Dynamic Stiffness of Barium-Contractured Cardiac Muscles With Different Speeds of Contraction

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The dynamic stiffness of excised cardiac muscles that would be likely to have different intrinsic speeds of contraction, as judged by previous biochemical reports of their myosin ATPase rates, was compared. This study included muscles from thyrotoxic rabbits and newborn rabbits, rabbit atria, and normal papillary muscles at different temperatures. The usual excitation-contraction coupling process was bypassed by replacing bathing Ca²⁺ with Ba²⁺. The ensuing actively maintained contracture allowed us to focus more specifically on the contractile properties of the myofilaments. Dynamic stiffness was determined by sinusoidally oscillating muscle length at many different frequencies over the range 0.05–50 Hz while holding average muscle length at 95% of the systolic length, thus giving maximal developed force. The form of the stiffness modulus spectrum was similar for all muscles studied: stiffness was fairly constant at low frequencies, decreased to a minimum at an intermediate frequency, and then increased steeply, followed by a milder rate of increase over high frequencies. Differences in contraction speed were evident by shifts in the frequencies at which corresponding portions of the stiffness spectrum appeared. The clearest landmark was the frequency where stiffness became minimum (fₘᵢₙ). This varied strongly with temperature (Q₁₀ = 2.9). Compared to normal adult papillary muscles (fₘᵢₙ = 1.2 Hz), fₘᵢₙ was 2.2 times faster in thyrotoxic myocardium, 1.9 times faster in 1-week-old rabbits, and 3.7 times faster in atrial trabeculae. These ratios of functional speed are similar to the corresponding ratios of myosin Ca²⁺-ATPase activities reported in the literature. (Circulation Research 1987;60:770-779)

The intrinsic speed of cardiac muscle contraction is a biological variable. It differs between species¹ and between atrial and ventricular muscle in the same species² and seems to be chronically adapted to aging³ and other long-term variations in demand on the heart.⁴⁻⁵ Much of the chronic variation in the speed of contraction appears to be accomplished by changes in the intrinsic composition of the contractile proteins, and 3 isoforms of cardiac myosin have been identified biochemically.⁶⁻⁹ The factors that lead to chronic adaptions in speed also shift the relative amounts of the myosin isoforms; faster speeds of contraction are correlated with a greater proportion of the myosin isozymes that has the higher biochemical rate for hydrolyzing ATP.¹⁰¹¹

The speed of contraction is usually measured during twitches of cardiac muscle. However, the results from isotonic shortening experiments, the most common method of measuring the speed of twitch contraction, depend not only on the contractile proteins but also on the process of excitation-contraction coupling. For example, acute changes in heart rate or external calcium concentration can have a large influence on the measured speed of contraction.¹²¹³

To evaluate changes in the intrinsic speed of cardiac myofilaments, a method was used that would be specific for changes in the contractile proteins. Therefore, the excitation-contraction coupling process was bypassed in excised papillary muscles by replacing the bathing Ca²⁺ with Ba²⁺. Barium produces a stable state of contracture with a steady force level (approximately two thirds of peak twitch force), which is believed to be maintained by the same active process of cross-bridge cycling as during twitches.¹⁴¹⁵

To examine the rate of the contractile process in such tonically activated muscles, the muscle length was oscillated over a wide range of frequencies to observe how the dynamic stiffness of these muscles varied with frequency.¹⁶ The frequency-dependent characteristics of dynamic stiffness for papillary muscles from normal adult rabbits were compared with muscles from adult rabbits pretreated with thyroxine, with muscles from newborn rabbits, and with atrial trabeculae of adult rabbits. In summary, the relation between frequency-dependent mechanical properties and the rate of cyclic movement of cross-bridges was explored.

Materials and Methods

Animal Groups

New Zealand white rabbits, adult males weighing 3–4 kg and 1-week-old newborns, were used in this
study. Adult rabbits were divided into 3 groups: 1) normal control group without treatment, 2) control group, sham-treated with daily intramuscular injection of 0.2 ml saline for 2 weeks, and 3) thyroxine-treated group, intramuscularly injected with 1-thyroxine (0.15–0.20 mg/kg/day) for 2 weeks. If the body weight of the rabbit decreased below 80% of its initial weight, the treatment was skipped for 3 days.8

The heart rate of awake rabbits was recorded by surface electrocardiography in a dark room after the heart rate had become stable. Serum concentrations of triiodothyronine (T3) and thyroxine (T4) were measured by radioimmunoassay.

**Muscle Preparation**

Adult and newborn rabbits were anesthetized with an i.v. injection of 40–50 mg/kg sodium pentobarbital in the adult and 50 mg/kg i.p. in the newborn. The heart was excised rapidly, the aorta cannulated above the aortic valve, and the heart perfused with Tyrode’s solution (37° C) equilibrated with a 98% O2–2% CO2 gas mixture. The perfusate contained (in mM) NaCl 130, KCl 4, NaHCO3 10, MgCl2 1, CaCl2 2.5, NaH2PO4 0.43, glucose 5.56, pH 7.2–7.4. Papillary muscles were dissected from the right ventricle and mounted in a tissue bath through which the same oxygenated solution as above flowed. The tendinous end of the muscle was tied by 6-0 black silk to a force transducer (Kulite Corp., Ridgefield, N.J.), and the nontendinous end was fixed by a clamp connected to a linear motor. Atrial trabecular muscles were obtained from the right atrium of normal adult rabbits. One side of the excised atrial trabecula was tied to the force transducer, and the other side was clamped in the same way as the papillary muscle. All muscles were stimulated 30 times/min. After mounting a muscle in the bath, the temperature of the perfusate was gradually decreased from 37° to 24° C over approximately 30 minutes. The muscle was then equilibrated at 24° C for 2 hours.

Two tungsten pins were inserted vertically into the papillary muscle by a micromanipulator. The pins were separated by 2–3 mm and were used to define the central segment between them. The length of this segment was measured by an optical method, as described previously.16 Briefly, images of 2 pins were projected in a given muscle were completed within 1 hour after the induction of Ba2+ contracture.

The criteria for accepting a given adult papillary muscle preparation were the same as used previously: 1) diameter less than 1 mm, 2) maximal developed force more than 4 g/mm², 3) resting force less than 17% of total force at L_{m,max}, and 4) small transverse inhomogeneity of fiber shortening.16 For newborn papillary muscles and atrial trabeculae, 4 successive preparations were analyzed after a preliminary series of 2–4 trials for each type of muscle.

**Protocols**

After the equilibration period in normal Tyrode’s solution, the dynamic stiffness of normal and thyrotoxic muscles was studied at resting state at segment lengths that corresponded to 95 and 100% of systolic L_{m,max}. Stimulation was stopped for a short period, and muscle length was sinusoidally oscillated at 9 different frequencies ranging from 0.05–50 Hz. The amplitude of muscle length perturbation was adjusted so that the central segment length would change approximately 1% of L_{m,max} at any of the different frequencies of perturbation. Stimulation was resumed as soon as the last frequency was measured.

To induce Ba2+ contracture in a muscle, the muscle was incubated in Ca2+-free Tyrode’s solution for 5–15 minutes until the force developed during a twitch became almost zero, after which BaCl2 was added to the Ca2+-free Tyrode’s solution. Some thyrotoxic muscles, especially muscles with a diameter greater than 0.9 mm, showed persistent spontaneous contractions after adding barium to the calcium-free Tyrode’s solution; these muscles were discarded. All muscles were activated with 0.5 mM Ba2+ except newborn muscles, which required 1.0 mM Ba2+ for sufficient activation.

The dynamic stiffness of normal and thyrotoxic papillary muscles was measured at 95% L_{m,max}. The dynamic stiffness of newborn papillary muscle and atrial trabecular muscle was measured at the length of 95% L_{m,max}. The muscles were perturbed by sinusoidal length oscillations with an amplitude of approximately 1% L_{m,max} (1% L_{m,max} in newborn and atrial muscle) at 15–21 different frequencies (0.05–50 Hz). Experimental measurements in a given muscle were completed within 1 hour after the induction of Ba2+ contracture.

**Data Analysis**

The modulus of dynamic stiffness (K) was defined as the ratio: K = ΔF/ΔL, in which ΔF is the amplitude of the force oscillations and ΔL is the amplitude of muscle length oscillation. The oscillation of central segment length was measured for normal and thyrotoxic muscles and of whole muscle length for newborn and atrial muscles. Each dynamic stiffness value was normalized to the value that would be obtained if each unit (whole muscle or central segment) had a length of 10 mm and a cross-sectional area of 1 mm².

The time difference between the peaks of the force and length oscillations was expressed as a phase shift. A positive value of the phase shift means that the force wave leads the length wave.
The table shows the comparison of control, thyroxine-treated, and newborn animal groups. The results indicate that thyroxine treatment produced a hyperthyroid state, as evidenced by elevated hormone concentrations and changes in cardiac hypertrophy and heart rate.

### Results

#### General Features of Hyperthyroid State

Measurement of serum concentrations of triiodothyronine (T3) and thyroxine (T4) confirmed that the protocol of thyroxine treatment produced a hyperthyroid state. As Table 1 shows, both hormone concentrations were elevated to levels more than 5 times control. Consequently, the thyroxine-treated rabbits lost 22% of their initial body weight, while the sham-treated rabbits that had saline injections increased their weight slightly (7%). Cardiac hypertrophy, judged by the ratio of heart weight to body weight, was clearly recognized in the thyroxine-treated rabbits (Table 1), even when the initial body weight before thyroxine treatment was used for normalization. Heart rate was also elevated approximately 60% by thyroxine. All of these alterations match the typical pattern of changes observed in a hyperthyroid state. Since none of the variables in Table 1 showed any significant difference between sham-treated or untreated control rabbits, muscles from both sets were combined into one control group.

Newborn rabbits showed significantly higher ratios of heart to body weight and higher heart rates than normal adult rabbits.

#### Isometric Twitch Contraction

Despite the overall cardiac hypertrophy, the papillary muscles from thyroxine-treated rabbits were not longer or thicker than muscles from control rabbits (Table 2). The percent shortening of the central segment during twitches also showed no significant difference among normal, thyrotoxic, newborn, and atrial cardiac muscles of the rabbit.
ence between normal and thyroxine-treated muscles nor any statistical difference compared with our previous report. The major functional difference between thyrototoxic muscles and normal muscles (Table 2) was the rate of contraction, judged by the time to peak force (TPF), time from peak force to half relaxation (T½R), and normalized maximal rate of force development. The amount of developed force and resting force between the thyrotoxic and normal groups, on the other hand, was not different. These results are consistent with earlier reports.

Atrial trabeculae showed significantly shorter TPF and T½R in comparison with normal papillary muscles (Table 2), a result consonant with an earlier study. Developed force was also significantly less in atrial trabeculae and in newborn papillary muscles compared with normal adult values. However, in the newborn, the timing of twitch contractions did not reveal a clear-cut increase of speed. Relaxation (T½R) was faster, but the timing of force development (TPF and maximal dF/dt normalized for developed force) did not differ significantly from the adult.

Mechanical Properties of Thyrotoxic Muscles in Barium Contracture

The total force during contracture of thyrotoxic muscles was not significantly different from normal muscles. A representative frequency spectrum of the stiffness modulus and phase shift of a thyrotoxic papillary muscle is presented in Figure 1 and compared with that from a normal cardiac muscle. The stiffness modulus curve of the thyrotoxic muscle showed a constant value in the low frequency region, but it rapidly decreased from 0.8 Hz to a minimal value at 2.5 Hz. From the minimal value, the stiffness curve increased very rapidly at first and then more moderately. The marked change in stiffness with frequency was mainly due to the dramatic change in force response to sinusoidal length oscillations because the length oscillation of the central segment was kept approximately constant (about 1% Lmax) over the range of frequencies. The phase curve decreased gradually. This pattern of change in stiffness modulus and phase with frequency is almost identical to that reported for normal papillary muscles.

However, the frequencies at which comparable portions of the stiffness spectrum occurred were markedly higher in the thyrotoxic muscles (Figure 1, Table 3). One clearly discernible and meaningful marker is the frequency (fmin) at which stiffness became minimal. The fmin more than doubled for thyrotoxic muscles (2.6 ± 0.2 Hz, range 2.5–3 Hz) compared with normal muscles (1.2 ± 0.2 Hz, range 0.8–1.5 Hz). Also, the frequency at which phase shift became +45° was shifted similarly to fmin. The frequency at which phase shift became most negative also appeared to be significantly higher in the thyrotoxic state (Table 3), although the precise location of these points was much less certain. The frequency of maximum phase shift also tended to be elevated in thyrotoxic muscles, but significance could not be demonstrated.

While the dynamic stiffness spectrum clearly shifted to higher frequencies, the levels of stiffness at comparable regions of the spectrum did not change in thyro-
toxic muscles (Figure 1, Table 3). At low frequencies ($K_f$), at the minimum of stiffness ($K^*$), and at high frequencies ($K^h$), the thyrotoxic muscles had levels of stiffness statistically similar to normal muscles. Phase shifts were also similar, although the maximum positive shift was slightly greater in the thyrotoxic case.

To summarize the trends in Figure 1 and Table 3, the stiffness modulus and phase shift curves of thyrotoxic papillary muscles demonstrated a horizontal shift toward higher frequency regions from the curves of normal muscle, but they exhibited little vertical shift.

Nonlinearities observed in normal muscles in Ba$^{2+}$ contracture$^{16}$ were also recognized in thyrotoxic muscles: a mild distortion of the force waveform around the frequency of minimal stiffness (2.6 Hz in the case of thyrotoxic muscle), decreased mean force level (90.6 ± 3.7% of the unperturbed force level) at high frequencies of perturbations (50 Hz), and slightly increased mean length of the central segment (increment of 0.34 ± 0.18% $L_{\max}$) during high frequency oscillations (50 Hz). These phenomena seem to be related to the active processes of muscle contraction.

In 2 thyrotoxic muscles, the effect on stiffness modulus and phase shift curves of changes in the mean segment length around which oscillations occurred was observed. Figure 2 shows the spectra (both in contracture and at rest) for mean segment lengths of 90%, 95%, and 100% $L_{\max}$. The effect of length on thyrotoxic muscle was the same as that reported for normal muscle.$^{16}$

For the highest frequency studied, the relation between stiffness modulus and force is presented in Figure 3 for thyrotoxic and normal muscles. Because of the shift toward higher frequencies in thyrotoxic muscles, stiffness at 50 Hz was chosen for comparison with 30 Hz for normal myocardium. The stiffness-force relation of thyrotoxic muscles could be well described by a linear regression equation $Y = 5.26X + 1.48$ ($r = 0.91$), which was not significantly different from that of normal muscles ($Y = 6.38X - 2.60$, $r = 0.94$, solid line) and that of thyrotoxic muscle by $Y = 5.26X + 1.48$ ($r = 0.91$, dashed line), in which $Y$ is stiffness modulus in g/mm and $X$ is contracture force in g/mm².

Resting properties (resting force level as a fraction of developed force and dynamic stiffness as a function of frequency) did not show any difference between thyrotoxic and normal muscles.

Effects of Temperature

When temperature was increased from 18° to 28° C in 3 normal papillary muscles, the total force of Ba$^{2+}$ contracture decreased to 46% of lower temperature force.

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FIGURE 3. Stiffness-force relation of normal and thyrotoxic papillary muscles in Ba$^{2+}$ contracture. Solid symbols, normal muscles; open symbols, thyrotoxic muscles; square, $L_{\max}$; circle, 95% $L_{\max}$; triangle, 90% $L_{\max}$. Central segment stiffness was obtained for normal muscles at 30 Hz of length oscillations and for thyrotoxic muscles at 50 Hz. The stiffness-force relation of normal muscle could be described by linear regression equation $Y = 6.38X - 2.60$ ($r = 0.94$, solid line) and that of thyrotoxic muscle by $Y = 5.26X + 1.48$ ($r = 0.91$, dashed line), in which $Y$ is stiffness modulus in g/mm and $X$ is contracture force in g/mm².

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FIGURE 2. Effect of central segment length on segment stiffness (modulus and phase shift) for thyrotoxic muscle. Solid symbols show curves from Ba$^{2+}$ contracture at three different lengths. Open symbols show curves of same muscle at rest for same segment lengths. Mean force levels in Ba$^{2+}$ contracture were 3.53, 3.40, 3.09 g/mm² at 100, 95, 90% $L_{\max}$. Resting forces were 0.39, 0.21, 0.12 g/mm², respectively.
A typical set of stiffness modulus and phase shift curves obtained from a newborn papillary muscle (using whole muscle length instead of central segment length) is shown in Figure 5. In our previous study,\textsuperscript{16} stiffness modulus and phase shift values calculated from whole muscle length were slightly different from those calculated from the central segment, but \( f_{\text{min}} \) from the whole muscle was the same as that from the central segment. Therefore, only \( f_{\text{min}} \) using whole muscle data from newborn papillary muscles was compared with \( f_{\text{min}} \) using central segment data from normal adult papillary muscle. The \( f_{\text{min}} \) of 4 newborn papillary muscles was \( 2.3 \pm 0.3 \) Hz, which was significantly higher than that of normal adult papillary muscles (\( p<0.001 \)).

**Atrial Muscle**

Total contracture force of 4 atrial muscles at 95\% \( L_{\text{m,max}} \) was \( 0.98 \pm 0.46 \) g/mm\(^2\).

Figure 6 is a representative set of stiffness modulus and phase shift curves from atrial trabecular muscle in Ba\(^{2+}\) contracture. As with newborn muscle stiffness, these data were calculated from the length of the whole muscle, not of the central segment. Therefore, \( f_{\text{min}} \) of atrial muscle was only compared with that determined in papillary muscle preparations using segment length for stiffness calculation. The average \( f_{\text{min}} \) of 4 atrial

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**Newborn Papillary Muscle**

With a Ba\(^{2+}\) concentration of 1 mM, the average total contracture force of 4 newborn papillary muscles was \( 0.94 \pm 0.38 \) g/mm\(^2\) at 95\% \( L_{\text{m,max}} \).
FIGURE 6. Whole muscle stiffness (modulus and phase shift) of atrial trabecular muscle (open circles) compared with central segment stiffness of normal ventricular papillary muscle (solid circles). Atrial trabecula was held at 95% L_{max}, and whole muscle length was used to calculate stiffness. L_{max} = 3.5 mm; diameter, 1 mm, total contracture force, 0.57 g/mm^2; Ba^{2+} concentration, 1 mm. Normal papillary muscle was same as that shown in Figure 1.

Discussion

Correlation Between Frequency of Minimum Stiffness and Intrinsic Contractile Rate

This study tested the hypothesis that an increase in the intrinsic contractile rate of cardiac myofilaments will be manifest as a shift to higher frequencies of the spectrum of dynamic stiffness in contracture. Biochemical studies by others (as will be described below) have demonstrated that ventricular myocardium from thyroxine-treated rabbits or from newborn rabbits and atrial myocardium from normal rabbits all contain a greater proportion of isozymes of myosin that have faster biochemical rates than the dominant isozyme in normal adult rabbit papillary muscles. Correspondingly, this study has shown that all of these muscles have spectra of dynamic stiffness that are shifted towards higher frequencies (Figure 7).

The most sharply defined frequency in any of the dynamic stiffness spectra in Figure 7 is the frequency of minimum stiffness (f_{min}). This landmark was used as an empirical indicator of the intrinsic contractile rate of cardiac myofilaments. It was shown previously\(^{16}\) that f_{min} is not altered by changes in length or degree of activation in normal papillary muscles. The present
Studies of skeletal muscle also demonstrate that the critical frequencies of mechanical response are sensitive to and reflect changes in the contractile proteins. Kawai found that the spectrum of dynamic stiffness was significantly different between fast- and slow-twitch skeletal muscle fibers. The frequency of minimum stiffness, for example, was approximately 30 times smaller in the slow fibers.

**Mechanical Properties of Thyrotoxic Myocardium**

Thyrotoxic treatment alters the distribution of myosin isoforms of adult rabbits from a V$_1$-dominant to a V$_m$-dominant pattern. The change in the myosin isozyme distribution correlates well with the higher activity of myosin Ca$^{2+}$-ATPase for the V$_m$-dominant pattern. Although the biochemical composition of the hearts was not measured in this study, the thyroxine-treated muscles were probably composed primarily of the V$_m$ myosin isoform because our protocol for thyroxine treatment was the same as in the two studies cited above and because data on heart weights and rates (Table 1) and for twitch contractions (Table 2) were quite similar to those reported by many others for thyroxine treatment.

The results from our sinusoidal length perturbation experiments on muscles in Ba$^{2+}$ contracture were compared with biochemical findings in the literature. The stiffness modulus curve and phase shift curve of thyrotoxic papillary muscle were found to shift to a higher frequency region without changing the shape of the curves or the levels of stiffness. This observation demonstrates in a functional way that the contractile machinery in thyrotoxic muscle became faster than normal. The average $f_{\text{in}}$ of thyrotoxic papillary muscles was 2.2 times higher than normal. This functional ratio (2.2) is within the range of biochemical ratios of contractile rates (1.9–3.0) found when the Ca$^{2+}$-ATPase activity of thyrotoxic myocardium was compared with that of normal myocardium. While this correspondence between ensemble means of mechanical function (from our data) and biochemical function (from the literature) is encouraging, the statistical strength of the inference drawn may be limited by several sources of biologic variability. Since the content of myosin isoforms varies between rabbits and between regions of the normal heart, a more powerful comparison could be achieved by performing the biochemical tests on the papillary muscle that was tested mechanically. Note that in thyrotoxic hearts, however, the distribution of myosin isoforms was more homogeneous.

The series elastic property of thyrotoxic myocardium does not seem to be different from that of normal muscle. The relation between stiffness at high frequency and force was nearly identical for both groups of muscles. This finding is contrary to a previous report by Parmley et al, but the effect of damaged ends was not excluded in that early study.

Because of the increased metabolic demand in thyrotoxic myocardium, especially during contraction, diffusion of oxygen to the core of the papillary muscle...
Mechanical Properties of Newborn Rabbit Papillary Muscles

Similar to thyrotoxic-treated muscles, the average f_{min} of newborn papillary muscles was nearly twice as fast as that of normal adult papillary muscles (2.3 vs. 1.2 Hz). This finding is consistent with biochemical studies of 1-week-old rabbit hearts, which showed a V_{1} dominant myosin isozymic pattern. In general, the myocardium of immature animals exhibits a higher activity of myosin ATPase than adults, which we believe would be the basis for a higher f_{min}.

Pins were not inserted in newborn papillary muscles because they were so short. Coping with this constraint illustrates another of the potential advantages of f_{min} as a functional correlate of myofibrillar contractile rates, namely, that there may be little error introduced by damage at the ends of the muscle. Quite possibly, reliable information may be obtained from a simpler preparation than one in which the shortening of the central segment must be observed to obtain accurate velocities. These potential advantages are based on our previous work, which showed no difference between f_{min} measured using central segment length or whole muscle length. Newborn papillary muscles also showed significantly smaller stiffness moduli than adult muscles over the entire frequency range. This observation is probably related to the low contracture force of newborn muscle, which was less than 40% of the adult value.

Mechanical Properties of Atrial Rabbit Muscles

The electrophoretic pattern of atrial myosin differs from that of ventricular myosin, and the ATPase activity of atrial myosin is higher than that of ventricular myosin. These biochemical data support the functional difference between atrial and ventricular mechanical stiffness spectra that were observed in the present study. On average, f_{min} was 3.7 times faster for atrial muscle (4.4 vs. 1.2 Hz). This functional ratio is comparable to the ratio of atrial to ventricular myosin Ca^{2+}-ATPase activities (2.1–3.5) reported for rabbits by Yazaki et al. and Banerjee. For the atrial vs. ventricular comparison, the changes in twitch properties parallel the changes in f_{min} and biochemical rates. For example, canine atrial muscles have a higher shortening velocity than ventricular papillary muscles. Among the four different kinds of heart muscle examined, TPF and T/VtR were shortest for atrial muscle (Table 2). Similarly, f_{min} of atrial muscle was the fastest of all (Figure 7). Such a correlation between TPF during twitches and the rate of myofilament contraction was previously recognized in skeletal muscles.

Effect of Temperature on Normal Adult Papillary Muscle in Barium Contracture

In an earlier study, the dip of the stiffness modulus curve became so shallow and wide at physiologic temperature (36°C) that it was considered an insignificant phenomenon. In contrast, our results (Figure 4) suggested that the drop in stiffness near f_{min} could become even sharper and deeper as temperature approached the physiologic level. However, the earlier findings may have been due to partial rigor developing in the muscle. In a preliminary study at 34°C, evidence for partial rigor was obtained immediately after the induction of contracture, which is why 28°C was chosen as the highest temperature during contracture.

Of particular interest is the observation that the negative phase shift at low frequencies became larger when temperature was increased from 18°C to 28°C. This increase would correspond to an increase in the area contained within the counterclockwise-rotating loop in the force-length diagram (Lissajous’ loop) at higher temperature. Since the area within the loop is presumed to be the work performed by the muscle, this observation can be interpreted as showing that cardiac muscle generates more work at higher temperature. A similar temperature effect was observed in glycerinated insect fibrillar muscles.

The stiffness modulus measured at high frequencies decreased in magnitude at 28°C. This drop was most likely due to the decreased force level at higher temperature because high-frequency stiffness is nearly proportional to the force level during contracture.

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