Stretch reflex responses in the human elbow joint during a voluntary movement

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1. The responsiveness of the stretch reflex is modulated during human voluntary limb movements. The influence of this modulation on the limb mechanical properties (stiffness) was investigated.

2. Subjects were taught to replicate accurately a rapid (4·0 rad s^{-1}) targeted elbow flexion movement of 1 rad. From the onset of 12 % of the trials a sinusoidal position disturbance (0·05 rad) was superimposed on the normal (trained) movement trajectory. The net joint torque (muscle torque) resisting these stretches was computed from measurements of applied torque, acceleration and limb inertia. Electromyographic (EMG) responses in the triceps brachii (TB), brachialis (Br) and biceps brachii (BB) were monitored.

3. The EMG responses to sinusoidal stretches applied early in the movement were less than those responses to perturbations applied when the arm neared the target (especially in the antagonist muscle TB). These EMG responses caused fluctuations in the resistance to the perturbation (stiffness), as described below.

4. When the perturbation frequency was low (< 4 Hz) the resistance of the elbow muscles to the stretch increased as the arm approached the target (48 % increase). In contrast, when the stretch frequency was 7 Hz the resistance *decreased* by 63 %. This decrease can be explained by the increased reflex response, since at 7 Hz the reflex response is probably timed so that it assists, rather than resists, the stretching as a result of loop delays. This reflex timing was confirmed by observing that, after abruptly stopping the sinusoidal stretch, the reflex response persisted for 100 ms and was indeed in a direction that would have reduced the resistance, had the perturbation continued.

5. The time course of the net muscle stiffness was estimated for frequencies ranging from 4 to 8 Hz and for each 40 ms interval a Nyquist plot was constructed, forming a C-shaped curve as frequency was varied. The size of this curve gave a measure of the stiffness resulting from reflex activity. When the arm neared the target this reflexive stiffness reached a maximum, and was probably comparable in size to the intrinsic (non-reflexive) muscle stiffness. Also, in four of the five subjects the viscous component of stiffness at 7 Hz dropped significantly below zero when the arm neared the target, again indicating that at this frequency the reflex was large and acted inappropriately.

6. In summary, the contribution of the stretch reflex to the mechanical response of the arm is relatively small during a movement but increases as the target is approached, to a maximum level where it contributes substantially to the total resistance to low frequency perturbations. This increase is, however, insufficient to cause limb instability resulting from reflex delays since: (i) the reflexive component of stiffness is only transiently high and (ii) at the frequencies where the delayed reflex responses act inappropriately (6–7 Hz) the limb inertia dominates the response to disturbances.

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During a voluntary limb movement the responsiveness of the stretch reflex is continuously modulated. Electromyographic (EMG) responses to muscle stretch (Gottlieb & Agarwal, 1979; Soechting, Dufresne & Lacquaniti, 1981) and nerve activation (Gottlieb, Agarwal & Stark, 1970; Capaday & Stein, 1987; Llewellyn, Yang & Prochazka, 1990) are modulated according to the phase of the movement. However, due to the complexity of muscle force production (e.g. Hill, 1938; Bawa & Stein, 1976; Aldridge & Stein, 1982) the mechanical consequences of these EMG modulations are difficult to predict.

Various methods have been used to characterize the reflex contribution to muscle stiffness. Usually total muscle stiffness is assumed to be a linear sum of the immediate mechanical response of the muscle (intrinsic stiffness), and the delayed response produced by changes in muscle activation (reflex stiffness). While such a distinction between the intrinsic and reflex stiffness is an approximation that neglects the non-linear properties of muscle (e.g. Aldridge & Stein, 1982), it has proved useful in predicting behaviour resulting from reflex activity, such as tremor (Joyce, Rack & Ross, 1974; Jacks, Prochazka & Trend, 1988; Prochazka & Trend, 1988). In this paper a similar distinction is adopted between the intrinsic and reflex stiffness.

In the case of isometric contractions, the contribution of reflexes to the total mechanical stretch response has been investigated by deafferentation. For example, in decerebrate cats Nichols & Houk (1976) compared muscle stretch responses with and without afferent feedback, while assuring similar patterns of muscle activation under both conditions. The results indicate that reflex activity can compensate for muscle yielding upon stretch, contributing substantially to the total stiffness. The contribution from reflex activity to the total muscle stiffness appears to saturate at activation levels above one-third of maximum (Hoffer & Andreassen, 1981). Similar conclusions have been reached through studies in man employing electrical stimulation in lieu of deafferentation (Sinkjaer, Toft, Andreassen & Hornemann, 1988; Carter, Crago & Keith, 1990). However, in human studies it is more difficult to be certain that the subject's muscle activation patterns are the same with and without this effective deafferentation.

Another method used to study the reflex contribution during isometric contractions takes advantage of the delays in the reflex path to dissociate the reflex response from the intrinsic response of muscles. The simplest approach is to apply a very brief stretch (tendon jerk), and study the mechanical consequences of the reflex activity that ensues (e.g. Aldridge & Stein, 1982). Likewise, taking advantage of the delay in the reflex path, Rack and colleagues (Joyce *et al.* 1974; Brown, Rack & Ross, 1982; Evans, Fellows, Rack, Ross & Walters, 1983; also see Matthews & Watson, 1981) have used sinusoidal perturbations to investigate the mechanical contribution from reflexes during isometric contractions. As a result of feedback delay, the contribution from reflexes to the total stiffness develops an increasing phase lag as the perturbation frequency increases. Thus, since the intrinsic stiffness of muscle is relatively insensitive to changes in frequency, it is possible to determine approximately the reflex contribution as that component of the total stiffness which changes with frequency.

At a 'critical frequency' the forces developed from the reflex activity are delayed to be 180 deg out of phase with the velocity of the imposed movements; the reflexes aid the imposed movements rather than resist them. If the reflex gains are sufficient, the muscles then produce a net positive amount of work on the torque motor imposing the movements, that is, the viscous stiffness is negative (Joyce *et al.* 1974). Depending on the reflex latencies, this critical frequency ranges from 4 to 12 Hz (Joyce & Rack, 1974; Brown *et al.* 1982; Prochazka & Trend, 1988). If the mechanical resonant frequency happens to match this critical frequency and the reflex gains are sufficiently high ($> 1{\cdot}0$), then spontaneous unstable oscillations ensue (Joyce & Rack, 1974; Jacks *et al.* 1988; Prochazka & Trend, 1988).

The objective of the present work was to use sinusoidal perturbations to characterize the reflex contribution to the muscle stiffness during voluntary human elbow movement. A technique of applying controlled *position* disturbances during well-learned targeted movements was employed (Bennett, 1993*a*), thus avoiding length-dependent non-linearities of muscle stiffness. The time-varying mechanical response of the limb was estimated at frequencies ranging from 4 to 8 Hz, using a modification of an ensemble averaging method developed by Bennett, Hollerbach, Xu & Hunter (1992). The present actuator and the method of limb attachment were improved significantly over that of Bennett, Hollerbach, Xu & Hunter (1992), allowing a 0·05 rad perturbation amplitude to be maintained well above 10 Hz and allowing sufficiently accurate estimates of limb inertia to be obtained. The latter allowed direct estimation of the net muscle force, which was often less than 10 % of the total measured force, by subtracting the large component resulting from the forearm inertia.

The results show that there was a large modulation in the magnitude and phase of the torque response during the second half of a movement. This modulation was consistent with that expected from the observed modulation of reflexes as measured by EMG activity. At certain critical frequencies the total viscous stiffness of the arm was unexpectedly low and at times negative. Parts of the present study have been presented in abstract form (Bennett, 1991).

METHODS

Experiments were performed on the left arm of five normal human subjects, all of whom gave informed consent to the experimental procedures. Experiments were approved by the local ethical committee. Subjects were aged between 25 and 30 years old; three were female, two male.

The apparatus and protocol were identical to those described in earlier papers (Bennett, 1993*a*,*b*), except that sinusoidal rather than step position errors were imposed starting at the onset of elbow movement. Only moderately fast (about 4 rad s^{-1}) movements were investigated. The effects of movement speed on stiffness are reported elsewhere (Bennett, 1993*a*).

Briefly, the subject's forearm, wrist and hand were immobilized in a custom-fitted cast that was bolted to a low

inertia composite beam. The beam was coupled through a strain gauge torque sensor to a torque motor (RS0608FN005; NSK Nippon Seiko, Tokyo, Japan) such that the elbow joint was aligned with the motor rotation axis. This allowed free elbow movement in the horizontal plane. During most trials the torque motor remained off; the subjects executed self-paced 1 rad elbow flexion movements from a 'start' position to a 'target' position, and attempted to match a velocity target displayed on an oscilloscope. This display showed only the velocity measured at the time of passing a point 0·015 rad from the start (onset time). Nevertheless, by insisting on a 1% velocity-matching error before a movement was deemed 'successful', subjects soon learned to repeat the whole trajectory with less than 0·03 rad error at any point in time (Fig. 1). The display of velocity thus allowed subsequent movements to be improved, but did not allow tracking during a given movement. Movements which did not match the velocity criterion at the onset were discarded; these amounted to about 50 % of the movements (depending on the subject's skill). The running average of all such 'successful' unperturbed trials was computed on-line, and termed the 'normal' or 'nominal' trajectory, $\theta_n(t)$.

At the onset of 25 % of successful trials the torque motor was turned on. The motor was operated in a position servo-control mode, and held on a trajectory that was the sum of the 'normal' trajectory plus a desired sinusoidal displacement: $\theta_n(t) + A\sin(2\pi ft + \phi)$. The amplitude ($A$) of the superimposed sinusoid was usually 0·05 rad, the frequency (f) ranged from 2 to 8 Hz and the phase (ϕ) was chosen at random. When the phase was not zero or π (a zero crossing of the sinusoid) the motor was not turned on until the time of the next zero crossing. There was thus no large torque transient at the onset of the perturbation.

The subjects were told that when they were perturbed they must ignore the perturbations and continue the movement as usual. As before, we found that subjects could readily ignore the perturbations, and could not report the direction (phase) of a perturbation (Bennett, 1993*a*). Catch trials were introduced during 50 % of the perturbed trials; that is, the motor came on as usual for 100 ms and was then turned off. These catch trials assured that the subject did not adopt a strategy that relied on the torque motor to bring the arm to the target during perturbed trials.

Elbow joint position, acceleration and applied motor torque were monitored, low-pass filtered at 200 Hz and sampled at 1400 Hz, as described in Bennett (1993*a*). All signals were digitally low-pass filtered at 25 Hz without phase distortion. EMG signals were measured with surface electrodes on the triceps brachii (TB), brachialis (Br) and the biceps brachii (BB). EMG signals were rectified and shown as a percentage of the peak EMG measured in a maximum-speed self-arrested 1 rad flexion movement (Bennett, 1993*a*).

Inertia estimation

Before elbow joint stiffness can be estimated, an accurate estimate of limb inertia must be obtained. A white noise (0–15 Hz) length perturbation was applied to the limb, and a second-order model, $\tau_s = I\ddot{\theta} + B\dot{\theta} + K\theta$, was fitted to the measured torque (τ_s) and length (θ) (Bennett, 1993*a*), where I is the inertia, B the damping and K the stiffness. Both conditions of relaxed and isometrically contracting flexor muscles (10 % MVC) were tested. Inertia estimates were the same regardless of the muscle contraction. This second-order model was also fitted with time-varying parameters, as described below (see also Bennett *et al.* 1992), to observe the influence of the onset of the perturbation. After 160 ms the parameters (I, B and K) always reached a steady state.

Net muscle torque

The net torque generated by all the muscles and connective tissue that acted about the elbow joint was computed by subtracting the forces that were due simply to limb inertia (Bennett, 1993*a*):

$$\tau = I\ddot{\theta} - \tau_s, \quad (1)$$

where τ_s is the measured applied motor torque; $\ddot{\theta}$, the measured joint acceleration and I, the total limb and apparatus inertia. Figure 1 illustrates the result of such a calculation. For

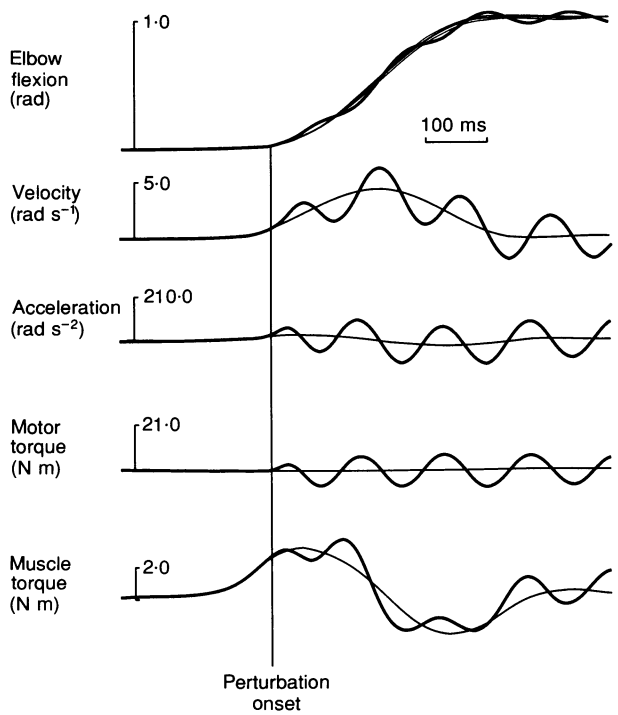

Figure 1. Elbow flexion movements to a target 1 rad from the starting position
Thin lines, unperturbed movement; thick lines, movement with superimposed sinusoidal position perturbation; very thin lines above and below the unperturbed movement path, one standard deviation. Net muscle torque is $\tau = I\ddot{\theta} - \tau_s$; where I is net inertia of the apparatus and arm; $\ddot{\theta}$, elbow joint acceleration; τ_s, motor torque measured in a strain gauge sensor coupling the motor to the arm. Averages of 10 trials. Subject S2; inertia = 0·071 kg m^2. Note the scale differences for the motor and muscle torques. A substantial portion of the motor torque simply accelerates the limb inertia on the perturbed trials.

convenience this computed torque (τ) is referred to simply as the net 'muscle torque'. The muscle torque is defined to be positive in the direction of torque produced by the flexor muscles (Br and BB). Joint angle is also positive in the flexion direction. Note that during unperturbed trials the computed muscle torque was simply proportional to the limb acceleration. Also, during perturbed trials the muscle torque was small in comparison to the applied motor torque accelerating the limb inertia (note scale differences between motor and muscle torques). Measurements of net muscle stiffness thus required an accurate estimate of limb inertia.

Stiffness estimation

Analysis of resistance to the perturbations was carried out using two methods. The first method was to average multiple trials with identical perturbation parameters, and compute the resistance to the perturbation by subtracting the unperturbed from the perturbed net muscle torques.

In the second method, the magnitude and phase of a fixed-frequency sinusoidal response was estimated at 40 ms intervals. For this purpose a time-varying correlation method similar to that developed in Bennett *et al.* (1992) was used as follows. An ensemble of 100 perturbed movements were recorded, in which the perturbations were all of an identical frequency, but of random phase onset. In a given time interval following the movement onset, the net muscle torque (τ) was assumed to be correlated with the elbow position (in-phase component) and velocity (out-of-phase component):

$$-\tau = K\theta + B\dot{\theta} + C, \quad (2)$$

where K is the elastic stiffness, B is the viscosity and C is a fixed offset. The offset (C) can be thought of as the contribution from the nominal trajectory (θ_n) and nominal muscle torque (τ_n) in a linearization of the mechanical properties such that $-\Delta\tau = K\Delta\theta + B\Delta\dot{\theta}$, where $\Delta\tau = \tau - \tau_n$, etc. (Bennett *et al.* 1992). That is, $C = \tau_n - K\theta_n - B\dot{\theta}_n$.

Since the perturbation is sinusoidal: $\dot{\theta} = \omega\theta$, where $\omega = 2\pi f$. The product ωB is thus the portion of the total limb stiffness produced by the viscosity, referred to as viscous stiffness (Brown *et al.* 1982). Thus, relative to the imposed position sinusoid, the magnitude of the torque response is $\sqrt{[K^2 + (\omega B)^2]}$ and the phase is $\tan^{-1}[(\omega B)/K]$.

From the ensemble of 100 perturbed movements, 100 equations of the form (2) were generated for each 40 ms time interval. Within an interval the parameters K, B and C were assumed constant and estimated with standard linear least-squares estimation techniques (p. 61, Norton, 1986). Parameter

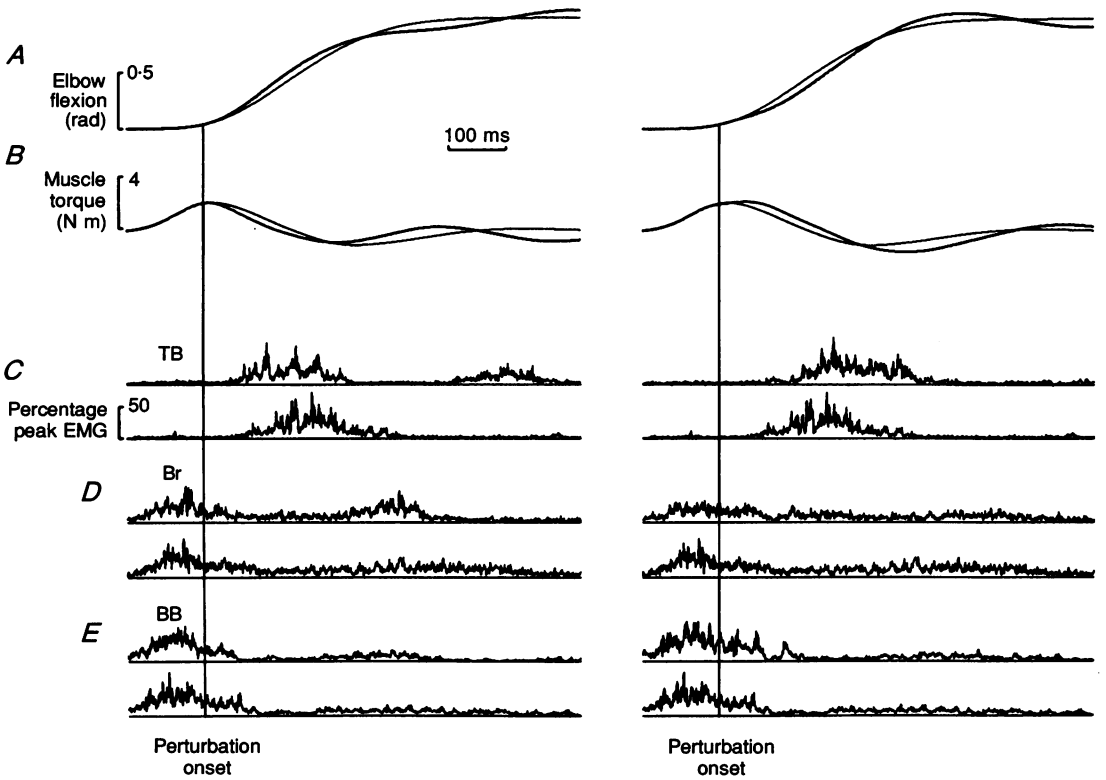

Figure 2. Low frequency (2 Hz) sinusoidal position perturbations imposed during voluntary elbow flexion

First column, perturbation applied with zero phase lag; second column same but with 180 deg phase lag. Peak velocity 3·6 rad s^{-1}. *A*, elbow flexion trajectories; *B*, computed net muscle torque. Thin lines, unperturbed movements; thick lines, perturbed movements. *C–E*, pairs of rectified EMG activity in extensor muscle (TB) and flexor muscles (Br and BB) during perturbed (upper record of each pair) and unperturbed movements (lower record of each pair). EMG shown as percentage of peak EMG of a maximal speed flexion movement (see Methods). Averages of 10 trials. Subject S2; inertia = 0·071 kg m^2. Note the modulation of the normal (unperturbed) EMG activity late in the movement.

variances were used to estimate confidence intervals (CI) for the parameters, as indicated. During many experiments the least-squares estimation was computed on-line by a recursive least-squares estimation procedure (p. 160, Norton, 1986), in which the parameter estimates at each time interval were updated at the end of each successful movement; this reduced data storage requirements. Identical results were obtained with the standard and recursive least-squares estimation procedures.

The resulting time-varying stiffness estimates can be interpreted by the following device based on a technique used by Soechting *et al.* (1981). Suppose that the ensemble of perturbed movement trials (perturbation frequency, f) are ordered according to the onset phase of the perturbation, and that for simplicity the phase increases by equal increments between each trial. If we then observe the amplitude of the perturbation at a fixed time (T) during all trials, then as we proceed in order through the ensemble of trials, this amplitude forms a sinusoid, which is a function of the onset phase (given by the trial index number, α): $\sin(2\pi fT + \alpha)$. At time T, a similar sinusoid of the same period is formed by the muscle resistance to the perturbation: $A\sin(2\pi fT + \alpha + \phi)$. This sinusoid bears a particular phase (ϕ) and magnitude (A) relationship to the input perturbation sinusoid, which has been estimated by the correlation technique described above. Any standard sinusoidal analysis (i.e. linear least-squares estimation) may be used on these ensemble sinusoids, but the trial index (α), rather than time (T), must be used as the independent variable.

If this ensemble sinusoidal analysis is performed on a time-invariant system, then the magnitudes (A) and phase shifts (ϕ; i.e. stiffness parameters) estimated at all times (T) after the onset of the perturbation should be identical, and these parameters should be identical to the parameters obtained with standard time-invariant sinusoidal analysis techniques. Indeed, this was found to be approximately the case in initial tests of the technique, where either subjects held an isometric contraction, or a steel spring was used in lieu of the subject (D. J. Bennett, unpublished observations). Usually the stiffness estimates were unreliable (based on parameter variances) for the first half-cycle of the perturbation, as discussed in Results.

RESULTS

The main finding was that in a certain critical frequency range (6–7 Hz) the resistance to a sinusoidal perturbation was attenuated at the time of the peak deceleration of the arm. This time period corresponds to when the resistance was previously found to *increase* when measured with lower frequency position perturbations (step perturbations, Bennett, 1993*a*; pseudorandom perturbations, Bennett *et al.* 1992). We start firstly by presenting the responses to low frequency sinusoidal perturbations, verifying the earlier studies, and then turn to the main results.

Low frequency responses

When the forearm movement trajectory was altered by the imposed sinusoidal perturbation the muscles resisted this alteration with an approximately sinusoidal change in net muscle torque, as in Fig. 2*A* and *B*, where the thin lines indicate the unperturbed movements and forces, and the thick lines show the effects of the perturbations. The resistance to the perturbation is more readily seen in Fig. 3 where the force and position in the unperturbed movements are subtracted from those recorded in the perturbed movements. For the low frequency perturbations (< 4 Hz) this resistance usually led the imposed position changes, as expected of a mechanical system with viscosity as well as stiffness. The resistance increased as the movement progressed. For example, the peak resistance to the first half-oscillation cycle (0–250 ms) of the 2 Hz stretch applied in Fig. 3 was 8·2 N m rad^{-1}, and increased to 12·0 N m rad^{-1} on the second half-cycle (250–500 ms). This increase was significant and typical of that found in all subjects (48 ± 15 % increase, ± S.D.; $P < 0{\cdot}05$, $n = 5$ subjects). It was also similar to the increase found when step position changes were applied during movement (Bennett, 1993*a*). Notice that the EMG responses to the perturbation only occurred with about an 80 ms latency (Fig. 2).

Reflex asymmetry

The resistance to the perturbation was not always symmetrical about the unperturbed torque profile (Figs 4 and 5; see also Fig. 6 for a less prominent asymmetry). This asymmetry, or bias, could have resulted from muscle non-

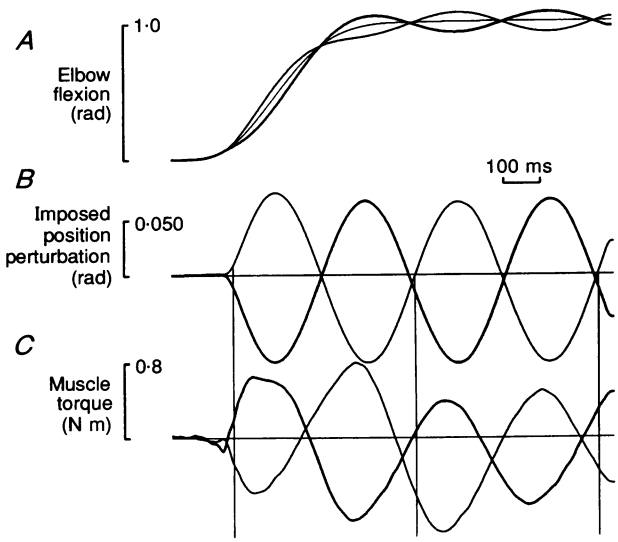

Figure 3. Resistance to perturbations shown in Fig. 2
A, elbow flexion trajectories. *B*, difference between perturbed and unperturbed trajectories (imposed position perturbation). *C*, difference between perturbed and unperturbed net muscle torque (resistance). Thin lines, unperturbed movement; medium lines, perturbation with zero phase lag (relative to onset time, first vertical line) and thick lines, perturbation with 180 deg phase lag. Note that resistance to the perturbation was greater in the second half-cycle of the perurbation than the first. Oscillation cycles indicated by the vertical lines.

linearities (e.g. Rack & Westbury, 1974). However, it appears to be correlated with asymmetries in reflexive EMG activity as follows. It was often found that the antagonist muscle (TB) did not respond to stretch early in the movement, but by the time of the normal (unperturbed) antagonist burst a large reflex response was present (Figs 4 and 6; see also Gottlieb *et al.* 1970). In contrast, the agonist muscles (Br and BB) responded relatively quickly to stretch at all points in the movement. Thus, the mechanical response to early stretch of the agonist must have produced a reflexive torque, but an early stretch of the antagonist did not produce torque. Thus, the mean response was biased in the direction of the agonist. Late in the movement the reflex responsiveness of all muscles was increased. However, the antagonist EMG response magnitude was then typically greater than that of the agonist (relative to the EMG on the unperturbed trials; see Figs 4 and 6), reversing the direction of the torque bias.

The sinusoidal torque response can be seen independently of the slowly changing bias in the background muscle torque by observing the width of the envelope of an ensemble of sinusoidal responses applied with different onset phases. Figure 5 shows two superimposed perturbations with a 180 deg difference in onset phase. In later analysis the stiffness was computed by an ensemble time-varying technique. Implicit in this technique is subtraction of this low frequency 'bias' (see explanation of C in Methods); the technique is similar to computing the time variation in the amplitude of the envelope of responses in Fig. 5.

High frequency responses

The muscles resisted higher frequency perturbations with a similar sinusoidal modulation of EMG and torque to that seen for lower frequencies (e.g. see response to a 7 Hz perturbation in Fig. 6). However, there was a significant reduction in the amplitude and phase of the resistance at the time when the arm neared the target (roughly the time of peak deceleration). Again, this resistance is best seen by subtracting the unperturbed from the perturbed trials, as shown in Fig. 7. Across subjects, the mean resistance during the time (214–357 ms) following the start of perturbation (during the 4th and 5th half-cycles of the 7 Hz perturbation) was significantly less than that during the first perturbation cycle (0–143 ms; 63 ± 12 % decrease, $\pm$ S.D.; $P < 0{\cdot}05$, $n = 5$ subjects). Although less obvious in Fig. 7, the phase lead of the torque response (relative to the imposed position) was at the same time reduced; this is quantified in a later section.

Stopped perturbation trials

This reduction in 7 Hz muscle resistance (stiffness) late in the movement was hypothesized to result from delayed

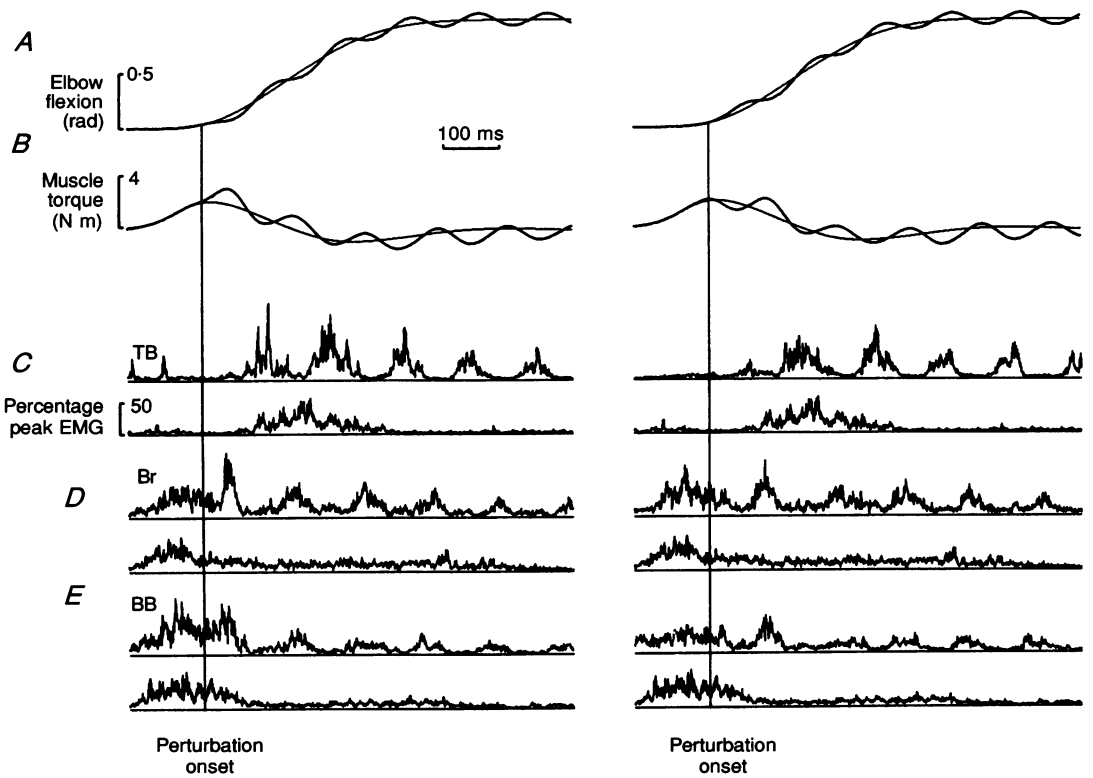

Figure 4. Non-linear bias in sinusoidal response
High frequency (8 Hz) sinusoidal position perturbations imposed during voluntary elbow flexion. Same format and subject as Fig. 2. Peak velocity 3·6 rad s^{-1}. Note that the modulation of the normal (unperturbed) EMG activity is asymetrical; the TB muscle (antagonist) did not respond to the first half-cycle of the imposed stretch, whereas the agonist muscles did.

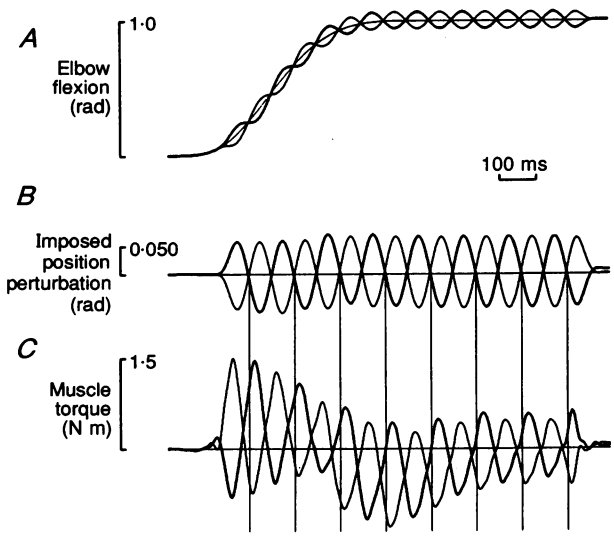

Figure 5. Non-linear bias; resistance to perturbations shown in Fig. 4
Same format as Fig. 3. Note that the mean muscle torque resistance is biased in the agonist (flexion positive) direction early in the movement, and the antagonist direction later.

reflex responses occurring half an oscillation cycle late, and thus aiding the torque motor rather than resisting it (see Introduction). This is consistent with the above observation that the reflex activity was particularly strong late in the movement, compared with an earlier stage when the antagonist response was usually absent (Soechting *et al.* 1931; Bennett, 1993*b*).

One method of testing this hypothesis is to terminate abruptly the imposed 7 Hz sinusoidal perturbance and hold the arm on the unperturbed trajectory. The delayed action of reflexes should then produce a torque that persists for a half-cycle after the termination of the mechanical disturbance. Indeed, as seen in Fig. 8, this did occur. During the trial corresponding to the thick line in Fig. 8, the agonist muscle was stretched (relative to its normal trajectory, negative displacement) by two complete cycles of the sinusoid only,

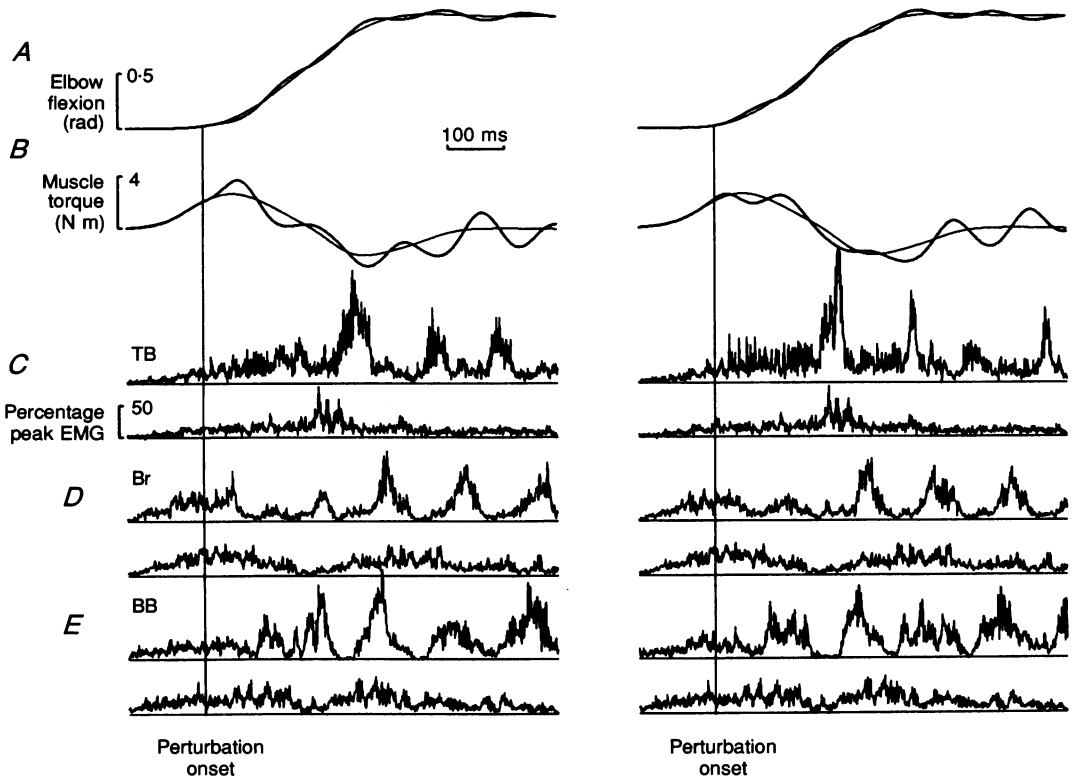

Figure 6. High frequency (7 Hz) sinusoidal stretches imposed during flexion movement
Same format as Fig. 2. Peak velocity 4·4 rad s^{-1}. Subject S4; inertia = 0·075 kg m^2. Note that the TB muscle (antagonist) does not respond to the first half-cycle of the imposed stretch, and then responds quickly during the period of the normal (unperturbed) antagonist burst.

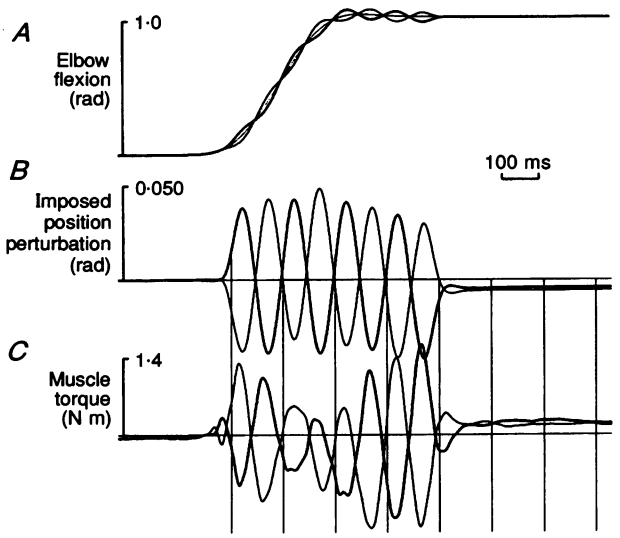

Figure 7. Resistance to high frequency perturbations shown in Fig. 6
Same format as Fig. 3. Note that the resistance to the perturbation decreased as the arm approached the target (note envelope of the response).

but the muscle torque response (positive torque) persisted for an additional 100 ms after the end of this stretching (downward-pointing arrow). The thin line in Fig. 8 shows the torque resisting the motor when the perturbation was continued for a third cycle. This resistance should be negative at the upward-pointing arrow, but note that, as in Fig. 7, it is greatly attenuated. It is suggested that this torque attenuation resulted from a delayed reflex torque, similar to that indicated by the thick line at the downward arrow, cancelling out the intrinsic resistance of the muscles. See Discussion.

Time-varying stiffness estimation

To further quantify the resistance to the perturbations, the elastic and viscous stiffness (K and $B\omega$; or equivalently response magnitude and phase) were estimated at 40 ms intervals during the movement, as described in Methods. Figure 9 shows a typical response computed from an ensemble of 100 perturbed movements similar to those shown in Fig. 6, but with a random onset phase. As anticipated from Fig. 7, both the elastic and the viscous stiffness decreased near the target, indicating, of course, that the magnitude of the net muscle resistance decreased.

An unexpected outcome was that, on average, the viscous stiffness (and thus the muscle torque phase lead) became negative at the time when the arm reached the target (see viscous stiffness computed 280 ms after the perturbation onset in Fig. 9). This negative viscosity occurred and was significant in four of the five subjects ($P < 0{\cdot}05$, $n = 100$). Rack *et al.* (1974) and Matthews & Watson (1981) also found negative viscosity in the elbow joint, although only during vigorous isometric contractions of about 80 N applied at the wrist. In the present study the peak background (unperturbed) muscle torque was only 2·5 N m (Fig. 2), or about 10 N applied at the wrist.

Parameter variances were computed at each point in time, and were generally found to be constant and small (Fig. 9). The parameter variance increased as the frequency of perturbation increased, since the torque required to accelerate the limb inertia increased proportionally to the

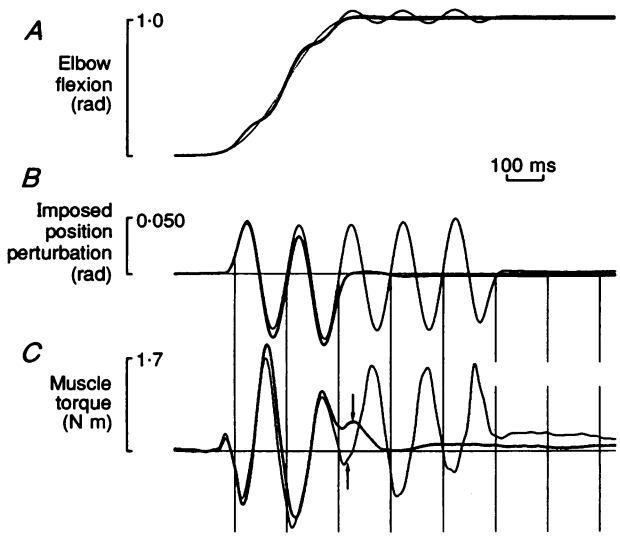

Figure 8. Stopped perturbation trials
Thin and medium lines, resistance to high frequency perturbations in a format similar to that of Fig. 7. Thick lines, same experiment, but perturbation was stopped after two cycles of oscillation, after which the servomotor held the arm on the unperturbed trajectory (i.e. at the target). Subject S1; inertia = 0·09 kg m^2. Note that, as seen in Fig. 7, the resistance to the perturbation decreased by the 5th half-cycle to nearly zero at the single upward-pointing arrow. This resistance decrease probably resulted from delayed inappropriate reflex activity, since in the trial where the perturbation was stopped, a torque response persisted for an extra half-cycle and was in a direction that would have aided the torque motor, rather than resisted it at the time indicated by the downward arrow.

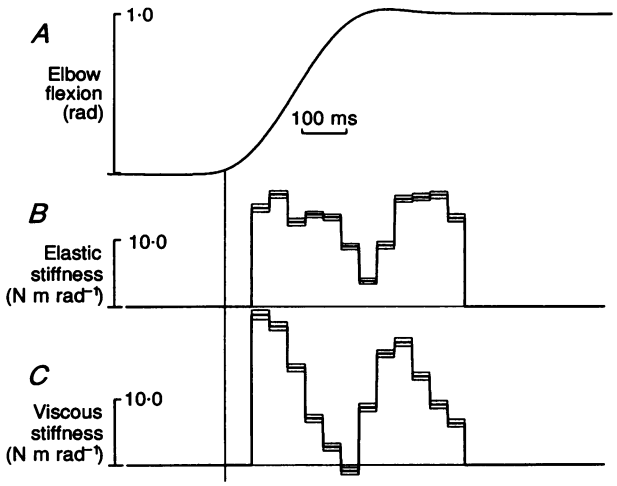

Figure 9. Time-varying stiffness
Stiffness estimated at 40 ms time intervals from an ensemble of 100 movements perturbed with a 7 Hz perturbation with a random onset phase. Perturbation amplitude 0·05 rad. *A*, elbow flexion trajectory (mean perturbed and unperturbed trajectories identical); *B*, elastic stiffness. *C*, viscous stiffness resisting the perturbation. Confidence intervals (CI) shown by thin lines ($P < 0·05$; $n = 100$; S.D. = 5 CI). Subject S1; inertia = 0·09 kg m^2. Note that the viscous stiffness dropped significantly below zero in the interval at 280 ms latency relative to the vertical line.

frequency squared, with corresponding decreases in the signal-to-noise ratio in the estimated net muscle torque (see Fig. 1). We have thus not studied perturbations above 8 Hz. The validity of the method was assessed by predicting the applied motor torque from the estimated parameters and comparing it with the actual applied torque. Correlation coefficients between these two variables were always greater than 0·9. Note that to avoid amplitude-dependent non-linearities (Rack & Westbury, 1974), the amplitude of perturbation was kept constant (constant input variance). Also, the randomization of the onset phase of the sinusoid ensured that the applied perturbations had a zero ensemble mean value (upper panel in Fig. 9).

Nyquist diagrams

To determine the 'critical frequency' range over which the

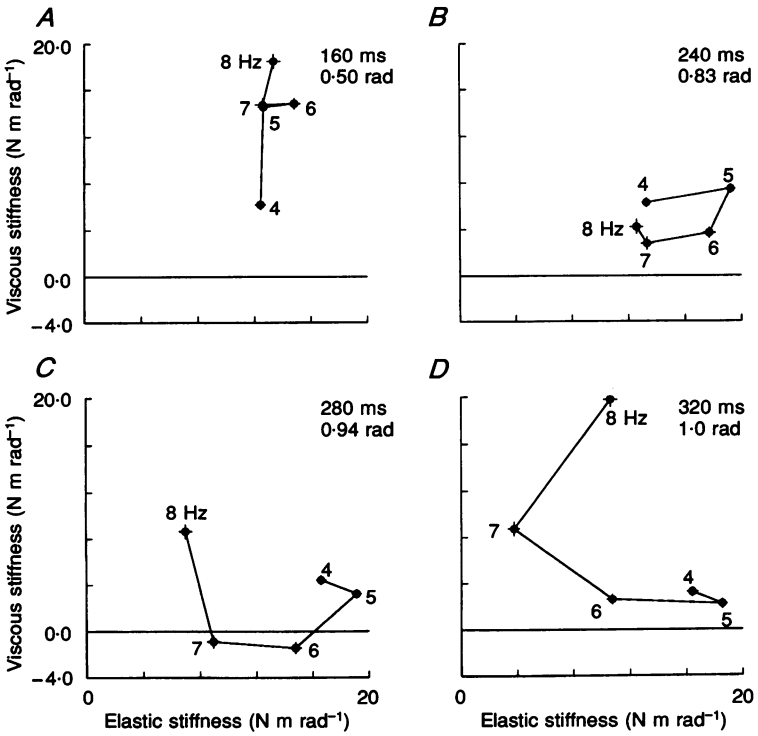

Figure 10. Nyquist plots of elbow joint stiffness at different times during a movement
Plots of elastic stiffness against viscous stiffness, as a function of the frequency of perturbation (perturbation amplitude: 0·05 rad). Frequency of perturbation indicated by each point; latency relative to the perturbation onset and angular position of stiffness estimate indicated in the upper right corner. Estimates for 7 Hz perturbation came from Fig. 9. Confidence intervals (CI) crosses ($P < 0·05$; $n = 100$; S.D. = 5 CI). Subject S1; inertia = 0·09 kg m^2. Note the large C-shaped curve that developed with time (*B–D*) and the significant reduction in the viscous stiffness to below zero in *C*.

viscosity was a minimum the experiments in the previous section were repeated at frequencies ranging from 4 to 8 Hz. Since a session at one frequency (e.g. Fig. 9) took more than 1 h, and since it was necessary for calibration purposes to keep the arm clamped onto the apparatus the whole time, this whole procedure took 5–8 h. Fatigue did not seem to influence the results, as repeated sessions at the same frequency gave similar results (a 3 s rest was taken at the end of each movement, and numerous 10 min rest periods were allowed to release pressure from the wrist cuff). Retesting on three separate days in one subject gave qualitatively similar results.

Figure 10 summarizes the typical results at representative phases during the movement. The response from one time instant and for one frequency of perturbation is depicted by a point in a plot of 'in-phase' resistance to the sinusoidal perturbation (elastic stiffness) *vs.* 'out-of-phase' resistance (viscous stiffness). A curve was drawn connecting points of increasing frequency (Nyquist plot). The format of these plots (sign convention) is identical to that used by Brown *et al.* (1982), although the inertial contribution is subtracted (see Methods). At short latencies relative to the perturbation onset time (marked by the vertical line in Fig. 9) the plots have no distinct shape, with the different frequency points clustering together (see Fig. 10*A*). However, at increasing latencies a characteristic C-shape is seen, which grows to a maximum size at 320 ms latency (Fig. 10*D*; i.e. at the end of the movement). At 280 ms latency the C-shaped plot dips below the zero viscosity axis (Fig. 10*C*; as also shown in Fig. 9). This negative (minimum) viscosity occurred in a range of 6–7 Hz, which is thus the 'critical frequency' range.

In Fig. 10 only responses beginning at 160 ms latency are shown. For the low frequencies (e.g. 4 Hz) responses occurred earlier than this and were corrupted by the onset of the perturbation. This is because the perturbation occurred with random phase relative to the onset time. The motor was not started until the time of the first zero crossing (to avoid a high frequency transient; see Methods). Thus, within the first half-cycle duration (125 ms for the 4 Hz perturbation) there were trials where the motor did not come on. Within this time the parameter variances were high, and have thus been ignored. For the same reasons the stiffness and viscosity parameters for the lower frequency perturbations, such as the 2 Hz perturbation shown in Fig. 2, were not computed.

As explained in the Discussion section, the size of the C-shaped response roughly corresponds to the magnitude of the reflex response. To quantify this, the diameter of a circle fitted to the C-shaped curve was used. The circle was centred at the centroid of all of the points in a given Nyquist plot. Such a computation gave diameters ranging from 8 to 20 N m rad^{-1}, with the largest values at 280–320 ms latency relative to the perturbation onset time (Fig. 10*C* and *D*).

DISCUSSION

Evidence has been presented which indicates that at times during a movement the reflex activity substantially increases or decreases the resistance to a perturbation (stiffness), depending on the frequency of perturbation. The modulations of the normal voluntary EMG activity produced by the perturbations are consistent with this resistive action of proprioceptive feedback (see also Gottlieb & Agarwal, 1979; Soechting *et al.* 1981). However, it is difficult to infer net torque from EMG activity, given the non-linear velocity dependence of muscle (Hill, 1938), its low-pass properties (Bawa & Stein, 1976) and its activation history dependence (Aldridge & Stein, 1982). Thus, attention is focused instead on the mechanical responses, which give a more direct measure of muscle action.

The present results confirmed that the low frequency resistance to the perturbation increased as the arm approached the target (see also Bennett *et al.* 1992; Bennett, 1993*a*). As this increase also coincided with the increase in the normal torque decelerating the limb, it is likely that part of this resistance increase was due to an increased intrinsic (non-reflexive) stiffness of muscle (e.g. Joyce & Rack, 1969). This finding alone is not conclusive as to the role of reflexes. However, it was also found that the resistance to higher frequency (6–7 Hz) perturbations *decreased* at the same time that the low frequency resistance increased (i.e. when the arm approached the target). This paradoxical effect can be explained by delayed reflexes acting as described below, but not by intrinsic muscle frequency response properties (e.g. Rack, 1966), nor by non-linearities (see discussion of non-linear bias under 'Reflex asymmetry' in Results). Consider the delay (and thus the phase lag) in peak force developed from reflex activity at 7 Hz. In order of operation, the reflex delays (given in parentheses) are produced by: (i) the muscle spindle response properties (110 deg lead, 43 ms, for Ia spindle afferents; Matthews & Stein, 1969; Poppele & Bowman, 1970); (ii) the neuronal transmission delay (50 deg for a 20 ms delay); (iii) the excitation–contraction delay (43 deg for a 15 ms delay; Bawa & Stein, 1976) and (iv) the low-pass muscle activation–force characteristic (110 deg lag or 43 ms delay; Jacks *et al.* 1988). To check these numbers note that the combined spindle phase advance and transmission delay gives a 60 deg lead, meaning the EMG response should be better correlated with velocity (90 deg lead) than position, as seen in Figs 4 and 6. Note also that the force should develop with a 148 deg (38 + 110 deg) lag with respect to the EMG. This agrees well with the 60 ms delay (i.e. 151 deg at 7 Hz) that can be seen between the peak agonist EMG and the peak torque accelerating the limb on an unperturbed trials (e.g. Fig. 4; also see Bennett, 1993*b*). In total, the reflex force component lags behind the position perturbation by 88 deg at 7 Hz (35 ms), and lags behind the velocity by 178 deg (70 ms).

Thus, at 7 Hz the force developed by the stretch reflex acts approximately one half-cycle after the imposed stretch velocity (180 deg out of phase) and subtracts from, rather than adds to, the intrinsic viscous resistance of the muscles. Longer transmission delays than the 20 ms assumed above produce greater phase lags in force production and thus also reduce the elastic stiffness of the muscle. Finally, note that, as pointed out by Prochazka & Trend (1988), at higher levels of muscle activation more fast-twitch muscle fibres are recruited; this reduces the phase lag. This perhaps explains

why, contrary to the present results, Joyce *et al.* (1974) found negative viscosity at frequencies well above 7 Hz (i.e. at 8–12 Hz), since they studied isometric contractions at over 50 % of maximum.

The results from the stopped perturbation experiments (Fig. 8) provide direct evidence for the above interpretation of the role of reflexes in reducing the 6–7 Hz stiffness. When the imposed sinusoidal perturbance was abruptly stopped, there was a muscle torque that persisted for 100 ms. The peak of this torque occurred at about a 180 deg phase lag (one half-cycle delay) relative to the last cycle of imposed stretch, and this torque was thus in a direction to aid, rather than resist, the torque motor, had the perturbation continued. This delayed torque response had a broad shape similar to that of a single muscle-twitch or tendon-jerk response (Bawa & Stein, 1976; Aldridge & Stein, 1982). We thus suppose that it arose from reflex activity and indicates the reflex torque that would have been produced had the perturbation continued (as also suggested by the large EMG responses to stretch, Fig. 6).

Stretch reflex stiffness

Analysis of elbow stiffness as a function of time and frequency (Nyquist plots) provides further evidence that the reflexes caused the reduction in stiffness seen during the 6–7 Hz perturbations, and also shows that at times the reflexes contributed substantially to total muscle stiffness. To proceed with Nyquist analysis it is necessary to assume that total muscle stiffness is a linear sum of intrinsic and reflex stiffness, which is reasonable considering that the perturbation amplitude is small. A vector representing intrinsic stiffness is drawn in Fig. 11, based on the assumption that it is close to the total stiffness at high frequencies (Brown *et al.* 1982). (Recall that the stiffness magnitude and phase are represented by the length and angle of the vector.) A similar vector representing the reflex component is added to the intrinsic vector to give the total measured stiffness. Due to reflex delays, this reflex component has an increasing phase lag for increasing frequency, giving a C-shaped curve (Fig. 11). Note that, as described above, at 7 Hz the reflex phase lag is so large that the reflex stiffness vector points downwards, reducing the total stiffness. This reduction is sufficiently large that the total stiffness dips below the horizontal axis (Fig. 11); that is, viscous stiffness due to reflexes is negative and exceeds the viscous stiffness due to intrinsic muscle properties.

As the reflex stiffness component gives a C-shaped Nyquist curve, the size of this curve gives a measure of the reflex response, without exact knowledge of the intrinsic muscle stiffness (Brown *et al.* 1982). The average reflex stiffness was therefore estimated as the size, or 'diameter', of the C-shaped curve (i.e. the best-fit diameter; see last paragraph of Results). These diameters increased as the target was approached. The diameter may be interpreted as an upper limit on the reflexive component to the low frequency (4–6 Hz) stiffness (see Fig. 11), since from Brown *et al.* (1982) the total low frequency stiffness and the intrinsic muscle stiffness are typically on opposite sides of the C-shaped Nyquist curve, and, as mentioned before, the difference between these two stiffness components is the reflex stiffness. This upper limit on low frequency stiffness is 12 N m rad^{-1} for Fig. 11 when the arm is near the target. While this estimate is only approximate, it suggests that the reflex component to the total stiffness can be substantial. Furthermore, if the intrinsic muscle stiffness magnitude is similar to the stiffness measured at 8 Hz in Fig. 11 (which is reasonable, based on results such as those of Brown *et al.* 1982), then the reflex and intrinsic stiffness components are of comparable sizes. Precise quantification of the reflex gain cannot be carried out without direct estimates of the intrinsic muscle stiffness.

Mechanisms of reflex modulation during movement

There are a number of possible explanations for the observed modulation of the stretch reflex during movement, apart from direct presynaptic modulation of the reflex pathways (Capaday & Stein, 1987; also see review of Schieppati, 1987). Reflex responsiveness changes with the level of background EMG, which changes continuously with the normal triphasic muscle activation. The afferent feedback usually has a greater chance of recruiting more motoneurons when the background motoneuron pool activity is greater (e.g. Matthews, 1986). Also, increased muscle activity and tension is likely to allow the muscle spindle afferents to be more securely driven by the sinusoidal vibration, given (i) increased tendon stiffness

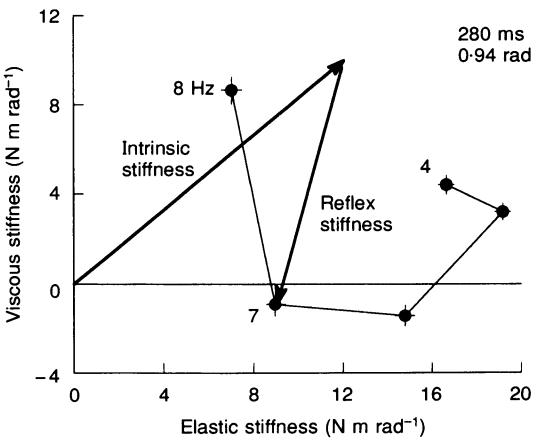

Figure 11. Nyquist plot of joint stiffness just prior to reaching the target during movement
Redrawn from Fig. 10. Hypothetical construction to demonstrate how at 7 Hz the reflex component to stiffness (reflex stiffness) can reduce the intrinsic stiffness (non-reflex) of the muscles. Intrinsic stiffness vector estimated arbitrarily but close to the highest frequency stiffness (Brown *et al.* 1982). The reflex stiffness vector is the difference between total stiffness and intrinsic stiffness (7 Hz case drawn). As a result of reflex transmission delays, when the frequency increases the reflex stiffness vector must rotate in a clockwise direction and thus its tip gives a C-shaped curve. Note that regardless of the location of the intrinsic stiffness estimate in the upper half-plane, the 7 Hz reflex stiffness must point downwards (as shown) since the total viscous stiffness is negative.

(non-linear tendon stiffness, Rack & Ross, 1984) and (ii) increased fusimotor drive (Burke, Hagbarth & Lofstedt, 1978). As there is likely be to be substantial co-contraction of flexor and extensor muscle activity and tension late in the movement (Bennett, 1993*b*), the above mechanisms contribute to the observed reflex responses that were on average higher late in the movement. These mechanisms also explain the phase-dependent bias in the torque responses (see 'Reflex asymmetry' in Results section).

As a consequence of the imposed sinusoidal muscle vibration, the mean contraction of both flexor and extensor muscles may increase as the movement (and vibration) progresses, particularly at vibration rates above the low-pass corner frequency of the muscle activation–force curve, 5 Hz for the elbow (Jacks *et al.* 1988). This vibration-induced co-contraction increases the intrinsic muscle stiffness, leading to an underestimation of the role of reflexes.

Finally, it is essential to rule out the possibility that the reflex modulation is simply an artifact of the measurement technique. Specifically, one might argue that since the sinusoidal vibration is turned on at the onset of the movement the reflex gain and negative viscosity might be higher than that in 'steady state' simply as a consequence of the onset of the vibration. The conclusions of reflex gain modulation during movement might therefore depend on a fortuitous choice of the duration of the movement. This appeared not to be the case, however, since no evidence was found for fluctuations in the stiffness estimates taken while subjects held a steady contraction. In the process of estimating the inertia of the forearm (see Methods), the time-varying stiffness (and viscosity) were routinely estimated with the subject holding a constant contraction level. These stiffness estimates were found simply to increase within 160 ms and then remain at a steady-state level.

Functional implications

Although at times the reflexes produced negative viscous stiffness, this does not necessarily predict that the system should become unstable, or oscillate in a limit cycle as found by Joyce & Rack (1974). Indeed, the arm did not oscillate during unperturbed movements. Two mechanisms restricted instability. First, the reflex gain was raised only transiently at the target, and so unstable oscillations had no time to develop. Second, the mechanical resonant frequency of the arm was far below the 'critical frequency' range of 6–7 Hz where the viscosity dropped below zero. The maximum stiffness obtained at any frequency was 25 N m rad^{-1} (see also Bennett, 1993*a*). Thus, the worst case (largest) resonant frequency was 2·7 Hz (using an inertia of 0·09 kg m^2). Resistance to perturbations above this frequency was dominated by inertia (see also Fig. 1), and thus self-sustained oscillations were impossible.

Limited duration oscillations of the forearm may occur if the mechanical resonant frequency of the arm is increased, by increasing the total stiffness. A sufficient stiffness increase might be achieved by having the subject operate against a stiff spring (200 N m rad^{-1}) near the end of a movement. Alternatively, a large constant force load would increase the intrinsic muscle stiffness, and thus may increase the resonant frequency sufficiently to cause transient oscillations at the end of a voluntary movement, as indicated by the isometric results of Joyce & Rack (1974).

REFERENCES

Aldridge, J. W. & Stein, R. B. (1982). Nonlinear properties of stretch reflex studied in decerebrate cat. *Journal of Neurophysiology* **47**, 179–192.

Bawa, P. & Stein, R. B. (1976). Frequency response of human soleus muscle. *Journal of Neurophysiology* **39**, 788–793.

Bennett, D. J. (1991). Relationship between stiffness and net joint torque during ballistic elbow joint movement. *Society for Neuroscience Abstracts* **17**, 1029.

Bennett, D. J. (1993*a*). Torques generated at the human elbow joint in response to constant position errors imposed during voluntary movement. *Experimental Brain Research* **95**, 488–498.

Bennett, D. J. (1993*b*). Electromyographic responses to constant position errors imposed during voluntary elbow joint movement in human. *Experimental Brain Research* **95**, 499–508.

Bennett, D. J., Hollerbach, J. M., Xu, Y. & Hunter, I. W. (1992). Time-varying stiffness of human elbow joint during cyclic voluntary movement. *Experimental Brain Research* **88**, 433–442.

Brown, T. I. H., Rack, P. M. H. & Ross, H. F. (1982). Forces generated at the thumb interphalangeal joint during imposed sinusoidal movements. *Journal of Physiology* **332**, 63–85.

Burke, D., Hagbarth, K.-E. & Lofstedt, L. (1978). Muscle spindle activity in man during shortening and lengthening contractions. *Journal of Physiology* **277**, 131–142.

Capaday, C. & Stein, R. B. (1987). Difference in the amplitude of human soleus H reflex during walking and running. *Journal of Physiology* **392**, 513–522.

Carter, R. R., Crago, P. E. & Keith, M. W. (1990). Stiffness regulation by reflex action in the normal human hand. *Journal of Neurophysiology* **64**, 105–118.

Evans, C. M., Fellows, S. J., Rack, P. M. H., Ross, H. F. & Walters, D. K. W. (1983). Response of the normal human ankle joint to imposed sinusoidal movements. *Journal of Physiology* **344**, 483–502.

Gottlieb, G. L. & Agarwal, G. C. (1979). Response to sudden torques about ankle in man: myotatic reflex. *Journal of Neurophysiology* **42**, 91–106.

Gottlieb, G. L., Agarwal, G. C. & Stark, L. (1970). Interactions between voluntary and postural mechanisms of the human motor system. *Journal of Neurophysiology* **33**, 365–381.

Hill, A. V. (1938). The heat of shortening and the dynamic constants of muscle. *Proceedings of the Royal Society* B **126**, 136–195.

Hoffer, J. A. & Andreassen, S. (1981). Regulation of soleus muscle stiffness in premammillary cat: intrinsic and reflexive components. *Journal of Neurophysiology* **45**, 267–285.

Jacks, A. J., Prochazka, A. & Trend, P. St. J. (1988). Instability in human forearm movements studied with feed-back-controlled electrical stimulation of muscles. *Journal of Physiology* **402**, 443–461.

Joyce, G. C. & Rack, P. M. H. (1969). Isotonic lengthening and shortening movements of cat soleus muscle. *Journal of Physiology* **204**, 475–491.

Joyce, G. C. & Rack, P. M. H. (1974). The effects of load and force on tremor at the normal human elbow joint. *Journal of Physiology* **240**, 375–396.

Joyce, G. C., Rack, P. M. H. & Ross, H. F. (1974). The forces generated at the human elbow joint in response to imposed sinusoidal movements of the forearm. *Journal of Physiology* **240**, 351–374.

JOYCE, G. C., RACK, P. M. H. & WESTBURY, D. R. (1969). The mechanical properties of cat soleus muscle during controlled lengthening and shortening movements. *Journal of Physiology* **204**, 461–474.

LLEWELLYN, M., YANG, J. F. & PROCHAZKA, A. (1990). Human H-reflexes are smaller in difficult beam walking than in normal treadmill walking. *Experimental Brain Research* **83**, 22–28.

MATTHEWS, P. B. C. (1986). Observations on the automatic compensation of reflex gain on varying the pre-existing level of motor discharge in man. *Journal of Physiology* **374**, 73–90.

MATTHEWS, P. B. C. & STEIN, R. B. (1969). The sensitivity of the muscle spindle afferents to small sinusoidal changes in length. *Journal of Physiology* **200**, 723–743.

MATTHEWS, P. B. C. & WATSON, J. D. G. (1981). Effect of vibrating agonist or antagonist muscle on the reflex response to sinusoidal displacement of the human forearm. *Journal of Physiology* **321**, 297–316.

NICHOLS, T. R. & HOUK, J. C. (1976). Improvement in linearity and regulation of stiffness that results from actions of stretch reflex. *Journal of Neurophysiology* **39**, 119–142.

NORTON, J. P. (1986). *An Introduction to Identification.* Academic Press, Orlando, FL, USA.

POPPELE, R. E. & BOWMAN, R. J. (1970). Quantitative description of linear behaviour of mammalian muscle spindles. *Journal of Neurophysiology* **33**, 5972.

PROCHAZKA, A. & TREND, P. ST. J. (1988). Instability in human forearm movements studied with feed-back-controlled muscle vibration. *Journal of Physiology* **402**, 421–442.

RACK, P. M. H. (1966). The behaviour of a mammalian muscle during sinusoidal stretching. *Journal of Physiology* **183**, 1–14.

RACK, P. M. H. & ROSS, H. F. (1984). The tendon of flexor pollicis longus: its effects on the muscular control of force and position at the human thumb. *Journal of Physiology* **351**, 99–110.

RACK, P. M. H. & WESTBURY, D. R. (1974). The short range stiffness of active mammalian muscle and its effect on mechanical properties. *Journal of Physiology* **240**, 331–350.

SCHIEPPATI, M. (1987). The Hoffman reflex: a means of assessing spinal reflex excitability and its descending control in man. *Progress in Neurobiology* **28**, 345–376.

SINKJAER, T., TOFT, E., ANDREASSEN, S. & HORNEMANN, B. C. (1988). Muscle stiffness in human ankle dorsiflexors: intrinsic and reflex components. *Journal of Neurophysiology* **60**, 1110–1121.

SOECHTING, J. F., DUFRESNE, J. R. & LACQUANITI, F. (1981). Time-varying properties of the myotatic response in man during some simple motor tasks. *Journal of Neurophysiology* **46**, 1226–1243.

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