Maximum lengthening velocity during isotonic relaxation at preload in canine papillary muscle

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TAMIYA, KOUICHI, MOTOAKI SUGAWARA, AND YASUHISA SAKURAI. *Maximum lengthening velocity during isotonic relaxation at preload in canine papillary muscle.* Am. J. Physiol. 237(1): H83–H89, 1979 or Am. J. Physiol.: Heart Circ. Physiol. 6(1): H83–H89, 1979.—The characteristics of the lengthening velocity during isotonic relaxation at preload in the blood perfused canine papillary muscle were investigated. The force imposed on the papillary muscle is shifted from total load to preload at the instant the muscle reaches the end-systolic fiber length in the conventional afterloaded contraction. Therefore, the isotonic bar is kept at the end-systolic position until the contractile force of the papillary muscle falls to the predetermined preload. Then the isotonic lengthening at preload follows. The lengthening continues until the muscle length reaches its initial fiber length. The results are *1)* maximum lengthening velocity of the cardiac muscle, $-dL/dt_{max}$, is linearly related to the extent of muscle shortening (ΔL); *2)* $-dL/dt_{max}$ divided by ΔL is independent of preload, total load, and contractility under conditions of constant temperature of 37°C; *3)* $\ln[(-dL/dt_{max})/\Delta L]$ decreases linearly with the reciprocal of absolute temperature over a range of 25.3–38.5°C under conditions of constant preload and total load; and *4)* frequency of contraction ranging from 45 to 180 min^{-1} has almost no effect on $(-dL/dt_{max})/\Delta L$ at 37°C.

blood-perfused papillary muscle; papillary muscle mechanics; electromagnetic lever system

THE RELAXATION OF THE CARDIAC MUSCLE in a functioning ventricle in vivo is divided into two phases: the phase of isometric tension decay and the phase of quasi-isotonic lengthening. We have recently reported the mechanical properties of the mammalian papillary muscle in the first phase (13), but the second phase is not clearly understood. In the conventional afterloaded contraction of the papillary muscle (11), the isotonic lengthening at the predetermined total load immediately follows the isotonic shortening, and after the muscle is stretched to its initial length the isometric relaxation occurs. In contrast to this, the relaxation of an intact ventricle begins with the isovolumic phase and the quasi-isobaric filling phase follows it.

To simulate such a process occurring in the intact ventricle, we performed a new experiment on papillary muscle in which an isotonic lengthening at predetermined preload occurs after the isometric relaxation at the end-systolic fiber length. The effects of changes in loading conditions, contractility, frequencies of contraction, and the temperature of the cardiac muscle on the maximum lengthening velocity during isotonic relaxation phase were investigated.

METHODS

For all the experiments we used canine papillary muscle obtained from the right ventricles of dogs anesthetized with pentobarbital. The muscle was perfused at a constant pressure with arterial blood from a donor dog by a cross-circulation technique (3). The temperature of the perfusing blood was changed in a range of 25.3–38.5°C. Resting tension of the muscle is not as easily identified as it is in the skeletal muscle. Therefore the specimen muscle was held at a length at which developed force is maximal (L_{max}) at the beginning of the experiment in order to determine the reference muscle length. Then we measured the average cross-sectional area of the muscle and calculated its volume assuming that the muscle is an elliptical cone. The number and basic characteristics of the papillary muscles studied in the experiments are shown in Table 1.

The muscle was mounted vertically, with the upper end connected to the tip of an electromagnetic lever system. A force transducer was attached to the tip of the lever to measure the muscle force. A noncontacting displacement measuring system was set up to measure changes in muscle length. To shift the force imposed on the muscle from total load to preload and vice versa, a reset-set flip-flop system was used, which was switched when the lever was at the end-systolic position and at the end-diastolic position.

Electromagnetic lever system and measuring system. A simplified diagram of the apparatus employed in the present study is shown in Fig. 1. The aluminum lever is attached by epoxy cement to the shaft of a rotary coil suspended in a strong field of a permanent magnet and grounded electrically. This system is basically analogous to that previously reported by Brutsaert et al. (1) and provides torque of more than 0.4 kg·cm when the current through the coil is 1.0 A. A light strain-gauge force transducer (compliance less than 6×10^{-7} cm/dyn) is attached to the tip of the lever. A disk of thin aluminum foil (thickness 0.1 mm, diameter 10 mm) to function as the target of a noncontacting displacement sensor is attached near the force transducer. The force transducer is calibrated by hanging known weights and has a linear output over a range of 0–30 g, which encompasses the range of force encountered in the present experiments.

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The noncontacting displacement measuring system (KAMMAN KD-2300 2S-SPL, measuring range 0.5–5.0 mm, nonlinearity less than ±0.5%, frequency response 0–20 kHz at −1 dB point) is set immediately above the target with small clearance (within about 5 mm) and is calibrated with a micrometer.

To measure the total equivalent mass of the lever system, a light spring was attached instead of a papillary muscle. An isotonic load of 10 g generated by a constant current through the rotary coil was imposed on the spring. Then the lever system was set in free oscillation. The measured frequency of oscillation f (Hz), amplitude of displacement S (cm) and force F (dyn) acting on the tip of the lever are 29.0 Hz, 0.082 cm, and 490 dyn, respectively. The equivalent moving mass of the lever system ($F/4\pi^2f^2S$) is 180 mg (mass), which is insignificant compared with the magnitude of present measurements.

Control unit. The control unit consists of a reset-set flip-flop, a summing amplifier, and two potentiometers. While the lever is kept in contact with the end-diastolic stopper (upper point of contact), the electromagnetic lever system is controlled to provide the predetermined total load for the specimen papillary muscle, but the substantial force imposed on the muscle is determined by the end-diastolic muscle length, which is controlled by the position of the end-diastolic stopper and the stress-strain relation of the muscle at rest. Following the electrical stimulation, the muscle develops the contractile force. At the instant the contractile force of the muscle overcomes the predetermined total load, the lever begins to leave the end-diastolic stopper and the muscle begins to shorten against the predetermined total load. The electromagnetic lever system continues to impose the predetermined constant total load on the muscle until the lever touches the end-systolic stopper (lower point of contact) because the current through the coil is kept constant by the nature of the current amplifier. Thus the phases of isometric and isotonic contraction of the papillary muscle are obtained.

At the instant the muscle length reaches its shortest length, the isotonic bar touches the end-systolic stopper, the position of which is carefully and empirically adjusted. This contact resets the flip-flop, and the force provided by the electromagnetic lever system is shifted to the predetermined level (corresponding to the preload in the succeeding contraction) within 5–8 ms. In spite of the reduction of the lever force, the muscle does not shorten any more as it is held by the end-systolic stopper. Then the muscle relaxes isometrically at the end-systolic fiber length until the tension decreases to the predetermined lever force (preload in the succeeding contraction). As soon as the contractile force of the papillary muscle decreases below the predetermined lever force, the lever leaves the end-systolic stopper, and the muscle begins to lengthen isotonically at the predetermined preload until the muscle length reaches its end-diastolic fiber length corresponding to the preload. The force imposed on the muscle during the isotonic relaxation phase is set by the adjustment of the preload potentiometer to be equal to the force determined from the stress-strain relation of the resting cardiac muscle and the end-diastolic muscle length. In this way, the isotonic lengthening of the papillary muscle at the predetermined preload is accomplished. The maximum lengthening velocity of the papillary muscle dL/dt_{max} is measured during this phase. At the instant the muscle reaches its end-diastolic fiber length, the lever touches the end-diastolic stopper (upper point of contact), which sets the flip-flop to shift the

TABLE 1. *Physical characteristics of papillary muscles*

Muscle	L_{max}, mm	Volume, mm^3	Avg Cross-Sectional Area, mm^2	Maximum Stress, g/mm^2	Maximum $\Delta L/L_{max}$
1	10.0	85.1	8.5	2.9	0.11
2	16.0	136.7	8.5	2.6	0.19
3	13.0	53.9	8.5	4.9	0.19
4	13.0	137.8	10.6	1.5	0.13
5	12.5	75.6	6.0	3.4	0.14
6	12.0	78.3	6.5	2.8	0.12
7	12.0	90.5	2.5	1.7	0.17
8	15.0	105.7	2.0	1.7	0.08
9	12.0	53.9	4.5	3.1	0.13
10	14.5	79.9	5.5	1.5	0.11
Mean ± SD	13.0	89.74	6.87	2.61	0.14
	±1.75	±9.42	±1.99	±1.07	±0.04

L_{max}, resting muscle length at which developed force is maximum.

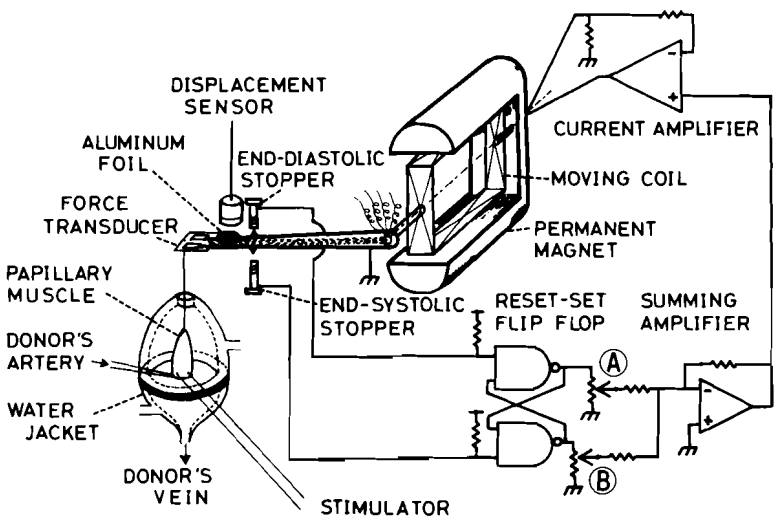

FIG. 1. Diagram of apparatus for measurement of lengthening velocity of the papillary muscle during isotonic relaxation at preload following the isometric relaxation at the end-systolic fiber length. Ⓐ: total load adjusting potentiometer. Ⓑ: preload adjusting potentiometer.

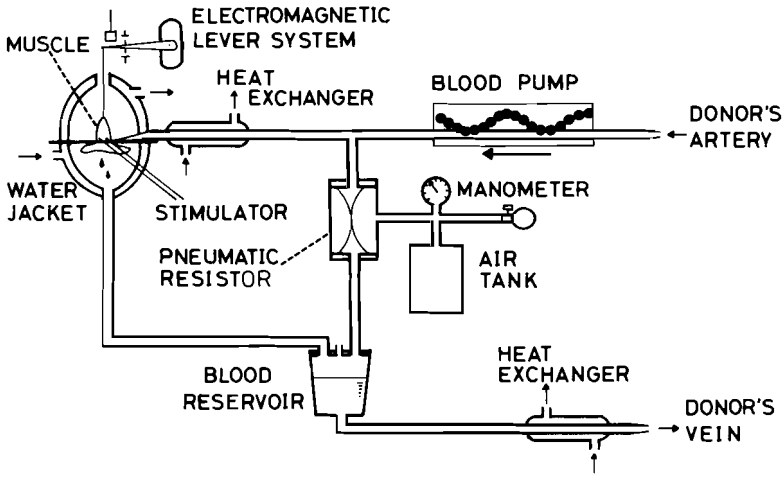

FIG. 2. Experimental setup for perfusion of papillary muscle by cross circulation.

force provided by the lever system to total load. But a significant force remains (preload) imposed on the muscle as previously mentioned. Then the cycle of contraction and relaxation similar to intact ventricle is accomplished.

Stimulation. Most of the muscles were stimulated at a rate of 60 pulses/min with rectangular pulses of 1 ms duration about 10% above threshold, while some were stimulated at a rate of 45–180 pulses/min, as desired. The stimuli were provided through two platinum electrodes arranged longitudinally along both sides of the papillary muscle.

Preparation of the papillary muscle. The preparation of the papillary muscle and the blood donor animal was basically the same as that of Endoh and Hashimoto (3), which is described in detail elsewhere (13). Briefly, two dogs were used for each preparation: one to provide the papillary muscle and the other as the blood donor supplying arterial blood for the papillary muscle preparation through the blood circuit. Dogs were anesthetized with the intravenous injection of sodium pentobarbital (25 mg/kg body wt) and heparinized with the intravenous administration of sodium heparin (1 mg/kg body wt) and ventilated with an artificial respirator. The common carotid artery and the external jugular vein of the donor dog were cannulated and connected to the arterial and venous cannulas of the blood circuit, respectively, as shown in Fig. 2.

The dog used to provide a specimen papillary muscle of the right ventricle was bled and thoracotomized; then the heart was excized and immediately plunged into normal saline at 4°C. While spontaneous contraction was inhibited, the septal artery was exposed and cannulated with a fine polyethylene tube, and the right and left ventricular walls were removed. All branches except the anterior branch to the anterior papillary muscle were ligated with silk thread. Then the base of the papillary muscle was fixed on an acrylic resin board by piercing the endocardium with two needles, and the tendinous end of the papillary muscle was connected to the strain-gauge force transducer by a short segment of a fine copper wire (0.08 mm in diameter). No pins were placed in the area that was suspected to have a branch of the septal artery perfusing the papillary muscle.

Blood circuit. The blood circuit used in the present experiments for the perfusion of the papillary muscle preparation at constant pressure and constant temperature is shown in Fig. 2. A pneumatic resistor was placed in parallel with the papillary muscle in order to maintain the perfusion pressure at 100 mmHg (13.33 kPa). The temperature of the papillary muscle was maintained at 37°C in most experiments by a double-walled glass chamber and a heat exchanger placed on the arterial perfusion tube; both of them were perfused with water from a servo-controlled thermostat bath (Coolnics Circulator CTE-240), which can provide fluid at a temperature range of −10°C to +100°C for the heat exchangers. The temperature of the muscle was monitored by a certified mercury thermometer soaked with the blood in the arterial tube near the specimen muscle. The surface of the papillary muscle was kept moist, for it was covered by the double-walled glass chamber. The blood leaving the papillary muscle through Thebesian veins was collected and returned to the donor dog via the external jugular vein. The blood returned to the donor dog was maintained at 38°C by another heat exchanger placed on the venous tube and supplied by another thermostat bath in all the experiments to avoid a hypothermic state of the donor dog.

Recording systems. Force, muscle length, and electronically differentiated changes in force (dF/dt) and muscle length (dL/dt) were displayed as a function of time on a multichannel recorder and recorded on the paper at a speed of 250 mm/s. The recorder has a flat response over a frequency range of 0–50 Hz. The maximum lengthening velocity of the papillary muscle during the isotonic relaxation at preload ($-dL/dt_{max}$) was obtained by the record on the paper.

RESULTS

The left panel of Fig. 3 shows a typical record of muscle length, force, and their first derivatives with respect to time in the conventional afterloaded contraction and the following relaxation. As Jewell and Wilkie (7) reported concerning the skeletal muscle, and as Parmley and Sonnenblick (11) reported about cardiac muscle, the rapid extension of the muscle by total load in the course of muscle relaxation is followed by a phase of isometric relaxation at the initial fiber length.

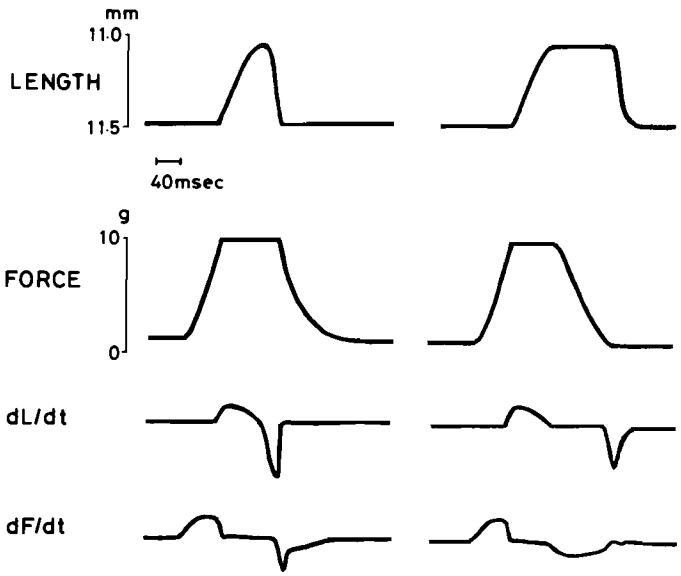

FIG. 3. *Left*: representative isotonic relaxation at total load and isometric relaxation at end-diastolic fiber length following afterloaded contraction. *Right*: representative isometric relaxation at the end-systolic fiber length and the isotonic relaxation at preload following afterloaded contraction. dF/dt is first derivative of force with respect to time.

In contrast to this, in the right panel of Fig. 3 is a typical record of the quantities described above when the muscle was allowed to change its length at constant preload after the phase of isometric relaxation at the end-systolic fiber length. In this case, the characteristic muscle lengthening curves were different from those obtained from relaxation process following conventional afterloaded contraction.

We measured the maximum lengthening velocity ($-dL/dt_{max}$) of the papillary muscle in this process under conditions of various total loads and preloads. The end-systolic fiber length is affected by the changes in preload in this kind of preparation (13). Then, neither the end-diastolic fiber length nor the end-systolic fiber length by itself can be the primary determinant of $-dL/dt_{max}$. Both increase in preload and decrease in total load resulted in the increase in the extent of muscle shortening (ΔL). The effects of increase in ΔL on $-dL/dt_{max}$ during isotonic relaxation at preload are plotted in Fig. 4. Because $-dL/dt_{max}$ was dependent only on ΔL and was practically linearly related to ΔL, $-dL/dt_{max}$ divided by ΔL can be regarded as constant under conditions of various combinations of preload (0.24–2.03 g/mm^2) and total load (0.32–4.87 g/mm^2). This constant will be a good index reflecting the mechanical characteristics of the cardiac muscle during isotonic relaxation after isometric relaxation.

To evaluate the effects of changes in contractility on $-dL/dt_{max}$, isoproterenol (0.2 μg), calcium dichloride (1 mg), and propranolol (50 μg) were injected into the perfusion blood under conditions of various combinations of preload and total load. The results are also shown in Fig. 4. These interventions showed almost no effect on the linear relation between the extent of muscle shortening and $-dL/dt_{max}$.

The effect of changes in frequency of contraction on $-dL/dt_{max}$ were evaluated over a range of 45–180 beats/min. Although the increase in ΔL and $-dL/dt_{max}$ were observed, the value of $(-dL/dt_{max})/\Delta L$ appeared to remain constant at a constant temperature of 37°C (Fig. 5). The slopes of the curves in the ΔL vs. $-dL/dt_{max}$ plots are different in different muscles. The summary of the relation between ΔL and $-dL/dt_{max}$ is shown in Table 2.

Thus the relation between ΔL and $-dL/dt_{max}$ appears to be characteristic of an individual muscle under given conditions. By changing these conditions the relation between ΔL and $-dL/dt_{max}$ can be altered. For instance, the effects of changes in temperature on the relation between ΔL and $-dL/dt_{max}$ is shown in Fig. 6: semilogarithmic plots of $(-dL/dt_{max})/\Delta L$ against reciprocals of absolute temperature ($1/T$). The linear relation between $1/T$ (K^{-1}) and $(-dL/dt_{max})/\Delta L$ (s^{-1}) was obtained over a

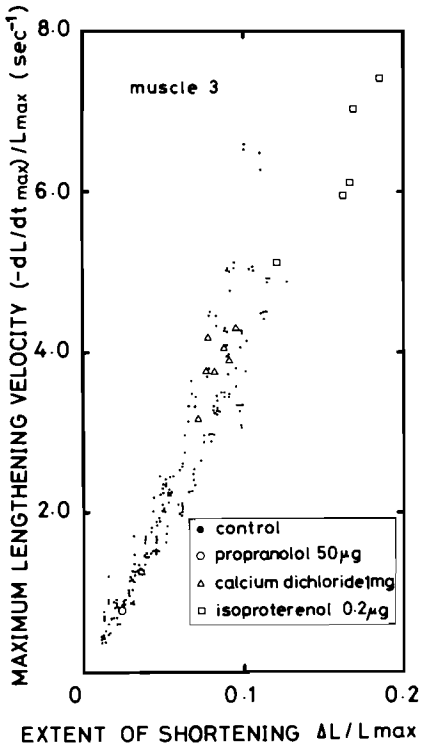

FIG. 4. Values of $(-dL/dt_{max})/L_{max}$ in one papillary muscle under conditions of constant contractility, various preloads, and total loads. Effects of three different inotropic interventions are also shown. Note that $(-dL/dt_{max})/L_{max}$ is linearly related to the extent of muscle shortening ($\Delta L/L_{max}$) and that this relation is not affected by the inotropic interventions.

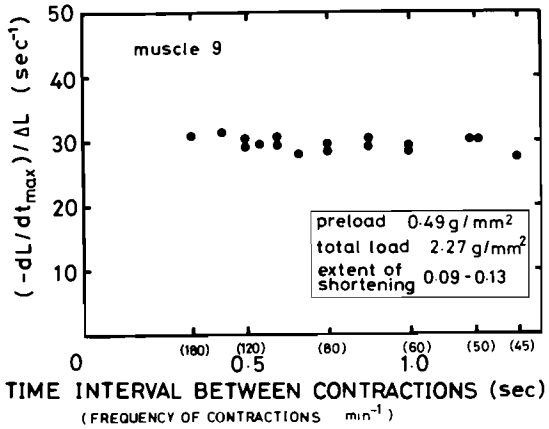

FIG. 5. Effect of changes in the frequency of contraction on the value of $(-dL/dt_{max})/\Delta L$ in one papillary muscle.

range of 25.3–38.5°C under conditions of constant preload and total load.

The experimental apparatus described in the present article determines the stretching force imposed on the muscle during the isotonic relaxation phase (which is equal to preload of the succeeding contraction in the isotonic relaxation process at preload) independently of the end-diastolic fiber length of the muscle. Figure 7 demonstrates the effect of increasing stretching force. The onset of isotonic relaxation advanced along the trace of muscle force during isometric relaxation as the stretching force increased under conditions of constant total load and constant end-systolic and end-diastolic fiber length. Figure 7*A* shows a typical trace of the isotonic relaxation at preload. Figure 7*B* is a record of the case when the stretching force was adjusted to a magnitude between preload and total load. Figure 7*C* is a record of the case when the stretching force was adjusted to the same magnitude as total load, which shows a conventional afterloaded contraction and the following relaxa-

TABLE 2. *Functional correlates in blood-perfused papillary muscle*

Muscle	Preload, g/mm²	Total Load, g/mm²	Relation between $-dL/dt_{max}$ (Y) and Extent of Shortening (X)		
1	0.09–1.13	0.46–2.83	$Y = 0.05 + 19.23X$	$r = 0.886$	(163)
2	0.09–0.98	0.40–2.58	$Y = 0.72 + 21.95X$	$r = 0.908$	(134)
3	0.24–2.03	0.32–4.87	$Y = -0.19 + 46.17X$	$r = 0.925$	(169)
4	0.18–0.63	0.57–1.55	$Y = 0.05 + 18.58X$*	$r = 0.980$	(33)
			$Y = -0.07 + 12.91X$†	$r = 0.884$	(35)
5	0.13–1.34	0.71–3.72	$Y = 0.07 + 48.16X$	$r = 0.906$	(180)
6	0.32–0.76	0.78–2.76	$Y = 0.30 + 29.03X$	$r = 0.937$	(99)
7	0.13–0.94	0.40–1.72	$Y = 0.14 + 32.89X$	$r = 0.922$	(88)
8	0.28–1.16	1.14–1.72	$Y = 0.11 + 34.44X$	$r = 0.971$	(38)
9	0.45	2.72	$Y = 1.35 + 40.71X$	$r = 0.952$	(16)
10	0.35–0.82	0.73–1.49	$Y = 0.11 + 11.64X$†	$r = 0.847$	(14)

Numbers in parenthesis denote number of measurements. * Muscle temperature 36°C; † muscle temperature 32°C. In other cases, muscle temperature was maintained at 37°C.

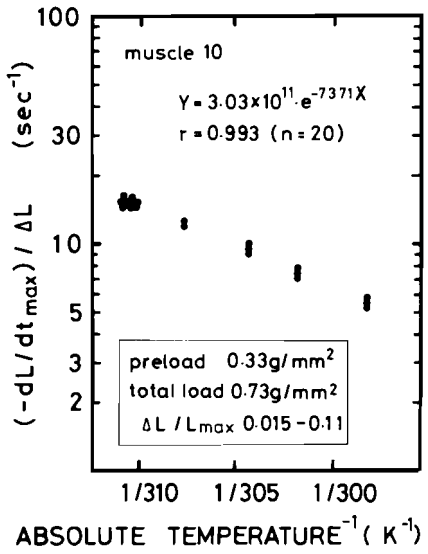

FIG. 6. Relation between $(-dL/dt_{max})/\Delta L$ and reciprocal of absolute temperature of muscle.

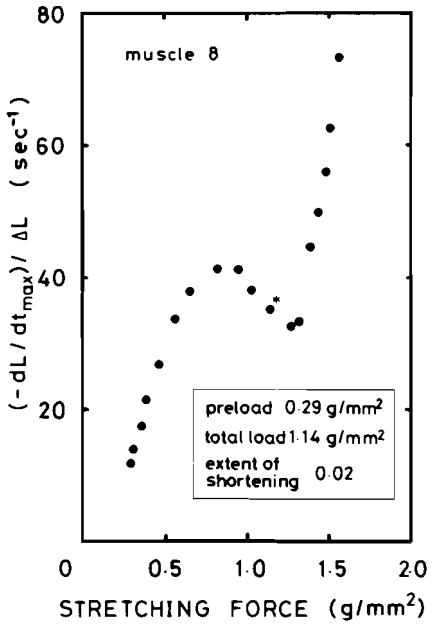

FIG. 8. Relation between stretching force imposed on muscle during isotonic relaxation phase and value of $(-dL/dt_{max})/\Delta L$. Asterisk shows $(-dL/dt_{max})/\Delta L$ values obtained from the case of conventional afterloaded contraction and following relaxation.

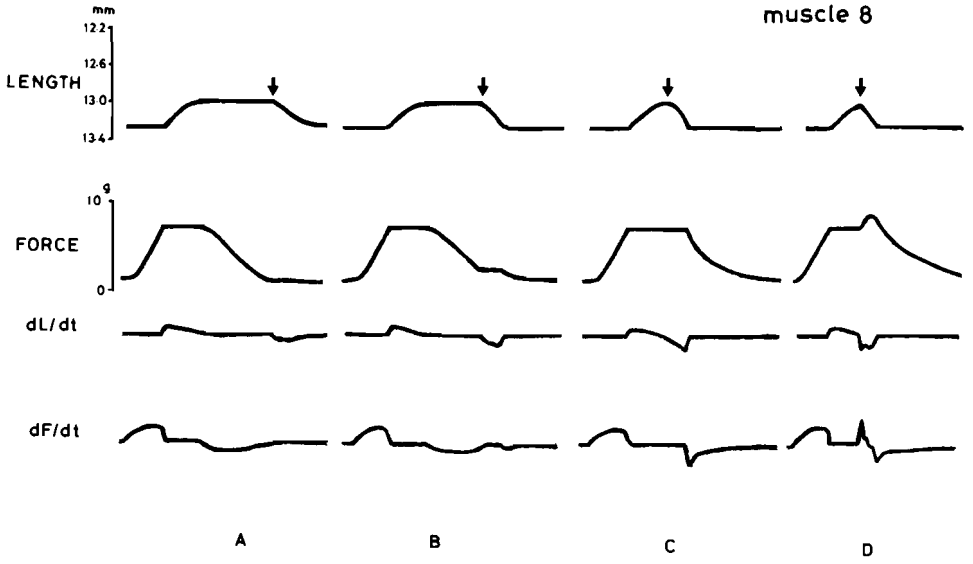

FIG. 7. Representative recordings of muscle length, muscle force, and their first derivatives with respect to time. *A*: the isotonic relaxation at preload following afterloaded contraction and isometric relaxation at end-systolic fiber length (standard of reference). *B*: stretching force imposed on the muscle during relaxation phase adjusted to value between total load and preload. *C*: conventional afterloaded contraction and following relaxation; stretching force was set equal to total load. *D*: higher stretching force than total load was imposed on the muscle during relaxation phase. Arrows indicate onset of the isotonic lengthening.

tion. In the case shown in Fig. 7*D*, the stretching force was increased above total load. Figure 8 shows the relation between the stretching force imposed on the muscle during the isotonic relaxation phase and $(-dL/dt_{max})/\Delta L$ under conditions of constant total load, end-diastolic fiber length, and contractile state of the muscle, consequently constant extent of muscle shortening.

DISCUSSION

Although the relationship between the mechanics of contraction and relaxation in cardiac muscle has been investigated in diverse preparations (2, 10, 11, 14, 15), the fundamental regulatory characteristics of the lengthening velocity of the cardiac muscle, which are expected to correlate with the rate of changes in ventricular volume during the filling period in the cardiac cycle of the heart, have not been elucidated. This is the first experimental study in which the relations between the maximum lengthening velocity of the muscle $(-dL/dt_{max})$, loads, the contractility of the muscle, the extent of muscle shortening, and the temperature of the muscle have been determined quantitatively under conditions of hysteretic force-length loop: a contraction very similar to an actual contraction in vivo in the blood-perfused canine papillary muscle preparation.

Because changes in muscle length are measured isotonically, the series elastic element of the mechanical muscle model (SE) (5, 6) remains unchanged in its length. Therefore, the lengthening velocity of the contractile element of the model (CE) is considered to be equal to the lengthening velocity of the muscle. In the present experiments, because the papillary muscle lengthens, roughly speaking, linearly with time, the CE velocity during isotonic relaxation phase would practically be constant. This constancy of CE lengthening velocity during isotonic relaxation at preload is curiously consistent with the results obtained by Parmley and Sonnenblick (11) from the calculation of tension decay and the exponential SE extension curve during isometric relaxation at the initial fiber length following afterloaded contraction. However, the calculated maximum lengthening velocity of the CE during the phase of exponential tension decay following afterloaded contraction is about $-0.25\ s^{-1}$ from their report (11). This value is considerably less than that of $-dL/dt_{max}$ obtained from the present experiments. For example, at an extent of muscle shortening of 0.1 (the ratio of the substantial amount of muscle shortening (mm) to the resting muscle length at which developed force was maximum (mm)), the maximum lengthening velocity of the CE during isotonic relaxation at preload was about $4.0\ s^{-1}$.

The three inotropic interventions, $CaCl_2$, isoproterenol, and propranolol, do not modify the relation between the extent of muscle shortening and the maximum lengthening velocity. Because, in the present experiments, the extent of lengthening is equal to that of shortening, an increase in the maximum lengthening velocity is accompanied by a proportional increase in the extent of lengthening. This suggests that the time required to accomplish the relaxation is constant under conditions of constant temperature, although the precise biochemical processes during relaxation is unknown. The stimulating effect of catecholamines on the relaxation characteristics of the cardiac muscle at lower temperature has been emphasized. (10, 11) However, Morad and Rolett (10) reported that at higher temperature, although there was a marked positive inotropic effect on addition of epinephrine, the time to peak tension and the rate of tension fall divided by peak tension hardly seemed to be augmented. The present experimental results are consistent with this observation, as most of our experiments were carried out at 37°C. At higher temperature, the rate of calcium sequestration of the sarcoplasmic reticulum is higher; therefore the stimulating effects of administration of catecholamines, $CaCl_2$, and the increased frequency of contraction are considered to be less pronounced than at lower temperature.

An increase in attained tension (total load) was accompanied by an almost parallel increase in the level of maximum rate of tension fall $(-dT/dt_{max})$ during isometric relaxation at the end-systolic fiber length (13). This suggests a close relation between the amount of calcium ions released to the myofilaments and the rate of sequestration of calcium from myofilaments by the sarcotubular system. Langer and Brady (9), and Harigaya and Schwartz (4) suggested that the enzyme associated with the sarcotubular system is sensitive to the concentration of calcium ions and temperature. The linear relation between the reciprocal of the absolute temperature ($1/T$) and the logarithm of $-dL/dt_{max}$ divided by ΔL is analogous to that between temperature and the reaction rate constant in a chemical reaction, i.e., the Arrhenius equation.

The specimen papillary muscles used in the present study are considered to have been maintained well enough for the investigation of the regulatory mechanisms of contraction and relaxation because the maximum developed force and other characteristics are similar to those obtained from nonexcized papillary muscle preparations (8, 12).

In summary, the present work demonstrates that $-dL/dt_{max}$ is linearly related to the extent of muscle shortening, independent of the frequency of contraction and the contractile state of the cardiac muscle, and that $-dL/dt_{max}$ increases with the temperature of the muscle under the condition that preload is equal to the stretching force imposed on the muscle during lengthening after isometric relaxation at the end-systolic fiber length. From these experimental results, $-dL/dt_{max}$ divided by the extent of muscle shortening might be a useful index of relaxation characteristics of cardiac muscle.

APPENDIX

The frequency of this test oscillation is given by

$$f = \frac{1}{2\pi}\sqrt{\frac{F}{MS}}$$

where M is the equivalent mass of the lever system. Then, f depends on the ratio F/S, i.e., the spring constant. If we choose a spring with larger spring constant, the measured f will be higher.

The only significant quantity obtained by this test oscillation is the equivalent mass of the lever system, and that was 180 mg. The effects of the equivalent mass can be examined in two ways. One approach is

to evaluate the effect on the measurement of the velocity of lengthening and shortening. The second is to evaluate the effect on the measurement of force.

Effect of the equivalent mass on the velocity measurements. We take the case of Fig. 3 as an example and consider the lengthening process (in which the effect of equivalent mass is larger than in shortening). In this case, preload $\simeq$ 1 g (generated by a constant current through the rotary coil), extent of lengthening $\Delta L \simeq 0.05$ cm, acceleration, α, of the tip of the lever without papillary muscle is

$$\alpha = \frac{\text{preload}}{M} = \frac{1\ (\text{g}) \times 980\ (\text{cm/s}^2)}{0.18\ (\text{g})} \simeq 5400\ \text{cm/s}^2$$

where M is the equivalent mass.

The time required for the lever to travel the distance ΔL without muscle is

$$\sqrt{\frac{2\Delta L}{\alpha}} = \sqrt{\frac{2 \times 0.05\ (\text{cm})}{5400\ (\text{cm/s}^2)}} \simeq 0.004\ \text{s} = 4\ \text{ms}$$

This is within the range of tolerable error in this sort of experiment.

Effect of the equivalent mass on the force measurement. In the case of Fig. 3, mean acceleration, α, of the tip of the lever during lengthening of the muscle is about 100 cm/s^2. Then, the inertia force caused by the equivalent mass is

$$M\alpha = 0.18\ (\text{g}) \times 100\ (\text{cm/s}^2) = 18\ \text{dyn} = 0.018\ \text{g (force)}$$

This is 1/50 of the preload (1 g) and does not cause any significant error. Therefore, the properties being measured are surely those of the muscle, not of the lever.

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