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Compensation for mechanically unstable loading in voluntary wrist movement

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Abstract. In order to study the roles of muscle mechanics and reflex feedback in stabilizing movement, experiments were conducted in which healthy human subjects performed targeted wrist movements under conditions where the damping of the wrist was reduced with a load having the property of negative viscosity. If the movement speed and negative viscosity were sufficiently high, the wrist oscillated for several hundred milliseconds about the final target position. Subjects increased the activation of both wrist flexor and extensor muscles to increase joint stiffness to damp the oscillations. With practice, both the tendency to oscillate and the level of muscle activation were reduced. A small bias torque in either direction, added to the negative viscosity, enhanced the oscillations as well as the amount of flexor and extensor muscle activation during the stabilization phase of fast movements. The tendency for the wrist to oscillate was also seen during slow movements where the oscillations were superimposed upon the voluntary movement. We suggest that this reduction in mechanical stability is primarily of reflex origin. As wrist stiffness increases, the natural frequency of the wrist also increases, which in turn produces an increase in the phase lag of the torque generated by the myotatic reflex with respect to wrist angular velocity, effectively reducing damping. The oscillation frequency was often close to a critical frequency for stability at which torque, due to the myotatic reflex, lagged angular velocity by 180° (6–7.5 Hz). Nevertheless, subjects were able to damp these oscillations, probably because the torque due to intrinsic muscle stiffness (in phase with position and hence lagging velocity by only 90°) dominated the torque contribution of the myotatic reflex. Increasing stiffness with declining oscillation amplitude may also have contributed significantly to damping.

Key words: Wrist – Mechanical stability – Stiffness – Damping – Human

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Introduction

Mechanical stability is important in the control of mechanical systems such as the human musculoskeletal system, where sensory feedback is delayed. Human movement is generally stable despite the potential for oscillation such as that in physiological and pathological tremor. To investigate the stability of maintained posture, Rack and his colleagues (Brown et al. 1982; Joyce et al. 1974) conducted experiments on healthy human subjects, examining the frequency response characteristics of the neuromusculoskeletal system by sinusoidally rotating various joints. These earlier studies suggested that, under most conditions, the natural frequency of a limb segment is low enough so that the myotatic reflex helps to provide mechanical stability by increasing the joint stiffness. However, the delayed sensory feedback of the myotatic reflex can endanger mechanical stability with particular combinations of voluntary force and frequencies of joint rotation.

The mechanical properties of joints, such as stiffness and viscosity, change with active joint torque. Hunter and Kearney (1982) showed that, under quasi-isometric conditions, stiffness and viscosity both increase with joint torque, thereby increasing the natural frequency of the joint and possibly modifying the damping ratio. Joyce et al. (1974) and Prochazka and Trend (1988) showed that the myotatic reflex can engender self-sustaining oscillations of the elbow for certain combinations of inertial load and muscle activation. Rack (1981) suggested that the only means of avoiding such instability is either never to enter these regions or to enter them so briefly that the instability cannot be completely expressed.

When two mechanical systems interact, the risk of instability may increase, since the stability of the coupled system may be lower than that of each system in isolation. For example, Joyce et al. (1974) showed that adding mass to the forearm can shift the elbow from an operating region that is mechanically stable to another that is unstable. Colgate and Hogan (1988) have analyzed "cou-

pled stability", a property of systems, stable in isolation, which allows them to remain stable when coupled to any passive system that is also stable in isolation. An example of a passive system is a combination of rigid bodies and springs. Colgate and Hogan suggested that the human arm is able to interact stably with a diverse set of environments that are stable in isolation because it behaves like a passive system.

The achievement of stable interaction requires that the mechanical behavior of the human arm be adapted to the task. Evidence that reflex responses are modulated or gated in a task-dependent manner (Capaday and Stein 1986, 1987; Lacquaniti et al. 1991; Brooke et al. 1992), as well as during different phases of a task (Dufresne et al. 1980; Soechting et al. 1981; Lacquaniti et al. 1982), indicates that neural control is adaptive, although such adaptation may be limited.

The potential for mechanical instability might be particularly high at the end of a fast goal-directed movement, because the gain of the myotatic reflex is relatively high at this point (Soechting et al. 1981). Since joints oscillate naturally, owing to the spring-like characteristics of muscles, particular combinations of joint stiffness and limb segment inertia at the movement endpoint could reinforce oscillations at critical frequencies through the action of the myotatic reflex. Since mechanical instability is not common in healthy human subjects, either these critical frequencies are rarely encountered or it is possible to counteract instability with neuromuscular mechanics.

Under quasi-isometric conditions, oscillation of a joint at its natural frequency may be reinforced by the myotatic reflex. However, the mechanical behavior of a limb segment is different during movement than during posture (Bennett et al. 1992; Milner 1993). Relatively low joint stiffness during movement might keep the natural frequency of the elbow below the critical frequency where oscillations are reinforced by the myotatic reflex. Joints have low stiffness during movement because relatively little muscle activation is required, even for fast movement. Increasing the inertial load results in increased muscle activation and joint stiffness, but this additional inertia could prevent the natural frequency from increasing. Since the most common loads encountered in normal movements are inertial, and since fast movements with large inertial loads are rare, the musculoskeletal system may generally operate outside of the mechanically unstable region.

To gain greater insight into the capacity of neural control to adapt when the potential for mechanical instability is present, it is first necessary to create instability. One way to create instability is to remove the natural damping from the musculoskeletal system. This can be done by coupling the limb to an active mechanical system with negative viscosity (Lakie et al. 1984). If the resulting mechanical behavior is undamped or underdamped, the frequency and amplitude of the oscillations could provide information about the musculoskeletal mechanics and the neural mechanisms that maintain stability. This approach was employed in the study reported in this paper, using positive velocity feedback to an electromechanical torque motor to generate negative viscosity.

Materials and methods

Seven normal healthy subjects (three women and four men ranging in age from 19–45 years) participated voluntarily in this study. All subjects were right-handed, and the experiments were carried out with the right hand only. All of the subjects gave their informed consent to the procedure.

Apparatus

A torque motor (PMI U16M4) was used to generate loads under computer control. The maximum torque that could be produced was 1.7 Nm. 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 control signal to the torque motor consisted of a constant torque which was summed with a second torque acting in the direction of movement and proportional to the velocity. The latter torque constituted positive velocity feedback. The negative viscosity created by positive velocity feedback reduced the damping of the wrist and manipulandum.

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, restraining the thumb, but leaving the fingers free. These pads were positioned to align the axis of rotation of the wrist over the motor axis.

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; the center-to-center separation of the zones was 30°. The cursor position on the screen corresponded to the angular position of the wrist. The angular position of the initial target zone was adjusted to be at the neutral position of the wrist for each subject.

Prior to each movement, the subject held the manipulandum within the initial zone for 1 s. The subject was instructed to reduce any oscillations about the target position as quickly as possible. Subjects were instructed to rotate the wrist at one of three angular velocities, corresponding to movements performed in 550, 235, or 100 ms. Movement duration was defined as the time taken to move the 28° between the boundaries of the initial zone and the target zone. A movement duration target was displayed throughout the trial to help subjects attain the correct velocity. The width of the target allowed for an error of $\pm 20\%$ of the desired movement duration. The actual movement duration was superimposed on this display immediately after each trial. A trial was considered successful only if the movement duration was within $\pm 20\%$ of the requested duration and if the subject met a "stability" criterion that no movement should occur outside of the target zone beginning 800 ms after movement onset until the end of the trial. Movement outside of the target zone (e.g., due to overshooting or oscillation) was permitted prior to this point.

There were 18 different movement conditions consisting of all possible combinations of the 3 movement durations, 3 bias torque levels and 2 positive velocity feedback gains. Bias torque levels were 0 Nm (no load), 0.5 Nm (opposing flexion), and -0.5 Nm (assisting flexion). These loads were small, representing less than 15% of maximal isometric voluntary contraction. The positive velocity feedback gain was set to create negative viscosity of either $-0.043\ \mathrm{Nm \cdot rad^{-1} \cdot s}$ or $-0.057\ \mathrm{Nm \cdot rad^{-1} \cdot s}$, referred to as "low" negative viscosity and "high" negative viscosity, respectively. The

value chosen for the higher positive velocity feedback gain allowed a practiced subject to achieve the stability criterion 100% of the time. The lower value made this feedback almost undetectable in slow movements, while still producing oscillations in the fastest movements with bias loads. The magnitudes of these negative viscous loads are substantially higher than the viscosity of the relaxed wrist, estimated to be 0.015–0.025 $Nm \cdot rad^{-1} \cdot s$ (Gielen and Houk 1984). Thus, the manipulandum and relaxed wrist were negatively damped with either of the positive velocity feedback gains.

Our first subjects had considerable difficulty in achieving a movement duration of 235 ms when the bias torque assisted flexion, with either high or low negative viscosity. Therefore, these two movement conditions were eliminated, leaving 16 conditions to be investigated in all subjects.

The subjects participated in two recording sessions on separate days. During the first session, they began by practicing several movements without loading to become familiar with each of the movement durations. Subsequently, they performed five trials with 550 ms, 0 Nm, $-0.043\ Nm \cdot rad^{-1} \cdot s$, followed by five trials with 100 ms, -0.5 Nm, $-0.057\ Nm \cdot rad^{-1} \cdot s$. Following these preliminary trials they completed five sets of all 16 movement conditions presented in random order. This gave the subjects an opportunity to try different control strategies in order to achieve the best performance. If a trial was unsuccessful, that condition was repeated until success was achieved before continuing to the next condition in the random sequence. Finally, the subjects again performed five trials for the two preliminary movement conditions. Only the two sets of five trials at the beginning and end of this session were stored and analyzed to examine the effect of practice.

The second recording session consisted of eight sets of the 16 movement conditions presented randomly. The data from the last five of these sets were stored and analyzed.

Recording

Electromyographic activity (EMG) 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). The EMG was recorded using active, bipolar, stainless steel surface electrodes (Liberty Mutual MYO 111) with a bandpass of 45-550 Hz and fixed gain of 4600. 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: radial deviation for ECU and 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 quantified by computing the root-mean-square (rms) values over four intervals corresponding to different phases of the task: (1) premovement; (2) movement to the target; (3) stabilization at the target; (4) postmovement. The interval boundaries were chosen such that EMG occurring within the interval would be expected to contribute to wrist torque during the corresponding phase of the task, given the delay between electrical and mechanical muscle activation. The time at which the wrist position crossed the boundary of the initial zone (R in Fig. 1) was used as a reference point for defining the EMG intervals. The premovement interval constituted a 50-ms period

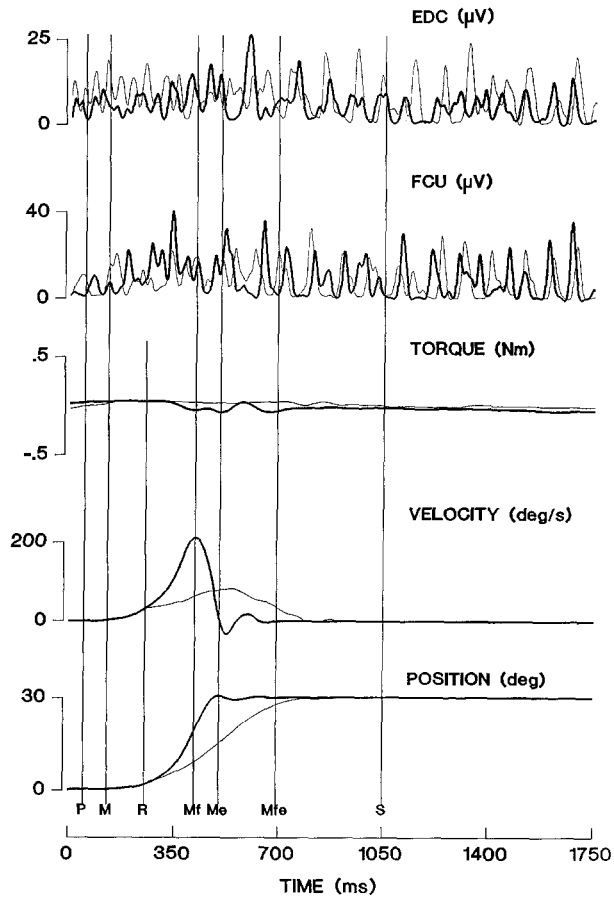

Fig. 1. Movements made at low velocity (550 ms movement duration, *thin lines*) and at intermediate velocity (235 ms movement duration, *thick lines*) with activity from representative muscles. Electromyographic activity (EMG) was rectified and low-pass filtered at 30 Hz for display purposes. Motor viscosity was $-0.057\ Nm \cdot rad^{-1} \cdot s$. *Labelled vertical lines* indicate interval divisions for computation of rms EMG (*P*, end of pre-movement interval; *M*, onset of movement interval; *R*, time at which wrist position crossed boundary of initial zone; *Mf* and *Me*, end of movement interval for flexors and extensors, respectively, during fast and intermediate speed movements; *Mfe*, end of movement interval of flexors and extensors for slow movements; *S*, end of stabilization interval and onset of postmovement interval). *EDC*, extensor digitorum communis, *FCU*, flexor carpi ulnaris

beginning 250 ms prior to R, from 0 ms to P in Fig. 1. The interval used for analysis of movement-associated EMG began 125 ms prior to R (M in Fig. 1). This interval had different durations for the agonist (flexor) and antagonist (extensor) muscles in the cases of 235 ms and 100 ms movement durations. The movement interval ended at peak wrist velocity for flexors (Mf in Fig. 1) and at peak wrist displacement overshoot for extensors (Me in Fig. 1). However, for the relatively slow, 550-ms movement duration, the interval was the same for the two muscles, ending when the wrist entered the target zone (Mfe in Fig. 1). The stabilization interval was defined as the period from the end of the movement interval (Me or Mfe) until 800 ms following movement onset (S in Fig. 1). The post-movement interval was defined as the remaining 700 ms of the trial (beginning at S).

One measure of oscillation about the target position was the number of oscillations in each velocity record. Oscillations were counted only if they had a regular appearance, i.e., the velocity

waveform appeared to be a smooth sinusoid centered about zero. This quantification was carried out only if there was at least one complete oscillation cycle.

For the slowest movement condition, regular fluctuations in the velocity were often present during movement to the target. These fluctuations were isolated from the underlying voluntary movement by digital high-pass filtering using a sixth-order Butterworth filter with a 2.5-Hz cut-off frequency. Phase distortion was eliminated by filtering backward as well as forward in time.

When regular oscillations were observed, mechanical parameters (inertia, viscosity, stiffness, and oscillation amplitude) were estimated by fitting the velocity record with the equation representing the solution to the following differential equation.

$$I\ddot{\theta} + B\dot{\theta} + K\theta = -B_m\dot{\theta}$$

where I is the combined inertia of the wrist, manipulandum and motor, B is the wrist viscosity, K is the wrist stiffness, and B_m is the motor viscosity (B_m is always negative). This equation was not intended to model control of the movement, but rather to model the perturbation response, i.e., the oscillation about the trajectory or final position resulting from the negative viscosity of the motor. The analytical solution to this differential equation for the angular velocity, $\dot{\theta}$ [with $\dot{\theta}(0)=0$] is

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

where θ_0 is the oscillation amplitude

$$\omega = \sqrt{\frac{K}{I} - \left(\frac{B+B_m}{2I}\right)^2}$$

and

$$\zeta = \frac{B+B_m}{2I}$$

Parameter estimation was carried out using a modified Marquardt-Levenberg procedure which minimized the residual sum of squares. It was often evident that either the stiffness or damping changed with time, since the frequency of oscillation varied. Therefore, parameters were assumed to be constant only over a half-cycle of the oscillation. They were re-estimated over successive half-cycles, which were defined as beginning and ending at successive velocity zero-crossings.

The data recorded during the second recording session were analyzed by computing the mean EMG and parameter values for all subjects together under each of the 16 conditions and then comparing various conditions for statistically significant differences using a one-sided Student t-test for means. Differences were considered statistically significant only if $P<0.05$.

Results

Stability

When positive velocity feedback control of the torque motor was implemented during voluntary wrist flexion, the combined mechanical system of wrist and manipulandum became severely underdamped if the movement speed and feedback gain were sufficiently high. Figure 2 compares a movement made by a naive subject, after approximately 20 practice trials without a load, with the fourth movement in the first block of trials in which the motor had low negative viscosity ($-0.043\ \mathrm{Nm \cdot rad^{-1} \cdot s}$). The movements were nearly identical until the first velocity peak, but with no load, stable posture was achieved after only two cycles of oscillation while six cycles can be clearly distinguished prior to stabilization when the motor was negatively damped. A considerable increase in the activation of both wrist flexor and extensor muscles was also evident with negative viscosity.

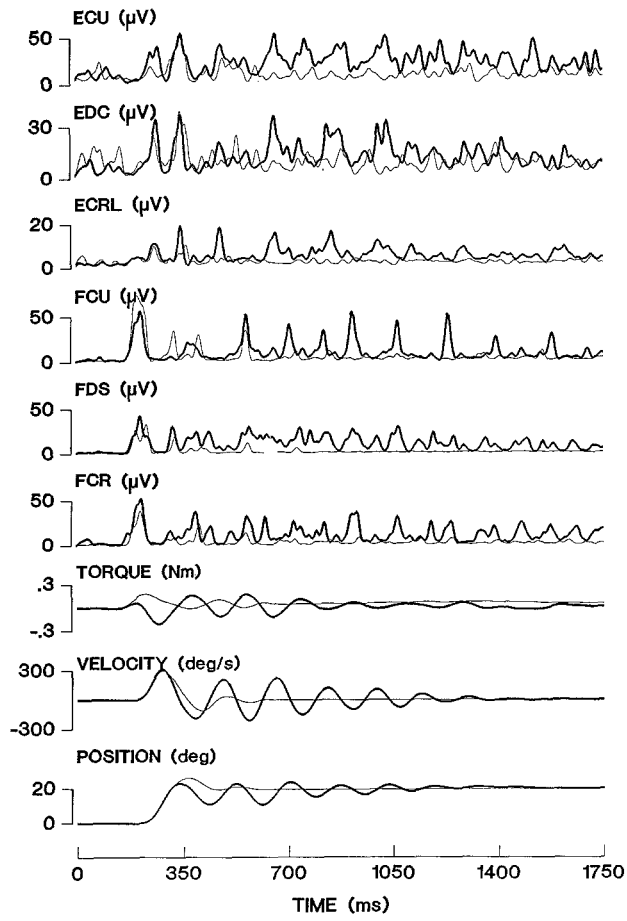

Fig. 2. Single-trial comparison of fast movements (100 ms movement duration) made with no load (*thin lines*) and when positive velocity feedback to the torque motor was used to create negative viscosity of $-0.043\ \mathrm{Nm \cdot rad^{-1} \cdot s}$ (*thick lines*). Note the increased flexor/extensor activation when the motor was negatively damped. Subject AS. *EDC*, extensor digitorum communis; *ECU*, extensor carpi ulnaris; *ECRL*, extensor carpi radialis longus; *FCU*, flexor carpi ulnaris; *FDS*, flexor digitorum superficialis; *FCR*, flexor carpi radialis

The greatest tendency for oscillation occurred when the movements were fast (movement duration 100 ms), the motor had high negative viscosity ($-0.057\ \mathrm{Nm \cdot rad^{-1} \cdot s}$) and there was a bias torque. In the case of a bias torque assisting flexion, 30 of 35 movements had one or more oscillations, with a bias torque opposing flexion the proportion was 21 of 35 and with no bias torque it was 11 of 35. Without bias torque, stabilization was consistently achieved within a single oscillation cycle (Fig. 3).

Oscillations were extremely rare for movements made at the intermediate speed (235 ms movement duration), whether or not there was a bias torque opposing flexion.

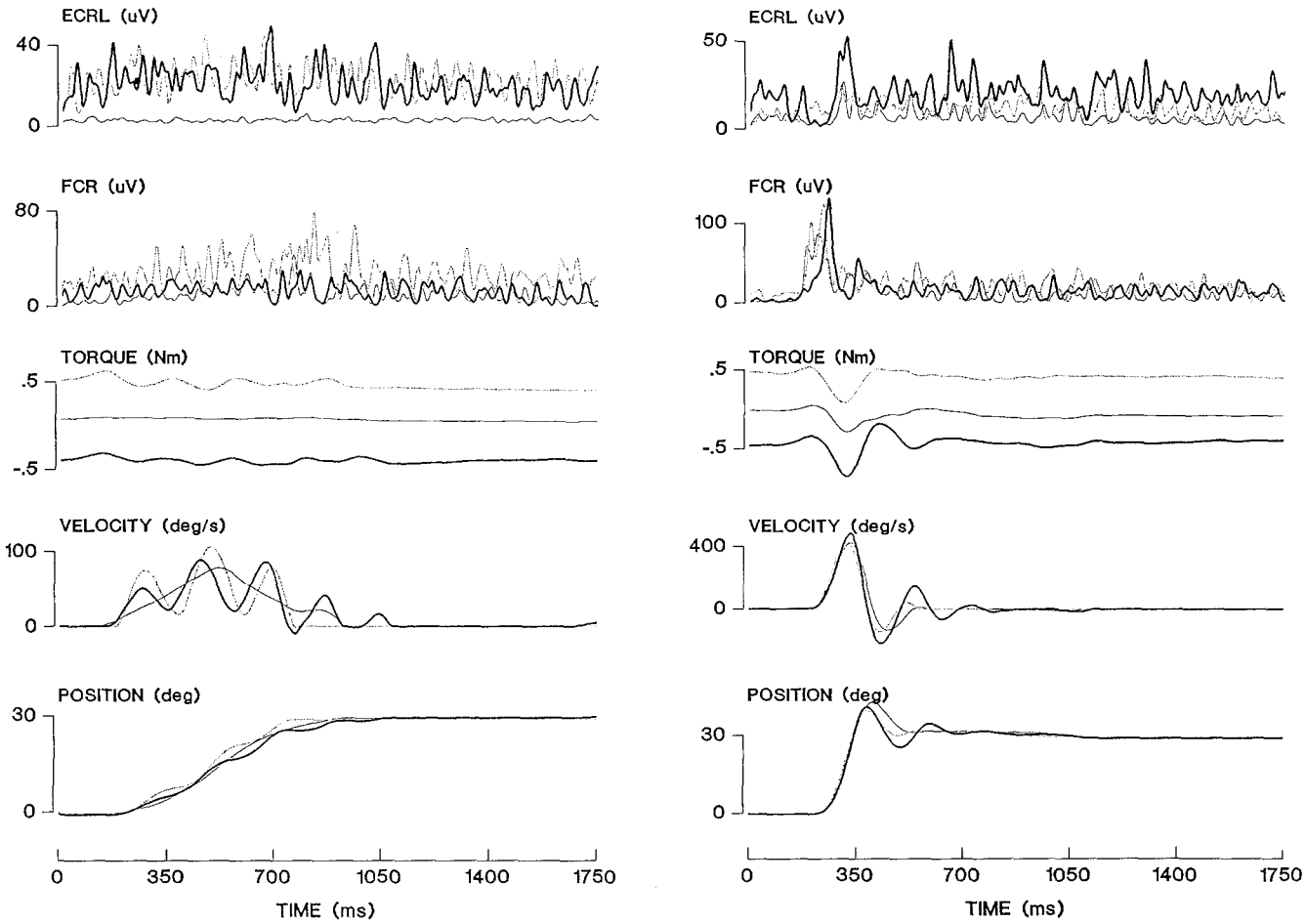

Fig. 3. Single-trial comparisons of slow movements (*left*) and fast movements (*right*) for different bias torques (0 Nm, *thin lines*; 0.5 Nm, *dotted lines*; −0.5 Nm, *thick lines*) with a negative viscosity of $-0.057\ \mathrm{Nm \cdot rad^{-1} \cdot s}$. The most active flexor and extensor muscles are displayed (see Fig. 2 for abbreviations). Positive torques opposed wrist flexion. Subject SM

Note that movements at this speed were not performed with a bias torque assisting flexion because subjects had great difficulty in achieving target movement duration.

At the lowest velocity, there were frequently small amplitude oscillations during movement towards the target when there was a bias torque either assisting or opposing flexion. These oscillations were much larger and more regular than any oscillations during slow movements without a bias torque (Fig. 3).

During the first recording session, subjects initially moved too rapidly when the bias torque assisted movement, greatly overshooting the final target zone which led to large-amplitude oscillations (Fig. 4). With practice, though, they learned to initiate the movement with less flexor muscle activity while also decreasing the subsequent activation of both flexor and extensor muscles.

Data from all subjects obtained during the second recording session (after practice) were grouped according to bias torque, movement speed, and motor viscosity. In order to determine whether there was a greater tendency for oscillation when the motor had high negative damping than when it had low negative damping, the mean number of oscillations per trial was compared. A significant increase was found only in the case of the fastest movements. The increase was greatest when the bias torque assisted flexion ($P < 0.0005$) and least when there was no bias torque ($P < 0.05$).

Electromyographic activity

The mean rms EMG values for all subjects were grouped by movement condition and compared across conditions. For the fastest movements with high negative viscosity, activity in *all* muscles was significantly greater during the stabilization and post-movement intervals when a bias torque was present in either direction than when there was no bias torque ($P < 0.05$). This increased activity became less pronounced when the tendency to oscillate was reduced by reducing the movement speed or decreasing the negative viscosity of the motor. Thus, subjects increased their activation of both flexor and extensor muscles during the stabilization phase of the task when confronted with mechanical instability. This led to an increase in wrist stiffness, as will be evident from our analysis of mechanical behavior.

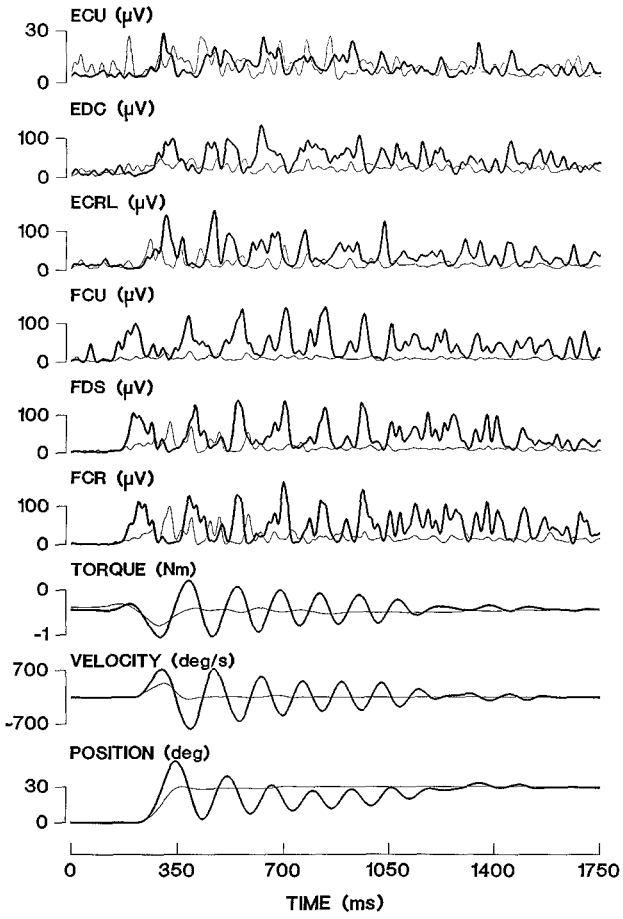

Fig. 4. Single-trial comparison of fast movements (100 ms movement duration) made with a bias torque of -0.5 Nm and negative viscosity of $-0.057\ \text{Nm}\cdot\text{rad}^{-1}\cdot\text{s}$ before (*thick lines*) and after (*thin lines*) several hours practice. Note the lower peak velocity and reduced flexor/extensor activation following practice. Subject JG. (See Fig. 2 for abbreviations)

Under conditions which produced oscillation, the rms EMG in all muscles was greater during stabilization than during postmovement ($P<0.05$). When the bias torque assisted flexion, this difference was statistically significant (except for ECU), but when the bias torque opposed flexion the differences were not significant (except for FCU). It should be noted that cocontraction of ECU and FCU could create a net force, pushing against the support surface of the manipulandum (in the direction of ulnar deviation), without flexing or extending the wrist if the contributions of these two muscles to the net wrist torque were equal. This might explain why the behavior of these two muscles differs slightly from that of the other muscles.

The rms EMG was compared for trials matched for movement duration and bias torque for the movement condition when the motor had low negative viscosity with that when the motor had high negative viscosity. In one movement condition (100 ms, -0.05 Nm), flexor muscle activity was significantly increased during the stabilization and post-movement phases ($P<0.05$). However, this higher level of muscle activity did not produce higher wrist stiffness. An analysis of the mechanical behavior is presented in the following section.

Large-amplitude oscillations were often associated with alternating bursts of activity in at least one pair of flexor and extensor muscles (Fig. 4). However, frequently flexors and extensors were tonically very active and it was not possible to distinguish bursts. Oscillations during slow movements were not associated with identifiable bursts of activity (Fig. 3).

Changes in the timing and relative intensity of agonist and antagonist muscle activity were often linked with improved performance. This was most evident during fast movements where the bias torque assisted flexion. With practice, subjects were able to reduce the general level of flexor and extensor muscle activation throughout the movement, stabilization. and postmovement phases of the task and to reduce the rate of change of activation of the flexor muscles at movement onset. This resulted in a lower initial acceleration (a more gradual increase in the slope of the velocity) without altering peak velocity Fig. 5).

Mechanics

During the stabilization interval following fast movements, where a number of oscillations followed the arrival in the target zone (Fig. 6A), the frequency of oscillation often changed as the amplitude of these oscillations decreased. The model equation of an underdamped, harmonic oscillator adequately described the variation in velocity over each half-cycle of oscillation. Changes in the oscillation frequency presumably resulted from changes in wrist stiffness. The parameter values estimated by fitting the model to the oscillations of Fig. 6A are listed in Table 1.

The frequency of oscillation and the wrist stiffness varied with the amount of flexor and extensor activity associated with movement initiation and subsequent levels of activation. This correspondence is illustrated in Fig. 7, where large initial bursts and subsequent high activation during the stabilization phase of one trial resulted in a higher frequency of oscillation (7.7 Hz as compared to 4.4 Hz) and values of wrist stiffness which were 3–4 times greater than those on the subsequent trial, when both flexor and extensor activation were relatively low (stiffness values for Fig. 7 are listed in Table 1).

There is a critical frequency of oscillation at which the torque contributed by the myotatic (stretch) reflex will lag the wrist angular velocity by 180°, acting like negative viscosity. We have computed this critical frequency and the corresponding wrist stiffness (Appendix). Instability could arise when the stiffness is in the range of 7.5–11 $\text{Nm}\cdot\text{rad}^{-1}$ (corresponding to natural frequencies of 6–7.5 Hz). Though the stiffness reached a critical level of 10 $\text{Nm}\cdot\text{rad}^{-1}$ in both Fig. 6A and in the high frequency oscillation of Fig. 7, the subjects nevertheless succeeded in damping the oscillations. This suggests that the gain of the myotatic reflex is sufficiently low that mechanical stability is ensured.

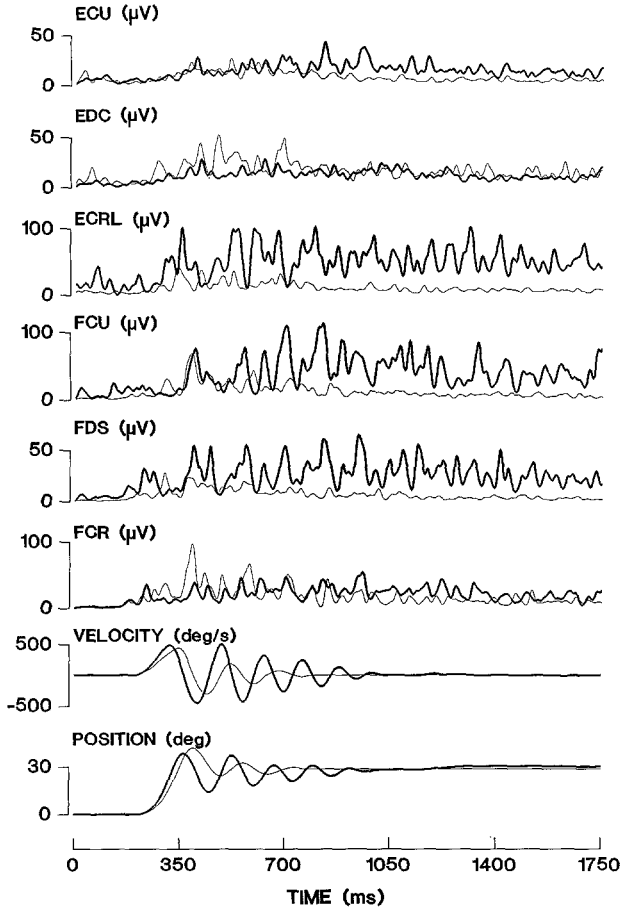

Fig. 5. Same format as in Fig. 4, but for another subject (NP). Peak velocity is the same, but the initial acceleration is lower and flexor/extensor activation is reduced following practice. (See Fig. 2 for abbreviations)

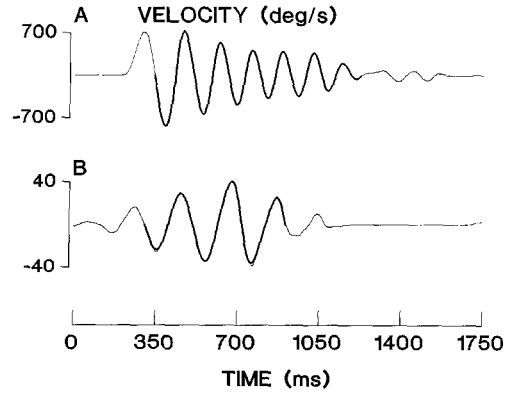

Fig. 6. A Velocity record from Fig. 4. Data was fit with the equation for an underdamped oscillator (*thick line*). Parameters were estimated separately for each half-cycle of oscillation following the first velocity zero-crossing. **B** Velocity record from slow movement in Fig. 3 (torque assisting wrist flexion) after high-pass filtering (2.5 Hz cut-off) and fit as in **A**

The estimated parameter values for conditions where oscillations were present were averaged across all subjects (see Table 2). During the stabilization of fast movements, the stiffness was usually lowest during the first half-cycle of oscillation and increased as the oscillations were damped. Again, despite the fact that the stiffness was often in the critical range of 7.5–11 $Nm \cdot rad^{-1}$, subjects were always able to damp the oscillations.

As noted above, we found one situation (100 ms, −0.5 Nm) where rms EMG during the stabilization and postmovement phases was greater for trials where the motor had high negative viscosity than when it had low negative viscosity. We compared the estimated wrist stiffness and oscillation amplitude (Table 2). Despite higher muscle activation, the wrist was actually less stiff during the first oscillation ($P < 0.0005$), although the oscillation amplitude was significantly larger ($P < 0.0005$). We showed previously in a postural task that static wrist stiffness decreases as displacement amplitude increases for a variety of loads (De Serres and Milner 1991). This nonlinear characteristic of wrist stiffness evidently also contributes significantly to the dynamic behavior of the joint.

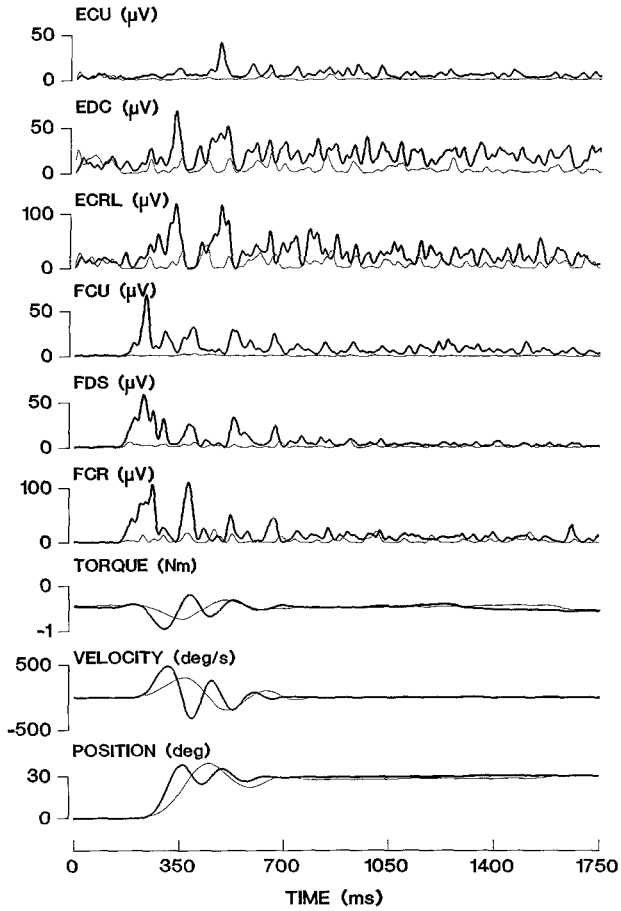

Fig. 7. Single-trial comparison of fast movements (100 ms movement duration) made with a bias torque of −0.5 Nm and negative viscosity of $-0.057\ Nm \cdot rad^{-1} \cdot s$ on two successive trials at the beginning of the first recording session. Note the differences in initial acceleration, muscle activation, and frequency of oscillation. Subject MB. (See Fig. 2 for abbreviations)

Table 1. Estimated mechanical parameter values

Half-cycle[a]	Wrist stiffness ($Nm \cdot rad^{-1}$)	Wrist viscosity ($Nm \cdot rad^{-1} \cdot s$)	Inertia[b] ($kg \cdot m^2$)	Oscillation amplitude (rad)
Fig. 6A (fast movement, 100 ms)				
EXT (1)	6.40	0.032	0.00518	−0.371
FLX (2)	8.56	0.098	0.00538	0.363
EXT (3)	8.88	0.028	0.00539	−0.245
FLX (4)	11.04	0.056	0.00551	0.209
EXT (5)	11.12	0.037	0.00554	−0.177
FLX (6)	12.79	0.053	0.00557	0.148
EXT (7)	13.98	0.022	0.00562	−0.120
FLX (8)	12.96	0.045	0.00569	0.140
EXT (9)	13.16	0.024	0.00571	−0.113
FLX (10)	12.65	0.037	0.00571	0.130
EXT (11)	16.05	0.103	0.00575	−0.094
FLX (12)	12.91	0.108	0.00589	0.085
Fig. 6B (slow movement, 550 ms)				
EXT (1)	4.88	0.029	0.00490	−0.0112
FLX (2)	4.62	0.055	0.00500	0.0167
EXT (3)	4.30	0.042	0.00503	−0.0186
FLX (4)	4.77	−0.003	0.00497	0.0160
EXT (5)	5.26	0.157	0.00497	−0.0290
FLX (6)	6.96	0.000	0.00485	0.0090
Fig. 7 (high frequency oscillation)				
EXT (1)	11.01	0.079	0.00504	−0.128
FLX (2)	10.81	0.085	0.00500	0.105
EXT (3)	8.76	0.221	0.00494	−0.084
FLX (4)	11.06	0.078	0.00504	0.033
Fig. 7 (low frequency oscillation)				
EXT (1)	2.66	0.082	0.00509	−0.171
FLX (2)	3.79	0.074	0.00511	0.072
EXT (3)	5.79	0.115	0.00537	−0.026

[a] Each half-cycle of oscillation is identified by its direction, i.e., wrist flexion (FLX) or extension (EXT). Numbers in parentheses indicate chronology of half-cycles
[b] Combined inertia of manipulandum and hand

Consistent with these observations is the fact that estimated wrist stiffness increased as oscillation amplitude decreased when several oscillation cycles occurred during the stabilization phase of fast movements (Tables 1, 2). When a bias torque was present, extensor half-cycles were not compared with flexor half-cycles, since the bias torque required that one muscle group be more highly activated than the other. As a result, for the same oscillation amplitude, there was always an asymmetric resistance to muscle stretch (stiffness), which was higher in the direction of the bias torque.

Not obvious in the figures, but readily apparent when the scale is enlarged, are the variations in skewness of the oscillations from one half-cycle to the next. These variations were due to differences in wrist viscosity. The greater the wrist viscosity, the earlier in time the occurrence of the peak velocity.

When a bias torque was present, the wrist viscosity, like the wrist stiffness, tended to be lower during the half-cycle when the loaded muscles were being stretched than when their antagonists were being stretched.

In order to analyze the oscillations that occurred during the slowest movements, it was necessary to remove the voluntary component with digital high-pass filtering. The initial acceleration phase of the movement was excluded from this analysis to bypass the initial transient response of the filter to the sudden change in velocity and to avoid the possibility of including a component of the voluntary movement among the oscillations. For similar reasons, the final deceleration phase was also excluded (Fig. 6B).

When multiple oscillations were present during slow movements, we compared the wrist stiffness at different points in the movement. The stiffness did not change significantly during the movement (Table 1). Furthermore, the wrist stiffness did not differ significantly between the half-cycles of the oscillation that stretched the flexor muscles and those stretching the extensor muscles. Nor was there much difference in the amplitude of oscillation. Wrist viscosity tended to be asymmetric, but considerably less so than during the stabilization of fast movements (Table 2).

When the negative damping of the motor was high, the wrist stiffness during slow movements was lower ($P < 0.0025$) and the oscillation amplitude was higher ($P < 0.01$), than when the motor was less negatively

Table 2. Mechanical parameter means and standard deviations

Bias torque (Nm)	Motor viscosity ($Nm \cdot rad^{-1} \cdot s$)	Movement time (ms)	Half-cycle[a]	Wrist stiffness ($Nm \cdot rad^{-1}$)		Wrist viscosity ($Nm \cdot rad^{-1} \cdot s$)		Oscillation amplitude (rad)		n
				Mean	SD	Mean	SD	Mean	SD	
0	−0.057	100	EXT (1)	4.08	1.26	0.094	0.043	−0.197	0.107	11
			FLX (2)	9.22	6.60	0.123	0.052	0.059	0.050	11
0.5	−0.057	100	EXT (1)	5.73	1.66	0.056	0.051	−0.107	0.037	20
			FLX (2)	9.14	3.54	0.094	0.055	0.057	0.041	20
			EXT (3)	8.94	6.06	0.065	0.055	−0.039	0.021	12
			FLX (4)	13.58	8.83	0.051	0.063	0.030	0.026	5
	−0.057	550	EXT	5.67	1.76	−0.010	0.031	−0.016	0.006	21
			FLX	5.74	1.98	0.011	0.042	0.015	0.006	23
	−0.043	550	EXT	8.34	8.38	0.023	0.082	−0.013	0.008	15
			FLX	7.80	5.12	0.000	0.090	0.011	0.006	17
−0.5	−0.057	100	EXT (1)	5.80	1.55	0.081	0.035	−0.150	0.069	29
			FLX (2)	6.78	2.43	0.024	0.043	0.076	0.048	29
			EXT (3)	9.52	4.08	0.068	0.049	−0.053	0.033	22
			FLX (4)	7.60	2.24	0.003	0.056	0.048	0.034	16
			EXT (5)	13.87	6.82	0.073	0.081	−0.031	0.022	11
	−0.043	100	EXT (1)	9.37	5.04	0.140	0.031	−0.067	0.027	18
			FLX (2)	7.76	3.12	0.044	0.070	0.026	0.013	18
	−0.057	550	EXT	5.90	1.90	0.030	0.068	−0.022	0.017	36
			FLX	5.55	1.33	0.012	0.056	0.018	0.008	39
	−0.043	550	EXT	8.72	5.19	0.032	0.071	−0.013	0.010	24
			FLX	9.16	7.22	0.022	0.069	0.013	0.010	26

[a] Each half-cycle of oscillation is identified by its direction, i.e., wrist flexion (FLX) or extension (EXT). Numbers in parentheses indicate that half-cycles have been grouped chronologically. Where no numbers appear (i.e., for slow movements) half-cycles have been grouped by direction only

damped, even though there was no significant difference in the rms EMG of any of the wrist flexor or extensor muscles. This is further evidence of the nonlinear, amplitude-dependent behavior of wrist stiffness.

The inertia of the manipulandum and hand was similar for all seven subjects. A mean value was calculated for each subject across all conditions where oscillations were present. The mean values ranged from approximately 0.0052–0.0054 $kg \cdot m^2$ with standard deviations of less than 10% of the mean, principally because the inertia of the manipulandum was several times as large as that of the hand.

Discussion

We have shown that when mechanical stability is reduced by decreasing the effective viscosity of the wrist, subjects adopt a strategy of increasing wrist stiffness by increasing the activation of wrist flexor and extensor muscles. This strategy was effective in damping oscillations about the endpoint of the movement. Another contribution to damping was the nonlinear, amplitude-dependent behavior of wrist stiffness, which increased as oscillation amplitude decreased. When a constant bias torque was added to the negative viscosity of the motor, the number of oscillations increased, whether movements were fast or slow. We attribute this to a decrease in wrist viscosity owing to reflexes. Subjects usually reduced the amount of wrist flexor and extensor activity after they had practiced the task for several hours. This decrease in muscle activity produced a decrease in wrist stiffness, accompanied by a decrease in the oscillation frequency and in the total number of oscillations during stabilization. These findings are discussed in more detail below.

Increased wrist stiffness during the stabilization and postmovement phases effectively reduced the amplitude of oscillations about the target position of the wrist. This reduction in oscillation occurred because any net torque acting to displace the wrist, whether originating internally or externally, was met by more elastic resistance. Oscillation was also reduced owing to the nonlinear properties of muscle stiffness which increase as muscle stretch decreases (Joyce et al. 1969; Nichols and Houk 1976). As the oscillation amplitude decreased, the wrist stiffness increased without requiring additional muscle activation. In fact, the wrist stiffness increased despite a concomitant decrease in muscle activation during the stabilization and postmovement phases. Similar behavior during forced oscillation of the wrist was reported by Lakie et al. (1984).

The amplitude dependence of wrist stiffness had the effect of damping wrist oscillation. Because the elastic resistance to wrist displacement (wrist stiffness) increased as oscillation amplitude decreased, oscillation amplitude was attenuated more rapidly than if the stiffness had remained constant. This may partly explain why oscil-

lations following fast movements are rare, even though limb segments such as the forearm are apparently underdamped during voluntary movement (Bennett et al. 1992).

The introduction of a bias torque clearly increased the number of oscillations, whether the oscillations occurred while moving toward the target with slow movements or following the arrival of the wrist at the target with fast movements. This increased oscillation indicates that, with the bias torque, the movements were less damped, suggesting that the reduction in damping was of reflex origin. The increased muscle activation necessary to counter the bias torque produced higher wrist stiffness, thereby raising the natural frequency of the wrist. This increase in natural frequency would reduce damping by increasing the phase lag of wrist torque with respect to wrist angular velocity.

The torque produced by motion of the wrist is the sum of the torque due to the intrinsic stiffness of wrist muscles and the torque due to the activation of muscles by the myotatic reflex. The intrinsic stiffness increases as muscle stiffness increases with increasing muscle activation. The reflex stiffness increases with increasing reflex responsiveness ("gain"). The intrinsic stiffness is in phase with wrist angular position and hence lags wrist angular velocity by 90°. The reflex stiffness also lags wrist angular velocity, but the phase lag depends on the oscillation frequency, increasing with increasing oscillation frequency (see Appendix). When the phase lag reaches 180°, the myotatic reflex torque acts like negative viscosity, reducing the intrinsic damping. If the negative viscosity becomes sufficiently large, it can produce mechanical instability.

Clearly, this did not happen. Even though negative viscosity was already present in the form of positive velocity feedback to the torque motor, subjects were nearly always successful in damping oscillations of the wrist. The main reason must have been that the torque contributed by the intrinsic muscle stiffness completely dominated any torque due to the myotatic reflex. This would have prevented the net phase lag from reaching 180°. This conclusion is supported by our observations in a previous study that most of the torque produced by stretch of the wrist flexor muscles came from intrinsic rather than reflex sources (De Serres and Milner 1991).

By reducing the amount of flexor and extensor muscle activity, subjects reduced the metabolic cost of performing the task. They likely also benefitted from increased mechanical stability by reducing the phase lag of myotatic reflex torque with respect to wrist angular velocity (as the natural frequency of the wrist decreased) and by reducing the reflex "gain" (shown to decrease with decreasing muscle activation by De Serres and Milner, 1991).

The results of this study indicate that the strategy of agonist/antagonist muscle cocontraction is effective in damping externally induced oscillations, even at relatively high frequencies of oscillation where the myotatic reflex could potentially create mechanical instability. Mechanical stability is apparently guaranteed because the torque due to intrinsic muscle stiffness exceeds that due to the myotatic reflex at these frequencies. In addition, the fact that joint stiffness increases as oscillation amplitude decreases helps to increase the rate at which oscillations are attenuated. Under normal circumstances, it appears quite unlikely that the wrist would ever become mechanically unstable.

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Appendix

As the frequency of oscillation of a joint increases, there is an increasing phase lag of joint torque, produced by reflexively generated muscle force, with respect to oscillation velocity. This is due to the fixed delay between the onset of muscle stretch and the myotatic reflex response. There is a critical frequency of oscillation at which the phase lag will equal 180°. At this frequency, the myotatic reflex torque will act like negative viscosity (Rack 1981), assisting rather than resisting motion, causing oscillation amplitude to grow. For this to happen, the reflex torque must be in phase with velocity. For example, the peak torque produced by wrist extension (flexor torque) must occur at the same time as peak flexion velocity. Wrist extension begins when the angular velocity of the wrist crosses zero, becoming negative; peak flexion velocity occurs three-quarters of a cycle later. Thus, reflex torque must be delayed by three-quarters of a cycle from the onset of muscle stretch.

The critical frequency can therefore be calculated from the relation $f=0.75/\Delta T$, where ΔT is the delay between the zero velocity crossing (onset of muscle stretch) and the peak flexion torque. We can estimate ΔT as follows: the delay to the onset of torque, following rapid stretch of wrist flexor muscles is approximately 40 ms (Gielen and Houk 1984); the delay from onset of torque to peak torque during twitch-like activation, such as would occur as the result of a short burst of activity, is about 60 ms (Riek and Bawa 1992); this gives a ΔT of 100 ms and a critical frequency of 7.5 Hz. Given that the total inertia of the manipulandum and wrist was approximately $0.005\ \mathrm{kg \cdot m^2}$, the critical frequency for stability corresponds to a wrist stiffness of approximately $11\ \mathrm{Nm \cdot rad^{-1}}$.

The above calculation was based only on the short-latency response to muscle stretch. If the long-latency response is included, the torque response is broadened, delaying the peak by another 20–30 ms. This brings the critical frequency down to 6 Hz with a corresponding wrist stiffness of approximately $7.5\ \mathrm{Nm \cdot rad^{-1}}$.

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