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EVIDENCE FROM THE USE OF VIBRATION THAT THE HUMAN LONG-LATENCY STRETCH REFLEX DEPENDS UPON SPINDLE SECONDARY AFFERENTS

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SUMMARY

1. The electromyographic activity of flexor pollicis longus has been recorded in normal human subjects on moving the tip of the thumb with the proximal phalanx clamped. Ramp and hold displacements (stretches) were compared with high-frequency sinusoidal movement (vibration). The subject exerted a constant flexor force between stimuli and made no voluntary response to them.

2. On stretching the muscle by forcibly extending the thumb at various constant velocities the usual combination of short-latency (*ca.* 25–30 ms) and long-latency (*ca.* 40 ms) components of response were observed. The short-latency response progressively predominated as the velocity was increased (60–900 deg s^{-1}, 9 deg joint displacement). One subject still showed only a long-latency response with the fastest stretch, arguing that it is a distinct reflex entity.

3. On commencing vibration (143 Hz, 3 deg movement peak-to-peak) a short-latency response was regularly obtained, but any long-latency response was always small in relation to that elicited by stretch. This was equally so when the short-latency responses to the two types of stimulation were matched by using appropriate parameters of stimulation. The time course of the vibration response did not change appreciably with change of amplitude of vibration, so that its temporal profile was always quite different from that of the stretch response.

4. The observed differences are in accordance with the hypothesis that the spindle group II afferents produce the long-latency excitation, with the time lost peripherally in afferent conduction rather than centrally. In relation to the strength of their Ia excitatory actions, stretch is known to excite secondary afferents more powerfully than does vibration. The findings are not readily accommodated on the hypothesis that the long-latency response is a transcortical reflex elicited by the initial Ia input, since vibration should then also have had a powerful long-latency action.

5. Similar responses to vibration were obtained when it was applied percutaneously to the tendon of flexor pollicis longus 6 cm above the wrist. Also, those elicited by thumb vibration persisted largely unchanged when the thumb was anaesthetized. This confirms that they were dependent upon the excitation of receptors in flexor pollicis longus, presumably the Ia afferents, rather than upon cutaneous or joint receptors in the thumb. The stretch responses also depended upon muscle receptors, since they too survived anaesthesia.

6. On termination of a pre-existing stretch or vibration, differences similar to those seen at their onset were found. Cessation of vibration led to a moderate short-latency reduction of activity and no appreciable extra component of long-latency reduction. Termination of stretch ('let go') normally failed to produce a detectable short-latency effect above the noise, but it was regularly followed by a large long-latency reduction of e.m.g. activity. This powerfully supports the group II hypothesis; these differences, unlike those at the onset of stimulation, cannot readily be explained by postulating appropriately different patterns of Ia firing for stretch and for vibration with corresponding differences in their short-latency reflex actions. The weakness of the short-latency Ia 'off' effects is notable but not entirely surprising.

7. The long-latency reduction of activity seen on releasing stretch was due to a withdrawal of excitation rather than to an inhibition, as might arise from the concomitant stretch of the antagonists. This was shown by comparing the response to ramp stretches lasting 15–30 ms with that to continued stretch. The reduction of activity again occurred with a long latency. But now there was increased activity above the base-line level in the period after the expected cessation of short-latency activity, which can only have been due to continuing long-latency excitation.

8. It is concluded that the only available single unifying explanation for the various findings is that in addition to its short-latency actions stretch evokes a distinct reflex with a longer latency, and that this is attributable to the spindle group II afferents exerting a powerful autogenetic excitatory action.

INTRODUCTION

When a contracting human muscle is stretched its electromyogram shows a series of waves. The earliest or short-latency M 1 response is generally agreed to depend upon the spinal reflex action of the Ia afferents from the spindle primary endings. The immediately ensuing part of the response, occurring well before any voluntary reaction, has been styled the long-latency or M2 component of the stretch reflex and has been a matter of continued controversy. At one extreme, there are those who deny its existence as a separate reflex entity and see it merely as a manifestation of continued Ia spinal reflex action, albeit segmented into waves by various subsidiary factors. Others have urged that it is a long-loop reflex with the Ia activity routed to the motoneurones via the motor cortex with an additional delay of some 10–15 ms over and above that required for their spinal reflex actions. The neural circuitry for such a cortical reflex certainly exists, as shown by animal recording, and operating in parallel with spinal pathways it provides an attractive route for the reflex regulation of muscles of the hand and wrist which are under a high degree of cortical control. However, even among enthusiasts the feeling seems to be spreading that the cortex has less part to play in the reflex control of more proximal muscles, and that segmented reflex responses may also occur for a variety of other reasons. The continuing tangled debate on these matters can be found in several recent reviews (Desmedt, 1978; Evarts & Fromm, 1981; Wiesendanger & Miles, 1982).

The present experiments pursue the matter by using timed trains of high-frequency sinusoidal movement (vibration) as the stimulus in addition to stretching the muscle by the conventional joint rotation of appreciable extent. Moreover, by virtue of the

improved frequency response obtained by using a large vibrator as the mechanical actuator, rather than the more standard torque motor, constant velocity 'stretches' of a better wave form and of a wider than usual range of velocities have been readily available, thus assisting interpretation. The present study has been restricted to the flexor pollicis longus, which is the sole muscle acting to flex the top joint of the thumb and so provides a favourable situation for detailed analysis. This preparation was pioneered and has been extensively exploited by Marsden, Merton & Morton from 1972 onwards, with their numerous subsequent collaborators. Some of their findings on this muscle, moreover, seem to have received scant attention from those who have attacked the suggested existence of a long-latency reflex solely on the basis of results on more proximal muscles in other species, thus leaving it open that at least for the human flexor pollicis longus the case is genuine. Indeed, recent animal work makes it rather unlikely that the genesis of the M2 response can be identical for all muscles (Lenz, Tatton & Tasker, 1983*a*, *b*).

The point about using vibration is that both animal and human recordings show that under isometric conditions it has a far more powerful excitatory action on the Ia afferents than on the group II afferents from the spindle secondary endings (Brown, Engberg & Matthews, 1967; Burke, Hagbarth, Löfstedt & Wallin, 1976). Since both types of afferent are well excited by stretch, even though not identically, there can be little doubt that relative to the activity of the Ia fibres the group II afferents will be excited much more by stretch than by vibration. If the stretch reflex in all its phases depends solely upon the Ia afferent input and the group II input is without effect, then this difference between the afferent composition of the input evoked by stretch and by vibration would be irrelevant. Their reflex effects should be much the same, each with short- and long-latency components of response, and able to be matched by adjusting the relative potency of the two forms of stimulation by varying their velocity and amplitude. In fact, as described in part I of the results, vibration has been found to produce rather little effect at the time when the long-latency response to stretch is appearing, at about 40 ms from the beginning of the stimulus. This makes it very unlikely that the delayed stretch response can be attributed to Ia activity, which had been initiated at the very beginning and then taken a circuitous route to the motoneurones. It is, however, just what is to be expected if the group II afferents with their slower peripheral conduction were to be contributing excitation to the stretch reflex. This idea has been aired before by others, only to be rejected with a greater finality than the evidence warranted (Marsden, Merton & Morton, 1976*b*; Chan, Melvill-Jones, Kearney & Watt, 1969); indeed, some recent workers simply ignore the possibility altogether when debating the origin of long-latency responses.

To further the present case it has proved essential to extend the analysis to the 'off' effect seen on completion of the period of stretch or vibration and now consisting of a reduction of e.m.g. activity. Again, as described in part II of the results, a much greater long-latency effect has been found with stretch as compared to vibration, though the Ia activity may be expected to shut off at the same time in each case. This largely disposes of any suggestion that the differences at the commencement of the response simply reflect different temporal patterns of Ia or other afferent firing elicited by the two modes of stimulation and acting via short-latency purely spinal

pathways. On the present hypothesis, by virtue of the delay in its peripheral transmission, the group II activity set up during stretch continues to reach the spinal cord and maintain the reflex response for a longer period after removal of the stimulus than does the Ia activity.

In essence, the various present experiments combine to support two conclusions. First, following Marsden, Merton & Morton (1976*a*, *b*), flexor pollicis longus does indeed possess a distinct long-latency stretch reflex that is quite separate from the short-latency action of the Ia afferents. Secondly, departing from their view, this reflex is predominantly if not entirely due to the spindle group II afferents producing spinal excitation, with the time lost peripherally through the relative slowness of their conduction rather than centrally through the operation of a transcortical reflex dependent upon the initial Ia discharge. The essentials of this new claim have already been published in brief (Matthews, 1983*b*). However, the present experiments do not aim to, and cannot be taken to, exclude the existence of any contribution from a transcortical reflex to the various later responses. But unless the present evidence can be faulted, probably all the existing human work with mechanical inputs that has been held to favour the long-loop hypothesis seems better explained otherwise, thereby almost entirely removing the experimental basis for the idea that with the growth of his cortex the stretch reflex of man has come to receive an appreciable transcortical contribution, especially for muscles under a high degree of voluntary control.

METHODS

Six normal adult subjects were studied, each on a number of occasions. The present author was investigated in particular detail; consequently it has been found convenient to base a majority of the illustrations upon his data, but nothing so illustrated is unique. Similar responses have been observed to a more limited range of stimuli in another eleven subjects studied in the course of other work in which the whole thumb was free to move (Matthews, 1984).

Mechanical arrangements. The subject sat with the forearm and the ulnar border of the hand resting on supports with the arm horizontal, pointing forwards, and the thumb uppermost. The proximal phalanx was firmly clamped, leaving the thumb free to move only into flexion and extension at the interphalangeal joint; the rounded jaws of the clamp squeezed the phalanx on either side, rather than antero-posteriorly, so as to minimize interference with the relevant tendons and vessels. The fingers were loosely flexed. Fixation of the wrist was provided by a yoke pressing gently from above on the end of the radius. With the thumb flexed about 25 deg the subject pressed down with its pad against a moulded impression mounted on the extended shaft of a large electromagnetic vibrator which was described in detail earlier when it was used for stretching the cat soleus (Goodwin, Hulliger & Matthews, 1975). The externally mounted position transducer used for the servo control also permitted recording of the actual movement as shown in the present illustrations; it was constructed to record faithfully to at least 1 kHz. The motion of the vibrator shaft was linear rather than along an arc centred on the axis of the joint, so that there must have been a small component of movement along the length of the thumb as well as of the desired rotation about the joint. However, with the amplitudes of movement used (maximum 3 mm, 2 cm away from centre of rotation) this will have been below 0·2 mm and did not exert any appreciable effect; it was presumably taken up by the soft tissues of the pad of the thumb. The vibrator was used to apply ramp stretches of up to 3 mm extent at velocities of 20–300 mm s^{-1}, and brief bursts of sinusoidal movement at 143 Hz (7 ms period) at amplitudes up to 1 mm peak-to-peak. Such vibration was made symmetrical about the zero position in the hope of minimizing any excitation of group II afferents. Some must still be presumed to have occurred, not only because of the vibration *per se* but also because it may have produced a small component of steady stretch by virtue of the driving force on the muscle (upward thrust of the vibrator) being more powerful than

the muscles' own visco-elastic restoring force so that the thumb may have tended to be pushed very slightly away from the vibrator when it was moving sinusoidally. Calibrations have been given throughout in mm s^{-1}, since from the way the apparatus was set up this yielded round numbers; 1 mm corresponded approximately to 3 deg, though the precise conversion factor will have varied slightly from subject to subject. These stimuli were usually repeated at 1·25 Hz, though when some of the briefer stimuli were used repeat rates of 2·5 or 5 Hz were employed and still allowed a steady level of electromyographic activity to be restored between stimuli. This steady state may, however, have differed slightly from that which would have obtained if the repeat rate had been lower. The movement of the vibrator was unaffected by the loading applied by the subject and was in any case always recorded. From the subject's point of view the vibrator provided an approximately isometric resistance (stiffness 50 N mm^{-1}).

The subject was instructed to exert a constant force of 6 N with the thumb against the vibrator. This corresponded to approximately 20% of the maximal voluntary contraction and, with the 'standard' thumb length of 2 cm (rotation axis to pad), to a torque of 0·12 Nm. The subject was provided with an oscilloscope display of the force exerted on the vibrator, derived from a separate force transducer mounted on the shaft. In pilot experiments the signal was low-pass filtered to remove the transient changes of force associated with the stimuli, but subsequently the filter was removed and the subject told to maintain a constant force in the interval between stimuli and to avoid any voluntary response to the stimuli. The strategy adopted to perform this task was simply to exert a constant effort, making any slow adjustments required on the basis of the force level between stimuli. This proved easy to do without appreciable training, partly because the force transients associated with the stimuli were too rapid to be tracked visually. Control experiments showed that the results obtained were the same, within experimental limits, as those obtained when the display was low-pass filtered and the same also as those when the subject endeavoured to maintain a constant force while his eyes were shut or he was looking away. The subject maintained the contraction, and the stimuli applied, for 34 s and then rested for 26 s before repeating the cycle. The first 7 s of recording from the beginning of the contraction was discarded and the responses from the ensuing 26 s averaged; the responses to two to eight such periods of stimulation were pooled. Facilities were not available for randomly alternating different types of stimuli and then sorting out their individual responses, but when two particular responses were to be compared their several 1 min periods of data collection were interleaved.

In control experiments the tendon of flexor pollicis longus was vibrated more directly with a separate small vibrator (Goodmans, V 47) applied over the tendon about 6 cm proximal to the wrist. This vibrator was run open-loop, without servo control, but had a similar position transducer mounted on its shaft so that its movement too could be continuously monitored. Vibration that was symmetrical with regard to the zero position was again used; for both vibrators this was generated by an input consisting of an appropriate combination of square-wave pulses, rather than by a sinusoidal wave form. The small vibrator was mounted on a pivoted arm which was counterbalanced by weights so that it was pressed against the arm with a force of 2 N. A tendency for this lever arm to oscillate slightly at the repeat period of the stimulus (usually 1·25 Hz) was counteracted by viscous damping provided by a syringe filled with oil thinned by paraffin. The tip attached to the vibrator was approximately oval in cross-section (6 × 9 mm) and cylindrically rounded in its long axis. It was placed with its long axis along the arm, just lateral to the tendon of flexor carpi radialis and just medial to the radial artery. Percutaneous contact with the tendon was assured by observing movement of the vibrator on its counterbalance when the subject flexed the terminal joint of his thumb.

Recording. The electromyographic activity of flexor pollicus longus was recorded with surface electrodes placed approximately 8 and 12 cm above the wrist crease as described by Marsden *et al.* (1976*a*). After amplification it was stored, along with various marker signals, on an FM tape recorder for subsequent analysis. Together these gave a recording bandpass of 10 Hz to 1·25 kHz. On replay the e.m.g. was full-wave rectified and the response to 64–256 cycles of stimulation averaged with a small hard-wired averager (NL 750; Digitimer). Normally a bin width of 0·8 ms was used; although it was immaterial for most of the present work, the average was actually taken of the integral of the signal throughout the course of the bin rather than just of a momentary sample from within it. No further smoothing on filtering was performed and the total lag in recording was below 1 ms. The output of the averager was displayed on a digitial oscilloscope and from this plotted to give graphic records for study, as in the illustrations. The final stage of digitizing introduced

slight additional noise which is obvious in some of the illustrations of the stimuli which were recorded similarly (but without averaging); these recordings of the actual movement were, in fact, quite smooth when observed on a normal oscilloscope. In many experiments cumulative sums (cusums) were also determined of the deviation of the electromyographic signal from its initial value before the response, as done in single unit studies (Ellaway, 1977). The computation, however, was done by analog rather than by digitial means by feeding the output of the averager into an integrator that was reset at the beginning and provided with a steady bias to counteract the initial level of e.m.g. activity, so that the base line runs flat in the period before the response; the bias was readjusted for each individual cusum. The recordings are thus similar to a single limb of the 'tulips' of Marsden *et al.* (1976*a*) with the initial slope adjusted to zero.

RESULTS

Part I. The effects of onset of stimulation

Response to constant velocity displacement

Fig. 1 illustrates the behaviour of the author who has a brisk short-latency response when the velocity of movement is appropriately high. The initial response then occurs with a latency of about 28 ms and is followed by a complex segmentation, the precise form of which varies with the velocity. At the two lowest velocities there is very little response before 40 ms. In their earliest work using relatively slow displacements Marsden *et al.* (1976*a*) suggested that the early component hardly existed for flexor pollicis longus, but on subsequently using higher velocities of movement and increasing the number of subjects studied they observed it repeatedly (Marsden, Merton, Morton, Adam & Hallett, 1978). Thus the recording of responses broadly like those of Fig. 1 from all but one of the present subjects largely reduplicates their observations, albeit now using a stimulus of more constant velocity and, due to the low inertia of the present system, a slightly more clearly defined moment of starting. Taken on their own, however, it is impossible to decide whether any of the later responses depend upon a specific 'long-latency' reflex.

The delayed response seen with the slow stretches might depend equally upon a slow build-up of afferent activity coupled with a need for central facilitation before a weak input produces an appreciable effect. Others have already attributed the greater delay of the response to parabolic as compared to ramp stretching 'to the soft thumb pad taking up the initial slow movement... before rotation of the joint began' (Marsden, Merton, Morton, Rothwell & Traub, 1981). The segmentation of the responses evoked by the faster stretches can just as well be ascribed to irregularities in the continuing afferent discharge and the complex interplay of excitatory and inhibitory mechanisms within the spinal cord, as to the arrival of delayed reflex activation. The fall-off of the later part of the response with the rapid stretches is presumably due partly to the refractoriness of the motoneurones that have just fired in the early response with accompanying Renshaw inhibition of the others, and partly to the delayed effects of the inevitable rapid reduction of afferent input on completion of the dynamic phase of stretching.

None the less, the findings from one subject in particular suggest that there is indeed a genuine long-latency response, as illustrated in Fig. 2. The response of this subject to the slowest stretch is not very different from that of the subject of Fig. 1, but with a 10-fold increase of velocity the main response still occurs with a latency of about 40 ms rather than falling to about 30 ms. This makes it very unlikely that the delay in the response to the slow stretch is solely due to a progressive slow build-up of excitation, whether peripheral or central. Marsden *et al.* (1976*a*, *b*) also noted that a more restricted change in velocity might have rather little effect on the latency of

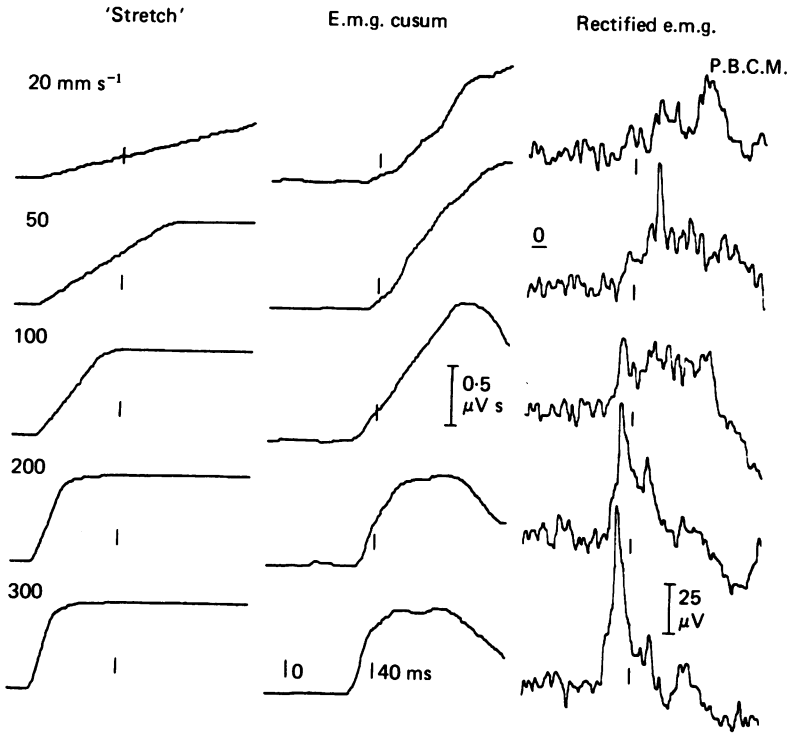

Fig. 1. Electromyographic responses of flexor pollicis longus to ramp 'stretches' of increasing velocity in a subject with a brisk short-latency response. The stretch velocity is given in mm s^{-1} of displacement of the thumb pad at the level of the base of the nail; the movement in millimetres should be multiplied by 3 to convert approximately to degrees angular movement at the interphalangeal joint. Maximum movement, 3 mm. The timing mark in the middle of each record is at 40 ms from the beginning of the stimulus. Each rectified e.m.g. trace is the average of 128 individual responses, repeated at 1·25 Hz (stretch duration, 200 ms). The 'cusum' is the integral of the rectified e.m.g. after subtracting the initial level throughout. The zero level is shown for the top right e.m.g.; all responses started from a similar initial level. Between stimuli the subject exerted a flexion force of 6 N with the pad of his thumb, and he avoided making any voluntary reaction to the stimuli. The irregularities on the position recordings are due to digitizing in playback and were not detectable on an analog display.

a late, but then rapidly developing, response. They likewise considered that this indicated that there is a separate late reflex, with a latency of around 40 ms, quite different from the short-latency reflex corresponding to the tendon jerk.

Within a given experimental session the reflex behaviour of any particular subject remained reasonably stable; similar responses were obtained for a given rate of stretching applied at the beginning and end of a session and when a given set of data was averaged in two or more sub-groups rather than altogether. On different occasions, however, the relative balance between the initial short-latency response and the later responses might alter, for reasons which were not apparent. There was a tendency for the short-latency response to decrease in the later sessions for a given subject, possibly associated with the loss of any apprehension that might have been felt about the experiment. However, any particular subject showed less variation than the population as a whole. For example, the author continues to show brisk short-latency responses after more than thirty

experimental sessions whereas another subject (S.J.J.) continues to be without an appreciable such response over a period of half a year. It may be noted that he possesses clinically normal jerks to routine testing whereas the special subject studied by Marsden *et al.* (1976*b*), who lacked appreciable short-latency action for a variety of muscles, was entirely without tendon jerks. It is believed that S.J.J. merely represents one extreme of the normal range rather than possessing some abnormality.

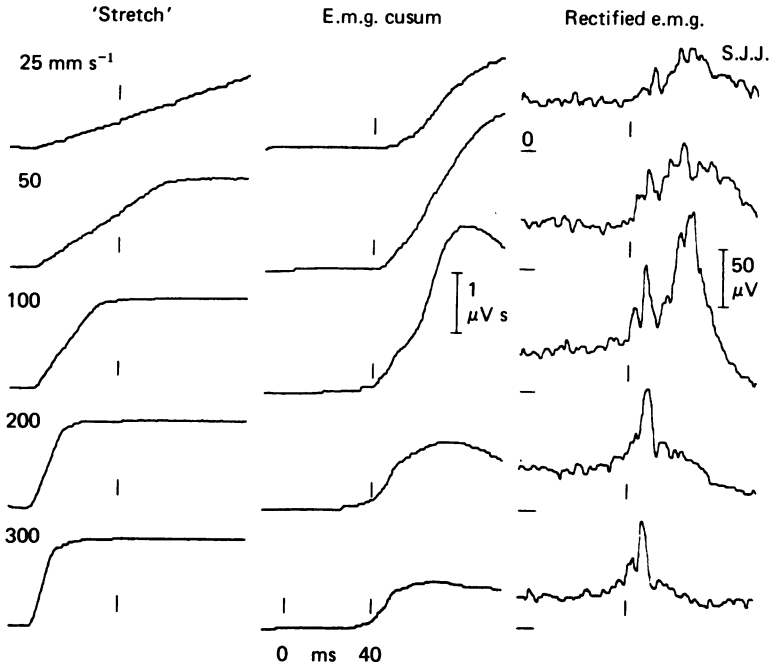

Fig. 2. Responses to ramp 'stretches' in a subject with virtually no short-latency response. Note the timing of the main e.m.g. wave in relation to the 40 ms timing mark. Details as Fig. 1.

Response to vibration

On the transcortical hypothesis the delayed responses of the subject of Fig. 2 are attributed to conduction around a 'long-loop' within the C.N.S. so that the initial Ia activity, set up at the very beginning of the stimulus, takes much longer to reach the motoneurones than it does via the monosynaptic spinal pathway, which for some unknown reason is largely without effect. On this view high-frequency sinusoidal movement of the joint (vibration) should elicit a similar response at 40 ms. However, as illustrated in the top trace of Fig. 3, this was not so. Instead, there is a small response at just under 30 ms followed by a reduction of activity at 40 ms. Comparison with the next trace down shows that up to about 50 ms S.J.J. now had a much smaller but otherwise broadly similar response to that of P.B.C.M. Similar findings were obtained when each subject was studied on different occasions and with different amplitudes of vibration, and with the vibration applied directly to the tendon (see later). The responses to vibration of the other four subjects showed the same general pattern, with an invariable initial short-latency response irrespective of whether or not it was apparent for the lower rates of stretch.

Fig. 4 complements Figs. 1, 2 and 3 by showing some of the same reflex responses to stretch and vibration, but now with the e.m.g. averaged *without rectificaton*; under certain circumstances either method of analysis alone can fail to disclose everything of interest (Evarts & Vaughn, 1978). In this case the two methods of analysis entirely support each other. The response to vibration is again much less for S.J.J. than for P.B.C.M., but the latency and wave form of the response is roughly similar and S.J.J.

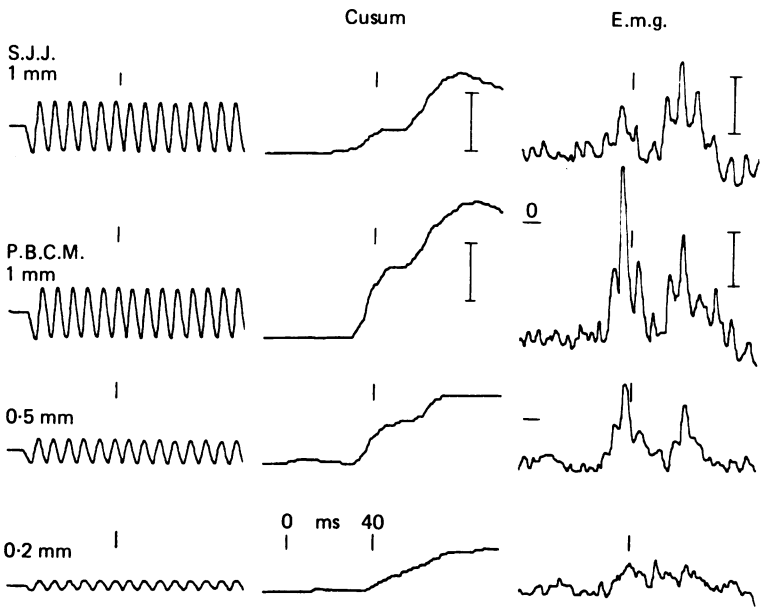

Fig. 3. Responses to vibration applied to the pad of the thumb. The subject of Fig. 2 (S.J.J., top) shows a clear but small short-latency response at about 30 ms, but no ensuing long-latency response at 40 ms; there is, however, a later wave at about 55 ms. The subject of Fig. 1 (P.B.C.M., bottom three responses) shows a much larger short-latency response to vibration, but likewise no build-up of response at 40 ms; moreover, the wave form of the response changes little with variation of stimulus amplitude. Time zero for the stimulus is taken as the beginning of the first stretching (upwards) phase of the vibration. Vibration frequency, 143 Hz (its peak-to-peak amplitude is shown beside each record); 128 responses averaged for each, repeat rate 1·25 Hz; duration of vibration, 200 ms. Calibration bars, 25 μV and 1 μV s.

has a clear, albeit small, short-latency response. The response to a rapid stretch shows much more early activity for P.B.C.M. than for S.J.J., and for S.J.J. there is a large wave starting about 40 ms from the beginning of the stimulus betokening a strong synchronizing influence on his motor discharge. A response at 40 ms is also to be seen for both subjects for the slower stretch. For P.B.C.M. it is preceded by a small short-latency wave, whereas for S.J.J. it is not. These various findings would seem to eliminate the possibility that the conduction velocity of S.S.J.'s Ia afferents was simply below normal, and thereby delayed his Ia spinal stretch. If this had been so, then his response to vibration should have been similarly delayed but of normal size.

The reason for S.J.J.'s deficit of Ia short-latency action for flexor pollicis longus remains obscure. Perhaps a normal population of Ia afferents have certain of their normal connexions missing or

of unduly low potency. Even more simply, the poverty of obvious Ia reflex action might just reflect a relative lack of Ia afferents. In other words, instead of normal afferents simply lacking certain of their spinal connexions it may be the afferent composition of the muscle which differs from that which, on remarkably little evidence, we have come to consider the essential normal complement.

Comparison of the time course of the responses to stretch and to vibration

As will become apparent from Figs. 1–4, the time course of the normal reflex responses to stretch and to vibration is quite different, suggesting that there is an

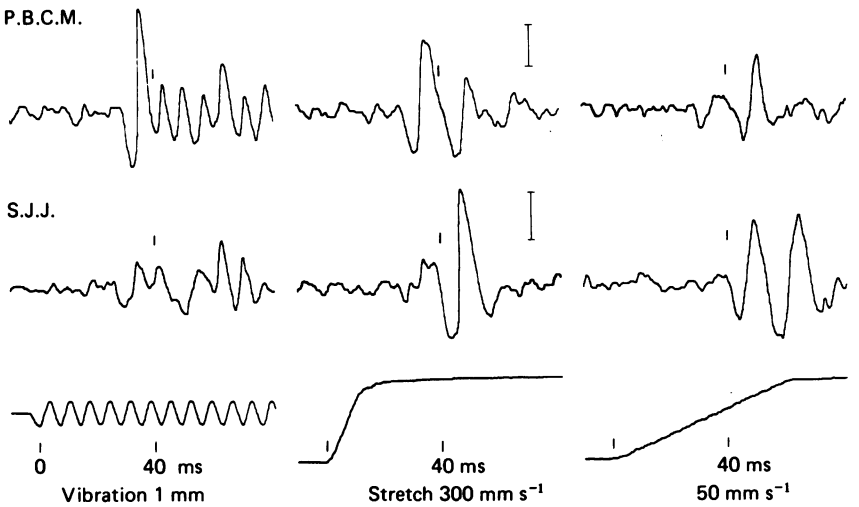

Fig. 4. Unrectified e.m.g. responses for the two subjects of Figs. 1–3 (P.B.C.M. and S.J.J.) and based on the same original data. Left, responses to vibration with that of S.J.J. again being much the smaller. Middle, responses to rapid stretch with the major part of S.J.J.'s activity occurring after 40 ms. Right, responses to slower stretch with S.J.J. showing a large late response but no significant short-latency response. A total of 128 responses averaged; bin width, 0·4 ms. Calibrations, 50 μV.

essential difference between their underlying mechanisms. For stretch, except at the highest velocities, any short-latency response is merely the prelude to a continued enhanced e.m.g. activity up to 50 ms or more from the beginning of the stimulus. In contrast, after an abrupt early response to vibration the e.m.g. activity drops down to, or even below, the pre-existing base line at 40–50 ms from the beginning of the stimulus before rising up again thereafter to a further wave, which showed considerable variation between subjects (see also Fig. 12). Though varying in detail, such differences between the effects of stretch and of vibration were found in all subjects.

The validity of the comparison of the two types of response depends *inter alia* upon their being of comparable magnitude. The fastest stretches may evoke a large initial response with little immediately maintained follow-up (see Fig. 1) and not so very different from the response to vibration. It might be suggested that the characteristic differences between the two modes of stimulation arose through vibration being much the more powerful stimulus to Ia action, and thus eliciting a much greater initial motor discharge and subsequent refractoriness. However, as already illustrated in Fig. 3, the wave form of the response to vibration altered relatively little with

reduction of the amplitude of vibration, and did not then become at all like that to stretch. Fig. 5 hardens the argument by comparing the responses to stretch and to vibration when the two members of each pair have been selected from among others recorded on the same occasion, so as to show approximately the same initial short-latency response. As was always found on making such comparisons, in the period from 40 to 50 ms the stretch response was by far the larger. This disposes of the suggestion that the differences described hitherto arose simply from non-linear

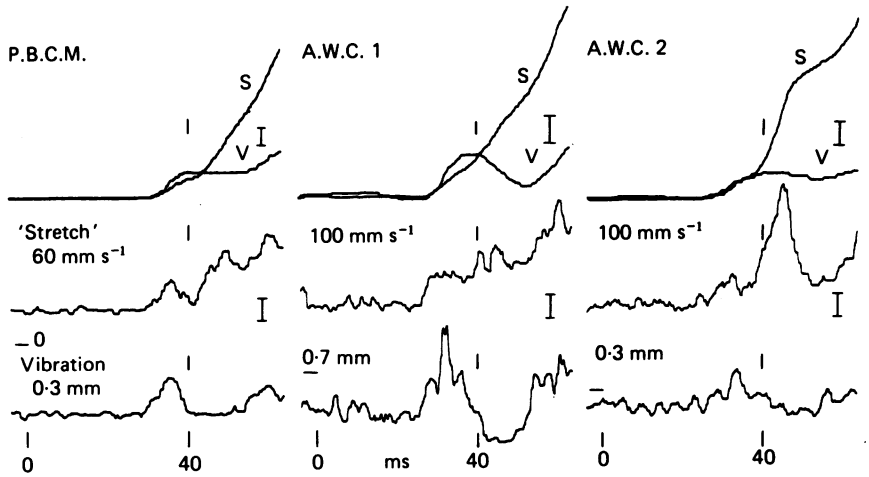

Fig. 5. Comparison of responses to 'stretch' and to vibration. These have been selected so that the two types of stimuli, by virtue of their particular parameters, excited comparable short-latency responses, thus emphasizing the differences in their long-latency components of response. The responses for P.B.C.M. were from his left, non-dominant, hand instead of from his right as before; they happened on this occasion to show particularly clear 'segmentation' in response to stretch, though not to vibration. The responses for A.W.C. were obtained on two separate occasions, at different repeat frequencies and when his reflex status would seem to have altered slightly; in A.W.C.2 the long-latency component of response was relatively enhanced in the response to stretch but not in that to vibration. From left to right various parameters were as follows: number of responses averaged, 128, 128, 256; repeat rate, 2·5, 1·25 and 5 Hz; stimulus duration, 46, 220 and 65 ms; stimulus magnitude, 2·3, 3 and 3 mm. E.m.g. calibration, 25, 10 and 10 μV; cusum calibration, 0·25, 0·1 and 0·1 μV s.

scaling effects obscuring the issue through the vibration always producing a very much larger initial motor discharge.

Interpretation of findings. While supporting the idea that the stretch response includes a special *long-latency* contribution, these observations simultaneously argue against its being due to the operation of a *long-loop* reflex of the conventional kind with a Ia input. Since in Fig. 5 the initial reflex responses are similar, the initial Ia volleys which caused them should also have been similar, and so these should again have evoked similar activity in any long-loop circuit responding to Ia input. The conspicuous absence of any response at 40–50 ms for vibration is incompatible with the idea of a Ia-activated long-loop reflex via the cortex or any other central site (including the spinal cord), unless some undefined characteristic of the afferent discharge set up by vibration somehow overrides the postulated delayed action. On

the other hand, the findings are entirely compatible with the suggestion that, in addition to its short-latency Ia spinal actions, stretch evokes a separate delayed reflex through the excitation of a different group of afferents that are not so readily excited by vibration. The spindle group II afferents comprise such a group. They have the added advantage for explaining the present results that, since they conduct more slowly than the Ia afferents, any spinal reflex action that they evoke of necessity occurs with a greater delay than the initial Ia action, so removing the need to postulate a long reflex pathway within the C.N.S.

Evidence for the dependence upon muscle afferents of the characteristic phasic response to vibration

Vibrating the pad of the thumb must inevitably excite a variety of cutaneous afferents as well as those in the muscles affected by the high-frequency to-and-fro motion of the digit. Moreover, the excitation of cutaneous receptors is well known to produce reflex effects on nearby muscles quite apart from that produced by nociceptors (see especially Eklund, Hagbarth & Torebjörk, 1978; Garnett & Stephens, 1980). An inescapable question is thus whether the characteristic differences between the reflex effects of stretch and of vibration arise merely because of superadded cutaneous reflex effects for the latter, whether excitatory or inhibitory. Two separate sets of control experiments argue strongly against this possibility.

1. Tendon vibration. By using a separate small vibrator with a fine tip, vibration could be relatively selectively applied to the tendon of flexor pollicis longus about 6 cm above the wrist. A lever arrangement pressed the vibrator against the skin over the tendon with a constant force of 2 N; this caused appreciable indentation of the surface so that the tip bore closely upon the tendon. The subject still flexed his thumb in the normal way against the tip of the now-stationary large vibrator. As illustrated in Fig. 6 vibration applied by this means produced essentially the same effect in the electromyogram as moving the terminal phalanx at high frequency. Again the vibration produces an initial short-latency phasic response, with no sign of the maintained activity at 40–50 ms that may be produced by a stretch with much the same initial response. Similar results were obtained in four other subjects.

In contrast to the similarity of the reflex effects of applying vibration at the two sites, the subjective sensory effects were totally different, suggesting that there was little similarity in the activation of cutaneous receptors in the two cases. Vibrating the thumb causes the most intense local sensation in the digit, with some spread into the hand. Vibrating the tendon is chiefly notable for a slowly increasing discomfort caused by the tip of the vibrator being forced into the arm, both in the presence and absence of vibration. The vibration itself produces a slight local vibratory sensation near its tip, but practically none in the thumb or hand. There was an inevitable slight spread of vibration into the thumb, as could be detected by the experimenter by feeling with his own hand, but it was one or two orders of magnitude less than when the vibration was applied to the thumb itself. This makes it very unlikely that any reflex effects of cutaneous or joint afferents can be held responsible for the characteristic phasic form of the reflex response to vibration, thereby leaving the field to the muscle afferents. Furthermore, afferents in the antagonist muscle would seem to be excluded from being in any way uniquely responsible for the typical form of response; although they were presumably regularly affected by thumb vibration, they would not have been expected to be excited by vibrating the agonist tendon. Even when vibrating the thumb their action may be weak because the agonist is lying flaccid and so its muscle receptors would be expected to be deprived of intrafusal bias and relatively insensitive to vibration.

It should be noted that on vibrating the tendon the vibration probably spread somewhat to

nearby muscles with excitation of their afferents, and that any consequential reflex activity on their part would have contributed to the recorded e.m.g. Thus it cannot be guaranteed that the various responses depend equally upon the uncontaminated activity of flexor pollicis longus. However, any such effect seems likely to have been slight since these other muscles should not have been contracting initially and so their intrafusal bias and central reflex gain were presumably low. Moreover, even if an appreciable proportion of the responses to tendon vibration were to have been derived from other muscles it would have little effect on the point at issue, namely that vibration confined to muscle fails to elicit a continuously maintained reflex response.

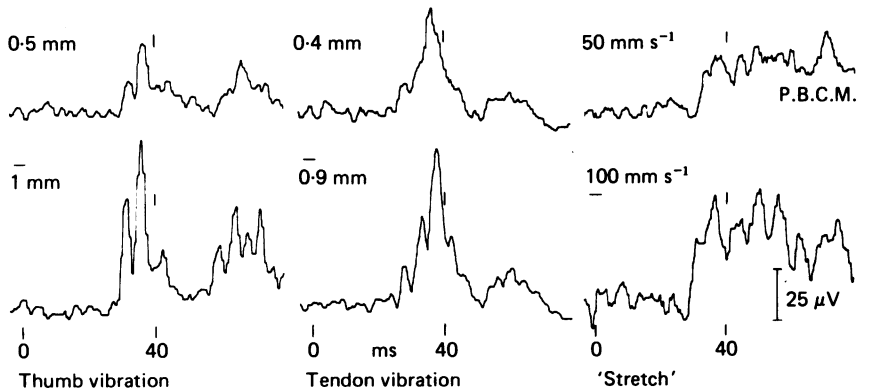

Fig. 6. Comparison of the response to thumb vibration, as in earlier Figures, with that evoked by vibration applied transversely to the tendon of flexor pollicis longus 6 cm above the wrist, with a separate vibrator with a fine tip. In both cases, for two amplitudes of vibration, the well-developed short-latency response is decaying rapidly at 40 ms, the time of expected occurrence of any long-latency component of response. 'Stretch' responses obtained later in the same recording session are shown on the right. Repeat rate, 1·25 Hz. Stimulus duration, 220 ms; 128 responses averaged for each. The initial e.m.g. level for the top traces is shown by the horizontal bars; it was coincidentally greater for the 'stretch' trials.

2. *Local anaesthesia.* The effect of eliminating local cutaneous and joint afferents by ring-blocking the base of the thumb with lignocaine (2 ml of 2% without adrenaline) was studied in the author and one other subject, who also had a brisk short-latency response. For both, the reflex response to vibration maintained its normal wave form with several amplitudes of stimulation on repeated study during the period that the thumb was totally insentient to standard clinical testing, as illustrated in Fig. 7. (It may be noted that the region under the clamp holding the thumb was also insentient.) The size of the initial component of response was indistinguishable from the normal, but there was a slight reduction for components of response after 40 ms.

Fig. 7 also shows that the response to stretch was barely altered by the anaesthesia; the other subject had a slightly greater reduction in the components after 40 ms. Thus the usual differences between the stretch and vibration responses occur equally in this state. It is concluded that whatever the reflex contributions of cutaneous and joint afferents to the present responses (and on the present findings these appear surprisingly slight) they cannot be held responsible for the essentially different effects produced by the two modes of stimulation.

The present findings with stretch are entirely in accordance with the experience of Marsden, Rothwell & Traub (1979), who in similar experiments on flexor pollicis longus examined 'seven subjects (five of whom had never undertaken the task before)' and found that 'on average, the stretch reflex decreased by 30% after anaesthesia, but in only one subject was it dramatically reduced, and in two (who were practised in the task) it was not altered'. It contrasts with the earlier widely disseminated view of Marsden, Merton & Morton (1972, 1977), based on two subjects only, that when a relatively naive subject has not previously been anaesthetized the reflex is abolished,

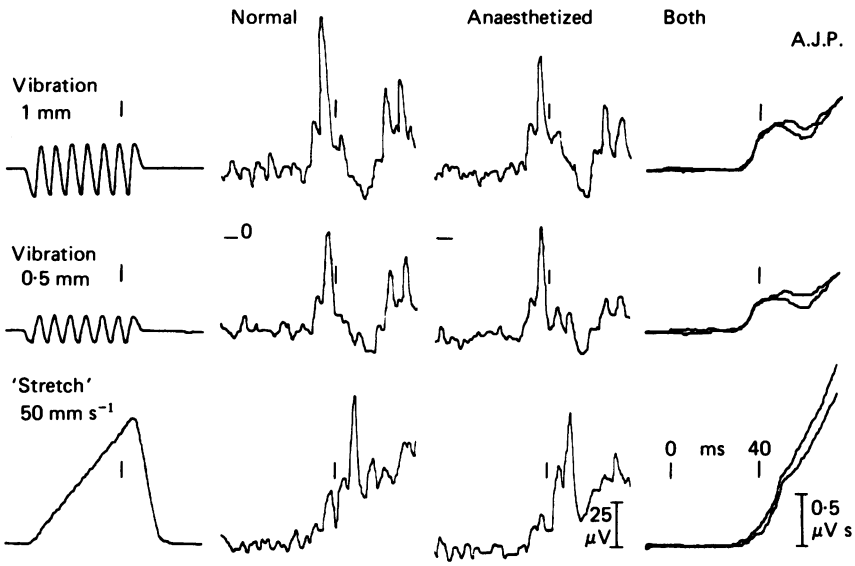

Fig. 7. Persistence of the characteristic response to thumb vibration after anaesthetizing the thumb by injecting local anaesthetic at its base. The thumb was then quite insentient to local stimulation, showing that the typical wave form of the vibration response could not have depended in its essentials upon the reflex action of cutaneous or joint afferents, whether acting to excite or inhibit the flexor pollicis longus motoneurones. The response to stretch also showed little change, arguing that it too depended upon the excitation of muscle receptors. A total of 128 responses averaged; repeat rate 2·5 Hz. Stimulus duration 47 ms (the length of record shown just excludes the 'off' response). The base line is shown for the top traces only; it was similar in all cases and did not alter appreciably with anaesthesia.

but that after repeated periods of anaesthetization and/or experimentation the subject becomes habituated so that, though reduced, the reflex may persist. The 'jerk' response elicited by rapid stretch of flexor pollicis longus has, however, always been seen as resistant to anaesthesia (Marsden *et al.* 1977). The present behaviour falls into line with that of all other muscles studied, the reflex responses of which have also been found to survive anaesthesia of the moving part, notably of the big toe, foot and hand (Marsden *et al.* 1977; Iles, 1977; Chan, Melvill-Jones & Cutchlove, 1979; Bawa & McKenzie, 1981).

Part II. Effect of cessation of stimulation

Value of studying 'off' effects for distinguishing between alternative spinal mechanisms. The case so far is that *if* it be accepted that all subjects (as well as special ones like S.J.J.) possess a long-latency reflex *then* this should be attributed to the spindle group II afferents producing autogenetic excitation, rather than to a transcortical reflex

activated by Ia input. However, it might be argued that the long-latency reflex does not occur universally and that the regularly contrasting effects of stretch and vibration depend simply upon conventional short-latency actions of the Ia afferents, and that when the discharges of these are recorded they will be found to be firing in an appropriately different manner in response to the two modes of stimulation. In the absence of such recordings, the debate rapidly becomes deadlocked when based solely upon the responses at the onset of stimulation, since the afferent volleys may well then possess a complex structure with a different temporal structure and degrees of 'segmentation' in the two cases. However, the relatively neglected study of the effects of terminating the stimulus, whether stretch or vibration, provides a more discriminating situation for analysis, since the behaviour of the afferents may then be predicted more confidently. On cessation of either type of stimulus the Ia afferents can be expected to show an immediate reduction of firing, greater for vibration than for stretch. Just how deeply the Ia afferents will be silenced and when they will commence firing again remains problematical, but on the short time scale of 10–20–30 ms there seems most unlikely to be anything corresponding to the successive variably synchronized discharges set up at the onset of stimulation. If differences in Ia short-latency reflex action were to be responsible for the contrasting responses to stretch and vibration at the onset of stimulation, then the responses on termination of the two types of stimulation could be expected to be similar rather than different, and both with a short latency. But if the late response at the onset of stretch is due to the group II afferents, it should occur equally on the release of stretch; these afferents also, like the Ia afferents, can then be expected to show a reduction of firing, with a consequent delayed reduction in their postulated excitatory contribution to the motor discharge.

The rest of this paper is concerned with establishing that the prediction of the group II hypothesis is indeed fulfilled. An unexpected complication has been that the short-latency effects of withdrawal of Ia input have proved to be surprisingly weak, perhaps because of a self-evoked presynaptic inhibition. A variety of experiments, differing chiefly in detail, have thus been required to show unequivocally that release does indeed produce an 'off' response with two separate components corresponding, it is suggested, to the successive withdrawal of Ia and then II reflex support from the motoneurone pool; a staggered delay is, of course, again to be expected by virtue of the different conduction velocities of the two types of afferent.

Termination of stretch. As would be expected, the subject S.J.J. with the very poor short-latency response to the onset of stretch (Fig. 2) showed solely a long-latency 'off' response on its completion. As illustrated in Fig. 8, an 8-fold change in the velocity of 'let go' had a negligible effect on the latency of the consequent reduction of activity, which in all cases was around 45 ms. This again favours the view that his long reflex delay is attributable to a 'transport delay', due to a finite time of transmission of activity around some neural loop, rather than to a 'distributed delay', due to a slow progressive withdrawal of afferent activity during the course of the let go.

Fig. 9 shows the effect of removing a pre-existing stimulus in the author whose brisk short-latency responses to stretch have been illustrated in Figs. 1 and 7. For both velocities of release there is a large 'off' effect, with the motor discharge temporarily

dropping almost to zero. The latency of this 'off' effect is marginally under 50 ms and thus 'long' rather than 'short'. Since the 4-fold change in the velocity of release produces practically no effect on the value it may be concluded again that the prolonged delay owes little to any lag in the response of the spindle afferents to the graded removal of the pre-existing stimulus. (The very fastest rates of release cannot usefully be employed since the 'off' response becomes yet more liable to be mixed up with the effects of reaching the end of the release; the spindle afferents would then

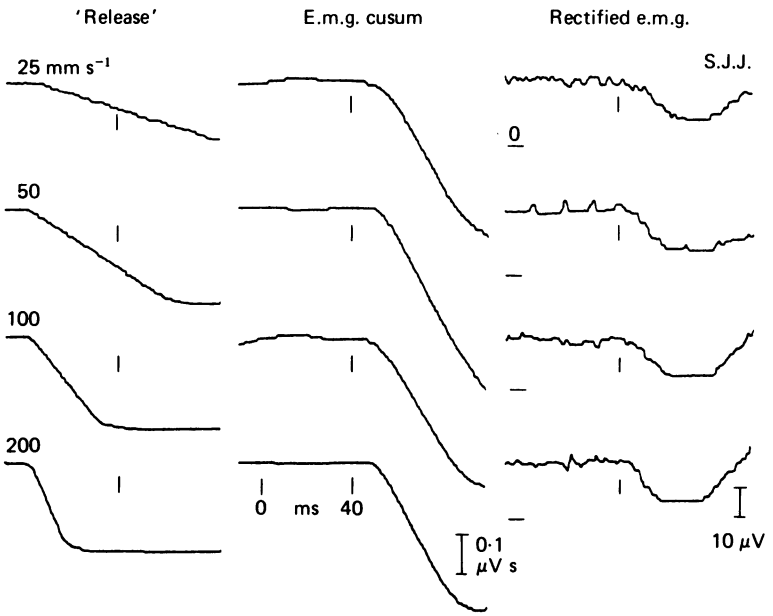

Fig. 8. Responses to 'let go' of the thumb at various velocities in the subject of Fig. 2 (S.J.J.) with a very poor short-latency response. The latency of the 'off' response is always greater than 40 ms and shows little change with an 8-fold increase in velocity, indicating that the delay is caused by the slowness of neural transmission rather than by a lag between the beginning of the release and the reduction in afferent firing. Stimulus repeat rate, 1·25 Hz; 80 ms from the beginning of the release the muscle was stretched back to its initial length at a velocity of 200 mm s^{-1}. (N.B. The background e.m.g. is appreciably less than in Fig. 2, presumably because of a less favourable electrode placement.)

be expected to increase their discharge again and to respond to any intramuscular oscillations that may then occur.) A separate 'short-latency' release effect might now also be expected, but none is immediately apparent above the base-line noise. In the light of further evidence (see later) it is presumed that this is simply due to its being too small to manifest itself after even quite extensive averaging, rather than to its complete absence.

Similar results were obtained in the other four subjects with short-latency responses of varying degrees of briskness to the onset of rapid stretch. The findings thus amplify those of Marsden *et al.* (1976*a*), who found similar long latencies for the response to a relatively slow 'release' applied in the course of a continuous voluntary movement. By using a wider range of velocity and studying a holding task rather

than a movement the present experiments provide more definitive evidence that flexor pollicis longus has a maintained stretch reflex mediated via long-latency pathways, while any continuing short-latency contribution is negligible. For some other muscles Marsden *et al.* (1976*b*) observed appreciable short-latency components of response on release, as has also been found with the present arrangements on studying flexor pollicis brevis (Matthews, 1984).

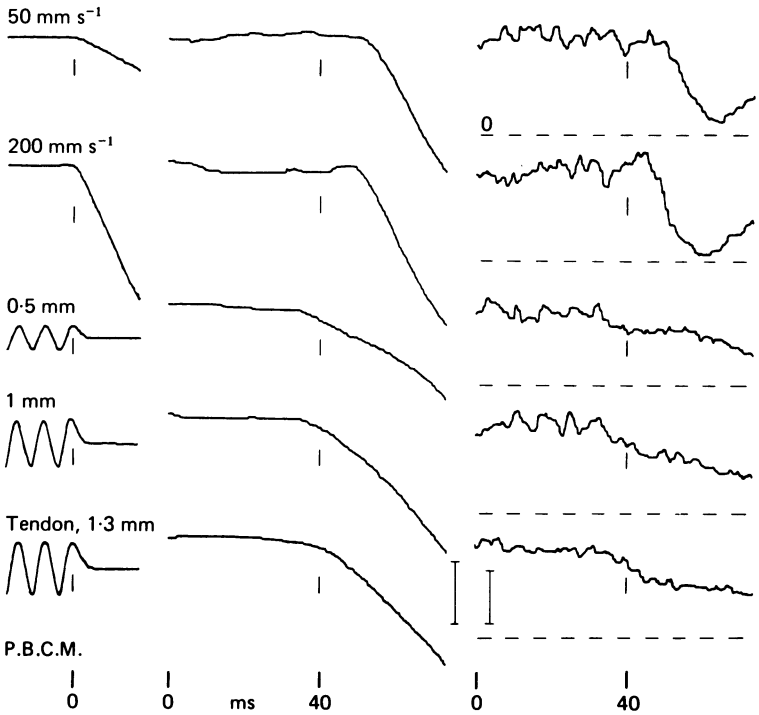

Fig. 9. Behaviour on cessation of pre-existing 'stretch' or vibration for the subject with brisk short-latency 'on' effects. With 'let go' the latency of the consequent reduction of activity is nearly 50 ms for both velocities of release, whereas on cessation of vibration (whether to the thumb or to the tendon) the latency is below 40 ms. Left, stimuli; middle, cusums; right, rectified e.m.g.s, with 512 responses averaged for each; repeat rate 2·5 Hz. The stretch or vibration was maintained continuously, except for successive periods of 66 ms the beginning of which is shown. The return stretch was at 200 mm s^{-1} for both velocities of release. The maintained target force was reduced to 3 N from the normal 6 N for the tendon vibration; this was studied earlier in the same day with different electrode positioning. The thumb vibration records were taken at the same time as the stretch records but are shown on lower gain to make the initial deflexion from the base line approximately the same in the two cases; in absolute terms e.m.g. activity was greater during vibration. (Calibration bars 10 μV and 0·1 μV s for the tendon vibration, twice these values for the stretch records, and twice this again for the thumb vibration.)

Cessation of vibration. In contrast, cessation of vibration produces a clear though modest short-latency reduction of electromyographic activity, as shown in the lower half of Fig. 9. The latency of this action is about 35 ms and thus substantially less than that of the only definite effect of 'let go'. But now, reversing the situation with release, there is no obvious long-latency component of response on terminating the

vibration. This was equally so when the amplitude of vibration applied to the thumb was varied, and when the vibration was applied directly to the tendon of flexor pollicis longus above the wrist. It is notable also that the reduction of e.m.g. activity is relatively less marked on cessation of vibration than on release of stretch, whereas if they had the same cause the response with the shorter latency might be expected to be the larger. Three other subjects with good responses to the onset of vibration gave similar short-latency 'off' responses without any appreciable ensuing long-latency response, though for one the difference in latency between the 'off' effect of stretch and of vibration was rather less than that in Fig. 9. It may be concluded that the paucity of long-latency actions for vibration in comparison with stretch is even more marked on the termination of stimulation than on its commencement.

Most of these observations were actually made with stimuli of about 200 ms duration as in Fig. 10, rather than with the very prolonged stimuli of Fig. 9, but this does not affect the situation. The responses of S.J.J. of Figs. 2 and 8 showed only a long-latency response on the cessation of vibration with a latency similar to that of termination of stretch, and any short-latency response was lost in the background noise. The final subject showed brisk vibration responses in an early session and the normal short-latency 'off' effect, but in a later session showed much weaker short-latency 'on' effects with both stretch and vibration, and their 'off' effects then had a similar long latency. In assessing these latter findings in the light of the group II hypothesis it should be remembered that vibration probably produced some group II excitation, both by its direct action and by the slight maintained stretch of the muscle that it is likely to have caused (see Methods).

A minor complication in Fig. 9 which would not appear to affect the point at issue is that the level of base-line e.m.g. activity was different for the stretch responses and for the top two with vibration, although they were obtained on the same occasion with the same target force and with the two types of stimulation alternated. The higher level of e.m.g. activity for a given force during nearly continuous vibration was seen also on other occasions, but did not seem to occur on the same scale when the 'off' responses to relatively short stretches were compared with those to short periods of vibration (see Fig. 10). This makes it unlikely to be due to differences in motor unit synchronization occurring in the two cases, which on sporadic testing seemed to be as great for short as for prolonged periods of vibration (see Fig. 5). Such differences might perhaps be associated with differences in the mean firing rates of the active motor units in the two conditions; this is thought to affect strongly the relation between force and e.m.g. (Rymer, Houk & Crago, 1979). The response in Fig. 9 to tendon vibration cannot be compared in this respect with the others, since it was taken earlier in the same day with a lower target force and slightly different positioning of the electrodes; on other occasions, however, the same effect was seen with tendon vibration.

Stretch and vibration combined. Fig. 10 provides a direct comparison of the short- and long-latency responses at 'on' and at 'off' for one and the same period of stimulation (220 ms duration); those in Figs. 1 and 9 were obtained on widely separated occasions. In addition, the simultaneous application of the two types of stimuli allows the early and the late 'off' effects to manifest themselves sequentially in a single recording, thus further demonstrating their independence. The response to stretch is shown on the left. At its onset a separate short-latency response appears; but on release only the long-latency component stands above the noise. In the middle of Fig. 10 the onset of vibration can be seen to evoke the usual short-latency response, with no obvious subsequent long-latency response in the 40–50 ms period. On cessation of vibration, the latency of the 'off' response is slightly longer than that of the 'on' response; but it is still below 40 ms and so counts as 'short', at any rate in comparison with the latency of the 'off' effect of stretch. (Comparison with the comparable responses on the right and also with those in Fig. 9 suggests that in the

middle cusum the 'off' latency appears unduly long because of irregularities in the e.m.g. base line.) The right of Fig. 10 shows the effect of combining vibration and stretch, with both types of stimuli starting and stopping simultaneously. For the onset of stimulation the response throughout its course is equal to or slightly larger than that to either stimulus alone and shows no definite separation into early and late components. However, at the end of stimulation two separate phases of reduction

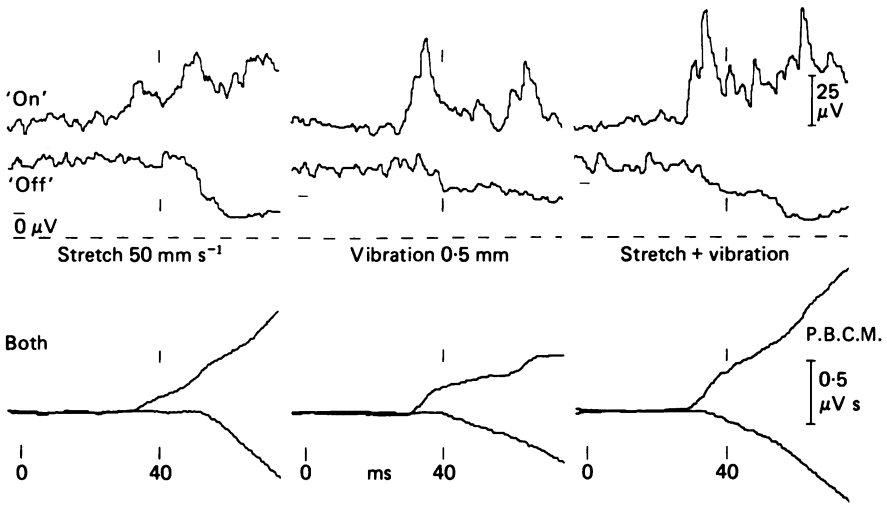

Fig. 10. The asymmetry of the 'on' and the 'off' responses. Left, responses to stretch. 'Off' does not elicit any definite short-latency effect but has a large long-latency action. Middle, responses to vibration. There is a clear but small short-latency 'off' effect, with a slightly greater latency than the large 'on' effect and of a quite different wave form; there is no appreciable further delayed long-latency 'off' effect. Right, responses to a combination of stretch and vibration, starting and stopping simultaneously. The 'on' response is larger than that for either stimulus alone. The 'off' response now shows separable short- and long-latency components, with 256 responses averged for each. Repeat rate, 1·25 Hz. 'Stretch', 3 mm at 50 mm s^{-1} at both 'on' and 'off'. Stimulus duration, 220 ms. Rectified e.m.g.s above; their cusums below.

of activity occur, corresponding to the early and late responses when the two types of stimuli were delivered independently and in accordance with the view that two separate mechanisms are involved. The particular importance of finding short-latency 'off' effects with vibration, whether alone or combined with stretch, is that it provides a yardstick by which to assess the long-latency 'off' effect with stretch and so excludes the possibility that, through the operation of adventitious factors, this latter represents the shortest time at which the cessation of activity in a short-latency pathway can become apparent.

Some additional delay for the 'off' response, above that of the 'on' response, might be expected to arise from the finite duration of the potentials recorded from individual motor units coupled with their diphasic shape. (Indeed, the first effect of a reduction of motor activity on the gross potential recorded from a number of units might be an increase in its over-all size, since the initial phases of newly activated motor units would no longer be interfering with the oppositely going phases of previously

activated motor units.) In the present recordings, the unitary potentials might last for 5–10 ms. Further, even 'short-latency' spinal pathways might perhaps show a degree of 'after-discharge' on termination of their afferent input. Thus even when a response is mediated solely by a short-latency pathway there is no necessity for the change in electromyographic activity to occur with an identical latency at the beginning and end of the stimulation; rather, there is every reason to suspect that the latency of the 'off' responses will be slightly greater than the minimum delay seen for the tendon jerk, as would appear to be the case in the present situation. But

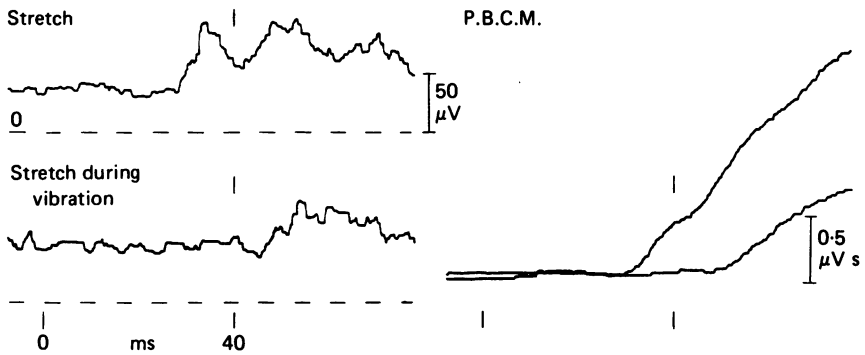

Fig. 11. The effect of continuous vibration on the response to stretch. Left, rectified e.m.g. in response to 100 mm s^{-1} applied on its own (above) and during continuous 1 mm vibration of the thumb (below). Right, corresponding cusums. Note that the initial response is abolished by the vibration and that the later response is also slightly reduced. A total of 256 responses averaged; repeat rate 2·5 Hz; stretch duration 60 ms; return velocity 200 mm s^{-1}.

the excess latency for the 'off' effect of stretch over and above that for vibration cannot be explained away in this manner, and so provides strong evidence for the occurrence of two separate reflex mechanisms.

Effect of continuous vibration. The poor development of the short-latency 'off' effects may well be due to a self-induced inhibition of the relevant pathways. As Fig. 11 shows, combining vibration with stretch has such an inhibitory action when it is maintained continuously, rather than commencing at the beginning of stretch as in Fig. 10. The short-latency component of the stretch response may then be virtually abolished while an appreciable long-latency response persists, as already described at the wrist (Hendrie & Lee, 1978; Jaeger, Gottlieb, Agarwal & Tahmoush, 1982; see also Marsden *et al.* 1976*b* for a similar observation on the effect of a preceding tendon tap). Moreover, whatever the underlying mechanism, such selective depression of the early response fits readily with the hypothesis that the early and late responses depend upon separate afferent pathways.

Presynaptic inhibition of the Ia afferents is a likely cause of at least part of this reduction in reflex responsiveness to stretch (Delwaide, 1973; Iles & Roberts, 1981), probably partly elicited by the activity of the Ia afferents themselves. Any such reduction in the responsiveness of the Ia pathway with continued activity will have the inevitable consequence that cessation of the activity will have much less effect than it otherwise would have done. This must at least partly explain the comparatively modest reduction of e.m.g. activity on stopping vibration, in spite of the fact that a very considerable fall in Ia input will then occur. On release of a pre-existing stretch the

change in Ia firing will be much less than at the end of vibration since the initial firing rate will then be fairly low; in a comparable task involving steady finger flexion, the firing of a carefully studied Ia afferent was always below 30 s^{-1} (Hulliger, Nordh & Vallbo, 1982). Moreover, the Ia afferents again seem likely to have been subjected to a certain amount of inhibition, whether self-induced or other. Thus there is no particular surprise that the short-latency 'off' effect of release should have been lost in the noise.

The functional properties of the motoneurones can also be expected to contribute to the asymmetry between the 'on' and the 'off' responses seen in Fig. 10. At the beginning of stimulation a number of new motor units can be expected to discharge within a few milliseconds, as well as the time of firing being advanced in a number of those which were already discharging; both factors would contribute to a large initial response. On cessation of stimulation, however, the motoneuronal discharge cannot drop below zero; moreover, the motoneurones will be continuing to receive a central 'voluntary' drive so that on withdrawal of the peripheral component of their excitation (as opposed to an inhibition) their activity will be delayed rather than totally eliminated.

It should be noted that in the present experiment the size of the late response shows a slight decrease rather than the increase described by Hendrie & Lee (1978); moreover, the latency of the response would appear to be increased. Thus it would be unwise to assume that vibration has a completely selective effect upon the short-latency mechanism, though this is clearly much more powerfully affected. In any case, an increase of the late response occurring at the time of a decrease of the early response tells one little about the strength of the late action *per se*, since it will no longer be so strongly opposed by the refractoriness and the recurrent inhibition brought into play by the initial motor activity. It may be noted also that in the present experiments the vibration was applied in such a manner as to mainly influence the agonist, whereas in Hendrie & Lee's (1978) experiments the effect of vibrating over the antagonist tendons was chiefly studied. On vibrating the agonist at the wrist, Matthews, Bawa & Matthews (1982) found no consistent effect on the size of the e.m.g. responses. In the cat, under conditions approximating to those in man, Matthews & Watson (1981) found that the afferent discharge elicited by stretch of a muscle might be increased by vibrating it, rather than 'clamped' as suggested by Jaeger *et al.* (1982). Thus the situation is a far from easy one to analyse.

Is the effect of release due to an inhibition rather than to a withdrawal of pre-existing excitation?

Any release of flexor pollicis longus is inevitably accompanied by a stretch of its antagonists, and so it must be asked whether the delayed reduction of activity seen with release could be due to an inhibition of the flexor pollicis longus motoneurones by afferents from the antagonists. The Ia inhibitory pathway would seem the only likely candidate for such an action, but the latency of the presently observed effects seems too long for this to be held responsible; the latency of this effect should be similar to that found on the withdrawal of Ia excitation on cessation of vibration. Given the complexity of spinal organization, however, other reflex pathways can readily be postulated. Direct evidence against the 'off' effect of stretch being due to inhibition was obtained by examining the response to brief periods of stretch and of vibration. In essence, a brief stretch may show an 'off' response with the usual long latency arising now, not from the steady base line, but from a brief period of elevated e.m.g. activity that betokens a continuing excitation of the motoneurones by the stretch at a time when the short-latency response should have been over. Put more simply, though less rigorously from the point of view of alternative interpretation, a suitable brief stretch may evoke a long-latency excitatory response at a time when the short-latency response is completed.

Effects of brief stimuli. Fig. 12 develops the slightly complicated argument. On the left are shown the responses to two separate periods of vibration. Time zero on the

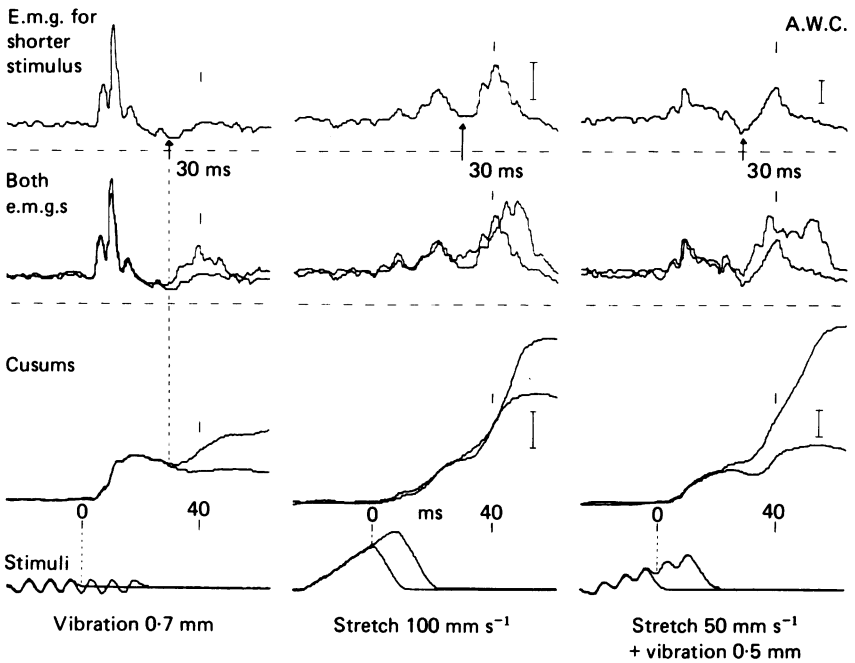

Fig. 12. The effect of terminating the stimulus soon after it began, whether stretch or vibration or both combined; the long-latency response to stretch develops at a time when any short-latency response should be decaying. Left, responses to thumb vibration of either 3 or 6 cycles duration. The superimposed e.m.g.s and cusums show that the two records deviate with short-latency timing, namely about 30 ms after the deviation between the stimuli (time zero in this illustration). In the top record the arrow shows this time in relation to the e.m.g. response to the 3 cycles stimulation; there is no further wave rising above the initial base line. Middle, responses to stretches of similar duration to the vibration and obtained on the same occasion. The deviation between the responses now occurs with long-latency timing (over 40 ms). Moreover, as indicated by the arrow which has the same positioning as for the vibration response, the shorter stimulus now elicits a large delayed response at the time when any residual short-latency response should be disappearing. Right, responses to stretch and vibration combined (separate recording session with deliberately different parameters of stimulation). The deviation between the responses now shows both short- and long-latency components, with 256 responses averaged. Repeat rate, 5 Hz. Stretch amplitudes and durations as indicated on records; the releases were deliberately rapid at 200 mm s^{-1}. Even the 'long' stimuli have been made rather short to allow a high repetition rate to be used. Calibrations 25 μV and 0·25 μV s.

scale corresponds to the time at which the stimuli begin to separate. The responses separate 30 ms later. The difference between them may be seen equally as due to an 'on' response to the continuing vibration or, preferably for present purposes, as due to an 'off' response on cessation of the shorter period of vibration; the same two ways of looking at things apply also to the effect of a normal release from a pre-existing steady state. (The longer period of vibration was also made deliberately short so as to minimize its over-all reflex effects and allow more rapid recovery before its next application. None the less it provides a perfectly suitable reference for assessing the effect of terminating the shorter stimulus, though perhaps less simply so than a period of vibration lasting throughout the record.) Either way, the findings provide a

measure of short-latency action in this particular subject. It may also be noticed that in this case the conspicuous late wave produced by the longer period of vibration (see also Fig. 3) depends upon continued short-latency excitation, since it is not seen with the shorter period of vibration; the late wave was again abolished in two out of the three other subjects in whom the matter was tested.

The middle records show the responses to two periods of stretch, with the shorter being of the same duration as the shorter period of vibration. (The longer stretch again provides a reference by showing the effect of continuing the excitation, so it is immaterial that its duration differs from that of the longer period of vibration.) The superimposed cusum and e.m.g. records now first separate clearly with the long latency of 40 ms. There is also a temporary deviation between the traces whose significance cannot be decided upon, but which occurs at the expected time of withdrawal of the short-latency contribution from the shorter stretch. Thus once again the 'off' effect of stretch is seen to have a longer latency than that of vibration. The new point is that in the period just before the traces separate they are running together *above* the pre-existing base line in a well-developed 'long-latency' response, with regard to their common beginning; moreover, they continue to do this beyond the time (30 ms from the point of separation of the stimuli) when any short-latency response evoked by the shorter stretch would have been disappearing. This all fits in with the idea that, although it has ceased, the shorter stretch was responsible for a continuing long-latency excitation, and the responses only separate after a corresponding delay. On the other hand, if the deviation were to be attributed to an inhibition from the antagonist it would remain to be explained why excitation (as shown by the enhanced e.m.g. activity) should be continuing when, on the evidence of the parallel experiment with vibration, any short-latency action should have ceased to be important.

The uppermost middle trace in Fig. 12 emphasizes the point at issue by showing that the shorter stretch evokes a large excitatory response at the very time when short-latency action should be ceasing, as indicated by the arrow based upon the responses with vibration, and when no such wave develops to a similar short period of vibration. This wave would appear to be the 'long-latency reflex' occurring on its own, without the support of the short-latency reflex. It might be suggested, however, that this wave should be attributed to a completely new phase of short-latency excitation, due to a burst of Ia firing triggered by intramuscular oscillations initiated by the sudden change in the direction of movement of the digit. And, if the response to the shorter stretch only had been studied there would be no gainsaying this possibility. However, this suggestion is contradicted by the finding that this delayed excitatory wave is seen equally for the continuing stretch, and so cannot be attributed to an abrupt disturbance in the movement. An apparently similar delayed response may be evoked in the first dorsal interosseus by a brief stretch, and as it is absent in Huntington's disease again cannot be attributed merely to the effect of mechanical oscillations (Noth, Friedemann, Podoll & Lange, 1983). A rapid tap to this muscle, however, evokes very little later response (Buller, Garnett & Stephens, 1980) while, as with the present vibration, producing a powerful short-latency effect.

The whole argument is strengthened by studying the effects of combining stretch and vibration, both stimuli being co-terminous, thus providing an internal comparison

of the two phases of reflex action. As shown on the right of Fig. 12, the response to the shorter stimulus now moves away from that to the longer stimulus in two separate phases, with a small separation at short latency and a larger deviation at long latency. Again the long-latency separation of the traces occurs at a time when the e.m.g. response to the shorter stimulus is above the base line, after having developed a new excitatory wave at a time when the short-latency activity is disappearing. These

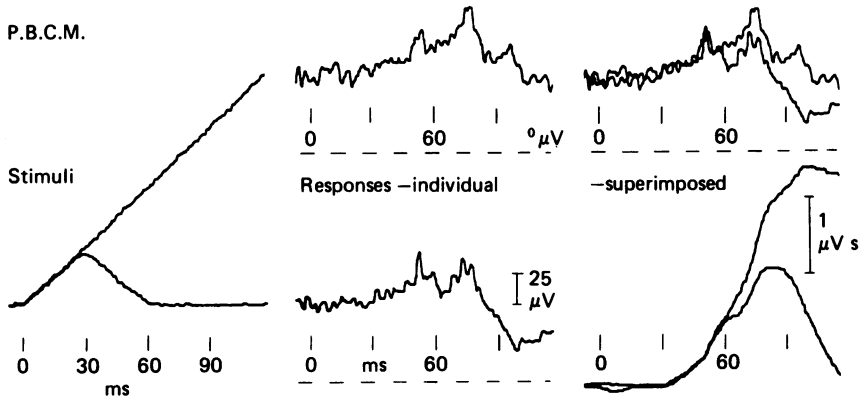

Fig. 13. Two components seen in the 'let go' response to a short stretch at a low velocity (20 mm s^{-1}) compared with a continuing long stretch; this was used with the aim of producing a weak but prolonged initial short-latency response. Left, the stimuli, with the short stretch having a rising phase of 230 ms. Middle, the resulting e.m.g. responses; as is usual, even that to the prolonged stretching (above) failed to produce maintained excitation. Right, superimposed e.m.g.s and cusums showing distinct short- and long-latency deviations between the two records at about 30 and 55 ms respectively, with 128 responses averaged, 1·25 Hz repeat. Large stretch, amplitude 3 mm, duration including its rising phase 200 ms, with a similar velocity of release. Small stretch, 0·6 mm.

records were obtained from the same subject as the rest of Fig. 12, but on a separate occasion and with deliberately less powerful stimuli of either kind, so that neither type of response should overwhelm the other. Such observations have been made in three subjects with a range of slightly different parameters of stimulation and have all been entirely in accord with the view that stretch produces a regular long-latency response, whereas vibration does not. However, they appear quite incompatible with the suggestion that the delayed 'off' effect of terminating a stretch might be attributable to an inhibition, rather than to the withdrawal of a pre-existing excitation.

Brief slow stretch. Fig. 13 fortifies this conclusion by showing an experiment in which, by persevering in the selection of the parameters of stretching, a 'release' in the absence of vibration elicited definite both short- and long-latency 'off' responses. In examining Fig. 13 it should first be noted that the response to the continued stretch fails to be maintained at a high level beyond about 70 ms from the beginning of the stimulus, even though the augmented Ia input may be presumed to be continuing (see also Marsden *et al.* 1981). The short stretch starts being released 30 ms from its beginning, and shortly thereafter this causes its reflex response to fall away below

that evoked by the continuing stretch. The separation occurs in two distinct stages, one at just below 30 ms from the time of separation of the stimuli and one at about 55 ms. The slightly greater latency of the long-latency 'off' response on this occasion as compared with others for the same subject (cf. Fig. 9) finds ready explanation on the group II hypothesis, since with such a slow release group II afferents, with their low dynamic sensitivity, might take an appreciable time to diminish their firing. The detection of a short-latency 'off' effect with stretch alone is important, since it excludes the possibility that the long latency of the normal 'off' response is merely due to a lag in the Ia afferents in responding to the release, as a result of mechanical lags or otherwise. Fig. 13, with its much slower release velocity than in Fig. 12, further supports the view that the late excitatory wave (occurring after the beginning of the release) cannot reasonably be attributed to intramuscular oscillations evoked by a rapid release.

Additional clear examples of a double 'off' response to brief stretches alone (without vibration) were also obtained on several other occasions from the author, using somewhat different stimulus parameters, and including the time when his thumb was anaesthetized. Further convincing examples were obtained from two other subjects (A.W.C. and A.J.P., on one occasion each). Otherwise in spite of further attempts a short-latency 'off' effect could not be confidently identified, just as on the termination of a prolonged stretch. The difficulty in successfully recognizing two separate 'off' responses in the face of the background irregularities is compounded by the complex form of the control response obtained with continuing stretch.

DISCUSSION

Genuine existence of a long-latency stretch reflex

The existence of a distinct long-latency stretch reflex, quite separate from short-latency Ia action, would seem to be put beyond question by the present experiments, at any rate for flexor pollicis longus. In this respect Marsden *et al.*'s views of 1976 have been vindicated. In part, the present experiments have simply extended their approach by employing ramp stretches of a wider range of velocity and more clearly defined wave form. The findings with stretch gained their force from the behaviour of one particular subject who had practically no short-latency response to complicate the issue. With release of stretch the results were uniformly clear-cut, since in all subjects a virtually pure long-latency 'let go' reduction of activity was seen. This interpretation of the findings with release depended crucially upon the newly observed short-latency effects of cessation of vibration, which provided the yardstick by means of which it could be established that the 'off' effects of stretch were not simply due to the removal of short-latency Ia action. There is no necessity for this to be identical with that of the short-latency 'on' effect; among other things the muscle action potential has a finite duration. Moreover, the situation was complicated by the smallness of the short-latency 'off' effect for stretch so that it could not usually be detected above the noise. Control experiments with brief stretches eliminated the possibility that the delayed action of release might really have been an inhibition produced by the concomitant stretch of the antagonist muscles, rather than due to the removal of a pre-existing excitation.

Comparison of the effects at the onset of stretch and of vibration supports the existence of a separate long-latency reflex evoked by stretch. In past discussions on

the stretch reflex it often seems to have been tacitly assumed that a step increase in Ia activity should evoke a similar, but delayed, step increase in the level of α motor activity in the situation when it acts upon the motoneurones solely by a single route (such as the monosynaptic pathway). Approximately 'square-wave' responses, such as those in Fig. 6 to stretching, might thus be taken to be 'natural' and the rather phasic responses to maintained vibration as 'unnatural', and requiring an explanation. In fact, however, motoneurone rhythmicity and refractoriness and Renshaw inhibition would seem to ensure that a sudden increase in afferent activity beginning with a near synchronous discharge would elicit at least one burst of motor firing above the subsequent level as seen with vibration. On this view the continued response evoked by stretch becomes the one requiring further explanation. The development of a long-latency reflex response at about 40 ms from the beginning of stretch would provide an entirely adequate such explanation for the continued activity. The occurrence of a late reflex for the long thumb flexor is further supported by observations on the timing of the response when the thumb is displaced sinusoidally at 5–10 Hz (Brown, Rack & Ross, 1982).

The arguments that have recently been arrayed against the existence of a long-latency response would not seem to disturb the present case. The fact that even a smoothly applied movement may still lead to mechanical irregularities in the muscle and bursts of Ia firing certainly makes it impossible to attach significance to any small bumps on the e.m.g. response at the beginning of stretch; but all this seems irrelevant to, and quite incapable of explaining, the present observations on release. Indeed, those who have questioned the very existence of a delayed reflex seem simply to have ignored this situation and the earlier though less crucial observations in this respect, whether they were extrapolating from results obtained in animals (Ghez & Shinoda, 1978; Miller & Brooks, 1981) or taking their stand on human data (Eklund, Hagbarth, Hägglund & Wallin, 1982*a*, *b*).

Accepting that a long-latency reflex exists, it follows that it inevitably plays a part in shaping the segmented reflex discharge produced by stretch. But in this respect it is not unique. In addition to any 'segmentation' of the afferent volley, it seems likely that the complexity of the spinal reflex centres is such that they respond with some sort of 'segmented' output even when presented with a 'square wave' of increased firing in a single type of afferent (Matthews, 1983*a*). Thus the mere observation of reflex segmentation provides little evidence as to whether or not a long-latency reflex is present in any particular case. It follows that even when a long-latency response is believed to be contributing to the response to stretch, some justification is required before equating any particular inflexion on the e.m.g. with the precise onset of the long-latency response. Most past estimates of its latency should thus be treated with caution, particularly if the response studied shows a well-developed short-latency component. Moreover, what is being sought may not always be as clear-cut as often tends to be supposed. If the delay of the late response were the result of a sudden initial volley in the Ia afferents taking a circuitous route to the motoneurones it should indeed have an abrupt onset. But if, as is being presently urged, it depends upon the spindle group II afferents the beginning may well shade into a continuing Ia response, and its sharpness of onset will depend crucially upon the distribution of conduction velocities of the population of group II afferents; this could well vary from subject to subject, and muscle to muscle. It

may be presumed that if a late response exists for flexor pollicis longus it will also exist for at least some other muscles, though on the evidence of Marsden *et al.* (1976*b*) its strength relative to that of Ia short-latency action is likely to show wide variation, thus complicating its recognition. Indeed, flexor pollicis longus may well be a peculiarly favourable muscle for the detailed study of long-latency effects, since here they may be relatively uncontaminated by short-latency effects and their aftermath.

The group II hypothesis: is it sufficient?

The essential finding of the present work is that in contrast to the effect of stretch, vibration largely fails to elicit the long-latency response, whether at its beginning or its end. If, as everything indicates, vibration produces its action by Ia excitation, then the late response to stretch must depend on something else. The spindle group II afferents are undoubtedly excited by stretch, and so if they were to have an autogenetic excitatory action the present findings would of necessity immediately follow. The latency of the late response is what one would expect if the conduction velocity of the group II afferents were to be about half that of the Ia afferents. No human data appear to be available, but extrapolation from our knowledge of the cat makes this a likely ratio. On the basis of limited evidence (Matthews, 1981) it is assumed throughout this discussion that it is appropriate to transfer our terminology for and knowledge about muscle spindles from the cat and monkey to man, though there may well be essential differences of detail. Given such uncertainties, the number of synapses involved in the postulated group II excitatory action cannot begin to be estimated, but there seems no possibility of sufficient time being available for it to have been mediated transcortically.

Animal work has left considerable confusion about the reflex actions of the spindle group II afferents (Matthews, 1972; Baldisserra, Hultborn & Ilert, 1981). The preferred view of the 1950s and the 1960s was that they belonged to a system of flexor reflex afferents producing a flexor reflex irrespective of their muscle of origin; however, as the specificity of the message transmitted by the spindle secondaries came to be appreciated it was conceded that they could well also have some other more specific action. In 1969 the present author launched the hypothesis that in the decerebrate cat their activity contributed powerfully to the well-marked tonic stretch reflex of that preparation. Comparison of the effects of stretch and vibration again provided the basis of the argument, but then in relation to the relative strength of their actions rather than to their timing; the timing has been highlighted in the present experiments by the use of an appreciably larger species than the cat. The idea then fell on stony ground and much informed opinion has failed to be swayed (cf. Baldisserra *et al.* 1981). Quite independently of this, however, spike-triggered averaging of intracellular recordings has shown that the group II afferents may have a monosynaptic autogenetic excitatory action, though in the preparations studied so far this action has been relatively weak compared to that of the Ia fibres. Any polysynaptic excitatory actions exerted by group II afferents would, on current thinking, be under supraspinal control so that their strength of action might differ greatly with the circumstances. Thus animal work leaves the matter open, and it certainly cannot be used to contradict the suggestion that in man, under the present circumstances, the group II afferents exert an excitatory action. This, it should be emphasized, is the sole unsupported postulate of the present work. Once granted, it

follows inevitably that stretch should elicit a long-latency reflex while vibration does not.

Not only does the group II hypothesis cover the present findings but, with a single exception, it would also seem to provide for all the results in the literature obtained with mechanical stimulation. The exception is that the excess delay of the long-latency reflex over the short-latency reflex appears in some cases to be the same for proximal and for distal arm muscles (Marsden *et al.* 1976*b*), instead of increasing with distal progression as required by the group II hypothesis. This would be a crucial deficit if the experiments could be performed so as to provide an unequivocal comparison. However, in the face of various complicating factors it was recognized from the beginning that 'the answer obtained is not straightforward', and even the authors claimed no more than that the group II hypothesis 'is made improbable', rather than being excluded. This essential matter requires further investigation; initial studies have been used to argue against the transcortical hypothesis on other grounds (Matthews, 1984). It should be noted that the frequently quoted finding that the excess latency of the late response over the early response is greater for muscles of the leg than of the arm (Marsden *et al.* 1976*b*; Chan *et al.* 1979) is to be expected on the group II as well as on the transcortical hypothesis, since in this comparison the peripheral conduction distance increases as well as the postulated central conduction distance.

Is the group II hypothesis unique?

On assuming the role of devil's advocate the present author has been able to devise only one other single hypothesis to cover the present findings and it fails to carry conviction, as well as lacking all independent support. It is that the powerful late reflex seen with both stretch and release depends upon Ib autogenetic excitatory action transmitted via some uncharted supraspinal route so as to achieve the appropriate delay. However, given the indirect nature of the evidence a series of reasonably plausible interlocking *ad hoc* suggestions could almost certainly be developed to explain the various results seriatim, without invoking the group II afferents. But every previous explanation for the long-latency response fails to accommodate all the present new findings and would appear to become untenable, except by inventing fresh explanations for, or producing objections to, the particular observation that refuses to conform with the preferred hypothesis. The suggestion that the late response is an artifact based either upon muscle resonances, with accompanying bursts of spindle firing, or upon the complicated interplay of excitation and inhibition within the cord, could certainly be made to cover the complex range of responses to stretch seen in the majority of subjects; but as already emphasized these explanations do not provide for the 'let go' responses.

On accepting that for flexor pollicis longus there is a genuine long-latency response to stretch it becomes unreasonable to attribute it to Ia activity, since it is not seen with vibration although this undoubtedly provokes a powerful Ia discharge. This would seem to exclude both the hypothesis that it is due to a Ia transcortical reflex, and any suggestion that the initial Ia activity sets up a delayed response within the spinal cord in the manner described by Hultborn & Wigström (1980) in the decerebrate cat. This latter response, it may be noted, differed from the present long-

latency response in showing a greater latency, in persisting after removal of the stimulus, and in being elicited by vibration rather than by stretch, instead of vice versa. The failure of the late excitation to appear with vibration might be suggested to be due to a separate inhibitory response intervening at the time of arrival of the putative cortical volley with vibration but not with stretch. But it is not apparent where this could originate. It did not come from cutaneous or joint receptors in the thumb. Pacinian corpuscles seem an unlikely candidate for such a supposedly powerful effect. Moreover, if such an inhibitory mechanism were to exist it would provide little help in explaining the delayed responses to release. Thus the group II hypothesis cannot yet be considered to provide a unique explanation for the present findings and requires further testing. But on current showing it would appear to provide by far the most reasonable explanation for the long-latency excitatory effects of stretch.

Does the group II hypothesis exclude the long-loop hypothesis?

Animal work has provided a rather powerful case for the existence and functional operation of a neural pathway running from a displaced limb to the motor cortex and back to excite the motoneurones of the stretched muscle (see Evarts, 1981; Evarts & Fromm, 1981; Chofflon, Lachat & Ruegg, 1982; Wiesendanger & Miles, 1982). For its fastest responses it would appear to rely upon Ia input, but group II afferents might well contribute to its later components. It may be noted, however, that the work on cortical units has tended to be concentrated upon those which fire appropriately to contribute to the desired spinal excitation; less emphasis is usually placed on those which fire inappropriately and which might contribute a counter-balancing inhibition, so there is no necessity for a powerful response to be mediated by this route. In man, electrical stimulation of a mixed nerve elicits certain late responses that may perhaps be due to Ia long-loop action (Iles, 1977). Transcortical reflexes have also been suggested to be produced by cutaneous inputs, as by Jenner & Stephens (1982); however, this is an essentially different case, the outcome of which has little bearing on the situation for muscle.

As recognized for some years, any transcortical stretch reflex acts in parallel with the Ia spinal pathway, so that the only change created by the present hypothesis is that any long loop has also to function in parallel with a group II spinal pathway. The unchanging question is, what relative contributions are provided by spinal and by cortical pathways to the excitation of motoneurones? The Ia spinal route can continue to be accepted as uniquely responsible for any short-latency action, though for slower stretches this may be weak. However, on present showing, for the human flexor pollicis longus it contributes rather little to any maintained response. For this the spinal group II route emerges as far more powerful than any Ia supraspinal route, since it now appears as the agent chiefly if not wholly responsible both for the appreciable component of delayed excitation at about 40 ms after the onset of stretch and also for the powerful delayed 'off' effects of release, neither of which is apparent with vibration. Debate as to whether or not vibration also produces some effects at 40–50 ms does not immediately further the argument, since it would still be impossible to decide whether any such are due to Ia transcortical action or to some degree of spindle group II activation with accompanying spinal reflex action

supporting the short-latency effects of a continuing Ia impact. Conversely, the fact that vibration normally fails to produce appreciable excitation above the pre-stimulus base line at the supposed time of arrival of descending cortical activity (approx. 40 ms, referred to the muscle) does not show that such a cortical volley is absent; it might merely be acting to reduce the depth of an 'inhibition' following the initial short-latency discharge, rather than producing a frank wave of excitation.

It will have been noted that the present analysis of 'late' response has been concentrated on the period 40–50 ms from the beginning of the stimulus. This corresponds to the initial or A segment of the long-latency response of Marsden *et al.* (1978), who suggest various adventitious reasons why some other workers have apparently found a greater latency for the beginning of the supposed long-loop response. Excitatory effects occurring at 50–60–70 ms from the onset of stimulation are equally of interest, but are still less open to exact interpretation, though continued Ia and II spinal reflex action can be safely presumed to be at least partly responsible. The effect of shortening the duration of vibration (see Fig. 12) shows that continuing short-latency Ia action is usually crucial in determining whether a frank late excitatory wave occurs with a Ia input. An additional complication is that at about this time the possibility first arises of further short-latency reflexes evoked by the mechanical consequences of any initial short-latency reflex response. The possibility of some sort of 'voluntary' response also has by then to be considered. An important related question, to which there is no immediate answer, is whether the 'neural set' in the present experiments was such as to favour spinal group II action, perhaps at the expense of transcortical Ia action. This cannot, however, be ascribed to the subjects exerting an isometric force before the stimulus, rather than to their performing a movement; in their later work Marsden, Merton and their collaborators seem to have employed these two conditions interchangeably without affecting the findings (see Marsden *et al.* 1978). The successful recognition or exclusion of a transcortical contribution to the response becomes extraordinarily difficult when, as now suggested, it has to be distinguished from the spinal contributions of the group II afferents as well as those from the Ia afferents. Further, as suggested by Crago, Houk & Hasan (1976), the cortex may well respond to its proprioceptive input with a pre-set 'triggered response' considerably in advance of the classical reaction time and which may be difficult to distinguish from a graded 'proportional response'. Human work, it would appear, has as yet shown nothing that can be unequivocally attributed to a rapid transcortical servo-type reflex evoked by muscle stretch; but, given the experimental difficulties, there seems no immediate prospect of excluding its existence. How far the hypothesis remains credible must thus be a matter of opinion, but on the basis of the present findings it would not appear to provide a serious alternative to the group II hypothesis in providing an explanation for the delayed component of the stretch reflex.

Final considerations

Once assimilated, the idea that the spindle group II afferents act co-operatively with the Ia afferents in mediating a spinal stretch reflex produces very little disturbance to classical thought. It is the conception now under attack of a Ia transcortical reflex that has always been proclaimed as providing the radical new

approach. There is nothing intrinsically surprising about the present view that such a basically simple response as the stretch reflex should fall entirely into the sphere of action of the spinal cord. Some of the implications of the present hypothesis were listed over a decade ago when the possibility was first raised that the spindle group II afferents might contribute to the stretch reflex of the decrebrate cat (Matthews, 1969); they remain pertinent, but do not merit reiteration. Perhaps the chief current interest of the suggested dual afferent input for the stretch reflex is that it allows for a greater modifiability of spinal action by higher centres than would otherwise be possible. In particular, the balance between the phasic and tonic components of the reflex could potentially be altered by selectively enhancing or depressing the contribution of one or other of the two afferent inputs, as by operating upon the interneurones via which some parts of their action may expect to be mediated. The spindle secondaries are, of course, much less sensitive to dynamic stimuli than are the spindle primary endings. Even the monosynaptic actions of one or other pathway might be depressed independently by means of presynaptic inhibition, as is probably the case for the Ia afferents during maintained vibration (see Fig. 11). Such modification of the functional properties of the stretch reflex could thus comprise far more than a simple gain control and might well be valuable when performing different types of task. Occurring in an unwanted and unplanned manner with neurological disease, derangements in the balance between Ia and II reflex contributions seems likely to underlie at least some of the bewildering number of ways in which muscle 'tone' may change. Thus the present hypothesis suggests certain new approaches to classical problems, and the pursuit of these should expose the hypothesis itself to further testing.

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