Journal of Physiology (1988), **404**, *pp.* 515–534
With 8 text-figures
Printed in Great Britain

ON THE LONG-LATENCY REFLEX RESPONSES OF THE HUMAN FLEXOR DIGITORUM PROFUNDUS

BY PETER B. C. MATTHEWS AND TIMOTHY S. MILES*
From the University Laboratory of Physiology, Parks Road, Oxford OX1 3PT

(*Received 22 February 1988*)

SUMMARY

1. Electromyography (surface and intramuscular) has been used to study the reflex responses of the human flexor digitorum profundus (FDP) to angular rotation of the distal interphalangeal joint of the 4th finger. This has been done with the hand in three separate positions which, owing to the arrangement of the various tendons, allow the movement to be transmitted to (a) both the flexor and extensor muscles, (b) FDP alone (extensors disengaged) and (c) neither flexor nor extensor muscles (all muscles disengaged, but cutaneous and joint receptors still potentially activated). The stimuli were applied while the subject was voluntarily contracting FDP to produce a constant level of EMG activity; this remained possible when the muscle was disengaged from the joint.

2. With all muscles connected, FDP behaved similarly to the analogous long flexor of the thumb. 'Stretch' elicited a prolonged complex response starting with a short-latency component corresponding to the tendon jerk. Unloading of the contracting muscle caused a pronounced reduction of its on-going EMG activity. The latency of this latter effect was approximately 20 ms greater than that of the initial stretch-evoked response, thereby demonstrating that it was not due to a disfacilitation via the short-latency pathway (on reduction of the tonic spindle afferent firing from FDP as it shortened).

3. With all muscles disengaged, movement of the joint in either direction evoked simply a weak, variable excitatory response, with a latency somewhat greater than that of the normal unloading response. This was attributed to the activation of cutaneous and/or joint receptors. The effectiveness of the disengagement of the flexor was demonstrated by the abolition of its normal stretch-evoked short-latency response.

4. With the flexor engaged and the extensors disengaged both stretch and release evoked their normal types of response. In control experiments, surface EMG recordings from the interosseus muscles confirmed that the procedure used for extensor disconnection was effective. These findings exclude the possibility that the reduction of EMG activity of the unloading response of FDP might be attributable to an inhibition evoked by the concomitant stretch of its antagonists.

5. The long-latency unloading response (whether with the extensors engaged or

* Present address: Department of Physiology, The University of Adelaide, Adelaide 5001, Australia.

disengaged) remained when the sensory receptors in the finger itself were inactivated, confirming that these were not responsible. This was seen both when the finger was ring-blocked with local anaesthetic and when the whole hand was made insentient by occluding its circulation at the wrist and so also paralysing the intrinsic hand muscles. The stretch response was also relatively unaffected by these procedures except that its later components, at the time of putative digital afferent reflexes, might be reduced.

6. It is concluded that the long-latency unloading response is primarily a disfacilitation due to a reduction of the tonic afferent input to a long-latency 'stretch' reflex arising from the muscle itself.

INTRODUCTION

The extensive work of Marsden, Merton and Morton and their subsequent collaborators strongly argues that the long flexor of the human thumb has a specific 'long-latency' stretch reflex utilizing neural pathways which differ, at least in part, from those responsible for the familiar short-latency tendon jerk (Marsden, Merton & Morton, 1976; Marsden, Rothwell & Day, 1983). Whether a specific such reflex exists, and if so which pathways are involved, remains the subject of extensive debate for this and other muscles. Several distinct mechanisms probably contribute to the observed responses with their relative importance varying from muscle to muscle, so that each case must be taken on its own.

Matthews (1984*a*) supported the original view of the existence of a specific long-latency reflex arising from the long thumb flexor, while taking his own stand on its genesis. A feature on which he laid particular emphasis was the reduction of electromyographic activity seen on rotating the terminal joint of the thumb so as to release the already contracting long flexor. This unloading response is classically attributed to a disfacilitation occurring on removing a tonic reflex drive, as a result of a reduction of an on-going spindle afferent discharge. For many muscles the response occurs with a latency comparable to that of the tendon jerk and so is normally ascribed to reduced Ia activation of their motoneurones via a mono- or oligosynaptic spinal pathway. However, the unloading response of the long thumb flexor occurs too late for this to be so, and it has thus been attributed to a disfacilitation of the 'long-latency' stretch reflex pathway; the absence of an appreciable short-latency effect remains puzzling, especially since it is well developed for the short thumb flexor (Matthews, 1984*b*). An alternative possibility is that the delayed reduction of EMG activity represents an active inhibition rather than a disfacilitation, though certain observations already argue against this (Matthews, 1984*a*).

The most probable source of an inhibition is the activation of receptors lying in antagonist muscles, since the release of a given muscle is normally accompanied by stretch of its antagonist. However, unloading responses may persist for triceps surae and the elbow extensors after anaesthetizing the nerves to their antagonists (Herman & Mayer, 1969; Angel, Garland & Moore, 1973); Hagbarth (1967) has also noted that they can still be seen in patients in whom the antagonists have been denervated, although the muscles involved were not then specified. Thus it is unlikely that the

usual short-latency unloading response can depend at all crucially upon inhibition from the antagonist. But these early observations cannot be taken to resolve the origin of the long-latency unloading response, since at the time they were made this had yet to be recognized.

The present experiments attack the problem by taking advantage of the long-known anatomical fact that when the hand is fixed in certain positions it becomes impossible to move the terminal joint of the 4th finger (or alternatively of the 3rd), as well described in Gray's Anatomy (Williams & Warwick, 1980). This arises because of the arrangement and interconnections of the various tendons which has the effect of 'disengaging' the terminal part of the tendons and their insertions at the joint from the main muscle. This allows an experimenter to rotate the distal interphalangeal joint without the movement being transmitted to its controlling muscles, as has already been extensively exploited in studies of 'position sense' (Goodwin, McCloskey & Matthews, 1972; Gandevia & McCloskey, 1976; Gandevia, Hall, McCloskey & Potter, 1983). Three situations are available for study. First, the normal situation in which the sensory input is complete, with both the tendon of flexor digitorum profundus (FDP) and the common extensor expansion engaged (FDP is the sole flexor acting at the distal interphalangeal joint). Second, the extensor contribution can be eliminated by disengaging the extensor tendons while leaving the flexor tendon still engaged to allow the profundus to act on its own. Third, the sensory input can be reduced to that from the joint and the surrounding skin by disengaging both the flexor and the extensor tendons and so eliminating all muscular afferent input; the subject fortunately remains able to contract the disengaged FDP.

It would have been most in line with previous work to have been able to perform the experiments on the thumb, but its movements cannot be restricted in this way. However, FDP has been found to behave very similarly to flexor pollicis longus and to have a comparable, well-developed long-latency unloading response. This persists when the extensors have been disengaged, thus answering the main question as already briefly noted (Matthews & Miles, 1988). It has also been possible to show that the delayed unloading response does not depend upon joint or cutaneous inputs, in accordance with much previous evidence but conflicting with the view (Darton, Lippold, Shahani & Shahani, 1985) that these latter are solely responsible for long-latency effects.

METHODS

A single healthy subject (T.S.M.) was studied in particular detail, with observations made on some twenty separate occasions; this enabled him to become particularly adept at making the correct muscular contraction. The main findings were confirmed on two further subjects, each of whom was studied several times and who both gave the usual responses on the first occasion they were examined showing that these did not depend upon some particular neural set developed by training. The general experimental arrangements were the same as those used in earlier experiments on flexor pollicis longus (Matthews, 1984*a*). A few modifications of detail were required for the present study of FDP, which was upon that part of it which supplies the 4th (ring) finger. All three subjects gave informed consent.

Recording. The electrical activity of FDP was routinely recorded with a pair of 1 cm diameter surface electrodes. They were placed over the prominent bulge of the muscle on the ulnar side of the arm close to the elbow; typically, they lay 6 and 8 cm from the tip of the olecranon and 1·5 cm

from the posterior border of the ulna. As would be expected movement of each finger on its own produced recordable activity from these electrodes, thus showing that the recording was far from selective for any particular head of the muscle. The 4th finger was chosen for study rather than the 3rd, which has been used previously in proprioceptive studies, because flexing it produced larger EMG signals, due to its part of FDP lying more superficially. The activity recorded in this way was believed to come largely from this head of the muscle, because the subject was trying to flex the terminal joint of the 4th finger completely on its own.

Intramuscular recordings were also made on a number of occasions from T.S.M. to ensure that the responses observed with surface recording reliably indicated the activity of the correct part of the FDP. Fine Teflon-insulated silver wires, bared at their tips for up to 3 mm, were introduced into the muscle using a hypodermic needle which was then withdrawn; bipolar recordings were made from a pair of wires the relative position of whose tips was unknown. They were judged to be in the correct part of FDP by virtue of their picking up strong EMG activity when the subject moved the correct finger. Weaker activity might be seen on his moving adjacent fingers, but it was not decided whether this was due to the particular placement of the electrodes or to a failure of the subject to activate the various heads of FDP entirely on their own. The intramuscular electrodes picked up the activity of far more motor units than could be counted, and no one unit ever appeared to dominate the recordings.

After suitable amplification and filtering (band-pass 100 Hz–3 kHz) the EMG was stored on a multi-channel tape-recorder for subsequent analysis, along with timing pulses and the stimulus waveform. On play-back the EMG was full-wave rectified then averaged with a hard-wired averager (Neurolog NL750) for a number of trials of the same stimulus; this analysis was also done 'on-line' to guide the conduct of the experiment. Cusums (Ellaway, 1977) were also determined on occasion using an analog circuit.

Mechanical stimulating arrangements. A servo-controlled, electromagnetic vibrator with position feed-back was used to produce precise displacements of the distal interphalangeal joint of the 4th finger. The arm lay on a horizontal board, with the palm vertical. The middle phalanx of the finger was clamped in fixed relation to the vibrator in a manner which allowed the rest of the hand and the other fingers to be moved, and also the angle of the various joints of the 4th finger itself to be altered. The tip of the finger was held firmly in a clamp attached to the end of the vibrator. This consisted of a 7 mm wide metal strip shaped to the volar surface of the finger, and a post (4 mm diameter) against the middle of the nail; both were covered in resilient plastic. The linear motion of the vibrator was used to produce an angular rotation of the distal interphalangeal joint. There was thus a small shearing force between the clamp and the tip of the finger, possibly producing greater cutaneous excitation than if a pure angular torque had been applied to the finger. A pure torque was applied on occasion by replacing the finger clamp by a simple sling attached to the vibrator and against which the finger pressed; no obvious difference was then seen in the responses.

With the present arrangements, the positions of the two distal phalanges of the finger were kept in a fixed relation to the vibrator throughout the experiment, while the subject adopted the three separate postures required to 'engage' or 'disengage' the flexor and extensor muscles of the terminal interphalangeal joint. The same angular displacement, over the same range, continued to be imposed on the joint. The three postures used were:

(a) All muscles engaged. All fingers, including the 4th, and the wrist were nearly fully extended; it was sometimes found more comfortable to allow the other fingers to flex slightly.

(b) Extensors disengaged, FDP still engaged. First, the proximal interphalangeal joint of the 4th finger was vigorously flexed to its limits, and then the metacarpophalangeal joint of the same finger was flexed as far as was possible without its tip impinging on the palm. The other fingers were moderately well flexed at all joints.

(c) FDP as well as the extensors disengaged. The posture of the 4th finger was kept approximately the same as in (b), while the other fingers were all fully extended.

Maintenance of these postures was assisted by an arrangement of boards and straps which could be changed relatively rapidly. In each of the three subjects they were regularly found to prevent voluntary movement of the distal interphalangeal joint in the direction of action of the disengaged muscle or muscles. For extension, because of the elasticity of the surrounding tissues, this was most convincingly demonstrable for the last half of the range of extension so the stimuli were applied in this range. Thus the same angular displacement could be imposed on the joint in each of the three

experimental conditions, viz. both flexor and extensors engaged, flexor only engaged, or no muscles engaged.

The stimuli were ramp-and-hold movements of 3–6 mm amplitude, applied to the middle of the terminal phalanx of the 4th finger at velocities of 50–300 mm s^{-1}. The most commonly applied displacement of 6 mm at 150 mm s^{-1} corresponded approximately to 24 deg at 600 deg s^{-1} (these and other values given in degrees in the text have all been simplified by being rounded off). The stimuli were delivered regularly, at an interval of 411 ms for those acting to stretch FDP and of 811 ms for those releasing it; the initial position was restored within 200 ms of the beginning of the stimulus. The reflex pathways involved are unlikely to have returned to their 'resting state' in the interval between stimuli, but were probably in a steady state; normally 255 successive responses were averaged. The subject's task was to flex the terminal interphalangeal joint so as to maintain a steady mean level of EMG activity; this was displayed on a monitor oscilloscope after heavy smoothing (0·5 s time constant). The level of contraction chosen was that which could be maintained comfortably without undue effort when FDP was engaged, and when tested produced 10–20% of the maximum voluntary contraction force. Similar results were obtained with lower 'target' forces; higher forces were not used because of the risk of fatigue. (In our initial experiments, which gave similar results, the subject's task was to maintain a constant finger pressure.) When surface and intramuscular recordings were both available the intramuscular record was usually employed in preference (especially when the finger was anaesthetized as making the requisite limited movement then became more difficult), but no differences were found on alternating between them. The subject avoided responding voluntarily to the individual stimuli; this proved easy to do by making a steady effort and paying no particular attention to them.

Anaesthesia. In five experiments on T.S.M. the afferent inputs from the hand and/or the 4th finger were inactivated while preserving those from FDP itself. In three of these experiments the whole hand was made anaesthetic by inflating a pressure cuff around the wrist to well above the arterial pressure to produce anaesthesia by occluding the circulation (the pressures used of 180–200 mmHg might also have produced some direct pressure block of the underlying nerves). Anaesthesia with associated paralysis of the intrinsic hand muscles began to develop after half an hour of occlusion and was complete after about 50 min (except perhaps for a vague, poorly localized sensation of discomfort on firm squeezing). The cuff was left on for 1–1·5 h; apparently complete recovery occurred within a few minutes of releasing the pressure. In two experiments the 4th finger was ring-blocked by injecting 2 ml of 2% lignocaine (without vasoconstrictor) into each side at its base. The duration of anaesthesia was prolonged by winding a rubber band around its base to impede the circulation; it was released intermittently to prevent major local anoxia. On the first occasion this was done a narrow band was applied tightly and led to slight local sensory impairment for the subsequent few days; this did not occur on repeating the experiment with a broader band (5 mm wide).

RESULTS

'Normal' responses of FDP, with all muscles connected

Figure 1 illustrates the reflex responses of flexor digitorum profundus to rotation of the terminal interphalangeal joint of the 4th finger while all muscles were engaged. The subject was voluntarily flexing the joint to press the pad of the finger against the 'stretcher'; the middle phalanx was clamped. Rotation in the direction required to stretch FDP evoked an increase of activity, while that acting to release the already contracting muscle markedly reduced it. The records at the top were taken with surface electrodes while those at the bottom were recorded simultaneously with fine wires inserted into the portion of FDP supplying the 4th finger. The initial part of the stretch response has a latency of 34 ms. This value is about 5 ms longer than that found in other experiments on the same subject on flicking the finger (10 deg movement in 5 ms) and may equally be attributed to Ia-evoked short-latency spinal action. The difference between the two values seems likely to be due to the slower stretch eliciting a less-synchronous afferent volley. The response then continues for

some 50 ms with progressive increase in amplitude, and with the intramuscular record showing marked segmentation; some part of this prolonged response seems likely to be due to 'long-latency' reflex action over and above any continued short-latency action. The latency of the second stretch-evoked wave in Fig. 1 is just slightly less (about 5 ms) than that of the unloading response; although common, such approximate temporal correspondence between major components of the two types of response was not invariable.

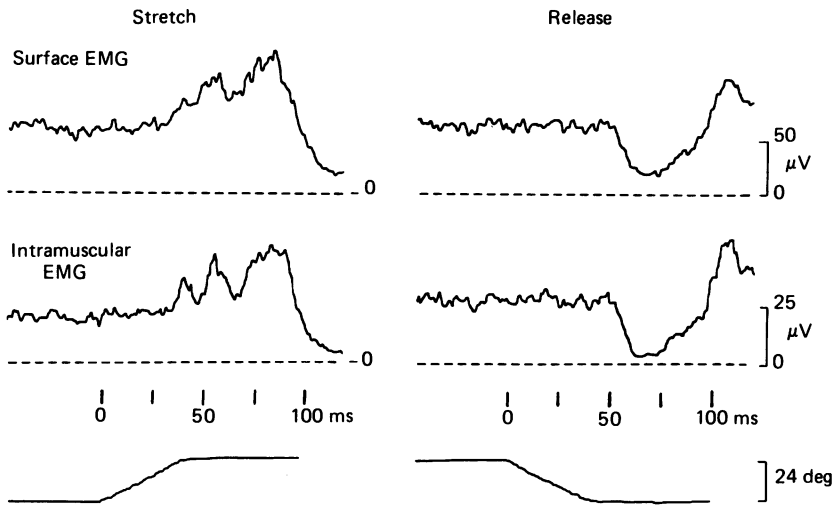

Fig. 1. Electromyographic responses of flexor digitorum profundus to 'stretch' (left) and to 'release' (right), on rotating the distal interphalangeal joint of the 4th finger while the muscle was contracting. Both FDP itself and its antagonists were 'engaged' and so directly affected by the applied movement. Top, averaged, rectified EMG recorded with surface electrodes ($n = 255$). Bottom, EMG simultaneously recorded with fine wires inserted percutaneously into the part of the muscle acting upon the finger studied. The subject (T.S.M.) was trying to maintain a constant mean level of EMG activity, recorded intramuscularly, and was avoiding responding voluntarily to the disturbance. The irregularities on the position recordings are due to digitizing on play-back and were not present on an analog display.

The unloading response of Fig. 1 has a typically abrupt onset, but its latency is 53 ms, and so is nearly 20 ms greater than that of the initial stretch response. This seems too much for it to be attributable to a reduction of activity in the classical Ia spinal stretch reflex. Instead, it may be reasonably equated with the more fully studied, delayed, unloading response of the long thumb flexor and attributed to long-latency reflex action (whether disfacilitation or inhibition). Rather surprisingly, as for the thumb, there is no clear sign of a preceding short-latency unloading response, as was typical for this subject under normal conditions (but see Fig. 8). Of the two other subjects, one again lacked a short-latency unloading response, whereas the other showed a possible small such effect (see Fig. 5, P.B.C.M.). Both gave well-marked long-latency unloading responses at around 50 ms (Fig. 5) and which were again approximately 20 ms later than the initial response to stretch, recorded on the same occasion. The latency difference tended to be slightly less for the smaller subject

(J.M.M., 19 ms) and slightly greater for the larger (P.B.C.M., 23 ms), with that for our usual subject falling in between (T.S.M., 20 ms; these are all modal values rather than means). As for the thumb flexor, the magnitude of the long-latency unloading response merits emphasis. Typically, it was around 50% of the pre-existing level of activity and quite commonly it was appreciably greater, as in Fig. 1.

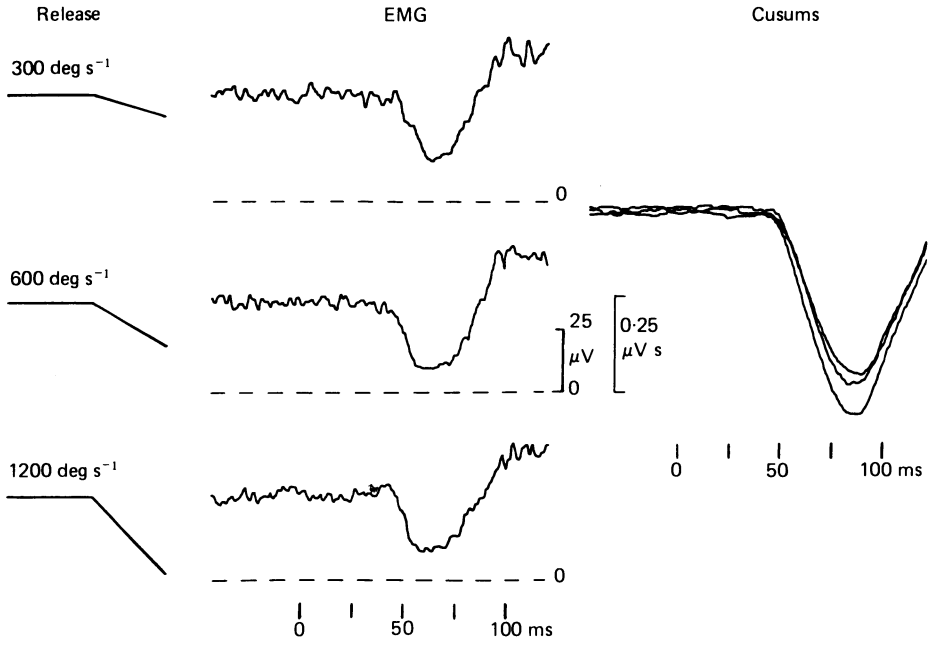

Fig. 2. The effect of varying the velocity of release. Left, the stimuli. Middle, averaged rectified surface EMGs ($n = 255$). Right, their cusums; these have been superimposed to emphasize the constancy of the latency of response. The cusum is the integral of the rectified EMG after subtracting the initial level throughout; thus a deflection in the cusum indicates the sum of the deviations of the EMG from its resting level, up to the time in question. Subject, J.M.M. The stimulus markers in this and all subsequent illustrations have been drawn in, and provide a better representation of the applied movement than digitized recordings like those in Fig. 1.

Type of recording. Intramuscular and surface recordings were compared on several occasions for our main subject to validate the use of surface recordings; these latter were used on their own for the other two subjects. One advantage of the intramuscular recordings is that they provide better temporal resolution and, as in Fig. 1, regularly showed more marked segmentation in stretch records. However, with their slower time course, the unloading responses were much less affected by the 'low-pass' filtering inherent in surface recording, as can again be seen in Fig. 1. Intramuscular recording also provides greater assurance that the observed EMG activity arose from the correct part of the correct muscle. Surface leads are potentially capable of picking up from a wide area and their efficacy for studies such as the present depends upon the subject being able to make an appropriately restricted voluntary contraction. The similarity of the two sets of recordings suggests

that this was achieved. One minor but typical difference was that the cessation of activity during the unloading response was slightly more complete for the intramuscular recordings. This would occur if the surface leads picked up EMG activity from a region of muscle which was contracting, perhaps another head of FDP, but which was not so strongly reflexly influenced by moving the tip of the 4th finger. It was concluded that surface leads provide a suitable signal for the present purposes; none the less, all the present findings have been confirmed with intramuscular recording.

Velocity of release. Figure 2 provides evidence that the long latency of the unloading response of Fig. 1 is indeed intrinsic to the effect and was not due to mechanical lags in transmission of the stimulus to the muscle. The records on the left, taken from another subject, show similar responses to those of Fig. 1 obtained for three different velocities of unloading. They have similar latencies, again of about 50 ms. The constancy of the latency on varying the velocity is demonstrated further by the superimposed records on the right; these are 'cusums' derived from the same data (see legend). Similar results were obtained for all three subjects. Control recordings of the force developed against the stretcher while the muscle was being released showed that the rate of tension decline varied with the velocity, thus strongly suggesting that the rate of shortening of the contracting muscle was also varying. The long latency of the unloading response would thus indeed seem attributable to delays introduced by reflex conduction, as already concluded for the long thumb flexor.

Effect of total disengagement

Residual responses. Figure 3 compares the normal responses with those seen when both flexor and extensor muscles were disengaged and the joint was rotated through the same angle. With the aid of the EMG monitor it still proved possible voluntarily to activate the disengaged flexor muscle, even though it was producing no external mechanical effect. This was rather harder and more tiring to achieve than when it was producing its normal action at the joint. Performance was improved by periodically looking at the tip of the finger and thinking of actively moving it; the task was also appreciably easier while the subject's sensory awareness of the finger was heightened when it was being passively moved in the course of the experiment, as compared with when it was simply being held still. The normal responses in Fig. 3 are very much like those for the same subject in Fig. 1, with contrasting actions of stretch and of release and with latencies of 34 and 53 ms for the first effects of stretch and release respectively. With the muscles disengaged, there is no significant response to be seen before 75 ms when a weak excitation occurred, whichever way the joint was rotated. The abolition of the short-latency response to stretch demonstrates that our particular arrangements had indeed disconnected FDP successfully. The residual excitation may be attributed to the excitation of cutaneous and/or joint receptors and no attempt has been made to separate these. Muscle afferents are thus shown to be responsible not only for the short-latency response but also for the various effects of intermediate latency, including the unloading response, that are found when the muscles are engaged.

In other experiments on the same subject the latency of the residual excitation on

disengaging all muscles varied between 65 and 80 ms, perhaps related to small uncontrolled changes in the stimulating conditions or in the subject's reflex responsiveness. In both other subjects, disconnection of all muscles again left a small delayed residual excitation, whether to stretch or to unloading. For one, the physically larger, the latency was approximately 70 ms; for the other, who was appreciably smaller, the latency varied from 63 to 69 ms. For all three subjects these

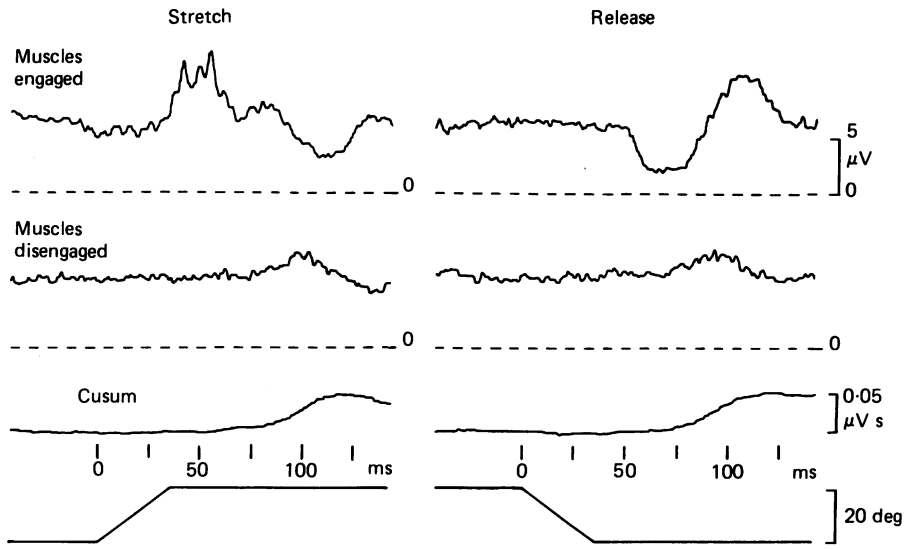

Fig. 3. The effect of 'disengaging' the muscles acting on the terminal phalanx of the finger; rotating the joint should have continued to excite some cutaneous and joint receptors, but should no longer have activated those in muscle. Top, stretch and release responses obtained with all muscles engaged and similar to those in Fig. 1, recorded from the same subject (T.S.M.) on another occasion. Middle, responses elicited on applying the same angular rotation to the terminal interphalangeal joint when both FDP and the extensors had been disengaged by appropriately posturing the hand; the subject was still attempting to flex the joint and so still contracting the disengaged FDP. Bottom, cusums of the middle records to emphasize the small residual response, with a latency of approximately 75 ms. Surface EMG recordings ($n = 510$); the small absolute size of the EMG is adventitious (leakage of electrode jelly; responses with the muscles engaged were also obtained earlier in the experiment, when they were of comparable waveform but of normal absolute voltage).

residual responses were uniformly weak, and rather variable from trial to trial. The examples shown in Fig. 3 are about average size, and their indefinite beginning illustrates the difficulty in measuring the latency precisely. However, with the present mechanical stimuli no sign of a residual excitatory response was ever found before 60 ms. The observed responses do not necessarily represent the minimum latency of the reflex, since subjectively the cutaneous stimuli were weak and considerable central temporal facilitation may have been required to produce a response. However, the minimum values presently observed are similar to that described by Bremmer & Stephens (1985) for the probably analogous E2 response of forearm finger flexors on single-shock stimulation of digital nerves (mean value

65 ms); this appears to be slightly greater than that of the E2 response of the 1st dorsal interosseus (Jenner & Stephens, 1982).

Size of normal cutaneous responses. It might be suggested that the muscular contribution to the normal responses, with the muscles engaged, could be determined simply by subtracting the cutaneous–joint responses seen when the muscles were disengaged, since the rotation applied to the joint was kept constant. However, this was not possible because the mechanical conditions and presumed consequent cutaneous–joint stimulation differed on several counts in the two situations. First, when the contracting flexor was engaged a considerable resting force was exerted on the finger pad, whereas when it was disengaged there was practically none. Consquently the pattern of discharge in the relevant afferents, whether from cutaneous receptors in the finger pad or from capsular receptors of the terminal interphalangeal joint, must be rather different on applying rotation in the two cases; it may be suspected to be much greater when the muscle is producing angular torque at the joint. The same considerations apply to any cutaneous effects elicited by changing pressure on the clamp which held the middle phalanx. Second, when the flexor was disengaged and a rotation was required in the 'release' direction this was achieved by a stop which pressed upon the middle of the nail. However, when the flexor was engaged the finger movement probably occurred largely under the drive being produced by the contracting muscle, without the finger being pushed so hard from behind. Mechanical stimulation of the nail is known to be a potent reflex stimulus (cf. Garnett & Stephens, 1980) and any change in the pattern of this stimulus is likely to produce changes in the reflex response. It should be noted, however, that very similar 'pure' unloading responses, at around 50 ms, were obtained when the nail stop was removed and, with FDP engaged, the joint was flexing solely under the action of the muscle. In addition, it seems quite possible that the effect of a given cutaneous afferent volley might be either facilitated or depressed when it is combined with a normal muscle volley (as when FDP is engaged), in comparison with when it is delivered on its own. But, in spite of these complications, the various responses occurring before about 65 ms can still be reasonably safely attributed to muscle afferents, since we have found no sign of cutaneous–joint effects so early with the stretch parameters currently used.

In line with the above, an appreciable contribution, larger than that shown in Fig. 3, of cutaneous–joint afferents to the later parts of the normal stretch response was strongly suggested by observing the effects of removing all such inputs by anaesthetizing the finger. It was anaesthetized either on its own (by local anaesthetic ring-block) or along with the rest of the hand (by a pressure cuff applied to the wrist); in both cases the afferents from FDP itself were not, of course, directly affected. A striking example is illustrated in Fig. 4 with both intramuscular and surface recording. When the hand was anaesthetic the duration of the response to a standard 'stretch' was appreciably shortened and it terminated at 70–75 ms from the beginning of the stimulus rather than continuing beyond 90 ms; in contrast, its earlier components showed virtually no change. This supports the conclusion that for this muscle, which operates on the densely innervated hand, normal 'stretch' responses typically contain a late contribution from cutaneous–joint receptors, but that this does not manifest itself below a latency of 65–75 ms. The afferents involved

seem likely to be large and fast, because the truncation of the 'stretch' response occurred as soon as the hand became significantly anaesthetic and then showed no further progression. The first of the shortened responses of Fig. 4 was obtained when the hand had only just become anaesthetic to touch, but while the subject was still aware of his finger being squeezed; when the second, virtually identical, such

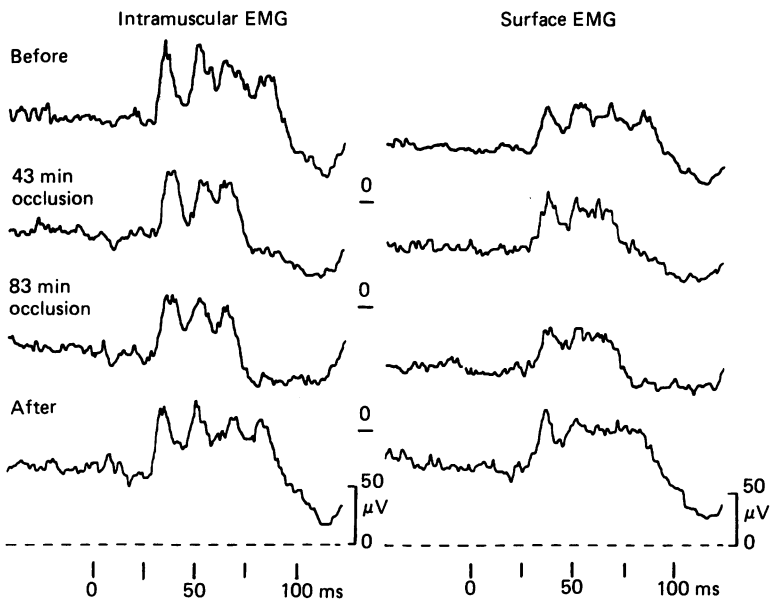

Fig. 4. The effect of anaesthetizing the hand, by occluding its circulation, on the response to 'stretch'. Left, intramuscular recordings; right, concurrent surface recordings (n = 255). Top, before occlusion, showing prolonged segmented response to standard stimulus (24 deg at 600 deg s^{-1}). Middle, responses while hand was anaesthetic after 43 and 83 min of continuous occlusion; the response is markedly shortened and would appear to have lost a component with a latency of 70–75 ms. Bottom, 35 min after circulation restored, with recovery of apparently normal sensation and reappearance of late component. Subject, T.S.M.; extensors disengaged throughout. The intramuscular recording was used as the 'target'.

response was obtained after a further 40 min of circulatory occlusion his hand was totally insentient. Results similar to those of Fig. 4 were obtained when just the finger was anaesthetized, eliminating the remote possibility that the effects of Fig. 4 could have depended upon removing a reflex contribution from the intrinsic hand muscles, especially the lumbricals.

Persistence of unloading response on extensor disengagement

As might be expected, disengaging the extensors had no readily recognizable effect on the 'stretch' responses of FDP; they were not, however, examined in sufficient detail to detect minor changes. The essential observation in the present context is that a typical unloading response was still present when any contribution from stretch of the extensors had been eliminated. Figure 5 illustrates examples from all three subjects, with the normal responses at the top. For T.S.M. (left) the unloading

response was virtually unchanged by extensor disengagement. On some other occasions it was slightly reduced for T.S.M. as is seen here for P.B.C.M. (middle). However, for J.M.M. (right) the response was, as here, typically larger on extensor disengagement. Thus in the series of experiments as a whole there was no consistent change, whether increase or decrease, in the size of the unloading response with disengagement. It may be noticed incidentally that the records for both T.S.M. and J.M.M. show no sign of a short-latency unloading response, whereas there is a

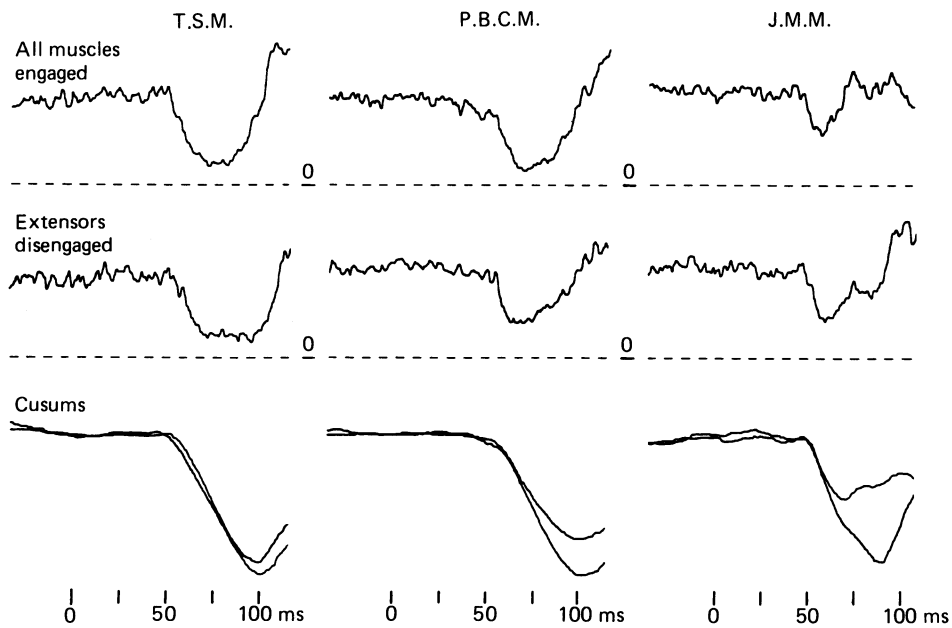

Fig. 5. The persistence of the response to release when the extensor muscles had been disengaged while FDP remained engaged. Top, averaged surface EMGs with the muscles engaged, for each of the three subjects studied. Middle, their responses to the same release with the extensors disengaged. Bottom, superimposed cusums of the responses above. For ease of visual comparison the rectified averages have been normalized so that their initial levels are the same; the absolute voltages were of the same order as those in Figs 1 and 2. (The angular stimuli were, from left to right: 24 deg at 600 deg s^{-1}, 18 deg at 450 deg s^{-1} and 20 deg at 600 deg s^{-1}; the numbers of responses averaged were 220, 255 and 510.)

suspicion of such an effect for P.B.C.M.; this difference between the subjects occurred regularly.

Verification of extensor disengagement. The efficacy of our particular technique for disengaging the extensors is demonstrated by the records in Fig. 6 which were obtained by recording the surface EMG from the intrinsic hand muscles involved in extending the joint studied. In this case the subject was instructed to extend the distal interphalangeal joint rather than flex it, so that the extensor muscles contracted and the direction of joint rotation employed for release of FDP produced a 'stretch' of the contracting muscles; the subject was now provided with a monitor of extensor EMG activity. The electrodes straddled the 4th metacarpal bone with a

longitudinal separation of 2–3 cm so that, under the present conditions, the recorded activity probably arose predominantly from the 4th dorsal interosseus muscle. When the extensors were engaged (top), stretch elicited a complex response starting with a typical short-latency component with a latency comparable to that already described for the 1st dorsal interosseus (Buller, Garnett & Stephens, 1980; Garnett & Stephens, 1980; Jenner & Stephens, 1982); for this subject the latency was remarkably similar to that of the more proximally placed FDP, whereas in the other

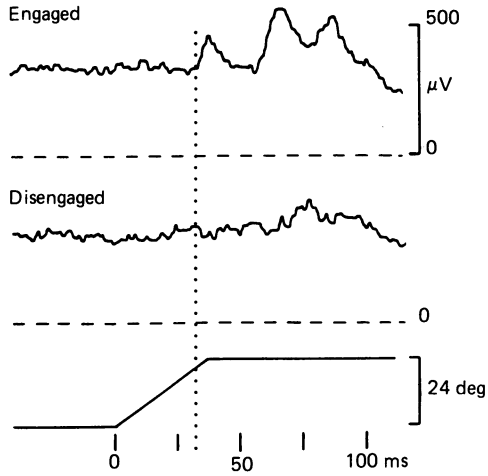

Fig. 6. Demonstration of the effectiveness of extensor disengagement. The EMG averages are now from the *intrinsic hand muscles*, rather than FDP as in all other illustrations, and the subject is endeavouring to *extend* the terminal interphalangeal joint (surface recordings; $n = 510$). Top, response to stretch of these extensor muscles (flexion of the joint) showing a prominent short-latency component with a latency of 33 ms. Bottom, same movement applied to the joint when the extensors were disengaged, but with the subject still contracting them on trying to extend the joint (FDP still engaged, but non-contracting); the short-latency response has been abolished.

subject in which the comparison was made it was about 6 ms greater. Disengagement eliminated the short-latency response, showing that the procedure had indeed been effective, while leaving a miniscule later response which may again be attributed to the excitation of skin and joint receptors. Similar results were obtained in another of the subjects, but the third (P.B.C.M.) had too small a short-latency response from the intrinsic muscles for this control experiment to be usefully performed. However, as for the other two subjects, extensor disengagement could be demonstrated by his inability to extend the terminal interphalangeal joint when the position for extensor disengagement was taken up.

In the two subjects studied electromyographically, disengagement sometimes left a low-level hump at or near the time of the normal short-latency excitation, 10% or below of the normal response, so it is possible that the intrinsic extensors were not invariably totally disengaged; alternatively this could have been equivalent to the short-latency E1 response described on stimulating digital (non-muscular) nerves by Jenner & Stephens (1982). Even if the intrinsic extensors were not always totally disengaged by our manoeuvre when they were contracting, any residual mechanical

effect of joint rotation upon them seems likely to have been still smaller in the normal situation in which they were relaxed and FDP was contracting; the extensor tendons would then have been flacid and so less-able to transmit mechanical stimuli than when they were taut with the extensors contracting.

The intrinsic hand muscles were used for this control experiment rather than the more obvious extensor digitorum, because even when it was indubitably engaged this

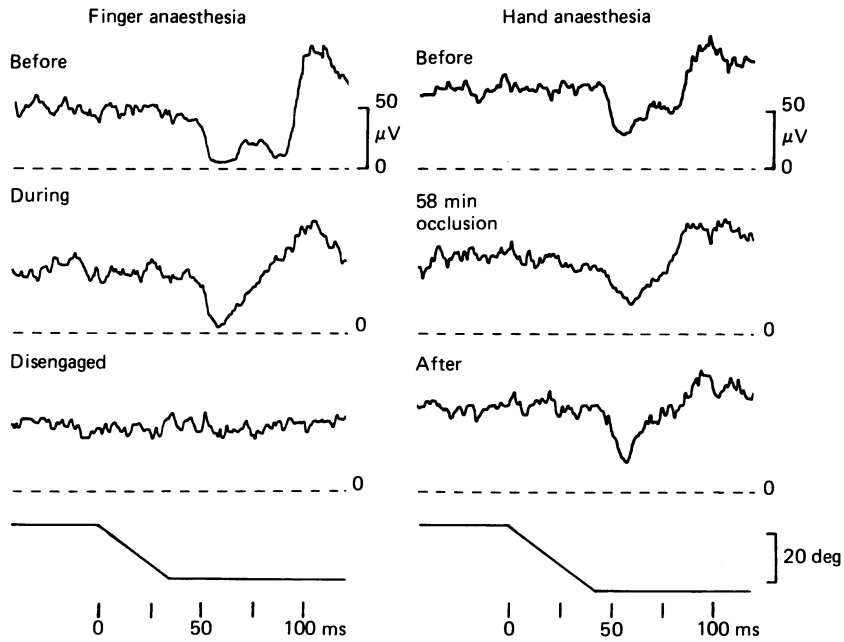

Fig. 7. The persistence of the long-latency 'release' response on removing local afferent input while sparing that from FDP. Left, effect of anaesthetizing the 4th finger with a ring-block at its base. Right, effect of anaesthetizing the whole hand by occluding its circulation at the wrist; this also paralysed its intrinsic muscles. Intramuscular recordings from T.S.M., obtained on two separate occasions, with FDP engaged and the extensors disengaged ($n = 255$). Top, responses to release before anaesthesia. Middle, during anaesthesia. Bottom right, 40 min after restoration of circulation. Bottom left, the total absence of response during anaesthesia when FDP was disengaged as well as the extensors (full recovery occurred only after termination of the experiment).

latter muscle did not show an appreciable short-latency stretch response under our conditions, as required to be able to test for disengagement. However, the interconnections of the various extensor tendons and their common insertion into the dorsal digital expansion should ensure that the control performed on the intrinsic hand muscles applies equally to the extensor digitorum (Williams & Warwick, 1980). It is concluded that a functionally effective extensor disengagement is indeed likely to have been virtually complete, making it highly unlikely that the unloading effects of Fig. 5 could be attributed to failure in this respect.

Persistence of unloading response with anaesthesia

The long-latency unloading response, whether seen with the extensors engaged or disengaged, was still present when the finger (two experiments) or whole hand (three experiments) was made anaesthetic, thereby confirming that it could not be attributed to cutaneous–joint afferents. This is illustrated in Fig. 7 where all the records were obtained with the extensors disconnected. Those on the left were obtained during ring-block of the finger, and those on the right when the hand had been made anaesthetic by occluding the circulation at the wrist. The bottom left record shows again the disappearance of the unloading response on disengaging FDP as well as the extensors (cf. Fig. 3); but now, with the finger anaesthetic, there is no residual delayed response, thus confirming that in the unanaesthetized state this is indeed due to receptors in the finger itself.

The particular virtue of making the whole hand anaesthetic is that as well as eliminating cutaneous and joint afferents it also inactivates afferents from the intrinsic hand muscles. These muscles were confirmed to be paralysed by the inability of the subject to make lateral movements of the fingers and by the disappearance of their normal EMG activity, recorded as for Fig. 6, when finger extension was attempted. There seems no possibility that their afferents could then have remained functional. Thus the persistence of the unloading response as in Fig. 7 confirms that any residual stretch of the interossei could not be crucially involved. More important, it also excludes any suggestion that the lumbrical muscles should be held responsible for the effect. The lumbrical muscles are attached both to the dorsal digital expansion and to the tendons of FDP (Stack, 1962; Williams & Warwick, 1980) and so would still have been stretched on unloading FDP when the extensors were disengaged.

Time course and magnitude of unloading response

The initial falling phase of the unloading response was relatively constant and under normal conditions its onset was usually quite sharp. However, the absolute depth of the response and more particularly the time course of its recovery phase were highly variable for reasons we have not established. This is illustrated by the recordings in Fig. 7 where it can be seen that the recovery phases of the responses obtained before and after the period of circulatory occlusion were quite different, although the conditions were supposedly the same and recovery from the anaesthesia was subjectively complete. Factors which may be expected to affect the time course of the response include the duration of the release (Alston, Angel, Fink & Hofmann, 1967) and the rate at which various motor units happen to be discharging (Miles, Türker & Nordstrom, 1987). In addition, we suspected that a super-added cutaneous reflex at 65–70 ms might introduce a hump into the bottom of the trough, like that seen in the top records of Fig. 7.

Of particular present interest is whether there was a systematic change, whether quantitative or qualitative, in the unloading response on engaging and disengaging the extensors, as would occur if they contributed to the response without being solely responsible for it. Because of the variability, both within and between subjects, this question remains unresolved though we had the qualitative impression that the

unloading response tended to be smaller and shorter when the extensors were disengaged; however, this was not invariable (cf. Fig. 5). We made no attempt to collate the data to provide a statistical description of the average change, because whatever the outcome this would still have left the main question unanswered, namely whether any such alteration was due to disengaging the extensors *per se* or to various other changes which were regularly associated with this (see Discussion).

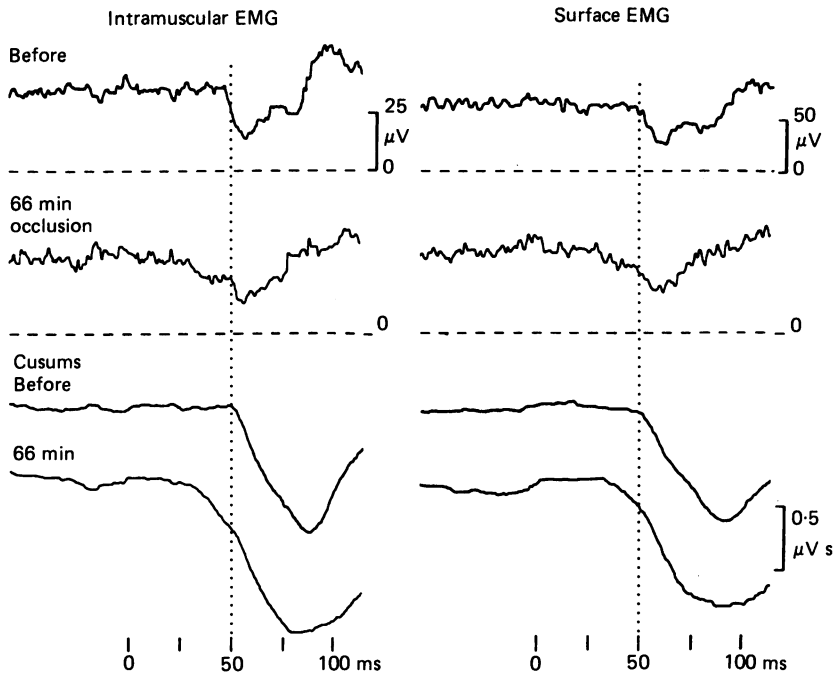

Fig. 8. A short-latency response to release seen when the circulation to the hand had been occluded at the wrist. Left, intramuscular recordings; right, concurrent surface recordings. Top pairs of traces, averaged rectified EMG ($n = 255$); bottom pairs, their cusums. After 66 min occlusion, when the hand was anaesthetic and its intrinsic muscles paralysed, the normal long-latency response at 50 ms is preceded by a weak short-latency reduction of activity with a latency of just over 30 ms. (Same experiment on T.S.M. as in Figs 4 and 7; FDP engaged, extensors disengaged; intramuscular EMG used for 'target'.)

Reflex set. The state or 'set' of the reflex centres may also be presumed to have contributed to the precise form of the observed responses. This view is supported by the observations shown in Fig. 8 which show the emergence of a definite short-latency unloading response in T.S.M. when his hand was made anaesthetic by circulatory occlusion. This change in the pattern of response is best seen in the intramuscular recordings (left), where it is recognizable in the rectified averages as well as in the more discriminating cusums. The short-latency effect starts at just over 30 ms, corresponding to the initial stretch response observed on the same occasion (Fig. 4); it then continues into the usual long-latency unloading response at 50 ms. On recovery from anoxia the short-latency response on release was no longer apparent (not illustrated). The emergence of a short-latency unloading effect can be taken to indicate a change in spinal set, since it seems unlikely that the afferent

discharge would have changed in the requisite manner. The short-latency unloading effect appeared in all three of the occlusion experiments, it emerged only when the hand became anaesthetic, and it was not seen in the two experiments in which just the finger was anaesthetized. This all suggests that the normal absence of a short-latency unloading effect may be due to an active inhibitory process set up by a widespread tonic segmental afferent input.

DISCUSSION

Flexor digitorum profundus has proved to behave very similarly to the previously studied flexor pollicis longus and both differ from the more commonly studied large limb muscles (cf. Marsden *et al.* 1983). This similarity occasions no surprise; both muscles are the sole flexor acting on the terminal phalanx of their own digit, suggesting that they must share many functions. For both, on allowing the contracting muscle to shorten, there is a well-marked unloading reduction of the pre-existing EMG activity with a latency of some 20 ms longer than the initial 'short-latency' response to stretch. Moreover, both normally lack an appreciable short-latency unloading response to mirror the initial excitation on stretch. The present experiments suggest that the absence may be due to the prevailing neural set, since an unequivocal but small short-latency unloading effect appeared when the hand was rendered insentient (Fig. 8); this incidentally provides further evidence against any suggestion that, by virtue of mechanical lags, a normal short-latency unloading response had been mistakenly characterized as a long-latency effect. It would thus appear that both muscles are controlled by a distinct long-latency reflex which may be anticipated to have the same mechanism in the two cases.

On stretch, both muscles show a prolonged complex response with a variable amount of segmentation; this latter was particularly prominent in the present intramuscular records. In some cases there was a marked increase in EMG corresponding approximately in time to the unloading response, and so potentially attributable to the action of the same long-latency pathway; but such temporal correspondence was not invariable. Taken on its own, the segmentation of a response provides rather weak evidence for the existence of multiple reflex pathways since it can have various other causes. The existence of a delayed long-latency reflex is perhaps only convincingly demonstrated when the effect it produces commences *de novo*, without being preceded by overt shorter-latency reflex action; hence the importance of the present unloading responses. Finally, it is noteworthy that in the squirrel monkey also, the deep finger flexor seems to have a prominent long-latency stretch response and a poor short-latency one (Lenz, Tatton & Tasker, 1983).

Analysis of unloading response

The novel feature of the present experiments has been the analysis of the unloading response by mechanically uncoupling the antagonists. This has been possible for the particular muscle studied by making use of the anatomical fact that by appropriately posturing the hand it is possible to functionally disconnect (disengage) some or all of the muscles acting at the terminal interphalangeal joint of the finger. The advantage of this method of eliminating a particular part of the

phasic afferent input normally associated with joint rotation is that it allows the tonic background input to persist. It thus seems relatively unlikely to produce changes in the normal central 'set', and the reflex responsiveness to the remaining components of the phasic afferent volley elicited by the stimulus. The occurrence of such changes is an ever-present risk on completely eliminating a source of afferent input, whether from surrounding skin or antagonistic muscles, by anaesthetizing the appropriate nerves; whatever the means employed, this inevitably abolishes tonic as well as phasic activity. Of course, even with constant set, the residual afferent input need not produce the same effect on its own as it would when combined with the missing phasic input, so the present method cannot be used to fractionate a given reflex arithmetically into its various components; it also has certain other limitations (see later). It has thus been found useful to combine the effects of disengaging muscles with those of local anaesthesia.

Autogenic disfacilitation. The various findings strongly support the view that the unloading response represents a disfacilitation resulting from a reduction of afferent activity from the shortening muscle, rather than an active inhibition from the excitation of other receptors. Cutaneous receptors were excluded on two counts as the prime source of the observed unloading response. First, it was not obtained when the distal interphalangeal joint was rotated while all muscles were disengaged; however, since the mechanical forces involved then differed appreciably from the normal this observation cannot be taken to decide the matter. Second, the unloading response persisted when either the finger or the hand was anaesthetized, confirming sporadic earlier observations for the long thumb flexor (Marsden, Merton & Morton, 1977; P. B. C. Matthews, unpublished; FPL stretch responses are now well known to survive thumb anaesthesia – cf. Matthews, 1984*a*). Stretch receptors in the antagonist muscle are excluded by the simple observation that the unloading response was still present when the extensor muscles had been disengaged while the deep finger flexor remained engaged, and this was equally so when the hand or the finger was anaesthetized. When the whole hand was anaesthetized its intrinsic muscles were paralysed thus excluding any possibility that the lumbrical muscles could have been responsible, as well as yet further excluding the interossei (extensors of the joint studied); the thumb, of course, lacks lumbricals.

It is concluded from all the above that the unloading response is autogenetic and originates from receptors in FDP itself (nearby paciniform corpuscles seem highly unlikely to be implicated, since they would be excited by vibration which signally fails to give a long-latency response; Matthews, 1984*a*). Only three types of muscle receptor are available with sufficiently fast axons to be able to contribute to the response; they are the spindle primary and secondary endings (group Ia and II afferents) and the tendon organs (group Ib). All three reduce their firing when a contracting muscle is allowed to shorten. Thus, without prejudice as to how far each is involved or which particular reflex centres are concerned, it may be concluded that the unloading response is a disfacilitation arising from the interruption of an on-going 'long-latency' stretch reflex. The power of this on-going reflex action is shown by the fact that on its withdrawal virtually all motor discharge may cease for 20 or so ms (present Figs 1, 5 and 7; Fig. 9 in Matthews, 1984*a*), even though the subject is endeavouring to produce a constant level of voluntary drive. Thus the long-latency

reflex contributes tonically during voluntary contraction and is not just a phasic reflex elicited at the commencement of stretch. This is particularly noteworthy if the long-latency reflex should prove to be mediated by the motor cortex since every neurone in the chain, including presumably the corticospinal neurones, would seem to be receiving appreciable steady drive from the periphery during an, apparently, purely voluntary contraction.

Role of other afferents. The residual question is whether the various non-autogenetic inputs which must have been excited by the present stimuli were contributing significantly to the observed responses, even though they have been excluded as being uniquely responsible. Cutaneous and/or joint receptors would certainly seem to be providing an excitatory component with a latency of some 60+ ms to the stretch responses (Figs 3 and 4; cf. Marsden, Merton & Morton, 1985; Matthews, 1987). Such an excitation may also be suspected to be acting against the disfacilitation of the unloading response and so affecting the depth of the trough and the time course of subsequent recovery from it. Given the complex mixture of inhibition and excitation produced on intrinsic hand muscles by digital nerve stimulation (Jenner & Stephens, 1982), yet other effects might well be produced by different modes of mechanical stimulation. However, there seems no possibility that cutaneous afferents can be held uniquely responsible for all long-latency effects, as was suggested by Darton *et al.* (1985); the late excitatory effects that they studied would seem analogous to those currently observed with all the muscles disengaged.

Whether receptors in the antagonistic muscles produced any effect at all must remain an open question. There is no positive evidence that they did so, but the possibility has not been excluded. It may be noted, however, that with the complex anatomy of the hand, none of the extensors acting on the finger functions in mechanical terms as a direct antagonist to FDP. Differences were usually apparent on comparing the responses with the extensors engaged and disengaged, while leaving FDP engaged, but no simple pattern was apparent (Fig. 5). Even if one were to be defined, several potential alternative explanations are available so that the extensors could not be unequivocally held responsible. The most significant of these is that it seems likely that the pattern of spindle afferent firing will differ on allowing FDP to shorten from two different initial lengths of the muscle; the presumption is that the change in firing will be the greater when the muscle is initially the longer in the posture with all muscles engaged. The level of fusimotor activity might well also differ in the two positions, and further affect the response to shortening.

In conclusion, a major part of the long-latency unloading response of the deep finger flexor has been shown to be due to disfacilitation of an on-going long-latency autogenetic stretch reflex. In addition, though it remains unproven, this mechanism seems likely to be wholly responsible for the effect. As a corollary, these conclusions seem likely to apply equally to the similar response of the more widely studied long flexor of the thumb.

The provision of a travel grant to T.S.M. by the British Council is gratefully acknowledged.

REFERENCES

ALSTON, W., ANGEL, R. W., FINK, F. S. & HOFMANN, W. W. (1967). Motor activity following the silent period in human muscle. *Journal of Physiology* **190**, 189–202.

ANGEL, R. W., GARLAND, H. & MOORE, W. (1973). Unloading reflex during block of antagonist muscle nerves. *Electroencephalography and Clinical Neurophysiology* **34**, 303–307.

BREMNER, F. D. & STEPHENS, J. A. (1985). Reflex responses in human forearm finger flexor and extensor muscles produced by digital nerve stimulation. *Journal of Physiology* **369**, 31*P*.

BULLER, N. P., GARNETT, R. & STEPHENS, J. A. (1980). The reflex responses of single motor units in human hand muscles following muscle afferent stimulation. *Journal of Physiology* **303**, 337–349.

DARTON, K., LIPPOLD, O. C. J., SHAHANI, M. & SHAHANI, U. (1985). Long-latency spinal reflexes in humans. *Journal of Neurophysiology* **53**, 1604–1618.

ELLAWAY, P. H. (1977). An application of cumulative sum technique (cusums) to neurophysiology. *Journal of Physiology* **265**, 1–2*P*.

GANDEVIA. S. C., HALL, L. A., MCCLOSKEY, D. I. & POTTER, E. K. (1983). Proprioceptive sensation at the terminal joint of the middle finger. *Journal of Physiology* **335**, 507–517.

GANDEVIA, S. C. & MCCLOSKEY, D. I. (1976). Joint sense, muscle sense, and their combination as position sense, measured at the distal interphalangeal joint of the middle finger. *Journal of Physiology* **260**, 387–407.

GARNETT, R. & STEPHENS, J. A. (1980). The reflex responses of single motor units in human first dorsal interosseus muscle following cutaneous afferent stimulation. *Journal of Physiology* **303**, 351–364.

GOODWIN, G. M., MCCLOSKEY, D. I. & MATTHEWS, P. B. C. (1972). Contribution of muscle afferents to kinaesthesia shown by vibration induced illusions of movement and by the effects of paralysing joint afferents. *Brain* **95**, 705–748.

HAGBARTH, K.-E. (1967). EMG studies of stretch reflex in man. *Electroencephalography and Clinical Neurophysiology* **25**, suppl., 74–79.

HERMAN, R. & MAYER, N. H. (1969). The silent period and control of isometric contraction of the triceps surae muscle. *Electromyography* **9**, 79–84.

JENNER, J. R. & STEPHENS, J. A. (1982). Cutaneous reflex responses and their central nervous pathways studied in man. *Journal of Physiology* **333**, 405–419.

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

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

MARSDEN, C. D., MERTON, P. A. & MORTON, H. B. (1977). The sensory mechanism of servo action in human muscle. *Journal of Physiology* **265**, 521–535.

MARSDEN, C. D., MERTON, P. A. & MORTON, H. B. (1985). New observations on the human stretch reflex. *Journal of Physiology* **360**, 51*P*.

MARSDEN, C. D., ROTHWELL, J. C. & DAY, B. L. (1983). Long latency automatic responses to muscle stretch in man. In *Motor Control Mechanisms in Man: Electrophysiological Methods and Clinical Applications*, ed. DESMEDT, J. E., pp. 509–539. New York: Raven.

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

MATTHEWS, P. B. C. (1984*b*). The contrasting stretch reflex responses of the long and short flexor muscles of the human thumb. *Journal of Physiology* **348**, 545–558.

MATTHEWS, P. B. C. (1987). Effect of arm cooling on long-latency reflex responses from the human first dorsal interosseus muscle – evidence for a group I contribution. *Journal of Physiology* **394**, 102*P*.

MATTHEWS, P. B. C. & MILES, T. S. (1988). Is the 'let go' response of the human flexor digitorum profundus due to stretch of its antagonists? *Journal of Physiology* **401**, 49*P*.

MILES, T. S., TÜRKER, K. S. & NORDSTROM, M. A. (1987). Reflex responses of motor units in human masseter muscle to electrical stimulation of the lip. *Experimental Brain Research* **65**, 331–336.

STACK, G. S. (1962). Muscle function in the fingers. *Journal of Bone and Joint Surgery* **44B**, 899–909.

WILLIAMS, P. L. & WARWICK, R. (1980). *Gray's Anatomy*, 36th edn. Edinburgh: Churchill Livingstone.