VELOCITY TRANSIENTS AND VISCOELASTIC RESISTANCE TO ACTIVE SHORTENING IN CAT PAPILLARY MUSCLE

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ABSTRACT When isotonic force steps were applied to activated papillary muscles, the velocity was almost never constant. Early rapid shortening associated with the step persisted for 2–7 ms after the step ends. The early rapid shortening is attributed to lightly damped series elastic recoil and velocity transients of the contractile elements. In most steps, the subsequent velocity declines progressively with shortening, and most of the decline in velocity can be accounted for by compression of a viscoelastic element in parallel with the contractile elements. To demonstrate this, the time course of isotonic velocity was compared with a model in which the force-velocity characteristics of the contractile element were assumed to be constant, and the decline in velocity was due to increasing compression of the viscoelastic element. This model predicted the observed results except that the predicted velocities rose progressively above the measured values for steps to light loads applied late in the twitch, and fell below the velocity trace for heavy loads applied early in the twitch. These deviations would occur if rapid shortening caused deactivation late in the twitch, and if activation were rising early in the twitch. A conditioning step applied to the muscle during the rise of force depressed both isometric force and maximum velocity measured at the peak of force; isometric force was more depressed with later conditioning steps than with earlier steps, while maximum velocity was depressed by about the same extent with both early and late steps. This difference between the effects on isometric force and maximum velocity are explained by a combination of deactivation and viscoelastic load.

INTRODUCTION

Physiological experiments frequently seek to isolate and study individual variables by holding other variables constant. Isotonic contractions of skeletal muscle are a good example. Both contractile element force and series elastic element length are presumed constant, so that overall muscle force and velocity directly reflect contractile element force and velocity. Considerations of the arrangement of passive elastic structures have suggested that these presumptions may be inappropriate in studying cardiac muscle (Hefner and Bowen, 1967; Pollack 1970). In 1968, Jewell and Blinks pointed out that the complications arising from these passive properties could be minimized if velocities were measured immediately after quick release to an isotonic load. Studies of this sort using mechanical levers (Brady, 1965; Sonnenblick, 1965, 1967; Edman and Nilsson, 1968) were complicated by the levers oscillating on the compliant tendons immediately following a step. More recent experiments have suggested that these oscillations might inactivate the muscle (Bodem and Sonnenblick, 1974; Edman and Nilsson, 1972). In an effort to resolve these questions, we have made a servo system capable of applying rapid, critically damped force or length steps to papillary muscles. When isotonic steps were applied to activated papillary muscles, the velocity was almost never constant following the step. The preceeding paper (Chiu et al., 1982) showed that very rapid early shortening could be accounted for by rapid shortening of a lightly damped, nonlinear, series spring and presented evidence that there is likely to be a substantial viscoelastic element in parallel with the contractile elements. The present paper shows that the later changes in isotonic velocity can be explained largely by the parallel viscoelastic elements. As with all theories, this model cannot be proved absolutely. We only show that the model can account for the data, and that certain mechanisms are inconsistent with the results.

The methods and muscles used are the same as in the preceeding paper (Chiu et al., 1982).

RESULTS

The general form of the twitch contraction with an isotonic force step applied at the peak of force is shown in Fig. 1. A servo system held the muscle at a constant length until a predetermined time in the twitch. After this time, force was held constant at a level equal to a preselected fraction of the isometric value before the step while the muscle shortened against this constant force. To prevent excessive shortening, the muscle length was held constant after it shortened a specified amount so that it contracted isometri-

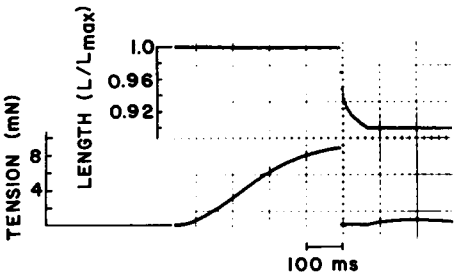

FIGURE 1 Time course of isotonic release. The muscle was stimulated to contract isometrically until a predetermined time and then released to an isotonic load. After reaching a pre-set length, the muscle was again held isometrically and force partially recovered. Muscle length 6.0 mm, weight 1.2 mg.

cally again to increase the force slightly before the muscle relaxed fully. The muscle was stretched to its original length when the muscle had relaxed. The muscles were held at a constant length and stimulated regularly for at least seven isometric twitch contractions before another isotonic contraction was studied to allow the muscle to reestablish its base line state and also for the computer to store the acquired data.

The changes that occur in association with different size steps made during three separate contractions are shown at a 20 times faster recording speed in Fig. 2. The first 2 ms are base line values recorded at the time the muscle was stimulated. Recording is stopped and restarted 2 ms before the step, so that the second 2 ms are isometric values recorded immediately before the step was applied. The rate of change of force during the steps was initially rapid and then declined progressively. The steps were 98% complete in <2 ms for the largest steps, and in <1 ms for the smaller steps. The changes in the length records become more obvious when these records are differentiated.

Velocity Changes

The gains of the velocity records are amplified more in steps to heavier loads to provide approximately the same degree of velocity resolution in different sizes of step (Chiu et al., 1982). These records show three common properties, which are illustrated in Fig. 2 *C*. (*a*) The rapid shortening associated with the force step continued well beyond the time the step ended, with no discontinuity associated with end of the force step. This early rapid shortening is more prolonged in large steps than in small steps and is attributed to shortening of a lightly damped, nonlinear series elastic spring (Chiu et al., 1982). (*b*) the deceleration of muscle length is not always a monotonic process, but frequently reaches a minimum within 3 ms after the end of the step and achieves a second maximum. (*c*) After these early transient changes, the isotonic velocity is not constant, but changes continuously. The time course of these changes depends on step size and time in the contraction cycle.

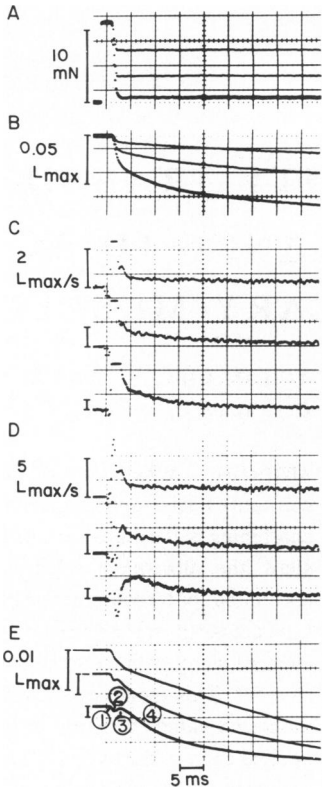

FIGURE 2 Three force steps applied to the active muscle. *A*, three force records superimposed. *B*, length. *C*, velocity. *D*, contractile element velocity (overall velocity minus calculated series elastic recoil), at 2.5 times greater gain. *E*, contractile element shortening (integral of contractile element velocity). The records are spliced so that the first 2 ms are recorded at the time of muscle stimulation and recording is then halted until the 2 ms before the step. First 2 ms are therefore base line values recorded at the instant of stimulation, and the next 2 ms are isometric values recorded immediately before the step. Note the gains of the velocity and integral traces are increased in steps to higher loads. In Fig. *C*, the very high velocities during the rapid recoil saturate the analogue output from the computer so that the records appear as short horizontal lines until the velocity declines to the range of the output amplifier. This truncation is not seen in *D*, even though the gain is higher, because the velocity attributed to series elastic recoil has been substracted. The four phases of the transients are labeled on the lowest record in *E*. Muscle length 5.4 mm, weight 2.0 mg.

Velocity Transients Associated with a Step. The shortening of the damped series was calculated and subtracted from the velocity records as previously described (Chiu et al., 1982). The constants describing the series elastic spring and its damping, as well as those used to obtain the calculated records in Fig. 6 and 8, are given in Table II of the preceding paper (Chiu et al., 1982). The corrected velocities are shown at a 2.5 times greater gain in Fig. 2 *D*. If the series elastic recoil has been described properly, the corrected velocity record should represent the contractile element velocity. As shown in Fig. 2 *D*, there is an initial rise in this velocity at the onset of the step, a decline to a minimum 2–3 ms after the onset of the step, and rise to a second maximum ~5 ms after the step. These

early velocity transients are similar to those of skeletal muscle near 0°C (Podolsky, 1960; Civan and Podolsky, 1966; Huxley and Simmons, 1972) except that they have a faster time course. To compare the velocity changes with the published records from skeletal muscle, the corrected velocity trace was integrated to show the time course of contractile element length, (Fig. 2 *E*). (Note that the gain of these length records is increased with heavier loads.) The four phases of the transients described by Huxley and Simmons (1972) are labeled on the lowest record Fig. 2 *E*. There is an immediate, rapid, small shortening associated with the step (phase 1), followed by an additional small, rapid shortening after the step ends (phase 2). The most conspicuous aspect of the record is the abrupt slowing, or even slight lengthening, of the muscle (phase 3). The steady state isotonic shortening (phase 4) continues for the remainder of the record.

The effects of these transient changes in contractile element velocity are less obvious but still apparent in the velocity records before the correction for series elastic recoil. The deceleration of the muscle (i.e., the second derivative of the length record) is not a monotonic process; velocity slows to a nearly constant value between 4 and 8 ms following the release, and declines at a more rapid rate after 8 ms (Fig. 3). This plateau in the velocity records, corresponding to phase 3, was most obvious, and always seen in steps to intermediate loads, as shown in Fig. 3; at low loads the persistence of the damped recoil obscured the velocity plateau, while at high loads, velocities were too low to resolve a plateau. This transient pause in the decline of velocity could not be caused by any series combination of monotonically decelerating processes, and further supports the interpretation that the contractile elements undergo the transient velocity changes shown in Fig. 2 *D*.

Afterloaded Contractions. The effects of series elastic recoil were minimized in afterloaded contractions, where the muscle was allowed to develop force isometrically to a certain level and then allowed to shorten against the same force. Because there was no change in force level, the series element did not change length, so the length data reflected directly the contractile element length. The length traces from these afterloaded contractions share several features with those obtained after quick releases, as shown in Fig. 4. Because there is no abrupt change in force, there is no distinct phase 1, but there is an initial rapid shortening associated with the transition from isometric to isotonic state (phase 2). Shortening slows to a minimum value after several ms (phase 3), and then accelerates to a second maximum (phase 4). In contrast to steps where the force is suddenly lowered, the late rise of velocity is more prolonged and the maximum velocity is not achieved for many milliseconds.

Isotonic Shortening. Corrected velocity reached a minimum shortly after end of the step, and rose to a maximum before beginning a second decline that continued throughout the remainder of the isotonic phase (Fig. 2 *D*). With heavy loads applied early in the contraction, the velocity maximum did not occur until after the normal 50 ms period of recording. The afterloaded contractions shown in Fig. 4 are an example of this, and the prolonged rise of velocity early in contraction is probably due to rising activation. In most isotonic steps, however, contractile element velocity achieved its maximum 5 to 7 ms after the onset of the step, and declined progressively thereafter. The deceleration was greater when the muscle was allowed to shorten faster, as in the case of shortening against very low loads. This deceleration was most pronounced when the step was applied later in the contraction. The length records shown in Fig. 2 *E* are an example of the greater

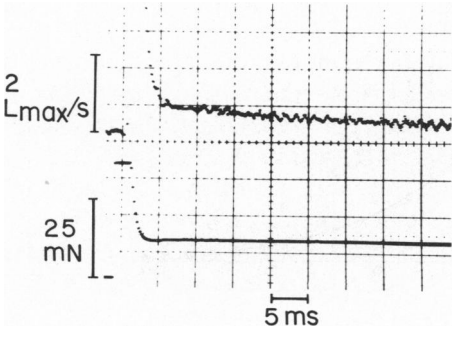

FIGURE 3 Transient velocity responses. The velocity record has not been corrected. Steps made from 100% L_{max} and at 300 ms after stimulus, shortly before the time of peak isometric force. The deceleration in the velocity trace slows between 4 and 8 ms after the onset of the step.

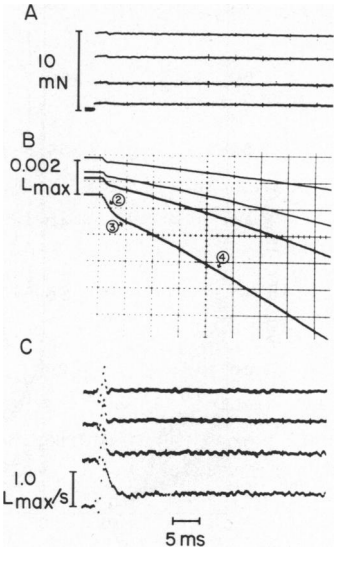

FIGURE 4 Four afterloaded contractions. *A*, superimposed force records. *B*, length records. *C*, velocity records. Muscle acceleration is manifested as an upward convexity of the length records in *B* and a rise of the velocity records in *C*. Phases 2–4 of the transients are indicated by numbers on the lowest record in *B*. Same muscle in Fig. 2.

slowing at higher velocities; there is considerably more curvature in the lowest trace than the two upper traces.

Force-Velocity Relations. The changes of velocity with time are better quantified by force-velocity plots using velocities measured at different times following the force step. Fig. 5 shows these velocities averaged for seven muscles and plotted as a function of normalized isotonic force. The times at which the velocities were measured are 1 ms after completion of the step, and 7, 20, and 40 ms after the onset of the step. The data in Fig. 5 *C* are for steps made at the peak of an isometric twitch. Those in Figs. 5 *A* and *B* are for steps made earlier in the twitch, when force had risen to approximately one-quarter and one-half of its peak value, respectively. Each individual value of velocity is an average taken over 1 ms. All steps were made at 90–92% L_{max}, to minimize the effects of rest force in the muscle, so that the peak developed force is approximately half that achieved at L_{max}. The 7 ms curves were all fitted to the Hill (1938) equations by a Newton-Raphson least-squares technique (Carnahan et al., 1969, p. 319). As can be seen, the 1 ms velocity values are 2–3 times larger than the 7 ms values, and for steps to low loads, the 20 and 40 ms values are lower than the 7 ms values by another factor of 2–3. For the steps made to heavy loads early in the contraction, the 20 and 40 ms values are the same or slightly higher than the 7 ms values.

Effect of Viscoelastic Resistance

The decline of contractile element velocity during isotonic shortening might be explained by the previously described viscoelastic elements (spring *d* and dashpot *p* in Fig. 7) in parallel with the contractile element (Chiu et al., 1982). To assess this hypothesis, the time course of measured velocity was compared with the time course of velocity calculated on the basis of a constant force-velocity relation for each length and time in the twitch, and an increasing viscoelastic load (Fig. 6). The model for the calculations is shown in Fig. 7. To make these calculations, series elastic recoil velocity was subtracted from the measured velocity to obtain contractile element velocity. This corrected velocity was then used to solve for the force in the viscoelastic element (spring *d* and dashpot *p* in series). This is done by solving the following equation, which relates the viscoelastic element velocity (equal to the contractile element velocity, V_{ce}) to its force T, by a fourth-order Runge-Kutta numerical method (Carnahan et al., 1969 p. 361)

$$dT/dt = h\,(V_{ce} - T/Q_p) \quad (1)$$

where h and Q_p are spring and damping constants respectively. The force in the parallel spring (spring *p*) is found by direct substitution of the change in length of the contractile element in the exponential spring equation:

$$T = (T_i + T_o)\,e^{\Delta L/k} - T_o \quad (2)$$

where T_i is the initial rest force at length L_i, T is the force at length $L = L_i + \Delta L$, and T_o and k are constants. The force on the contractile element is given by the measured isotonic muscle force minus the forces in parallel spring plus the force in the viscoelastic element. The total calculated force on the contractile element at 7 ms after the

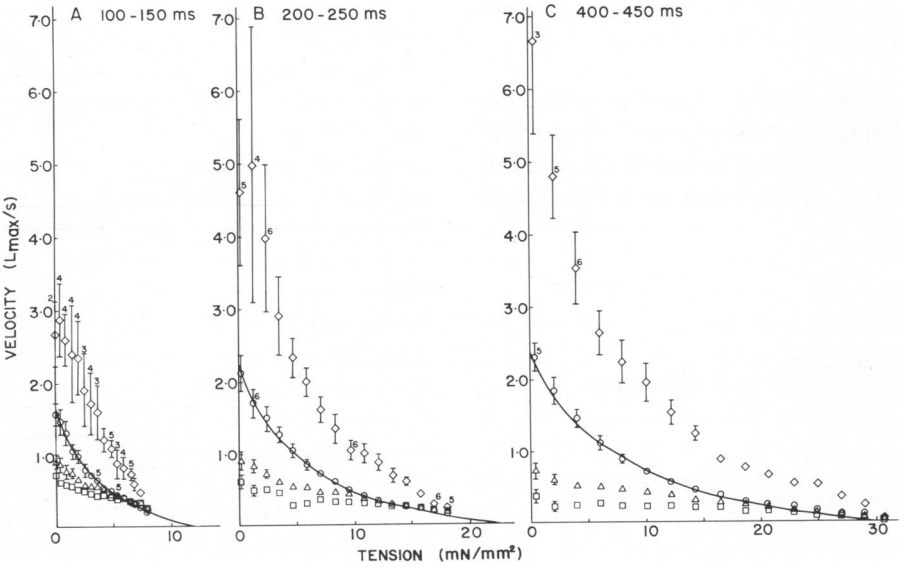

FIGURE 5 Force-velocity relations. *A*, when force had risen to about one-quarter of its peak value. *B*, at one-half of peak. *C*, at the peak of the twitch. Velocity measured at 1 ms after end of step (◇), 7 ms after onset of step (○), 20 ms after onset of step (△), and 40 ms after onset of step (□). Muscle lengths = 90–92% L_{max}. 7 ms data fitted with the Hill (1938) equation. Error bars indicate SE. Unless otherwise indicated, each point is the average of one value for each of the six muscles in *A* and seven muscles in *B* and *C*. Force steps were segregated into intervals and the standard error of the mean values of force in each interval is less than the width of the symbols. Not every muscle had a force step in every interval. The 1 ms velocities were not included unless the force step was 98% complete within 3 ms.

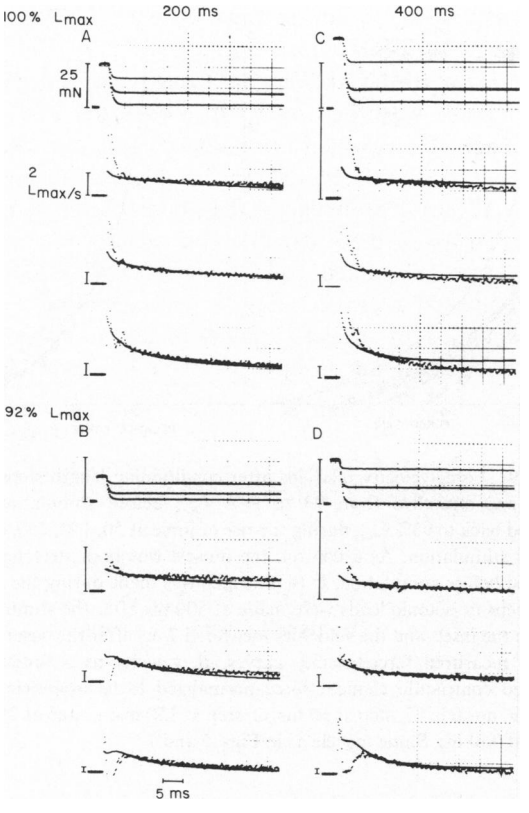

FIGURE 6 Comparisons of calculated and recorded velocity responses. Three sizes of force steps are shown at each of two lengths and two times in the twitch. The upper panel in each quadrant shows the three force records superimposed and the lower three panels show the calculated and measured velocity responses superimposed. The series elastic recoil velocity has been subtracted from the recorded velocity response, and the gain for the velocity traces is increased in steps to lower loads to give about the same degree of velocity resolution with each step. Muscle length 6.0 mm, weight 5.8 mg.

onset of the step and the velocity measured at the same time were fitted with the Hill (1938) hyperbola to describe the force-velocity characteristics of the contractile element (Fig. 8). This fitted equation was then used to calculate the time course of velocity from the time course of contractile element force. Comparisons of the calculated and measured velocity responses for 12 isotonic releases are shown in Fig. 6. The calculated and measured velocities are superimposed for three different force steps made at each of two lengths and two times in the contraction cycle. The shorter length, 92% L_{max}, was sufficiently short that the parallel elastic element bore almost no force at rest, and very little correction for this rest force was required. A correction for transfer of load from the parallel elastic was required. A correction for transfer of load from the parallel elastic element to the contractile elements was required at the longer lengths. As can be seen, the calculated velocities became progressively smaller than the measured velocities when the muscle was made to shorten against a heavy load early in the twitch, and larger when it was made to shorten against a light load at the peak of the twitch, with good

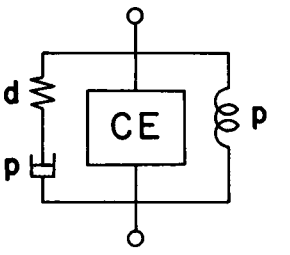

FIGURE 7 Relationship of parallel elastic and viscous elements to the contractile elements (CE). The spring in series with viscous elements is compressed during shortening and so is drawn as a folding structure. The series elastic elements are not shown because they do not change length during isotonic shortening, and are therefore not taken into account in calculating the velocity responses shown in Fig. 6. The constants used for the example here are listed in Table II of the preceding paper (Chiu et al., 1982). The time constant of relaxation for the series combination of dashpot and compressive spring, determined as the quotient Q_p/h, was 63 ms.

agreement for the other steps. This would be expected if activation were rising in spite of the slow shortening with heavy loads early in the twitch, falling because of the rapid shortening with light loads late in the twitch, and if there was very little change in activation with the other steps. This figure also shows that the initial velocities measured at the short length were substantially higher than the later velocities measured at the long length, indicating that the progressive decline in velocity was not due to the shorter length itself. In none of the contractions beginning at the longer length did the muscle shorten to the shorter initial length.

Corrected Force-Velocity Relations. The force-velocity relationship measured at 7 ms after the onset of the step is shown as Fig. 8. The measured values are shown as the solid lines and open circles. The rapid shortening associated with recoil of the lightly damped series elastic element (Chiu et al., 1982) persists at this time only in steps to the very lowest loads, as shown by the corrected curves (· · ·, ◊). Although small, the persistence of this recoil velocity at 7 ms indicates that velocity measurements could not be made much earlier without requiring more substantial corrections. Correction of the curves for the calculated increase of internal compressive force on the contractile element was of a much larger order, as shown by the dashed line in Fig. 8. The maximum velocity calculated from extrapolation of this corrected curve is four times larger than the values obtained by extrapolation of the measured values. If these corrections are appropriate, they indicate that it is not possible to measure directly the steady-state unloaded shortening velocity in this preparation. Immediately after the contractile elements are unloaded their velocity is not in a steady state because of the transients. By the time these transients are over, the compressive load on the contractile elements has increased to a substantial fraction of the isometric value.

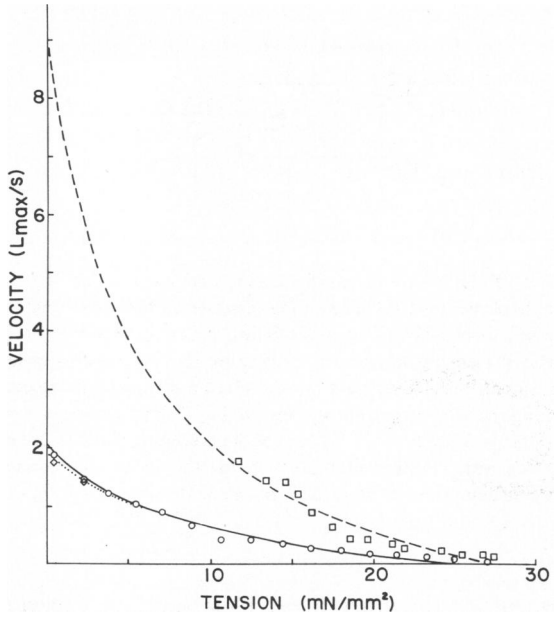

FIGURE 8 Corrected force velocity relations. Isotonic releases made from 92% L_{max}, 400 ms after stimulation. Velocity data recorded 7 ms after onset of step. Measured values (O); corrected for damped series recoil (◇); corrected for viscoelastic load (□). The curves are fitted with the Hill (1938) equation. The curves fitted to the data corrected for viscoelastic compression were used in calculating the velocity responses. The dashed curve here, for example, was used in calculating the responses shown in the lower right quadrant of Fig. 5.

Changes in Force-Velocity Capabilities during the Twitch. Fig. 5 illustrates that maximum shortening velocity rises much more rapidly than isometric force during the twitch. For example, the V_{max} measured at 7 ms after the onset of the step reached 65% of its ultimate value at a time when isometric force had reached only 25% of its full value. This observation, which will be used in interpreting double-step experiments below, reflects the fact that the contractile element V_{max} reaches an early peak and does not change much during the twitch, while isometric force rises relatively slowly. This finding suggests that changes of activation have relatively little effect on V_{max} and a large effect on isometric force.

Double Step Experiments. To further evaluate the effects of internal compression, force-velocity relations were measured at varying intervals after a shortening step had been applied to the active muscle. The muscle was stretched by 6% L_{max} before stimulation and shortened to its original length during the rise of force at 50, 150, 250, or 350 ms after stimulation. The force-velocity relations were measured 500 ms after stimulation, just after the peak of the twitch. In control experiments, both stretch and release were performed befored stimulation, so that the muscle was held isometrically at its original length during the rise of force. All the velocities were measured at 7 ms after the

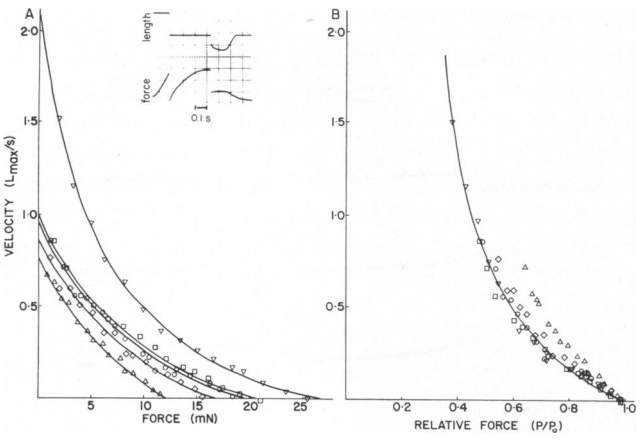

FIGURE 9 Force-velocity relations after conditioning length steps. The muscle was stretched from 93 to 99% L_{max} before stimulation and shortened back to 93% L_{max} during the rise of force at 50, 150, 250, or 350 ms after stimulation. As a control, the muscle was both stretched and shortened before stimulation, so that no step was made during the rise of force. Steps to isotonic loads were made at 500 ms after the stimulus, as shown in the inset, and the velocities measured 7 ms after the onset of the step. *A*, measured force-velocity curves. *B*, velocity as a function of calculated contractile element force normalized to its isometric value (p/p_o). ▽, no step; □, step at 50 ms; O, step at 150 ms; ◇, step at 250 ms; △, step at 350 ms. Same muscle as in Figs. 5 and 7.

onset of the step. The five force-velocity curves are plotted in Fig. 9 *A*, and the protocol is illustrated in the *inset* of that figure.

The force-velocity curves were depressed by the conditioning steps, and the depression was greater with steps made later in the twitch. The variation of isometric force with timing of the step was about twice as great as the variation in V_{max}. This finding argues fairly strongly against the depression in the force-velocity curves being due to a single mechanism, such as inactivation due to shortening. If V_{max} was influenced by changes in activation, changes in V_{max} should be proportional to the changes in isometric force. The observation, described above, that V_{max} is not much affected by changes in activation further suggests that the changes in the force-velocity curves cannot be explained entirely by changes in activation. The conditioning step depressed all values of V_{max} to less than half the control value, which is substantially less than the values measured early in the twitch (Fig. 5 *A*). A possible explanation for the observed curves is that the conditioning steps impose an internal viscoelastic load on the contractile elements as well as causing deactivation. This possibility was tested by calculating the total load on the contractile elements using the model shown on Fig. 7, and comparing the corrected force-velocity curves with each other. If V_{max} is not affected by changes in activation, plots of velocity vs. the relative force (P/P_o) should overlie each other (Thames et al., 1974). Although there is some scatter in the data, the data points in these plots superimpose fairly well (Fig. 9 *B*).

DISCUSSION

The main finding in these experiments is that isotonic shortening velocity in papillary muscle is not constant, and that the velocity changes are due largely to passive structures. Assessment of the contractile element behavior, therefore, depends heavily on the modeling of the passive structures. Because of this dependence on the modeling it is important to show that evidence for the different mechanisms exist independent of the models. Evidence for the velocity transients can be seen both in the uncorrected records from afterloaded contractions and in the small plateaus in the uncorrected velocity traces from the usual isotonic releases. Evidence that the later changes in isotonic velocity are not due to inactivation is provided by the double step experiments and by the observation that V_{max} is not much affected by changes of activation.

Velocity Transients

The velocity transients associated with a step have a similar, but 3–5 times faster time course than those described for frog skeletal muscle near 0°C (Podolsky, 1960; Civan and Podolsky, 1966; Huxley and Simmons, 1972). Their principal significance in these experiments is that they provide additional evidence that the contractile mechanisms of cardiac and skeletal muscle are similar.

The maximum velocity measured 7 ms after the onset of the force step was approximately the same as that of frog skeletal muscle near 0°C, while the transients were several times faster. This difference in the relative speeds of the two processes suggests that the true maximum velocity of the contractile elements is greater than the measured value, as implied by the corrected curve in Fig. 8.

Changes in Isotonic Velocity

As shown in Fig. 6, the decline in isotonic velocity after the initial transients can be accounted for largely by an increasing viscoelastic load. Neither viscosity nor elastic compression alone will account for the change in velocity. A simple viscosity would impose a constant load at a given speed, rather than a load that increased with shortening. A simple compressive spring, without a series viscosity would impose a load that would be strongly dependent on the initial length (Ford and Forman, 1974).

In addition to the viscoelastic resistance, there are at least three factors that might contribute to decreased velocities with rapid shortening: (*a*) decrease in activation due to the process of shortening (Brady, 1966; Briden and Alpert, 1972): (*b*) inactivation due to the shorter length (Taylor and Rudel 1970; Schoenberg and Podolsky, 1971); and (*c*) exhaustion of a substrate required for rapid shortening. The present evidence excludes the first two of these factors as the sole cause of the velocity changes. The observation that V_{max} is not much affected by changes in activation and the double step experiments in which V_{max} is depressed following a conditioning step indicate that inactivation due to shortening is not the only factor causing a progressive decrease in shortening, as discussed above. Experiements done at different lengths indicate that inactivation due to a shorter length is also not the major cause of the decreasing isotonic shortening; velocities measured immediately after force steps at shorter lengths are much higher than those seen after 50 ms of isotonic shortening at a longer length. Although the third possibility, rapid substrate exhaustion, cannot be excluded definitely by these experiments, the double-step experiments make this possibility seen unlikely. The muscle liberates much less mechanical energy, and presumably consumes less substrate, with the conditioning step placed early in the twitch, and yet V_{max} is depressed by about the same amount with all conditioning steps.

It should be emphasized that changes in activation also affect isotonic shortening velocity. This is most obvious in the afterloaded contraction made early in the twitch, where velocity actually increased during shortening, undoubtedly because of rising activation. Similarly, with large releases made after the time that 90% of peak twitch force had developed, isotonic velocity declined more rapidly than would be expected from internal load alone, and this is probably caused by deactivation secondary to shortening.

Relation to Earlier Experiments

The observation that isotonic velocity progressively following a quick release to a relatively low load is not a new finding. It can be seen in virtually all of the published records of such experiments in cardiac muscle (e.g., Sonnenblick, 1965, 1967; Brady 1965, 1966; Edman and Nilsson 1969; Noble et al., 1967). The present interpretation of this phenomenon is new, however, and can possibly help to resolve some of the questions in the literature. Experiments using afterloaded isotonic contractions produce very different conclusions than experiments using quick releases to isotonic loads (cf. Brady, 1965, and Sonnenblick, 1965 vs. Brutsaert et al., 1971). The present experiments indicate strongly that the interpretation of the two types of experiments will be different when the data are not corrected for the effects of progressive shortening.

Two studies have suggested that the length oscillations produced by undamped quick releases partially inactivate the muscle (Edman and Nilsson, 1972; Bodem and Sonnenblick, 1974). In both studies, velocities at specified times following a quick release were higher when the force step was overdamped than when the step was undamped and there was substantial lever oscillation. Effects of lever oscillations seem likely to be small because the oscillations seen in the length records are the result of the lever oscillating on the compliant muscle attachments, and probably do not reflect large oscillations in sarcomere length. The extent of inactivation following a release can be estimated from the decrease in the extent of shortening

during the remainder of the twitch. In both studies, the extent of shortening was the same with either type of release, provided that the release was made before the peak of the twitch. This observation suggests rather strongly that the oscillations associated with the undamped release did not cause much inactivation. The depressed velocity associated with the more rapid release could be explained by the model described here. The more rapid early shortening immediately following an undamped release would cause a more rapid accumulation of viscoelastic resistance. If this interpretation is correct, velocity at any load should depend almost entirely on the distance shortened, and be independent of the time after the release, since the internal load depends almost entirely on the extent of shortening. This dependence is clearly seen in the records of Bodem and Sonnenblick (1974), who plotted velocity as a function of muscle length. The velocity-length plots made with the two types of releases overlie each other exactly when the release is made before the peak of the twitch. In both studies, the extent of shortening and the velocity at each length is reduced following the undamped releases made after the peak of the twitch. While these reductions are undoubtedly due to inactivation, it is unclear how much of the inactivation is due to the oscillations, and how much to the higher initial velocities associated with the undamped releases.

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