A One-Dimensional Viscoelastic Model of Cat Heart Muscle Studied by Small Length Perturbations during Isometric Contraction

By Louis Loeffler, III, and Kiichi Sagawa

ABSTRACT

To develop a model of heart muscle, we studied cat papillary muscle contracting in a quasi-isometric condition under a fixed inotropic state. The properties of resting muscle were determined by using a step stretch of less than 1.2% of Lmax for initial lengths from 85 to 100% of Lmax. The passive force response suggested the model of the passive branch (Fig. 1). All five parameters were small at muscle lengths below 95% of Lmax but increased markedly at longer lengths. The properties of contracting muscle were studied with a sinusoidal length change (amplitude < 0.15% of LmM, frequency 0.1-35.0 Hz). The frequency response of active (total minus passive) stiffness suggested the model of the active branch (Fig. 1). We determined the dependency of the elastic elements (K, Ks) and the viscous element (C) on length and time by recording the frequency response at various combinations of length and time. Ks varied linearly with active force (FA). K and C exhibited time courses that paralleled FA up to 0.6Lmax, and they maintained their values until 1.4Lmax. K then fell toward zero, whereas C exhibited a secondary rise before it fell toward zero. K was independent of length up to 95% of Lmax and then began to decline, but C varied in proportion to muscle length.

KEY WORDS passive stiffness active stiffness cat papillary muscle time-varying elasticity time-varying viscosity stress-relaxation frequency response modulus of mechanical impedance

In identifying the mechanical properties of heart muscle, the investigator is faced with many difficulties (1-4). Unlike amphibian skeletal muscle, resting heart muscle exhibits significant passive elastic force in a range of lengths below Lmax, the muscle length at which the maximum isometric force is actively developed. Therefore, this passive force and its changes must always be considered in calculating active force during heart muscle contraction. But to do so, the investigator must choose some form of a mechanical analogue, e.g., the Maxwell configuration or the Voigt configuration, of a three-element model. Brady (1, 2) has discussed how difficult it is to decide by an experimental approach which configuration is more pertinent to heart muscle, although for some particular purpose one configuration may be preferable to the other. Fung (5) has pointed out that the two models are mathematically equivalent. But this equivalency does not always free an investigator, who usually does not have a unique reason to choose either model, from having to design two sets of loading conditions and calculate two sets of data to reduce the externally measured force and shortening to those of the contractile element (2). Pollack (6) has discussed the possibility that use of a three-element model, either the Maxwell or the Voigt configuration, might invalidate the once widespread thesis (7) that the maximum shortening velocity of the contractile element with zero afterload, which has been estimated using two-element models, is independent of initial muscle length.

Apart from this problem, the force-velocity relation of heart muscle has been found to be nonhyperbolic in the hands of many recent investigators (8-11). Edman and Nilsson (12) have only been able to obtain a hyperbolic force-velocity relation by a quick-release method when they maintain the length of the contractile element constant. However, there is an accumulating body of evidence (13-15) suggesting that, in addition to being nonlinearly dependent on the instantaneous force, the stiffness of the series elastic element changes with time after stimulation. If this phenomenon is really the case, then the length of the contractile element cannot be calculated by as simple a method as has been used previously. This fact compounds the already difficult situation that exists in heart muscle compared with that in skeletal muscle: heart muscle cannot be tetanized under physiological circumstances, and its active state develops more slowly than that of skele-
nal muscle, reaches a peak, and then decays without plateauing during measurements of the contractile element properties (16). One way to circumvent this time dependency is to measure shortening velocity following a quick release of the muscle to various isotonic afterloaded forces at an identical instant after the stimulus (16). However, there also is a family of evidence that shows uncoupling effects of large, quick releases or stretches of a muscle on the active state (2). Taken together, these findings emphasize that all of the mechanical properties of the subcomponents of muscle (not just the contractile element) need to be identified as a function of both time and length. Furthermore, since the natural contraction of heart muscle does not involve length changes as rapid and drastic as those used in quick-release experiments, it is by far preferable to use a small, smooth perturbation signal (either force or length).

The present paper describes a mechanical analogue of heart muscle. We did not initially decide to choose either the Maxwell or the Voigt configuration of a three-element muscle model, although one temporary assumption that we employed (see the first paragraph of Methods) endowed the model with a feature shared by the Maxwell model, i.e., the parallel combination of the active and the passive branch. We perturbed isometrically contracting heart muscle with extremely small changes in length. In characterizing the dynamic stiffness of the contracting muscle, we used a family of frequencies of sinusoidal length changes (frequency-response method). Finally, we extensively determined the parameter value of each component of our muscle model as a function of time at various muscle lengths. The model resulting from this exhaustive study is fairly complex and needs further testing, particularly of its applicability to contractions that involve muscle shortening. The model, however, consists of elements that are all clearly defined and quantified; therefore, it is ready for testing.

Methods

For a clear explanation of the methods, we need to describe the fundamental assumptions and the terminology used in our analysis. First, we termed the force responses to length changes observed in the resting muscle "passive force," whereas the responses of contracting muscle were termed "total force." We then made a temporary assumption that the total muscle force observed in contraction is the sum of the passive force and an active force generated in addition. This assumption was temporary, and we will present in Discussion experimental findings that support this contention. The implication of this temporary assumption is that passive and active properties of muscle are independent and that their respective models should be coupled in parallel (Fig. 1).

Second, we considered muscle mass to be negligible, i.e., inertial force is small compared with elastic and viscous forces. Loeffler (17) has shown that under the conditions employed in this study inertial force never exceeds 0.001 g as compared with elastic and viscous forces which range up to 0.4 g. Thus, we considered the assumption to be well justified.

Third, we treated the muscle as a one-dimensional, lumped parameter system. The validity of this assumption depends on the uniformity of both the cross-sectional geometry and the mechanical properties along that length of the muscle utilized experimentally. To ensure uniformity of cross-sectional geometry, we reported data only from muscles with a cylindrical shape between muscle clips and a length-diameter ratio at the base and the tendinous end of the muscle. We used length perturbations (either step or sinusoidal) that were quite small compared with those used in most of the earlier studies. The amplitude of the length perturbation never exceeded 1.2% of Lmax when we were probing the properties of resting muscle and was less than 0.15% of Lmax when we were probing the properties of contracting muscle. Because we expected the properties of the elements of the model to be nonlinear functions of length, we used an amplitude small enough to obtain a piecewise linear approximation of these properties about a specific average length. By repeating this procedure at various lengths within the full range of physiologically important muscle lengths, the length dependence of each property could be determined.

Finally, the perturbation techniques employed in this study assumed that muscle properties were uninfluenced by the probing signal itself. We examined this assumption by comparing the isometric force curves obtained from a preparation with and without the sinusoidal length perturbation used in the rest of the preparations. Over the time interval considered in this study, 0 ≤ τ ≤ 2Tmax (Tmax = time from stimulation to peak force), the average value of the perturbed quasi-isometric force deviated no more than 6% from the unperturbed isometric force, and this deviation occurred only at 2Tmax. When Templeton et al. (18) used a similar sinusoidal length perturbation with
an amplitude of 0.6% of \( L_{\text{max}} \) (four times as large as our largest perturbation), they observed a 30% reduction in the peak developed force. On the basis of this comparison, we think that the length perturbation which we used did not affect the development of the active state significantly.

**EXPERIMENTAL PREPARATION**

Nine papillary muscles from nine chloroform-anesthetized cats were used in this study. The sole criterion for muscle selection was that the length-diameter ratio at \( L_{\text{max}} \) had to be greater than or equal to 4.3. A papillary muscle, quickly removed from the heart and kept in a bath of oxygenated Krebs-Ringer's solution, was clipped on each end well inward from both the base and the tendinous tip. It was then placed in the muscle bath; a stainless steel wire (0.01 inches) was connected with the force and length transducers. The muscle was stretched until it supported 0.5-g of passive tension (depending on its cross-sectional area), stimulated via platinum plate electrodes at 10 stimuli/min, and allowed to sit overnight (about 14 hours) at 25°C while it was contracting isometrically. The temperature of the bath was reduced to 20°C, and the experiment was conducted at this temperature.

**APPARATUS**

The details of the apparatus and other technical points have been described elsewhere (17). Only a brief account will be given in this paper. Muscle length was controlled by a Brush recorder penmotor (model 864750) and drive amplifier (model 869223). The signal generator was offset to zero and just the step response was displayed at high gain. Absolute force and length were measured. The step increase in length was introduced 5 seconds after muscle stimulation (the muscle was stimulated every 6 seconds, whereas measurable active force vanished in less than 3.5 seconds). The major portion of the transient response occurred within 1 second following the step extension. The passive force level at the end of 6 seconds (after the next beat) was taken as the steady-state force.

From a recording of the step response, several quantities were measured: (1) \( \Delta L = \) step increase in muscle length, (2) \( \Delta F_p = \) difference between the steady-state force and the initial force before the stretch, and (3) \( \Delta F_A = \) difference between the transient and steady-state forces at 20, 40, 60, 80, 100, 140, 200, 300, 500, and 700 msec after the step.

**DETERMINATION OF ACTIVE MUSCLE PROPERTIES**

We used the sinusoidal length-perturbation technique to probe the properties of active muscle; we perturbed an isometrically contracting muscle with sinusoidal length changes of an amplitude less than 0.15% of \( L_{\text{max}} \) over the frequency range from 0.1 to 35 Hz. This range spans the frequency components of in situ length changes of heart muscle. At a particular value of frequency (\( f \)), length (\( L \)), and time (\( t \)), we defined the active stiffness \( K_A(f, L, t) = \Delta F_A(f, L, t)/\Delta L \), namely, the ratio of the amplitude of changes in the active force at time \( t \) to the amplitude of the given change in length about the mean length \( L \). The stiffness varied with the frequency of the input perturbation and therefore was plotted as a function of frequency. We obtained such frequency-dependent characteristic curves of active stiffness at many muscle lengths between 80 and 100% of \( L_{\text{max}} \) and also at many instants of time between 0.0 and 2\( L_{\text{max}} \).

The frequency-response method has been extensively used in a black-box approach to an unknown system (19). It requires that a system respond to a train of steady sinusoidal inputs with a train of steady sinusoidal outputs. Naturally, this requirement could not be realized in our muscle system which was periodically contracting and relaxing every 6 seconds. Therefore, strictly speaking, we cannot call our experiments an application of the frequency-response method. To compensate for this deficit of...
steady state particularly for the low frequency range, we used an envelope technique that will be described later in this section. A theoretical analysis of the use and the limitations of this technique for the present study is available elsewhere (17). The step-response method, another way to probe the dynamic properties of a system, is equally unsuited, because it also requires that the system of interest does not change its properties while the transient response is being observed. We think that, although our approach is not fully justifiable, the sinusoidal force responses as studied do contain valuable information.

For the determination of total stiffness, the reference signal to the servomotor was varied sinusoidally about a d-c voltage. This procedure produced a sinusoidal length change with an amplitude of less than 11μ about a preset constant muscle length. The resultant force response manifested itself as sinusoids superimposed on the slowly varying level of isometric force. To cancel this slow change in baseline, we generated a sine-wave signal that matched very closely the time course of isometric force over periods of time up to 1 second. The remaining signal, which consisted mainly of sinusoidal responses, was displayed at a high gain on the oscilloscope (Fig. 2) along with muscle length and absolute force signals amplified at low gains. When the frequency of the length perturbation was as high as 30 Hz, the response could be faithfully recorded simply by passing the total force through the high-pass filter and amplifier (Fig. 2).

To calculate total stiffness at a particular instant in time, the peak-to-peak force excursion at that instant resulting from the sinusoidal length variation must be measured. At relatively high frequencies (> 20 Hz) the envelope of maximum and minimum force excursions was clear, but at lower input frequencies the muscle stiffness changed significantly over one cycle of length variation and the maximum and minimum force excursions could not be recorded over a reasonably short interval of time within which the muscle stiffness could be regarded as time independent. To overcome this problem, we input each one of 16 length perturbation frequencies (0.1–35 Hz) repeatedly with a 60, 90, or 120° phase shift with respect to the onset of each contraction cycle. When the consecutive force responses to these repetitive inputs with an identical frequency but variable phase relations were superimposed on the memory oscilloscope, a well-defined envelope of peak-to-peak force excursion built up as shown in Figure 3. From such data, total stiffness (K_T) was calculated as

\[ K_T(L, t, f) = \frac{\Delta F_T(L, t, f)}{\Delta L} \]

where \( \Delta F_T(L, t, f) \) is the amplitude of the sinusoidal total force response measured at length \( L \), time \( t \), and frequency \( f \), \( \Delta L \) is the amplitude of the input length change, and \( E_L \) is elastance of the equipment coupled in series with the muscle. The term involving \( E_L \) corrects for the contribution of the compliance of the equipment to the measured total stiffness.

Based on the temporary assumption that the passive and active branch properties are independent, active stiffness \( (K_A) \) was found by subtracting out passive stiffness:

\[ K_A(L, t, f) = K_T(L, t, f) - K_R(L, t, f) \]

where \( K_R(L, t, f) \) is passive stiffness determined at length \( L \) and frequency \( f \) from the step response to stretch during the resting phase prior to muscle stimulation.

Rigorously speaking, the estimation of active muscle stiffness by Eq. 2 contains a problem. The amplitude of the total force response \( \Delta F_T(L, t, f) \) depends not only on the amplitudes of \( \Delta F_P(L, t, f) \) and \( \Delta F_A(L, t, f) \) but also on the phases of these componental sinusoidal forces. That is, Eq. 2 is valid only if the componental branches of the muscle model are purely elastic so that the force response has no viscous element. In fact, the muscle force responses, either in rest or in contraction, exhibited viscous properties. Therefore, we should first identify the real part of the active force response \( |\Delta F_A(L, t, f)| \) by subtract-
ing the instantaneous $F_p(L, t, \phi)$ curve from the instantaneous $F_t(L, t, \phi)$ curve, determine the amplitude of the frequency component of interest in the difference wave, and then divide this value by $\Delta L$. This procedure was technically difficult to carry out with accuracy. Besides, the approximation of $K_A(L, t, \phi, t')$ by Eq. 2 introduced a relatively small error (underestimation), because the values of the elastic and viscous parameters of resting muscle were small compared with those of the active muscle in the major range of $L$.

After normalizing $K_A$ as explained earlier, log$_{10}K_A$ was plotted vs. log$_{10}f$. That is, we constructed a Bode plot of active muscle stiffness at a specified length $L$ and a specified time $t'$ from the onset of the stimulus. From many such Bode plots specified at various lengths and times, an appropriate model for active muscle with pertinent values for the components of the model can be ascertained.

The measurement of $K_A$ at the very low frequency range ($K_A L^2 \tau$) was unreliable because of the large variation of the data. Therefore, we attempted to supplement the data on $K_A L^2 \tau$ by the following analysis. We caused the muscle to contract isometrically from various initial lengths ($L_i$) changed after a time interval of 1 minute or longer in steps of 150/$\mu$s from 80 to 100% of $L_{max}$. $K_A L^2 \tau$ was calculated as the ratio of the increase in active force (at a particular time $t'$), $\Delta F_A(L_i, t')$, to the increase in initial muscle length, $\Delta L_i$. This value was regarded as a zero-frequency (i.e., static) stiffness and was associated with $\phi$ and length $L$, the midpoint of the length interval over which the muscle was extended, i.e., $L' = L_i + \Delta L_i/2$.

Phase Shift.—We found that the determination of phase shift between the output force and the input length signal was difficult in the low-frequency range below 5 Hz because of the small output signal. Moreover, most phase shift occurred in this range. Therefore, the following special technique was used. Because we biased the phase of two successive inputs, the length traces "intersected" on the memory oscilloscope at an instant of time. Likewise, the force-response curves to these two inputs also intersected on the memory oscilloscope. We measured the time interval between these intersections of the length and force traces. Phase could then be calculated as

$$\phi = \pm \Delta t (360^\circ),$$

where $\phi =$ phase shift at some particular length, time, and frequency, $\Delta t =$ time interval between length and force intersections, and $\Delta t_o =$ period of the sinusoidal frequency. Finding the time interval at various time regions in one contraction cycle and repeating this procedure at several frequencies gave the phase-response data.

Results

$L_{max}$ AND $F_{max}$

The mean value of $L_{max}$ determined in nine muscles was 7.1 ± 0.3 (SE) mm. The mean value of $A_{max}$ was 1.44 ± 0.23 mm$^2$. The mean ratio of $L_{max}$ to the diameter at $L_{max}$ amounted to 5.7 ± 0.4. The mean value of normalized maximum active muscle force was 4.1 ± 0.4 g. The mean $\Delta L$ used for sinusoidal perturbation was 7.0 ± 0.8$\mu$s or 0.10 ± 0.01 % of $L_{max}$.

PASSIVE MUSCLE PROPERTIES

Figure 4 is an example of semilog plots of $\Delta F_p$, the change in passive force with time after a step increase in length. The amplitude of the input step increase in length was kept constant at 75$\mu$s, but the initial muscle length was varied. Evidently, the force response decayed with time. The straight lines (drawn in visually) emphasize the double-exponential nature of the step responses regardless of the initial muscle length from which the step was initiated. This double-exponential response was a consistent finding in all of the muscles and at all initial muscle lengths.

Based on this finding, we modeled the passive branch of heart muscle with two viscoelastic units ($K_1, C_1$ and $K_2, C_2$), both coupled in parallel with an elastic element ($K_3$) (Fig. 1). The chosen structure of the passive branch model is not unique, i.e., it is not the only one that is consistent with the observed properties of passive muscle. However, this model does contain the minimum number of linear elements needed to describe the observed step response and results in the simplest expression for evaluating the five elements. Mathematical analysis of the passive model is described in Appendix A. Eq. A-3 describes the theoretical response of the passive branch to a small step change in length.

The data for $\Delta F_p$ were first normalized and fitted numerically by a sum of two decaying exponentials using a Varian 620 computer. The program first peeled off the slower exponential and then identified the faster exponential via linear regression in the semilog domain. This process was iterated 50 times even though the values converged to within 1% of
their final values in 30 iterations. This procedure fitted the data with a standard deviation of less than 6 mg and more typically 1–3 mg.

The curve-fitting procedure yielded an initial value, $\Delta F_1$ and $\Delta F_2$, and time constant, $\tau_1$ and $\tau_2$, for both exponentials. These four numbers along with $\Delta F_{po}$ and $\Delta L$, all properly normalized, were used to calculate the five passive parameters via Eq. A-3. The relations are:

$$K_0 = \frac{\Delta F_{po}}{\Delta L}, \quad K_1 = \frac{\Delta F_1}{\Delta L}, \quad K_2 = \frac{\Delta F_2}{\Delta L}, \quad C_1 = K_1\tau_1, \quad C_2 = K_2\tau_2.$$ 

The normalized pooled data for the five parameters from experiments 1–8 are shown as a function of initial muscle length in Figure 5. Each length grouping contains five to eight data points. The symbols represent the average of each grouping, and the vertical and horizontal bars represent ±1 SE. All five passive parameter values increased with muscle length. The mean value of normalized static passive force at $L_{ma}$ was 2.4 g.

**ACTIVE MUSCLE PROPERTIES**

**Structure of Active Branch Model.**—Figures 6 and 7 present typical examples from experiment 5 of the relation between $K_T$ and input frequency (Bode plot) at several combinations of $L$ and $t$. In Figure 6 muscle length was fixed at 85% of $L_{max}$ and time was varied, whereas in Figure 7 time was fixed at 0.6$L_{max}$ and length was varied. The symbols are raw data, and the solid and broken lines are asymptotes (to be explained later).

Two things should be noted from these figures. First, the frequency response was influenced quantitatively by both $L$ and $t$. Second, the shape of the frequency response was independent of $L$ and $t$ and had a double-plateau nature, i.e., $K_T$ was relatively independent of frequency at both high and low frequencies and increased in a first-order manner (20 dB/decade) over the region of intermediate frequencies. The solid and broken lines in Figures 6 and 7 are asymptotes drawn in visually to emphasize the first-order, double-plateau nature of the frequency responses (19).

The relation between $K_A$ and frequency was presumed to be very similar to the $K_T$-frequency relation, because the magnitude of active stiffness greatly dominates that of passive stiffness at all frequencies (except for $L > 0.95L_{max}$ and $t < 0.2L_{max}$), and this situation was indeed found to be the case. We fitted all 18 frequency characteristic curves of $K_A$ from experiment 5 (obtained at 18 different combinations of $L$ and $t$) with the general expression for

**FIGURE 5**

Five passive branch parameters as functions of length. All variables are normalized. See text for abbreviations.

**FIGURE 6**

Unnormalized total stiffness vs. frequency relations at 85% of $L_{max}$ and three different instants of time ($T$) after stimulation. Crosses in this and the next figure represent the upper break frequency (19). The slope of the lines over the intermediate frequency range is 20 db/decade.

**FIGURE 7**

Unnormalized total stiffness vs. frequency relations at 600 msec after stimulation and three different muscle lengths. Only the asymptotes are shown at 98% of $L_{max}$. 

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the double-plateau response, which is
\[ K_A(\omega) = D_1 \left( \frac{1 + D_3 \omega^2}{1 + D_2 \omega^2} \right)^{1/2}, \]
where \( \omega = 2\pi f \) (radians/sec), and \( D_1, D_2, \) and \( D_3 = \) arbitrary constants. The program we used was NLLSQ, Share Distribution no. 1428 DPE 2135 (20). It fitted the nonlinear expression, Eq. 4, to the experimental data in the semilog domain via least squares. After curve fitting all 18 frequency responses, we normalized the upper and the lower plateau to arbitrary stiffness units of 10 and 1, respectively. The upper break frequency was also normalized to a nondimensional frequency of 10. The 16 data points in each frequency response were then scaled accordingly. Figure 8 shows the composite of the data from all 18 frequency responses. The solid and broken lines are the theoretical asymptotes and the stiffness-frequency relation curve expected from Eq. 4, respectively. Each square in Figure 8 represents a mean value of 5-18 data points. The very satisfying degree to which the normalized experimental data agree with the theoretical response curve clearly supports the supposition that the frequency response of active muscle can be represented by a first-order, double-plateau response characteristic. But even more importantly, it indicates that this particular characteristic is invariant with respect to muscle length and time during the contraction.

An infinite number of mechanical systems will manifest a frequency response similar to that in Figure 8 (21). Of these systems, only two are irreducible (i.e., cannot be made simpler), consisting of a minimum number of elements. These two models are labeled the active branch and the alternative active branch in Figure 1; they are the only models of active muscle that we considered in this paper. Because of their equivalence we have derived the equations for the frequency responses of both models in Appendix B, along with the algebraic relations that allow conversion from one model to the other. Eqs. B-2 and B-7 are the specific expressions of the first-order, double-plateau frequency response in terms of the elements in the active and the alternative active branch of our model, respectively. They were used repeatedly along with their limiting forms, Eqs. B-3 and B-4 and B-8 and 9, to evaluate the \( L \) and \( t \) dependency of the active parameter values.

\( K \) and \( K' \).—The active stiffness represented by the "lower plateau," or \( K_A, \) is related in a simple manner to \( K, K', \) and \( K_A \) by Eqs. B-3 and B-8. An estimate of the stiffness of this plateau was obtained by measuring \( \Delta F_A / \Delta L \), the static stiffness, as explained in Methods. Thus, the relation for the active branch model is
\[ K_{A,lp} = \lim_{\omega \to 0} K_A(\omega) = K, \]
and that for its alternative is
\[ K_{A,lp} = \lim_{\omega \to 0} K_A(\omega) = \frac{K'K_A}{K + K'A}. \]
Rearranging these equations, we obtain
\[ K = K_{A,lp} \]
\[ K' = \frac{K_{A,lp}K_A}{K_A - K_{A,lp}}, \]
where the value of \( K' \) can be independently evaluated as described later. Based on Eq. 5a and b, we could determine how \( K \) and \( K' \) varied with length and time without resorting to difficult curve-fitting techniques.

In general, \( K \) and \( K' \) are functions of both time and length. Figure 9 indicates that \( K \) and \( K' \) increased with time in parallel with \( F_A \) but maintained their near maximum values even after \( F_A \) declined to 60% of its maximum value, i.e., \( K \) and \( K' \) manifested a more prolonged plateau than did \( F_A \). Although this particular data set was obtained at \( L = 88 \% \) of \( L_{\text{max}} \), the time course of \( K \) was essentially independent of \( L \) over the interval of muscle lengths between 80 and 95% of \( L_{\text{max}} \). Figure 10 shows that for lengths from 80 to 95% of \( L_{\text{max}} \) \( K \) varied less than 10%, whereas \( K' \) varied consider-
ably. Both $K$ and $K'$ approached zero at $L_{max}$, because both quantities are related to the slope of the static relation of peak $F_A$ to $L$. Naturally, this slope becomes zero at $L_{max}$.

$K_s$ and $K'_s$—The stiffness of the "upper plateau" is related to $K_s$, $K$, and $K'_s$. This condition is explained by Eqs. B-4 and B-9. $K_{AHP}$, defined as the 30-Hz value of active stiffness, is a good estimate of the stiffness of the upper plateau, because the upper break frequency ($F_s$ in Appendix B) determined from the experimental data never exceeded 8 Hz. Thus, 30 Hz represents a frequency nearly two octaves above the maximum observed $F_s$. Therefore, the error in using $K_{AHP}$ as an estimate of the upper plateau stiffness is less than 1.3% (19). Utilizing these relations we have:

$$K_{AHP} \approx \lim_{\omega \to 0} K_A(\omega) = K + K_s,$$

and for the alternative model we have:

$$K_{AHP} \approx \lim_{\omega \to 0} K_A(\omega) = K'_s.$$

Therefore,

$$K_s \approx K_{AHP} - K,$$  \hspace{1cm} (6a)

$$K'_s \approx K_{AHP},$$  \hspace{1cm} (6b)

where $K$ can be evaluated independently as described in the previous section. Thus, $K_s$ and $K'_s$ along with $K$ and $K'$ can be evaluated without resorting to the difficult nonlinear curve-fitting procedures.

Figure 11 depicts a set of experimental data which suggests a very simple relation between $K_{AHP}$ (the total 30-Hz stiffness) and $F_T$ (total force). These data were taken from three contractions at three different muscle lengths. The data points represent $K_{AHP}$ measured at 300-msec intervals throughout the contraction phase. The broken line was drawn visually through the three points which relate $K_{AHP}$ to $F_T$ at $t = 0$, i.e., the relation of $K_{AHP}$ (passive 30-Hz stiffness) to $F_T$ (passive force). The solid lines were drawn through those points which represent the increases in stiffness resulting from active force generation. Several points can be made from Figure 11: within the scope of length between 0.92$L_{max}$ and $L_{max}$, (1) $K_{AHP} \approx K_{AHP} - K_{AHP}$ is a linearly increasing function of $F_A$, (2) the slope of $K_{AHP}$ vs. $F_T$ is much steeper than the slope of $K_{AHP}$ vs. $F_A$ (about
Normalized relation of $K$, and $K'$ to $F_A$ at 99% of $L_{max}$ from experiment 1. The straight lines were calculated by linear regression through the origin.

100% steeper), (3) the slope of $K_{A,nr}^H$ vs. $F_A$ does not change appreciably with muscle length, and (4) $K_{A,nr}^H$ depends on time indirectly through the dependency of $F_A$ on time.

$K_*$ and $K'_*$ can be calculated from data such as those in Figure 11. Figure 12 illustrates the relation of $K_*$ and $K'_*$ to $F_A$. The obvious linearity of $K_*$ and $K'_*$ to $F_A$ was a consistent finding in all muscles at all muscle lengths. Linear regression lines through the origin fit the data with a standard deviation of rarely more than 1 g/mm and with a standard deviation of the slope of less than 0.02 mm$^{-1}$ (values of the slope ranged from 3 to 4.5 mm$^{-1}$).

Although the slopes of the curves relating $K_*$ and $K'_*$ to $F_A$ ($S$ and $S'$, respectively) are independent of time, they might still be functions of muscle length. Figure 13 illustrates the relation of $S$ and $S'$ to $L$. The plot contains the pooled data from all nine experiments. Because $S$ and $S'$ appeared to be roughly linear functions of $L$, we fit each by linear regression as depicted by the broken line. The slope ± sd was 0.177 ± 0.062 for $S$ vs. $L$ and -0.392 ± 0.046 for $S'$ vs. $L$.

A $t$-test was used to determine if $S$ and $S'$ were independent of $L$ by testing the degree of significance of the deviation of the slope of $S$ or $S'$ vs. $L$ from zero. The slope of $S$ was significantly different from zero at > 0.025 level, whereas the slope of $S'$ was significantly different from zero at > 0.001 level. Because of these statistically significant differences, we cannot say that $S$ and $S'$ are independent of $L$. However, $S$ and $S'$ deviated from their average values by no more than 6% and 10%, respectively, over the length range from 80 to 100% of $L_{max}$. Therefore, as a first approximation, we can regard $S$ and $S'$ as length-independent constants.

$C$ and $C'$.—$C'$ was evaluated by curve fitting the empirical active stiffness vs. frequency relation with the theoretical expression given by Eq. B-7 via the nonlinear least-squares method mentioned previously. This procedure resulted in a set of values of $C'$, $K'$, and $K'_A$ for each frequency response. $C$ was calculated by using Eq. B-10c. Repeating this procedure for responses obtained at many combinations of $L$ and $t$, the full length and time dependency of $C$ and $C'$ were determined. Figure 14 illustrates the time course of $C$, $C'$, and $F_A$ at one muscle length, and Figure 15 gives the time course of $C$ alone at three different muscle lengths. The data in these figures are from experiments 1-8, and each grouping of data contains 5-11 data points.

These plots illustrate several points: (1) $C$ and $C'$ exhibit a plateau near $t_{max}$, (2) $C$ and $C'$ exhibit a further increase in the relaxing phase ($t > t_{max}$),
and (3) $C$ increases roughly in proportion to muscle length. The implications of these observations will be considered in Discussion.

**Phase Shift.**—The phase shift between the sinusoidal length perturbation and the resulting force response as a function of frequency was measured in muscles 4 and 8 at $L_{max}$ and 0.95$L_{max}$. Utilizing Eq. B-5, we calculated the theoretically expected phase shift–frequency relation with previously obtained values of $K$, $K_r$, and $C$ (determined at $L_{max}$ and 0.95$L_{max}$). We compared this relation with the actual phase shift–frequency relation at 16 different frequencies (0.1-35 Hz). The data deviated from the theoretical curve with a standard deviation of 8.5°. Although the data did not closely follow the theoretical prediction, the qualitative agreement was good enough to further support the assumption that the perturbing signals used in this work were small enough to obtain a piecewise linear approximation of the globally nonlinear properties of muscle.

**Discussion**

Although we excluded any preconceived notions of a muscle model as much as possible, the model proposed in this paper still needed several assumptions described in Methods. These assumptions are mostly related to technical convenience of measurements and data analysis rather than to speculative concepts on the contractile process with reference to the known microscopic structure of muscle. We sought the simplest mechanical analogue(s) which explains the extensive set of data obtained from relaxed and isometrically contracting muscle with small length perturbations. Every model parameter value was then determined from the experimental data. For these reasons, the present model is a complete model of a descriptive nature. One exception to this principle exists, i.e., the assumption of the parallel combination of the passive and active branches in the model. This initial assumption was found to be consistent with the data in Figure 11 as will be discussed later in the subsection on total muscle properties. We will also attempt in following sections to compare the parameter values that we found with those available in earlier reports.

**Passive Branch Parameters**

The analysis of the transient response of resting papillary muscle to a small step stretch led us to a model of the passive branch which consists of five subcomponents—three elastic elements and two viscous elements. The magnitudes of these parameters are highly sensitive, increasing functions of muscle length. Two points require emphasis. First, the proposed model is not unique but is one of the simplest models that can explain the observed transient response to a step elongation. Nevertheless, it is far more complex than the conventional representation of the passive behavior of heart muscle by a single nonlinear elastance with no viscosity (1). Second, the proposed model was derived by focusing on a short-term transient response to step extension, whereas most of the earlier analyses of the viscoelastic properties of resting heart muscle have paid attention only to long-term stress-relaxation over many seconds (22, 23) or minutes (15, 24). Also, little attempt has been made previously to formulate an analytical expression and quantify the parameter values to describe the observation with the exception of the work by Pinto and Fung (15). Long-term stress-relaxation was evident in our preparations, too. However, its magnitude was quite small and difficult to measure with accuracy primarily because of the extremely small extension (75µ) that we gave to the muscle. For this reason, we concentrated on analysis of short-term stress-relaxation in the present study.

Templeton and his associates (18) studied the dynamic response of papillary muscle to sinusoidal length perturbations (10 and 30 Hz) during the relaxation and contraction phases. They found that the curve relating relaxed muscle stiffness to passive force was identical to that relating total stiffness of contracting muscle to total muscle force. Namely, a single rectilinear line adequately describes the relation of dynamic stiffness to muscle force whether the muscle is contracted or relaxed. Lundin (25)
studied a bundle of frog ventricular muscle by using a crudely sinusoidal length perturbation at a frequency of 3–5 Hz. He also obtained similar results. Such was certainly not the case with our analysis, as the different slopes of the total stiffness–total force relation for relaxed muscle and activated muscle (Fig. 11) indicate. A recent brief report by Joseph and Huntsman (26) also suggests different dynamic stiffnesses at the same muscle force between contracting and relaxing muscles. The source of these differences in findings is not clear. It could be the difference in the amplitude of the forcing function. Lundin (25) used an amplitude as large as 6–8% of the fiber length and Templeton et al. (18) used 0.6% of $L_{\text{max}}$ as opposed to an average of 0.1% of $L_{\text{max}}$ in our experiments. Joseph and Huntsman (26) increased the amplitude of the sinusoidal length perturbation from 0.5 to 3.0% of $L_{\text{max}}$ and found that the slope of the stiffness–force relation curve for contracting muscle decreased with the amplitude. This finding supports our use of the very small perturbation, but it does not explain the discrepancy between our findings and those of Templeton et al. (18). Using step length changes, Brady (27) noted a decrease in the slope of the stiffness–force relation as muscle length was increased beyond about 93% of $L_{\text{max}}$. Unfortunately, his report is not detailed enough to allow a quantitative comparison between his data and ours.

The presence of the viscoelastic elements in the passive branch (with time constants of about 0.1 seconds and 1.5 seconds) greatly complicates the analysis of contracting muscle. Even so, if an investigator wanted to eliminate the contribution of the passive properties to total muscle force and shortening, he could use a computation based on Eq. A-3 or an equivalent expression. But instead of assigning fixed numerical values to passive elements as has been done in previous analyses, the functional relation of all of the variables on $L$ must be substituted as shown in Figure 5.

Since the present experiment was conducted at 20°C, the parameter values shown in Figure 5 may be too large for higher temperatures. We cannot predict the magnitude of the temperature dependence of these parameters. According to the study by Pinto and Fung (15), the time course of the long-term stress-relaxation is not significantly influenced by a temperature change from 5 to 37°C. Whether inotropic interventions such as paired stimulation, hypoxia, catecholamines, etc. affect the parameter values of the passive branch components has been a controversial subject (23). We did not investigate this topic and therefore cannot comment on it. Neither Templeton et al. (18) nor Lundin (25) found any significant effect of catecholamines on the dynamic stiffness, except via alteration of the active muscle force.

**ACTIVE BRANCH PARAMETERS**

The active branch of the proposed model is characterized by the absence of a contractile element as such. Instead, the contractile property is imparted among $K$, $K_c$, and $C$ or $K'$, $K'_c$ and $C'$. Note that the active branch is nonexistent when the muscle is relaxed; it suddenly comes into play when the muscle is activated.

$K$ and $K'$ in the active branches are closely related to the low-frequency active stiffness of muscle. $K$ is identical to the low-frequency stiffness and thus is the slope of the $F_A$–$L$ relation at all values of $L$ and $t$. At $L_{\text{max}}$, $K$ corresponds to the slope of the frequency measured maximum $F_A$–$L$ relation (3). Because this relation is approximately linear from 80 to 95% of $L_{\text{max}}, K$ should be constant over this interval of length (Fig. 10). In the proposed model, $K$ or $K'$ is the element responsible for isometric force development. However, the $K$ element alone cannot represent the time course of those contractions that involve large and quick shortening as well as force development because of the presence of the other elements described in the passive and active branches.

$K$, and $K'_c$—$K$, and $K'_c$ constitute the high-frequency elastic properties of muscle; both were found to be linear functions of $F_A$. No one has previously measured $K_c$ as it is presented in this paper. However, $K_c'$ corresponds to the series elastic component of the two-element model of muscle in which the contractile element represents the parallel combination of $K'$ and $C'$ in our alternative active branch model. Over a series of three studies using a variety of methods, Sonnenblick and co-workers (7, 28, 29) have shown that the total series elastic component stiffness of heart muscle is a linear function of $F_A$ with a finite value of stiffness $b$ at $F_A = 0$, namely,

$$\frac{dF_A}{dL} = aF_A + b.$$  

To convert this expression to the appropriate equation for the series elastic component of the Maxwell model, the contribution of the passive stiffness, i.e., $b$, must be subtracted out. Hence, we have:

$$\frac{dF_A}{dL} = aF_A.$$  

This expression for the series elastic component is the same as that for $K_c'$ (Fig. 12). At 24°C, Yeatman...
et al. (30) have reported a value for $a$ of 5.1 mm$^{-1}$, whereas the corresponding average value for $a$ from this study ($S'$ in Fig. 13) is 4.1 mm$^{-1}$ at 20°C.

More recently, Templeton and his associates (18) probed the high-frequency elastic properties of papillary muscle with sinusoidal length perturbations at two frequencies, 30 and 10 Hz. They found a similar linear relation between total dynamic stiffness and mean muscle force, $F_R$. They reported a mean value of 4.1 mm$^{-1}$ for the slope constant at 95% $L_{max}$, which is in good agreement with our value of $S'$ at 95% of $L_{max}$ (3.9 mm$^{-1}$).

From studies using quick release and stretch, Pollack et al. (13) and Noble and Else (14) recently suggested that the stiffness of the series elastic component varies not only with the force imposed on it but also with the time during muscle contraction. This hypothesis differs from the present finding on $K_s$ and $K_s'$ in that these variables are dependent solely on active force and only indirectly dependent on time. However, we measured only the slope (i.e., $K_s'$) of the force-length relation for the "series elastic component" about $\Delta L = 0$, i.e., about the isometric force level; Pollack et al. (Fig. 7 [13]) and Noble and Else (Fig. 3 [14]) measured the entire $F$-$L$ relation of the series elastic component. Our value for the stiffness of the series elastic component ($K_s'$) applies only to a narrow range of quick length changes about $\Delta L = 0$ and not to the entire $F$-$L$ relation of the series elastic component. Furthermore, neither Pollack et al. (13) nor Noble and Else (14) presented normalized mean data. For these reasons, a meaningful comparison cannot be made between their findings and ours at this moment.

$C$ and $C'$.—These two parameters constitute the viscous property of active muscle and endow the muscle with a reciprocal (but not necessarily hyperbolic) relation between maximum shortening velocity ($V$) and afterloaded force ($F$) during isometric contraction. Although $C$ and $C'$ have not previously been measured as such, $C$ is related to the slope of the $F$-$V$ plot at $V \approx 0$. This relation can be established by solving Eq. B-1 for a ramp shortening (constant velocity) input and neglecting the transient and length-dependent portions of the solution.

Meiss and Sonnenblick (31) have published several $F$-$V$ plots which they determined by controlling the shortening velocity (a ramp length change) of the contractile element instead of the afterloaded force. From Figure 6 of their paper (31), we estimated the slope of the $F$-$V$ plot at $V = 0$ as 0.8 g sec/mm at 95% of $L_{max}$ and 1.2 g sec/mm at 98% of $L_{max}$. These values compare favorably with our values of $C$ (Fig. 15) of 0.8 g sec/mm at 93% of $L_{max}$ and 0.9 g sec/mm at 99% of $L_{max}$ ($t = l_{max}$ in all cases). This agreement is quite impressive when the substantial differences in the method of determining the viscosity parameter, $C$, between the two studies is considered. Although the agreement may be coincidental, it may indicate that the present approach to identification of a mechanical analogue of active muscle is indeed a promising method. We cannot evaluate our finding of the further increase in $C$ and $C'$ after $l_{max}$ (Fig. 14), because, to our knowledge, there has been no measurement of the time course of the viscous property of active muscle. It may merely be an artifact produced by the proposed model, or it may turn out to represent an interaction between the bridges of the myosin and actin filaments in the relaxation process.

**TOTAL MUSCLE PROPERTIES**

One distinct assumption that we initially proposed for our analysis of muscle properties is the parallel combination of passive and active muscle properties. This assumption is supported by the experimental finding on the relation between total muscle dynamic stiffness and total muscle force (Fig. 11). This finding was consistent in all of the experiments; more importantly, it is independent of the aforementioned assumption. Figure 11 shows that at 92% of $l_{max}$ the passive stiffness (represented by the solid square on the broken line) was quite small and hence total stiffness was virtually equal to active stiffness. On stimulation active stiffness increased and decreased linearly with $F_A$. At 99 and 100% of $L_{max}$ passive stiffness was not negligible, but on stimulation the active stiffness of the total muscle increased linearly with developed force with essentially the same slope as that at 92% of $L_{max}$. Apparently, passive stiffness simply established the starting point for further increases in stiffness caused by activation. This additive property of stiffness is what one would expect for a parallel combination of passive and active properties; it therefore lends support to the assumed structure of the total muscle model. In contrast, if the Voigt configuration of Hill's three-element model were assumed to represent heart muscle, one would expect the relation between total muscle stiffness at 30 Hz and total muscle force to converge into a single curve shortly after stimulation regardless of the initial length and the initial muscle force. Figure 11 shows that this phenomenon does not occur.

After critically reviewing the Maxwell and Voigt configurations of a muscle model against the findings on quick length perturbations, Pollack et al. (13) concluded that the only structure consistent with the currently available information is one that has a
parallel combination of a passive elastic component and a contractile component that remains to be defined but has no inert elastance in series. The model presented in the present paper has such a structure except that the parallel passive branch consists of several viscous and elastic subcomponents instead of a single elastic element.

If we were forced to choose a single model of active muscle properties, we would prefer the active branch in Figure 1 as opposed to its alternative for the following reasons. (1) $K$ is essentially independent of $L$ from 80 to 95% of $L_{\text{max}}$, whereas $K'$ is not. Probably heart muscle operates at lengths below 95% of $L_{\text{max}}$ under most physiological conditions. Under this circumstance, the active model in Figure 1 will constitute a simpler model when it is applied to normal muscle lengths. (2) $S'$ is more nearly independent of length than is $S''$, although neither is greatly affected by length. Again, this fact would help to simplify the model. (3) To simulate muscular contraction with the alternative active branch, one needs to know the force-length relation of $K'$ when $K' = \frac{dF_A}{dL_a} = S' F_A$ ($L_a$ is the extension of $K'$). However, the only solution of this differential equation with initial conditions $F_a = 0$ at $L_a = 0$ is the trivial solution, i.e., $F_A = 0$. This solution assumes that the series elastic component is an inert elastic element; this assumption might not be true in light of the recent work by Pollack et al. (13) and Noble and Else (14). Hence, it is awkward to attempt to describe active muscle behavior with the alternative active branch. In the case of the active branch in Figure 1, one does not need to know the force-length relation for $K_a$ and the active branch does lend itself to simulation without this difficulty.

These reasons for preferring the active branch are of an operational nature and do not in any way reject the structure of the alternative active branch. In fact, the dependence of $K_s$ on $F_A$ during a single isometric contraction indicates that $K_s$ in the active branch is a controlled parameter which is somehow influenced by the contractile process. This fact in turn implies that the apparent simplicity gained by the choice of the active branch over the alternative branch may be more incidential than real. The models presented in this paper are of a descriptive nature and definitely need further testing as to their predictability. Simulations of muscle contraction involving shortening by the proposed model require reasonably complex calculations because of the multiple viscous elements in both the passive and the active branch. Nevertheless, the calculation is entirely possible now because every component of the model is clearly defined and its value is available in the present report within the length range from 80 to 100% of $L_{\text{max}}$ and the time range from 0 to $2.0\text{max}$. Once the calculation is done with the aid of a computer, the validity of the proposed model can be judged more definitely by comparing the simulated behaviors against the reported data on the time course of shortening muscle force, length, and velocity.

Appendix A

THE PASSIVE BRANCH

In analyzing the response of the passive branch of the model to a sufficiently small step change in length from a given initial length, the parameters can be treated as constants (i.e., the piecewise linear approximation). Under this condition, the values of $K'$ are linear springs; hence, $\Delta F_i = K \Delta L_i$, where $\Delta F_i$ is the change in force across the elastic element caused by a change in its length of $\Delta L_i$, and $K$ is the coefficient of elasticity. Likewise, the values of $C_i'$ are linear viscous elements; hence, $\Delta F_i = C_i(\Delta L_i)$, where $\Delta F_i$ is the force across a viscous element $C_i$ caused by a rate of change in length of $\Delta L_i$, or $d(\Delta L)/dt$, and $C_i$ is the coefficient of viscosity.

Because the passive branch model consists of three subunits coupled in parallel, the total passive force is simply the sum of the forces supported by the individual subunits. Hence, the force-length relation for the passive branch will consist of the sum of the force-length relations of the individual subunits. Furthermore, each subunit has the same general configuration, i.e., an elastic element in series with a viscous element. As to the leftmost subunit, we can consider that there is a viscous element $C_i$ in series with $K_i$, which has an infinite value. Therefore, one needs only to solve for the force-length relation of the general configuration and then sum over each subunit.

For such a general subunit, let us call $L_c$ the length across the viscous element and $L$ the length of the series combination of elastic and viscous elements; then, the equations of motion appropriate to such a system for small length changes are:

\begin{align}
\Delta F_i &= K_i(\Delta L - \Delta L_c), \\
\Delta F_i &= C_i(\Delta L_c).
\end{align}

Transforming to the complex frequency (or Laplace) domain with zero initial conditions, Eq. A-1 a and b becomes:

\begin{align}
\Delta F_i(S) &= K_i[\Delta L(S) - \Delta L_c(S)], \\
\Delta F_i(S) &= C_i[\Delta L_c(S)].
\end{align}

where $\Delta L(S)$ is the Laplace operator and can be taken as $j\omega$ for the present analysis. Eliminating $\Delta L_c(S)$ yields

\begin{align}
\Delta F_i(S) = \frac{C_i[\Delta L(S)]S}{1 + (C_i/K_i)S}.
\end{align}

Therefore, $\Delta F_p$, the change in the total passive force from
its initial condition, is
\[ \Delta F_p(S) = K_o \Delta L(S) \]
\[ + \frac{C_d \Delta L(S)}{1 + (C/K_o)S} + \frac{C_2 \Delta L(S)}{1 + (C_2/K_2)S}. \]  
(A-2)
The first term on the right side of Eq. A-2 results because \( C_i = \infty \).

The time domain response of the passive branch to a step change in length of magnitude \( A \) impressed at \( t = 0 \) is found by substituting \( \Delta L(S) = A/S \) into Eq. A-2. Thus,
\[ \Delta F_p(t) = AK_o + AK_i e^{-t/(C_i/K_i)} + AK_t e^{-t/(C_t/K_t)}. \]  
(A-3)
Because the initial conditions were assumed to be zero, \( \Delta F_p(t) \) represents only the increase in force caused by the step change in length. \( AK_o \) is the steady-state response, \( AK_i \) and \( AK_t \) are the initial amplitudes, and \( C_i/K_i \) and \( C_t/K_t \) are the time constants of the two exponentials.

### Appendix B

#### THE ACTIVE BRANCH

Because the active branch of Figure 1 is of the same general form as the passive branch, we need merely to modify Eq. A-2 to obtain the relation between force \( \Delta F_A \) and length \( \Delta L \) in the active branch. Namely,
\[ \Delta F_A(S) = K_1 \Delta L(S) + \frac{C_1 \Delta L(S)S}{1 + (C_1/K_1)S}. \]
The transfer function is:
\[ \frac{\Delta F_A(S)}{\Delta L(S)} = K \left[ \frac{1 + (K_1 + K_2)S}{1 + (C/K_1)S} \right]. \]  
(B-1)
By substituting \( j\omega \) for \( S \) in Eq. B-1 and taking the absolute value of the resulting complex number, one obtains the gain of the system to a sinusoidal input of frequency \( \omega \) (radians/sec). We define this gain as the active stiffness of the muscle, \( K_A(\omega) \). Hence,
\[ K_A(\omega) = \left| \frac{\Delta F_A(j\omega)}{\Delta L(j\omega)} \right| \]
or
\[ K_A(\omega) = \left[ \frac{K^2}{1 + \left( \frac{K_1 + K_2}{K_1} \right)^2 \omega^2} \right]^{1/2}. \]  
(B-2)
Taking the limits of Eq. B-2 as \( \omega \) approaches zero and infinity, one finds:
\[ \lim_{\omega \to 0} K_A(\omega) = K, \]  
and
\[ \lim_{\omega \to \infty} K_A(\omega) = K_1 + K_2. \]  
(B-3)
The phase shift, \( \phi \), of the system's output with respect to the input is
\[ \phi(\omega) = \tan^{-1} \frac{\text{Im} \left[ \frac{\Delta F_A(j\omega)}{\Delta L(j\omega)} \right]}{\text{Re} \left[ \frac{\Delta F_A(j\omega)}{\Delta L(j\omega)} \right]}, \]
where
\[ \phi(\omega) = -\tan^{-1} \left[ \frac{\left( \frac{C_1}{K_1} \right)^2 \omega^2}{1 + \left( \frac{C_1}{K_1} \right)^2 \omega^2} \right]. \]  
(B-5)
The break frequencies in the stiffness curve are
\[ F_1 = \frac{K_1 K}{2\pi C(K + K_1)} \]  
(Hz)
and
\[ F_2 = \frac{K_1 K}{2\pi C} \]  
(Hz),
whereas the maximum phase shift is expected to occur at a frequency midway between \( \log F_1 \) and \( \log F_2 \), i.e.,
\[ F_{\text{max}} = \sqrt{F_1 F_2}. \]

#### THE ALTERNATIVE ACTIVE BRANCH

The model of the alternative active branch is equivalent to the model of the active branch in the sense that by proper choice of parameter values, the mechanical impedance of each model can be made identical. Because of their equivalence, unique algebraic relations exist that allow one to convert from one model to the other. The derivation of these relations and the transfer function for the alternative active branch are the topics of the remaining portion of this appendix.

Referring to Figure 1, let \( L_c \) be the length of the parallel combination of \( K' \) and \( C' \) and \( L \) the length of the entire branch. The equations of motion for a small length change are
\[ \Delta F_A = K'(\Delta L - \Delta L_c), \]
\[ \Delta F_A = K'\Delta L_c + C'\Delta L_c. \]
Converting to the complex frequency domain with zero initial conditions and eliminating \( \Delta L_c \), yields
\[ F_A(S) = K' \left( \Delta L(S) - \frac{\Delta F_A(S)}{K' + C'S} \right). \]  
(B-6)
Rearranging Eq. B-6 results in
\[ \frac{\Delta F_A(S)}{\Delta L(S)} = \left( \frac{K' + K_1}{K' + C'} \right) \left[ \frac{1 + \left( \frac{C'}{K'} \right)^2 \omega^2}{1 + \left( \frac{C'}{K'} \right)^2 \omega^2} \right]^{1/2}, \]
the transfer function of the alternative active branch. Again,
\[ K_A(\omega) = \left| \frac{\Delta F_A(j\omega)}{\Delta L(j\omega)} \right| \]
or
\[ K_A(\omega) = \left( \frac{K'}{K' + K_1} \right)^2 \left[ \frac{1 + \left( \frac{C'}{K'} \right)^2 \omega^2}{1 + \left( \frac{C'}{K'} \right)^2 \omega^2} \right]^{1/2}, \]  
(B-7)
while
\[ \lim_{\omega \to 0} K_A(\omega) = \frac{K' K'}{K' + K_1}, \]  
(B-8)
and
\[
\lim_{u \to \infty} K_A(u) = K'_A.
\]  
(11-9)

Equating similar coefficients in Eqs. 15-2 and B-7 gives rise to the conversion relations between the two models of active muscle. These relations are
\[
K = \frac{K'_A K'}{K' + K'_A},
\]  
(B-10a)
\[
K_A = \frac{(K'_A)^2}{K' + K'_A},
\]  
(B-10b)
and
\[
C = \frac{C'(K'_A)^2}{(K' + K'_A)^2},
\]  
(B-10c)
or
\[
K' = K + \frac{K^2}{K'_A},
\]  
(B-11a)
\[
K'_A = K + K,
\]  
(B-11b)
and
\[
C' = \frac{C}{K'_A} \left( \frac{K^2}{K'_A} + K + 2K \right).
\]  
(B-11c)

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