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ELECTROMYOGRAPHIC REFLEXES EVOKED IN HUMAN WRIST FLEXORS BY TENDON EXTENSION AND BY DISPLACEMENT OF THE WRIST JOINT

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SUMMARY

1. The electromyographic (EMG) reflexes evoked in the wrist flexor muscle, flexor carpi radialis (FCR), by percutaneous extension of its tendon and by forcible extension of the wrist joint have been studied. Reflexes were elicited during steadily maintained voluntary flexor contraction of 10% of each subject's maximum.

2. Tendon extension, using 'ramp and hold' displacements, evoked fairly prolonged (*ca* 50 ms) increases in EMG activity. These responses were usually subdivided into two main excitatory peaks of respectively short (SL, *ca* 20 ms) and long (LL, *ca* 45 ms) latency. This pattern contrasted with that observed following brief tendon taps when only a single, SL peak was elicited.

3. 'Stretch' reflexes evoked by 'ramp and hold' wrist extensions, as has been noted by numerous earlier investigators, were also protracted and comprised two main excitatory components. These responses resembled those produced by tendon extension both in their general form and in their behaviour upon altering the velocity of mechanical stimuli. Quantitatively, however, two main differences were evident. The reflexes evoked by wrist extension, including their SL and LL peaks, were generally somewhat larger. Additionally, when parameters of the two modes of stimulation were adjusted to elicit SL responses of equivalent amplitude, the LL responses elicited by tendon extension were regularly smaller and of shorter duration than those elicited by wrist extension.

4. Termination of the two forms of mechanical stimulation, by releasing tendon or wrist extension, each elicited a SL reduction in EMG activity. Such troughs were more pronounced and more consistently observed upon release of wrist extension.

5. Neither local anaesthesia of the skin overlying the flexor tendons at the wrist nor ischaemia of the hand and lower forearm produced any systematic modification of reflex response patterns.

6. It is concluded that intramuscular receptors (presumably muscle spindles) in FCR mediate both the SL and LL reflexes evoked in this muscle by extension of its tendon. Intramuscular receptors also seem certain to be very largely responsible for the EMG responses generated in this muscle by wrist extension.

INTRODUCTION

Tendon taps form the classical method by which myotatic reflexes are elicited in man. Although still widely used in clinical practice, this relatively direct mode of applying stretch to muscles has been largely abandoned in laboratory studies of human motor control. Instead, recent investigations of 'stretch reflexes' have concentrated upon analysis of the EMG responses evoked in a muscle upon its extension by forcible displacement of the joint about which it acts. Particular interest has been aroused by the finding that comparatively protracted joint displacement commonly produces a prolonged reflex response comprising a short-latency (SL or M1) component, believed analagous to the single EMG burst characteristic of the tendon jerk, and a long-latency (LL or M2) peak of activity (Hammond, 1954, 1956; Tatton & Lee, 1975). The reflex mechanisms responsible for M1 and M2 responses are likely to act co-operatively in the continuous reflex regulation of voluntary contraction and thereby to help counteract external disturbances and the effects of muscle fatigue as well as to contribute to muscle tone.

Joint displacement, as a means of evoking 'stretch reflexes', is a more physiological form of stimulus than tendon extension since it more closely resembles naturally occurring bodily movements. Unfortunately, resolution of the afferent origin of such 'stretch reflexes', and especially their later components, has proved difficult. In addition to muscular receptors, a variety of non-muscular receptors (e.g. skin, joint) are likely to be simultaneously activated. Indeed, M2 responses, of different muscles, have been alternatively attributed to the reflex action of muscle spindle group Ia (Phillips, 1969; Marsden, Merton & Morton, 1972; Eklund, Hagbarth, Hagglund & Wallin, 1982) or group II (Matthews, 1984) afferents, both muscular and non-muscular (cutaneous and possibly joint) afferents (Marsden, Merton & Morton, 1985) and solely cutaneous afferents (Darton, Lippold, Shahani & Shahani, 1985). Furthermore, joint displacement usually causes combined extension of several synergistically operating muscles and reciprocal release of their antagonists. Thus, the contribution of receptors from individual muscles to the overall reflex response is hard to ascertain. Of course, it may also be noted that the precise mechanisms involved in the genesis of stretch reflex patterns, including the relative importance of the various sources of sensory input, are likely to differ between muscles and according to the task being undertaken.

In the present experiments we have studied the reflex responses elicited in human wrist flexor muscles by forcible transverse 'ramp and hold' extensions and releases of their tendons. Wrist flexors have especially long and accessible tendons so that this mode of stimulation allows relatively selective stretching and unloading of individual muscles with reduced concomitant activation of non-muscular receptors. Our results, using tendon extension, indicate that both SL and LL reflex responses can be evoked in the flexor carpi radialis (FCR) by predominantly muscular afferent activity. M1 (SL) and M2 (LL) responses produced by wrist displacement resembled these respective reflex components, in their general form and resistance to anaesthesia of non-muscular receptors, suggesting that they also arise largely from muscle afferents. Preliminary observations have been reported briefly (Cody, Plant & Richardson, 1988*a*).

METHODS

Subjects

The main series of experiments, comparing reflex responses to tendon and wrist extension, was performed on twenty-six subjects (fifteen male, eleven female, aged between 19 and 54 years). Twenty-four of these subjects were studied on a single occasion and the other two were studied on two occasions. Reflexes following release of tendon and wrist extension were studied in six subjects (four male, two female, aged between 20 and 42 years). The effect of anaesthesia of the skin overlying the flexor tendons upon reflex patterns was tested in eight subjects (four male, four female, aged between 23 and 48 years) whilst the effect of ischaemia of the hand and lower forearm was investigated in two subjects (the authors, aged 25 and 39 years). None of the subjects had any history of neurological or cardiovascular disease. All subjects participated with their informed consent (Code of Ethics of the World Medical Association, Declaration of Helsinki) and protocols were approved by the local ethical committee.

Experimental arrangements

Detailed accounts of the general techniques and equipment used have been reported previously (Cody, MacDermott, Matthews & Richardson, 1986).

Subjects exerted a constant wrist flexor torque (typically about 1·0 N m) of 10% of their individual maximum throughout the experiment, aided by an oscilloscope display of their force production. They were instructed to try to relax their fingers to reduce activation of finger flexor muscles.

Mechanical stimulation

Tendon extension. A small electromagnetic vibrator was used to apply 'ramp and hold' extensions percutaneously to the tendon of FCR. The arm was semipronated and the hand orientated in line with the axis of the forearm. The body of the vibrator was rigidly clamped with its probe directed at right angles to the tendon about 5 cm above the wrist crease. The plastic, grooved head of the probe was positioned so that prior to commencement of the extension stroke it pressed lightly upon the tendon of FCR; this ensured that the movement of the probe was transmitted to the tendon as completely as possible. Stimulus waveforms were monitored by a length transducer attached to the shaft of the vibrator. The stroke of the vibrator probe was usually of 2 mm extent (sixteen subjects) although for some subjects larger displacements of 3 or 4 mm were used.

Wrist extension. 'Ramp and hold' movements of 10 mm extent were applied (see Cody *et al.* 1986) which corresponded to approximately 10 deg of angular displacement of the wrist joint.

The range of velocities of wrist extension was typically 100–400 mm s^{-1} whilst the velocities of tendon extension ranged from 50 to 286 mm s^{-1}. Repetition rate of each of the two forms of extension was usually 0·66 s^{-1}. An interval of at least 900 ms was allowed following the return of the mechanical stimulator from its 'hold' to its rest position before commencement of the next dynamic phase of extension. Responses to release of extension were studied using a different repetition rate, 0·33 s^{-1}, and more prolonged 'hold' phase of 1250–1350 ms.

Selectivity of tendon stimulation

In two subjects (the authors) the reflex responses evoked in FCR were studied when the stimulus probe was positioned over its own tendon and then serially repositioned over its neighbouring superficial flexor tendons (PL, palmaris longus; FDS, flexor digitorum superficialis). Stimulation (2 and 4 mm, velocities 100–150 mm s^{-1}) over FCR tendon elicited the usual well-developed SL and LL responses (see Results). Transfer of the stimulator probe to the immediately adjacent PL tendon produced a dramatic decline in amplitude of the EMG responses recorded over FCR. In some trials stimulation now failed to evoke any definite response and on average the SL and LL components (normalized for background activity, see below) were, respectively, about 15 and 30% of their former size. Stimulation over FDS tendon, which in both subjects was deeper and more inaccessible, was even less effective, although a small residual LL response was occasionally observed. These findings strongly indicate that during application of extension to FCR, using a probe directly located over its own tendon, any spread of stimulus to nearby tendons (and stretching of PL and FDS) will have had negligible action in production of the SL responses of FCR and only a minor influence on its LL response. This is especially so since any extension of PL

and/or FDS due to stimulus spread seems certain to have been far smaller than that produced by a probe placed over their own tendons.

Electromyography

Differential surface recordings were made from the belly of FCR using pairs of electrodes placed 3 cm apart. Signals were filtered (bandpass 0·1 Hz to 3 kHz) and rectified (time constant 1 ms) before on-line averaging (Neurolog NL 750, binwidth 2 ms).

Cutaneous anaesthesia

Anaesthesia of an area of skin of approximately 6 cm^2 over the flexor tendons at the wrist was produced by the intradermal injection of a solution of 2% Xylocaine (without adrenaline). Typically four to five injections were made and a total of about 2·5 ml of anaesthetic was used.

Ischaemia

Ischaemia of the hand and lower forearm was produced by inflation of a narrow blood pressure cuff, applied just below the belly of FCR, to around 200 mmHg. Reflex recordings were made before and at 10 min intervals throughout the 1 h period that the cuff was inflated.

Measurement and data analysis

Areas of rectified EMG averages were made by tracing the relevant sections of the records using a graphics tablet linked to an Apple II computer. The areas of SL responses were measured over a period of 20 ms commencing at the onset of the initial, excitatory peak. The LL response areas were measured over a period commencing 25 ms after the SL onset and finishing at the point at which the later excitatory peak returned to background levels. Areas are expressed as 'normalized' values with respect to background activity (i.e. fractional increases over background). These values were calculated by computing the mean height of response above pre-stimulus background level and then dividing it by the mean height of a 50 ms pre-stimulus period of the background activity.

RESULTS

Reflex responses evoked by tendon extension

The records of Fig. 1 illustrate the main new finding of the study. 'Ramp and hold' extensions of a range of velocities, applied percutaneously to the tendon of FCR evoked excitatory, rectified, EMG responses lasting some 50 ms. In addition, apparently separate short- (SL, *ca* 20 ms) and long- (LL, *ca* 45 ms) latency components are evident, especially at the intermediate rates of extension. This pattern contrasted markedly with that produced by a brief tendon tap (Fig. 1, top trace) when only a SL response was elicited.

Alterations in the velocity of tendon extension caused several consistent changes in the reflex pattern. Increasing velocity produced a slight reduction in the latency of the initial excitatory peak. More notably, a progressive increase in the amplitude of the SL component occurred. The behaviour of the LL component was more complex. At the highest velocities studied the LL responses were inevitably modest. As extension rates were progressively reduced the LL responses became more prominent only to decline again at the slowest rates when the overall reflex was attenuated.

Upon releasing tendon extension small, but definite reductions in EMG activity could be identified in all subjects. These troughs commenced at about 25 ms, i.e. at SL (see Fig. 3*A*). Their occurrence was, however, rather inconsistent and, for any individual subject, SL reductions in EMG could be confidently distinguished in only a proportion of trials. No unequivocal indication of a separate LL depression was

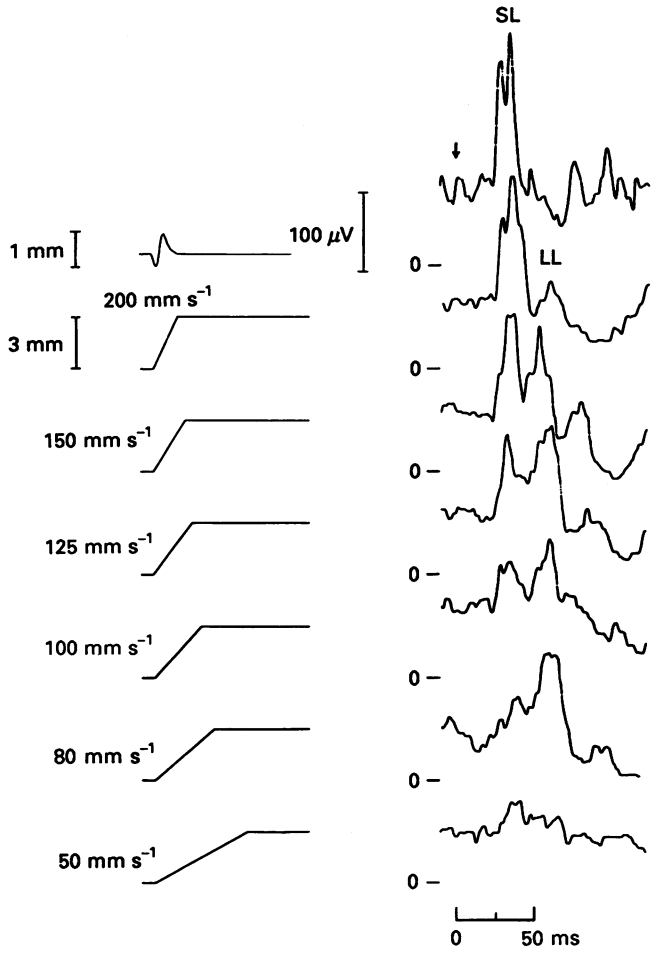

Fig. 1. Patterns of reflex EMG responses evoked in FCR by a brief tap (top trace) and 'ramp and hold' extensions, at a range of velocities, of its tendon. The subject maintained a steady wrist flexor torque of 10 % of his maximum. Displacement waveforms are shown to the left. EMG responses are rectified averages (sixty-four sweeps; repetition rate, 0·66 s^{-1}) of surface recordings. The horizontal bar at the start of each EMG average indicates, in this and subsequent figures, zero activity level.

ever noted; instead the SL troughs were often terminated at about 50 ms by the appearance of an excitatory wave. Such late excitatory peaks did not depend critically upon a preceding decrease in motor discharge since they were observed in the absence of SL reductions in EMG.

Reflex action of synergistic afferent input

In two subjects (the authors) the reflexes simultaneously elicited in FCR and flexor carpi ulnaris (FCU) were compared when the stimulus probe was sited over (i) FCR tendon and (ii) FCU tendon. Tendon extensions of 2 and 4 mm amplitude were applied at rates of 100–150 mm s^{-1}.

The reflex patterns elicited in FCR and FCU muscles by stimulation over their own tendons differed systematically from those following stimulation over the tendon of the synergist. Stimulation over the muscle's own tendon typically evoked well-defined SL (*ca* 20 ms) and LL (*ca* 45 ms) responses; the SL responses were on average larger (SL/LL amplitude ratio > 1). Stimulation over the synergist's tendon elicited either no obvious SL activity or SL peaks which were far smaller (on average about 15%) than those seen with direct tendon stimulation. Distinct LL peaks were, however, regularly observed and SL/LL amplitude ratios ranged from 0 to 0·6.

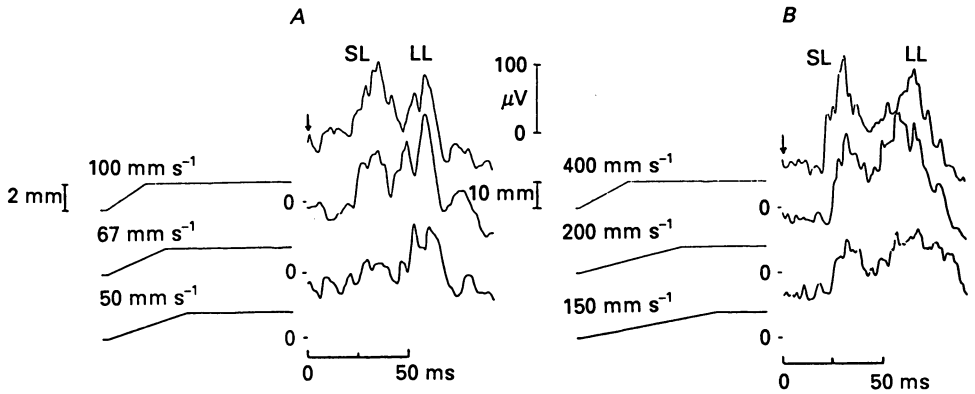

Fig. 2. Comparison of the rectified, averaged (sixty-four sweeps; repetition rate, 0·66 s^{-1}) reflexes evoked in FCR by tendon extension (*A*) and wrist extension (*B*), of varying velocities, in an individual subject.

Comparison of reflex responses evoked by tendon and wrist extension

Figure 2 compares the reflex responses to tendon extension with those to the more usual method of applying 'stretch' to FCR of wrist extension.

A striking similarity in the general pattern of the reflex responses to the two modes of 'stretch' is evident. As has been previously reported by several groups of investigators (Lee & Tatton, 1975, 1978; Bawa & McKenzie, 1981; Cody *et al.* 1986; Cody, Richardson, MacDermott & Ferguson, 1987) following extensions of the wrist the reflex responses are also rather prolonged and comprise SL (*ca* 20 ms, M1) and LL (*ca* 45 ms, M2) components. Over the range of velocities studied, the average latencies of the initial excitatory responses evoked by the two modes of stimulation corresponded closely, being, respectively, $22·2 \pm 0·3$ and $21·7 \pm 0·3$ ms (mean $\pm$ S.E.M.) for tendon and wrist extension.

Velocity-dependent alterations in the behaviour of reflexes elicited by wrist extension resembled those already described for tendon extension in several major respects: (i) latencies of M1 (SL) peaks increased slightly as the velocity of wrist extension decreased, (ii) increasing the stimulus velocity produced consistent increases in M1 (SL) amplitudes, and (iii) the amplitudes of M2 (LL) components were regularly greatest for intermediate stretch rates, being less pronounced at the fastest rates when M1 (SL) responses dominated and at the slowest rates when the overall size of the excitatory reflex waveform diminished.

It should be noted, however, that the displacement waveforms used to generate comparable reflex patterns varied considerably between the two types of stimulation. The amplitudes of movement of the transverse probe, used to apply extension of the FCR tendon, were necessarily limited (typically 2 mm, occasionally 3 or 4 mm). Application of larger displacements was impractical due both to the need to minimize stimulus spread between neighbouring tendons and the physical obstruction provided by deep tissues. Wrist extensions, which produced longitudinal stretches of several tendons, were routinely of 10 mm amplitude. The 'ramp' phases of displacements used for transverse tendon extension were, on average, shorter-lasting than those producing wrist extension.

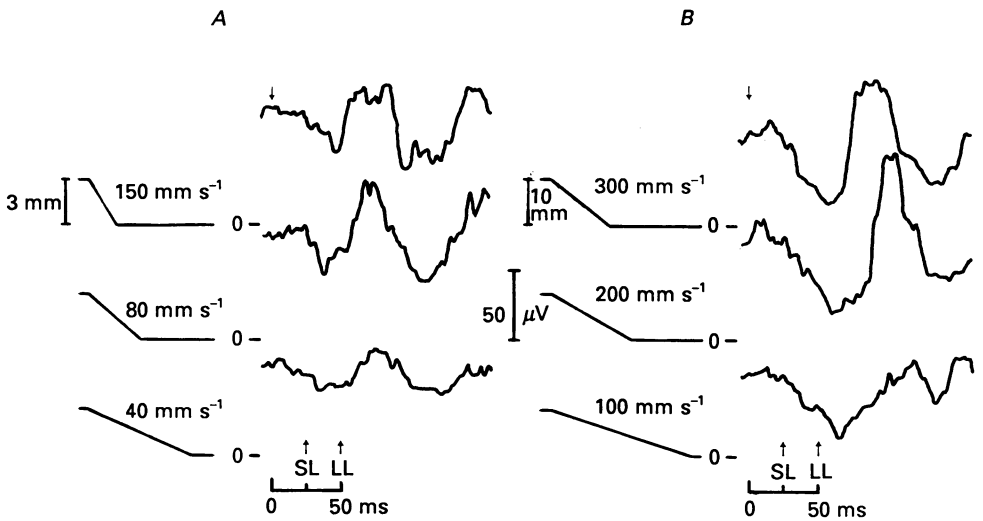

Fig. 3. Comparison of the rectified, averaged (128 sweeps; repetition rate, 0·33 s^{-1}) reflexes evoked in FCR by release of tendon extension (*A*) and of wrist extension (*B*), of varying velocities, in an individual subject.

Release of wrist extension consistently evoked clear-cut reductions in EMG activity, as shown in the records of Fig. 3*B*. These troughs had a comparable latency (*ca* 25 ms) to those observed upon release of tendon extension (Fig. 3*A*) but were more pronounced and longer lasting. Occasionally an inflexion was present on the falling slope at around 50 ms (see Fig. 3*B*, bottom trace) suggesting the possibility of an additional LL contribution.

Quantitative analysis of the effect upon reflex patterns of altering the stimulus velocity

Quantitative analysis of the amplitudes of SL and LL responses evoked, in the same subjects, by varying rates of tendon and wrist extension confirmed that the response patterns to these two forms of stimulus shared similar basic velocity-dependent characteristics. Figure 4 presents histograms of the mean amplitudes of SL and of LL responses (and the ratio of SL/LL responses) for a range of velocities of the two forms of stretch.

Mean SL response amplitudes, to both types of mechanical stimulus, decline as rate of extension falls ($P < 0{\cdot}05$, Kruskal–Wallis one-way analysis of variance). A similar relationship was also found for each individual subject (Kendall rank correlation coefficient). Mean LL response amplitudes are relatively less affected by

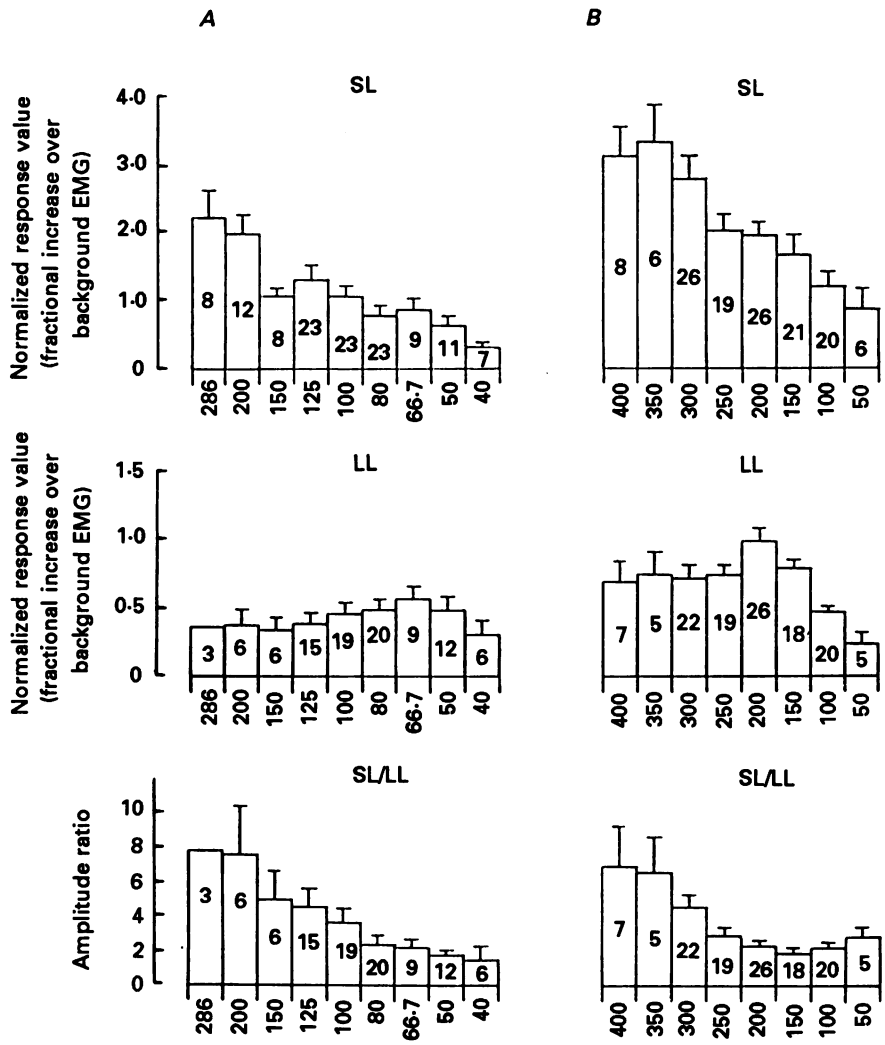

Fig. 4. Histograms showing the effects of altering the velocity of tendon extension (*A*) and of wrist extension (*B*) upon the mean (±S.E.M.) amplitudes of SL and LL responses and upon SL/LL ratios. Response amplitudes are expressed as normalized values in relation to background EMG (see Methods). The values below each column of the histograms indicate the velocity of stimulation in mm s^{-1}. The values appearing within each column indicate the number of observations.

changes in stimulus velocity. These responses do, however, tend to be greatest for intermediate rates and show reductions at the lowest velocities. The ratios of grouped mean and individual SL/LL responses decrease with decreasing velocity ($P < 0{\cdot}05$, Kruskal–Wallis and Kendall respectively), largely reflecting the accompanying reduction in the amplitudes of SL responses.

Thus, clear similarities in velocity dependence existed for the reflexes evoked by tendon and wrist extension. Two obvious quantitative differences, however, were

found between the responses to these respective stimulus modes. Firstly, both SL and LL reflexes tended to be larger when elicited by wrist than by tendon extension ($P < 0{\cdot}01$, Mann–Whitney U test). Secondly, the times of termination of LL reflexes were on average greater ($P < 0{\cdot}01$, t test), and thus responses more prolonged, when generated by wrist compared to tendon extension. LL responses to tendon extension were generally briefer and more synchronous even when the period of phasic tendon extension outlasted that of wrist extension (Fig. 2*A*, middle and bottom records *versus* Fig. 2*B*, top record).

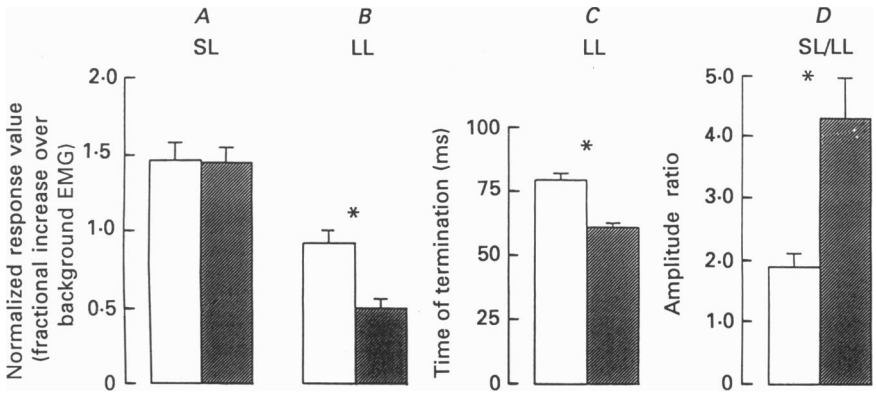

Fig. 5. Comparison of the relative mean (±S.E.M.) amplitudes (*B*) and times of termination (*C*) of LL responses evoked by wrist extension (open areas) and tendon extension (hatched areas) for reflexes in which SL response amplitudes were matched to within 10% (see *A*). The corresponding SL/LL amplitude ratios are shown in *D*. Analysis was based on thirty-five paired observations made in seventeen subjects. Asterisks indicate differences which are significant at the 1% level (amplitudes, Wilcoxon; times of termination, paired t test).

In order to allow a better comparison between the LL responses evoked by the two stimulus modes in individual subjects, stimulus parameters (velocity and amplitude of extension) were varied to match the sizes of SL reflex components. Under these circumstances, inequalities in motoneuronal refractoriness and recurrent inhibition will be minimized. A rate of wrist extension, most commonly around 150 mm s^{-1}, was first selected which elicited both a moderate-sized SL response and a distinct LL response. The rate of tendon extension was then adjusted, by trial and error, until a value (most commonly around 125 mm s^{-1}) was found which gave an equivalent SL component.

Figure 5 illustrates data from thirty-five paired reflexes, elicited in seventeen subjects by tendon and wrist extension, for which SL responses were matched within 10%.

No significant differences existed between either the mean amplitudes (Wilcoxon matched-pairs test) or the latencies (paired t test) of the SL responses evoked by two types of stimuli. The amplitudes of the LL components of the responses elicited by tendon extensions are, however, on average only 54% of those evoked by wrist

extensions and, thus, significantly smaller ($P < 0{\cdot}01$, Wilcoxon). Correspondingly, the SL/LL response ratios for the tendon extensions are significantly greater ($P < 0{\cdot}01$, Wilcoxon) than their wrist extension counterparts. In addition, the durations of LL responses elicited by tendon extensions were, on average, 88% of those generated by wrist extension and were significantly shorter ($P < 0{\cdot}01$, paired t test).

Thus, tendon and wrist extension each evoke reflexes comprising SL and LL responses, which react in very similar ways to the changes in stimulus velocity. It seems reasonable, therefore, that the SL and LL components produced by tendon extension may be considered to be essentially analagous to those observed following wrist extension and to be largely mediated by corresponding reflex mechanisms.

Use of local anaesthesia of the skin and of ischaemia

The effects of local anaesthesia of the skin over the flexor tendons at the wrist and of anaesthesia of the hand and lower forearm (induced by ischaemia) upon reflexes evoked by tendon and wrist extension were tested for two complementary reasons. The former procedure will minimize any reflex action arising from excitation of cutaneous afferents due to distortion of the skin over the flexor tendons by the probe used to apply extension to the tendon of FCR. The latter procedure will additionally minimize, for both forms of stimulation, any reflex contribution from receptors in the skin of the hand and around the wrist and from joint receptors. Any reflex responses which survive anaesthesia may, therefore, be more securely attributed to intramuscular receptors.

Figure 6*A* illustrates that local anaesthesia of the skin over the flexor tendons failed to produce any appreciable modifications of the reflex patterns evoked either by tendon or wrist extension. The lower ('anaesthesia') recordings were made whilst the skin was insentient to light touch (cotton wool) and pinprick. Under these circumstances subjects could still identify the occurrence of tendon displacement although with less accuracy regarding rate and amplitude.

Similar findings were obtained for the range of velocities (typically 50–200 mm s^{-1} for tendon extension and 100–400 mm s^{-1} for wrist extension) tested in each subject. In the eight subjects investigated, the amplitudes of neither the SL nor the LL responses elicited by either stimulus were significantly altered (Wilcoxon) following anaesthesia. Equally, neither were the onsets of SL responses nor the times of termination of LL responses significantly affected (paired t test).

Figure 6*B* shows the reflexes evoked, in a different subject, before and 60 min after inflation of a narrow blood pressure cuff below the belly of FCR to occlude the arterial supply to distal structures. At the time of the 'ischaemia' recordings cutaneous sensation was absent in the fingers, palm and dorsum of the hand and ventral surface of the lower forearm. Cutaneous sensibility over the dorsal aspects of the wrist area and lower forearm was markedly dulled. Joint position sense was also appreciably impaired so that the speed and extent of passive movements of the wrist could not be accurately identified.

Despite these gross disturbances of cutaneous and joint sensibility, the reflex patterns, including their SL and LL components, evoked by each mode of stimulation showed no obvious change. Similar findings were obtained in a second subject, in whom ischaemia was used to induce a comparable degree of sensory impairment.

In two subjects, with intact cutaneous sensibility, mechanical stimulation was directly applied, using the stimulator probe, to a variety of areas of skin over the carpal bones and along the lateral and medial aspects of the forearm. Particular care was taken to locate and orientate the probe so as to minimize any displacement of nearby muscles. The only indication of any increase in FCR activity, elicited by such stimuli, commenced at about 60 ms. These late peaks, which were always far smaller than the excitory responses to tendon stimulation, were sometimes preceded, at around 40–45 ms, by slight reductions in EMG.

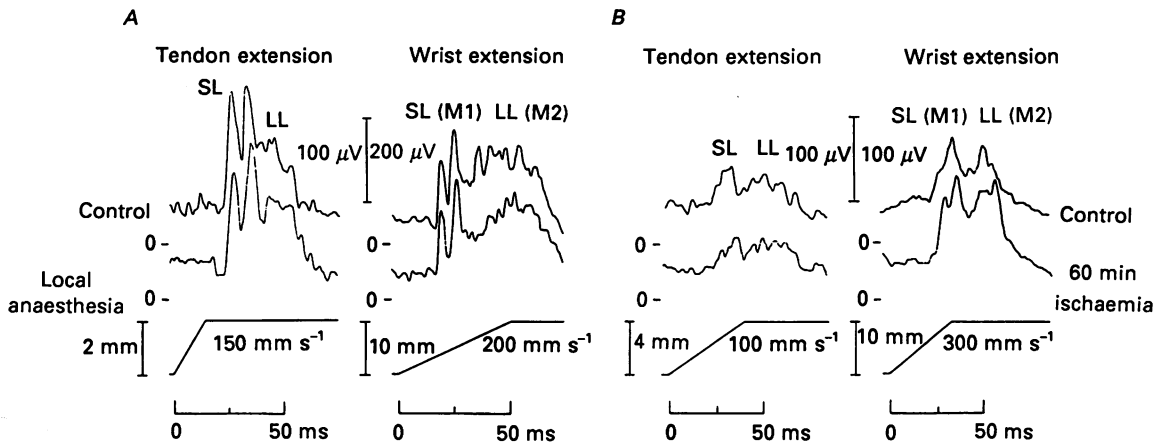

Fig. 6. Effects of local anaesthesia (2% Xylocaine without adrenaline) of an approximately 6 cm^2 area of skin over the flexor tendons (*A*) and of ischaemia of the hand and lower forearm (*B*) upon reflexes evoked in FCR by tendon extension and wrist extension. EMG responses are rectified, averages (sixty-four sweeps; repetition rate, 0·66 s^{-1}). The records obtained with local anaesthesia were from a different subject than those with ischaemia.

DISCUSSION

The present observations raise two principal issues. Firstly, what reflex mechanisms underlie the complex EMG responses, including their SL and LL components, to the relatively simple and discrete stimulus of flexor tendon extension? Secondly, to what extent can similar mechanisms, and especially their afferent components, account for the M1 and M2 reflexes elicited by the less selective stimulus of wrist displacement when a comparatively diverse ensemble of sensory discharge is generated?

Mechanisms underlying reflexes to tendon extension

The fairly prolonged (*ca* 50 ms) EMG responses to 'ramp and hold' tendon extension can be safely regarded as entirely reflex in nature since they terminated before the voluntary reaction time (*ca* 90 ms; Lee & Tatton, 1978) of wrist muscles. Their initial SL component is readily attributable, on the basis of latency and velocity dependence, to spinal reflex actions arising from muscle spindle primary endings located in FCR. The afferent origin and associated neural pathways of the later response peak, which appeared to be a genuinely separate entity, are less clear.

The temporal characteristics, e.g. relatively constant latency and limited duration, of the LL peak suggests that it resulted largely from a rather synchronous afferent volley generated at the onset of the dynamic phase of extension and which operated with little temporal dispersion within the reflex pathway. The relationship between the size of the LL peak and the rate of tendon extension was, in contrast to that of the SL response, not straightforward (see Fig. 4.). It is unlikely that this difference between SL and LL behaviour arose from these responses being mediated by separate subpopulations of motor units with reflex inputs of varying potency, as proposed for M1 and M2 stretch reflexes in the monkey (Bawa & Tatton, 1979). Recent recordings of human FCR motor units have shown that individual units may contribute both to M1 and M2 (Calancie & Bawa, 1985). Instead, the complex LL reflex behaviour, with increasing stimulus strength, probably reflects the counteracting influences of (i) progressively stronger activation of reflex afferents and (ii) greater motoneuronal refractoriness and recurrent inhibition following more pronounced SL discharge (see Matthews, 1984; Cody, Goodwin & Richardson, 1987).

Regarding afferent origin, neither skin nor joint afferents played an essential role in determining the reflex pattern to percutaneous tendon extension. The responses, including both their SL and LL components, were largely unaltered by local anaesthesia of the skin over the flexor tendon and by rendering the wrist and hand insentient during ischaemia. Additionally, the only excitatory changes in EMG elicited by direct activation of cutaneous receptors, upon applying the stimulator probe to various areas of skin over the wrist and forearm, were small and delayed (> 60 ms). Thus, cutaneous afferents, although capable of exerting well-defined reflex actions upon FCR in man when stimulated electrically (Becker, Hayashi, Lee & White, 1986; Cody, Plant & Richardson, 1988*b*), made no substantial contribution in the present context.

This leaves muscle receptors, in the stretched muscle, as the most probable origin of the afferent input responsible for the LL, as well as SL, response to tendon extension. Human M2 responses, elicited by joint displacement, have been alternatively attributed to spindle group Ia (Marsden, Merton & Morton, 1972) and group II (Matthews, 1984) reflex action; both group Ia and group II input, as shown in animal studies, can produce autogenetic excitation whereas the effects of tendon organ input are mainly inhibitory (for review see Baldiserra, Hultborn & Illert, 1981). The present data do not, however, allow any definite discrimination between the two groups of spindle afferents.

The small SL (*ca* 25 ms) reductions in EMG, observed on releasing tendon extension, can confidently be attributed to disfacilitation upon removal of tonic group Ia reflex excitation arising from spindles in FCR. Reciprocal inhibition can be ruled out since there was no accompanying stretch of extensor muscles. The small amplitude and rather limited occurrence of these troughs indicates that tonic group Ia excitatory influences must have been fairly modest under present circumstances.

The absence of an overt LL reduction in EMG upon release of the FCR tendon (and upon flexion movements of the wrist) contrasts with findings for unloading responses, by joint displacement, of muscles controlling the digits (flexor pollicis longus, Marsden, Merton & Morton, 1977; Matthews, 1984; flexor digitorum

profundus, Matthews & Miles, 1988). This difference presumably reflects a variation in the strengths of long-latency reflex action between wrist and finger muscles. For FCR, any small, separate LL depression may have been masked by its merging with the preceding SL response. Additionally, the EMG trough was often terminated by a sharp increase in activity which then rose well above baseline levels. Similar late peaks were also observed in the absence of preceding reductions in EMG so that they cannot be considered to be exclusively 'rebound' discharges. Instead, the steep onset of these delayed peaks suggests a strong active excitatory influence. The origin of such responses, which may be related to the paradoxical shortening contractions originally described by Westphal (1880), is unclear. It may be noted, however, that since only the muscle receptors of FCR are likely to have been appreciably affected during tendon displacement autogenetic reflexes emerge as the most likely explanation.

Comparison with reflexes evoked by joint displacement

The general similarities of the reflex patterns evoked by tendon extension and wrist extension suggest basically common reflex origins. Non-muscular afferent input was not an essential factor in generation of either the M1 or M2 reflexes produced by joint displacement since its elimination, by various anaesthetic procedures, failed to significantly alter these peaks. Thus, intramuscular receptors must be very largely responsible for the M1 and M2 reflexes of FCR as well as the SL and LL responses to extension of its tendon. These findings provide further support for the earlier conclusion that skin and joint afferents make little contribution to the development of M1 and M2 responses in FCR (Bawa & McKenzie, 1981; Cody *et al.* 1986, 1987).

Quantitatively, however, the M1 and M2 responses to wrist extension tended to be larger than their SL and LL counterparts to tendon extension suggesting that the former stimulus was generally more effective in causing muscle stretch. However, LL responses elicited by wrist extension were also usually more pronounced and of longer duration when the parameters of two stimulus modes were adjusted to produce matched SL reflexes. Variations in LL response behaviour, with the two types of stimuli, probably resulted from wrist extension producing not only stretch of FCR but also of its synergists (and simultaneous release of its relaxed antagonists) and thereby having evoked additive, and mainly LL, excitatory (and reciprocal disinhibitory) reflex actions. The finding that extension of FCU, by a probe over its tendon, evoked predominantly LL excitatory responses in FCR supports the hypothesis that autogenetic group Ia spindle reflex action was predominantly responsible for the SL responses of FCR whereas afferent inputs from synergistic muscles made a greater contribution to the development of LL responses.

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