

Critical Sarcomere Extension Required to Recruit a Decaying Component of Extra Force during Stretch in Tetanic Contractions of Frog Skeletal Muscle Fibers

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ABSTRACT 29 single frog skeletal muscle fibers were stretched during fused tetanic contractions. The force increase during stretch exhibited a breakpoint at a critical length change (average: 16.6 nm per one-half sarcomere) that was independent of velocity of stretch and of sarcomere length between 1.8 and 2.8 μm . After stretch there was an early decaying force component with a force-extension curve similar to that during stretch, which disappeared over ~ 2 s. This component was removed by a small, quick release, leaving a longer-lasting component. The critical amplitude of release required to produce this result was found by clamping the fiber to a load at which there was zero velocity of shortening. This amplitude increased with time up to the angle in the force record during stretch, was constant for the remainder of the stretch, and decreased with time after the end of stretch; it was consistently less than the critical amplitude of stretch required to reach the breakpoint of force enhancement during stretch but was also independent of sarcomere length. The force drop accompanying the critical release showed a small increase up to an optimum magnitude at 2.4–2.7 μm sarcomere length, with a decrease at longer lengths.

INTRODUCTION

Stretch of a contracting muscle at a moderate velocity causes tension to increase above the isometric level (Fenn, 1923; Abbott and Aubert, 1951; Hill and Howarth, 1959; Deleze, 1961; Cavagna and Citterio, 1974; Sugi, 1972; Flitney and Hirst, 1978*a* and 1978*b*). This phenomenon was confirmed and further investigated in a previous study on single muscle fibers (Edman et al., 1978*a*).

The present study extends our previous work (Edman et al., 1978*a*) and elucidates some aspects of the phenomena resulting from stretch in more detail. Special emphasis is put on the time and length dependence of the force

enhancement after stretch and on the relationship between force enhancement during and after stretch. Some of the results have been reported in preliminary communications (Edman et al., 1978*b* and 1980).

METHODS

Experiments were performed on single muscle fibers from the semitendinosus and the tibialis anterior muscles of *Rana temporaria*. The fibers were studied with the same techniques as described previously (Edman et al., 1978*a*).

Fibers were mounted in a muscle chamber between an electromagnetic puller and a tension transducer. In some of the early experiments an RCA 5734 mechano-electric transducer (RCA Corp., New York) was used (Edman et al., 1978*a*). In later experiments tension was measured with a semiconductor strain-gauge transducer (AE 801; Aksjeselskapet Mikroelektronikk, Horten, Norway) as described by Edman (1979). The chamber contained 8 ml of the bathing solution (NaCl, 115.5 mM; KCl, 2.0 mM; CaCl₂, 1.8 mM; Na phosphate buffer, 2.0 mM; pH 7.0). Between experiments the bath temperature varied from 0.8 to 3.8°C. During the experiments temperature was maintained to within $\pm 0.2^\circ\text{C}$. Temperature control was obtained by circulating a water-glycol solution through the jacket of the muscle chamber from a Colara Ultrathermostat (Colara Messtechnik, GMBH, Lorch, West Germany). The solution in the chamber was exchanged at least once every 2 h. For stimulation, rectangular pulses of 0.2-ms duration were delivered between two platinum plate electrodes placed symmetrically on either side of the fiber. A train of pulses (frequency: 8–22 Hz) of 1-s duration was applied every 2 min. Alternatively, longer tetanuses lasting between 5 and 8 s were produced at 5-min intervals.

Sarcomere length was measured by the laser diffraction technique described by Cleworth and Edman (1972). A 1.5-mm diameter light beam was shone through the fiber and the diffraction pattern was displayed on a ground-glass screen. Sarcomere length measurements were read to 0.02 μm accuracy from this calibrated screen. In some experiments, movements of the first-order beam were recorded on continuously moving 35-mm film (Gevapan 36; Agfa-Gevaert) at 50 or 100 mm/s with a Grass oscilloscope camera (Grass Instrument Co., Quincy, Mass.). This permitted measurements of internal shortening and sarcomere length during the tetanus. Measurements were made at 0.5-mm intervals along the fiber. Changes in mean sarcomere length were calculated from changes in fiber length and from careful measurements of both sarcomere length and fiber length in the resting fiber when it had just become taut. The resting-fiber length was measured to the nearest 0.05 mm in a Zeiss Stereo II microscope (Carl Zeiss, Inc., Oberkochen, West Germany) at 10 \times magnification. Tension and length changes were displayed on a Tektronix 5103N storage oscilloscope (Tektronix, Inc., Beaverton, Ore.) and photographed on 35-mm film. Measurements from the film (oscilloscope and laser diffraction recordings) were made on a Nikon model 6C (Nippon Kogaku K. K., Tokyo) comparator at using the stage-micrometer readings.

All force measurements were corrected for passive force. After recording force during the tetanus on the storage oscilloscope a fresh trace was recorded at the same fiber length in the resting state. If a stretch had been applied during the tetanus, the same stretch was applied to the resting fiber immediately before the tetanus to provide the baseline. These resting forces were subtracted from tetanic force to obtain the results presented here.

RESULTS

Force Enhancement during Stretch

Force responses during stretch at one velocity were studied in 17 fibers. Velocity of stretch was varied in a further 12 fibers. At the onset of stretch force rose very steeply. For stretches exceeding a critical amplitude a break-point occurred in the force record and the steep rise was followed by a plateau (Fig. 1). The amplitude of stretch needed to obtain the angle in the force trace was found to be little dependent on the velocity of stretch in all fibers studied (Fig. 1 *B*). The results of four fibers in which a very wide range of velocities was applied are presented in Fig. 2 *A*. The force at the angle was dependent on velocity (Fig. 1 *B*). This dependence of force enhancement during stretch on velocity of stretch is shown for the same four fibers in Fig. 2 *B*. The amplitude of stretch required to reach the angle in the force trace was also independent of sarcomere length between 1.8 and 2.8 μm as shown for two velocities of stretch in one fiber in Fig. 3 *A*; the means and standard deviations for altogether 15 fibers are shown in Fig. 3 *B*. The average magnitude of the critical amplitude averaged over all sarcomere lengths was 16.6 ± 3.4 (SD) nm per one-half sarcomere.

Decay of the Force Enhancement after Stretch

After the end of the stretch force decayed but remained elevated (inset of Fig. 3 *A*; see also Edman et al. [1978 *a*]). As previously emphasized (Edman et al., 1978 *a*), this force after stretch is independent of the velocity of preceding stretch.

The relationship between the preceding-stretch amplitude and the force enhancement *after* stretch was studied. An example, superimposed on the length-tension relationship obtained from isometric tetanic contractions, is shown in the *upper-left inset* of Fig. 4 *B*. Soon after the end of stretch (100 ms), the relationship between the amplitude of stretch and the enhanced force consisted of two parts: an apparent stiff component that was recruited by small stretches and a more compliant portion that was apparent when larger stretches had been used. This relationship is also shown in Fig. 4 *B* of Edman et al. (1978 *a*). The relationship had similar characteristics to those of the force record during stretch (Fig. 1), i.e., a sharply rising portion, angle, and flatter portion. The force decayed for >2 s after the end of the stretch leaving, at longer sarcomere lengths, a residual force enhancement after stretch (Fig. 6 of Edman et al. [1978 *a*]).

The critical amplitude of stretch needed to attain the angle of the relationship between the amplitude of stretch and the enhanced force early after stretch was determined in six experiments at different lengths. The results obtained were similar at the different sarcomere lengths studied within the range between 1.80 and 2.80 μm (Fig. 4 *A*). The mean critical amplitude after stretch for these six fibers was 11.5 nm per one-half sarcomere (± 1.78 SD). This value is significantly ($P < 0.05$) lower than the 18.0 nm per one-half

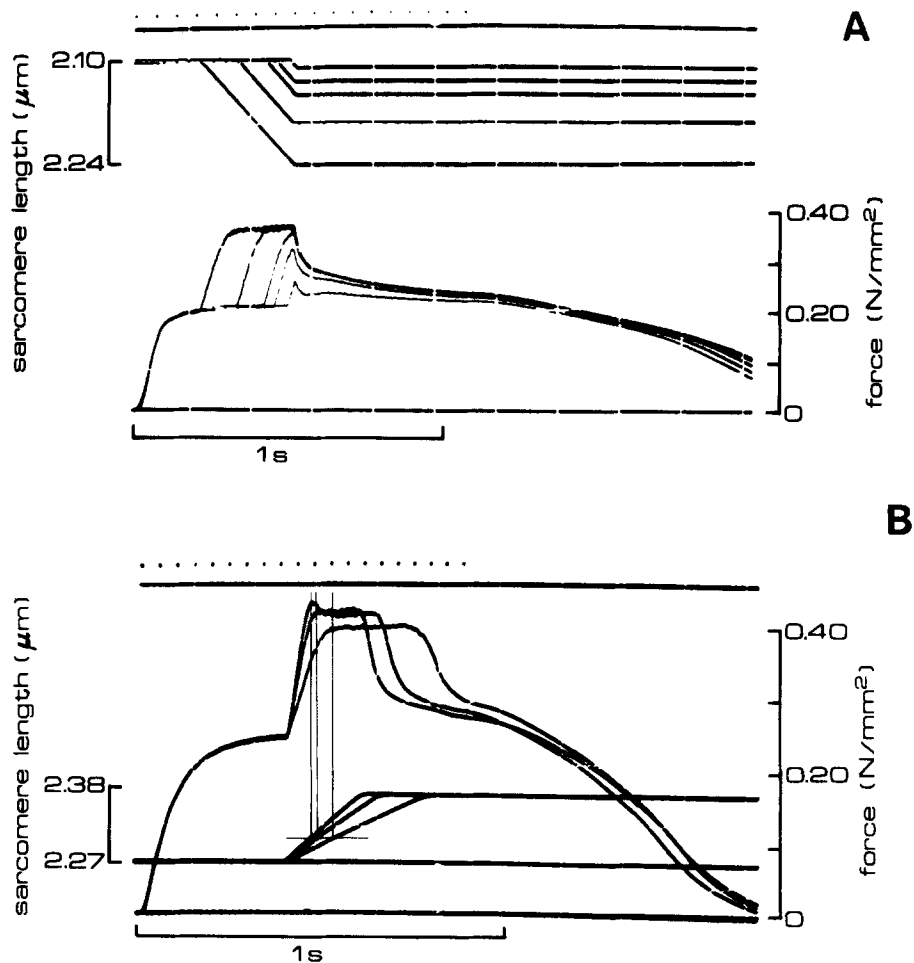
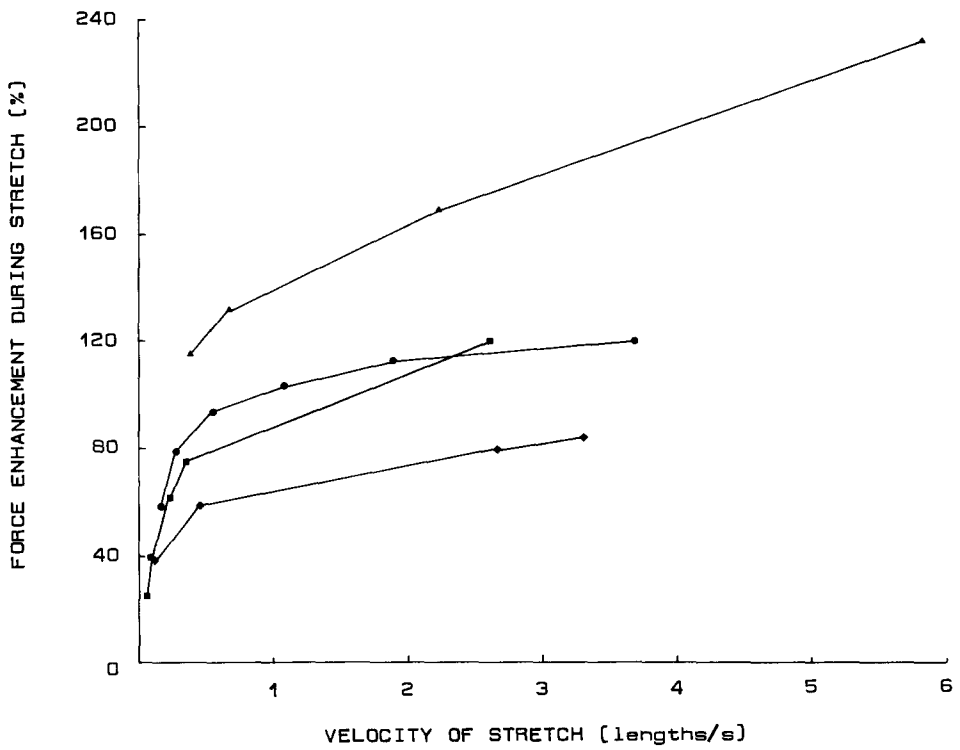
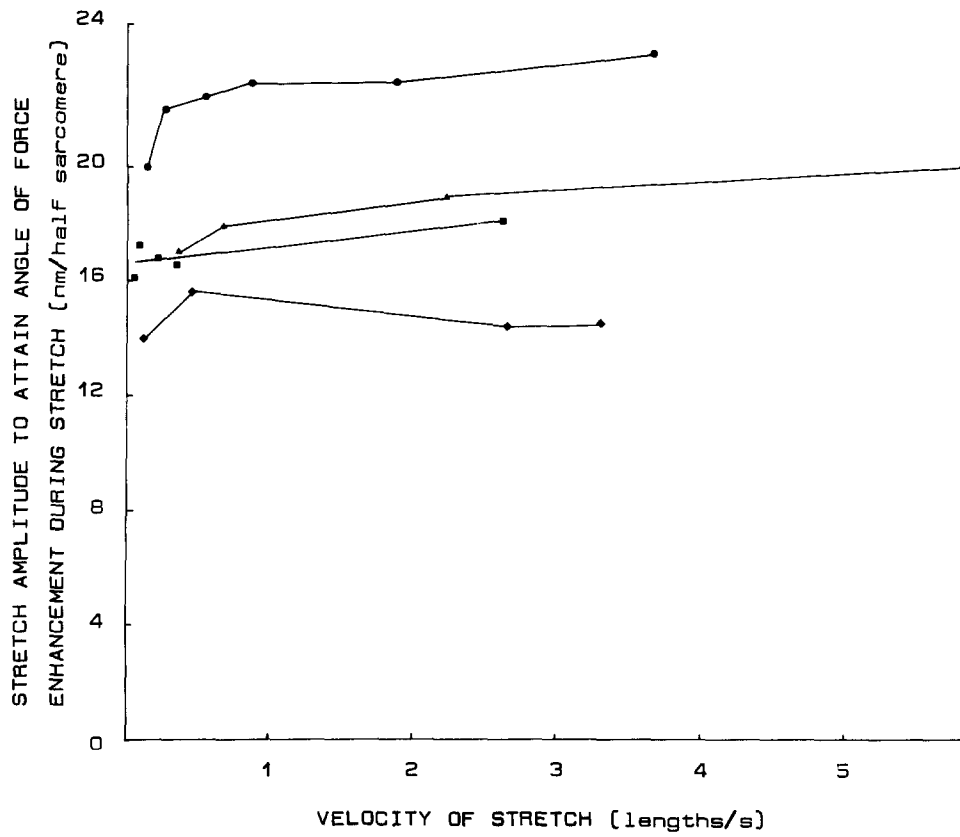


FIGURE 1. Changes in force as a result of stretch of a single fiber during tetani of 1-s duration. (A) The effects of different amplitudes of stretch are superimposed; these amplitudes in terms of sarcomere length are indicated by the length records above the myograms. Note that beyond a critical amplitude of stretch there is an angle in the force record during stretch and an identical decaying force after stretch. Fiber length, 8.9 mm at 2.25- μ m sarcomere length; temperature, 2.6°C; cross-sectional area, 15.7×10^{-3} mm². (B) The effects of different velocities of stretch are superimposed; these are indicated in terms of sarcomere length by the length records superimposed on the myograms. Note that the force during stretch increased with increased velocity of stretch and that the critical amplitude of stretch required to reach the angle in the force record during stretch is not influenced by velocity of stretch. Fiber length, 12.1 mm at 2.25- μ m sarcomere length; temperature, 1.62°C; cross-sectional area 7.9×10^{-3} mm².

FIGURE 2 (opposite). (A, top) The critical amplitude of stretch required to obtain the angle in the force record during stretch in four fibers, plotted against a wider range of velocities of stretch than those used in Fig. 1B. (B, bottom) The corresponding values for force enhancement during stretch.



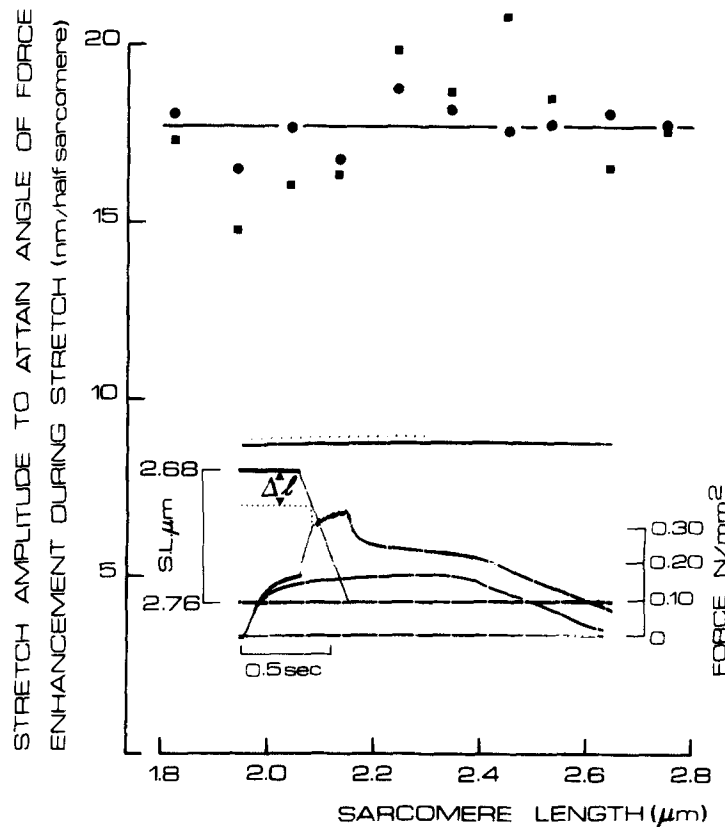


FIGURE 3. The amplitude of stretch needed to attain the angle of the force enhancement during stretch measured at different sarcomere lengths. The method of analyzing the tracings is shown in the *inset* of *A*. $\Delta \ell$ equals the critical stretch amplitude plotted on the ordinate of the main graphs. (*A*) Measurements for two different velocities of stretch in one fiber at 2.3°C. Stretch: ●, 0.14 lengths/s; ■, 0.22 lengths/s. Fiber length, 9.05 mm at 2.25- μ m sarcomere length; cross-sectional area, 5.4×10^{-3} mm². (*B*) For each fiber the mean value of the critical amplitude measured at different sarcomere lengths was taken as 100%. 15 fibers were used for the analysis. The number of fibers studied at each length is given in brackets. Bars, ± 1 SD.

sarcomere found for the critical amplitude during stretch in the same six fibers (Fig. 4 *B*). For these six fibers the mean critical amplitude during stretch was slightly greater than the overall mean of 16.6 nm for the 15 fibers of Fig. 3 *B*.

These values were calculated from the overall length changes in the fiber and they therefore averaged all the sarcomere length changes along the fiber. It is necessary, however, to make a correction for tendon compliance (see Discussion).

Further experiments were carried out to explore the nature of the decaying component of force enhancement after stretch.

Critical Release Required to Remove the Early Decaying Component of Force Enhancement after Stretch

The discrepancy between the critical amplitudes (see above) might be accounted for by assuming that they were the same at the end of stretch, but

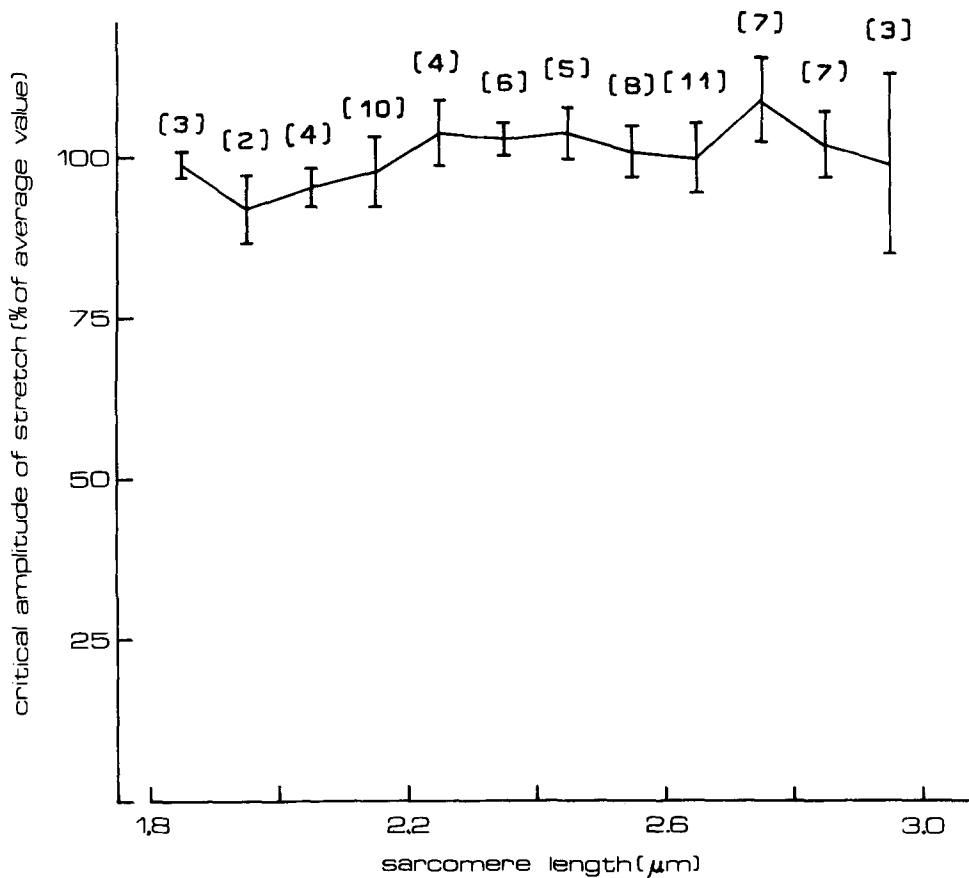


FIGURE 3B.

that there was some decay before the measurement of critical amplitude after stretch could be made. It was found in four preliminary experiments that the decaying component of force enhancement after stretch could be removed by a small, quick release (Edman et al., 1980). To determine the critical amplitude of release that just removed this component completely, load clamps were applied in nine fibers after stretch by the same method that was used previously to determine force-velocity curves (Edman et al., 1978a). The load

was adjusted so that velocity of movement, after the initial shortening step, was zero (Fig. 5). It was found that the same load was required for this condition for any time after the end of stretch that the load clamp was applied. However, the shortening step that occurred at the onset of such load

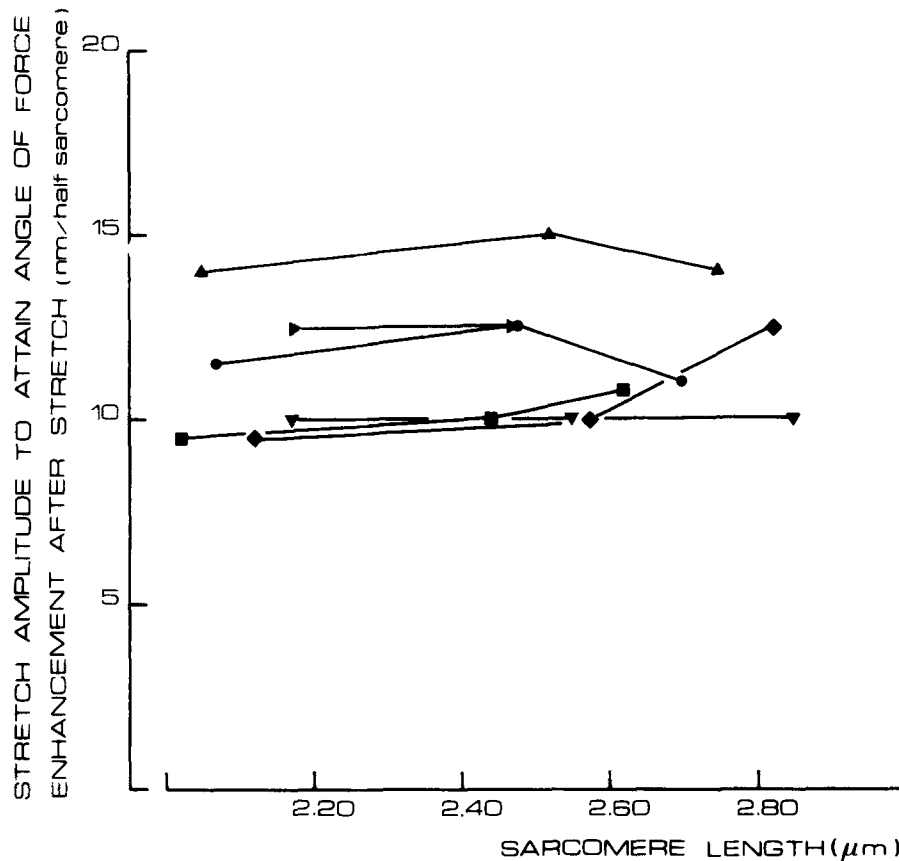


FIGURE 4. The amplitude of stretch needed to attain the angle in the relationship between the stretch amplitude and the enhanced force early after the end of stretch. The method of analysis is shown in the *upper-left inset* of *B* where part of the length-tension curve is shown (●) together with points obtained early after stretch (★), using different amplitudes of stretch as in Fig. 1*A*. The different symbols in *A* and *B* indicate different experiments ($n = 6$). (*A*) Relationship to sarcomere length. (*B*) Comparison with stretch amplitude to attain angle of force record during stretch. Insets indicate methods of analysis for the two axes.

clamps, i.e., the critical amplitude of release, decayed with time after the end of stretch (Fig. 5 and Fig. 6).

To explore the relationship between the critical amplitude of release and the critical stretch required to reach the angle in the force record during stretch, load clamps to zero velocity were performed during stretch in four

fibers. The results of such an experiment are shown in Fig. 6A. The critical release increased in amplitude during the initial part of stretch up to the angle in the force record, remained constant during the plateau phase of the force record during stretch, and decayed after the end of stretch (Fig. 6A). There was a steep initial fall in force after the end of stretch that was not accompanied by a correspondingly steep initial decline in critical release (Fig. 6A). The critical release during stretch was always clearly less than the stretch required to reach the angle in the force record (Fig. 6B).

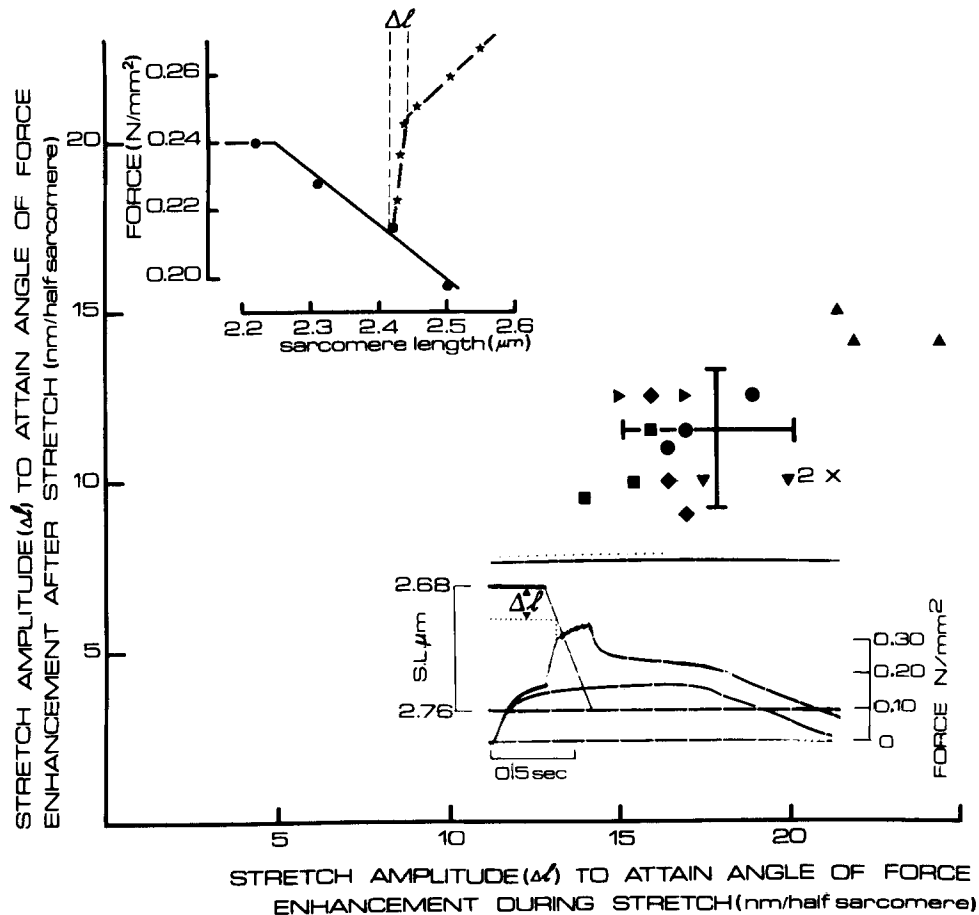


FIGURE 4B.

Sarcomere Length Dependence of the Early Decaying Component of Force Enhancement after Stretch

The critical release required to remove the decaying component of force enhancement was studied in fibers over the range of sarcomere length 1.95–3.15 μm . As can be seen for five fibers in Fig. 7, the critical amplitude was little affected by sarcomere length.

The drop in force accompanying the critical release showed a variable increase with sarcomere length from 2.0 to 2.4–2.7 μm and decreased at longer sarcomere lengths (Fig. 7B). The total force in the fiber early (~ 20 ms) after the end of the stretch, at sarcomere lengths up to 2.7 μm , was higher than the isometric tetanic force at 2.1 μm and reached a maximum at ~ 2.4 μm (also see Fig. 4 of Edman et al., 1978a).

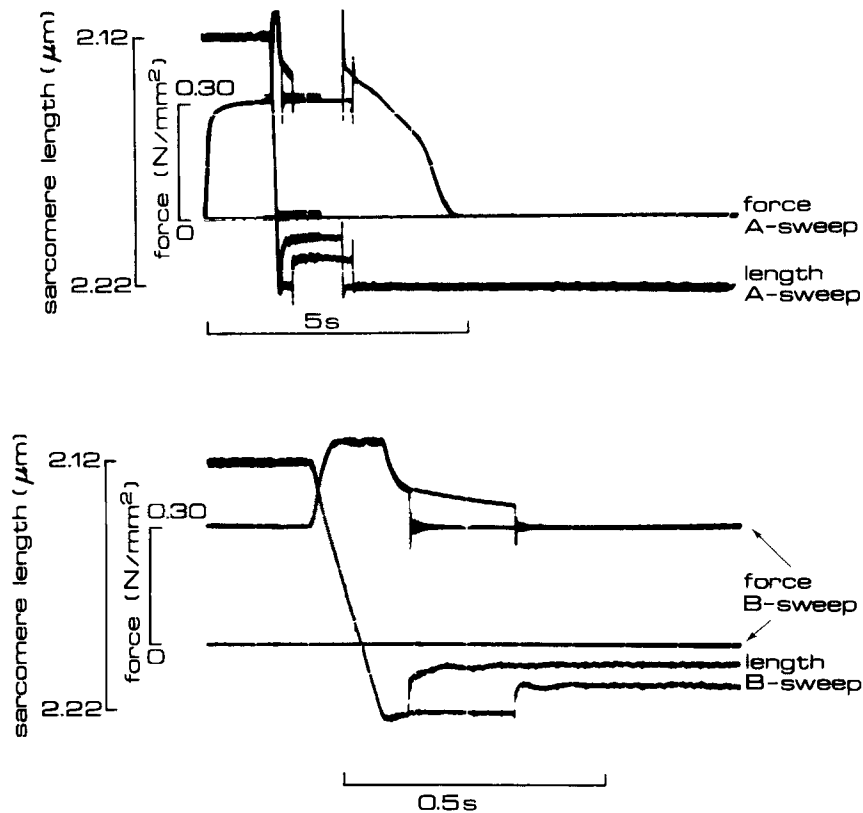


FIGURE 5. Force and fiber length recorded at two different sweep speeds: *A*, slow; and *B*, fast. The *A* sweep shows the whole myogram. The *B* sweep starts just before the application of the stretch; the *arrows* indicate clamp force and zero force. Two identical stretches are superimposed. Each stretch was followed by a release to a zero-velocity load clamp. Two different times of release are shown. Note that the amplitude of the release was smaller for the later release. Fiber length, 9.3 mm at 2.25- μm sarcomere length; temperature, 2.25°C; cross-sectional area, $18.1 \times 10^{-3} \text{ mm}^2$.

Stress-Strain Relationship of Component Decaying after Stretch

The stress-strain characteristic of the decaying component after stretch was explored in 12 fibers. For this purpose the relationship between the amplitude of release and the amplitude of the accompanying force drop was determined

at various times after the end of stretch using the critical release technique (Fig. 5). The example shown in Fig. 8 was a fiber in which the relationship between force change and release was explored at different sarcomere lengths. A curvilinear stress-strain relationship was found at all sarcomere lengths studied.

DISCUSSION

The idea that the response of a contracting muscle fiber to an externally applied stretch consists of several components has recently been proposed in a preliminary communication (Edman et al., 1979). In the present paper we present in detail some of the features of the response to stretch that are associated with forces above the isometric tension exerted at optimum sarcomere length (Fig. 4 of Edman et al., 1978*a*). This force enhancement is therefore a property of the sarcomeres when they are in the stretched state.

Relationship between a Component of Force Enhancement during Stretch and the Early Decay of Force Enhancement after Stretch

The force enhancement during stretch and the early decay of force enhancement after stretch may be considered to comprise a single functional component. This concept is supported by the findings that: (a) the breakpoint in the force record during stretch occurs at a given stretch amplitude independent of velocity of stretch (Figs. 1 and 2*A*); (b) a similar breakpoint also occurs in the relationship between force enhancement soon after stretch and the amplitude of stretch (Fig. 4*B*, upper left inset); (c) the force enhancement soon after stretch is removed by a critical amplitude of release that, after an initial rapid decline in force, decays with the same time-course as the force (Fig. 6*A*); and (d) all these critical distances (a-c above) are independent of sarcomere length (Figs. 3, 4, and 7). Such independence of sarcomere length rules out the possibility that the results are influenced by the small variations in sarcomere length along the fiber.

This component appears to behave as an elastic element up to a critical strain, beyond which sarcomere extension (and presumably filament sliding) occurs with much reduced stress increment. At the plateau of the length-tension curve, the component seems to be extremely compliant after the breakpoint and to become stiffer at longer sarcomere lengths (Fig. 4*B* of Edman et al. [1978*a*]). Thus, the stress-strain characteristics contain a striking plastic characteristic (Fig. 9).

We assumed that the decay of force and of critical release after the end of stretch represents the return of this viscoelastic element down the steep part of its stress-strain curve. The latter was obtained by plotting the force drop accompanying the release against the amplitude of the release. The stress-strain curves for a number of different sarcomere lengths in one fiber are shown in Fig. 8. The stress-strain relationship is non-Hookean.

The finding that the breakpoint in the force record during stretch occurs after a given stretch amplitude, independent of sarcomere length and velocity of stretch, suggests that this critical amplitude results from an interaction of fixed periodic structures along thick and thin filaments. The critical amplitude

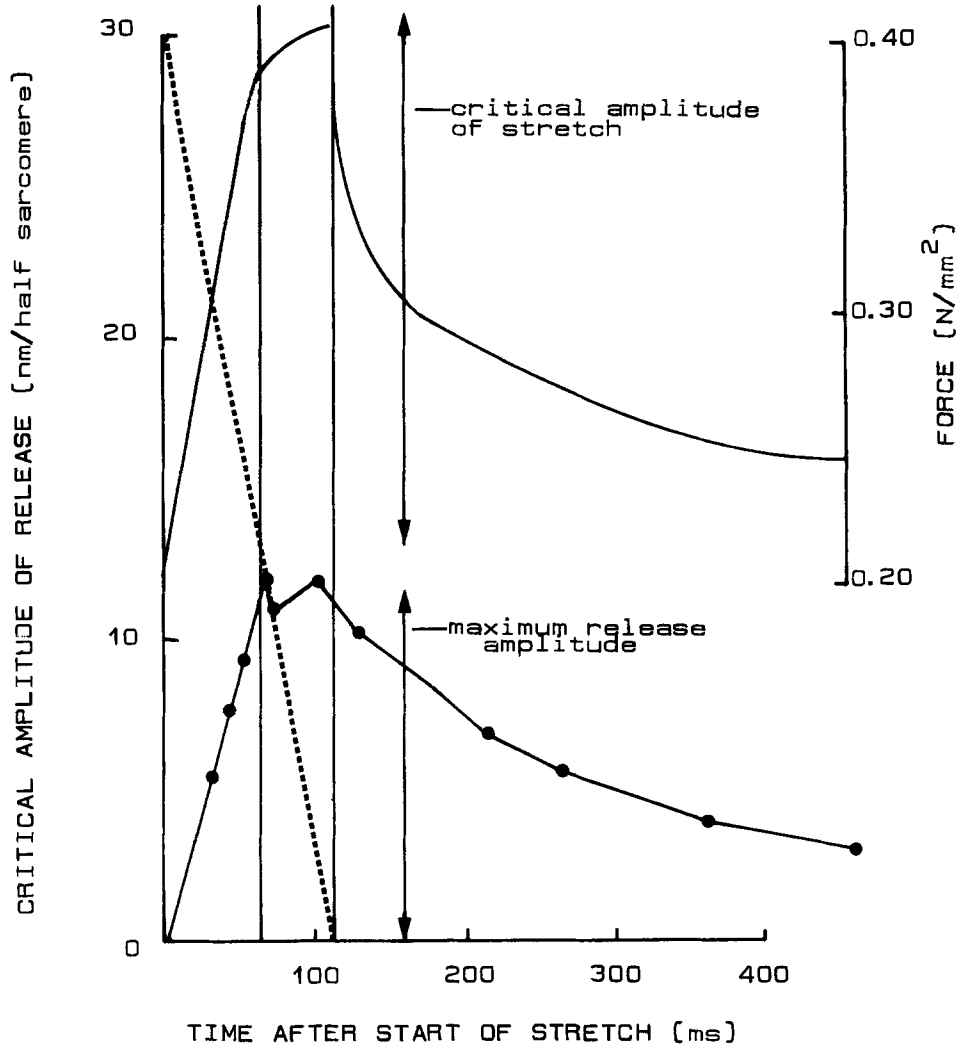


FIGURE 6. Data from experiments with interventions as shown in Fig. 5. (A) The amplitude of critical release (*filled circles*) is plotted against time after the onset of stretch in one fiber. Force myogram (*upper continuous line*) and fiber length change (*dashed line*, same scale as critical release) superimposed. Vertical lines indicate time of angle in force record and time of end of stretch. Fiber length, 7.1 mm at 2.25- μm sarcomere length; temperature, 2.1°C; cross-sectional area, $22.2 \times 10^{-3} \text{ mm}^2$. (B) Critical release amplitude plotted against time from the onset of stretch; each symbol represents a different fiber. Shaded area encloses the times when the angles in the force records were attained in these four fibers. The end of stretch is indicated by the vertical line (*arrow*). The amplitudes of stretch required to reach the breakpoints in the force records during stretch are indicated by the separate symbols within the shaded area.

of stretch may represent the amount of sliding required to disrupt this interaction.

The scatter in our data for this measurement is a limitation that may be overcome in the future by the use of sarcomere-length- or segment-length-clamp methods. Our value of 17 nm would include a contribution from tendon compliance of ~ 2 nm (Cleworth and Edman, 1972). Thus, we would estimate the critical sliding to be ~ 15 nm. This is greater than the 12 nm found by Flitney and Hirst (1978*a* and 1978*b*) and considerably greater than predicted by the stress-strain relation of the cross-bridges (Bressler and Clinch, 1974; Ford et al., 1977). However, the critical release found at the force angle

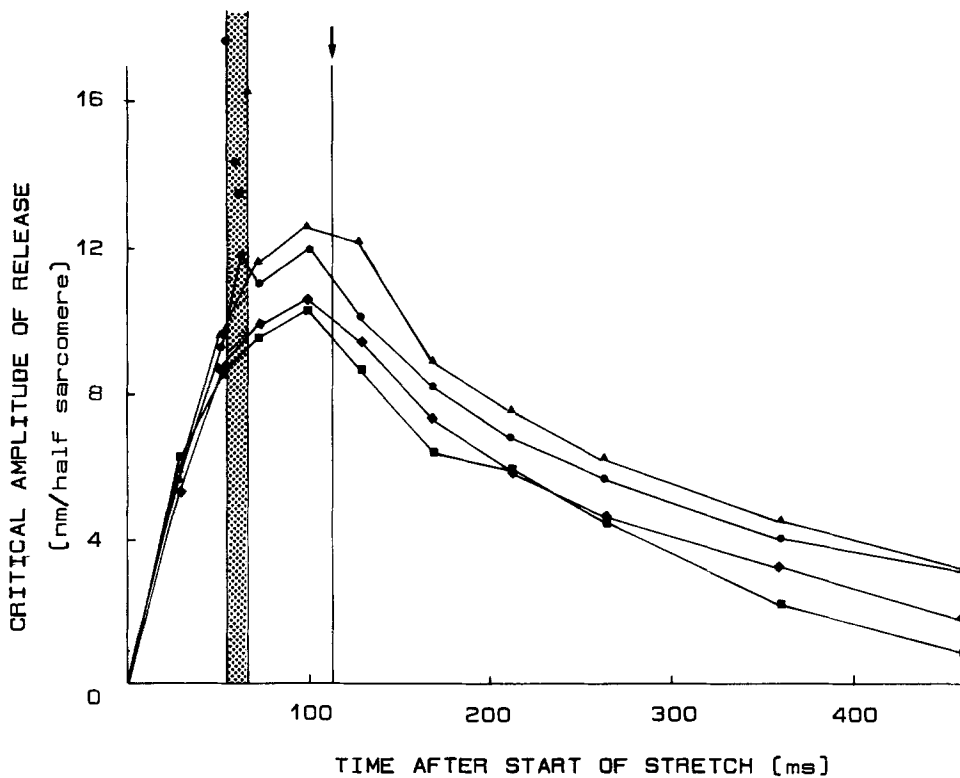


FIGURE 6B.

during stretch was 4 nm less than the stretch required to reach the breakpoint in the force record (Fig. 6*B*), i.e., ~ 11 nm. This value of 11 nm is therefore pertinent to the stress-strain curve explored after stretch (Fig. 8), and its interpretation in terms of cross-bridges depicted in Fig. 9.

The extra extension present during stretch (Fig. 6*B*) and the extra force that accompanies it (Fig. 6*A*) indicates the presence of an extra component *during* stretch. In contrast to the component that persists and decays *after* stretch, the extra force during stretch is velocity dependent, indicating a

viscous phenomenon. One could postulate that the critical release at the force angle during stretch (Fig. 6 *B*) represents the amplitude required to stretch the cross-bridges until the point where they break from the thin filaments and slide (Fig. 9).

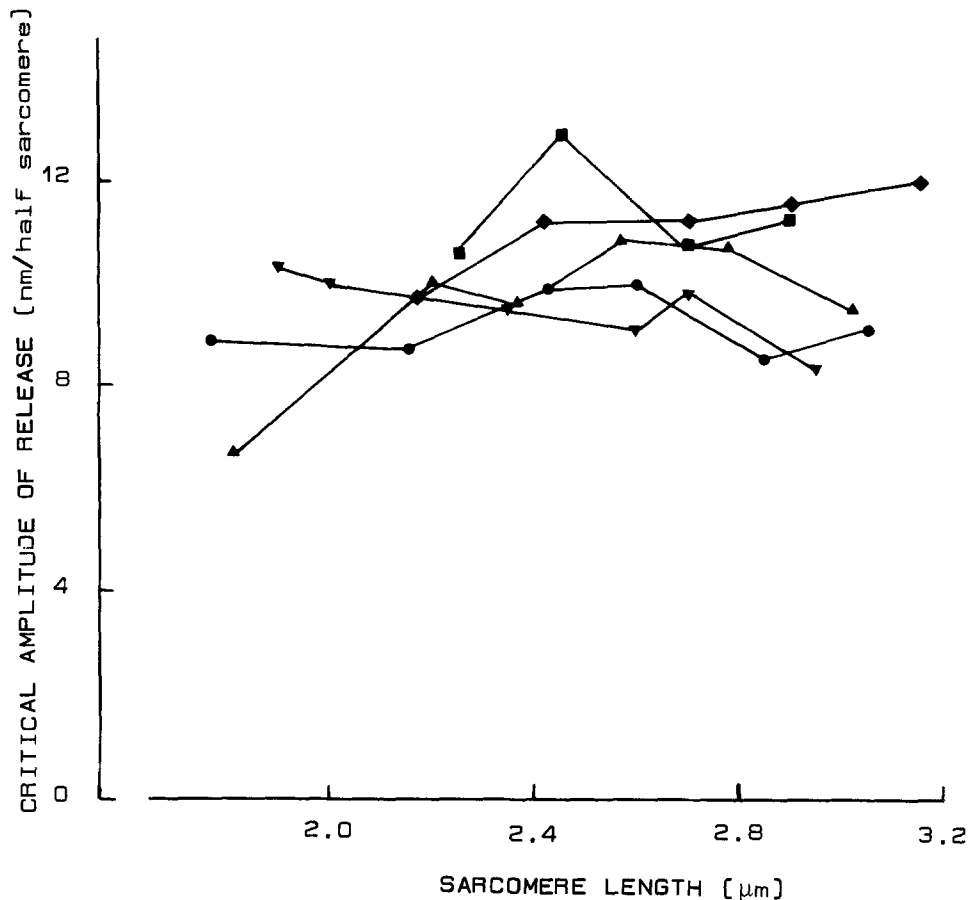


FIGURE 7. Critical release after stretch carried out at different sarcomere lengths. Data from five experiments with interventions as shown in Fig. 5 but with the time of release fixed. (*A*) Release amplitude; (*B*) associated change in force. *Dashed line in B* represents schematic typical isometric length tension curve for comparison.

The above considerations lead to the conclusion that interactions of regularly spaced active sites along the filaments must be involved. However some features of our results require us to postulate additional features of a simple model of this type:

(*a*) The stress-strain curve of our recruited component (Fig. 8) is non-Hookean and shows much greater compliance than obtained for cross-bridges

by Bressler and Clinch (1974) and Ford et al. (1977). The reason for this discrepancy is unclear. However, the larger (and slower) stretch used in our experiments may be thought to involve a back-rotation of the myosin head in addition to a strain of the bridge structures. We also cannot exclude the possibility that the greater strain measured here (Fig. 8) includes some extension of the thin filaments and Z disks. The difference between the critical stretch and critical release distances (Figs. 4 *B* and 6 *B*) is unclear but may be

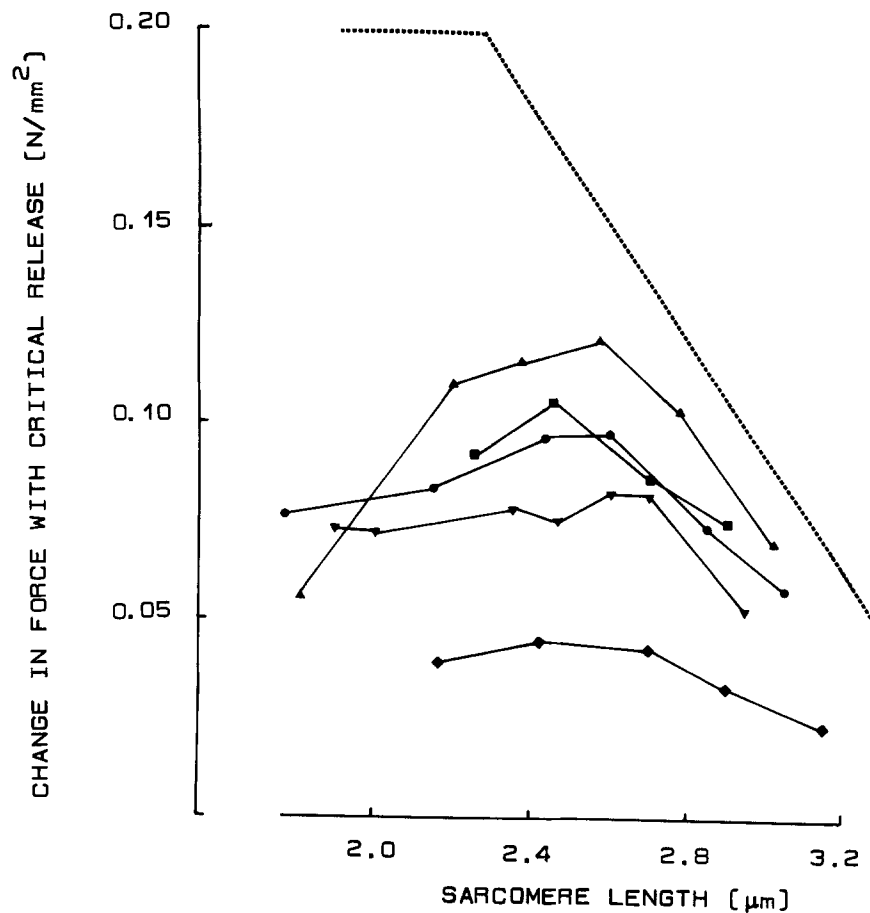


FIGURE 7B.

related to the adoption of regular orientation of the structures by stretch as suggested in Fig. 9.

(b) We would need to assume that the cross-bridges remain fully stretched during sliding so that they are still fully stretched at the end of the applied extension (Fig. 9). One would expect such sliding to reduce the opportunity for cross-bridge attachment as it does with shortening. This is the explanation

for the inverse relationship between force and shortening velocity (Huxley, 1957). A similar inverse relationship between force and lengthening velocity does not exist. In fact, there is a positive relationship (Katz, 1939; Aubert, 1956; Edman et al., 1978*a*; Flitney and Hirst, 1978*a*).

(c) During stretch, we would expect decreased cross-bridge cycling, in accordance with the decreased heat production (Fenn, 1923; Abbott and Aubert, 1951; Hill and Howarth, 1959; Curtin and Davies, 1972; Curtin and Woledge, 1979).

The time-course of stress and strain decay after the end of stretch is slow,

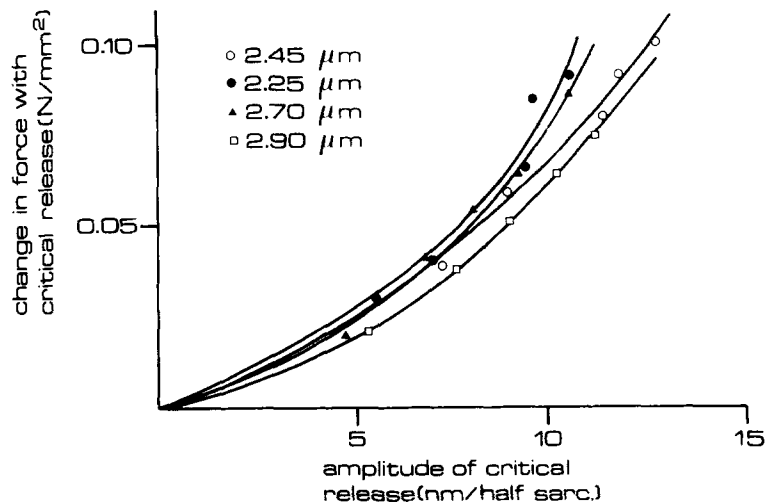


FIGURE 8. Data from an experiment similar to that shown in Fig. 5. For each sarcomere length studied the change in force with release is plotted against the release amplitude for releases at different times after the end of stretch. These relationships are the apparent stress-strain curves of the decaying component of force enhancement after stretch. Fiber length, 8.8 mm at 2.25- μm sarcomere length. Temperature, 1.0°C; cross-sectional area, $11.8 \times 10^{-3} \text{ mm}^2$. Similar results were obtained in a further four fibers.

taking ~ 2 s. We need to assume that this slow decay implies slowed detachment and therefore decreased cycling of cross-bridges. In Fig. 9 we illustrate the possibility that stretch brings the cross-bridges into a fixed attitude. It takes ~ 2 s for a return to the prestretch conditions. This hypothesis requires the assumption of decreased cross-bridge cycling (and, therefore, of ATP splitting) during the early force decay after stretch. However Curtin and Woledge (1979) found an increase in heat production during the early decay phase of force enhancement after stretch (early decaying component plus residual force enhancement). The increase in ATP splitting after stretch was not statistically significant, but Curtin and Woledge thought it probable that if they could resolve the early decaying part they would find an increased ATP splitting at that time,

matching the increased heat output. This increased heat production and ATP splitting would be consistent with increased cross-bridge cycling, whereas our mechanical measurements lead us to postulate reduced cross-bridge cycling.

(d) The decaying component of force enhancement after stretch showed an optimum sarcomere length at 2.4–2.7 μm (Fig. 7 B). This result confirms our previous finding that the total force during stretch exhibits a maximum at a

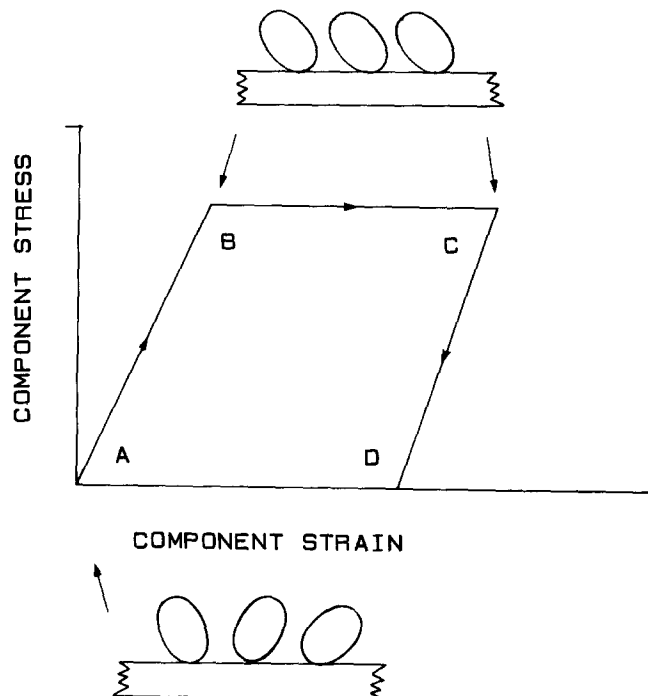


FIGURE 9. Scheme of the apparent stress-strain characteristics of the component responsible for the angle in the force record during stretch and the decaying component of force after stretch. Hypothetical positions of cross-bridges are indicated before and during stretch. *A*, start of stretch; *B*, angle in force record during stretch; *C*, end of stretch; *D*, return of force enhancement.

sarcomere length of 2.4 μm (Fig. 4 of Edman et al. (1978*a*)), i.e., a considerably greater length than the optimum for the isometric force. The effect of stretch therefore appears not to be a simple function of the overlap between the thick and thin filaments but involves recruitment of some other, overriding factor.

These experiments were performed at the Department of Pharmacology, University of Lund, Lund, Sweden.

Supported by the Swedish Medical Research Council (project 14X-184) and by a Wellcome Trust European Collaborative Grant.

Received for publication 4 May 1981.

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