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Vibration-induced Changes in Movement-related EMG Activity in Humans*

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Summary. The effect of muscle tendon vibration during voluntary arm movement was studied in normal humans. Subjects made alternating step flexion and extension movements about the elbow. A small vibrator was mounted over either the biceps or the triceps muscle and vibration was applied during flexion or extension movements. The vibrator was turned off between movements.

After a period of practice, subjects learned the required movements and were able to make them with their eyes closed. Application of vibration to the muscle antagonist to the movement being performed produced an undershoot of the required end-movement position. The undershoot was 20–30% of the total movement amplitude. In contrast, vibration of the muscle agonist to the movement resulted in no change in movement end position. The vibration-induced undershoot was associated with an increase in the EMG activity of the vibrated (antagonist) muscle and a resultant increase in the ratio of the antagonist to agonist EMG activity. The increase in antagonist EMG produced by the vibration occurred with a latency of approximately 60 ms from vibration onset.

The observed results are consistent with vibration-induced activation of muscle spindle receptors in the lengthening muscle during movement. It is suggested that, during movement, the sensitivity of the spindle receptors in the shortening muscle is decreased and the information concerning limb position during movement comes primarily from the lengthening muscle.

Key words: Muscle vibration – Spindle afferents – EMG

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Introduction

The role of muscle spindles in the control of movements has long occupied physiologists and much information has been gained of their central projections and physiological properties. At the same time little is known about how the information they relay is used in controlling movement. The studies of Goodwin and his collaborators demonstrated that muscle spindles can provide the central nervous system (CNS) with information used in the conscious perception of static limb position (Goodwin et al. 1972a, b and 1974). Utilizing muscle vibration, a technique known to phase lock spindle afferent discharge with each vibration cycle (Bianconi and van der Meulen 1963; Brown et al. 1967; Burke et al. 1976a, b), Goodwin et al. (1972a, 1974) observed a systematic distortion of the sense of position at the elbow joint when one of the muscles at that joint was vibrated. For instance, when the vibrator was applied over the biceps tendon, the forearm was always perceived to be more extended than it actually was. This finding was consistent with activation of length sensitive receptors such as muscle spindles.

The studies of Goodwin and co-workers were directed primarily at the role of spindles in the conscious perception of static limb position. How, or indeed whether, the CNS utilizes information from muscle spindles during active movements has remained unclear in spite of the fact that spindle information is known to reach area 3a of the sensorimotor cortex (Philips et al. 1971) and the demonstration by direct recording from afferent fibres that muscle spindles are active during isometric contraction (Vallbo 1970). In addition, recent studies relating spindle discharge to movement during walking in cats (Prochazka et al. 1976) support the idea of alpha-gamma co-activation during movement. Such co-activation was thought to be necessary since the

spindles in the shortening muscle would become unloaded and lose their capacity to signal muscle length changes. These studies, however, provided no indication about the use to which information from the spindles was put during the evolving movements.

In the present study, muscle vibration was used to activate spindles during voluntary, target-directed human arm movements. In an earlier report (Capaday and Cooke 1981) we showed that muscle vibration affects the attainment of the the final end position of voluntary arm movements. We here show that muscle vibration applied during the course of an ongoing movement affects the attainment of the movement final end position through characteristic changes in the movement-related electromyographic (EMG) activity. In addition, we present evidence that vibration of the lengthening muscle is most effective and that only a brief period of vibration during a movement is needed to effect changes in the movement.

Methods

Experimental Arrangement

Twenty normal humans were used in the various aspects of this study. During the experiment the subject was seated in a chair and grasped a manipulandum handle whose proximal end supported the elbow. The handle was connected to the shaft of a linear torque motor and could be moved in the horizontal plane by either flexion or extension movements about the elbow joint.

Subjects were instructed to track a target displayed as a vertical bar on an oscilloscope screen placed in their line of sight, one meter in front of them. Also displayed on the screen, as a vertical line, was the angular position of the manipulandum and thus the subject's arm. The target switched every 3 s between two fixed positions whose angular separation could be set by the experimenter. The targets were not mechanically detectable and were not bounded by mechanical stops. The subject was thus required to superimpose the position cursor on the target bar by making alternating flexion/extension movements about the elbow.

Subjects were instructed to move "briskly and accurately" between the targets (Brown and Cooke 1981). In order to force the subjects to rely exclusively on proprioceptive guidance they were required to learn to perform the task without visual guidance, that is with their eyes closed. During such trials an auditory cue was provided to signal the change in the target position and, thus, when the subject should move. After an initial training period and during the actual experimental trials no information was given to the subject as to whether he was attaining the required target positions. All subjects were well practised at the task and could move between the target position equally well with or without visual guidance.

Vibration Method

The vibrator consisted of a small D.C. motor (Wilson's of Cleveland, Model 875) with an eccentric head enclosed in a metallic casing. The vibrator was mounted over either the biceps or triceps tendon using adjustable Velcro straps. Vibration frequency was set to 120 Hz. The vibrator could be activated when the angular velocity of movement by the subject exceeded a threshold level. The threshold was normally set so that vibration was started shortly after movement initiation and ceased when the movement was terminated.

Data Recording and Analysis

All data was digitized online by a PDP 11/40 computer with an effective sampling rate of 250 Hz (500 Hz with two point averaging). Handle position and angular velocity (derived from a precision potentiometer and a tachometer) were recorded as well as a signal indicating the time of application of vibration. In some experiments, in addition to recording the kinematic variables, surface EMGs were obtained from the biceps muscle and the lateral head of the triceps muscle using silver disk electrodes. Data was collected on disk during the experiments and was later transferred to magnetic tape for storage and off-line analysis.

Results

Changes in Movements Produced by Vibration

As was described previously (Capaday and Cooke 1981) vibration of the muscle antagonistic to the movement resulted in an undershoot of the desired movement end position. Thus, vibration of the triceps during flexion movements (Fig. 1A, B) or the biceps during extensions (Fig. 1C, D) resulted in an undershoot of the desired target. In contrast, vibration of the muscle agonistic to the movement did not affect the attainment of the desired end position (Fig. 2). In the present experiments in which vibration was applied only during movement, vibration of the antagonist muscle resulted in an undershoot of the final position by twenty to thirty percent of the movement amplitude. In some experiments the vibrator was loosely mounted over the belly of the biceps muscle. The vibrator was on continuously for a period of about one minute during which approximately 30 movements (flexions and extensions) were made. This produced no effect on the attainment of the correct end position for either the flexion or extension movements. Similar results were found when the vibrator was attached over the forearm.

EMG Changes Produced by Vibration

How the final pattern of motor commands establishing movement trajectory is modified by muscle vibration was investigated by recording the pattern of EMG activity during muscle vibration. The EMG pattern associated with control, non-visually guided movements is shown in Fig. 1A, C and Fig. 2A, C. This pattern was similar to the well-known triphasic

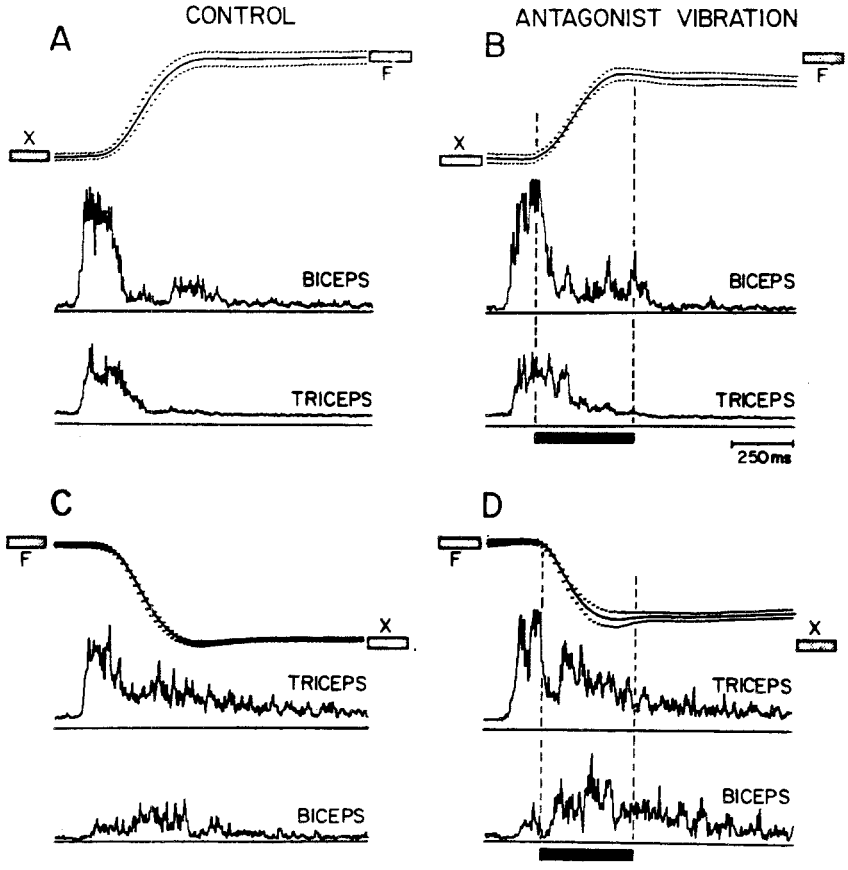

Fig. 1A–D. Effect of antagonist vibration on EMG activity. In **A–D** are shown records of averaged position records and the associated biceps and triceps EMGs from flexion (**A, B**) and extension (**C, D**) movements. All movements were performed without visual guidance. For **B** and **D** vibration was applied to the muscle antagonistic to the movement (triceps in **B** and biceps in **D**). Vibration was applied for the period indicated by the *solid bars*. Vibration resulted in an undershoot of the desired movement end position in each case and there was an associated increase in the EMG activity of the antagonist muscle. Each trace is the average of 15 movements and the inter-target distance was 65 deg in each case

pattern (Hallett et al. 1975; Brown and Cooke 1981). An initial burst of activity in the agonist was followed, in sequence, by activity in the antagonist (which could be co-extensive with the initial agonist burst) and a more prolonged period of agonist activity. Following vibration of the antagonist muscle (triceps in Fig. 1B and biceps in Fig. 1D, the subject undershot the required targets as already described. At the same time the antagonist burst was prolonged and increased in magnitude. Vibration of the antagonist had variable effects on the second agonist burst, a slight increase in activity being most common. In contrast, vibration of the agonist muscle produced no consistent changes in either the agonist or the antagonist EMG (Fig. 2B, D).

The increase in antagonist EMG produced by vibration occurred rapidly upon the onset of vibration. This is illustrated in Fig. 3 in which are shown EMGs from six extension movements. Each pair of records (A–C) shows biceps EMG activity from control movements (lower traces) and movements in which vibration was applied to the biceps tendon (upper traces). The period during which vibration was applied is indicated by the solid bars. As indicated by the vertical lines, the biceps EMG increased relative to control levels approximately 60 ms after vibration onset. A similar latency was found in eight subjects.

Relation Between EMG Changes and Movement Undershoot

Qualitative inspection of EMG records indicated that the magnitude of the vibration-induced undershoot was related to the magnitude of the change in EMG. Thus, the greater the vibration-induced EMG activity in the antagonist, the greater would be the undershoot. However, this was not always so: as noted earlier, vibration of the triceps (for instance) sometimes produced an increase in the activity in both triceps and biceps muscles. Therefore, to quantitatively relate EMG changes to movement undershoot, a measure of the relative change in EMG during vibration was calculated. The measure chosen was the ratio of EMG activity in the antagonist (vibrated) to agonist (non-vibrated) muscles. Movements and their associated EMGs were aligned about the start of the movement (Fig. 4A, B). The period during which vibration produced an increase in antagonist EMG activity (vertical dashed lines, Fig.

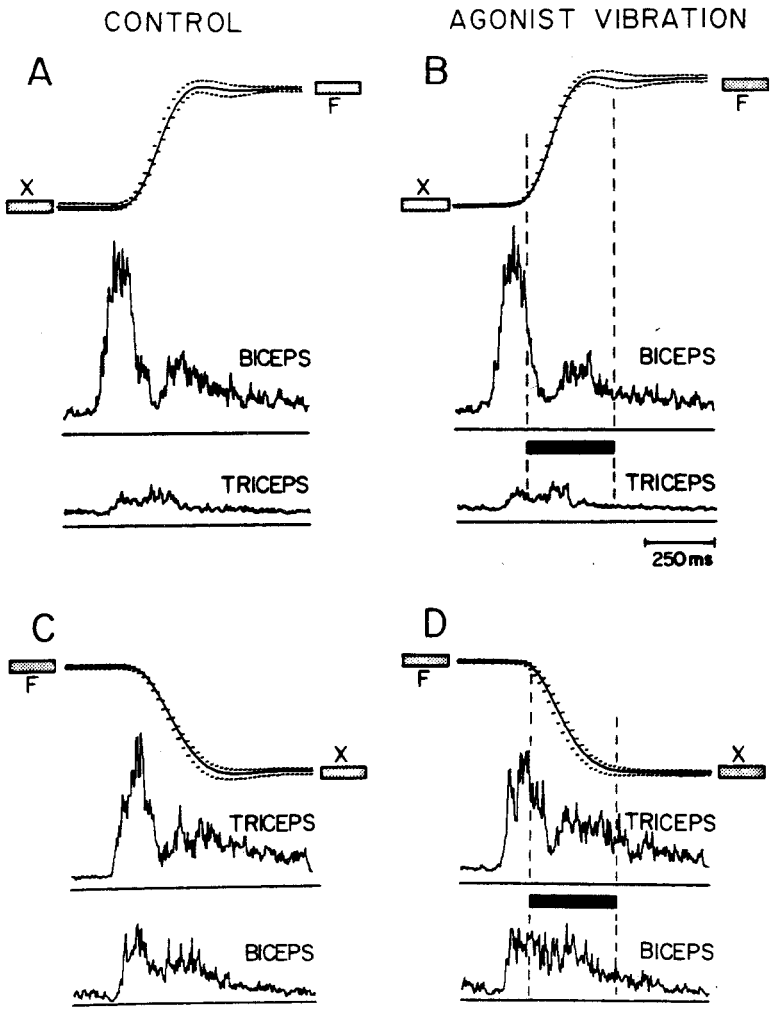

Fig. 2A–D. Effect of agonist vibration on EMG activity. Average position records and the associated biceps and triceps EMGs are shown for flexion (**A, B**) and extension (**C, D**) movements. For **B** and **D** the muscle agonist to the movement was vibrated during the period indicated by the *solid bar*. Thus, vibration was applied to the biceps during flexions (**B**) and to the triceps during extensions (**D**). Vibration produced no change in the end position of the movement. Each trace is the average of 15 movements. Intertarget distance was 65 deg

4B) was visually identified by superimposing the EMG traces. The integrated EMG activity in this period was determined and the ratio of antagonist to agonist activity calculated for both vibrated (Rv) and control (Rc) movements. The change produced in this ratio by vibration was then expressed as a percentage of the control ratio (% change = (Rv – Rc)/Rc).

In Fig. 4C the relation between movement undershoot (as a percentage of control movement amplitudes) and the change in the EMG activity is plotted. Each of the points was obtained from a different subject (filled circles – no visual guidance; open circles – visual guidance). Data was obtained from both flexion and extension movements with the antagonist being vibrated in each case. Undershoot of the target was directly related ($r = 0.92$) to the change in the ratio of antagonist to agonist EMG activity: the greater the relative increase in antagonist EMG activity the greater the undershoot. Note that with visual guidance (open circles) there was no undershoot and no change in the relative EMG activity.

It should be noted that vibration-induced changes were observed only in the phasic portion of the EMG associated with the movements. No changes were observed in the tonic EMG activity, that is, the EMG activity during maintenance of the acquired final position. It appeared that the end-position of the movement was determined by the phasic EMG activity and the limb was not held in this position by altered tonic activity. Indeed, in our experimental arrangement no correlation was found between resting limb position and the ratio of agonist to antagonist EMG activity as has been found by other workers (Lestienne et al. 1981; Sakitt 1980).

Discussion

Whether the information from length sensitive elements in muscle contributes to control of ongoing

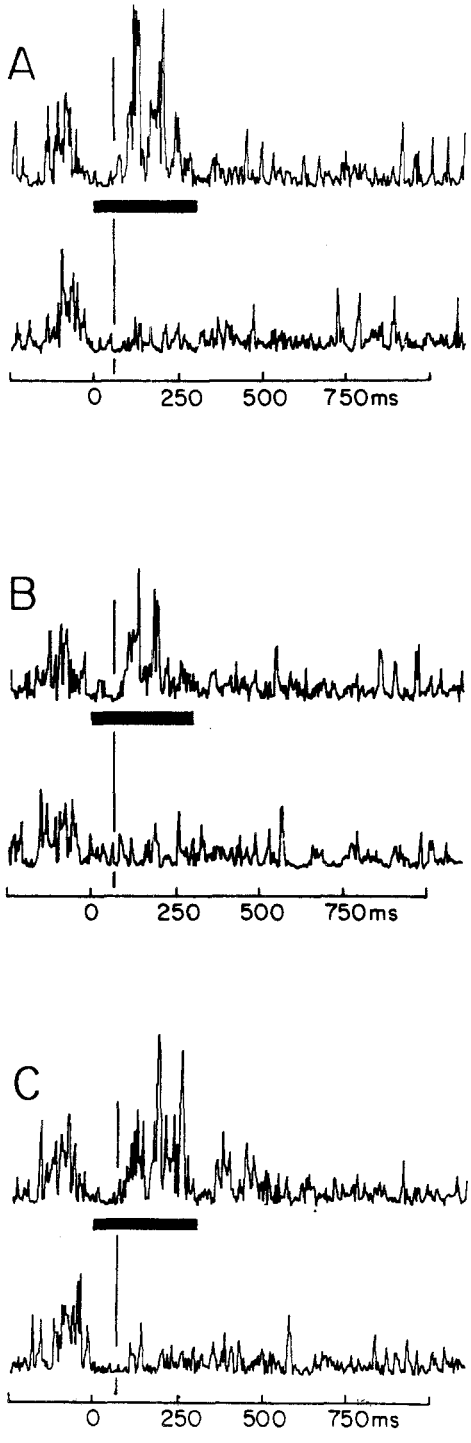

Fig. 3A–C. Latency of onset of vibration-induced increase in EMG activity. In A to C are shown pairs of records of biceps EMGs from individual flexion movements made without visual guidance. The EMG records are from sequential movements and were aligned about the start of movement. The lower record in each pair is from a control, non-vibrated movement and the upper record from movements in which vibration was applied to the biceps muscle during the period indicated by the *solid bar*. EMG activity in the vibrated movements increased relative to the control movements starting some 60 ms onset of vibration

movements has been unclear for some time. Indeed, until rather recently it was felt that muscle spindle afferents did not project to sensori-motor cortex. The finding of a projection from muscle spindles to area 3a (Philips et al. 1971) suggested the possibility of a cortically-mediated stretch reflex, analogous to the classic spinal myotatic reflex. Indeed, EMG responses to brief limb perturbation have been observed at latencies commensurate with a pathway involving cortical and/or sub-cortical structures (Hammond et al. 1956; see Wiesendanger 1978 for review).

The first reasonably convincing evidence that information from muscle spindles is utilised in limb motor control came from the studies of Goodwin and his colleagues. Their main interest was whether or not spindle information was utilised in or could affect the conscious perception of static limb position. The technique they used was that of vibration of the muscle tendon. Such vibration has been shown to be a powerful activator of muscle spindles (Matthews 1972; Burke et al. 1976). In addition, it has been found that activity in neurones in the motor cortex can be modified by perturbations applied to the limb (Conrad et al. 1976; Lee and Tatton 1975; Evarts and Tanji 1976). However, in none of these studies in behaving primates or humans has it been clearly established that muscle spindles were mainly or exclusively involved in the changes seen in firing of cortical neurones.

Modification of active movement by muscle vibration has been noted by several authors (Goodwin et al. 1972b; van Beekum 1979; Capaday and Cooke 1981). Van Beekum for example, commented that when vibration was applied during brisk arm movement, he observed "A very peculiar effect which was seen in all subjects . . . the asymmetrical behaviour of flexion and extension movements". In the experiments described here, vibration-induced changes in spindle afferent inflow during target-directed forearm movements produced changes which depended on whether the vibrated muscle was shortening or lengthening. Antagonist vibration resulted in an undershoot of the required end-movement position whereas agonist vibration had no effect on the correct attainment of final position. These changes of movement trajectory were accompanied by specific changes in EMG activity in the agonist and antagonist muscles: vibration of the antagonist resulted in an increase in the ratio of antagonist to agonist activity whereas vibration of the agonist did not alter this ratio.

These effects of muscle vibration during movement are consonant with the activation of the length-sensitive muscle spindle receptors. Thus, vibration of

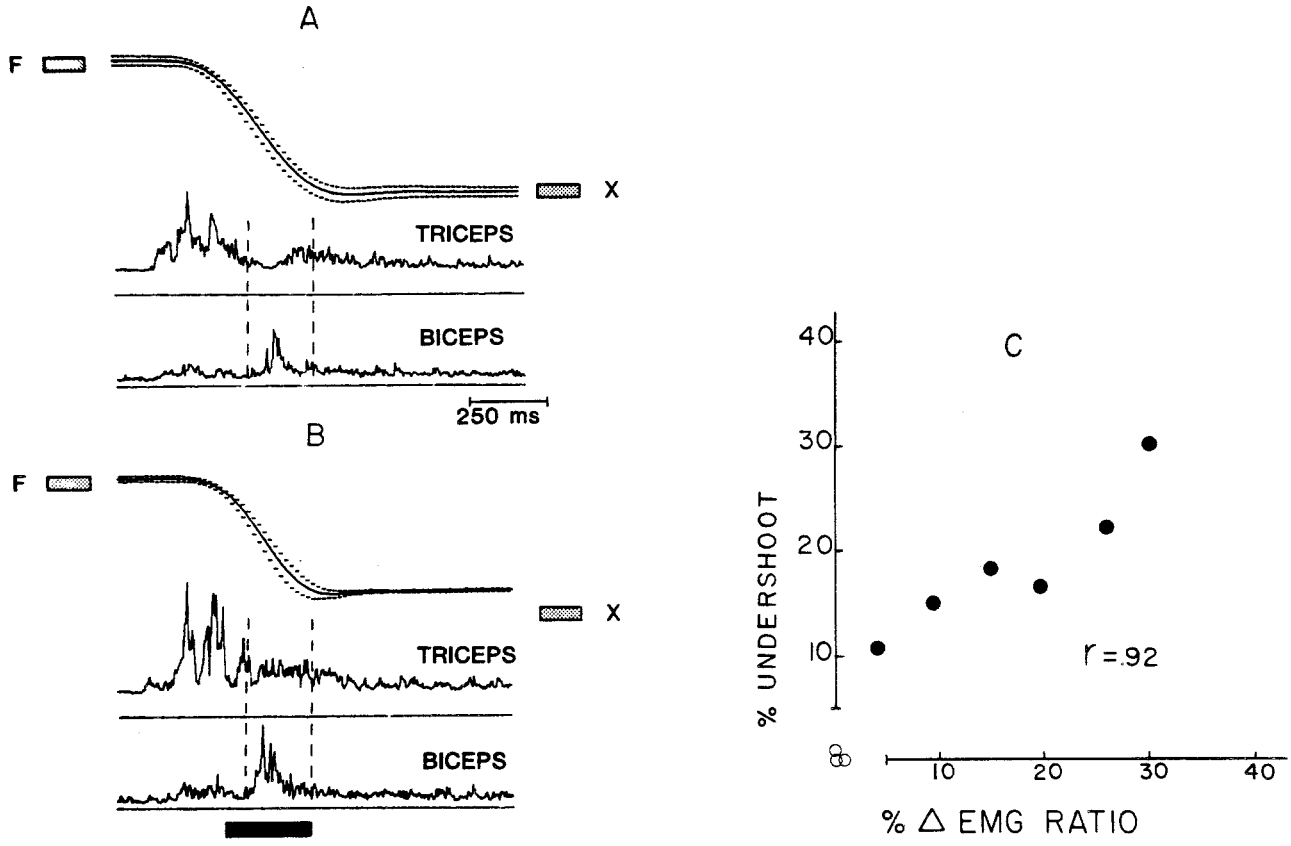

Fig. 4A–C. Relation between vibration induced undershoot and EMG changes. In **A** and **B** are shown average records of position, and biceps and triceps EMGs from extension movements. **A** shows records from control movements and **B** from movements in which vibration was applied during the period indicated by the *solid bar*. The *vertical dashed lines* indicate the period during which biceps EMG activity was increased by vibration relative to control. In **C** the percent undershoot is plotted as a function of the change in EMG activity in the vibrated antagonist relative to the agonist (see text for details). *Solid symbols* were obtained from trials without visual guidance and *open symbols* with visual guidance. Each point was obtained from a separate subject

the lengthening antagonist would increase the latter's spindle afferent discharge. This increased discharge would 'misinform' the CNS about limb displacement in a direction consistent with the observed results. For example, vibration of the triceps during flexion would increase the spindle afferent discharge from the triceps. This would correspond to an apparently overflexed forearm resulting in an undershoot of the required flexion target. The lack of effect of agonist vibration on movement is explained if the muscle spindle receptors have a decreased sensitivity during rapid muscle shortening. In this regard, it should be noted that a differential effect of vibration on slow and rapid movements has been reported previously (Goodwin et al. 1972; van Beekum 1979).

This conclusion that spindle receptors in the shortening muscle are relatively insensitive to vibration during rapid movement is consistent with results obtained by recording spindle afferent activity during rapid finger, jaw and hindlimb movements in both man and behaving animals (Vallbo 1970; Goodwin and Luschei 1975; Prochazka et al. 1976; Lund et al. 1979; Loeb and Duysens 1979). These investigators reported that spindle afferents were silenced during rapid muscle shortening. The shortening velocities involved in these studies were, for the most part, well below 100 deg/s (rate of joint rotation); less than those in the present study. In all of the above studies it was also found that the muscle spindles of the antagonists discharged at high frequencies during the movements.

It may be argued that the observed effects of muscle vibration were due to activation of force-sensitive receptors such as the Golgi tendon organs. In response to increased muscle tension Ib afferents induce ipsps in homonymous alpha-motoneurones and epsps in antagonist motoneurones (Watt et al. 1976). In this case, vibration of the antagonist, if it activates tendon organ receptors, should result in an overshoot of the required target rather than the

observed undershoot. It thus appears unlikely that the observed changes could be due to activation of tendon organs. Data was presented in the present paper indicating that stimulation of cutaneous receptors was also ineffective.

The undershoot of the required end-position produced by antagonist vibration was associated with an increase in the ratio of antagonist to agonist EMG activity. Activity in the antagonist always increased whereas activity in the agonist remained the same or increased somewhat. The undershoot was thus the result of an active braking process whose magnitude was modified by muscle spindle information from the antagonist. What is the origin of the increase in antagonist EMG activity with antagonist vibration? The increase could, presumably, be due to an action of spindle afferents at the spinal level via the monosynaptic reflex arc. However, in view of the relatively long latency (some 60 ms) of the vibration-induced changes of the antagonist EMG it is likely that this effect is, at least in part, mediated by supraspinal structures, possibly involving the cerebellum. It has, for example, been postulated that the cerebellum forms part of a proprioceptive loop involving spindle information from the antagonist muscle and which functions to decelerate movements (Mackay and Murphy 1979; Burton and Onada 1978).

The present results support a model for movement control in which afferent information from the lengthening antagonist muscle is used to provide the nervous system with position information from the limb. In this regard, the recent findings of Burgess et al. (1982) are of interest. They have shown differential activation of muscle spindels in antagonistic muscles of the cat leg depending on the joint position. "Under passive conditions, muscle receptors signal over the joint's entire range, with agonist and antagonist muscles dividing the range about equally between them". We would suggest that, during movement, there is a progressive shift in the source of position information from the agonist to the antagonist muscles. At the same time, of course, it is clear that large perturbations to the limb stretching the agonist during movements do produce "reflex" activity. It is not clear whether this activity arises exclusively from muscle spindle receptor activation. It is, however, possible, that large perturbations which stretch the shortening agonist could produce sufficient stretch of the spindle receptors for them to contribute to these reflex responses. Thus, we would suggest that in learned, non-perturbed movements the CNS obtains little information from the muscle receptors of the shortening muscle but that these receptors can be used in situations in which movement is perturbed.

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