Series Elasticity in Heart Muscle
ITS RELATION TO CONTRACTILE ELEMENT VELOCITY AND PROPOSED MUSCLE MODELS

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ABSTRACT
The series elasticity (SE) of cat papillary muscle was measured in 6 muscles by the isotonic quick-release method, with careful correction for the compliance of the equipment. The resultant SE curve was exponential in form with an extension of 4% to 5% of initial muscle length for a preload of 0.5 g and a developed tension of 10 g; this demonstrates a stiffer SE for cardiac muscle than has previously been reported. The quick-release SE extension curve was unaffected by inotropic interventions or changing the time of quick release, and was less compliant than the SE curves calculated by two different isotonic methods. These differences arise from the fact that contractile element velocity has a secondary rise during the transition from the isometric to isotonic phase of any afterloaded contraction. With increasing preload (and initial muscle length) the SE became stiffer, suggesting that a substantial part of it is in series with both the contractile and parallel elastic elements.

ADDITIONAL KEY WORDS isotonic quick release papillary muscle parallel elastic component series elastic modulus paired electrical stimulation Frank-Starling mechanism equipment compliance force-velocity relation cat

A. V. Hill has described active muscle in terms of a mechanical model with three functionally separate components: (1) an active contractile element (CE) which is assumed to be freely extensible at rest, but which can shorten and develop force with activation, (2) a passive series elastic element (SE) arranged in series with the CE, and (3) a passive parallel elastic element (PE) which serves to sustain resting tension. Both the SE and PE are characterized by their length-tension curves, while the shortening of the CE is described by a hyperbolic relation between force and velocity (1–6). During isometric contraction, the activated CE shortens and stretches the SE with the development of force in the SE in accordance with its stress-strain relations. The rate of force development is thus determined by the contractile element velocity and the stiffness of the SE.

Initial measurements of the SE in skeletal muscle by isotonic quick-release methods demonstrated a SE extension of 10% to 15% of initial muscle length during the development of tetanic tension (1, 2). Subsequent studies with more complete corrections for stray compliances of the apparatus have reduced this figure to 2% to 3% (6–10).

In cardiac muscle the SE would appear to be more compliant than in skeletal muscle. Abbott and Mommaerts (11) found a SE extension of 6% of muscle length at loads of 2 g/mm², using an isotonic quick-release method. Sonnenblick (12, 13) measured SE by two different isotonic methods and found a SE extension of 10% of initial muscle length with a developed tension of 10 g. At a developed tension of 2.2 g the SE extension was 6%, a finding similar to that of Abbott and Mommaerts.

Because of the reported differences between the SE compliance in skeletal and cardiac muscle and the different methods used to measure SE, the present study was undertaken to simultaneously measure the SE extension curve in heart muscle by the three
previously reported methods, with very careful attention to measurement of the compliance of the equipment and correction for it. Inotropic interventions, changing the time of quick release, and different preloads were also studied relative to their effect on series elasticity.

Results indicate that cardiac muscle SE is less compliant than previously reported, although not as stiff as in skeletal muscle. Some reasons for the overly compliant values previously reported (11-13) are demonstrated, and these differences give insight into the magnitude of the CE velocity during isometric contraction as compared to isotonic shortening. The results also permit some speculation about muscle models and the anatomical location of the SE.

**Methods**

Six single papillary muscles were obtained from the right ventricles of cats (1.5 to 2.0 kg) anesthetized with intraperitoneal sodium pentobarbital (25 mg/kg). Muscle lengths varied from 5 to 8 mm (excluding the tendon) with an average calculated cross-sectional area of 0.98 mm². Each muscle was rapidly removed from the right ventricle and quickly suspended in a 20-ml bath containing Krebs bicarbonate solution bubbled with 95% O₂ and 5% CO₂ as shown schematically in Figure 1. The lower nontendinous end of the muscle was held firmly by a spring-loaded lucite clip fixed to a rigid stainless steel rod extension of a force transducer (Statham model G1-4-250). The upper tendinous end of the muscle was tied with a short length (3 to 4 cm) of Ethicon 4-0 braided surgical silk to a straight wire connection from the tip of the isotonic lever system. The muscle bath was kept at a temperature of 30°C by an outer water jacket connected to a circulating constant-temperature bath.

The isotonic lever (14) was fashioned from magnesium with a lever ratio of 20:1, and an equivalent mass of 40 mg. Lever movement was detected by a photoelectric transducing system with a linearity less than 1% over a 2-mm range of movement. The output signal of the length transducer was electronically differentiated to measure instantaneous velocity with a time constant of 0.6 msec. The preload and afterload were added to a weight holder suspended from the lever by a rubber band to produce damping.

The muscle was stimulated (Laboratory Stimulator AEL 104A) with platinum mass electrodes (2.0 x 0.5 cm) placed parallel to the muscle using pulses of 7 msec duration, and voltages not more than 20% above threshold (15). The rate of stimulation was 12/min for all experiments except when the effects of changing frequency were analyzed. Muscle shortening, velocity of shortening, force, and a stimulation artifact were recorded on an Electronics for Medicine Recorder.

Quick-release experiments were performed with an air jet apparatus (16). With a preset afterload in place, the muscle was stimulated but initially prevented from shortening by an air jet which held the lever against the micrometer stop. After a preselected delay, the air jet was diverted from the lever by a solenoid activated by a second stimulator, allowing for both rapid lever shortening and an instantaneous fall in tension.

To determine the compliance of the equipment (tendon, knots, thread, and lever system), the spring clip was moved to the upper edge of the muscle at the end of each experiment as illustrated in Figure 1. A series of increasing afterloads were suddenly added without the lever stop in place and the initial rapid displacement was used as a measure of the equipment distensibility for each specific muscle. With time, there...
was a very small additional displacement of the lever, secondary to stress relaxation of the tendon, thread, and knots. This was ignored as a correction factor since the series elastic changes with quick release were virtually instantaneous.

After 1 hour of isometric stabilization the following experiments were performed. With a 0.5-g preload, the muscle contracted isotonically with afterload progressively increased from zero to isometric force. Quick-release experiments were next performed with the time of release occurring at successive 100-msec intervals after the stimulus throughout the duration of an isometric contraction (up to 900 msec). At each 100-msec interval, afterload was increased from zero to isometric force. The above sequence was repeated 3 or 4 times in alternate fashion, after which passive and active length-tension curves were determined. With a fixed time of quick release the stimulation rate was changed from 6/min to 36/min, after which paired stimuli were used as another inotropic intervention. The effect of changing initial muscle length was examined by keeping the time of quick release constant while varying the preload from 0.1 to 3.5 g. At each preload the afterload was varied from zero to isometric force.

**Results**

Series elastic extension curves were calculated by the following methods for each muscle: (1) quick release, (2) isotonic series elastic modulus, and (3) CE velocity integrated as a function of time.

**Quick Release.** After a predetermined period of isometric force development, the muscle was released abruptly and allowed to shorten to a given afterload, as illustrated in Figure 2. The abrupt fall in tension (ΔP) and rapid muscle shortening (ΔL) reflect the sudden shortening of the SE, as it adjusts to the lower tension of the afterload according to its characteristic elastic properties. All measurements were made with a small preload (0.5 g) so that the contribution of the PE could be neglected. Thus, ΔP and ΔL are a direct measure of the stress-strain relations of the SE.

This analysis presupposed (1) that the time interval of rapid shortening was too brief to allow for any significant CE shortening, and (2) that the basic contractile mechanism was essentially unaffected by the quick-release intervention. Firstly, the time required for the quick changes in length and tension was generally less than 10 msec, during which only negligible CE shortening could occur. Second, oscillographic phase plane analysis (velocity of shortening relative to length) of individual contractions after either quick release or quick stretch revealed only minor differences when compared to control records, indicating that no basic change in the contractile mechanism had occurred (unpublished observations). The SE load-extension curve was determined four to eight times for each muscle and then averaged and corrected for equipment compliance. The equipment distensibility curve had the general char-
acteristics of an exponential spring with a maximum extension of 0.2 mm for a 10-g load. Quick-release experiments without a muscle in the system revealed that the equipment (E) responded as rapidly as the tension and length changes of the muscle following quick release. Therefore, E was treated as a small additional series elastic element and was subtracted from the experimental SE curve to obtain the corrected SE curve.

The corrected average SE extension curve for all 6 muscles obtained by the quick-release method is shown by the open circles in Figure 3 together with the standard error of the mean. This curve is exponential in shape, and may be represented by the equation $L = \frac{1}{k} \log_e (kP + C) + C$. Thus, the SE modulus of elasticity is defined as $\frac{dP}{dl} = kP + C$. The constants of this equation which best fit the quick-release data in Figure 3 are: $k = 4/\%$ muscle length and $C = 0.8 \text{ g/\% muscle length}$ for a 0.5-g preload. The units of C are g/\% muscle length since 1/C is the initial slope of the SE extension curve. The closed circles and dashed line in Figure 3 represent the theoretical SE curve, using the above constants. For a muscle normalized to a length of 1 cm, $k = 4/\text{mm}$ and $C = 8 \text{ g/mm}$. These data indicate a SE extension of 4\% to 5\% of initial muscle length with a preload of 0.5 g and a developed tension of 10 g.

Isotonic Series Elastic Modulus. Theoretically, the series elastic modulus can also be calculated from a series of afterloaded isotonic contractions (3). During any one contraction, isometric tension rises until it matches the afterload, at which point the muscle begins to shorten with tension remaining constant (Fig. 4). At this transition point, the rate of rise of the tension, $\frac{dP}{dt}$,
and the maximum shortening velocity, \( \frac{dl}{dt} \), are measured respectively as the slopes of the tension and shortening traces. Assuming that this peak velocity equals CE velocity just prior to shortening, then the isotonic SE modulus, \( \frac{dP}{dl} \), is defined by the formula:

\[
\frac{dP}{dl}(\text{iso}) = \frac{dP}{dt} / (\frac{dl}{dt})
\]

for each afterload \( P \). In every series of afterloaded contractions (Fig. 4) the isotonic SE modulus calculated in this way was found to be approximately linearly related to the afterload:

\[
\frac{dP}{dl}(\text{iso}) = kP + C.
\]

Integration of this equation gives the exponential SE extension curve. Part of this calculated curve, however, represents equipment distensibility which was subtracted to give the corrected SE curve for each muscle. The corrected average SE extension curve for all six muscles, as determined by the isotonic modulus method, is shown as the closed triangles in Figure 3, together with the standard error of the mean. This method yielded an extension of 8% to 9% of muscle length with a 0.5-g preload and a developed tension of 10 g.

**CE Velocity Relative to Time.** The maximum velocity of shortening during afterloaded isotonic contractions (Fig. 4) was used to calculate the SE extension curve, again based on the assumption that this velocity is identical with CE velocity at the point where rising isometric tension matches the afterload. Using these data the peak velocities of shortening for different afterloads were plotted as a function of time to represent the course of CE velocity during isometric contraction. Integration under this curve, \( L = \int dl/dt \cdot dt \), should give the series elastic extension curve if the initial assumption concerning velocity is correct. Again, multiple determinations in each muscle were averaged and corrected for equipment distensibility. The resultant SE extension curve is plotted in Figure 3 as the closed squares. It is similar to the curve calculated by the isotonic modulus method and reveals a SE extension of 9% to 10% with a 0.5-g preload and a developed tension of 10 g. It is apparent that the SE curve obtained by the quick-release method is significantly different from the curves obtained by the other two methods (see Discussion).

Each of the above methods of calculation assumes that the SE behaves like an inert spring whose length is dependent only on the developed force, and that its properties do not change with time during the course of a contraction. This assumption was verified by performing quick-release experiments at 100-msec intervals throughout the course of contraction for each of the six muscles. Representative curves from one muscle are identical (Fig. 5), independent of the time of quick release. Multiple determinations in all six muscles verified the fact that the stress-strain relations of the SE are constant throughout the time course of contraction. Sustained postextrasystolic potentiation (paired electrical stimulation) or changing the rate of stimulation between 6/min and 36/min likewise did not alter the SE extension curve, as confirmed by multiple experiments in all six muscles.

**Discussion**

A. V. Hill's classic model for muscle is illustrated as Model I in Figure 6, together
Three proposed muscle models, with I representing Hill's classic model. The CE is freely extensible at rest, but with activation, shortens and develops force. The elastic elements PE, SE, and the equipment distensibility, E, have the general properties of exponential springs.

with two other possible model arrangements. With small preloads (0.5 g or less) the three models shown become equivalent, since the contribution of the PE can be neglected. At higher preloads, however, the contribution of the PE becomes important. For example, in Model I a large initial resting tension (large preload) is carried entirely by the PE, while total tension is the sum of the contributions of the PE plus the SE. Therefore, as the muscle shortens isotonically, the PE contributes progressively less to the total tension as it also shortens. Since total tension remains constant (isotonic contraction), the tension across the PE decreases and the tension across the SE increases. Therefore, the SE is being stretched during isotonic shortening although total tension remains constant. Thus the CE shortens relatively more than the muscle does, and CE velocity is greater than the velocity of shortening of the entire muscle. By using only very small preloads (0.5 g or less), however, this correction factor is negligible, and the velocity of the CE can be assumed to equal the isotonic shortening velocity of the whole muscle.

The compliance of the equipment (E) requires careful consideration since it is present when any experimental measurements are made. In the present study, E had the general characteristics of an exponential spring, with a maximum extension of 2 mm with a 10-g load (e.g. Fig. 5). In effect, E provides an additional series elasticity which allows the muscle to shorten slightly during "isometric" contraction. This reduces the rate of force development (dP/dt) and peak isometric force, which are dependent on CE velocity (dI/dt) and SE stiffness (dP/dl): dP/dt = (dP/dl) • (dl/dt). With the addition of equipment compliance to the muscle, the effective dP/dl (SE + E) is more compliant, thus reducing the rate of force development (dP/dt) if CE velocity (dl/dt) is essentially unchanged. Indeed, with the addition of springs of known compliance in series with a papillary muscle, we have observed the expected reduction of dP/dt and developed isometric force, in proportion to the increasing compliance of the added springs (unpublished observations).

After the appropriate corrections for equipment compliance were made, the resultant SE curves in Figure 3 illustrate a significant difference between the quick-release method and the other two methods of calculation. A reason for this difference is found in the assumption upon which the latter two methods are based. These methods assume that at the point during contraction where rising tension matches the afterload, CE velocity is identical with the maximal velocity attained when the muscle begins to shorten. The validity of this assumption is doubtful when one examines instantaneous muscle velocity, as illustrated in Figure 7. In the first few milliseconds after shortening there is a rapid rise component of the velocity trace (point A), and then a slower rise to a rounded peak (point B). Thus peak velocity (dl/dt), as measured by the slope of the shortening trace, occurs some 80 msec after shortening begins, and most likely is not identical with the CE velocity 80 msec earlier. Possible reasons for this delay in reaching peak velocity are the continued rise in active state, inertial changes of acceleration, and/or internal viscous limitations in the muscle. The
Representative afterloaded isotonic contraction. Record reads from right to left. Point A indicates the quick-rise component of the velocity trace, while peak velocity occurs at point B and is not reached until 80 msec after shortening begins. Note that the visual slope of the length trace, $dl/dt$, measures only peak velocity. The rate of force development, $dP/dt$, is shown at the point just prior to the onset of shortening.

quick-release method, however, does not depend on any such assumptions about velocity and appears to be the most direct and accurate method of measuring SE.

Accepting the quick-release SE extension curve as correct, CE velocity during isometric contraction can then be calculated from the relationship:

$$(dl/dt)_{CE} = (dl/dP)_{BE} \cdot (dP/dt)_{isom}$$

$(dl/dP)$ is the slope of the quick-release SE extension curve, and $(dP/dt)_{isom}$ is the slope of the force curve during isometric contraction. Since $(dP/dt)_{isom}$ can only be obtained with equipment distensibility as part of the system, the above equation is valid only for an effective series elastic of SE + E. Therefore $(dl/dP)_{BE}$ refers to the uncorrected SE quick-release data, and the resultant $(dl/dt)_{CE}$ will also be uncorrected for equipment distensibility. Figure 8 illustrates the results of such a calculation of CE velocity in one muscle, using the slope $(dP/dt)$ of the actual force trace and the slope of the uncorrected quick-release SE curve from that muscle. Similar calculations for all six muscles were identical. The closed circles and dashed line show the calculated CE velocity during isometric contraction as a function of time. An actual velocity trace for a 2-g afterloaded contraction is also shown, with the arrow indicating the quick-rise component of velocity. The open circles indicate the quick-rise component of velocity for various afterloads and the open triangles indicate the peak velocities of these same contractions at the time they are reached. The appropriate afterload is indicated for each pair of quick rise and peak velocity points. Two conclusions are apparent in
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this illustration. (1) The quick-rise component of isotonic shortening velocity is identical with the calculated CE velocity during isometric contraction. Thus, the quick-rise component and not peak velocity best represents CE velocity just prior to the onset of shortening. (2) CE velocity has a secondary rise at the transition point between the isometric and isotonic phases of any afterloaded contraction. It should again be emphasized that the above analysis of CE velocity is valid only at low preloads when the contribution of the PE can be neglected and muscle shortening velocity is identical with CE velocity. The discrepancy noted between the curves in Figure 3 is thus explained. The quick-release data represent the actual SE extension curve, while the other two curves are overestimates because of their false assumption regarding CE velocity.

The fact that CE velocity has a secondary rise during any afterloaded isotonic contraction has already been alluded to in both the papillary muscle and the intact dog (17). The reason for this secondary rise is not immediately apparent. One explanation could be that the active state is still rising, since active state in cardiac muscle reaches a broad peak only just before the peak of isometric tension (16). This is in contrast to skeletal muscle, where peak active state is reached very early in the course of a contraction (3). This may not be the sole explanation, however, since this secondary rise in velocity occurs virtually up to the point where the afterload equals peak isometric tension (Fig. 8).

A simpler explanation for this secondary rise in CE velocity may be that the stiffness of the SE limits CE shortening to only 4% to 5% of initial muscle length during isometric contraction, and thus limits CE velocity. During an isotonic contraction (preload only), however, the CE shortens 10% to 15% of initial muscle length in only a slightly longer time interval, thus achieving a higher velocity. The major factor, therefore, may be the stiffness of the SE, which limits the distance the CE can shorten isometrically and thus limits isometric CE velocity as compared to isotonic velocity. That this same phenomenon may be operative in skeletal muscle is suggested by the work of Jewell and Wilkie (8). They measured the SE extension curve by the quick-release method, and CE velocity during afterloaded isotonic contractions. Assuming that their measured CE velocity during shortening was equal to CE velocity just prior to shortening, they then calculated the isometric force curve. Their calculated curve rose more steeply than the experimental isometric force curve; a finding they explained by assuming that the velocity depended not only on the force, but on some aspect of the history of the muscle. The discrepancy noted can be resolved if there is a secondary rise in CE velocity during the transition from the isometric to isotonic phase of an afterloaded contraction.

Previous studies of SE in heart muscle have demonstrated that it is unaltered by changing the stimulation rate (11), by adding norepinephrine (12), or by post-extrasystolic potentiation (11). Data presented here confirm the fact that the SE is unchanging throughout the time course of a contraction (Fig. 5), and unaltered by inotropic interventions. The SE thus behaves as an inert spring, whose length is uniquely determined by the force across it.

All discussion to this point has concerned data obtained at very low preloads only. Under these conditions, the contribution of the PE can be neglected and all three models in Figure 6 are equivalent. As resting tension rises, however, the relative arrangement of the PE and SE assumes some significance. In model I, for example, the passive length-tension curve defines the properties of the PE, while in models II and III both the SE and PE contribute to resting tension. Quick-release SE measurements with increasing preload (and longer initial muscle length) were used to decide which model arrangement is most appropriate.

In model I, if one corrects for the contribution of the PE, the SE extension curve should be independent of preload (initial muscle length), since the nonactivated CE is freely extensible. This hypothesis was tested by measuring quick-release SE extension curves.
Quick-release SE extension curves in one muscle, with variable preload and corrected for equipment distensibility. The superimposed passive length-tension curve is shown for comparison. If model I is correct, these curves should become identical after correction for the PE. The appropriate model I PE corrections (Fig. 10) are shown for the end point of each curve; note that the corrected curves are still widely divergent.

In model I the PE contribution is 2.5 g of the total force. The muscle shortened .16 mm ($\Delta L$) with quick release, thus reducing the contribution of the PE to only .25 g of the total force. The fall in tension measured at the time of quick release (7 g in Figure 9 for a 2.5-g preload) must be reduced by 2.2 g ($\Delta P = 2.5 - .3$) in order to correct for the PE, and measure only the force affecting the SE. The dashed line corrections in Figure 9 are shown only for the end points of each extension curve. It is apparent that the curves do not become identical after correction for the PE, indicating that the SE does indeed become stiffer with increasing preload. Accordingly, model I is invalid since part or all of the SE must be in series with both the CE and the PE, as shown in either models II or III.

In the muscle shown in Figures 9 and 10, model III is the only applicable one for the following reasons. In model II the passive length-tension curve is a summation of the SE and PE. In five of the six muscles studied, the passive length-tension curve was more compliant than the quick-release SE curves with small preloads. In the muscle shown in Figure 9, however, the superimposed passive length-tension curve is less compliant than the quick-release SE curve for a 0.1-g preload. Thus, in order for the relationship PE...
+ SE = passive length-tension curve, to hold as required by model II, the PE must be represented by a negative curve, which is an impossibility. Therefore, model III is the only valid model of the three, applicable to this particular muscle.

Deciding between models II and III in the other five muscles is more difficult. Figure 11 plots the series elastic modulus as a function of afterload. The solid line is the line best fitting the quick-release data for a 0.5-g preload. If model I were correct, this line would remain unchanged with changing preload. The dashed lines, however, indicate the general shifts noted with changes in preload. If model II were correct, these shifts would be precise parallel shifts (with K remaining constant), and the vertical axis intercept (C) varying linearly with the preload. Within the limits of measurement, however, the intercept C did not vary as a linear function of the preload, which would tend to favor model III.

In the present experiments it is reasonable to assume that part of the SE is located just above the point where the spring clip firmly grips the muscle. This segment may not function entirely normally, and thus may provide some SE at the end of the muscle. Similarly the tendinous attachment at the top of the muscle may also provide additional SE since it is virtually impossible to define an exact demarcation between muscle and tendon, when moving the spring clip to this point to measure equipment distensibility. It is thus tempting to apply model III to these experimental results by assuming that SE₂ represents the attachments of the upper and lower ends of the muscle as noted above. SE₁ would then represent that portion of the SE in intimate association with the CE itself. The relative magnitude of SE₁ and SE₂ would appear to be individually determined for each muscle. Recent experiments have suggested that there is a small series viscous element which should be included as part of any representative muscle model (22). The experiments reported here were not designed to measure such, and thus no viscous element appears in the muscle models.

Huxley (18) and Civan and Podolsky (19, 20) have suggested that SE₁ may be found in the force-generating cross-links between the two types of myofilaments. This could account for a SE extension of up to 2%. It is conceivable that the stretching of the myofilaments themselves may also be part of this SE which is in such intimate association with the CE. CE velocity during isometric contraction may thus represent slight relative movement of the actin and myosin cross-links and/or filaments themselves, although overall sarcomere length is virtually unchanged.

The fact that the SE becomes stiffer with increasing preload, directly relates to the Frank-Starling mechanism (21). The rate of isometric tension development (dP/dt) varies directly with CE velocity (dl/dt) and the series elastic modulus (dP/dl). Since CE velocity as a function of time is little changed with changing preload (13), then increasing preload (initial muscle length) results in a stiffer SE and a greater dP/dt. Peak isometric force is thus increased, since the time to peak tension is unchanged (13).

Compared to previous work, these data
indicate a substantially stiffer SE for heart muscle than has been reported. Using quick-release methods, Abbott and Mommaerts found a SE extension of 6% of initial muscle length for a developed tension of 2 g/mm² with a preload of 0.25 g (11). Data presented here show a SE extension of 2% for a load of 2 g/mm² with a 0.5-g preload. Corrections for preload do not wholly resolve this discrepancy, and the difference may be due to unrecognized stray compliances in this previous study. Measurements of SE compliance by isotonic methods (13) have also been shown to be excessive because of an overestimate of CE velocity during isometric contraction. It would appear, therefore, that the 4% to 5% compliance of the SE of cardiac muscle is close to that of skeletal muscle, which is 2% to 3% (6-10).

The application of these studies of the papillary muscle to the intact heart is complicated by the irregular shape of the heart chambers, variable wall tension due to the Laplace relation, nonuniformity of fiber direction and activation, and the difficulty of defining preload and “isotonic” contractions. Further, the possible presence of strong muscle in series with weak muscle may exist in various types of heart disease, and may even play a role in the normal heart. In this regard, Forwand et al. have recently measured the active stiffness of the intact canine left ventricle before and after experimental myocardial infarction (23). Their results indicate a SE extension of 5.7% to 7.6% of initial muscle length during isometric contraction, a value comparable to the results reported here. Thus, there appears to be good correlation between in vitro and in vivo studies of cardiac muscle series elasticity.

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