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THE CONTRASTING STRETCH REFLEX RESPONSES OF THE LONG AND SHORT FLEXOR MUSCLES OF THE HUMAN THUMB

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SUMMARY

1. The electromyographic activity of flexors pollicis longus and brevis (with its synergists) has been compared on forcibly extending the thumb at various velocities with the muscles initially contracting. Both muscles gave short- and long-latency responses, but these differed in their relative magnitude with short-latency responses being better developed for the short flexor.

2. With jerk-type stimuli both muscles gave short-latency responses with the expected slight difference in latency due to their different position in the arm. That of the long flexor was sometimes immediately followed by a long-latency response to the same stimulus.

3. With slower displacements the short flexor regularly showed much more short-latency response than did the long flexor. The ensuing long-latency activity of the short flexor was normally appreciably less than that of the long flexor. However, since the short-latency response may be presumed to leave the motoneurones refractory it cannot be definitively concluded from this that acting in isolation long-latency pathways would be less potent for the short flexor, though this seems quite likely to be so.

4. In some cases the first reflex activity occurred nearly synchronously for the two muscles in spite of their different separation from the spinal cord. That for the more distal short flexor was a short-latency response, whereas that for the more proximal long flexor was a long-latency response.

5. The findings conflict with the provisional generalization that for muscles of the primate hand short-latency responses have been regularly supplanted by long-latency responses. They also provide the basis for a teleological argument against the view that the long-latency response is mediated transcortically.

INTRODUCTION

Stretching the human flexor pollicis longus by forcibly extending the thumb elicits a vigorous stretch reflex from it, provided that it is already being voluntarily activated. With high rates of movement its electromyographic response is normally dominated by the initial short-latency reflex, corresponding to the tendon jerk, and this largely bedevils the attempt to define any separate later reflex responses. However, with slow movements the first appreciable response often occurs with a

longer latency (*ca.* 40 ms as opposed to 25 ms). Recent experiments (Matthews, 1984) support the original view of Marsden, Merton & Morton (1976*a*,*b*) that this later response largely depends upon a special 'long-latency' stretch reflex, operating in parallel with but quite distinct from the Ia short-latency pathway, rather than to vagaries in the action of the latter as suggested by others (Eklund, Hagbarth, Hägglund & Wallin, 1982). With intermediate rates of movement both components of response may or may not be readily identifiable. The present experiments extend the previous work by recording simultaneously from flexor pollicis brevis in addition to flexor pollicis longus when they are both stretched at the same time by displacing the thumb without clamping its proximal phalanx so as to restrict the movement to the long flexor, as was done before. Anatomical measurements, given later, show that in spite of the apparent difference in their size the two muscles were now exposed to approximately equivalent rates of stretch, expressed as a proportion of their length.

As already briefly described (Matthews, 1983*b*) the short flexor has been found typically to show a much more vigorous short-latency response and relatively less long-latency response than does the long flexor. The difference between them shows up particularly clearly with lower velocities of stretch when the long flexor frequently fails to show a short-latency response while the short flexor still does so. Such behaviour would be very odd if the long-latency reflex were to be a transcortical reflex, since the cortex would then be involved in mediating an apparently meaningless fragment of the over-all mechanical response of the digit to the disturbance. But, the findings fit readily with the alternative hypothesis that the spindle group II afferents are primarily responsible for the late response (Matthews, 1983*a*, 1984). In addition, the observations conflict with the generalization that the relative importance of short-latency mechanisms for the reflex control of a muscle decreases progressively 'as one proceeds distally in the primate upper limb' (Lenz, Tatton & Tasker, 1983*a*); this view was supported by earlier human work (Marsden *et al.* 1976*b*; Marsden, Rothwell & Day, 1983). However, for reasons that are dealt with in the Discussion it has not proved possible, as was initially hoped, to determine the conduction velocity of the afferents responsible for the later response on the basis of the different separation of the two thumb flexors from the spinal cord. Finally, it should be emphasized that by virtue of using surface electromyography the recorded responses that for simplicity are here attributed to the 'short flexor of the thumb' will have arisen in part from other thenar muscles. Several of these will have been acting synergistically with the true flexor pollicis brevis in producing the initial force and will likewise have been stretched by the thumb displacement. For present purposes, nothing would seem to be lost by lumping these functional flexors together.

METHODS

Fifteen normal adult subjects were studied, of either sex. Most were studied on a single occasion only, but the reflex behaviour of those tested on more than one occasion was reasonably stable, just as before (Matthews, 1984). The methods followed closely those used earlier when further detail was provided (Matthews, 1984). The only difference was that in the present experiments the whole thumb was free to move, whereas in the previous experiments the proximal phalanx was clamped so as to restrict the movement to the terminal phalanx and the interphalangeal joint. In the present experiments the thumb was held nearly straight and, with its tip protruding, inserted loosely into

a cylinder so that little or no movement occurred at the interphalangeal joint. Displacement of the tip of the thumb was then largely taken up by movement at the carpo-metacarpal joint, with the metacarpo-phalangeal joint playing a much smaller part. No attempt was made to determine the precise distribution of the movement between the two joints and it may have varied from subject to subject; this seems immaterial, since movement at either joint affects both flexors. Surface electromyograms were taken from both muscles. The electrodes for the short flexor were placed on the ulnar side of the thenar eminence, about 3 cm apart. They must also have picked up activity from some of the other short thumb muscles; these will also have been co-operating in the initial flexor task, and contributing to the reflex response elicited by displacing the thumb.

The forearm lay horizontally on supports with the thumb uppermost and restrained by a yoke pressing on the wrist from above. The hand was also supported from below; it was slightly dorsiflexed but otherwise in line with the arm. The fingers were loosely flexed. The thumb itself was horizontal, pointing forwards, with its metacarpal bone running upwards at about 30°, but otherwise in line with the arm. The subject pressed down with the pad of his thumb against a large electromagnetic vibrator which was used to apply the mechanical stimuli. With the aid of a visual monitor the subject was instructed to develop a constant force of 6 N (about 20 % of maximal voluntary contraction) in the period between stimuli and to avoid making any voluntary response to their occurrence. They achieved this simply by exerting a constant effort throughout and ignoring the rapid transients with stretch displayed on the monitor, which were in any case too rapid to be tracked visually. The displacements usually lasted 270 ms and were repeated every 800 ms for a period of just over 30 s. They consisted of ramp and hold movements of the thumb of 5 mm amplitude, measured near the base of the nail, and velocities of 50–300 mm s^{-1}. A movement of 1 mm corresponded approximately to a rotation of 0·7° at the carpo-metacarpal joint. The elongation of the muscles produced by these movements is considered in the Results. The response to different velocities of stretching were studied in separate 30 s recording periods, rather than being randomly alternated within a single period. The displacements and resulting e.m.g. responses were recorded for subsequent analysis on a FM tape-recorder, along with various timing signals. The e.m.g. was then full-wave rectified and averaged, usually for 128 successive repetitions of the stimulus from four 30 s recording periods. No smoothing was applied in averaging and the time resolution was set by the bin width employed, which was always below 1 ms.

RESULTS

Response to brief stretch. As illustrated in Fig. 1, for all but one of the fifteen subjects a short-latency tendon jerk-type response could be readily elicited from both muscles by applying a brief rapid displacement to the thumb. The rising phase of the stimulus, which is likely to have been responsible for the observed excitation, lasted just under 7 ms. The whole stimulus was completed within 15 ms, though the movement of the thumb and of its muscles may have lagged slightly behind this. As in Fig. 1, the latency of the response was always some 8 ms less for the long flexor. This is to be expected from its lying some 15 cm closer to the spinal cord than the short flexor so that its conduction pathway is shorter on both its afferent and efferent limbs. Both muscles have the same segmental origin (C8 and T1). Given the relatively slow maximum conduction velocity of human nerves, as opposed for example to those in the cat, the latencies are in accord with the initial part of each response being attributable to autogenetic Ia action elicited by rapid stretch of its own muscle. The sizes of the initial responses are broadly comparable for the two muscles when considered in relation to the initial base-line level of activity, with that of the long flexor being slightly the smaller. The same has been found throughout the series, though no attempt has been made to quantify the matter. Thus with a brief rapid stretch of the muscles there is nothing much of note about the very earliest part of the response.

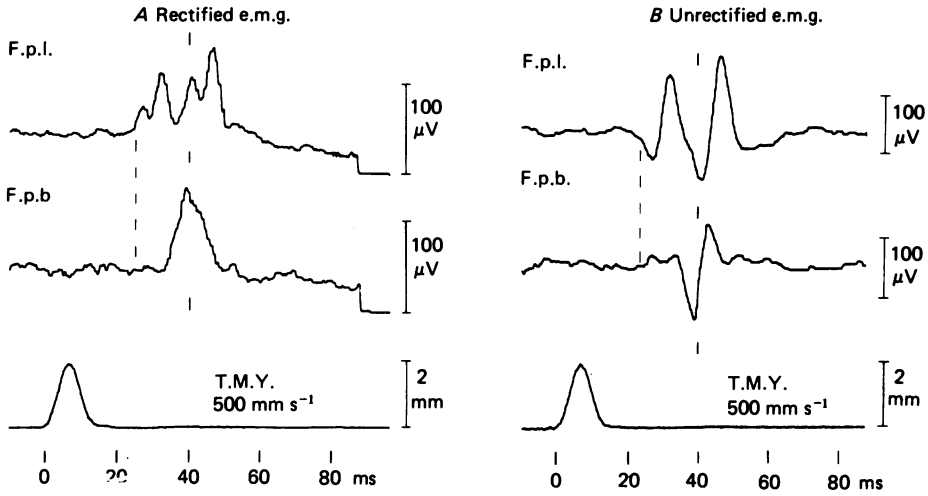

Fig. 1. The e.m.g. responses of flexor pollicis longus (F.p.l.) and flexor pollicis brevis (F.p.b.) to a brief rapid displacement of the thumb, applied at 500 mm s^{-1} for the subject T.M.Y. 1 mm corresponds approximately to 0·7° movement at the carpo-metacarpal joint. 128 responses averaged in *A* after rectifying the e.m.g. (with the zero shown on the right), and in *B* without doing so. Between stimuli the subject exerted a flexion force of 6 N with the pad of her thumb, and she avoided making any voluntary reaction to them. The dashed vertical line indicates the beginning of the first obvious response for the long flexor and is not to be confused with the widely separated dashes at 40 ms from the beginning of the stimulus, which help to assess the latencies of the various responses. Stimuli repeated at 1·25 Hz. Details similar in all subsequent Figures unless specified otherwise.

However, in this subject as in five others there was a marked difference in the duration and wave form of the response, with that for the long flexor lasting nearly twice as long and with the appearance of separate early and late components. This impression is most strongly conveyed by Fig. 1*B* which shows the e.m.g. responses averaged *without rectification*. Observation of individual responses showed that the complex wave form of the average arose from the summation of shorter, diphasic potentials occurring in variable proportion at the two latencies from trial to trial, thus excluding any suggestion that through some oddity of recording the potentials from individual motor units had this prolonged polyphasic form. The second wave in the e.m.g. has a latency of just under 40 ms and is thus correctly located to represent a long-latency response to the brief displacement. It seems unlikely to result from mechanical oscillations of the thumb leading to repetitive Ia short-latency action since the short flexor might then also be expected to show a second response. But this was seen in only one of the six subjects who showed 'double' responses for the long flexor and seems likely to have been due to a long-latency reflex for the short flexor as well as for the long. Another subject showed a 'double' response for the short flexor, but not for the long. The remaining eight did not show convincing double responses for either. The simplest explanation is that in spite of the brevity of the stimulus it may none the less elicit a long-latency response when the reflex mechanisms for this are well developed, which will vary from muscle to muscle and subject to subject, but more usually for the long than for the short flexor of the thumb.

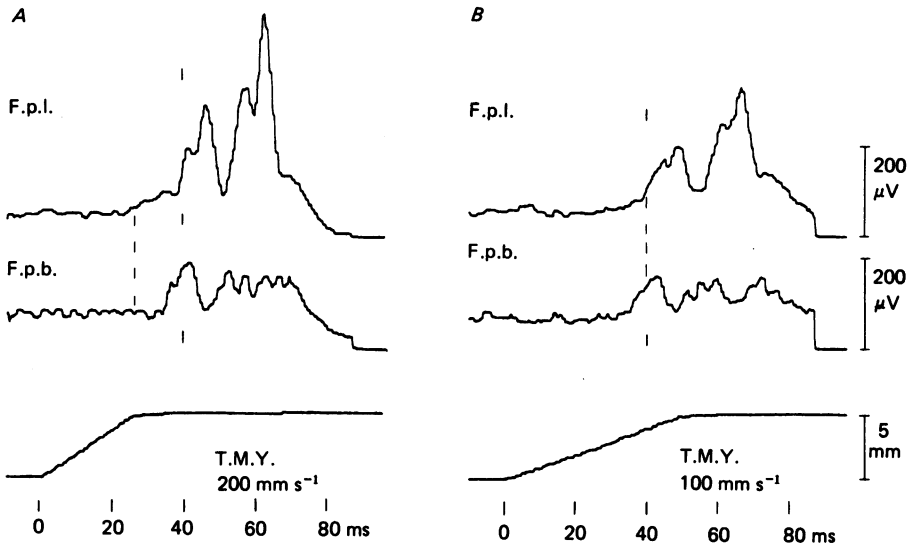

Fig. 2. The rectified e.m.g. responses of the subject of Fig. 1 to slower, larger displacements of the thumb. The lower velocity failed to elicit a short-latency response from the long flexor though it still did so for the short one. The 'noise' in the displacement records is due to digitizing in play-back and was not present in the stimuli themselves.

Response to ramp movements. When displacements were applied at a slower velocity differences were regularly observed in the relative strengths of the short- and long-latency components of response of the two thumb flexors, as illustrated in Fig. 2 for the same subject as in Fig. 1. On displacing the thumb at 200 mm s^{-1} the long flexor showed only a very small short-latency response, which was none the less followed by a well developed long-latency response with a latency of just on 40 ms. On reducing the velocity to 100 mm s^{-1} (right) the short-latency response virtually disappeared, whereas a large long-latency response persisted. In contrast, the short flexor continued to show an appreciable short-latency response for both velocities of stretch, and later components of response were much less prominent than for the long flexor. As before the vertical dashed line shows the first obvious response for the long flexor so that its latency can be compared with that of the short flexor. This helps to highlight an apparent paradox, namely that with 100 mm s^{-1} stretching the first definite response for the long flexor occurs *after* what on cursory inspection might be taken to be the equivalent response for the short flexor. Yet the former lies closer to the spinal cord and so would normally be expected to respond first! As Fig. 3 shows the contrast between their responses was just as great when the *unrectified* electromyogram was averaged.

Two further points in Figs. 2 and 3 merit attention. First, changing the velocity of stretch had relatively little effect on the response of the short flexor. Reducing the velocity to 50 mm s^{-1} had a slight further effect, but an appreciable short-latency response still remained. In some other subjects the effect of changing velocity was appreciably greater, making it unlikely that the response being studied was a non-specific reaction to the movement rather than a continuously graduated stretch

reflex. Secondly, in this subject as in some others the long flexor showed a prominent second 'late' wave with a latency of about 50 ms from the beginning of the stimulus, and which might be suggested to represent yet another type of reflex response and most notably a transcortical one. This cannot be finally excluded, but it seems much more likely to be due to the continued action of the pathways responsible for the 40 ms

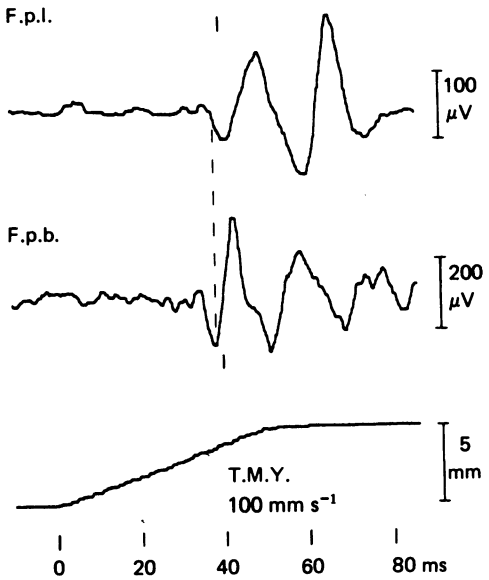

Fig. 3. The unrectified e.m.g. responses from the same raw data as used for Fig. 2*B*.

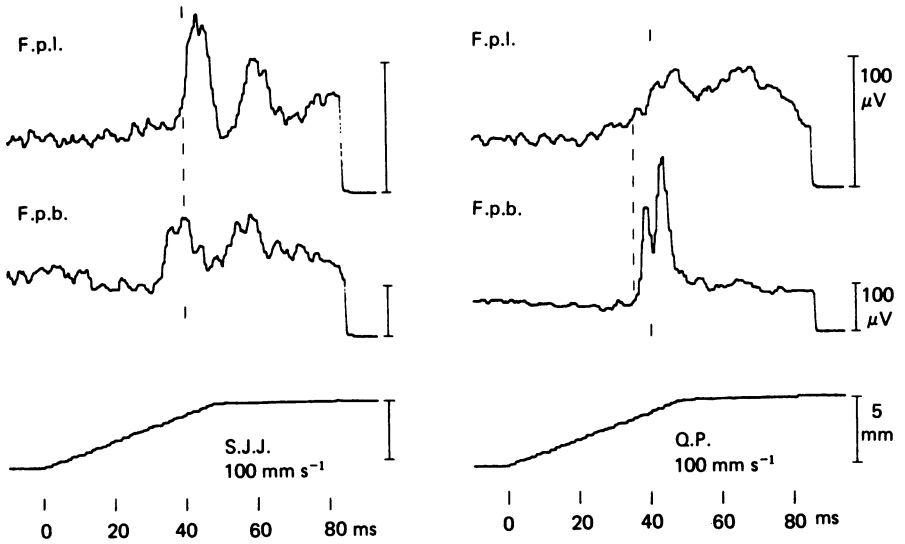

Fig. 4. The rectified e.m.g. responses to thumb displacement at the same velocity as in Fig. 2*B* for two further subjects. The short-latency response is again absent for the long flexor, so that its first obvious response occurs at about the same time as, or lagging behind, that of the short flexor.

response, possibly assisted by a contribution from yet slower afferents. The pronounced 'segmentation' could perfectly well arise from the operation of adventitious factors such as motoneurone rhythmicity and refractoriness and Renshaw inhibition. It should be noted that even with the more rapid stretch the rising phase of the stimulus continues long enough (25 ms) for this to be so, and that the brief rapid stretch evoked no appreciable such late response (Fig. 1). In previous experiments (Matthews, 1984) similar late waves could be abolished by reducing the duration of the rising phase of stretch while maintaining its velocity constant.

Fig. 4 shows the response of two further subjects to 100 mm s^{-1} stretching to demonstrate that the subject so far illustrated (T.M.Y.) was in no way unique. S.J.J. (left) showed a slightly greater excess latency for the long flexor than did T.M.Y. Even the brief rapid stretch failed to evoke an appreciable short-latency response from his long flexor (not illustrated here, but see Matthews, 1984), whereas that for his short flexor was well developed with all the present stimuli. Q.P. (right) showed a particularly large short-latency response for the short flexor with little or no sign of a superadded long-latency response, while for his long flexor the situation was approximately reversed.

Eight of the fifteen subjects studied gave similar responses to those in Figs. 2 and 4; on stretching at 100 mm s^{-1} the first appreciable response of the long flexor occurred at the same time as or just after that of the short flexor. In the remainder the long flexor showed a clear short-latency response (latency around 30 ms), but in only one of these was it larger than that of the short flexor (which in this subject was now the muscle almost lacking a short-latency response). Except for this and one other subject the later components of response were regularly less well developed for the short flexor than for the long flexor, as in Figs. 2 and 4. The same general pattern of behaviour was found on stretching at 50 mm s^{-1}. On stretching at 200 and 300 mm s^{-1} a short-latency response could usually be detected for the long flexor as well as the short, but it was normally not as large. It is concluded that for a range of velocities of stretching the short flexor regularly shows better developed short-latency activity than does the long flexor, and concomitantly usually less long-latency activity. It must, however, be immediately emphasized that this statement does not necessarily apply to the strength of the long-latency excitatory mechanisms considered in isolation. The size of the late 'response' in the two cases will also depend crucially upon the differing extent of the refractoriness and Renshaw inhibition of the motoneurones resulting from differences in the initial short-latency response.

Such comparisons could readily be extended quantitatively, but to do so would achieve little in the face of the other uncertainties involved. Among these is the choice of the time at which to separate the short- from the long-latency components of response for each muscle, and how far the complex temporal structure of the over-all response with its various waves should be attributed to the delayed effect of successive fresh types of reflex action and how far to the complex interplay of spinal mechanisms in the face of continued afferent bombardment (cf. Matthews, 1984). For velocities of stretching of 50–200 mm s^{-1} the following points seem established on the basis of qualitative inspection of the records.

(1) The short-latency response is regularly appreciably larger for the short flexor when expressed as a percentage of the pre-existing base-line activity.

(2) The long-latency component of response assessed over a period of 10 ms starting at 40 ms for the long flexor and 50 ms for the short flexor is regularly somewhat greater for the long flexor.

But the extent to which this is so varies appreciably between subjects, and it tends to vary inversely with the size of the short-latency response.

(3) As a corollary of (1) and (2), the amount of long-latency response relative to that of the short-latency response is appreciably less for the short flexor than for the long flexor.

The particular examples shown provide, it is hoped, sufficient illustration of these generalities; tabular comparisons could well be misleading in the face of the considerable variation in the time course of the response between the two muscles and between subjects.

Responses to vibration. Sinusoidal movement of the thumb at 143 Hz (0·5–1 mm peak-to-peak) elicited a vigorous excitation of both thumb flexors. This began with a short-latency response with the same latency as that elicited by a brief rapid stretch. The wave form of the response was generally similar for the two muscles, with the initial excitation rapidly decaying away by the time of the long-latency response to stretch, so that by then the level of e.m.g. activity was back to or even below the pre-existing level, as already amply illustrated for the long flexor (Matthews, 1984). An 'inhibitory' dip at this time tended to be slightly more prominent for the short flexor. This might or might not be followed by a subsequent wave during the continued vibration, and which in the case of the long flexor requires the continued action of short-latency mechanisms (Matthews, 1984). The size of the initial response tended on average to be slightly greater for the short flexor, but as with the rapid brief stretch the difference was small. Thus again with a sufficiently powerful stimulus the short-latency response of the long flexor can more or less match that of the short flexor.

Previous systematic comparison of the responses to stretch and vibration for the long flexor have led to the suggestion that under the present conditions the spindle group II afferents produce autogenetic excitation and should be held responsible for the long-latency stretch reflex (Matthews, 1984); this is largely absent with vibration which has relatively much less effect on these afferents. When the late response to stretch of the short flexor was reasonably developed, as in Fig. 2, a respectable case could again be made out that for this muscle also the group II afferents were contributing delayed activity to the stretch reflex, and this was so for about a third of the subjects studied. But for the remainder the matter could not be confidently decided since there was insufficient difference in the responses to the two modes of stimulation. An extreme example was provided by Q.P. in Fig. 4, who showed little or no late component in response to stretch. This is not to say that the group II afferents were not having an excitatory action, but merely that its detection inevitably becomes uncertain if it is small and is preceded by a large short-latency response.

It should be noted that the cutaneous effects of vibration might well be much greater for the short flexor than for the long, since with the thumb unclamped there was the most powerful sensation from the whole of the thumb and its surroundings, presumably leading to massive excitation of cutaneous receptors above and around the muscle. Cutaneous receptors above the distant long flexor muscle would be much less affected. In the previous experiments the responses of the long flexor were shown to be independent of receptors in the thumb itself since they were not significantly altered by local anaesthesia of the thumb (Matthews, 1984). A similarly effective control cannot be performed in the present situation. However, for latencies below 60 ms excitation of receptors in the tip of the thumb by stretch or vibration was without significant effect on the short flexor when these stimuli were applied in the usual way but with the proximal phalanx clamped so that the movement was no longer directly transmitted to the muscle and although it was contracting tonically.

Release of stretch. On terminating a stretch, flexor pollicis longus regularly shows a well-marked 'let go' reduction of activity. This occurs with a latency of over 40 ms and thus corresponds to the withdrawal of maintained excitation mediated by the 'long-latency' pathway, while the expected effect of withdrawal of the 'short-latency' component of maintained excitation is normally below the noise level, although detectable for vibration (Matthews, 1984). Such behaviour of the long flexor occurs equally under the present very slightly different conditions. Flexor pollicis brevis generally showed less well developed 'off' responses, and their latency could not always be determined reliably; this was particularly so with vibration. But as with the excitation at the onset of stretch, the reduction in activity of the short flexor often

occurred at very much the same time as did that of the long flexor, and with any difference much less than that seen for their short-latency responses elicited by a jerk-type stretch. Fig. 5 illustrates an example in which the 'let go' responses of the two muscles have an identical latency. Similar responses were obtained in a further seven subjects, though with a tendency for the response of the short flexor to lag slightly

Fig. 5. The responses to 'let go' of the thumb at the end of the stretch (220 ms duration). The consequent reduction in the level of the rectified e.m.g. activity occurs at the same time for both muscles. The gain of the system has been adjusted to make the initial average deflexion approximately the same in the two cases; the calibration bar applies to flexor pollicis longus (F.p.l.) and corresponds to 11 μV for flexor pollicis brevis (F.p.b.). (During the response the lower trace, when they are separate, is that for F.p.l.)

behind that of the long flexor, rather than sometimes being in advance as seen with the excitation produced by stretch. The obvious interpretation of such findings is that the maintained excitatory effect of the short-latency pathways is greater for the short flexor than for the long, as already suggested for the phasic effects at the onset of stretch. The near simultaneity of action of the two flexors would again be quite paradoxical if their responses were held to depend upon precisely the same reflex mechanisms, given their different separation from the spinal cord.

For four of the remaining seven subjects, the latencies of the weak 'let go' effect was not reliably determined for the short flexor. For the other three, however, it was appreciably greater than that for the long flexor with the excess being 10–20 ms, though not always apparently constant from trial to trial perhaps because of the variability in the base line. The form of the 'off' effects of vibration might be complex for the short flexor. No systematic difference was observed between the 'off' effects of stretch and of vibration for the short flexor, but because of the smallness of the effects and the consequent difficulty of measuring their latency no precise comparison was attempted.

In the subject of Fig. 5 short-latency excitation of the short flexor occurred at approximately 40 ms from the onset of a rapid stimulus, so that the latency of the 'off' effect was appreciably longer than that of the corresponding 'on' effect, and this was often so. The same is found with the long flexor (Matthews, 1984). Such differences probably depend largely upon the finite duration of the muscle action potential, but might perhaps also arise from any variation in the population of afferent fibres or of motor units responsible for the various components of response (for example, the earliest part of the 'on' response might depend upon fast motor units which then cease firing).

Extent of muscle elongation

Measurements were made bilaterally upon the prosected arms of an embalmed cadaver to determine the change in muscle length produced by the present stimuli.

With the hand in the same position as that studied physiologically and with the movement largely taken up at the carpo-metacarpal joint a 10 mm extension of the thumb (measured at the base of the nail) produced 1·5 mm movement of the tendon of the long flexor just above the wrist. The same stimulus produced about 1 mm change in length of the short flexor, which agrees with measurements made externally upon the author; these latter were made possible for this muscle by virtue of its lying subcutaneously. The measurements were made with a millimetre scale and were accurate only to about $\pm 0{\cdot}5$ mm, with that for the long flexor being slightly the more accurate since its tendon could be impaled by a needle which then moved directly against the scale; the measurement for the short flexor was made by laying a thread alongside. The length of the actual fibres of the two muscles was very much the same, namely 4–5 cm, in spite of the considerable difference in their gross length. In the short flexor the fibres run parallel along the whole length of the muscle, whereas in the unipennate long flexor they run obliquely and arise in sequence from along a considerable length of the tendon, so that they are individually much shorter than the muscle as a whole.

Thus both in absolute terms and in proportional terms the stretch applied to the long flexor in the present experiments was slightly greater than that applied to the short flexor. The difference would have been yet further accentuated by any yielding at the interphalangeal joint, which was only lightly splinted, since this would have the effect of increasing the displacement applied to the long flexor while reducing that of the short flexor. As an approximation, for both of the thumb flexors the 'standard' displacement of 5 mm at 100 mm s^{-1} (cf. Figs. 2–5) would have produced a 1·5% increase in muscle length at a velocity of 30% of the length of the muscle per second. The stimulus to their muscle spindles, which may be presumed to be responsible for the present reflex effects, should have been very similar for the two muscles, if anything being the greater for those in the long flexor.

DISCUSSION

There is nothing remarkable about the present finding that the relative strengths of the short- and long-latency mechanisms of reflex control should differ for two different muscles. This has already been described for a number of other muscles (for example, Marsden *et al.* 1976*b*; Rack, Ross & Brown, 1978). The interest arises because the two muscles studied were acting directly on the same part of the body and taking an apparently similar part in its control, and from the fact that it was a vital part of the primate hand which was being studied. The findings thus stand against the view that has recently been urged by two separate groups that long-latency mechanisms have largely supplanted short-latency mechanisms in the reflex regulation of the fine voluntary movements of the hand (Lenz *et al.* 1983*a*; Marsden *et al.* 1983). This provisional generalization would appear to have been based upon the study of an insufficient number of muscles. Under the present conditions the short flexor of the thumb, which lies in the hand itself, has frequently shown brisk short-latency responses, and a paucity of long-latency action in a manner which has hitherto been believed to be appropriate for more proximal muscles. In contrast, the more proximal long flexor has shown poor short-latency and good long-latency responses as in all

previous studies with the proximal phalanx clamped. The recently reiterated statement that its short-latency response can 'only be recorded in about 50% of normal human beings' (Marsden *et al.* 1983) is presumably based on studying a more limited range of velocity of movement and thus entirely in line with the present findings.

Since only a single task was studied in the present experiments it is impossible to decide whether the observed differences depended upon inherent differences in the reflex connectivity of the two muscles or whether upon some centrally controllable difference in their reflex set. It might be suggested that one was being used as the prime mover and the other as a postural fixator, since this can influence the type of response observed (Marsden, Merton & Morton, 1981; Cordo & Nashner, 1982). However there was no obvious indication that this was so, or which should be considered which. Rather, both seem to be called upon to play similar roles in the present task in which the subject was simply pressing down with his thumb to produce the required force, and both would seem to be under full voluntary control. The responses were not altered in the one subject tested when the terminal phalanx was held in partial flexion rather than extension. Anatomical measurements exclude the possibility that the greater short-latency response of the short flexor might be due to its being exposed to a relatively more rapid stretch by the present stimuli; if anything, it was the long flexor which was acted upon more powerfully. It thus seems most likely that there is a systematic difference in the reflex wiring or receptor properties of the short and long flexors of the human thumb, with short-latency mechanisms being favoured for the short flexor, just as it seems to be for certain other muscles. Alternatively, it may be that it is the poor development of the short-latency mechanisms for the long flexor that should be regarded as the unusual feature, along with the apparent enhancement of its long-latency mechanisms.

Obstacles to the estimation of the conduction velocity of the afferents responsible for the late response by comparison of the behaviour of different muscles. Marsden *et al.* (1976*b*) have already compared the timing of the responses to stretch of the two thumb flexors in the hope of throwing light upon their afferent origin. In the two subjects studied they noted that 'the results lean towards the slow afferent rather than the transcortical hypothesis' since the difference in latency between the responses they studied was slightly above the value estimated to be required for those dependent upon the fastest afferents. But in the light of other findings and since the differences to be determined 'are at the limit of the technique' they were not 'inclined to give much weight to this'. The original aim of the present experiments was to improve upon the precision of their measurements, but the hope that this would lead to a clear-cut answer was soon abandoned. The reasons for this seem worth discussing in order to prevent premature conclusions being drawn from the sporadic data that already exists in the literature for this and other pairs of muscles. The principles involved at first sight appear disarmingly straightforward. If the late response depends upon the spinal action of slow afferents, and the time is lost peripherally, then the excess of its latency over that of the jerk response should increase in nearly direct proportion to the value of the latter, on considering muscles at different distances from the spinal cord. On the other hand, if the late response is due to the same afferent volley that initiates the early response and the delay occurs centrally, as by transmission of activity to the cortex and back, then its excess latency might be expected to be very much the same for all muscles controlled by the same spinal segments; deviation from this would, at the least, demand some further explanation.

The first problem is to be able to homologize the various separate components of response of the two muscles compared and so be sure that like is compared with like. As will have become amply apparent it is quite insufficient to compare somewhat similar looking waves elicited by an arbitrary velocity of stretching, on the tacit assumption that they will depend upon the same underlying

mechanisms. If this had been done for the initial responses of Figs. 2 and 4 the supposed conduction velocity of the afferents involved would have come out at a negative value, as a result of comparing the short-latency response of one muscle with the long-latency response of the other! But even when one is alert to such gross confusion, considerable difficulties remain, especially when as in the present case the muscles compared differ in the relative strength of their short- and long-latency mechanisms of control. The problem is to identify the beginning of the 'true' long-latency response in each case and to distinguish it from any upswing of activity resulting from continued short-latency excitation, produced by continued fast afferent bombardment, on recovery of the motoneurone pool from its initial discharge with consequent refractoriness and Renshaw inhibition. This can be more confidently achieved with slower stretches, but delays in the build-up of afferent activity might then be suggested to be significant.

In spite of these difficulties, on several occasions equivalent components of the delayed responses of the two muscles were believed to have been identified. But the measurements of delay still failed to fall into a consistent pattern, favouring one or other hypothesis. This led to the tardy realization that an unjustified assumption was involved. It is that the conduction velocity of the slow afferents that are presumed to be involved is precisely the same for different muscles. If the values should differ somewhat, as in the cat (Boyd & Davey, 1968, p. 32), then the extra latency of the second response of the more distal muscle would depend not only upon the time taken for the afferent signals to traverse the segment of nerve between the two muscles, but also upon the differences in time taken by the functionally corresponding afferents from the two muscles to traverse that part of the route which they had in common. Since in the present case the distance between the two muscles is only about a quarter of the shared distance (that from the long flexor to the cord) even a 10–15% difference in conduction velocity could have a crucial effect on the measurements. (On the assumption that the slow afferents conduct at half the velocity of the fast afferents and the motor fibres, then depending upon which set of afferents were the faster, a 10–15% difference would either reduce the difference in latency of the late responses for the two muscles to the value found for their jerk responses, or increase it so that the velocity of the slow afferents would appear to be only half its true value.) Of course, the velocities can still be estimated from the over-all length of the conduction paths for the two muscles, but this is to beg the present question since it involves the assumption that the afferents involved *are* slow, and that the delay arises peripherally rather than centrally.

It should be noted, however, that if the delay should arise centrally and the late response be due to the initial discharge of fast afferents then the second problem vanishes. The excess of latency of the late over the early response should then be independent of the length of the conduction pathway, the velocities of the afferent and efferent fibres involved, and whether or not these values were the same for the two muscles in question. This would not be true if the early and late responses of a given muscle were to be mediated by different subsets of motor fibres with different conduction velocities, as at one time suggested (Bawa & Tatton, 1979), but this is no longer believed to be so for man (Bawa & Calancie, 1983).

A new argument against the transcortical hypothesis based on teleological considerations. The mechanisms underlying the long-latency responses continue to attract debate. The classical view is that they are mediated by a transcortical stretch reflex that is brought into action by the same initial Ia volley that elicits any short-latency response. The later responses may, of course, be supported by continued short-latency action produced by continued Ia bombardment of the motoneurone pool. In the face of accumulating evidence, however, the protagonists of the classical hypothesis now agree that for many muscles (notably those in the leg and upper arm) other mechanisms can be responsible for late responses that are superficially similar to those attributed to the transcortical reflex (Lenz *et al.* 1983*a*, *b*; Marsden *et al.* 1983). But the original hypothesis is still being powerfully defended for the long flexor of the thumb, and the findings generalized into the 'belief that the transcortical stretch reflex mechanism is a system evolved to its greatest extent for the control of the human hand' and that this 'has supplanted spinal stretch reflex mechanisms'

(Marsden *et al.* 1983). The present findings undermine this view and cast doubt on any supposed teleological advantages of transferring most of the machinery for the stretch reflex from the spinal cord to the cortex. From Phillips (1969) onwards this seems to have been felt to allow the cortex to 'dominate' the spinal cord more effectively and to prevent the latter from acting autonomously, presumably so as to improve fine voluntary control by the cortex by putting it in full command of the muscles most directly concerned with delicate movements. The control of postural muscles and so on, however, might more readily be largely entrusted to the spinal cord. But the possible advantages of transferring the main neural machinery for the stretch reflex from the cord to the cortex would seem to fail to be achieved if the translocation were to be seriously incomplete, and the spinal cord left in charge of some muscles that were functionally equivalent to those that the cortex had taken over. Moreover, when several muscles are co-operating in performing some particular task the integration of their behaviour would seem to be made yet more complex if their reflex control mechanisms were to be spatially separated on such a gross scale.

In the present situation the mechanical response of the thumb to disturbance must depend upon the contraction of both of the thumb flexors. It seems pointless for the cortex to be concerned with regulating the response of only one of them, since it would then be dealing with only an apparently meaningless fragment of the over-all mechanical response. For many of the present disturbances, by the time the supposed descending reflex signal from the cortex could have reached the motoneurones of the long flexor those of the short flexor would have already responded, so putting the initial command for mechanical action beyond recall. The inclusion, as must have occurred, of the response of other synergistic thenar muscles along with those of the true flexor pollicis brevis would not seem to alter the case. Even if with subsequent investigation such behaviour should not be found under all conditions, the fact that it does occur in some perfectly physiological circumstances still strikes at the heart of the teleological case for the occurrence of transcortical stretch reflexes.

On the other hand, if the long-latency response depends upon the spinal action of the spindle group II afferents as recently suggested (Matthews, 1984), then the much more mundane question arises as to why the balance between this and the corresponding action of the spindle Ia afferents should differ for different muscles. Nor does it seem at all surprising that this should occur, since in many essentials these two spinal pathways would seem to operate similarly. In the particular present case the difference in reflex balance may be to provide for a greater synchrony of mechanical action of the two muscles than would be achieved if their reflex control were identical. A disturbing mechanical stimulus would otherwise elicit restoring forces from the two muscles with some 8 ms difference in onset which in some cases might prove mechanically deleterious. But since in one apparently normal subject the short-latency response was more poorly developed for the more distant short flexor this would not appear to be an overwhelming consideration in the operation of the reflex control mechanisms.

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REFERENCES

BAWA, P. & CALANCIE, B. M. (1983). Reflex firing pattern of single motor units in human subjects. *J. Physiol.* **345**, 100*P*.

BAWA, P. & TATTON, W. G. (1979). Motor unit responses in muscles stretched by imposed displacements of the monkey wrist. *Exp. Brain Res.* **37**, 417–437.

BOYD, I. A. & DAVEY, M. R. (1968). *Composition of Peripheral Nerves*. Edinburgh: Livingstone.

CORDO, P. J. & NASHNER, L. M. (1982). Properties of postural adjustments associated with rapid arm movements. *J. Neurophysiol.* **47**, 287–302.

EKLUND, G., HAGBARTH, K. E., HÄGGLUND, J. V. & WALLIN, E. U. (1982). The 'late' reflex responses to muscle stretch: the 'resonance hypothesis' *versus* the 'long-loop hypothesis'. *J. Physiol.* **326**, 79–90.

LENZ, F. A., TATTON, W. G. & TASKER, R. R. (1983*a*). Electromyographic response to displacement of different forelimb joints in the squirrel monkey. *J. Neurosci.* **3**, 783–794.

LENZ, F. A., TATTON, W. G. & TASKER, R. R. (1983*b*). The effect of cortical lesions on the electromyographic response to joint displacement in the squirrel monkey forelimb. *J. Neurosci.* **3**, 795–805.

MARSDEN, C. D., MERTON, P. A. & MORTON, H. B. (1976*a*). Servo action in the human thumb. *J. Physiol.* **257**, 1–44.

MARSDEN, C. D., MERTON, P. A. & MORTON, H. B. (1976*b*). Stretch reflex and servo action in a variety of human muscles. *J. Physiol.* **259**, 531–560.

MARSDEN, C. D., MERTON, P. A. & MORTON, H. B. (1981). Human postural responses. *Brain* **104**, 513–534.

MARSDEN, C. D., ROTHWELL, J. C. & DAY, B. L. (1983). Long latency automatic responses to muscle stretch in man: their origins and their function. In *Motor Control Mechanisms in Man: Electrophysiological Methods and Clinical Applications*, ed. DESMEDT, J. E. New York: Raven.

MATTHEWS, P. B. C. (1983*a*). Does the 'long-latency' component of the human stretch reflex depend after all upon spindle secondary afferents? *J. Physiol.* **341**, 16*P*.

MATTHEWS, P. B. C. (1983*b*). Paradoxical timing of the responses to stretch of the long and short flexors of the human thumb. *J. Physiol.* **345**, 99*P*.

MATTHEWS, P. B. C. (1984). Evidence from the use of vibration that the human long-latency stretch reflex depends upon spindle secondary afferents. *J. Physiol.* **348**, 383–415.

PHILLIPS, C. G. (1969). Motor apparatus of the baboon's hand. *Proc. R. Soc.* B **173**, 141–174.

RACK, P. M. H., ROSS, H. F. & BROWN, T. I. H. (1978). Reflex responses during sinusoidal movement of human limbs. In *Cerebral Motor Control in Man: Long Loop Mechanisms*, ed. DESMEDT, J. E. Basel: Karger.