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RESEARCH ARTICLE

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Damping of the wrist joint during voluntary movement

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Abstract Damping characteristics of the musculoskeletal system were investigated during rapid voluntary wrist flexion movements. Oscillations about the final position were induced by introducing a load with the characteristics of negative damping, which artificially reduced the damping of the wrist. Subjects responded to increases in the negatively damped load by stronger cocontraction of wrist flexor and extensor muscles during the stabilization phase of the movement. However, their ability to counteract the effects of the negatively damped load diminished as the negative damping increased. Consequently, the number and frequency of oscillations increased. The oscillations were accompanied by phase-locked muscle activity superimposed on underlying tonic muscle activation. The wrist stiffness and damping coefficient increased with the increased cocontraction that accompanied more negatively damped loads, although changes in the damping coefficient were less systematic than the stiffness. Analysis of successive half-cycles of the oscillation revealed that the wrist stiffness and damping coefficient increased, despite decreasing muscle activation, as oscillation amplitude and velocity declined. This indicates that the inverse dependence of the damping coefficient on oscillation velocity contributes significantly to damping of joint motion. It is suggested that this property helps to offset a negative contribution to damping from the stretch reflex.

Key words Wrist · Stiffness · Voluntary movement · Damping · Human

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Introduction

The spring-like nature of muscles in isolation and under reflex control has long been recognized for its role in providing postural stability and permitting compliant interaction with the mechanical environment (Asatryan and Feldman 1965; Partridge 1966; Rack 1966; Grillner 1972; Bizzi et al. 1976; Nichols and Houk 1976; Hoffer and Andreassen 1981). Damping properties of muscle are less frequently the primary focus of research (Neilson and Neilson 1978; Gielen and Houk 1984), although a number of studies have touched on them to greater or lesser extents (Rack 1966; Rack and Westbury 1974; Agarwal and Gottlieb 1977; Hunter and Kearney 1982; Lacquaniti et al 1982; Becker and Mote 1990; Bennett et al. 1992; Milner and Cloutier 1993; Bennett 1994; Hajian and Howe 1997; Zhang and Rymer 1997). Without damping, it would not be possible to position a limb quickly and accurately, nor would it be possible to rapidly damp oscillations when the limb was subjected to an impulsive force. The term *viscosity* has frequently been used to refer to velocity-dependent mechanical properties of joints. However, this is suggestive of velocity-dependent resistance to joint motion arising from fluid viscosity (Zatsiorsky 1997). To avoid such implications, we have modeled the joint as a damped mass-spring system in which the damping force is a linear function of velocity. The coefficient that represents the proportionality between damping force and velocity will be referred to as the *damping coefficient* rather than the viscosity.

Without damping, a joint would tend to oscillate indefinitely following movement. Most studies that have estimated the damping coefficient of single joints such as the ankle, elbow, wrist, or finger articulations (Agarwal and Gottlieb 1977; Hunter and Kearney 1982; Lacquaniti et al. 1982; Becker and Mote 1990; Bennett et al. 1992; Milner and Cloutier 1993) have shown that these joints are underdamped. It is somewhat surprising then that humans are usually able to stop a rapid, voluntary limb movement without noticeable oscillation. Although it has been suggested that the stretch reflex is responsible for this rapid damping (Nielson and Nielson 1978), there is good evidence that stretch reflex responses may actually be destabilizing under some circumstances (Rack 1981; Stein and Lee 1981; Jacks et al. 1988; Prochazka and Trend 1988; Bennett 1994).

Damping in the musculoskeletal system originates primarily from two sources, namely, intrinsic velocity-dependent properties of muscle and reflex muscle activation. The intrinsic damping derives, in large part, from the dependence of contractile force on rate of change of muscle length (Hill 1938). Small-amplitude changes in muscle length, whether applied while a muscle fiber is held isometric or when it is undergoing a length change, suggest that attached cross-bridges are almost purely elastic. The damping must, therefore, derive from changes in the number of attached cross-bridges or some change in the cross-bridge strain as a function of velocity (Huxley 1957; Ford et al. 1985). It is highly unlikely that the damping arises from the resistance of the myoplasm to cross-bridge movement, since no velocity-dependent force is required to match the transient force response to rapid, small changes in muscle fiber length (Ford et al. 1985). The damping coefficient of muscle has been shown to increase with muscle force under isometric conditions (Agarwal and Gottlieb 1977; Hunter and Kearney 1982; Becker and Mote 1990; Kirsch et al. 1994; Hajian and Howe 1997; Zhang and Rymer 1997), suggesting further that it depends directly on the number of attached cross-bridges. However, it also decreases when either the amplitude or bandwidth of the input displacement increases (Kearney and Hunter 1982; Kirsch et al. 1994), indicating an inverse dependence on velocity.

The present study was designed principally to address two questions: first, whether during voluntary movement the damping of a joint is tightly linked to its stiffness, as has been shown under isometric conditions where the damping ratio remains relatively constant. This may not be the case during movement, since reflex torque probably contributes differentially to damping and stiffness depending on movement frequency. Second, whether the angular velocity of a joint has a significant effect on the damping coefficient. Because the slope of the force-velocity relation decreases dramatically as velocity increases, the damping coefficient is likely to decrease significantly as joint angular velocity increases unless counteracted by a substantial increase in muscle activation.

We have been studying the processes contributing to damping during voluntary movement by eliciting limb oscillations at the natural frequency of the neuromuscular system (Milner and Cloutier 1993). This is done by artificially reducing the damping of the joint, causing the limb to become severely underdamped. In the present study, the underdamping of the limb-load system was progressively increased to manipulate the number of cycles of oscillation about the target position as a means of investigating the questions posed above.

Materials and methods

Ten neurologically normal volunteers (six men, four women), aged 21–38 years, participated as subjects in these experiments. All subjects gave informed consent to the procedures, which were approved by the institutional ethics committee and were conducted in accordance with the principles of the Helsinki agreement.

Protocol

The subject was required to move a wrist manipulandum by flexing the wrist. The subject's hand was held securely in the manipulandum by clamping the palm, thus avoiding the need to grip the manipulandum with the fingers. The manipulandum was attached to a torque motor such that the shaft of the motor was aligned with the axis of rotation of the wrist. The movement was a 30° flexion to a 3° target zone, beginning from the neutral position of the wrist. The subject was required to move to the target zone with a peak velocity of approximately 500°/s and to stabilize the wrist within 800 ms of movement onset, such that there was no movement outside the target zone after that time.

The position and velocity of the manipulandum 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 the manipulandum. The torque motor was used to generate negative damping under computer control, using positive velocity feedback. The load was activated prior to the onset of movement and remained active until the end of the trial. In a previous study (Milner and Cloutier 1993), it was determined that subjects had little difficulty in stabilizing a negatively damped load of –0.057 Nm·s/rad. This was used as a starting value from which the stability limit of each subject was determined by incrementally increasing the negative damping until the subject could no longer successfully complete the task. For nine of the subjects, the stability limit was greater than –0.080 Nm·s/rad, the greatest being –0.114 Nm·s/rad. However, for one subject the stability limit was only –0.054 Nm·s/rad. The mean stability limit of the ten subjects was –0.088 Nm·s/rad. Generally, male subjects had higher stability limits than female subjects. We did not systematically investigate the effects of practice on the stability limit, although our earlier study (Milner and Cloutier 1993) suggested that stability improved as subjects became more familiar with the load.

Five different levels of negative damping, in equal increments from –0.057 Nm·s/rad to the stability limit, were then each presented to the subject ten times. In the case of the one subject with a low stability limit, the negative damping was incremented from –0.051 Nm·s/rad rather than –0.057 Nm·s/rad. The 50 presentations were completely randomized. Each subject was given as many attempts as he or she required to successfully complete a trial. Subjects were generally successful in completing ten trials with the lower levels of negative damping, although encountering difficulty with the two higher levels. For most subjects, fewer than five trials were successfully stabilized with the most negatively damped load. These trials were analyzed but were not included in the results because they could not be attributed the same statistical significance as conditions with many more trials. Although not reported, these data were entirely consistent with the results that are presented.

Surface electromyographic (EMG) activity 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 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 quantified by computing the rms (root mean squared) values during four time intervals, 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 postmovement epoch (800–1500 ms after movement onset).

The coupled system of wrist and torque motor was modeled as an underdamped mass-spring system. Mechanical parameters (damping coefficient, stiffness, and oscillation amplitude) were estimated by fitting oscillations occurring in the velocity record with the equation of an underdamped harmonic oscillator:

$$\dot{\theta} = -\frac{K\theta_0}{I\omega} e^{-\beta t} \sin \omega t \tag{1}$$

$$\omega = \sqrt{\frac{K}{I} - \beta^2} \tag{2}$$

and

$$\beta = \frac{B}{2I} \tag{3}$$

where θ_0 represents the oscillation amplitude, I, the moment of inertia of the manipulandum and hand, K, the stiffness of the wrist, and B the damping coefficient of the wrist and motor. Since the moment of inertia was essentially constant, the damping parameter β was directly proportional to the damping coefficient B.

If the damping parameter is small, as was the case in this experiment, then

$$\dot{\theta} \approx -\theta_0 \sqrt{\frac{K}{I}} e^{-\beta t} \sin \sqrt{\frac{K}{I}} t \tag{4}$$

It is apparent from the form of this equation that a procedure that simultaneously estimates K and I may yield an accurate value of the ratio K/I, without providing an accurate estimate of K or I. The potential for error was tested by estimating the moment of inertia of the manipulandum and hand using an independent procedure, which had been validated in tests with calibrated steel plates. The results demonstrated that fitting Eq. 1 to the data by simultaneously estimating I, B, K and θ_0, using a modified Marquardt-Levenberg procedure, which mimimizes the residual sum of squares (Marquardt 1963), could give estimates of the moment of inertia that were in error by as much as a factor of 2. Unfortunately, this procedure for independently estimating the moment of inertia was developed too late to apply it to the subjects of the experiment reported here.

Consequently, we decided to assign the same value of moment of inertia to all subjects to minimize the overall error in estimating K and B, the two primary parameters of interest. The rationale for doing this was that our subjects did not vary greatly in size and that the moment of inertia of the manipulandum was constant and was greater than the moment of inertia of the hand. The value used for the total moment of inertia, 0.002 $\text{kg}\cdot\text{m}^2$, represented the mean value for a similar group of subjects involved in a subsequent study where moment of inertia was independently estimated, using the validated procedure referred to above.

The potential error in using a single value for the moment of inertia was investigated by estimating K and B with values of I that were either 25% lower (0.0015 $\text{kg}\cdot\text{m}^2$) or 25% higher (0.0025 $\text{kg}\cdot\text{m}^2$) than the selected value. As anticipated, a 25% change in I led to a corresponding 25% change K, preserving the K/I ratio. However, the effect on B was minimal. Although B did change, the magnitude of the change amounted to only 2% of the original value. Furthermore, the change in B was not systematic. It did not matter whether I was made larger or smaller, the estimates of B over a series of ten trials always varied about equally both ways. Therefore, as long as the selected value of I closely approximated the mean moment of inertia of the ten subjects (which we believe was the case, as explained in the preceding paragraph), the estimates of K and B, averaged over the ten subjects, are unlikely to have been significantly in error.

It was often evident that the stiffness changed with time, since the frequency of oscillation varied. Our method did not allow us to easily estimate the instantaneous mechanical impedance. However, given the relatively slow response of joints to brief torque pulses during movement (Milner 1993), it is likely that the mean mechanical impedance over an interval of one-half cycle of oscillation (60–70 ms) is a more relevant mechanical parameter than the instantaneous impedance. Therefore, we modeled the mechanical impedance as remaining constant over half a cycle of oscillation. Parameters were reestimated for each half-cycle, delineated by successive velocity zero-crossings. The variance accounted for by the model was almost always greater than 99%, suggesting that the assumptions were reasonable. The damping coefficient B in Eqs. 2 and 3 represents the combined damping of the human wrist and the negative damping of the torque motor. To find the damping coefficient of the wrist, the negative damping command to the motor was subtracted from B. That the command accurately represented the actual negative damping generated by the motor was confirmed in tests that demonstrated that there was less than 5% error between the commanded and measured values.

For each subject, the rms EMG for each muscle during each task epoch was averaged over all trials (usually ten) for a given condition. This was then normalized for each subject by dividing all values by the largest value under any condition, thereby reducing the effect of variability across subjects. The effect of negative damping level on the EMG of each task epoch was tested by one-way ANOVA with damping level as the factor. A *t*-test for comparisons of means was used to test whether the EMG was significantly different during different task epochs for a given negative damping level. Differences were considered statistically significant only if $P<0.05$.

There were 5 levels of negative damping. For each negative damping level, there were generally 4 or more half-cycles in which parameters could be estimated. As previously noted, only 4 levels of negative damping were included in the statistical analysis owing to the low rate of success by subjects at the highest level of negative damping. We classified the estimated stiffness and damping coefficient according to the level of negative damping and the half-cycle in which they were estimated. The mean value was then calculated for all trials (usually ten) in a given condition, i.e., for each combination of negative damping level and half-cycle. The mean stiffness and damping coefficient for each subject under each condition were then used as dependent variables, and negative damping level and half-cycle number were used as factors in a two-way ANOVA to look for main effects.

Results

Muscle activation

The negatively damped load resulted in a series of damped oscillations about the final target position. The number of oscillations increased with the level of negative damping, as did the frequency of oscillation. Subjects adapted to increases in the negatively damped load by contracting both extensor and flexor muscles more strongly (Fig. 1).

The wrist oscillations during the stabilization epoch were accompanied by phase-locked EMG bursts that declined in amplitude as the oscillations were damped and the wrist stabilized at the final position (Fig. 2). The amplitude modulation of the EMG was more pronounced in flexor muscles than extensor muscles, although both muscle groups were tonically active as well. The tonic activa-

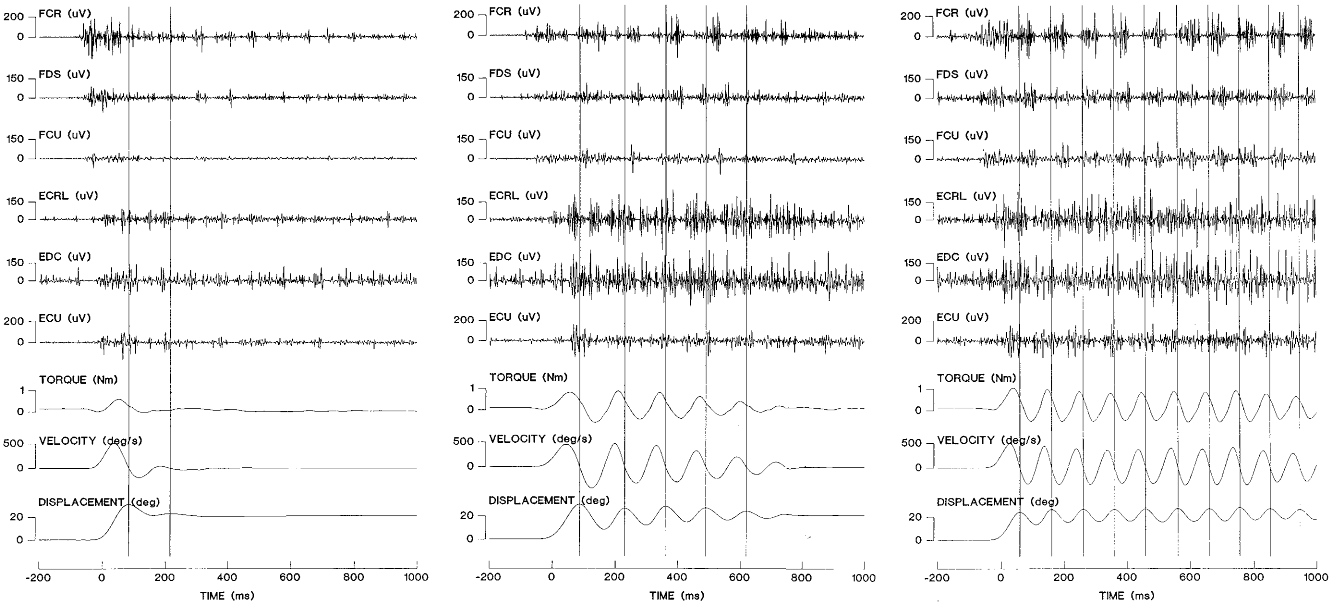

Fig. 1 Single-trial records for one subject with negative damping levels of 1, 3, and 5 *from left to right*, respectively. In order, *from top to bottom*, the first six traces (microvolts) are EMGs from flexor carpi radialis (*FCR*), flexor digitorum superficialis (*FDS*), flexor carpi ulnaris (*FCU*), extensor carpi radialis longus (*ECRL*), extensor digitorum communis (*EDC*) and extensor carpi ulnaris (*ECU*) muscles, the last three traces are wrist torque, angular velocity, and displacement (with respect to wrist neutral position). Note increases in muscle activation, number of oscillations, and oscillation frequency with increasing negative damping

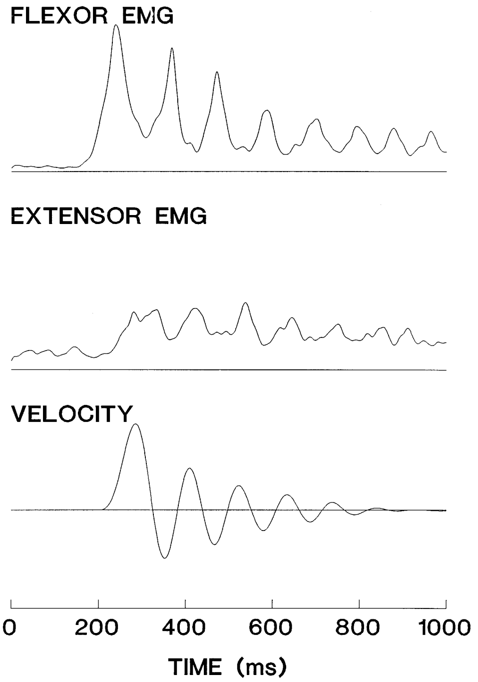

Fig. 2 Reciprocal modulation of flexor and extensor EMG during damping of oscillations. *Top two traces* Mean rectified EMG, low-pass filtered at 30 Hz. Flexor EMG is the sum of FCR and FCU muscle activity, while extensor EMG is the sum of ECRL and ECU muscle activity. *Bottom trace* Wrist angular velocity with flexion positive (upward) and extension negative (downward)

tion declined gradually, but remained above premovement levels, even after oscillation had completely subsided during the postmovement epoch.

Modulation of muscle activity during the task was quantified by computing the rms EMG during different task epochs. ANOVA did not indicate a significant main effect of negative damping level on the EMG of the premovement or movement phases, although the extensor EMG of the movement phase did increase with negative damping level (ECRL, P=0.06; ECU, P=0.2). This main effect was, however, significant for both stabilization (FCR, $P<0.0005$; FCU, $P<0.001$; ECRL, $P<0.0005$; ECU, $P<0.0005$) and post-movement phases (FCR, $P<0.001$; FCU, $P<0.03$; ECRL, $P<0.003$; ECU, $P<0.03$). The normalized rms EMG, averaged across subjects, is shown in Fig. 3. For clarity, only negative damping levels 1 and 4 are illustrated.

A change in the temporal variation of flexor and extensor muscle activation was noted as the negative damping level increased. At level 1, there was a progressive decline in the rms EMG from movement to postmovement, whereas at level 4 the rms EMG peaked during stabilization. For level 1, the rms EMG during movement was significantly higher than during stabilization in flexors ($P<0.05$), although not significantly different in extensors (Fig. 3). This changed systematically as negative damping level was increased, so that at level 4 the rms EMG of three muscles had become significantly greater during stabilization than during movement (FCR, $P<0.05$; ECRL, $P<0.01$; ECU, $P<0.05$). The rms EMG of the FCU was also greater during stabilization than during movement at level 4, although the difference was not statistically significant ($P<0.1$). These results suggest that subjects perceived a need for greater postural stabilization of the wrist as the negatively damped load increased and responded by stiffening the wrist through increased cocontraction of flexor and extensor muscles.

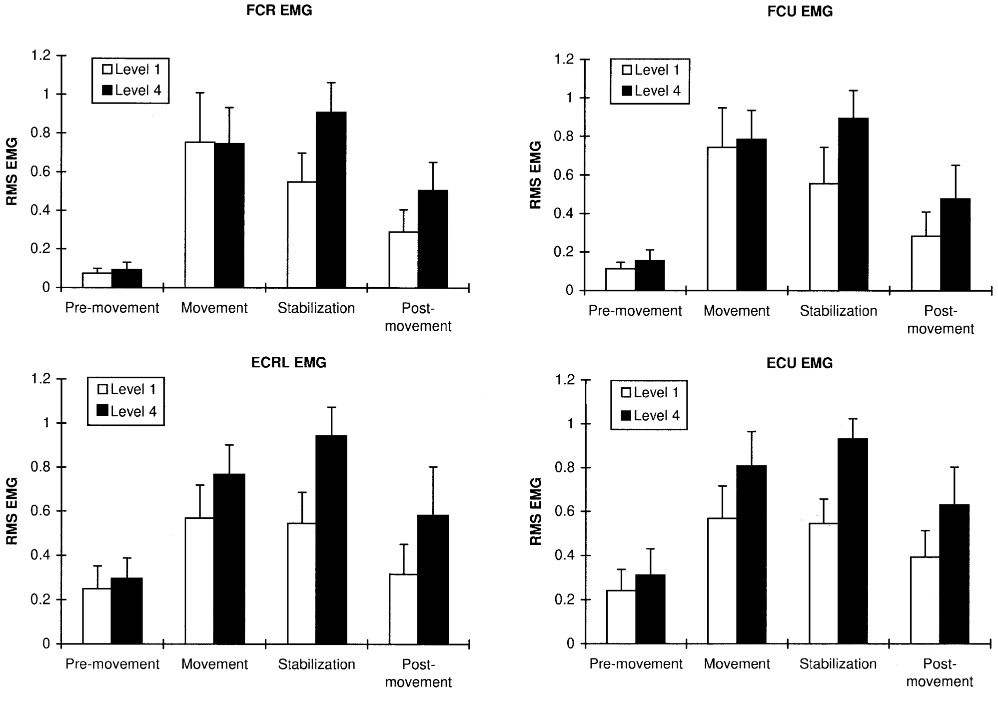

Fig. 3 Comparison of normalized rms EMG for low (1) and high (4) levels of negative damping, computed over time intervals corresponding to premovement, movement, stabilization, and postmovement phases of task. The principal wrist flexors FCR and FCU are shown at the *top* and the principal wrist extensors ECRL and ECU are shown at the *bottom*. Values represent the means and SDs for ten subjects

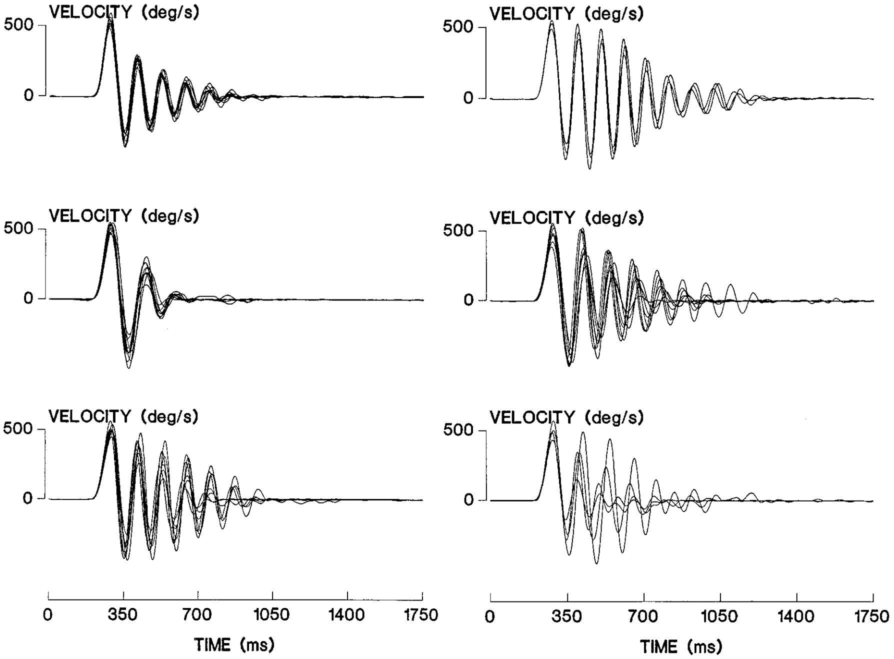

Fig. 4 Trial-to-trial variability in velocity for three different subjects, ranging from the least to the most variable (*top to bottom*, respectively). Trials performed with negative damping level 1 are shown on the *left* and trials performed with negative damping level 4 are shown on the *right*. There were fewer trials that met the acceptance criteria with level 4 than with level 1

Mechanical impedance

Because levels of negative damping were presented in random order, movements were less uniform than they would have been if the subject had been able to perform blocks of trials with one level of negative damping. Trial-to-trial variability for two different negative damping levels is compared for three subjects in Fig. 4, ranging from the least to the most variable.

This trial-to-trial variability was probably responsible for a large part of the intrasubject variability in the estimates of the stiffness and damping coefficient from trial to trial. We quantified the variability by computing the coefficient of variation (ratio of standard deviation to mean) for the stiffness and damping coefficient of each subject for each of the 16 conditions (4 negative damping levels×4 half-cycles of oscillation). In the case of stiffness, the mean coefficient of variation for the ten subjects was 11% (ranging from 5 to 16%). In the case of the damping coefficient, the mean coefficient of variation for the ten subjects was 13% (between 9% and 16% for all but one subject, where it was 24%). Thus, the coeffi-

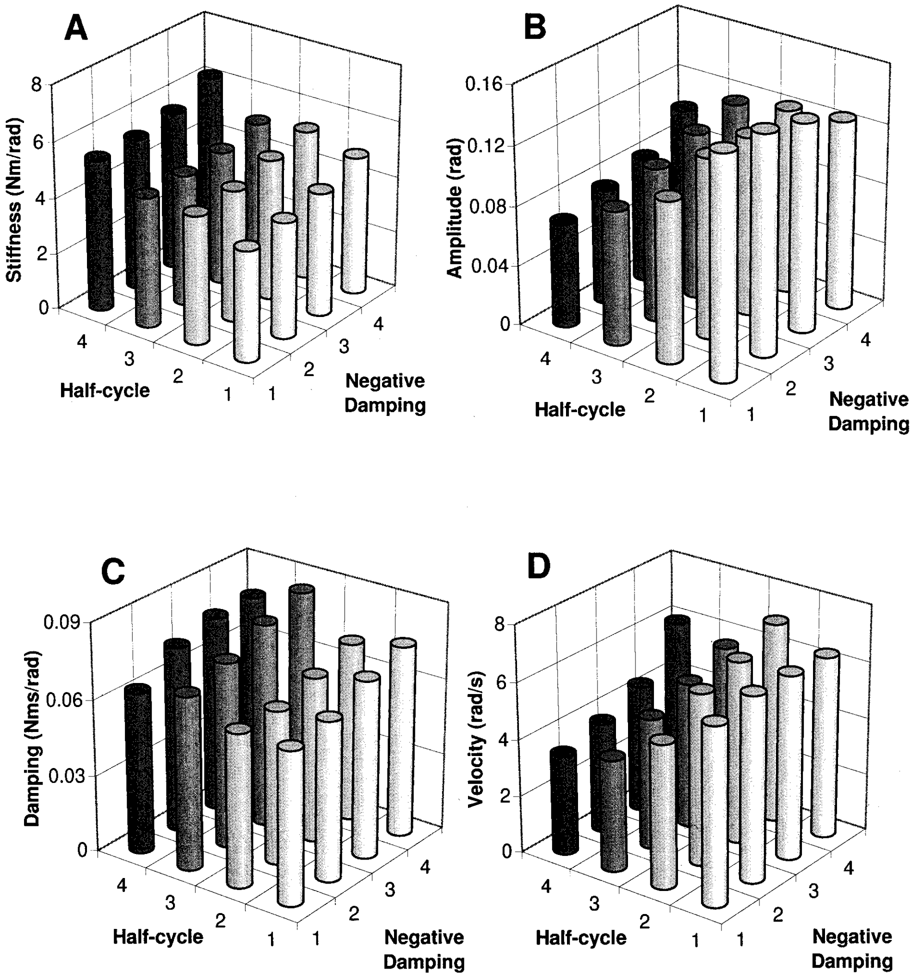

Fig. 5A–D Mean values of mechanical parameters for the ten subjects, displayed in relation to the oscillation half-cycle and negative damping level for which each estimate or measurement was made. **A** Stiffness. **B** Oscillation amplitude. **C** Damping coefficient. **D** Peak angular velocity

cient of variation from trial to trial was slightly greater than 10%. We quantified the intersubject variability in the stiffness and damping coefficient in a similar fashion, but now computing the coefficient of variation across subjects, using the subject means for a given half-cycle and negative damping level. The mean intersubject coefficient of variation across the 16 conditions was 22% for both the stiffness and the damping coefficient, about twice the intrasubject coefficient of variation.

There were clear and consistent changes in the mechanical impedance of the wrist with increasing negative damping of the load. The mean wrist stiffness across subjects increased progressively, for each oscillation half-cycle, as the negative damping increased from level 1 to 4 (Fig. 5A). Two-way ANOVA, using negative damping level and oscillation half-cycle as factors (see Materials and methods), showed a significant main effect of negative damping level (P=0.022).

The mean damping coefficient across subjects changed in a manner similar to stiffness, increasing as the negative damping of the load increased (Fig. 5C). However, this increase in the damping coefficient of the wrist did not produce a significant main effect in the two-way ANOVA (P=0.12).

The damping ratio ($B/2\sqrt{KI}$) reflects the combined effects of the wrist stiffness and damping coefficient. The damping ratio changed in a manner that generally paralleled the changes in the wrist stiffness and damping coefficient, i.e., it increased as the negative damping of the load increased (Fig. 6). Although the increases were relatively small, there was a significant main effect (P=0.013).

To examine the effects of oscillation amplitude and angular velocity on the damping coefficient, parameter estimates were classified according to the half-cycle of oscillation in which they were estimated. Both oscillation amplitude and peak angular velocity diminished as the oscillation was damped (Figs. 1, 5), with the sole exception of negative damping level 4, where the oscillations occasionally grew. Nevertheless, the main effect of half-cycle number on both amplitude and velocity was highly significant (P<0.0005). Hence, the half-cycle number classified the magnitude of oscillation amplitude and velocity in reverse order. The two-way ANOVA showed a highly significant main effect of half-cycle on both the wrist stiffness and damping coefficient (P<0.0005). Since the correlation was positive, it suggests that the stiffness and damping coefficient increased

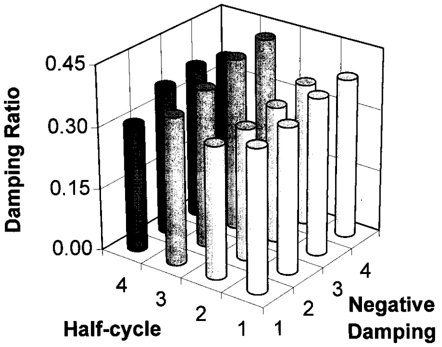

Fig. 6 Mean values of the damping ratio for the ten subjects, displayed as in Fig. 5

as the result of the systematic decrease in oscillation amplitude and/or velocity.

While the main effect was clearly significant statistically, the trend may not be evident when first looking at the data (Fig. 5). Breaking the data down into extension and flexion half-cycles makes it more evident that the mean stiffness and damping coefficient both increase, i.e., compare the first and third half-cycles (extension) or the second and fourth half-cycles (flexion). The damping ratio increased in a similar fashion (Fig. 6). While the increment was not large, it was highly significant ($P<0.0005$).

Discussion

Previous studies that have examined how muscle activation affects damping have shown that the joint stiffness and damping coefficient increase with joint torque under quasi-isometric conditions where voluntary muscle activation is kept constant (Agarwal and Gottlieb 1977; Hunter and Kearney 1982; Lacquaniti et al. 1982; Becker and Mote 1990; Hajian and Howe 1997; Zhang and Rymer 1997). From such results, it can be inferred that there is a positive correlation between joint stiffness or damping coefficient and muscle activation. Several studies have also shown that joint stiffness increases as joint angular displacement decreases (Cannon and Zahalak 1982; Kearney and Hunter 1982; De Serres and Milner 1991), while the damping coefficient appears to increase as joint angular velocity decreases (Kearney and Hunter 1982; Gielen and Houk 1984). The present study has extended these findings to the stabilization phase of voluntary movement and has examined the combined effects of agonist/antagonist muscle cocontraction and joint motion on damping.

Mechanical impedance

Wrist stiffness increased as expected with the increasing levels of agonist/antagonist co-contraction observed as the negative damping of the load increased. The wrist damping coefficient, on the other hand, did not change significantly. There may be two reasons for this. First, the oscillation velocity increased as the negative damping of the load increased. This would have counteracted the effect of increased muscle activation, since the damping coefficient of active muscle (Kirsch et al. 1994) and passive joints (Esteki and Mansour 1996) has been shown to decrease as velocity increases. Second, we did not distinguish between intrinsic and reflex contribution to the damping coefficient. As suggested below, the reflex contribution may have been negative and its effect would probably have increased as the level of muscle activation increased.

Perhaps of greater importance was the effect of oscillation half-cycle. The decreasing oscillation amplitude and velocity produced an increase in both the displacement and velocity-dependent components of the mechanical impedance. This more than compensated for the decreasing muscle activation as the oscillation progressed, such that the damping coefficient actually increased. It would appear that the mechanical properties of the musculoskeletal system are particularly appropriate for rapid damping of oscillations with minimal energy expenditure. Initially, the high elastic and damping torques necessary to reduce oscillation amplitude are achieved by strong agonist/antagonist muscle cocontraction. As oscillation amplitude declines, the muscles can be relaxed without a reduction in the stiffness or damping coefficient. Not only do the stiffness and damping coefficient increase as the oscillations die out, but the damping ratio increases slightly, as well.

Reflex contribution to damping

The contribution of phasic reflex activity to wrist damping must be considered separately from the mean level of voluntary activation. It depends on the phase relation between reflex torque and angular velocity and the size of the reflex response. The phase relation, in turn, depends critically on the frequency of oscillation (Rack 1981; Stein and Lee 1981; Milner and Cloutier 1993; Bennett 1994). In this study, the frequency was essentially determined by the natural frequency of the wrist. The oscillations that we observed ranged from 7 to 8 Hz.

If one assumes that joint angle and muscle length are in phase when the muscle is active (Fellows and Rack 1987; Amis et al. 1987) and that stretch reflex latencies obtained from ramp joint displacements are also valid under conditions where the joint undergoes sinusoidal displacement, then the frequency at which the short-latency stretch reflex will contribute in a negative way to the damping coefficient can be estimated. Short-latency reflex EMG of wrist muscles peaks 35–40 ms after the onset of muscle stretch (Jaeger et al. 1982; De Serres and Milner 1991), while the delay from motor unit action potential to peak twitch force is about 60 ms in wrist muscles (Riek and Bawa 1992). Therefore, if stretch reflex latency is measured from a zero velocity point, peak reflex torque will be aligned with peak muscle shortening velocity at frequencies in the 7.5- to 8-Hz range. Such reasoning would suggest that the short-latency stretch reflex was largely contributing in a negative way to the wrist damping coefficient in these experiments. It must be emphasized, though, that the magnitude of such a negative contribution cannot be determined accurately from the size of the phasic EMG bursts since the muscle may be rapidly shortening. A rapidly shortening muscle will generate considerably less force than an isometric or lengthening muscle. A negative contribution of the stretch reflex to the damping coefficient is also suggested by the results of Bennett (1994), where the reflex contribution to mechanical impedance during the stabilization phase of voluntary movement was shown to be negative for an oscillation frequency of 8 Hz. The results of Zhang and Rymer (1997) for isometric contractions of the elbow are similarly suggestive. They showed that estimates of the damping coefficient were lower for a model of the joint impedance that lumped intrinsic and reflex damping than one from which the reflex contribution was removed.

Modeling and parameter estimation

The model used in this study was that of a linear second-order system with parameters that were assumed to vary relatively slowly over time, i.e., they were assumed to remain constant over one half-cycle of oscillation. With this model, damping torque is directly proportional to joint angular velocity and the elastic and damping torques sum. Such models are commonly used to model joint mechanics and have been shown to be valid under a variety of operating conditions (Agarwal and Gottlieb 1977; Hunter and Kearney 1982; Lacquaniti et al. 1982; Becker and Mote 1990; Bennett et al. 1992; Hajian and Howe 1997). Caution must be exercised, though, in using these models for simulations or predictions, since the elastic and damping coefficients are not constant but vary with muscle activation, joint displacement, and joint angular velocity.

There are only a few published results with which to compare the estimates of the wrist damping coefficient obtained in the present study. Although the damping coefficient (usually referred to as viscosity in previous studies) was not expressed in the same units in some of these studies, all values have been converted to the same units for the purpose of this comparison. The passive damping coefficient of the relaxed wrist was reported to be between 0.02 and 0.03 Nm·s/rad (Gielen and Houk 1984), which is lower but of the same order of magnitude as the estimates for active wrist damping in the present study, as would be expected. The damping coefficient of the wrist, when subjects were required to exert a torque of 0.1 Nm (less than 1% MVC), was reported to be 0.61 Nm·s/rad by Sinkjaer and Hayashi (1989), with a corresponding stiffness of 26 Nm/rad. These values are much larger than the values obtained in the present study. Sinkjaer and Hayashi estimated stiffness, damping coefficient, and moment of inertia as parameters in a linear second-order model of the impulse response. An error in any one parameter could have led to errors in the others. For comparison, two other studies, in which wrist stiffness was determined in a more direct manner, found values of 2–3 Nm/rad for flexor torques below 5% MVC (Gielen and Houk 1984; De Serres and Milner 1991). Only when subjects were exerting torques of greater than 60% MVC (Milner et al. 1995) did wrist stiffness approach 26 Nm/rad. Based on these studies, the estimate of wrist stiffness in the study by Sinkjaer and Hayashi (1989) appears to be almost an order of magnitude too large. Perhaps, the wrist damping coefficient was similarly overestimated, in which case the value would correspond more closely to that of the present study.

The damping ratio has been reported for several joints for a range of muscle activation levels. In the case of the ankle joint (Gottlieb and Agarwal 1978; Hunter and Kearney 1982), the damping ratio was found to vary from about 0.25 to 0.45. A similar damping ratio (0.20–0.40) was reported for abduction/adduction of the metacarpophalangeal joint of the index finger (Becker and Mote 1990), although Hajian and Howe (1997) reported a value of approximately 1.0 for flexion/extension of the finger. In the case of the elbow, Bennett et al. (1992) reported that the damping ratio varied between 0.2 and 0.6 during voluntary sinusoidal motion, although they questioned the accuracy of their estimates. A damping ratio has also been reported for the elbow in a task involving cocontraction of antagonistic muscles. Lacquaniti et al. (1982) found that the damping ratio varied from 0.08 to 0.21, tending to increase as the level of cocontraction increased. It is noteworthy that only in the study of Hajian and Howe (1997) was the mechanical impedance estimated without the contribution of reflexes and that only in this case was the joint not underdamped. Like all other studies in which the reflex contribution was included, we found that the damping ratio was much less than 1, indicating that the wrist was underdamped.

It is clear from the results of the present study that, during voluntary movement, the mean level of muscle activation, the oscillation amplitude and the joint angular velocity all affect damping. The activation-dependent mechanical properties of muscle are complemented by length- and velocity-dependent properties, which effectively cascade the damping process by increasing the stiffness and damping coefficient as oscillation amplitude declines. Furthermore, these amplitude- and velocity-dependent properties may play an important role in offsetting the potential destabilizing effects of reflex delays.

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