	Fast	Vibration 5.5 Hz
14	Slow	Vibration 5.5 Hz

achieve a stable posture within 800 ms of movement onset. Movement duration was set at 550 ms for slow movements and 100 ms for fast movements. Movement duration was defined as the time taken to move the 18° between the boundaries of the initial zone and the target zone. Subjects were provided with information about movement duration immediately after each trial. A horizontal bar, with length proportional to movement duration, was displayed at the bottom of the screen with the target duration displayed just below it. A trial was considered successful only if the movement duration was within ±20% of the target duration and if the subject met the "stability" criterion, i.e., there was no movement outside of the target zone after the expiration of the 800-ms time limit. Movement outside of the target zone, e.g., due to overshooting or oscillation, was permitted prior to this time.

Subjects were presented with 14 different movement conditions, comprising the combinations of load and speed listed in Table 1. There were two levels of instability for each load type. The conditions were presented to all subjects in the order listed in Table 1. Subjects completed all trials for a given condition before going on to the next condition. For each condition, subjects first performed 25 practice trials. The first five successful trials after the practice trials were recorded for analysis.

The surface electromyogram was recorded from six wrist muscles: 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). Liberty Mutual MYO 111 electrodes were used, which bandpass-filtered the signal, with nominal cutoff frequencies of 45 Hz and 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: combined ulnar deviation and wrist extension for ECU, combined 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 absence of cross-talk in the raw EMG was confirmed by post hoc cross-correlation analysis between pairs of muscles. Even in condition 9, where EMG amplitude was large and strongly modulated, the absolute value of the cross-correlation was less than 0.2 in 56 of 60 cases and did not exceed 0.36. The mean cross-correlation across subjects and homologous muscle pairs for condition 9 was not significantly different from zero (–0.011, SD 0.17).

The position, velocity and torque of the wrist and the EMG signals were amplified and digitized at 2 kHz. Data acquisition was post-triggered when the subject had moved 0.5° beyond the boundary of the initial target zone. Data were acquired for 250 ms prior to the trigger and for 1,500 ms after the trigger.

Analysis

Temporal EMG patterns were quantified by computing the rms values during four phases of the task, comprising a premovement epoch (250–200 ms prior to movement onset), a movement epoch (125 ms prior to movement onset until peak velocity, in the case of flexor muscles, or until the first velocity zero crossing, in the case of extensor muscles), a stabilization epoch (end of the movement interval until 800 ms after movement onset) and a poststabilization epoch (800–1,500 ms after movement onset). The rms of the background noise (EMG signal with relaxed muscles) was subtracted prior to further analysis.

The temporal activation patterns of homologous muscles were found to be very similar (Fig. 1). The similarity was quantified by computing the correlation in activation between pairs of homologous muscles. The EMG was first rectified, the five trials for each condition were then averaged and low-pass filtered at 10 Hz, after which the correlation coefficient was computed. The mean correlation coefficients across conditions, for all subjects combined, were: FCR versus FDS 0.90 (SD 0.024), FCR versus FCU 0.86 (SD 0.039), FDS versus FCU 0.87 (SD 0.030), ECRL versus EDC 0.81 (SD 0.064), ECRL versus ECU 0.72 (SD 0.059), and EDC versus ECU 0.77 (SD 0.030) for fast movements; FCR versus FDS 0.75 (SD 0.093), FCR versus FCU 0.56 (SD 0.13), FDS versus FCU 0.57 (SD 0.12), ECRL versus EDC 0.58 (SD 0.11), ECRL versus ECU 0.43 (SD 0.16), and EDC versus ECU 0.51 (SD 0.17) for slow movements. The high temporal correlations for fast movements indicate that there is common modulation of activity in homologous muscles. The lower mean temporal correlations for slow movements are most likely due to weaker modulation and lower signal-to-noise ratios. This explanation is supported by the finding of high temporal correlations (0.72–0.82) in condition 14, where the activity of all muscles was 2–3 times greater than for any other slow movement condition. Because the activity of homologous muscles was modulated in common, the rms EMG activity was summed to obtain single representations for flexor and extensor muscles.

To quantify the change in muscle activity associated with a particular condition, the difference in rms EMG activity for each phase of the task was calculated with respect to the corresponding phase of the no-load condition. The change in EMG activity was normalized by representing it as a percentage of the subject's maximum EMG activity. A single maximum value was determined for each muscle by comparing the rms EMG activity for all movement phases of all trials. The maxima for the three flexor muscles were summed to obtain the normalization factor for flexor activity, and the same was done for the extensor muscles. The normalized change in muscle activity was tested for significance by applying a Wilcoxon signed-rank test to the data of the individual subjects. Differences were considered to be statistically significant for $P<0.05$.

To investigate whether EMG bursts associated with wrist oscillation could be attributed to the monosynaptic stretch reflex, the rectified electromyogram was aligned with respect to the velocity of the oscillations and averaged. The averaged electromyogram was smoothed using a zero-lag, second-order low-pass Butterworth filter with a cutoff frequency of 15 Hz. The time difference between peak EMG activity and peak wrist velocity, in the direction corresponding to muscle stretch, was then determined. This analysis was not carried out for the FDS and EDC muscles because finger motion may have initiated reflex activity, which was independent of wrist motion.

To obtain an estimate of damping during vibration trials, the wrist was modeled as a linear second-order system with stiffness, damping, and inertia. The model incorporated realistic values of stiffness and moment of inertia, based on experimental data. The damping parameter was varied by trial and error to match the response of the model to a sequence of bidirectional torque steps (square-wave vibration) with that of the wrist. The shape of the velocity profile was used to assess changes in damping.

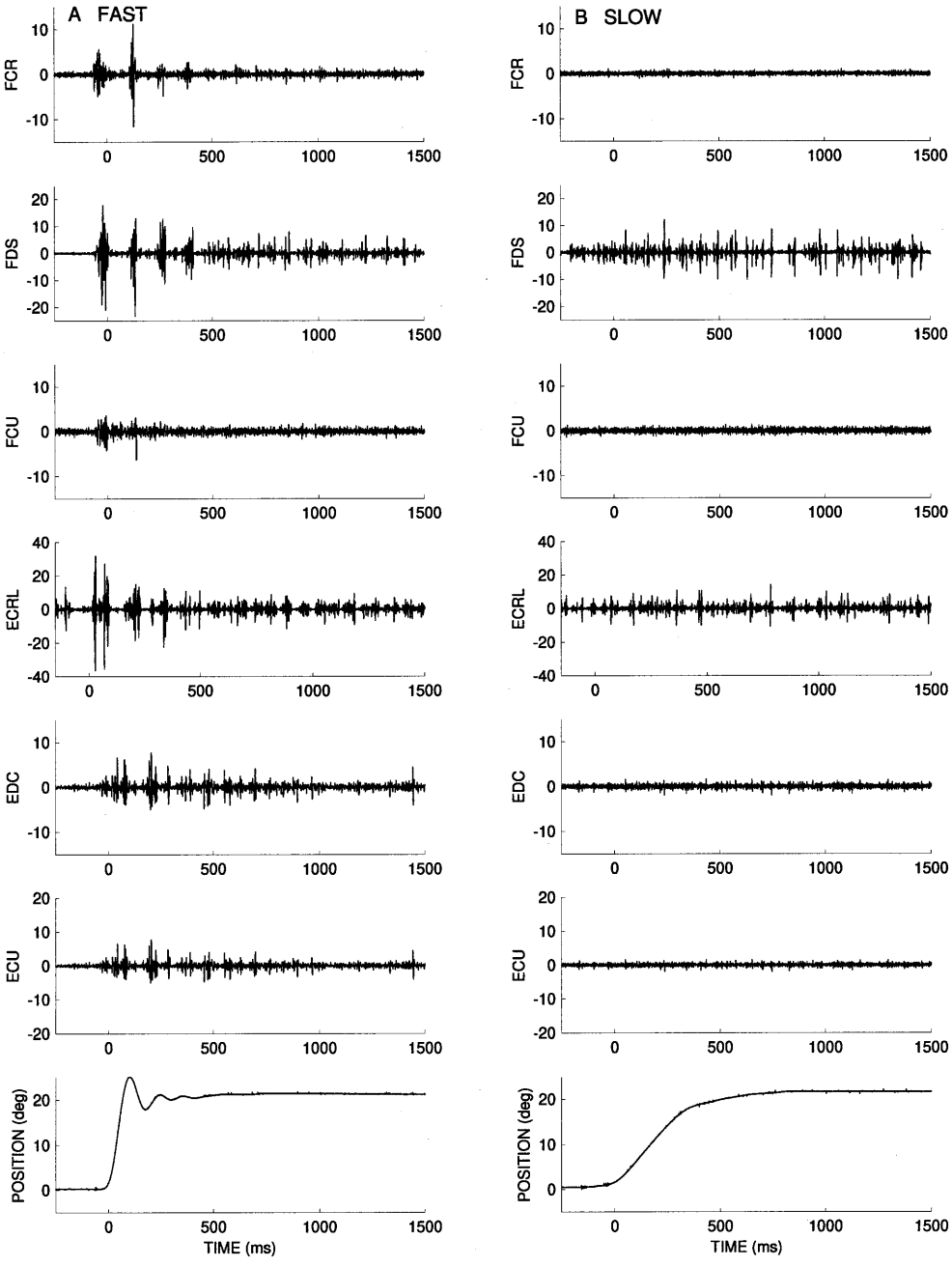

Fig. 1a, b Rapid wrist flexion (**a**) and slow wrist flexion (**b**) under the no-load condition. The subject (subject M) was required to stabilize the movement in a 3° target window within 800 ms of movement onset. The *top three traces* represent the surface EMG activity of three wrist flexor muscles, flexor carpi radialis (*FCR*), flexor digitorum superficialis (*FDS*), flexor carpi ulnaris (*FCU*), expressed in arbitrary units. The *middle three traces* represent the surface EMG activity of three wrist extensor muscles, extensor digitorum communis (*EDC*), extensor carpi radialis longus (*ECRL*), and extensor carpi ulnaris (*ECU*). The *bottom trace* is the angular displacement with respect to the start angle, expressed in degrees

Results

No load

The muscle activity patterns of fast movements, under mechanically stable conditions (no applied load), are shown in Fig. 1a. Flexion was initiated by a burst of activity in wrist flexor muscles and was terminated by a similar burst in the wrist extensors. Additional reciprocal bursts of flexor and extensor muscle activity were often observed during stabilization at the final position. During stabilization, mechanical oscillations were rapidly damped. Oscillations were in the 6- to 7-Hz frequency range and were damped, on average, within 1.5 cycles. During slow movement there was a small increase in activation of flexor muscles which remained relatively constant for the duration of the movement (Fig. 1b). Little extensor muscle activity was required to stop the movement.

Adaptation to destabilizing loads

The ratio of successful trials to attempts for destabilizing dynamics was as high as in the no-load condition for over 90% of the destabilizing load conditions, indicating that adaptation was successful. About half of the subjects had difficulty with one or two of the most destabilizing

Table 2 Mean change in rms EMG activity as a percentage of maximum rms EMG activity (RMS_{max}) compared with no load movements

Condition	Movement Flexor		Movement Extensor		Stabilization Flexor		Stabilization Extensor		Poststabilization Flexor		Poststabilization Extensor	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
3	0.73*	7.7	7.0*	10.0	4.0*	10.0	6.5*	12.0	3.7*	6.5	3.5*	7.6
4	13.0	6.6	8.5*	12.0	8.9	5.1	9.3	11.3	6.5	3.0	7.3	8.8
5	0.53*	8.0	9.1	7.2	4.2	7.4	3.4*	10.0	2.9	4.8	0.97*	8.2
6	15.0	5.0	8.4	8.9	10.0	5.0	10.0	9.2	6.9	4.0	7.3	7.0
7	–13.0	10.0	1.3*	6.3	9.8	13.0	11.0	13.0	6.3	9.3	5.6*	10.0
8	–0.35*	3.9	1.6*	6.6	0.75*	3.5	0.68*	7.6	0.64*	2.4	0.71*	5.6
9	–12.0	8.3	11.0	5.6	19.0	10.0	25.0	12.0	12.0	7.9	13.0	10.0
10	3.0*	4.7	7.4	9.3	4.5	5.6	7.7	10.0	3.5	4.8	6.4	8.4
11	–4.6*	10.0	4.7*	9.7	16.0	9.7	18.0	14.0	17.0	9.2	21.0	16.0
12	7.9	8.1	13.0	14.0	9.4	8.3	16.0	15.0	9.8	7.5	16.0	14.0
13	–0.75*	13.0	11.0	6.5	21.0	14.0	24.0	14.0	23.0	9.4	28.0	12.0
14	21.0	13.0	27.0	14.0	29.0	11.0	39.0	17.0	29.0	9.0	40.0	15.0

All values except those marked by *asterisks* are significantly greater or less than zero ($P<0.05$)

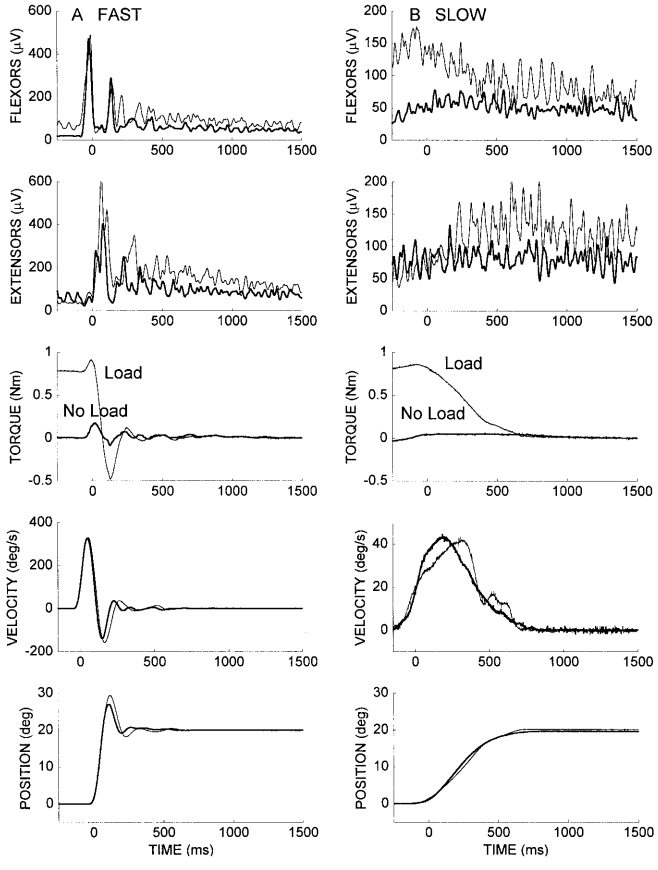

Fig. 2 a Rapid wrist flexion with negative load stiffness of –2.29 Nm·rad^{-1} (*thin lines*) compared with the no-load condition (*thick lines*). **b** Slow wrist flexion with the same load compared with the no-load condition. Traces represent the mean of 5 successful trials. The *top trace* is the sum of activity from three wrist flexor muscles, FCR, FDS, and FCU. The *second trace* is the sum of activity from three wrist extensor muscles, ECRL, EDC, and ECU. Surface EMG activity was rectified prior to summation and low-pass filtered at 30 Hz after summation. The *lower traces* are manipulandum torque, angular velocity, and angular position, respectively. Subject M

loads (conditions 9, 13, and 14) and required 3–4 times as many trials as during no load blocks. These low success rates were invariably due to difficulty in achieving the target movement duration rather than difficulty in achieving stable posture.

Negative stiffness

As shown in Fig. 2, the movement kinematics under the condition of negative stiffness (thin lines) were very similar to the no-load condition (thick lines), including the damping of oscillations during stabilization. However, the pattern of flexor muscle activity was clearly different. Activity in the flexor muscles was elevated in the premovement phase because of the opposing load (described in Materials and methods), which declined to zero in a linear fashion as the wrist was moved from the initial position to the target position. When the task was performed quickly, there was no more flexor muscle activity during movement to the target than under the no-load condition (Table 2, conditions 3 and 5). However, when performed slowly, flexor muscle activity was elevated by 13–15% of the maximum rms EMG activity (RMS_{max}) during movement to the target compared with the no-load condition. It is also evident from Fig. 2 that extensor muscle activity was higher than the no-load condition during both slow and fast movements. However, this change was statistically significant only when the negative stiffness was at the higher value of –2.29 Nm·rad^{-1}.

Flexor muscle activity during stabilization and poststabilization of fast movement was not significantly different than the no-load condition (Table 2, condition 3). However, when the task was performed slowly, there was a significant increase in activity of flexor muscles during stabilization, which persisted during poststabilization. Extensor muscle activity was elevated by an amount similar to flexor muscles (Table 2, conditions 4 and 6).

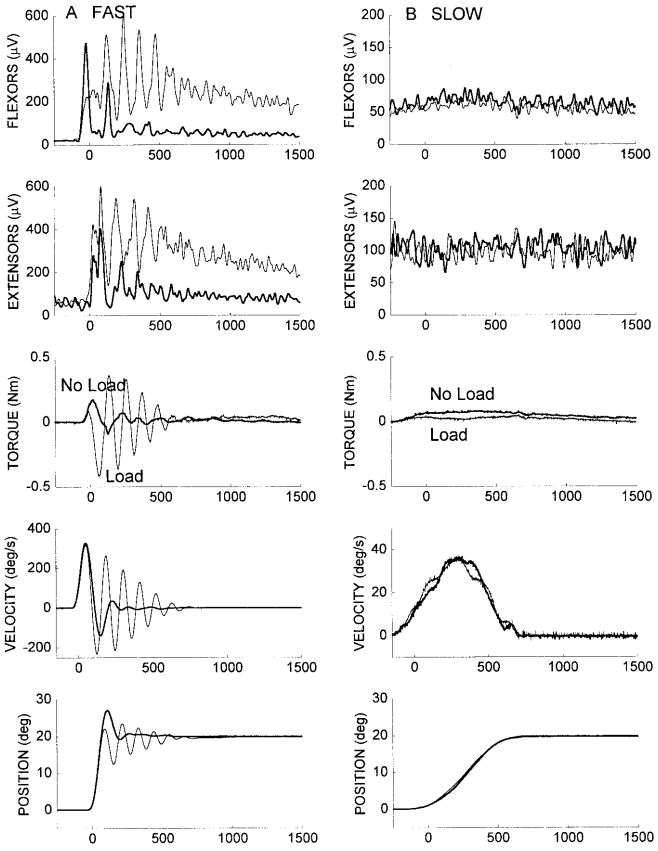

Fig. 3 a Rapid wrist flexion with negative load damping of –0.0859 Nm·s·rad^{-1} (*thin lines*) compared with the no-load condition (*thick lines*). **b** Slow wrist flexion with the same load compared with the no-load condition. Traces correspond to Fig. 2. Subject M

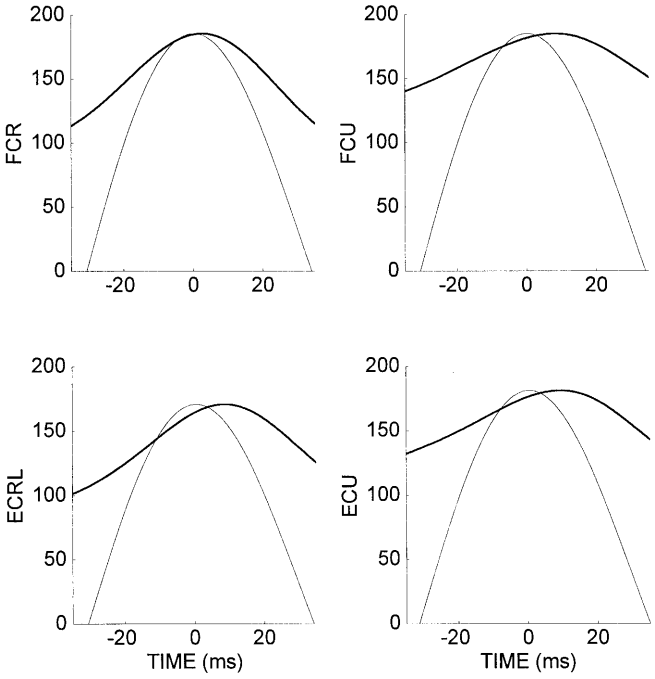

Fig. 4 Rectified EMG for the negatively damped load of Fig. 3 was aligned with respect to peak velocity in the direction of muscle stretch for each half-cycle of oscillation during the stabilization phase. The EMG activity and velocity were then averaged across subjects and subsequently low-pass filtered at 15 Hz. Velocity is depicted by the *thin line* and EMG by the *thick line* in each panel. FCR, FCU, and ECRL traces represent the averaged activity for ten subjects. The ECU trace represents the averaged activity for five subjects. Note that the EMG peaks occur several milliseconds after peak velocity and about 35–40 ms after zero velocity

Negative damping

When negative damping was used to destabilize the wrist, the number of oscillations increased dramatically, as can be seen in Fig. 3a. With the lower value of negative damping, there was a mean of three cycles of oscillation, while with the higher value of negative damping, the mean number of cycles increased to 4.5. Regular, periodic bursts of muscle activity, at the frequency of oscillation, were observed in both flexor and extensor muscles, although the bursts were less pronounced in extensor than flexor muscles.

As shown in Fig. 4, the peaks of these periodic EMG bursts, averaged across all subjects, occurred at about the time of peak velocity in the direction of muscle stretch. The frequency of oscillation of the averaged velocity was 7.5 Hz, while the delay between the averaged velocity and EMG peaks was 2 ms for FCR, 8 ms for FCU, and 9 ms for ECRL. For about half of the subjects, ECU EMG activity was too weakly modulated to produce distinguishable peaks, so averaging was only carried out for the five subjects for whom ECU EMG activity was most strongly modulated. For these subjects, the EMG peak occurred 9 ms after the velocity peak.

Movement to the target required significantly less flexor muscle activity than the no-load condition (Table 2, conditions 7 and 9), because the positive velocity feedback produced torque which assisted movement toward the target. At the higher level of negative damping (Table 2, conditions 9 and 10), there was a significant increase in extensor muscle activity during both fast and slow movement.

From Fig. 3 it is evident that both flexor and extensor muscle activity were greater during stabilization of fast movement than slow movement. In the case of slow movement, there was no significant change in activity of either flexor or extensor muscles during stabilization and poststabilization, compared with the no-load condition, for the lower level of negative damping (Table 2, condition 8), although flexor and extensor muscle activity did increase when negative damping was higher (Table 2, condition 10). When the task was performed quickly, even at the lower level of negative damping, activity of both flexor and extensor muscles increased by 10–11% RMS_{max} during stabilization and 6% RMS_{max} during poststabilization with respect to the no-load condition. With higher negative damping, the increase in activity of both muscle groups approximately doubled.

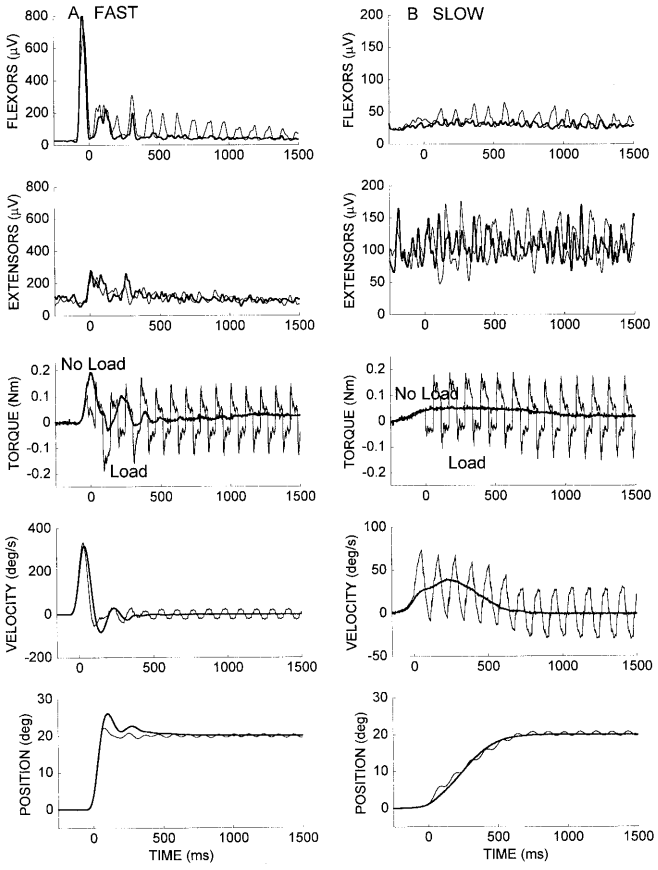

Fig. 5 **a** Rapid wrist flexion with 10 Hz square-wave vibration (*thin lines*) compared with the no-load condition (*thick lines*). **b** Slow wrist flexion with the same vibration compared with the no-load condition. Traces correspond to Fig. 2. Subject R

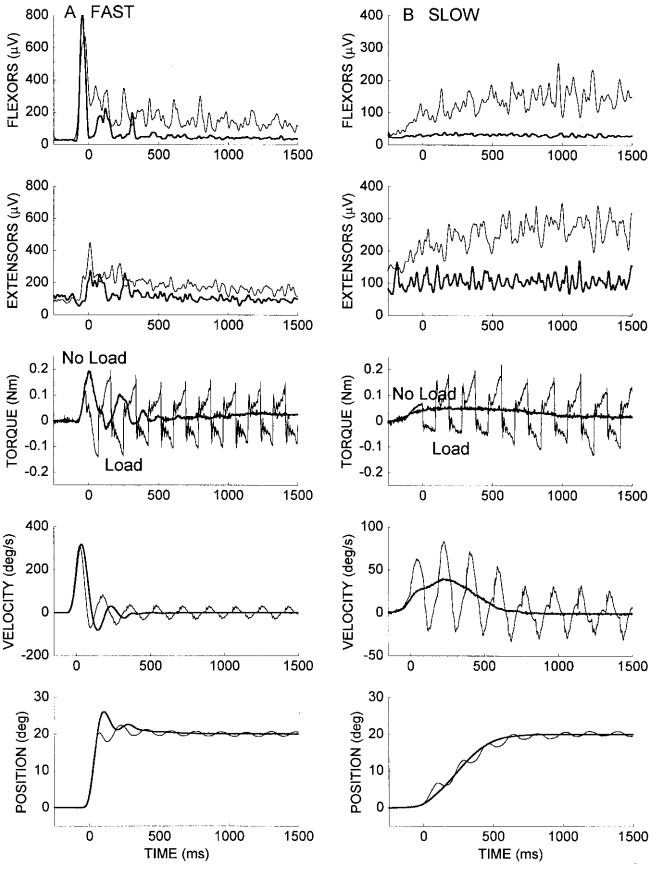

Fig. 6 **a** Rapid wrist flexion with 5.5-Hz square-wave vibration (*thin lines*) compared with the no-load condition (*thick lines*). **b** Slow wrist flexion with the same vibration compared with the no-load condition. Traces correspond to Fig. 2. Subject R

Vibration

The vibratory destabilization consisted of square-wave torque pulses alternating between flexion and extension at a regular frequency of either 5.5 Hz or 10 Hz. The oscillation of the position traces in Figs. 5 and 6 indicates that the vibration had a significant perturbing effect, despite its predictable nature. The lower-frequency vibration, shown in Fig. 6, produced larger displacements during the stabilization period (within 800 ms of movement onset) than the high-frequency vibration, shown in Fig. 5, even though the vibration amplitudes were chosen to produce the same torque impulse at both frequencies.

The vibration had no significant effect on the activity of flexor muscles during movement when the task was performed quickly (Table 2, conditions 11 and 13). However, the 5.5-Hz vibration produced an increase in extensor muscle activity of 11% RMS_{max}. The effect of vibration on muscle activity was much more pronounced during slow movements. Flexor muscle activity increased by 8% RMS_{max} for the 10-Hz vibration and 21% RMS_{max} for the 5.5-Hz vibration compared with the no-load condition, while extensor muscle activity increased by 13% RMS_{max} for the 10-Hz vibration and 27% RMS_{max} for the 5.5-Hz vibration. Regardless of whether the task was performed quickly or slowly, flexor and extensor activation during stabilization and poststabilization were significantly greater than under the no-load condition (Table 2, conditions 11–14). Activity increased by as much as 30% RMS_{max} in flexor muscles and 40% RMS_{max} in extensor muscles.

Activity in three of the muscles was noticeably modulated at the vibration frequency. At 10 Hz, the EMG peak, averaged across subjects, lagged the averaged velocity peak by 9 ms, 12 ms, and 11 ms for the FCR, FCU, and ECRL muscles, respectively. At 5.5 Hz, the EMG peak led the velocity peak by 18 ms for FCR, 4 ms for FCU, and 16 ms for ECRL. This analysis could not be performed for the ECU muscle, because there were only three subjects for whom modulation of ECU activity was evident at either vibration frequency.

Changes in shape of the velocity profile resulting from the torque pulses were used to assess damping of the wrist. The modeled velocity profiles, shown on the right in Fig. 7, indicate that velocity peaks become more rounded at 10 Hz (top panels) with greater damping, whereas corners become more pronounced at 5.5 Hz (bottom panels). Comparison of the first and last velocity

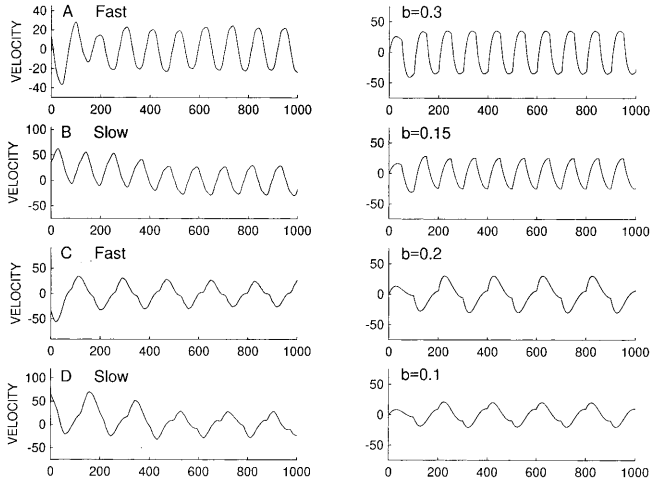

Fig. 7a–d *Left:* Recorded velocity profiles from Figs. 5 and 6, corresponding to the time interval beginning at 250 ms and ending at 1,250 ms. Velocity was low-pass filtered at 40 Hz. **a** corresponds to Fig. 5a, **b** to Fig. 5b, **c** to Fig. 6a, and **d** to Fig. 6b. *Right:* Simulated velocity profiles, generated using a linear second-order model of wrist mechanics (see Appendix). The damping parameter (*b*) was varied, as indicated at the *top* of each panel

peaks in Fig. 7a–d (left panels) reveals changes in shape, which signify lower damping during movement (first peak) than during stabilization (last peaks), i.e., damping increases as velocity decreases. Figures 7a and b show that in the case of the 10-Hz vibration damping was higher during the stabilization and poststabilization phases of fast movement than slow movement. It is somewhat more difficult to draw conclusions about differences in damping between fast and slow movement for the 5.5-Hz vibration, since the velocity profile changed in a more subtle way. However, careful examination of the simulations suggests that damping was also higher during the stabilization phase of fast movement than slow movement (Fig. 7c, d).

Discussion

The results of this study show that agonist-antagonist coactivation is used to varying degrees to stabilize the wrist when interacting with different types of destabilizing loads. Both the magnitude and temporal patterning of agonist-antagonist coactivation depend on the nature of the instability. In some situations coactivation must be effected early in the movement, whereas in others it is only required during stabilization at the final position. Some instabilities require a constant level of coactivation at the final position, while others allow coactivation to gradually decline once movement has stopped. Three distinctive instabilities were examined. The first was a position-dependent instability in which the final position was an unstable equilibrium point. The second was a velocity-dependent instability, which reduced the damping of the system. The third was a vibration, which alternately flexed and extended the wrist.

The suitably of adaptations in muscle activation to the characteristics of an instability must be interpreted in the light of their effects on wrist kinetics. Therefore, it is first necessary to distinguish between modulation of the EMG and modulation of the resulting wrist torque or wrist stiffness. Muscle force and stiffness change much more gradually than EMG. Partridge (1965) has shown that muscle acts like a low-pass filter, with significant attenuation of force modulation with respect to electrical activation above 4 Hz. For example, there would be insufficient time for either flexor or extensor muscles to relax completely during the period of alternating activity in Fig. 1, although the mean muscle force would gradually decrease. This type of activation pattern, consisting of high-frequency reciprocal bursts of gradually diminishing amplitude, is functionally equivalent to agonist-antagonist cocontraction. Wrist stiffness is effectively proportional to the sum of flexor and extensor muscle force magnitudes. Consequently, the gradually diminishing oscillations in muscle force will result in joint stiffness that decays at the same rate, but with greatly reduced ripple.

No load

Patterns of muscle activity were expressed in terms of differences with respect to the no-load condition, which involved movement of a small inertial load. With no load, slow movement was executed with little difference in surface EMG activity from premovement to poststabilization phases, suggesting little need for stabilizing action. Fast movement was characterized by clear modulation of both flexor and extensor surface EMG activity. There was a distinctive burst of activity in flexor muscles to initiate flexion, which was immediately followed by a burst of activity in the extensor muscles (Wachholder and Altenburger 1926). The activity in both muscle groups thereafter declined sharply, but remained elevated with respect to premovement levels, even 1,000 ms after the movement had stopped. This suggests a need for cocontraction of agonist and antagonist muscles to stiffen the joint in order to achieve postural stability at the final position. Hasan (1986) proposes that the relative amount of cocontraction during inertially loaded movements can be predicted by minimizing a cost function related to sense of effort. However, his model was addressed specifically to stiffness *during* movement and did not deal with stabilization at the final position.

Negative stiffness

When stabilizing posture at an unstable equilibrium position, there was a much greater increase in agonist-antagonist cocontraction compared with the no-load condition if the task was performed slowly than if it was performed quickly. This is probably because slow movements require much less muscle activation than fast movements.

As a result, the joint stiffness during slow movement is significantly lower than during fast movement (Bennett 1993; Milner 1993). Bennett (1993) has shown that elbow stiffness varies in an almost quadratic fashion with peak velocity. Assuming even a linear relationship between wrist stiffness and peak velocity, during slow movement the stiffness could have been less than one-fifth that during fast movement. Consequently, there would have been a much greater need to stiffen the wrist by agonist-antagonist cocontraction during the stabilization phase of slow movements than fast movements. The adaptations in muscle activation adequately compensated for the negative stiffness, as can be judged from the similarity in movement kinematics under conditions of negative stiffness compared with the no-load condition.

Negative damping

With negative damping, the relation between movement speed and the amount of cocontraction during stabilization was reversed. A greater increase in activity of both wrist flexors and extensors, compared with no load movements, was observed during stabilization when the task was performed quickly than when it was performed slowly. In particular, when the task was performed slowly with negative damping set to the lower value of –0.0573 $Nm \cdot s \cdot rad^{-1}$, muscle activity during movement and stabilization was no different than under the no-load condition. When the task was performed quickly, although flexor muscle activity during movement was lower than under the no-load condition and extensor muscle activity was the same, during stabilization both flexor and extensor activity were 10–11% RMS_{max} greater. Increasing the level of negative damping to –0.0859 $Nm \cdot s \cdot rad^{-1}$ induced an additional increase in muscle activation. It also led to greater activation of both flexor and extensor muscles in slow movements than under the no-load condition, though the increase was substantially less than during fast movements. This suggests that during slow movements there was little need to modify wrist damping with respect to the no-load condition, i.e., damping was close to the minimum level needed to ensure stability. The greater muscle activation during stabilization of fast movements would have produced a steeper force-velocity curve (Petrofsky and Phillips 1981), resulting in higher intrinsic damping.

Fast movement was executed by producing brief torque impulses in the flexor and extensor muscles. The underdamped nature of the system was already evident in the no-load condition. Adding negative damping to the system reduced the overall damping, which resulted in oscillation of greater amplitude and longer duration. The oscillations were accompanied by bursts of muscle activity, which could be attributed to the monosynaptic stretch reflex. The burst peaks of both wrist flexor and extensor muscles occurred slightly after peak velocity in the direction of muscle stretch. Since the oscillation frequency was about 7.5 Hz, the delay between onset of movement in the direction of muscle stretch and peak velocity was about 33 ms, which gave a latency of 36–42 ms between onset of muscle stretch and peak EMG. This is consistent with an earlier study (De Serres and Milner 1991) in which we determined that a monosynaptic reflex response, elicited by a sudden stretch of wrist flexor muscles, peaks 35–40 ms after the onset of the perturbation. We have argued previously (Milner and Cloutier 1998) that the monosynaptic stretch reflex contributes negatively to damping for frequencies of oscillation around 7 Hz, as observed here. The reason is that motor units in wrist muscles produce their maximum twitch force about 60 ms after the action potential (Riek and Bawa 1992), so that peak wrist torque would occur at approximately the same time as peak wrist velocity in the direction of muscle shortening. Thus, damping would be less during fast movements than slow movements, because the muscle force-velocity curve is flatter at higher velocities of shortening and because the monosynaptic stretch reflex contributes negatively to damping. Agonist-antagonist cocontraction would serve to counteract the reduced stability. Increasing the number of active muscles and their contraction strength would increase the intrinsic joint stiffness and damping. The stiffness serves to limit the amplitude of oscillation, while the damping progressively attenuates the oscillation.

Tonic cocontraction

It is apparent from Fig. 3 that muscle activity is modulated about a gradually decreasing tonic contraction level, in both flexors and extensors, during stabilization. Muscle activity increased above the tonic level when a muscle group was being stretched and fell below it, due to reciprocal inhibition, when the antagonists were being stretched. Once the oscillations ceased, the underlying tonic activity was readily apparent. It would appear that the tonic cocontraction was initiated early in the movement, perhaps at the same time as the antagonist burst. Thus, it could be considered equivalent to Feldman's coactivation command (Feldman 1980; Levin et al 1992). Such tonic cocontraction was even more evident during movements destabilized by the 5.5-Hz vibration (Fig. 6).

Vibration

The response to an instability induced by vibration depended critically on the frequency of the vibration. A 10-Hz disturbance was stabilized with less muscle activation than a 5.5-Hz disturbance. Activity in FCR, FCU, and ECRL showed evidence of modulation by the monosynaptic stretch reflex. Averaged EMG peaks for these muscles occurred 27–41 ms and 34–37 ms after the onset of movement in the direction of muscle stretch at 5.5 Hz and 10 Hz, respectively. Assuming a delay of 60 ms between peak EMG and wrist torque, as above, peak torque would have coincided with maximum muscle length at

5.5 Hz and with minimum muscle length at 10 Hz. That is, at 5.5 Hz the reflex torque would have resisted the displacement, while at 10 Hz it would have assisted it, in a position-dependent manner. It is, therefore, interesting that subjects used greater agonist-antagonist coactivation for the 5.5-Hz disturbance than the 10-Hz disturbance. This is counterintuitive to what would be expected if tonic cocontraction were compensating for the effect of the phasic reflex activity. To understand why this occurred, consider first that the muscles act as low-pass filters, modulating force less for phasic activity at 10 Hz than at 5.5 Hz (Partridge 1965). Thus, there would have been relatively little displacement produced by reflex EMG activity at 10 Hz. Second, with the muscles relatively relaxed (low tonic activation), the natural frequency of the system would have been much lower than 10 Hz, serving to limit the displacement produced by the 10-Hz torque steps. In contrast, higher joint stiffness, requiring greater tonic activation, would have been required at 5.5 Hz to move the natural frequency of the system above the frequency of the disturbance, i.e., to a natural frequency of approximately 7.5 Hz (see Appendix). Lower muscle activation would have resulted not only in less resistance to the disturbance due to lower stiffness, but also in greater displacement due to the natural frequency being closer to the frequency of the disturbance.

Damping

The relative changes in damping during transition from movement to stabilization, estimated from the torque step response, are consistent with our previous results (Milner and Cloutier 1998). Damping increased as movement velocity decreased during the transition from movement to stabilization. There also appeared to be greater damping during the stabilization phase of fast movements than slow movements, which may have been due to greater muscle activation.

Adaptive muscle coactivation

The close link between muscle force and muscle stiffness, due to the elastic nature of actin-myosin cross-bridges, guarantees a certain amount of stability to the musculoskeletal system without the need for adaptation. For example, when muscles are highly activated, as during fast movements, a mechanical instability, such as that produced by negative stiffness, goes unnoticed if the negative stiffness is less than the intrinsic stiffness of the joint. However, there are many situations in which the dynamics of the task does not result in sufficient muscle activation to guarantee stability of the joint. In these cases, coactivation of agonist and antagonist muscles is used to increase joint stiffness or damping. In other situations, coactivation may be used to raise the natural frequency sufficiently above the frequency of a disturbance to avoid mechanical resonance.

Acknowledgements This work was supported by the Natural Science and Engineering Research Council of Canada. The experiments were conducted at the Institut de Réadaptation de Montréal. I thank Mr. Steven Cooner and Ms. Caroline Cloutier for assistance in conducting the experiments.

Appendix

If the wrist is modeled as a second-order mechanical system, the equation of motion is given by:

$$I\ddot{\theta} + b\dot{\theta} + k\theta = \tau \tag{1}$$

where I is the moment of inertia, b is the damping parameter, k is the stiffness, and τ is the applied torque. If τ is a step input, then the Laplace transform for this system is given by:

$$\Theta(s) = \frac{\theta_o(s+\beta) + (\dot{\theta}_o + \beta\theta_o)}{(s+\beta)^2 + \omega^2} + \frac{\tau}{Is((s+\beta)^2 + \omega^2)} \tag{2}$$

where:

$$\beta \equiv \frac{b}{2I},\ \omega \equiv \sqrt{\frac{k}{I} - \beta^2}$$

$\dot{\theta}_o$ *and* θ_o represent the angular velocity and position, respectively, at the time of each torque step. For an underdamped system, the angular velocity as a function of time is then given by:

$$\dot{\theta}(t) = e^{-\beta t}\left\{\frac{\sin\omega t}{\omega}\left(\frac{\tau}{I} - \theta_o(\omega^2 + \beta^2) - \dot{\theta}_o\beta\right) + \dot{\theta}_o\cos\omega t\right\} \tag{3}$$

The moment of inertia of the wrist and manipulandum was approximately 0.005 kg·m^2. The ratio k/I for simulation of the 5.5-Hz vibration was estimated from the frequency of oscillation, f, in condition 9 (Table 1), using the relation between stiffness, moment of inertia, and natural frequency:

$$\frac{k}{I} = (2\pi f)^2 \tag{4}$$

The rationale behind this approximation was that the amount of muscle activation was similar in conditions 9 and 13. Hence the wrist stiffness should have been similar, resulting in a similar natural frequency (7.5 Hz). In the case of the 10-Hz vibration, the muscle activation was lower so the natural frequency was set to 6 Hz. Values of the damping parameter were chosen by trial and error to produce velocity profiles with shapes similar to those recorded in the experiment. The angular velocity and angular position at the time of the first torque step were set to zero for convenience. Thereafter, they were set equal to the position and velocity at the end of the previous torque step. The simulated velocity profiles are compared with the recorded profiles in Fig. 7.

References

Bennett DJ (1993) Torques generated at the human elbow joint in response to constant position errors imposed during voluntary movements. Exp Brain Res 95:488–498

Clément G, Rézette D (1985) Motor behavior underlying the control of an upside-down vertical posture. Exp Brain Res 59:478–484

De Serres SJ, Milner TE (1991) Wrist muscle activation patterns and stiffness associated with stable and unstable mechanical loads. Exp Brain Res 86:451–458

Feldman AG (1980) Superposition of motor programs. II. Rapid forearm flexion in man. Neuroscience 5:91–95

Gielen CCAM, Houk JC (1984) Nonlinear viscosity of human wrist. J Neurophysiol 52:553–569

Hasan Z (1986) Optimized movement trajectories and joint stiffness in unperturbed, inertially loaded movements. Biol Cybern 53:373–382

Hogan N (1984) Adaptive control of mechanical impedance by coactivation of antagonist muscles. IEEE Trans Autom Control 29:681–690

Houtz SJ (1964) Influence of gravitational forces on function of lower extremity muscles. J Appl Physiol 19:999–1004

Lacquaniti F, Maioli C (1987) Anticipatory and reflex coactivation of antagonist muscles in catching. Brain Res 406:373–378

Levin MF, Feldman, AG, Milner TE, Lamarre Y (1992) Reciprocal and coactivation commands for fast wrist movements. Exp Brain Res 89:669–677

Milner TE (1993) Dependence of elbow viscoelastic behavior on speed and loading in voluntary movements. Exp Brain Res 93:177–180

Milner TE, Cloutier C (1993) Compensation for mechanically unstable loading in voluntary wrist movement. Exp Brain Res 94:522–532

Milner TE, Cloutier C (1998) Damping of the wrist joint during voluntary movement. Exp Brain Res 122:309–317

Milner TE, Cloutier C, Leger AB, Franklin DW (1995) Inability to activate muscles maximally during cocontraction and the effect on joint stiffness. Exp Brain Res 107:293–305

Partridge LD (1965) Modifications of neural output signals by muscles: a frequency response study. J Appl Physiol 20:150–156

Petrofsky JS, Phillips CA (1981) The influence of temperature, initial length and electrical activity on the force-velocity relationship of the medial gastrocnemius muscle of the cat. J Biomech 14:297–306

Riek S, Bawa P (1992) Recruitment of motor units in human forearm extensors. J Neurophysiol 68:100–108

Thoroughman KA, Shadmehr R (1999) Electromyographic correlates of learning an internal model of reaching movements. J Neurosci 19:8574–8588

Wachholder K, Altenburger H (1926) Beiträge zur Physiologie de willkürlichen Bewegumg. Einzelbewegungen. Pflugers Arch Ges Physiol 214:642–661