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IN-SERIES COMPLIANCE OF GASTROCNEMIUS MUSCLE IN CAT STEP CYCLE: DO SPINDLES SIGNAL ORIGIN-TO-INSERTION LENGTH?

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SUMMARY

1. It has been claimed that stretch in the non-contractile (extramysial) portion of muscles is substantial, and may produce large discrepancies between the origin-to-insertion muscle length and the internal length variations 'seen' by muscle spindle endings.

2. In eight pentobarbitone-anaesthetized cats, we estimated stretch in the extramysial portion of medial gastrocnemius (MG) muscle with a method similar to the spindle null technique.

3. Length variations of MG previously monitored in a normal step cycle were reproduced with a computer-controlled length servo. The responses of test MG spindle endings were monitored in dorsal root filaments. Distributed stimulation of ventral root filaments, rate-modulated by the step-cycle EMG envelope, served to reproduce step-cycle forces. The filaments were selected so as to have no fusimotor action on the test spindle.

4. Spindle responses in active cycles were compared with those in passive cycles (stretch, but no distributed stimulation). In some cases concomitant tonic fusimotor stimulation was used to maintain spindle responsiveness throughout the cycle, both in active and passive trials. Generally, small discrepancies in spindle firing were seen. The passive trials were now repeated, with iterative adjustments of the length function, until the response matched the spindle firing profile in the active trial. The spindle 'saw' the same internal length change in the final passive trial as in the active trial. Any difference between the corresponding length profiles was attributed to extramysial displacement.

5. Extramysial displacement estimated in this way was maximal at short mean muscle lengths, reaching about 0·5 mm in a typical step cycle (force rising from 0 to 10 N). At longer mean muscle lengths where muscle force rose from say 2 to 12 N in the cycle, extramysial displacement was in the range 0·2–0·4 mm.

6. Except at very short lengths, the displacement was probably mainly tendinous. On this assumption, our results suggested that the stiffness of the MG tendinous

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compartment was force related, and about double that of cat soleus muscle at any given force. Calculations indicated that though the stretch was small, the MG tendon would store and release enough strain energy per cycle to contribute significantly to the E_3 phase of the step cycle. The discrepancies in spindle firing were generally quite subtle, so we reject the claim that extramysial stretch poses a serious difficulty for inferences about fusimotion from chronic spindle afferent recordings.

INTRODUCTION

The CNS controls movement by causing muscle fibres to contract, their force usually being transmitted to bony structures by in-series tendons. Being elastic, tendons stretch when loaded. The stress–strain properties of excised tendons have been studied in detail in many different species (reviews: Butler, Grood, Noyes & Zernicke, 1979; Bennet, Ker, Dimery, & Alexander, 1986) interest centring mainly on tensile strength and the kinetic and kinematic implications of the storage and retrieval of elastic strain energy (e.g. Alexander & Vernon, 1975; Alexander & Bennet-Clark, 1977; Proske & Morgan, 1987).

It was realized some time ago that if tendon length did change substantially during voluntary movements, this could have an important impact on the feedback control of movement (Goslow, Reinking & Stuart, 1973; Rack & Westbury, 1984). The issue is the following. It is well established that proprioceptive feedback to the CNS plays an important role in the control of movement (e.g. Rothwell, Traub, Day, Obeso, Thomas & Marsden, 1982; Sanes, Mauritz, Dalakas & Evarts, 1985; Cole, 1989). The sensors of movement are the 'classical' proprioceptive afferents from joints, tendons and muscles, and afferents from skin receptors. For various reasons, not least the limited reproducibility and dynamic range of skin and joint receptors, muscle spindles are probably the key proprioceptive movement sensors. Now spindles 'see' length changes only of the 5 mm or so of the muscle fibres they span. If the in-series tendon were inextensible and the muscle fibres uniformly compliant, spindles would 'see' an invariant fraction of origin-to-insertion length l_{oi} and would therefore qualify to monitor this variable. But if tendons stretched significantly in response to active force, the relationship between the time courses of origin-to-insertion length $l_{oi}(t)$ on the one hand and the muscle fibre length $l_m(t)$ or spindle response on the other, could be quite variable. In the extreme case, muscle spindles could be stretched (increasing l_m) as l_{oi} decreases (Fellows & Rack, 1986). In human biceps brachii most of the change in l_{oi} in unloaded movements seems to take place in the muscle fibre compartment (Amis, Prochazka, Short, Trend, & Ward, 1987; Fellows & Rack, 1987). In maximal contractions tendon stretch reduced the length spindles 'saw' by an amount equivalent to 4% of the range of motion of the elbow.

In an elegant set of experiments based on the sonocardiometry technique, the transit times of bursts of ultrasound travelling between piezoelectric crystals sewn along muscle fascicles were monitored in the conscious, walking cat (Griffiths & Hoffer, 1987; Hoffer, Caputi, Pose & Griffiths, 1989). The results indicated a substantial stretching of medial gastrocnemius tendon during the step cycle, to the extent that in the E_2 phase of the cycle, though $l_{oi}(t)$ increased, the contracting muscle fibres actually shortened. Taken at face value, the peak discrepancy l_e between $l_{oi}(t)$ and $l_m(t)$ was about 2 mm during the active E_2 stance phase of the cycle, and 5 mm

at the E_3–F transition at swing onset. We have chosen the symbol l_e to represent total non-muscle-fibre (extramysial) displacement from all sources, including tendon displacement (see below). These discrepancies are large in relation to the total $l_{oi}(t)$ excursion measured to be about 10 mm, and would suggest a high tendon compliance, given that the peak force in the step cycle is at most 25% of maximal physiological force in this muscle. However, the interpretation of the data has recently been modified, some of the discrepancy being attributed to changes in muscle fibre pinnation angle, deformation of aponeurotic sheets (Caputi, Hoffer & Pose, 1989) and reduction of $l_{oi}(t)$ below the slack length of the muscle at the E_3–F transition (J. A. Hoffer, personal communication). As regards what spindles 'see' in a particular muscle such as medial gastrocnemius, it is the existence and extent of the net discrepancy rather than its source which is important. But in more general terms, if pinnation angle, aponeurotic deformation and operating range are indeed factors to be reckoned with, then their relative contributions to net extramysial compliance could vary considerably from muscle to muscle, depending on their architecture, anatomical disposition and kinematics.

The issue is of general importance for motor control because if $l_m(t)$ as 'seen' by muscle spindles does differ substantially from $l_{oi}(t)$, then the job of sensing limb movement might default to joint, and possibly skin, receptors. Alternatively, 'muscle length' might not be signalled by any single type of receptor, but rather be computed centrally from various sensory signals. The role of muscle spindles would be quite specialized: to provide feedback about internal changes in muscle fibre length. Furthermore, a high in-series compliance would pose interesting control and stabilization problems in the movement of varying inertial loads: the use made by the CNS of the two length feedback signals $l_{oi}(t)$ and $l_m(t)$ would then require substantial rethinking.

In the present study we used a variant of the spindle null technique of Rack & Westbury (1984) to investigate in simulated step cycles how much the internal length 'seen' by gastrocnemius spindles is attenuated during active force production.

METHODS

Experiments were performed on eight cats weighing between 2·4 and 4·9 kg. Anaesthesia was induced with an intraperitoneal injection of pentobarbitone (40 mg/kg), intravenous supplements being given during the experiment to maintain deep anaesthesia throughout the procedure, after which the cat was killed with an overdose of pentobarbitone injected intraperitoneally. The cat's temperature was automatically maintained at 37 °C using a heating blanket and a thermistor taped to the abdomen. The right hindlimb was extensively denervated, leaving the tibial nerve intact. The triceps surae muscle was dissected free from surrounding tissue. The calcaneum was cut through, leaving a stump about 5 mm long attached to the Achilles tendon. The triceps surae and plantaris muscles were separated and three of these four muscles were completely excised from close to their origin leaving either medial gastrocnemius (MG; six experiments) or lateral gastrocnemius (LG; two experiments) intact. The common peroneal nerve and branches of the tibial nerve other than that to the chosen portion of gastrocnemius were cut. A hole was drilled through the free stump of calcaneum allowing a loop of thick stainless steel wire to be passed through. The loop was crimped closed and subsequently served as a secure and stiff coupling to the electromagnetic length servo. A lumbar laminectomy spanning the L2–S2 segments was performed. The right L7 and S1 dorsal and ventral roots were cut close to the spinal cord. The animal was mounted in a rigid metal frame. Its right hindlimb was immobilized by hip pins and clamps at the femur and the calcaneum, all rigidly attached to the frame. Special steps were taken to buttress the clamps and their supports, as it was found in preliminary experiments that 20 N of muscle force

could significantly deflect the supports normally used in passive experiments of this type. The skin around the muscle and laminectomy sites was dissected free and formed into separate pools which were filled with paraffin covering the spinal cord and muscles. The pools were automatically maintained at 37 °C with heating lamps and thermistors. Special care was taken to keep the portion of tendon which emerged from the leg pool covered with gauze swabs, which acted as wicks for the paraffin, and prevented the tendon from drying out. The loop of stainless steel wire at the tendon was hooked onto a 10 mm diameter proving-ring force gauge which was attached rigidly to the linear shaft of the length servo (Printed Motors, Ltd, Bordon, Hants). Displacement was monitored by a ten-turn potentiometer attached to the rotary shaft of the printed circuit motor. Conversion of rotary to linear motion was achieved by a cable-coupled rack and pinion gear.

System stiffness

The servo had a measured stiffness of 6 μm/N. Consequently, in trials with 20 N peak force, the actual changes in l_{ol} differed from the command signal by up to 120 μm. However, this did not introduce errors in comparisons between l_{ol} profiles in active and iterated passive trials, because the actual monitored displacements rather than the command signals were compared. The rack and pinion coupling and the in-series force transducer contributed 10 μm/N compliance, and the femoral clamp assembly 7 μm/N. The 'hidden' 17 μm/N compliance resulted in overestimates of inferred extramysial displacement l_e. Corrections were therefore calculated according to the force variation: e.g. for a 0–20 N variation, the correction for yield in the apparatus ('hidden compliance') was 0·34 mm.

Recording and stimulation

Figure 1 shows a schematic of the experimental arrangement. The dorsal roots of L7 and S1 were split and laid over platinum hook electrodes to obtain recordings from single Ia or II afferents from LG or MG. Two steps were taken to minimize cross-talk due to ventral root stimulation. First, where possible, the afferent recordings were bipolar. This required careful handling of the filaments to ensure that conduction was maintained beyond the proximal electrode. Second, the four pairs of stimulating electrodes were arranged so that the proximal electrode of each pair was the anode, referenced to a common indifferent electrode. Afferents were identified by standard procedures (conduction velocity and responses to maximal muscle twitches and stretch).

'α' and 'fusimotor' ventral root subdivisions

The ventral roots of L7 and S1 were split and subdivided. Filaments were selected and arranged in one 'fusimotor' and four 'α' divisions, so that stimulation of each of the latter produced about the same muscle tetanic force. Distributed stimulation was then used, i.e. each division was stimulated in turn in a cyclical sequence, to obtain a smooth contraction (Rack & Westbury, 1969). The aim was to reach a total isometric force at resting muscle length of between 10 and 20 N with a stimulus rate of 30 s^{-1} division^{-1} (100 μs pulse duration).

The 'fusimotor' division contained all filaments which had been found to contribute fusimotor action to the test spindle and had therefore been split away from the 'α' divisions. The screening was done as follows: each of the fledgling 'α' divisions was stimulated briefly at 100 impulses s^{-1} ($4 \times \alpha$-threshold), to reveal changes in spindle responses to sinusoidal or ramp-and-hold stretches characteristic of static and/or dynamic fusimotor action. If such action was seen, the filaments responsible were isolated by repeated subdivision and testing. Non-fusimotor subdivisions were returned to the 'α' division. Fusimotor sub-divisions were pared down so as to have minimal α-contamination, and added to the 'fusimotor' division. This process led to a progressive decrease in the total force elicitable from the 'α' divisions, but it was crucial to the experiment that these be entirely devoid of fusimotor action, and that powerful, tonic fusimotor stimulation be available to maintain spindle afferent discharge throughout the simulated step cycles.

Step cycle simulations

The cat step cycle was mimicked by using data obtained previously during chronic recordings (Prochazka, Trend, Hulliger & Vincent, 1989). Digitally stored averages of chronically recorded step cycles, as exemplified in Fig. 2, were reproduced repetitively by a CED 1401 laboratory interface and Olivetti M28 host microcomputer. The length functions (e.g. second trace in Fig. 2)

served as command signals for the muscle puller. In active force trials, the corresponding time courses of rectified, averaged gastrocnemius EMG (e.g. top trace in Fig. 2) were concomitantly reproduced, and were used to rate-modulate the pulse trains used for distributed ventral root stimulation: interleaved trains of 100 μs constant-current pulses were delivered to the four

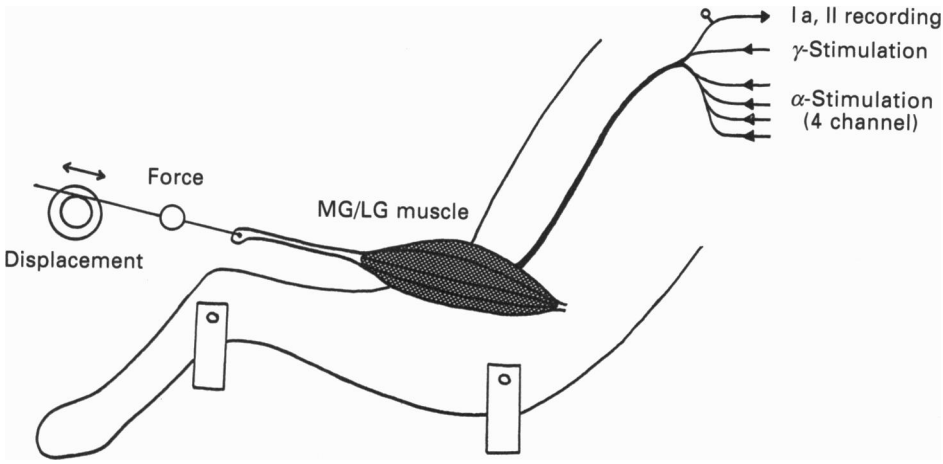

Fig. 1. Experimental arrangement. LG or MG muscle spindle group Ia or II afferents were recorded from dorsal root filaments. Ventral root filaments with pure fusimotor (γ) or skeletomotor (α) action were collected together. Displacement reproducing that in a normal step cycle (see Fig. 2) was imposed with a computer-controlled servo. Distributed stimulation of α-filaments reproduced step-cycle tensions. Spindle responses with and without active tension were compared.

α-divisions (rate range 0–30 impulses s^{-1} $division^{-1}$). Stimulus intensities were set independently for each division and adjusted to produce a total peak force of 10–20 N in the E_2 phase of the step cycle, and in some cases up to 30 N. For comparison, peak MG forces observed in normal cats range from 4–11 N in walking and 11–20 N in running (Walmsley, Hodgson & Burke, 1978; Whiting, Gregor, Roy & Edgerton, 1984). In most experiments, the fusimotor division was stimulated tonically with 100 μs pulses at 100 impulses s^{-1}. The overall effect of these combined inputs was to produce length and force changes in the muscle which mimicked those of step cycles, and ensured maintained firing of test spindles throughout.

Iteration

After an active force trial, consecutive passive trials were performed in which the length function was iteratively changed, with the aim of producing the same spindle firing profile as had just been obtained in the active trial. To this end, the active profile, or 'target', along with its associated length and EMG profiles, were first printed onto a transparent sheet using a Hewlett Packard Laserprinter. The transparency was trimmed and used as an overlay on the screen of a Tektronix 5111 storage oscilloscope. With a second CED/Olivetti computer system, iterative changes were made as follows: the length function to be changed was reproduced by the first computer at one-tenth speed, and summed with an externally generated analog 'error' signal, the resulting function being sampled and stored by the second computer. The 'error' signal came from a length transducer operated by the experimenter so as to add or subtract desired amounts from the signal being replayed. The error signal was a 'best guess' as to the length correction required to produce the desired firing profile, based upon known spindle response properties. When complete, the corrected function was loaded back into the first computer to be used in the next passive trial. This procedure was repeated until there was a good match between the spindle response to the latest length function (no active force) and the active 'target' response. Good fits were usually obtained

within three or four iterations. Indeed because this simple manual technique worked well, we cancelled plans to adapt the error minimization software of Hulliger, Horber, Medved & Prochazka (1987) to our present equipment. Displacement, force and afferent neurograms were recorded on a TEAC R61 cassette tape recorder for off-line analysis, which was performed with a CED 1401/Olivetti M28 system with averaging software.

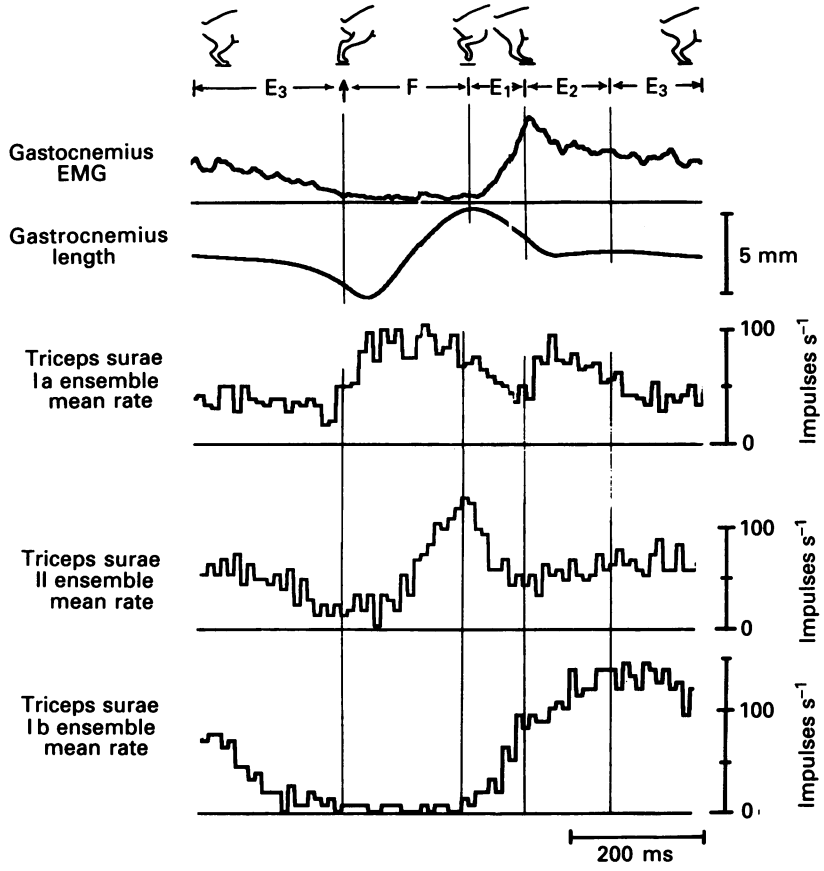

Fig. 2. Averaged time courses of electromyogram (EMG), muscle length and firing rate of group Ia and II muscle spindle endings and Ib tendon organ endings in cat step cycle (from Prochazka, 1990). The length and EMG profiles were used to drive the servo and the distributed stimulation concomitantly, as shown in Fig. 1. The effect of extramysial stretch on inferences about the fusimotor action underlying the spindle responses is dealt with in the Discussion.

RESULTS

In each of eight cats, a single afferent was studied, the distribution being: four group Ia and two group II endings in MG, and one Ia and one II ending in LG. In some trials the effect of active force on spindle responses to muscle stretch was barely perceptible. Figure 3*A* compares the responses of one such MG spindle primary ending to ramp-and-hold stretches before, during and after a period of tonic muscle contraction. The firing profiles are virtually identical throughout, with no more than a 2% drop in mean firing rate during the hold phase in the active force cycles.

Similarly, during sinusoidal stretching (Fig. 3*B*, same afferent), the firing rate profiles in trials with active force modulated from 7 to 24 N were nearly indistinguishable from those in passive trials.

Step cycles

Figure 4 compares the responses of a passive spindle secondary ending to the length variations of a step cycle with and without concomitant α-stimulation. In

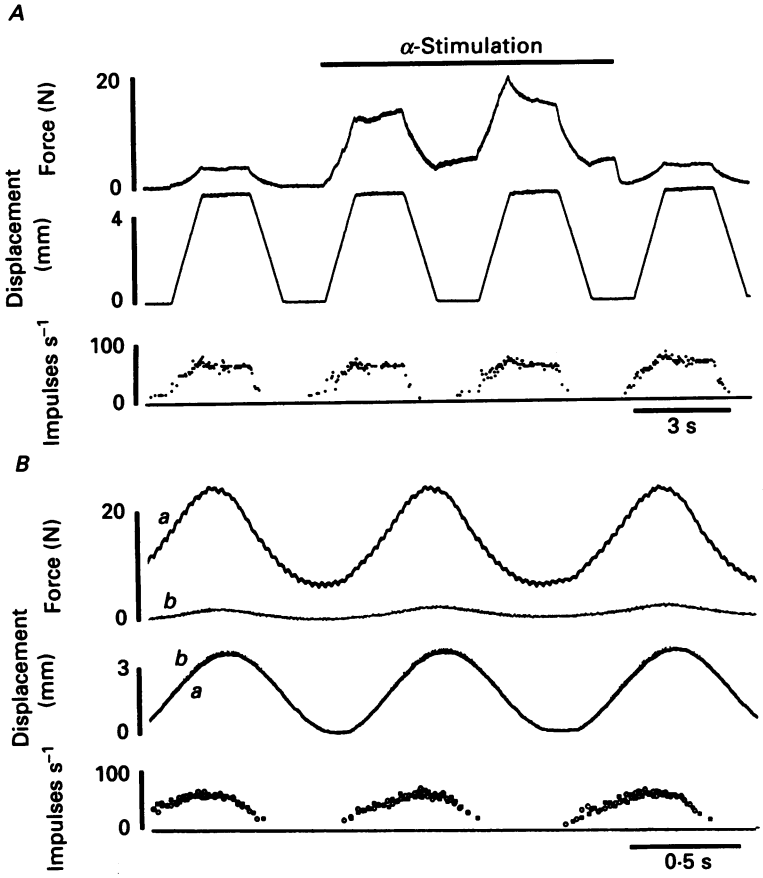

Fig. 3. Responses of a medial gastrocnemius (MG) Ia ending to: *A*, ramp-and-hold stretches with a period of tonic distributed α-stimulation indicated by the bar, and *B*, sinusoidal stretches, with and without tonic active tension (active traces *a* and ■, passive traces *b* and ○). In both sets of records, the spindle rate profiles are virtually the same with and without active tension. Note that the low dynamic index of this ending in *A* was probably due to the short mean muscle length; the afferent conduction velocity was 100 m s^{-1}.

this afferent there were more substantial differences between the active and passive firing profiles. The reduced firing in the active trial at the peak of force is consistent with in-series extramysial lengthening, which would absorb a portion of l_{ol}, thereby reducing the length 'seen' by the spindle. However, strain in the apparatus could also

have caused the discrepancy: a close examination of the length traces in Fig. 4 shows that at the 30 N peak of active force, l_{oi} was actually 0·18 mm less than in the passive trial, due to the 6 μmN^{-1} compliance of the length servo. An additional 0·51 mm reduction in l_{oi} due to 'hidden' compliance (see Methods) must be added to this,

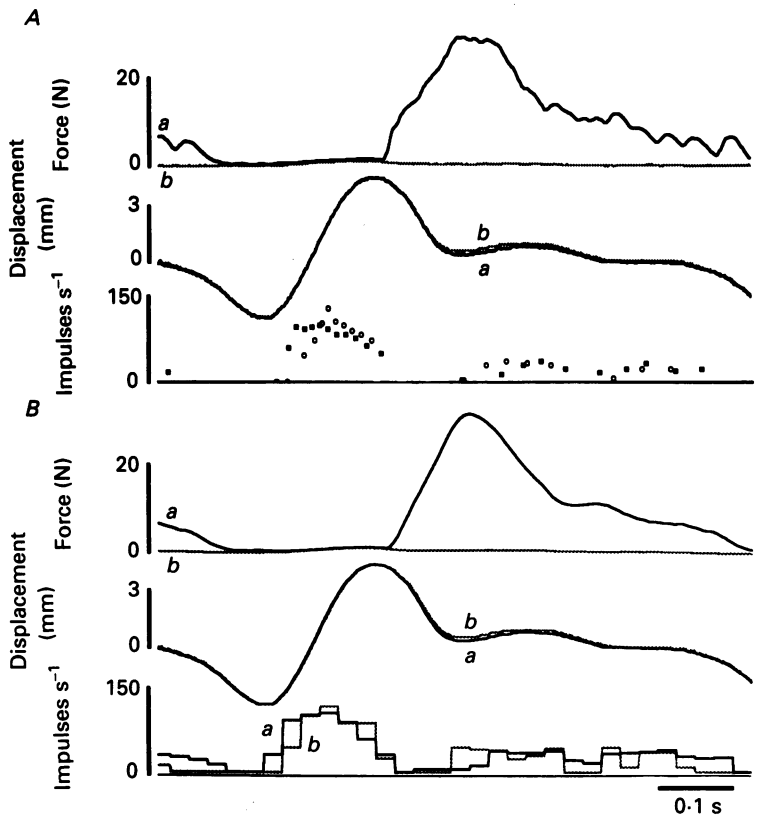

Fig. 4. Responses of a MG group II spindle ending in active (modulated distributed α-stimulation) and passive trials. *A*, a single active cycle (*a* and ■) compared with a single passive cycle (*b* and ○). *B*, averaged records of ten active (*a*) and ten passive (*b*) trials to demonstrate reproducibility. Note the discrepancies in spindle firing in this case. Note too that the length servo yielded slightly at high tension in the active trial.

giving a total shortfall in l_{oi} of 0·7 mm at peak force. From the data in Fig. 4 alone, it is impossible to deduce the relative contribution of these two sources of compliance and for this reason it was necessary to perform the iterative procedures to be described below, whereby spindles 'saw' virtually the same length changes in active and matched passive trials.

Another discrepancy in Fig. 4, which is more difficult to account for, occurred at the onset of the main stretch: in active trials the spindle firing profile was advanced by some 25 ms on that in the passive trials. This difference, which in fact was prominent in only two of the eight spindles studied, cannot easily be attributed to in-series compliance alone, as there was little difference in the active and passive forces at this point in the cycle. It is worth noting that force during the stretch,

whether in active or passive trials, was very low, rising to only 1·5 N in this example. This is because the range of length variation had been adjusted to be at the lowest end of that occurring in 'real' step cycles; slack length coincided with the onset of the cycle illustrated (zero on the length axis). The initial reduction in l_{oi} caused visible kinking in the tendon in passive trials. In active trials, kinking was reduced; the residue of muscle contraction carried over from the previous cycle was apparently sufficient to take up some of the slack. Thus in passive trials there was a 'dead' zone in which the imposed length variation was poorly transmitted to the spindle, whereas in the active trials, more of the length variation may have been transmitted, and so relengthening would have been 'seen' slightly earlier. This is not a tendon compliance effect, but it is an example of a mismatch between $l_{oi}(t)$ and $l_m(t)$ due to slackening, which might also occur in normal locomotion. We will return to this issue later.

Figure 5 illustrates an iteration experiment with a LG spindle II afferent. The small drop in firing rate at peak force (*A* and *C*: compare active and passive firing profiles) was matched by adjusting the length function in subsequent passive trials, the final results being shown in *B* and *D*. The maximal difference between the active and final passive length traces was about 0·6 mm. However, l_{oi} in the active trial at peak force (11 N) was actually about 0·2 mm less than shown, because of yield in the femoral clamp ('hidden' compliance 17 μm N^{-1}). We conclude that the net 0·4 mm difference represents the in-series extramysial stretch l_e due to active contraction. As will be seen, this is a fairly typical figure for inferred l_e in either LG or MG in a step cycle. Note that 11 N peak force in MG corresponds to a fast walk in the normal cat (Walmsley *et al.* 1978).

Fusimotor biasing

The drawback in the data of Figs 3, 4 and 5 is that the spindles did not fire throughout the cycle, and so length discrepancies occurring in periods of spindle silence would have gone undetected. To overcome this, in five of the eight experiments, tonic fusimotor stimulation (mixed static and dynamic) was used in both passive and active force trials. This is illustrated in Fig. 6, which shows three sequences of reproduced step cycles: (a) imposed displacement only, (b) displacement and tonic fusimotor stimulation, (c) displacement, fusimotor stimulation and α-stimulation. The tonic fusimotor stimulation produced a large increase in both stretch sensitivity and bias in the test MG primary ending. There was generally a slow, small decline of fusimotor action over the twenty to thirty cycles comprising a trial, this being particularly evident in early cycles such as the ones illustrated. Thus it was important when comparing cycles with and without active force, but with fusimotor stimulation, to pair them up according to their rank order within a sequence.

A close examination of Fig. 6 shows that there are small reductions in spindle firing rate associated with active force (cf. middle panel with right panel). This is detailed in the superimpositions of Fig. 7*A* and *C*. Iterated passive trials to match the spindle firing profiles (*B* and *D*) gave a maximal length discrepancy of 0·6 mm at peak force (18 N). Subtracting 0·3 mm of shortening due to the 'hidden' 17 μm N^{-1} compliance, peak l_e was 0·3 mm in this case. Even this is probably an overestimate because the

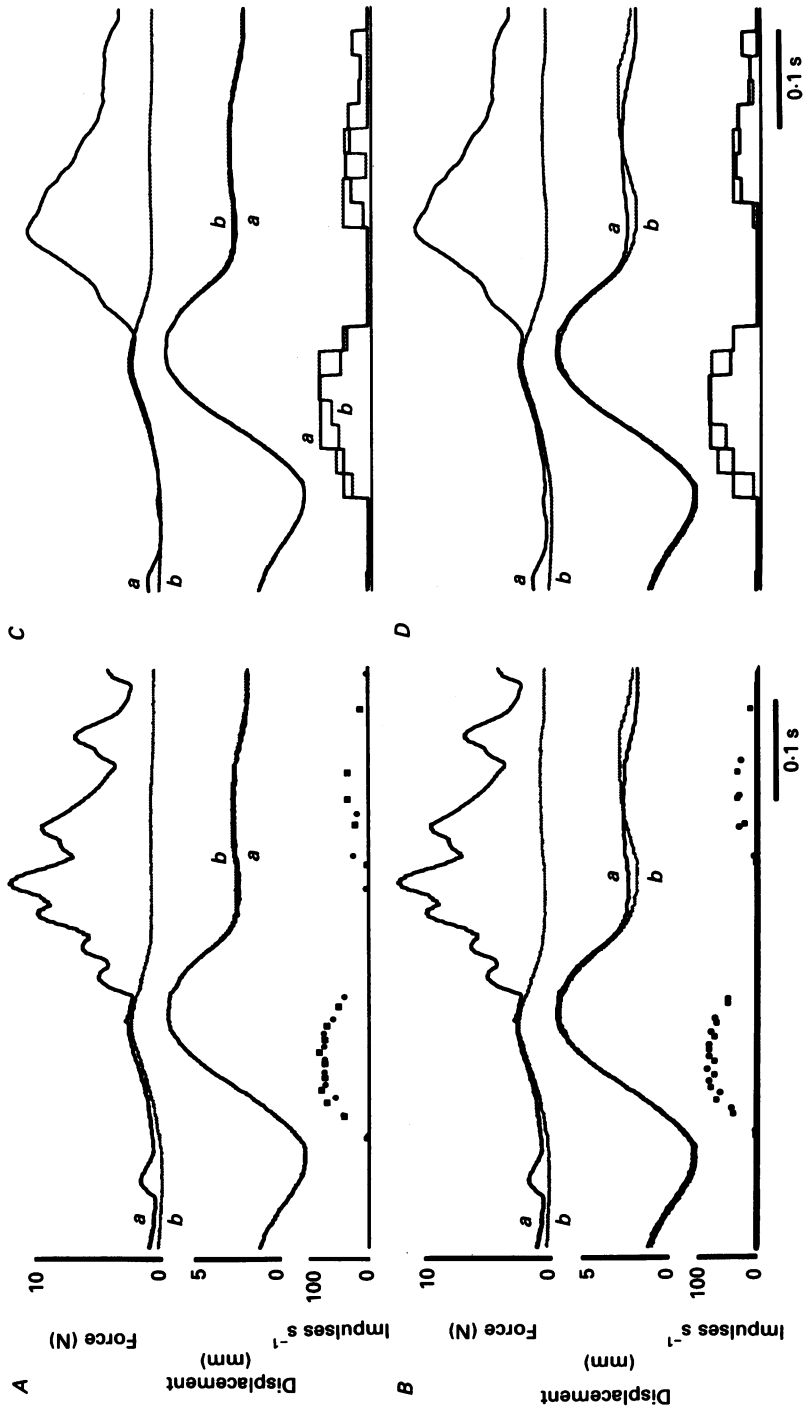

Fig. 5. Iteration experiment. Responses of a lateral gastrocnemius spindle secondary ending. *A*, single active (*a* and ■) and passive (*b* and ○) trials, as in Fig. 4. Note the small drop in firing during active tension (■). *B*, same active trial (*a*) as in *A* superimposed on the last trial (*b*) of a group in which on-line adjustments were made to the length signal to produce spindle firing which matched that in the active trial. *C* and *D*, averages of ten cycles corresponding to the trials in *A* and *B*. The differences between the length traces in *B* and *D* represent inferred extramysial stretch.

iteration overcompensated for the discrepancy in spindle firing. Note that a peak force of 18 N might only occur in a fast run.

Extremes of slackening

As seen above, if the muscle goes slack, $l_{oi}(t)$ may not be transmitted faithfully to the spindles. Does MG slacken in normal locomotion and if so, by how much? There

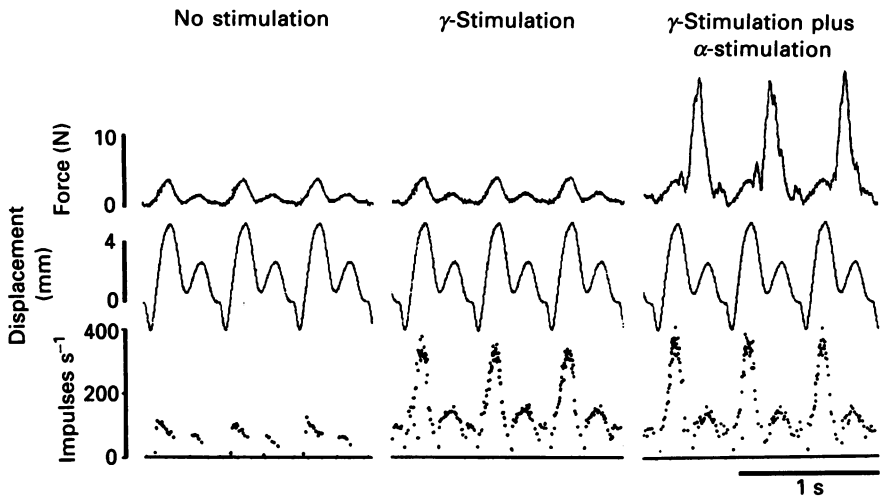

Fig. 6. Active and passive trials with concomitant, tonic fusimotor stimulation directed to a MG Ia ending to maintain its firing throughout the cycles. Left: responses to passive stretch alone. Middle: responses to stretch with concomitant 100 s^{-1} stimulation of a fusimotor filament containing both static and dynamic fusimotor fibres. Right: responses with concomitant (tonic) fusimotor stimulation and (modulated) active tension.

is not enough information in the literature to answer this with any certainty. Goslow *et al.* (1973) estimated that the minimal l_{oi} in cat MG was about 108 mm in walking and 105 mm in galloping, but slack l_{oi} was not determined. Force monitored in MG tendons in walking cats momentarily drops to zero or near zero once per step cycle, indicating that the muscle just slackens (Walmsley *et al.* 1978, Figs 2 and 3; Whiting *et al.* 1984; Hoffer *et al.* 1989). In cats with the skin over the Achilles tendon incised and reflected, but no other tissue disturbed, we found that with the knee at 90 deg, MG slackened at an ankle angle of 110 deg (20 deg plantarflexion). At these joint angles, MG l_{oi} measured on bones of a 3 kg cat was 101 mm. We passively moved the knee and ankle through the range of motion of step cycles. For simulations of erect locomotion (minimal knee angle 80 deg) MG never went slack. However, it did so at the end of the simulated stance phase of a crouched step, when hip and knee were at 90 deg and the ankle was plantarflexed to 135 deg.

Because slackening is likely to occur in real-life stepping, and because tendon compliance is maximal at minimal force, it was important to pursue this issue in our trials. First, we had to clarify the precise point at which the muscle went slack. In our experiments, the muscle was pulled at an upward slant of about 20 deg to the horizontal. With progressive shortening, it slackened, in the sense that its belly sagged, but because its tendon was held up, some residual force (< 1 N) was

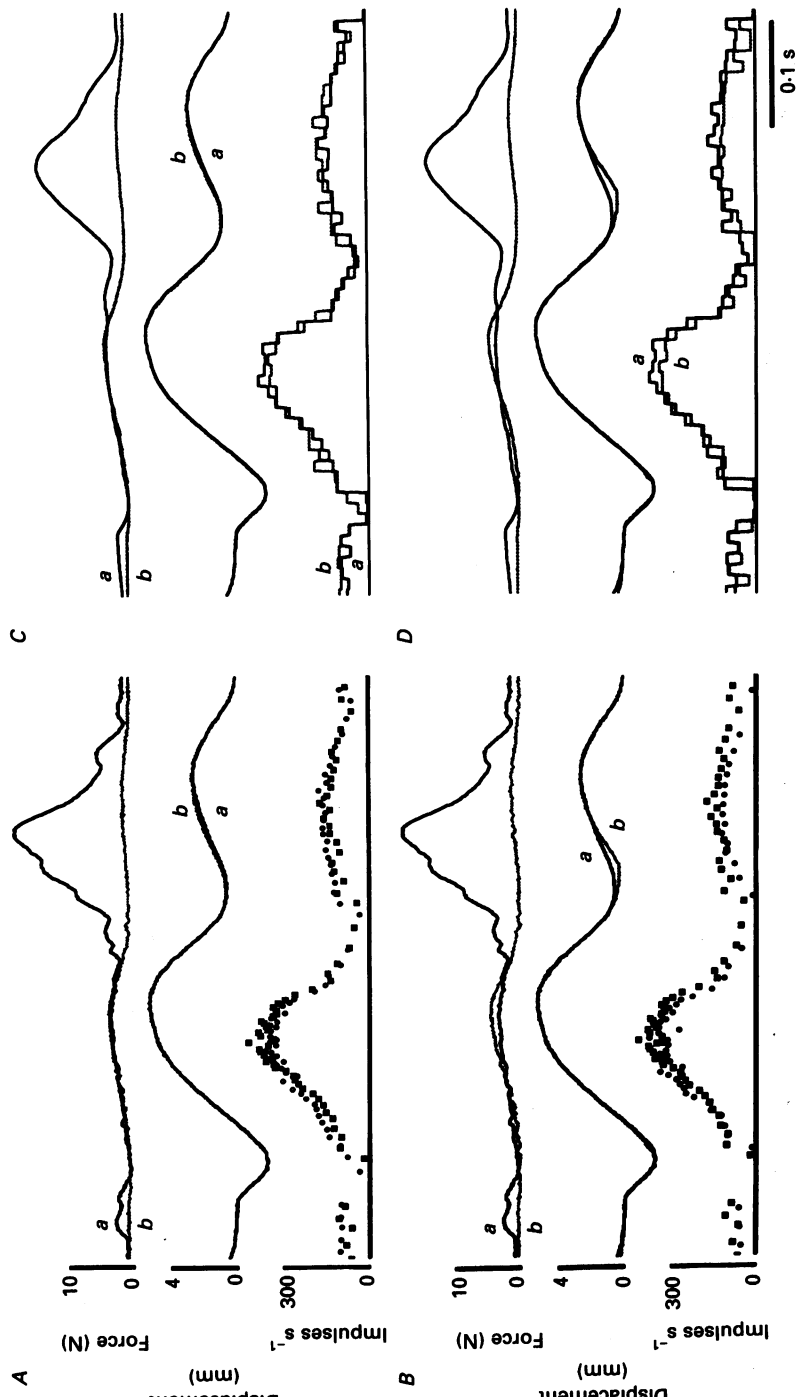

Fig. 7. Iteration experiment (as in Fig. 5) with the afferent of Fig. 6, under tonic fusimotor drive. *A* and *C*, active (*a* and ■) and passive (*b* and ○) cycles superimposed, showing small discrepancies in spindle firing. *B* and *D*, final iteration trials, indicating that the length adjustments had overcorrected the discrepancies thereby slightly overestimating extramysial stretch.

registered. Upon further shortening the tendon kinked, the muscle began to float in the leg pool, and eventually force dropped to zero. As there was no precise transition to zero force, we defined slack length as that at which the tendon began to kink during dynamic stretching. Under static conditions this corresponded to tendon

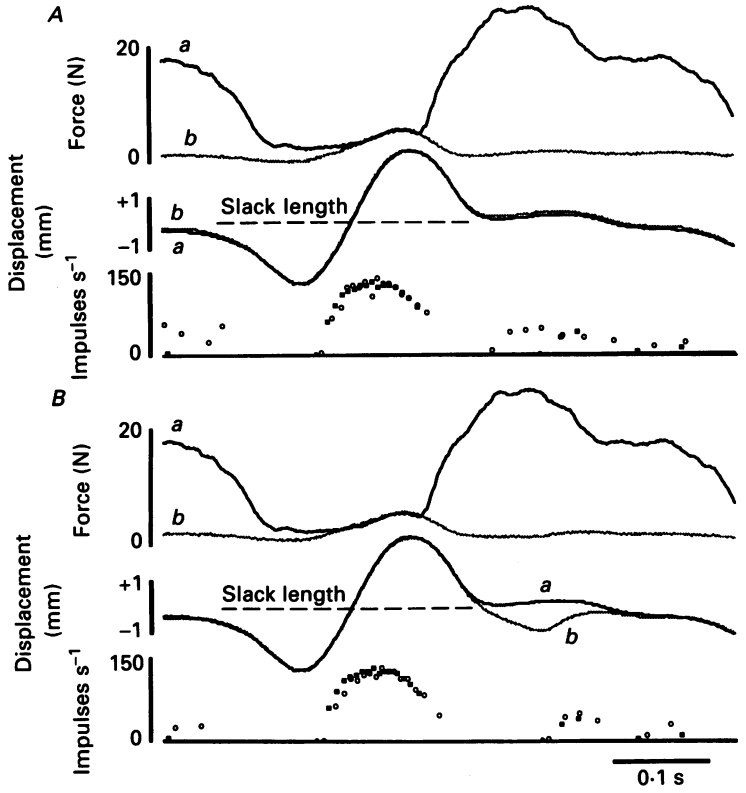

Fig. 8. Responses of a MG Ia afferent; shortening below slack length. *A*, active (*a* and ■) and passive (*b* and ○) trials. Note the large discrepancy during active tension. *B*, final iteration, showing that a substantial reduction in l_{oi} was required to correct for the discrepancy in *A*.

forces of 0·2–0·5 N. In normal stepping, MG muscle is oriented downwards at varying angles to the vertical and so even if force in the distal tendon dropped to zero, the proximal tendon would still be slightly loaded by the weight of the muscle.

We arranged for MG l_{oi} to decrease below slack length by up to half the total $l_{oi}(t)$ variation in each cycle. This probably exceeds the maximal slackening which would ever occur in real life, and thus represents a 'worst case' situation for spindle monitoring of l_{oi}. In such trials (Fig. 8*A*) there was indeed a clear difference in spindle firing with and without active force. The iterated result in Fig. 8*B* revealed a 1·1 mm discrepancy at the 25 N force peak, which, after subtraction of 0·4 mm 'hidden' shortening, gave a net extramysial l_e of 0·7 mm. Surprisingly, even though the muscle kinked very noticeably at shortest length in the passive trials, the spindle started responding to relengthening at well below the minimal 'slack length'. This

unexpected result, which was also mentioned in relation to the spindle II afferent in Fig. 4, will be dealt with in more detail in the discussion. With active force the relengthening response came 4–5 ms earlier, though the advance in this case was less than that in Fig. 4.

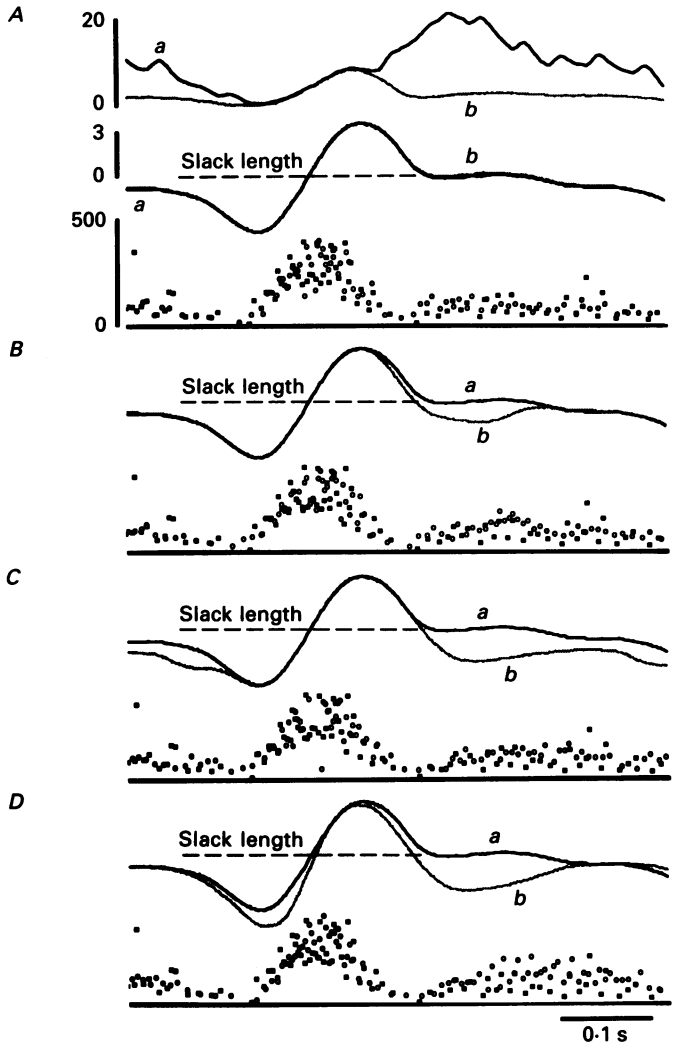

Fig. 9. Iteration sequence with shortening well below slack length. Responses of a MG Ia ending under tonic fusimotor drive. Active tension trial (*a*) is repeated in each panel, and superimposed with successive iterations (*A–D*). Consecutive increases in shortening only just correct for the small discrepancy in spindle firing at the time of peak active tension in *A*.

With the exaggerated slackening just described it was sometimes quite difficult to shorten the muscle enough in passive trials to match the firing in active force trials. In Fig. 9*A*, there was the usual small but clear shortfall in Ia firing during active force production. In successive passive iterations (Fig. 9*B*, *C* and *D*; *b* and ○), progressively larger length reductions in this part of the cycle were tried. The firing

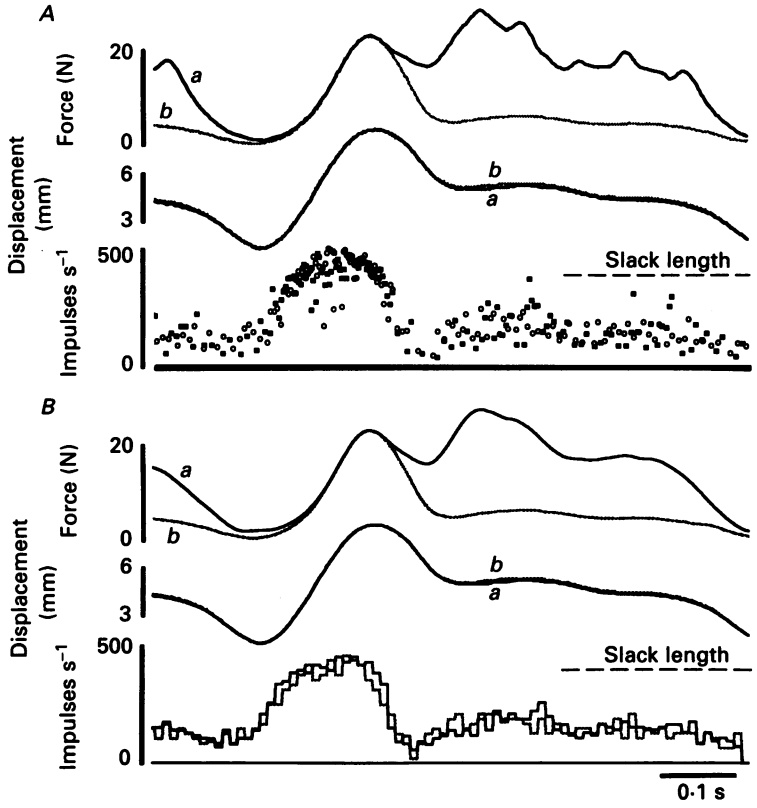

Fig. 10. Minimal discrepancy in step cycles at long muscle lengths. *A*, responses of the MG Ia afferent of Fig. 9 to active (*a* and ■) and passive (*b* and ○) trials where the muscle never shortened beyond a length 1·5 mm greater than slack length. The spindle firing discrepancy was very small, and only emerged in the off-line averages of *B*.

profiles were nearly matched in *B*, but a further large length adjustment was required for the good match in *C*. Reducing the length even more (*D*) did not reduce the passive firing below the active 'target' as desired. In retrospect this is not too surprising, because beyond a certain point the passive muscle started kinking, and so less and less of the externally imposed length change would have been transmitted to the spindle. Taken at face value, the inferred l_e after corrections for hidden compliance was 1·0 mm in *B* and 1·7 mm in *C*.

At the other extreme, with 5 mm of pre-stretch, which ensured that the muscle remained under tension throughout the cycle, there was very little difference in spindle firing in passive and active trials (Fig. 10). Indeed in this case the differences in individual trials were so small that they were obscured by the irregularity in firing, and so no iterations were performed (though with the benefit of the off-line histogram in *B*, the small discrepancies emerged more clearly).

DISCUSSION

In these experiments we have mimicked the length and force changes in MG muscle during the step cycle and indirectly estimated the amount by which in-series

extramysial compliance absorbs origin-to-insertion displacements $l_{oi}(t)$ thus distorting the signal 'seen' by muscle spindles. At short mean lengths the effect was greatest, the extramysial displacement l_e reaching about 0·5 mm in cycles corresponding to medium-paced stepping (force rising from 0·5 to 10 N). At longer

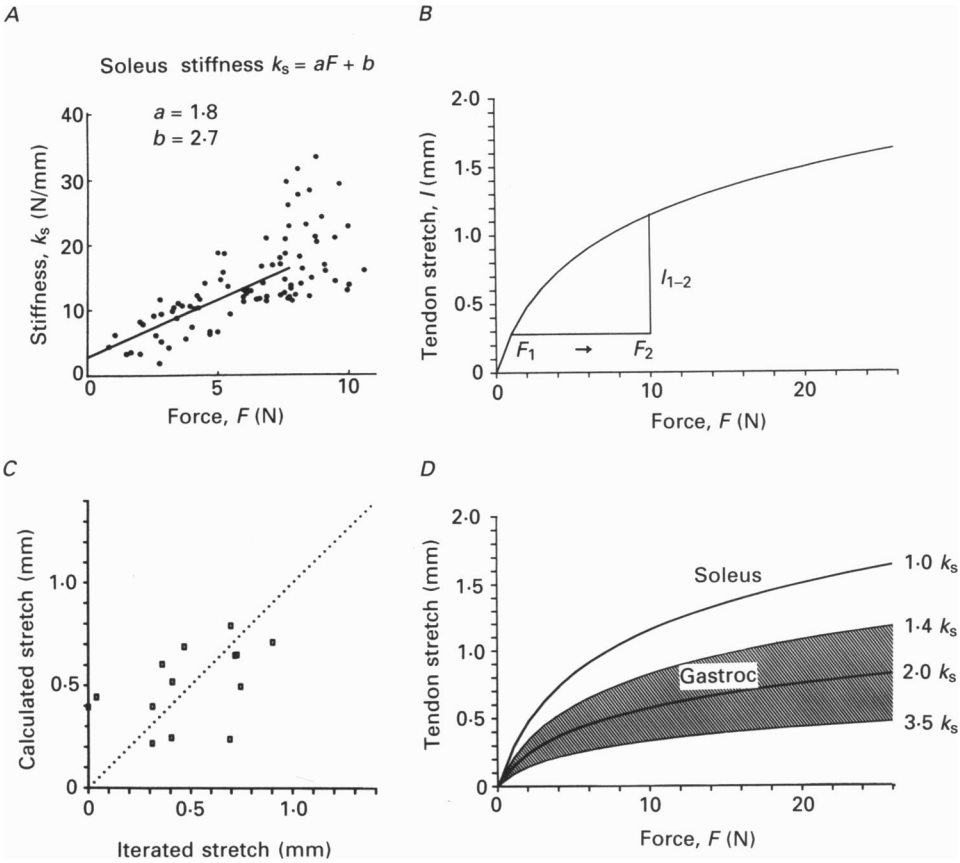

Fig. 11. *A*, stiffness k_s of cat soleus tendon, reproduced with permission from Rack & Westbury (1984). We fitted the regression line. *B*, calculation of tendon stretch expected from the regression line in *A*. Straight lines show the force increment (1–10 N) and resultant tendon stretch (0·9 mm) expected in a normal step cycle. *C*, plot of our experimentally derived estimates of extramysial stretch ('iterated stretch') against those calculated for a tendon of stiffness $2 \times k_s$ (points would all lie on diagonal if iterated stretch exactly matched stretch calculated for a tendon of stiffness double that of soleus). *D*, tendon stretch *vs.* force of tendons with stiffnesses 1·0, 1·4, 2·0 and 3·5 times that of soleus tendon respectively. Eighty per cent of our data points would be explained by a curve within the shaded area, the best fit being the central line.

mean lengths, where muscle force rose from say 2 to 12 N, l_e was in the range 0·2–0·4 mm. The dependence of l_e on the range of force is consistent with the dependence of extramysial stiffness on force inferred with the spindle null technique in cat soleus by Rack & Westbury (1984). We have coined the term extramysial to make it clear that the in-series compliance may be due to slackening, torsional displacement, and changes in pinnation angle as well as tendon strain. Figure 11*A*

shows Rack & Westbury's data, with a regression line calculated between 0 and 7·5 N. The regression line is described by a simple linear equation relating the inferred stiffness of soleus tendon k_s to force, with the coefficients a and b as shown in Figure 11.

Predictions of l_e from spindle null model

The spindle null technique provides a measure of the small-signal stiffness (i.e. force increment/0·25 mm length increment) under dynamic conditions (2 Hz stretching) so it is easy to develop an analytical expression for the cumulative length change l_{1-2} associated with a force change from F_1 to F_2:

since $dl = dF/\text{stiffness}$, and $\text{stiffness} = aF + b$,

$$dl = \frac{dF}{aF+b}$$

so

$$l_{1-2} = \int_{F_1}^{F_2} \frac{dF}{aF+b},$$

$$l_{1-2} = \frac{1}{a} \ln\left[\frac{aF_2+b}{aF_1+b}\right]. \quad (1)$$

The cumulative extramysial displacement predicted by this model is plotted against force in Fig. 11*B*. For a typical step cycle where force rises from say 1 to 10 N after foot contact, the displacement would be 0·9 mm, as shown. Now if the extramysial displacement can simply be read off the plot, one might ask why it was at all necessary to do the experiments described in this paper. In fact there are several reasons. First, the scaling of the curve in Fig. 11*B* is quite sensitive to the value of the intercept b in Rack & Westbury's stiffness *versus* force plot (Fig. 11*A*); the intercept is extrapolated from a limited number of fairly scattered data points, and cannot be considered to be definitive. Second, the use of small-signal stiffness to estimate cumulative displacement assumes linear behaviour and needs validation with large displacements. Third, in the step cycle, $l_{oi}(t)$ has a higher mean frequency content than the 2 Hz used in the spindle null experiments, and from Rack and Westbury's comparisons of static and dynamic testing, this might be associated with a higher stiffness. Last, the data of Hoffer *et al.* (1989), suggesting a 2·25 mm extramysial displacement of MG in the early stance phase of the step cycle (and up to 5 mm in late stance), differed so much from the 0·9 mm prediction above that an independent test mimicking the step cycle was clearly necessary.

Comparison of our l_e estimates with those of the model

Because we could easily measure the net force variation occurring in each of our iterated step cycles, it was possible to compare our experimentally derived length increments with those predicted from the model in Fig. 11*B*. This is shown in Fig. 11*C*: we found that by doubling the stiffness of the model (doubling a and b) we obtained a reasonably good fit to our data. This indicates that the incremental stiffness of MG tendon at any given force is approximately double that estimated for soleus tendon by Rack & Westbury (1984). Near the calcaneum, the cross-sectional area of MG tendon is about double that of soleus. Assuming similar Young's Moduli

of MG and soleus tendons (Bennet *et al.* 1986), MG tendon should have double the stiffness per unit length of soleus tendon. Now if MG tendons are also 50% longer than those of soleus (Walmsley & Proske, 1981) their overall stiffness should be about 33% larger (note that with the α-method, Walmsley and Proske found roughly equal tendon stiffnesses in soleus and MG).

Figure 11*D* summarizes the tendon displacement *versus* force profiles derived above. The top curve is a reproduction of that for soleus tendon (Fig. 11*B*). The other curves were obtained by multiplying the soleus stiffness by the scaling factors 1·4, 2·0 (best fit) and 3·5. Eighty per cent of our estimates of l_e due to specific force increments would lie within the shaded area.

The α-method

At this point we should briefly consider the alternative method of establishing muscle and tendon strain, the so-called α-method of Morgan (1977). In its original formulation, tendon compliance was assumed to be constant. Mean MG compliance derived by Walmsley & Proske (1981) using the α-method was 0·06 mm N^{-1}. So a 10 N change in force would result in 0·6 mm tendon displacement. This is in close agreement with our results in cases where force rose from zero, but overestimates the displacements we saw when initial force was higher. Proske & Morgan (1987) recently conceded that at forces up to 20–30% maximal isometric (i.e. 16–24 N for MG) tendon stiffness increased with force. Above 30% maximal force, it was suggested that stiffness might be invariant, and that the spindle null technique of Rack & Westbury (1984) lacked the resolution to disprove this. Peak MG force in fast running does not exceed 24 N (Walmsley *et al.* 1978), so for our purposes the approximation of a linear relationship between stiffness and force in Fig. 11*A* seems reasonable.

Non-tendinous extramysial displacement

Caputi *et al.* (1989) suggested that variations in pinnation, deformation of aponeurotic sheets and muscle slackening could all contribute to extramysial displacement. In our experiments, if the muscle did not go slack, extramysial displacement was that expected of tendon alone, with a stiffness twice that of soleus. We cannot rule out contributions from pinnation and aponeurotic deformation, because our estimates were of total displacement from all sources; but any sharing of displacement would imply an even great MG tendon stiffness than we inferred.

Exaggerated slackening in our trials did produce significant discrepancies between l_m and l_{oi} (up to 1·7 mm). But the corresponding changes in spindle firing were actually quite minor. We commented earlier that in trials where l_{oi} went 2 or 3 mm below slack length (e.g. Figs 4 and 8), Ia firing resumed quite early in relengthening, even in the absence of active force. There is no easy explanation for this. Perhaps during shortening some residual elastic strain energy in the muscle fibres is released, taking up some of the slack of the kinked tendon, thereby reducing the 'dead zone' upon relengthening. This, combined with the very high small-amplitude sensitivity of spindle afferents might explain the early resumption of firing. Whatever the mechanism, the effect is in fact to reduce the discrepancy in spindle firing caused by this form of extramysial displacement.

How significant is the extramysial displacement?

Energy storage

The strain energy E in an elastic element of invariant compliance stretched l by a force F is:

$$E = \frac{Fl}{2}.$$

A tendon whose compliance is double that of its in-series muscle fibres would stretch twice as much, and from the equation would store twice the energy (Alexander & Bennet-Clark, 1977). But as we have seen, tendon compliance decreases as force increases. Also, the compliance of active muscle increases with force once strain exceeds 1% or so (Rack & Westbury, 1974). This means that tendon stretches relatively more at low force, absorbing little energy. At high force, stretch (and therefore energy) shifts to muscle (see Appendix). But even this is far from general, for example during the load-bearing E_2 phase of the step cycle, MG muscle actively resists stretch; in slow steps, l_{oi} increases by as little as 0·5 mm (e.g. Fig. 2). All of this stretch and therefore all of the energy would be absorbed by the tendon (the muscle fibres remain isometric not because they are infinitely stiff but because their operating point varies with activation level (Houk & Rymer, 1981)). This shows that the proportions of strain energy absorbed by muscle and tendon depend on the kinetic and kinematic details of movements (Zajac, 1989). In the Appendix we estimate that MG tendon stores about 2 mJ in the E_2 stretch phase of a medium-paced step. Over 90% of this is probably recovered during E_3 shortening. How much of the net work done by MG in E_3 does this represent? Whiting *et al.* (1984) estimated that MG contributed 8 mJ during E_3 shortening in slow steps. But in view of the rapid decline in force and the small concomitant l_{oi} excursions in E_3 (Gregor, Roy, Whiting, Lovely, Hodgson & Edgerton, 1988; Walmsley *et al.* 1978; Figs 4 and 7 above), this may be an overestimate. So the 2 mJ recoverable from MG tendon represents at least 20% of the total requirement. This supports the idea that strain energy stored in tendons contributes significantly to gait (Cavagna & Kaneko, 1977; Morgan, Proske & Warren, 1978; Alexander, Maloiy, Ker, Jayes & Warui, 1982).

Significance for inferred fusimotor action

The current hypotheses about what muscle spindles signal during voluntary movement and how they are controlled by fusimotion are based in part upon comparisons between spindle afferent firing and $l_{oi}(t)$ as shown in Fig. 2. Hoffer *et al.* (1989) pointed out that large discrepancies between $l_{oi}(t)$ and $l_m(t)$ could seriously undermine both the qualitative (e.g. Loeb, Hoffer & Pratt, 1985) and quantitative inferences about fusimotion (e.g. Prochazka, Hulliger, Zanger & Appenteng, 1985; Hulliger *et al.* 1987). However, our present data indicate that the discrepancies are smaller than claimed, and that the attendant changes in spindle discharge are subtle, and sometimes scarcely detectable. This is in marked contrast to the prominent changes in spindle sensitivity and bias upon which, for example, the 'fusimotor set' hypothesis is based (Prochazka *et al.* 1985). On the other hand, it is quite true that

in specific situations, even a small extramysial stretch introduces some uncertainty as regards the underlying fusimotor action. For example, in Fig. 2 just after foot contact, mean gastrocnemius l_{oi} increased by no more than 0·3 mm. If we assume 0·5 mm extramysial displacement, l_m would undergo a net 0·2 mm shortening. Yet both group Ia and II endings increased their firing at this time. Up to a point, this would be expected to occur even with a tonic fusimotor background (e.g. Figs 9 and 10), but a small phasic (α-linked) component of fusimotor drive cannot be ruled out from the spindle firing profiles of Fig. 2. On balance, we reject the notion that extramysial displacement has introduced any serious or fundamental errors in inferences about fusimotor control to date, because these have been based upon very large changes in spindle firing. However, it can obviously introduce small errors and thereby restrict the resolution obtainable with indirect methods.

APPENDIX

Strain energy stored in tendons with force-dependent stiffness

From eqn (1), for a tendon of stiffness $aF+b$, the length change l due to an increase in force from 0 to F is

$$l = \frac{1}{a}\ln\left[\frac{aF+b}{b}\right],$$

rearranging terms,

$$F = \frac{b}{a}(e^{al}-1).$$

The strain energy E is given by:

$$E = \int_0^l F\,dl,$$

$$= \frac{b}{a}\frac{(e^{al}-1)}{a} - \frac{bl}{a}$$

$$= \frac{F}{a} - \frac{bl}{a}.$$

For the parameters of MG tendon referred to in the paper $F = 10$, $l = 0{\cdot}5$, $a = 3{\cdot}6$, $b = 5{\cdot}4$ and $E = 2{\cdot}05$ Nmm (mJ).

Note that for a tendon of invariant stiffness 20 N mm^{-1}, resulting in the same final force and stretch, the strain energy would be $Fl/2 = 2{\cdot}5$ mJ.

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