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PROPRIOCEPTIVE GUIDANCE OF HUMAN VOLUNTARY WRIST MOVEMENTS STUDIED USING MUSCLE VIBRATION

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SUMMARY

1. The alterations in voluntary wrist extension and flexion movement trajectories induced by application of vibration to the tendon of flexor carpi radialis throughout the course of the movement, together with the associated EMG patterns, have been studied in normal human subjects. Both extension and flexion movements were routinely of a target amplitude of 30 deg and made against a torque load of 0·32 N m. Flexor tendon vibration consistently produced undershooting of voluntary extension movements. In contrast, voluntary flexion movements were relatively unaffected.

2. The degree of vibration-induced undershooting of 1 s voluntary extension movements was graded according to the amplitude (0·75, 1·0 and 1·5 mm) of flexor tendon vibration.

3. As flexor vibration was initiated progressively later (at greater angular thresholds) during the course of 1 s voluntary extension movements, and the period of vibration was proportionately reduced, so the degree of vibration-induced undershooting showed a corresponding decline.

4. Varying the torque loads (0·32, 0·65 and 0·97 N m) against which 1 s extension movements were made, and thereby the strength of voluntary extensor contraction, produced no systematic changes in the degree of flexor vibration-induced undershooting.

5. Analysis of EMG patterns recorded from wrist flexor and extensor muscles indicated that vibration-induced undershooting of extension movements resulted largely from a reduction in activity in the prime-mover rather than increased antagonist activity. The earliest reductions in extensor EMG commenced some 40 ms after the onset of vibration, i.e. well before voluntary reaction time; these initial responses were considered to be 'automatic' in nature.

6. These results support the view that the central nervous system utilizes proprioceptive information in the continuous regulation of moderately slow voluntary wrist movements. Proprioceptive sensory input from the passively lengthening antagonist muscle, presumably arising mainly from muscle spindle Ia

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afferents, appears to be particularly important and to act mainly in the reciprocal control of the prime-mover.

INTRODUCTION

Our ability to produce accurate, purposive voluntary limb movements is thought to depend critically upon information about the progress of the task being fed continuously to the central nervous system (CNS). If the CNS is denied such proprioceptive information, e.g. by surgical or pathological deafferentation of a limb (Mott & Sherrington, 1895; Foerster, 1927; Rothwell, Traub, Day, Obeso, Thomas & Marsden, 1982) marked impairment of motor performance ensues. Conversely, artificial experimental activation of proprioceptors during the course of voluntary movements, a means of 'misinforming' the CNS as to the actual kinematics, also produces errors. Application of vibration, which is a powerful stimulant of muscle receptors and particularly of spindle Ia afferents (Burke, Hagbarth, Lofstedt & Wallin, 1976*a*, *b*; Roll & Vedel, 1982), can generate kinaesthetic illusions (Goodwin, McCloskey & Matthews, 1972*a*, *b*, *c*; Gilhodes, Roll & Tardy-Gervet, 1986) and can modify the trajectories of voluntary movements (Capaday & Cooke, 1981, 1983; Lackner, 1984; Appenteng & Prochazka, 1983; Sittig, Dernier van der Gon & Gielen, 1985, 1987; Bullen & Brunt, 1986).

In a number of studies of goal-directed elbow movements, an interesting general finding has been that vibration of the antagonist, i.e. passively lengthening muscle, produces an undershooting of the intended target position whereas stimulation of the actively shortening agonist has relatively little effect upon accuracy (Capaday & Cooke, 1981, 1983; Bullen & Brunt, 1986; Sittig *et al.* 1987). As noted by Capaday & Cooke (1981, 1983), this suggests that muscle receptors in the antagonist are a particularly important source of proprioceptive input during normal movements. Presumably, the CNS interprets the vibration-induced enhancement of antagonist spindle firing as representing an erroneously rapid rate of stretch (and movement) and initiates compensatory changes in motor activity which result in an abbreviated trajectory.

The central neural mechanisms responsible for vibration-induced movement errors remain unclear. Capaday & Cooke (1983) reported, however, that undershooting of rapid elbow movements arose principally from increases in motor discharge of the vibrated muscle (antagonist) rather than reductions in activity in the prime-mover. Furthermore, these authors proposed, on the basis of the fairly long latency (about 60 ms) of the EMG changes, that the responses were mediated, at least in part, by supraspinal centres.

The present experiments upon vibration-induced alterations of voluntary wrist movements and associated EMG patterns contribute in several respects to understanding the role played by proprioceptive input in guiding voluntary movements. In demonstrating that antagonist vibration elicits undershooting of target trajectories, they extend the generality of earlier findings for elbow movements to more distal muscle groups which are under a greater degree of cerebral cortical control. In addition, several quite new features were shown. First, the extent of undershooting was graded according to both the amplitude and the duration of stimulation. Such scaling of errors suggests the continuous operation of a selective

proprioceptive feedback loop. Second, comparable errors were found for movements requiring a range of agonist contraction strengths. This implies that sensory input can be utilized in regulating muscle length changes rather than force output. Third, undershooting of target trajectories of moderately slow (750–2000 ms) wrist movements resulted mainly from reductions of agonist activity commencing at a latency (*ca* 40 ms) well below voluntary reaction time. These findings support the operation of a proprioceptive guidance system which has a reciprocal organization and is, in part, 'automatic' in nature.

Some of the preliminary findings have been briefly reported (Cody, Schwartz & Smit, 1990).

METHODS

Subjects

The study was made on seventeen subjects (ten male, seven female) of mean age 32 years. Subjects were unaware of the theoretical issues guiding the investigation but experimental procedures were fully explained. All subjects participated in the experiments with their informed consent (Code of Ethics of the World Medical Association, Declaration of Helsinki) and protocols were approved by the Local Ethical Committee. None of the subjects had any history of neurological disease.

Experimental arrangements

Subjects were seated in a chair and grasped a vertical manipulandum handle with their right hand. The arm pointed forwards and was semi-pronated with the forearm resting on a horizontal support and clamped at its distal end by padded bars. Angular extension or flexion movements of the wrist, in the horizontal plane, were made against several pre-existing torque loads (0·32, 0·65 and 0·97 N m). The manipulandum handle was attached to a shaft pivoted directly below the wrist joint and linked by low-friction bearings to a pulley system from which pre-selected weights were suspended. Angular displacement and tension records were, respectively, provided by a precision potentiometer and a force transducer which were incorporated in the manipulandum.

Voluntary extension and flexion wrist movements of target amplitudes of 30 deg were studied. Extension movements started from a position in which the carpus was orientated in line with the forearm; flexion movements started from a position of 30 deg extension, i.e. the final position of extension movements. Subjects were required to make movements of different speeds and against different loads. Prior to experimental trials, subjects practised the required movements until a reasonable degree of reproducibility was attained. These movements were made with the non-actuated vibrator probe in place over the tendon of flexor carpi radialis (see below). An oscilloscope display of wrist angle together with desired initial and final positions was provided. During the practice period (approximately 5 min), subjects were given both auditory and visual cues of required movement time. A loudspeaker produced three clicks, at equal intervals, to signal 'get ready', 'start' and 'stop'. The oscilloscope sweep, of selected speed, was initiated at the first click. At the second click the beam reached the end of the initial level marker and subjects were required to start the movement. At the third click the beam reached the beginning of the final level marker. Two pre-determined movement times were used, 1 and 2 s; in addition, subjects performed 'self-paced' movements, i.e. were instructed to move between the initial and final positions at their most comfortable rate. The interval between successive movements was 7 s; this was sufficient to allow subjects to complete the on-going movement and to easily return to the initial position before the next one. Subjects were not instructed to move between initial and final positions with any particular form of trajectory; most, however, appeared to attempt an approximately constant velocity movement.

During experimental trials in which the desired movement times were 1 and 2 s, the 'get ready', 'start' and 'stop' auditory cues were presented as before; for self-paced movements the 'stop' click was turned off. The original oscilloscope display of movement trajectory was extinguished. Instead, only a visual indicator of desired initial position was shown; this was suppressed upon commencement of movement. Thus, the amplitudes of the active voluntary movements depended

upon purely proprioceptive guidance. Subjects were required to reproduce as accurately as possible the movements previously practised with visual feedback. Voluntary movements were now made either in the absence (non-vibrated) or presence (vibrated) of flexor tendon vibration and subjects were instructed to 'try to make the same movement every time'.

Experimental protocols. Five main series of experiments were performed: (i) effects of flexor tendon vibration (1·0 mm) upon extension *vs.* flexion movements (1 s movement time) against a 0·32 N m load (nine subjects), (ii) effects of varying amplitudes of flexor tendon vibration (0·75, 1·0 and 1·5 mm) upon extension movements (1 s movement time) against a 0·32 N m load (six subjects), (iii) effects of flexor tendon vibration (1·0 mm) upon extension movements (1 s movement time) against varying loads (0·32, 0·65 and 0·97 N m) (six subjects), (iv) effects of flexor tendon vibration (1·0 mm) upon extension movements of varying movement times ('self paced', 1 and 2 s) (nine subjects) and (v) effects of flexor tendon vibration (1·0 mm) of varying angular onset threshold (0, 10 and 20 deg) upon extension movements (1 s movement time) (six subjects). In series (i)–(iv) tendon vibration was initiated at the start of the movement. For some series (ii, iii, v) data for non-vibrated and vibrated movements were collected in separate blocks of twenty trials whereas in others (i and iv) equal numbers of non-vibrated and vibrated movements were interspersed in a pseudo-random fashion. To test whether differences in vibration-induced alterations in movements existed between these two conditions, several subjects were studied using both interspersed and continuous blocks of vibrated movements with otherwise identical experimental parameters. Paired statistical comparison indicated that similar vibration-induced movement errors occurred irrespective of whether non-vibrated and vibrated movements were studied as separate blocks or were interspersed.

Vibration. High-frequency (125 Hz), sinusoidal mechanical stimuli were applied transcutaneously to the tendon of flexor carpi radialis using a small electromagnetic vibrator. The vibrator was suspended from a frame and counterweighted so that it pressed upon the tendon, approximately 6–8 cm proximal to the wrist crease, with a constant force (about 2·5 N). A 1 × 1·5 cm plastic head was attached to the moving element of the vibrator, with a groove running along its axis to assist contact with the tendon of flexor carpi radialis. Palpation over the bellies of several muscles in the flexor compartment indicated that flexor carpi radialis was by far the most powerfully stimulated although some spread of vibration inevitably occurred. The vibrator probe was left in position throughout the experiments but sinusoidal stimuli were only applied during 'vibrated movements'. The stimulus waveform was monitored by a length transducer incorporated in the vibrator. Each train of vibration commenced and terminated at the mid-point of a releasing stroke so that each stretching stroke, including the first and the last, was of full size. The peak–peak amplitude of vibration was varied between 0·75 and 1·5 mm.

The vibrator was activated when the movement trajectory reached a pre-determined angular threshold. In the majority of experiments this corresponded to the onset (0 deg) of the movement and vibration then continued for a period equivalent to the instructed movement time. Vibration period for 'self-paced' movements was adjusted to equal the duration of the non-vibrated movement made by individual subjects. In some experiments, with 1 s movement times, a range of different thresholds (usually 0, 10 and 20 deg of extension or flexion) for initiation of vibration and matching periods of vibration (1000, 667 and 333 ms) were employed.

Electromyography. Electromyograms were recorded from the extensor and flexor compartments of the forearm using two pairs of surface disc electrodes positioned respectively, over the bellies of extensor carpi ulnaris and flexor carpi radialis. The signals were amplified and bandpass filtered (20 Hz–3 kHz). Unrectified EMG signals, displacement waveforms, vibration waveforms and trigger pulses were stored on an 8-channel digital tape-recorder during experiments for off-line computer analysis.

Data analysis. Simultaneous averages (twenty trials) of wrist movement trajectory parameters (displacement, velocity or acceleration) and of extensor and flexor rectified electromyograms were made by an Olivetti M 240 computer, stored on hard disc and displayed on a X–Y plotter. Averaging was either done on-line or by replaying signals previously recorded on a digital tape-recorder. Averages were usually initiated by a pulse coincident with the 'get ready' auditory cue (see above). In the case of high resolution EMG averages computed to determine the latencies of vibration-induced changes in EMG patterns, averages were started by the pulse used to trigger the onset of vibration.

Quantitative measurements of the amplitude of movement trajectories were made as follows.

The actual starting time of the averaged movement was first determined and the corresponding wrist angle calculated. The wrist angle attained at the end of the instructed movement time (1 and 2 s movement times) was then calculated. The difference between these two angles was used as the measure of movement amplitude. In the case of 'self-paced' movements, the amplitude (difference

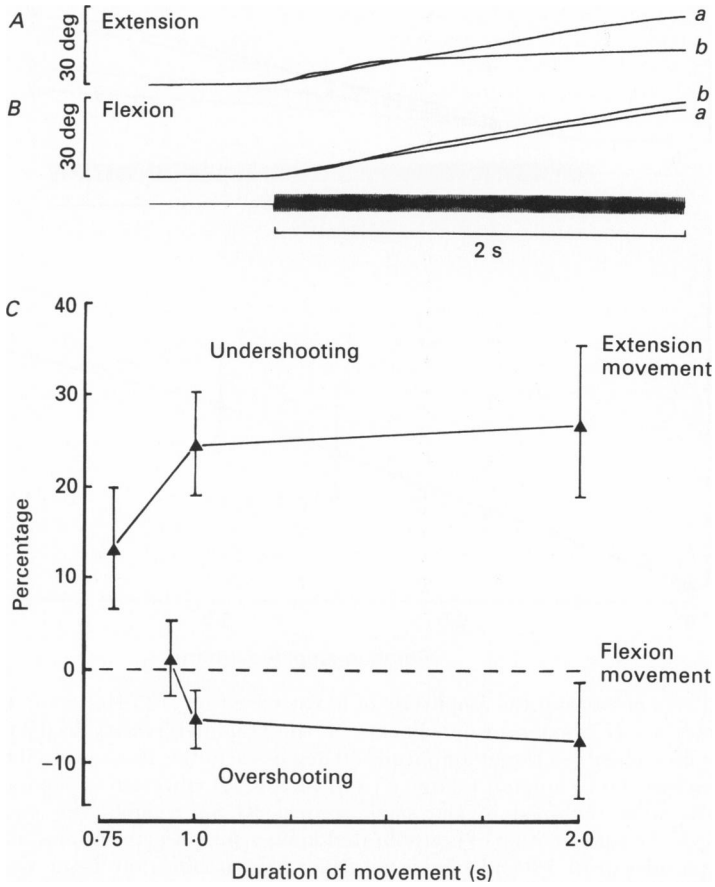

Fig. 1. Alterations in the trajectories of voluntary extension and flexion wrist movements by the application of vibration (1 mm, 125 Hz) to the tendon of flexor carpi radialis. *A* compares the averaged (twenty-one trials) trajectory of 2 s non-vibrated (*a*) extension movements of target amplitude 30 deg with that of vibrated movements (*b*). *B* compares non-vibrated (*a*) and vibrated (*b*) flexion movements. The records of *A* and *B* were obtained in the same subject (A.L.). *C*, plots of the mean (±S.E.M.) vibration-induced alterations of extension (upper) and flexion (lower) movements of three different movement times (self-paced, 1 and 2 s) recorded in nine subjects. Amplitudes of vibrated movements were expressed as percentages of corresponding non-vibrated ones. Self-paced movements varied in duration between subjects and for individual subjects between extension and flexion; mean durations are indicated. Movement errors, for both extension and flexion, were significantly greater for 1 s than for self-paced movements (paired *t* test, $P < 0{\cdot}04$).

between starting and maximal angular displacements) and duration (time from movement onset to attainment of maximal angular displacement) of the averaged non-vibrated movement were established. Measurement of the trajectory of the corresponding vibrated movement was made over an equivalent movement time.

Statistics. Conventional parametric tests were used for the calculation of mean values and their standard errors. Paired statistical comparisons of movement trajectory data from non-vibrated and vibrated trials were made using the parametric paired t test and the non-parametric Wilcoxon's matched-pairs signed-rank test. Correlation between movement errors and the

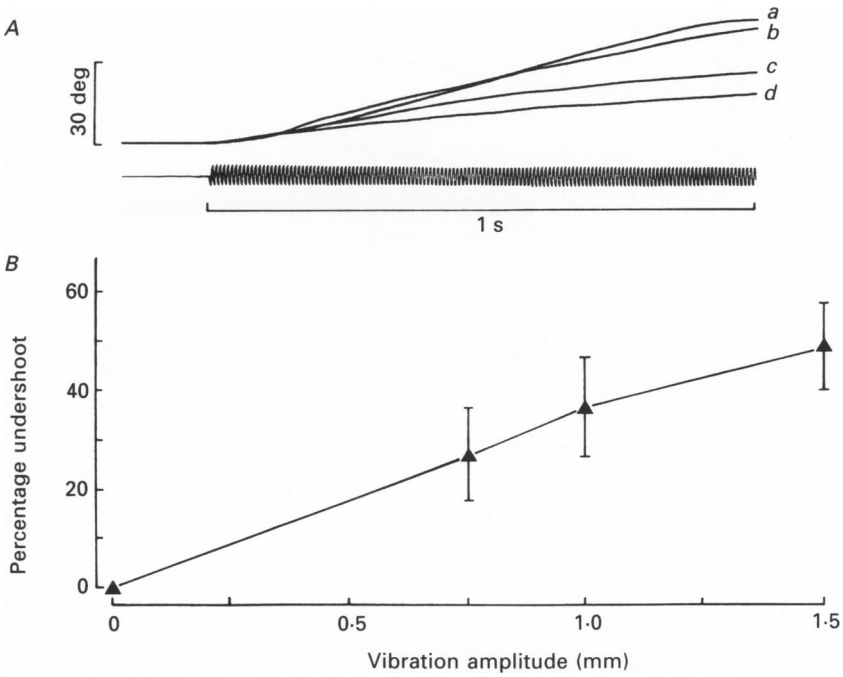

Fig. 2. Effects of varying the amplitude of flexor vibration (125 Hz) upon undershooting of voluntary 1 s wrist extensor movements. *A*, the averaged (twenty trials) trajectories of extension movements of target amplitude 30 deg made in the absence of vibration (*a*) and in the presence of 0·75 mm (*b*), 1·0 mm (*c*) and 1·5 mm (*d*) vibration of flexor carpi radialis. All records were obtained in the same subject (G.S.) *B* plots the mean (±S.E.M.) percentages of undershooting of non-vibrated movement trajectories produced by each of three amplitudes (0·75, 1·0 and 1·5 mm) of flexor tendon vibration. Data were obtained in six subjects. Percentage undershooting was significantly correlated with vibration amplitude (Kendall, $P = 0{\cdot}0005$).

amplitude of vibration, the movement time and the angular threshold for vibration onset were assessed using the parametric Pearson's correlation coefficient and non-parametric Kendall's rank correlation coefficient.

RESULTS

General form of alterations in wrist movements produced by vibration

The records illustrated in Fig. 1*A*, obtained from a representative subject, provide an example of a central finding of the study. Application of vibration (125 Hz, 1 mm) to the tendon of flexor carpi radialis throughout the course of each of a series of 2 s voluntary extension movements resulted in a marked (by about 40%) reduction in amplitude of the averaged movement compared to the mean trajectory of a series of undisturbed movements. In contrast, as shown in Fig. 1*B*, flexor tendon vibration produced a far smaller alteration of voluntary flexion movements compared to their

non-vibrated counterparts. Furthermore, antagonist vibration caused clear undershooting whereas stimulation of the agonist caused slight overshooting.

The quantitative plots of Fig. 1*C*, which present data from nine subjects studied during both flexion and extension movements, emphasize these differential effects of antagonist *vs.* agonist vibration. The mean amplitudes of vibrated movements, as percentages of corresponding non-vibrated ones, are shown for extension and flexion movements of three different durations (self-paced, 1 and 2 s movement times). Statistical analysis indicated that, for each movement duration, flexor vibration produced significant undershooting of extension movements (paired t test, $P < 0{\cdot}05$). Flexion trajectory amplitudes tended to show overshooting (for 1 and 2 s movement times) but overall the degree of overshooting was not significant.

Control experiments were performed to determine (i) whether mechanical stimulation of the skin over the carpus had comparable effects to that of flexor tendon stimulation and (ii) whether subjects were able to make accurate extension movements, in the presence of flexor vibration, provided that they were given a visual monitor of movement performance.

The first question was studied by repositioning the vibrator probe from the tendon of flexor carpi radialis to a more distal site over the angular ligament of the carpus. Three subjects, who had previously shown definite flexor vibration-induced undershooting of extensor movements were tested. In no case did vibration now produce significant alterations in trajectories. Thus, the vibration-induced movement errors appeared to depend upon stimulation of muscle, rather than cutaneous, receptors. The second question was investigated by providing four subjects, who had previously show vibration-induced extension undershooting, with an oscilloscope display of the trajectories of their on-going movements. All subjects were now able to accurately produce movements of target amplitude despite the presence of flexor tendon vibration. Thus, extension undershooting did not result from vibration impairing subjects' capability of producing contractions of requisite strength to attain the desired trajectory.

Effect of amplitude of flexor vibration upon undershooting of extension movements

Figure 2*A* shows the averaged trajectories of 1 s extension movements made by a subject whilst differing amplitudes (non-vibrated, 0·75 mm, 1·0 mm and 1·5 mm) of flexor tendon vibration (125 Hz) were applied. The degree of undershooting is graded with stimulus strength.

Pooled data from six subjects, for 1 s movements, are plotted in Fig. 2*B*. Each vibration amplitude is associated with a significant degree of undershooting (paired t test, $P < 0{\cdot}03$). The mean percentage of undershooting increases incrementally with vibration amplitude and a significant correlation was found between the degree of undershooting and the amplitude of vibration (Kendall's rank correlation coefficient, $P = 0{\cdot}0005$).

Relation between angular position of flexor vibration onset and the degree of extension movement undershooting

The averaged extension trajectories, made by an individual subject, presented in Fig. 3*A* illustrate the effect of initiating flexor vibration (125 Hz, 1 mm) at four different times during the course of 1 s movements. These averaged records are examples taken from a series in which the vibrator was activated at each of six different extension angular thresholds (0, 5, 10, 13, 16 and 25 deg). Vibration periods were adjusted, as fractions of 1 s, to the respective remaining angular components of the target movement amplitude. As vibration was activated at progressively greater

angles of extension, i.e. generally later in the course of the movement, so the degree of final extension undershooting decreased. The initial trajectories of the vibrated movements of Fig. 3*A*, with the exception of movement *b* which happens to commence at a relatively high velocity, closely resemble that of the non-vibrated

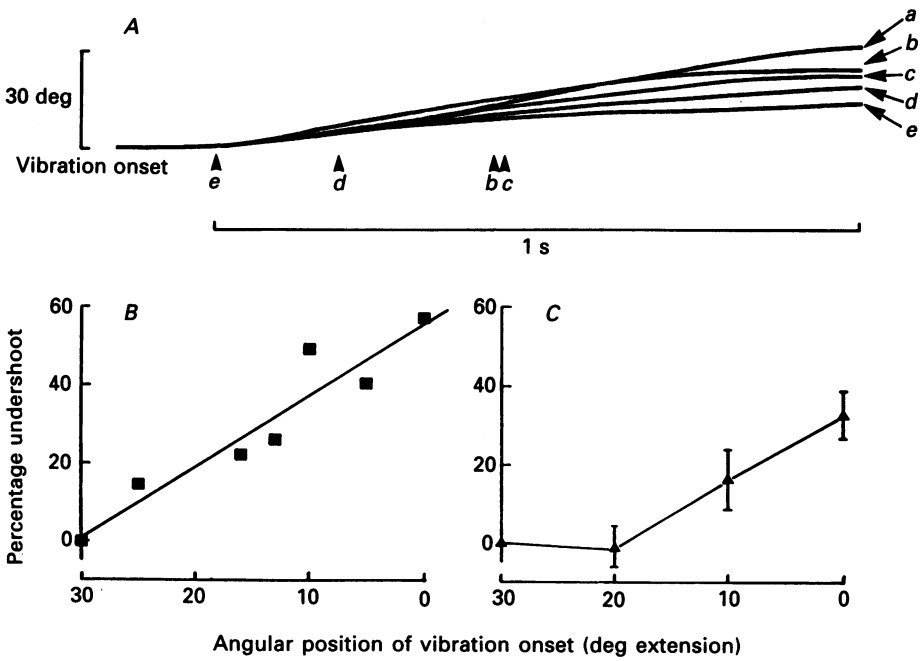

Fig. 3. Effects of varying the angular position of flexor vibration (1 mm, 125 Hz) onset upon the degree of extension movement undershooting. *A*, averaged (twenty trials) trajectories of 1 s non-vibrated movements of 30 deg target amplitude (*a*) and movements during which vibration was initiated at 16 deg (*b*), 13 deg (*c*), 5 deg (*d*) and 0 deg (*e*) during the course of the movement. The times of onset of vibration for the different trajectories are indicated by the arrows below the traces. Not that the trajectory *b* commenced at a relatively high velocity prior to initiation of vibration and that the corresponding angular threshold for vibration onset was attained relatively quickly. All movements were recorded from the same subject (G.S.) as featured in Fig. 2*A*, although those of Fig. 3*A* were obtained in a separate experiment. *B*, the degree of undershooting of extension movements made by the same subject, expressed as percentages of the non-vibrated movement amplitude, for each of six angular thresholds (including the four shown in *A*) for vibration onset. *C*, mean (±S.E.M.) undershooting, expressed as percentages of non-vibrated trajectory amplitudes, for three different vibration onset thresholds (0, 10, 20 deg extension) for data obtained in six subjects. Undershooting and threshold for vibration onset were strongly correlated (Kendall, $P = 0{\cdot}0006$).

movement; in these cases, the vibrated movement averages only start to diverge from the non-vibrated one after the onset of vibration.

Figure 3*B* plots the relationship between extension movement undershooting and the six angular positions of vibration onset studied in this subject. The linear correlation coefficient (Pearson's r value) was 0·95 and the slope differed significantly from zero ($P < 0{\cdot}002$). Pooled data from six subjects, in whom three vibration onset thresholds (0, 10 and 20 deg) were studied during 1 s movements, are shown in Fig.

3*C*. The mean degree of undershooting of extension movements increases progressively as vibration commenced earlier during the movement and a highly significant correlation was found (Kendall, $P = 0{\cdot}0006$).

Relationship between flexor vibration-induced undershooting of extension movements and extensor load

In six subjects flexor vibration (1 mm) was applied throughout 1 s extension movements made against three different torque loads of 0·32 and 0·65 and 0·97 N m. This range of loads corresponds approximately to 6–17 % of the maximum isometric voluntary extensor contraction of an average subject, although the precise percentages varied according to the strength of individual subjects. Considering pooled data for all loads, vibration produced significant undershooting (paired *t* test, $P < 0{\cdot}01$). However, there was no statistical evidence that any overall relationship existed between the size of the torque load and the degree of vibration-induced undershooting of extension movements.

Relationship between vibration-induced alterations in movement trajectories and EMG patterns

Figure 4 illustrates examples, from a representative subject, of the most common patterns of wrist extensor (extensor carpi ulnaris) and flexor (flexor carpi radialis) activities recorded during non-vibrated extension movements and during vibration-induced extension undershooting.

This subject, as did most others, normally generated extension movements, of each movement time (self-paced, Fig. 4*A*; 1 s, Fig. 4*B*; 2 s, Fig. 4*C*), by smoothly increasing extensor muscle activity. There was no appreciable co-contraction of the wrist flexors whose activity is displayed at a far higher gain than that of the extensors. Flexor vibration (1 mm, 125 Hz) applied throughout extension movements consistently resulted in undershooting. Comparison of the EMG patterns of the vibrated movements with their non-vibrated counterparts clearly indicates that undershooting arose from a sustained reduction in extensor activity whilst flexor activity remained of low level. There is, however, some hint of tiny transient increases in flexor activity, occurring at around the time of vibration onset. There is no indication of a tonic vibration reflex being elicited under present conditions.

Considering all subjects, in the large majority (about 70 %) of experiments vibration-induced extension undershooting was associated with reductions in extensor EMG with little or no change in flexor activity; in these cases the alterations in movement trajectories could be confidently attributed to decreased EMG in the prime-mover. In the remainder, a combination of reduced extensor and increased flexor activities was found. Even in these cases, however, the relative changes in extensor and flexor EMGs were asymmetrical with depression of extensor activity being the dominant feature.

The higher resolution records of Fig. 5 show the alterations in extensor and flexor activities occurring within the first 250 ms following onset of flexor vibration. These averages were initiated when the movement trajectories attained the threshold level at which vibration was triggered and comprise a proportion (16/20) of the trials used to compute the corresponding traces of Fig. 4.

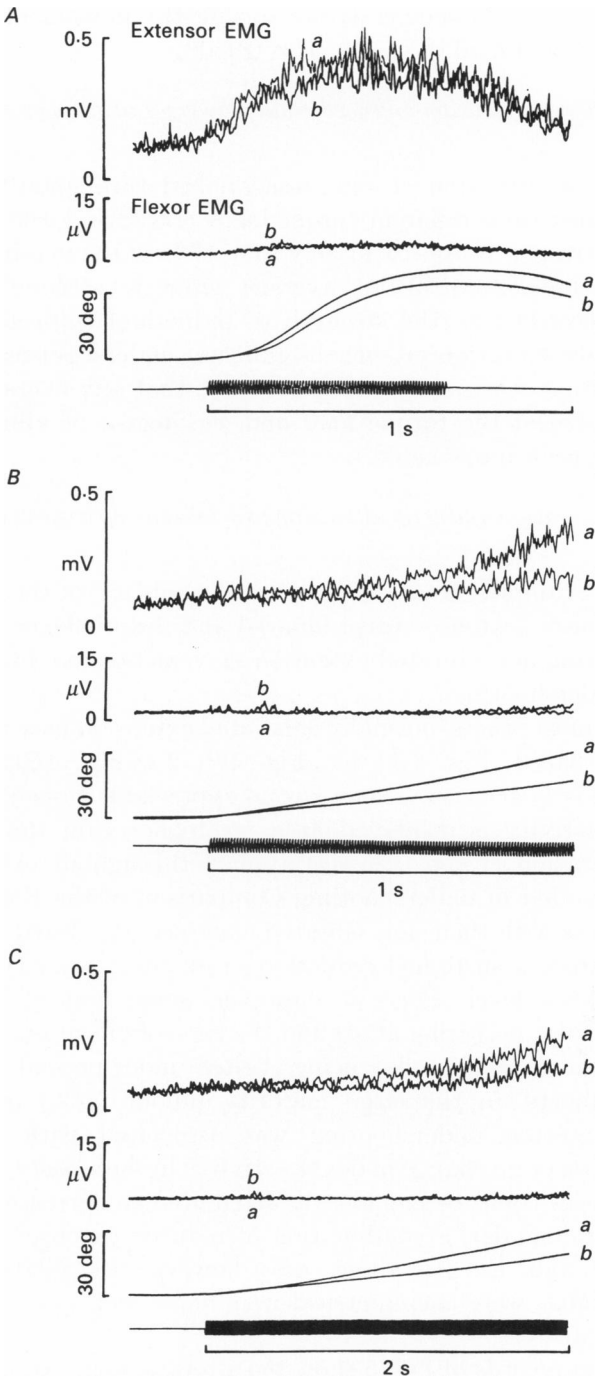

Fig. 4. Wrist extensor and flexor muscle EMG patterns accompanying non-vibrated wrist extension trajectories and vibration-induced undershooting of movements. *A*, self-paced movement; *B*, 1 s movement time; *C*, 2 s movement time. All records are averages of twenty trials. Vibration: 1 mm, 125 Hz. Non-vibrated trajectories and associated EMGs are labelled *a* and their vibrated counterparts are labelled *b*. All records obtained from a single subject (D.E.). Some of the small increases in flexor activity evident during

Clear, sharp reductions in extensor activity are evident commencing at about 40 ms (indicated by the filled arrows) after vibration onset for each of the three movement speeds (self-paced, Fig. 5*A*; 1 s, Fig. 5*B*; 2 s, Fig. 5*C*). These early depressions of extensor EMG are transient and last about 20 ms. No similarly well-defined and consistent reductions in extensor activity are apparent in the remaining sections of the averages. Instead, the extensor EMG builds up slightly more slowly than in the averages from non-vibrated movements (Fig. 5*D*, *E*, *F*) but with no demarcation point which can be readily distinguished from the on-going background fluctuations.

Small vibration-evoked excitatory responses may be identified in the averages of flexor EMG. It must be stressed, however, that these averages are displayed at high gain and that the responses themselves are relatively modest. These excitatory peaks precede the reductions in extensor activity, occur with a latency of *ca* 20 ms (marked by open arrows) and are short-lasting. On the basis of latency, which corresponds to the tendon jerk, they seem certain to represent reflexes elicited by group I a spinal action. Subsequently, there are no further systematic differences between the flexor activities of vibrated and non-vibrated movements.

The early reduction (40 ms) in extensor and increase (20 ms) in flexor activity occur considerably in advance of voluntary reaction time (*ca* 90 ms, Lee & Tatton, 1978) for wrist muscles and must be regarded as 'automatic'. Both types of response are transient and appear to be related to the onset of vibration. The initial short-lasting trough in extensor EMG constitutes, however, a rather small component of the overall vibration-induced depression of activity, as can be judged by comparison of Figs 4 and 5. The more protracted averages of Fig. 4 indicate that the extensor EMGs associated with vibrated and non-vibrated movements diverge progressively as the movements continue.

DISCUSSION

The basic observation of the present study was that experimental interference with the patterns of proprioceptive discharge normally generated during voluntary wrist movements, using muscle vibration, produced consistent errors of movement trajectories. As a corollary, this finding supports the operation of a proprioceptive feedback mechanism in the continuous regulation of wrist movements. Three main types of objections might be raised to this interpretation. First, that vibration rendered the peripheral motor apparatus incapable of responding naturally to efferent commands. This possibility was negated by the ability of subjects to readily achieve target trajectories, in the presence of vibration, if provided with a visual indicator of their movement performance. Second, that powerful 'artificial' stimulation of proprioceptors produced a physiologically nonsensical barrage of afferent input to which CNS centres might respond in an aberrant or stereotyped manner. Against this notion, however, is the observation that movement errors were clearly graded according to the strength and timing of afferent stimulation. In

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vibrated averages, particularly clear in *B*, appear to commence prior to the mean time of onset of vibration. This is presumed to result from vibration commencing at somewhat different times, with respect to the start of the averaging period, during individual movement trials so that in some instances it commenced relatively early in the sweep.

addition, the occurrence of movement errors selectively depended upon vibration over the tendons of antagonist muscles rather than resulting from arbitrary, non-specific excitation. Third, that the effect of vibration-induced sensory input might have been to disrupt a central programme of the learned movement or its 'recall'.

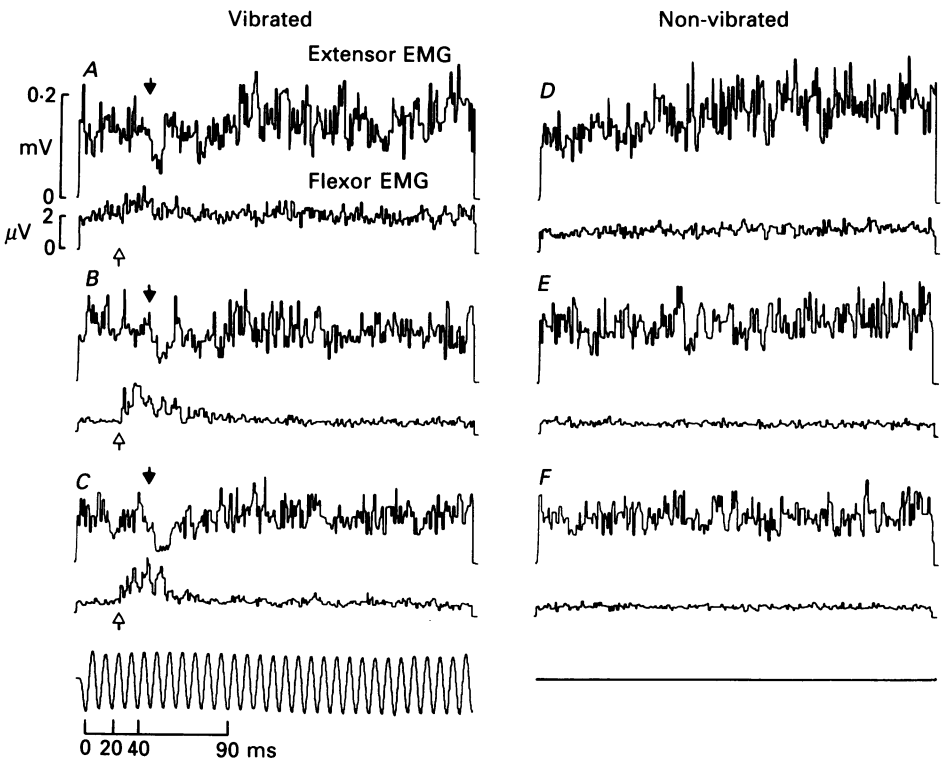

Fig. 5. High-resolution averages (sixteen trials, 1 ms per bin) of wrist extensor and flexor EMGs associated with the initial phases of vibrated (1 mm, 125 Hz) and non-vibrated extension movements. Records are from the same subject (D.E.) as in Fig. 4 and the averages were compiled from a proportion of the corresponding trials. Movements were of three speeds: self-paced, *A*, *D*; 1 s, *B*, *E* and 2 s, *C*, *F*. Averaging was triggered by a pulse generated when the trajectory had reached a specified angular displacement close to the beginning of the required movement. The same pulse was used to initiate vibration during pseudo-randomly interspersed trials. The filled down-going arrows, aligned at 40 ms following onset of vibration, mark the beginnings of reductions of extensor activity during vibrated movements. The open up-going arrows, aligned at 20 ms, mark the beginnings of small increases in flexor activity during the same trials.

This idea is improbable since interspersed non-vibrated movements were carried out normally. Furthermore, in trials in which vibration commenced in the mid-course of movements the initial parts of the trajectories were unaffected.

The present demonstration that the size of movement errors and of proprioceptive disturbances co-vary in a predictable manner extends a number of earlier observations. Several investigators have reported that vibration can modify the course of active limb movements (Goodwin *et al.* 1972*a*, *b*, *c*; Appenteng & Prochazka, 1983; Lackner, 1984; Bullen & Brunt, 1986). In keeping with the present

findings for wrist movements, these studies, which have largely concerned elbow movements, have generally shown that undershooting accompanies vibration of the antagonist whereas stimulation of the agonist has little effect.

Three principal issues arise concerning the mechanisms responsible for vibration-induced movement errors, namely, the origin of the afferent input, the relative modulations of agonist and antagonist motor discharge and the central neural pathways by which the effects are mediated.

Afferent origin of vibration-induced alterations of voluntary movements

A number of categories of sensory receptors, most notably muscle spindle receptors, Golgi tendon organs and cutaneous receptors, are sensitive to vibration and could potentially contribute to the observed effects. Of these, muscle spindle receptors, and especially their primary endings and associated Ia afferents, seem likely to exert the dominant influence. Human microneurographic recordings (Burke *et al.* 1976*a*, *b*; Roll & Vedel, 1982) have fully demonstrated that spindle primaries are extremely powerfully excited by vibration of both relaxed and contracting muscles and are relatively more sensitive than spindle secondaries or tendon organs. In addition, the most consistent movement errors were observed during vibration of the quiescent antagonist muscle when the responsiveness of tendon organs would be relatively low (Burke *et al.* 1976*a*). Whilst a contribution from cutaneous receptors cannot be totally excluded, several factors argue against skin afferents playing a major part. Vibration over the flexor tendons may be expected to activate skin receptors to a broadly comparable extent during both voluntary wrist extension and flexion movements; only in the former case were movement trajectories appreciably modified. Additionally, application of vibration to several areas of skin around the carpus, in a manner which minimized direct transmission to the tendons, failed to elicit significant movement errors. It may also be noted that earlier studies have shown that appreciable kinaesthesia survives cutaneous (and joint) anaesthesia (Goodwin *et al.* 1972*c*; but see Merton, 1964; Moberg, 1983).

Overall, the present observations strongly support muscle receptors, presumably mainly spindle primaries, in the passively lengthening antagonist providing a major source of proprioceptive input for the guidance of voluntary movements. As noted above, antagonist vibration produced distinct movement errors whereas agonist vibration did not. A probable explanation, as proposed by Capaday & Cooke (1981, 1983) to account for essentially similar findings for rapid elbow movements, is that muscle spindles in the actively shortening agonist are relatively insensitive (due to unloading) whilst those in the lengthening antagonist are progressively sensitized. Alternatively, the balance of presynaptic inhibition or central transmission of proprioceptive input from agonist and antagonist muscle afferents may differ during active movements.

Proprioceptive control of agonist and antagonist motor activities

Vibration-induced errors of wrist movement trajectories could potentially result from changes in either agonist or antagonist contractions or a combination. The electromyographic recordings of the present experiments, which were absent in most earlier related studies, have allowed this issue to be investigated. Extension

undershooting was typically associated with a sustained reduction in extensor EMG, whilst flexor activity was largely unaffected. In a few subjects, however, flexor vibration did produce a small but distinct excitatory response in the flexors. When present, such increases in flexor EMG were generally modest compared to concomitant decreases in extensor activity and were often transient; they appear incapable of having made appreciable contributions to net force production and movement. Thus, enhanced proprioceptive discharge, probably arising from antagonist muscle spindle afferents, exerted a predominantly reciprocal inhibitory effect upon motor activity.

These findings for vibration-induced alterations in EMG during wrist extension undershooting, contrast fundamentally with those of Capaday & Cooke (1983) for fast elbow movements. In the latter situation, undershooting was attributed principally to an increase in EMG in the vibrated antagonist, although increased activities in both agonist and antagonist were often noted. These discrepancies are probably related, at least in part, to differences in the underlying excitability levels of the agonist *vs.* antagonist motoneuronal pools under the different experimental conditions. The elbow movements studied by Capaday & Cooke were far faster (lasting about 250 ms) than the wrist movements presently investigated and their associated EMGs approximated to the 'triphasic' pattern (Hallett, Shahani & Young, 1975) characteristic of ballistic movements. They were initiated by a strong agonist burst which was often declining by the time of movement (and vibration) onset. Thereafter, antagonist activity increased during the movement itself. Many antagonist motoneurones were now active and, thereafter, susceptible to vibration-induced excitation from their homonymous proprioceptors; in contrast, during slower wrist movements antagonist motoneurones were generally essentially quiescent and hence relatively inexcitable. Conversely, the failure of antagonist vibration to reduce agonist activity during the course of elbow movements in Capaday & Cookes' experiments, as opposed to the findings for wrist muscles, may simply have resulted from the excitability of agonist motoneurones having already been depressed following their prior phasic discharge. It is also possible that in the experiments of Capaday & Cooke some spread of vibration to the agonist, via the straps used to secure the vibrator over the antagonist tendon, may have occurred; if so, any consequent stimulation of agonist muscle receptors may have exerted central autogenetic excitatory effects which counteracted co-existent reciprocal inhibitory actions from antagonist proprioceptive afferents.

Neural mechanisms mediating vibration-induced movement errors

The initial vibration-induced changes in EMG, during extension undershooting, were definitely 'automatic' rather than voluntary. Reductions in extensor (agonist) activity commenced at about 40 ms and well before reaction time (*ca* 90 ms for wrist muscles, Lee & Tatton, 1978). Small increases in flexor (antagonist) EMG occurred even earlier, at around 20 ms, although they probably had minimal mechanical effect.

The precise mechanism underlying the pronounced early depressions of agonist activity is uncertain. The latency of these EMG reductions, however, considerably exceeds that of classical group Ia-mediated disynaptic reciprocal inhibition for these

muscles (*ca* 23 ms, Berardelli, Day, Marsden & Rothwell, 1987). Instead, these responses probably correspond to a later phase of 'reciprocal inhibition' which may be evoked in isometrically contracting wrist muscles by vibration of their antagonists and whose possible origins have recently been discussed (Cody & Plant, 1988, 1989).

The tiny, excitatory responses recorded in the antagonists were of similar latency to tendon taps and may be confidently attributed to group Ia-mediated autogenetic spinal reflex action. They may be presumed to be basically analagous to the vibration reflexes which can be elicited in a variety of isometrically contracting muscles and whose characteristics have been well documented (Matthews, 1984). Rather surprisingly, comparable short-latency reflexes were not noted in the experiments of Capaday & Cooke (1983) despite a pre-existing level of antagonist motor discharge whose presence would be expected to favour their emergence. Instead, these workers estimated that the earliest increases in vibration-induced antagonist EMG activity, of elbow muscles, occurred at about 60 ms. Indeed, the rather delayed onset of these EMG bursts led Capaday & Cooke to propose that supraspinal centres were likely to be involved in their generation.

Whilst spinal reflex action was certainly responsible for some of the presently observed EMG changes, additional, more complex processes mediated by higher centres cannot be disregarded. The initial sharp depression of agonist activity was transient and its mechanism seems unlikely to solely account for the subsequent sustained reductions in extensor activity. Rather, a continuum of successive contributory processes, of varying degrees of 'automaticity' and including voluntary corrective reactions to perceived deviations from the desired trajectory, may be suspected.

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