Myocardial Mechanics:
Tension-Velocity-Length
 Relationships of Heart Muscle

By Donald L. Fry, M.D., Douglas M. Griggs, Jr., M.D., and
Joseph C. Greenfield, Jr., M.D.

The purpose of this paper is to present recent findings that define in greater detail the mechanical behavior of the myocardium. Investigation of the mechanical events associated with myocardial contraction involves at least three parts: 1) study of the contractile elements of the muscle itself, 2) study of the elastic and hydraulic machinery coupling the forces of the contractile elements to the ventricular cavity, and 3) study of the nature of the load imposed on the ventricular cavity by the distal circulation. Although all three parts bear on this presentation, it is with the first of these, namely, the mechanical nature of the contractile element, that this report is primarily concerned.

A. V. Hill's model of skeletal muscle has been adopted tentatively as a prototype for the mechanical behavior of cardiac muscle. A mathematical model consistent with these views is shown schematically in figure 1. It consists of a circumferential arrangement of contractile elements attached to one another by a series of relatively stiff springs. These springs represent the "series elastic component" of the heart. The elasticity represented by the other spring, shown in figure 1 to be running parallel to the contractile elements, represents the "parallel elastic component" of the heart. During systole both elastic systems will be acting in parallel. Since the stiffness of the heart during systole is many times greater than that during diastole, the stiffness of the parallel elastic component is much less than that of the series elastic component. Thus, to simplify analysis in this report, the presence of parallel elasticity was ignored during systole.

In this simple preliminary model one would visualize systolic ejection as a group of contractile elements which are shortening and applying tangential tension through a series of springs to a hollow cavity. The presence of these springs means that the shortening velocity of the contractile elements, in general, will not be the same as the shortening velocity of the muscle as a whole. It will be necessary to incorporate this complicating factor into the analysis of the contractile behavior of the myocardium.

Prior to this analysis it will be helpful to review briefly certain outstanding features of muscle behavior. Skeletal muscle exhibits two important mechanical properties. First,
the maximum isometric force that can be developed by a tetanically stimulated muscle increases with length in the physiologic range. The second important property is that for a given length the force that can be developed is a function of the velocity with which the muscle is contracting. The velocity of shortening decreases monotonically with the force developed. This "force-velocity" relationship is unique and reproducible for a given skeletal muscle, and, moreover, when muscles are corrected for size appears to be the same from muscle to muscle.

Abbott and Mommaerts applied these concepts to cardiac muscle. They demonstrated that the cat papillary muscle preparation exhibits a "force-velocity" relationship similar to that of skeletal muscle. They were also able to demonstrate the nature of the series elastic component of this muscle preparation. More recently Sonnenblick has studied the effects of initial length and of different chemical milieux on the force-velocity relationship of the cat papillary muscle preparation. The data shown in figure 2 taken from his work show a family of force-velocity curves. The velocity of muscle shortening appears on the ordinate and the force developed by the muscle appears on the abscissa. Each individual curve represents the force-velocity relationship for a specific initial muscle length. The shapes of these curves, as well as those of Abbott and Mommaerts, are very similar to the force-velocity curves for skeletal muscle obtained by Hill.

Preparations used by Abbott and Mommaert as well as those by Sonnenblick were necessarily deprived of blood supply and, a priori, cannot be considered metabolically normal. Moreover, the response of these electrically stimulated papillary muscle preparations does not necessarily reflect the behavior of a rhythmically beating, intact myocardium.

Therefore, it would be most useful to demonstrate that the force-velocity relationship applies also to the contractile elements of the intact, metabolically supported, normally beating, mammalian heart. It is the purpose of this report to present the experimental approach and results of studies designed to measure the force-velocity relationship of the intact heart. The relationships between muscle tension, velocity of shortening, and length to the measurable variables, ventricular ejection rate, intraventricular pressure, and external heart volume are derived in the appendix.

Methods

The animal preparation used in these studies is shown in figure 3. With this preparation it was possible to vary ejection rate and intraventricular pressure independently while perfusing the rest of the animal at a constant pressure of about 90 mm Hg. The heart rate did not vary under these conditions. With the vagus nerves cut this preparation was assumed to provide a heart with a constant chemical milieu.

A diagram showing the placement of instruments necessary for the three measurements, ejection rate, pressure, and heart segment length, may be seen in the upper left portion of figure 4. Ventricular ejection rate was measured with a Kolin-Kado electromagnetic flowmeter placed around the root of the aorta. Intraventricular pressure was measured through a short flexible catheter-manometer system having a uniform (±5%) dynamic response through 50 cycles/sec. Muscle segment length was measured with an electrical recording caliper placed circumferentially as shown in the diagram. An ellipsoidal ventricular configuration and a simplified pattern of contraction from the data of Rushmer and Thal are assumed in all computations.

Only the mechanical behavior of that portion...
of the intact ventricle located in the plane of the minor semiaxes of the chamber, as indicated by the shaded area in the heart diagram of figure 4, will be considered. All computations are referred to this imaginary slice of muscle which is redrawn schematically on the right of figure 4. The circumferential tension, $P$ (g/cm$^2$), exerted by this slice was computed from intraventricular pressure, $p$, left ventricular muscle volume, external left ventricular volume, and computations relating radial to circumferential stresses in curved walls (appendix, equation 6). The tension, $P$, is the "force" that would enter into the force-velocity relationship.

Average muscle fiber shortening velocity ($v$) per unit circumference of this muscle ring was estimated from ventricular ejection rate, $Q$, left ventricular muscle volume, and the instantaneous external left ventricular volume (appendix, equation 11). From the estimate of the nonlinear series elastic component spring-constant (last section of appendix) it was possible to calculate the shortening velocity of the contractile elements themselves (appendix, equation 19). It is this "velocity" that is entered into the force-velocity relationship. The instantaneous external heart size was calculated from instantaneous ejection rate, and instantaneous segment length, $S$, (appendix, equations 3a and 4e).

A recording of these three variables, $Q$, $S$, and $p$, along with certain ancillary information is shown in figure 5. Attention is directed to the middle three channels with marker arrows representing, from top down, instantaneous values of flow, volume, and intraventricular pressure. The middle or volume channel was obtained from the integral of the flow. The initial value of the integral for each beat, $V_o$, was obtained from the end diastolic point on the caliper tracing at the top of this figure which was calibrated in terms of volume as discussed. As indicated by the arrows, instantaneous values of flow and pressure at various selected instantaneous volumes could be measured from this tracing. Measurements such as these were done in six animal preparations.

**Results**

It was possible to obtain many different simultaneous pressure and flow values at selected instantaneous heart volumes with the above preparation. For example, in figure 6 representative data from two different experiments are shown. The velocity of shortening appears on the ordinate and muscle tension on the abscissa. Each of these plots represents the relationship of muscle tension to shortening velocity at a particular instan-

**FIGURE 3**

Diagram of animal preparation. EMF = electromagnetic flowmeter, $p_A =$ aortic pressure at point under flowmeter, $p_B =$ perfusion pressure to lower aorta and head, $p_V =$ left ventricular pressure, $R_4 =$ Starling Resistance for control of $p_V$, $R_{B} =$ Starling Resistance for constant perfusion pressure to lower aorta and head, $R_r =$ screw clamp used to regulate venous return to heart or pump reservoir.

**FIGURE 4**

Upper left: Heart diagram showing placement of recording instruments and the muscle ring oriented in the plane of the minor semiaxes to which all computations are referred: $R =$ minor semiaxis of heart chamber. $L =$ major semiaxis of heart chamber. $S =$ circumferential segment length included in caliper. $p =$ pressure. $Q =$ flow. Lower right: Schematic representation of muscle ring. $P =$ circumferential wall tension. $\theta =$ central angle subtended by $S$. $p =$ pressure. $Q =$ flow.
taneous external heart volume (LV Vol). For simplicity only one selected volume is shown for each dog. Each point on each plot represents the instantaneous tension and velocity of shortening at the moment that the volume of the contracting heart passed through the

**FIGURE 5**

Recording taken during an experiment showing the end diastolic volume point with the arrow in upper channel and instantaneous pressure, flow and volume points with the arrows in the middle channels. Body Press. = perfusion pressure of the body. Aortic Press. = aortic pressure. $\frac{dp}{dt} =$ time derivative of ventricular pressure.

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value selected for that particular volume isopleth. At a given instantaneous volume there is a reciprocal relationship between the tension developed and the velocity of shortening. Moderate scatter is present, that on the left of figure 6 being somewhat less and that on the right being somewhat greater than usually observed.

In the six animals studied the trends of these data were shown statistically not to vary significantly from straight lines. Therefore, the results from each animal were analyzed by calculating regression lines for each volume isopleth. The families of regression lines for each animal may be seen in the six graphs of figure 7. The length of the vertical bars drawn through the midportion of each line represents two times the standard error of estimates. Further statistical analysis showed that all lines have a significant negative slope and that in each family there is a significant progressive march of the grand means for each isopleth upward and to the right with increasing heart volume. Therefore, it can be concluded first that there is a reciprocal relationship between the tension in the muscle and the velocity with which it is shortening at any instantaneous volume, and second, for any instantaneous tension the velocity of shortening increases with increased heart volume. Finally, to suggest the possible presence of an underlying more classically shaped family of force-velocity relationships, hyperbolic curves have been lightly sketched through the regression lines for dog no. 1 in figure 7. It is of interest to compare this figure to the data of Sonnenblick shown in figure 2.

Discussion

Based on these consistent findings it is our belief that the mechanical behavior of contractile elements in heart muscle follows laws similar to those in skeletal muscle. However, the precision of these experiments is not high, as can be seen from the scatter of the data in figure 6, and from the length of the vertical bars shown in figure 7 representing twice the standard error of estimates for each curve. This lack of precision may be either instrumental or physiological. Although the measurements of pressure and flow may be considered reasonably accurate, the measurement of the third variable, heart volume (fiber length), was necessarily indirect and, therefore, subject to potential error. Although it was impossible to check the validity of the heart volume measurement directly, no obvious errors or inconsistencies were detect-

![Figure 6](http://circres.ahajournals.org/)

**Figure 6**

Plots of data from two experiments showing the relationship of muscle tension to the velocity of shortening at selected instantaneous left ventricular volumes (LV Vol).
ed. For example, the volume of the left ventricular muscle measured post-mortem by liquid displacement was less than the smallest heart volume measured by the caliper method during any experiment. Furthermore, the difference between the volume measured by the caliper method and the volume of the muscle measured post-mortem gave reasonable values for intraventricular cavity volume except for dog no. 4 where abnormally high volumes were calculated.

Several of the physiological factors possibly contributing to the scatter seen in figures 6 and 7 are worth mentioning. In the first place the data were analyzed around a model which was assumed to be uniquely characterized by only three variables, tension, \( F \), shortening velocity, \( v_c \), and length (heart volume) of the contractile units. It is not likely that the heart's action can be described so simply. For example it will be recalled from figure 5 that data were taken all through systole. The validity of this approach is based on several conditions:

1. It assumes that the "active state" begins instantly and simultaneously throughout the heart and, moreover, remains constant throughout systole.
2. It assumes that the contractile element reacts to stresses changing with time in the same manner as it does to constant stresses.
3. It assumes that when the load placed on the muscle is changed, internal readjustment of the ionic and other chemical milieu does not occur and does not cause compensatory changes in "contractility."

It is likely that many other variables enter in the description of myocardial behavior. However, the major fact remains that the
three most important variables describing the intrinsic behavior of the heart muscle appear to be tension, velocity of shortening, and fiber length.

The tension-velocity relationships in cardiac muscle appear to be much more dependent on muscle length than is the case for skeletal muscle. It might be more appropriate, therefore, to refer to the tension-velocity-length relationships of cardiac muscle to emphasize this dependence. Until other important variables can be defined more precisely, we would tentatively suggest that myocardial function might be usefully analyzed graphically as a three dimensional tension-velocity-length surface similar to that used in the study of pulmonary mechanics. Each heart beat would then be represented by a path traced in this surface. Since “length” (heart volume) is one dimension on this surface, it is clear that the initial fiber length or end diastolic volume is of significance only in that it determines the starting point of the heart-beat path. The path traced from this initial point throughout the ensuing contraction will be unique only for a unique pressure-time function. The pressure-time function in turn is determined by the hydraulic input impedance to the great vessels and the peripheral demands for mean flow. Furthermore, the area enclosed by the projection of the path on the tension-length plane of this three dimensional co-ordinate system represents the stroke work. Therefore, the power developed and work accomplished by the heart also must depend on the particular path traveled on the tension-velocity-length surface during the beat. It follows that stroke work is only indirectly, and not uniquely, related to end diastolic fiber length, but depends equally on the nature of the load imposed by the distal circulation.

Summary

An animal preparation was developed in which it was possible to vary the flow and pressure presented to the left ventricle independently while maintaining a constant pressure and flow to the rest of the animal. Instantaneous left ventricular ejection rate, ventricular pressure, and the separation of two points on the epicardial surface were continuously measured with suitable instruments. Using these measurements and making certain assumptions about the shape and sequence of contraction of the heart, it was possible to calculate instantaneous muscle tension, rate of muscle shortening, and length of an assumed circumferential ring of muscle in the mid portion of the ventricle. It was found that at any instantaneous muscle length there was a reciprocal relationship between muscle tension and the velocity of shortening, consistent with the “force-velocity” relationship of A. V. Hill. Regression lines describing the relationship between tension and rate of shortening at a given instantaneous muscle length were seen to become progressively elevated with increasing length.

The concept of the tension-velocity-length surface is suggested as a useful logic diagram that forms a fundamental framework within which it is possible to unify many other previous observations of myocardial function including Starling’s Law. Moreover this concept permits one to define such vague notions as “contractility” specifically in terms of the orientation, elevation, or depression of the tension-velocity-length surface.

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Appendix

COMPUTATIONAL DETAILS

It is the purpose of this appendix to define and derive the expressions used in the body of the paper that relate the shortening velocity, the fiber tension generated, and the instantaneous muscle fiber length in the myocardium to the measurable variables. As indicated in figure 4 these variables are the ventricular ejection rate (Q), intraventricular pressure (p), and an instantaneous muscle segment length (S). Derivation of these relationships requires precise knowledge of the distribution and orientation of muscle fibers.
the pattern of fiber excitation and the nature of the machinery coupling the forces of the contractile elements in these fibers to each other and ultimately to the ventricular cavity. The details of this information for the most part are unknown. Therefore, certain simplifying assumptions are necessary.

From the cineangiographic studies of Rushmer and Thal and from anatomical studies it appears that the approximate shape of the ventricular cavity is that of an ellipsoid with a major semiaxis from apex to base and equal minor semiaxes. This assumption has been made in the present paper. From the studies of Rushmer and Thal and Hawthorne the sequence of contraction may be viewed as an initial isovolumetric apex to base contraction with radial bulging followed by the ejection phase during which the apex to base dimension changes very little, i.e., ejection is accomplished mostly by decrease in radial dimensions. Thus, focusing on the ejection phase only, a model of the left ventricle may be considered approximately to be an ellipsoid with a fixed major semiaxis and a time varying set of equal minor semiaxes or radii (R). This view is also consistent with the anatomical presence of the essentially circumferential though gradually spiralling masses of deep constrictor muscles, i.e., the deep bulbospiral and sinospiral muscle components of the myocardium.

**Instantaneous Volume** (V)

The calculation of the instantaneous external volume of such an ellipsoidal model was accomplished as follows. A recording caliper was placed circumferentially in the plane of the minor semiaxes, as shown in figure 4, to record continuously the value of a segment length, S. The caliper feet have two pointed metal dowels that extend 1 cm into the ventricular wall to couple the feet with as many muscle layers as is practical. The instantaneous ejection rate, Q, was recorded with an electromagnetic flowmeter. The external volume, V, of the ellipsoidal left ventricle may be calculated from

\[ V = \frac{4}{3} \pi LR^2 \]  

where \( L \) is the “constant” major semiaxis and \( R \) is the time varying minor semiaxis of the heart.

Referring to figure 4 it can be seen that a relationship between \( R \) and the segment length, S, is given by

\[ R = \frac{S}{\theta} \]  

where \( \theta \) is the central angle subtended by S. If the heart contracts symmetrically in the plane of the semiaxes, then the angle \( \theta \) will remain constant. Substituting equation 2 into equation 1 gives

\[ V = K_v S^2 \]  

where

\[ K_v = \frac{4}{3} \pi L \]  

If \( K_v \) can be evaluated, then it is possible to express the external heart volume directly as a function of S. This can be done if the ejection rate, Q, is recorded simultaneously with S.

Recall that

\[ Q = -\frac{dV}{dt} \]  

and

\[ \frac{dV}{dS} = \frac{dV}{dt} \left( \frac{dS}{dt} = \frac{-Q}{ds} \right) \]

Also by differentiating equation 3a with respect to S

\[ \frac{dV}{dS} = 2 K_v S \]  

or

\[ K_v = \frac{1}{2S} \frac{dV}{dS} \]

Substituting equation 4b for \( \frac{dV}{dS} \) in equation 4d one finally obtains

\[ K_v = \frac{-Q}{2S \sqrt{\frac{dS}{dt}}} \]  

wherein Q, S, and \( \frac{dS}{dt} \) are parameters measured during systolic ejection from the simultaneous recording of the flowmeter and the caliper. The numerical evaluation of \( K_v \) from equation 4e was substituted into equation 3a.
to calibrate the caliper tracing in terms of volume as illustrated in the top channel of figure 5.

Since the caliper recording may have unknown errors or "artifacts" related to other variables, for example intraventricular pressure levels, the caliper recording is used only to estimate the end diastolic or initial volume of a particular contraction. The instantaneous volume thereafter (during the contraction) was evaluated from the continuous recording of the time integral of flow as explained in the text and as shown in figure 5. Since equation 4e uses systolic information to calibrate the caliper recording, an approximately constant error in the estimation of diastolic volume from the caliper tracing probably occurs. The magnitude of this error requires further study. Instantaneous muscle length could be calculated directly from instantaneous volume; however, no particular advantage is gained by expressing the data this way. Constant volume isopleths were used throughout this presentation to represent constant muscle lengths.

### Instantaneous Muscle Tension and Shortening Velocity

The thickness of the heart wall is about 20% to 30% of the end diastolic external radius of the heart. Since muscle is incompressible, the rate of strain or shortening velocity per unit circumference will vary across the wall, being greatest at the endocardial surface and least at the epicardial surface. If the tension exerted by a muscle fiber is a function of its rate of shortening, it follows that the tangential tension will also vary across the heart wall. Although the literature is not concordant on the details of muscle fiber orientation, there is general agreement that the statistical orientation of fibers is nearly circumferential in planes perpendicular to the major axis of the heart, except near the apex. It follows that without precise anatomical information it would be difficult to attempt any detailed analysis of the distribution of fiber shortening velocities and tensions. Consequently, a statistical approach was employed in this study. Moreover, only the behavior of an imaginary ring of muscle in the plane of the minor semi-axes will be considered as discussed in the body of the paper (fig. 4). Therefore, the results presented in this study represent the instantaneous spatial average of the shortening velocities across the heart wall and the instantaneous spatial average of the tension exerted in the circumferential direction of this muscle ring.

### Average Muscle Tension ($P$)

To the extent that the fibers are arranged circumferentially, only the wall curvature in the plane of the minor semiaxes need be considered. Thus, the curvature in the planes of the major semiaxis is not pertinent. The force, $F$, supported by the muscle ring in the tangential direction per unit slice width is given by

$$ F = p R_1 $$

where $p$ is the transmural pressure and $R_1$ is the radius of curvature of the cavity in the plane of the minor semiaxes. The spatial average tangential tension, $P$, will be the force divided by the area over which it is distributed.

$$ P = \frac{F}{R_2 \cdot R_1} = \frac{p R_1}{R_2 \cdot R_1} = \frac{p (V_2 - W)^{1/2}}{V_2^{1/2} - (V_2 - W)^{1/2}} \quad (6) $$

where $R_2$ and $R_1$ are the radial coordinates of the epicardial and endocardial surfaces respectively, $V_2$ is the external left ventricular volume, and $W$ is the left ventricular muscle volume. Equation 6 represents the tension, $P$, that corresponds to the “force” in Hill's force-velocity relationship.

The “velocity” in Hill's force-velocity relationship is the velocity at which the contractile element is shortening which we shall call $v_c$. Referring to figure 1, it can be seen that the velocity at which the contractile element is shortening will be the sum of the velocity at which the series elastic component is elongating, which we shall call $v_s$, plus the velocity at which the circumference of the ring is shortening which we shall call $v$. Therefore to estimate $v_c$ it is necessary to calculate the sum of $v$ and $v_s$. The derivations and computations involved in estimating $v$ and $v_s$ will be discussed now.
Average Muscle Shortening Velocity \( (\bar{v}) \)

The term "shortening velocity" is confusing when applied to an organ such as the heart wherein the muscle is continuous. However, since the term "shortening velocity" is firmly rooted in the language of muscle physiologists, it will be retained here with the understanding that it means the time rate of strain of the muscle fibers in the circumferential direction of the muscle ring shown in figure 4.

As mentioned earlier the muscle fibers near the endocardium are shortening more rapidly than those near the epicardium. The velocity of shortening per unit circumferential length (time rate of strain, \( v \)) at any distance, \( R \), from the center of the heart is given by

\[
v = \frac{1}{c} \frac{\partial V}{\partial t} = \frac{1}{2\pi R} \frac{\partial R}{\partial t} = \frac{1}{R} \frac{\partial R}{\partial t} \tag{7}\]

where \( c \) is the circumference passing through \( R \), \( R \) is the distance of the muscle fiber from the heart center, and \( \frac{\partial R}{\partial t} \) is the centripetal velocity of the fiber along the radius. Thus, \( v \) represents the time rate at which a unit length of the circumference through \( R \) is shrinking during ejection. The spatial average \( (\bar{v}) \) of this time rate of shrinking across the heart wall can be obtained by integrating equation 7 with respect to \( R \) from endocardial surface to epicardial surface and then dividing the integral by the wall thickness, \( R_2 - R_1 \). To perform this integration it is necessary to express \( \frac{\partial R}{\partial t} \) as a function of \( R \). This may be accomplished by differentiating equation 1 with respect to time and solving for \( \frac{\partial R}{\partial t} \)

\[
\frac{\partial R}{\partial t} = \frac{3}{8\pi LR} \frac{dV}{dt} \tag{8}\]

where \( R \) and \( \frac{\partial R}{\partial t} \) are the location and instantaneous centrifugal velocity of a fiber in the heart wall. This fiber is located on the surface of an infinitesimally thin-walled ellipsoid whose volume is contracting at an instantaneous rate of \( \frac{dV}{dt} \). The muscle wall is made up of the summation of an infinite number of such concentric shells. Since muscle is essentially incompressible, \( \frac{dV}{dt} \) will be the same for each shell, and, as a consequence, \( \frac{dV}{dt} \) may be considered as a constant when integrating with respect to \( R \).

Therefore substituting equation 8 into equation 7 and remembering that the ventricular ejection rate \( Q \) is equal to \( -\frac{dV}{dt} \), it may be seen that the spatial average of the time rate of circumferential muscle fiber shortening per unit circumference, \( \bar{v} \) will be

\[
\bar{v} = \frac{1}{R_2 - R_1} \int_{R_1}^{R_2} c dR = \frac{1}{R_2 - R_1} \int_{R_1}^{R_2} -1 \frac{R}{R} \frac{\partial R}{\partial t} dR = \frac{3Q}{8\pi L (R_2 - R_1)} \int_{R_1}^{R_2} \frac{R^2}{R_2 R_1} dR = \frac{3Q}{8\pi L R_2 R_1} \tag{9}\]

where \( Q \) and \( L \) are the ejection rate and major semiaxis of the ventricle respectively. The two radii \( R_2 \) and \( R_1 \) are the minor semi-axes of the ellipsoids forming the epicardial and endocardial surfaces of the heart respectively. These radii may be expressed in terms of external heart volume, \( V_2 \), and left ventricular muscle volume, \( W \), as follows:

\[
R_2 = V_2^{\frac{1}{3}} \left( \frac{3}{4\pi L} \right)^{\frac{1}{2}} \tag{10a}\]
\[
R_1 = (V_2 - W)^{\frac{1}{3}} \left( \frac{3}{4\pi L} \right)^{\frac{1}{2}} \tag{10b}\]
\[
R_2 R_1 = \left( \frac{3}{4\pi L} \right) (V_2^{\frac{1}{3}})(V_2 - W)^{\frac{1}{3}} \tag{10c}\]

Substituting equation 10c into 9, the spatial mean of the velocity of shortening per unit circumference \( (\bar{v}) \) becomes

\[
\bar{v} = \frac{Q}{2(V^{\frac{1}{3}})(V - W)^{\frac{1}{3}}} \tag{11}\]

where the subscripts have been omitted and \( V \) represents the external left ventricular volume.

Average Elongating Velocity of the Series Elastic Component \( (v_s) \)

The series elastic component of cat papillary muscle was studied by Abbott and Mom-
They measured the relationship between the muscle tension and the extension of the series elastic component of the muscle under a variety of conditions. A curvilinear relationship was obtained. It can be shown that their data may be fitted with an equation of the form

\[ P = k \left( \frac{\delta}{D} \right)^n \]  

(12a)

where \( P \) is the tension, \( k \) is an "elasticity modulus," \( \delta \) is the elongation, \( D \) is the resting length of the muscle fiber, and \( n \) is between 1.6 and 1.8.

In the present study this general expression will be assumed to describe the relationship between the tension and the elongation of the series elastic component, however, with the minor simplification that \( n = 2 \). That is,

\[ P = k \left( \frac{\delta}{D} \right)^2 \]  

(12b)

The rate of elongation \( \frac{\partial \delta}{\partial t} \) may be obtained by differentiating equation 12b with respect to time giving

\[ \frac{\partial P}{\partial t} = 2k \frac{\delta}{D^2} \frac{\delta}{\partial t} \]  

(13a)

Substituting \( \frac{P^{1/2}}{k^{1/2}} \) for \( \frac{\delta}{D} \) and solving for \( \frac{\partial \delta}{\partial t} \),

the time rate of the series elastance elongation, gives

\[ \frac{\partial \delta}{\partial t} = \frac{D}{2k^{1/2} P^{1/2}} \frac{\partial P}{\partial t} \]  

(13b)

If equation 13b is considered to apply to a circumferential muscle fiber in the muscle slice shown in figure 4, then \( D \) is equal to \( 2\pi R_0 \) where \( R_0 \) is the radial co-ordinate of this fiber at the resting volume of the heart. Moreover, equation 13b must be divided by the instantaneous circumference \( (2\pi R) \) in which the fiber is oriented to convert to time rate of elongation per unit circumference, \( v_s \). Thus:

\[ v_s = \frac{1}{2\pi R} \frac{\partial \delta}{\partial t} = \frac{1}{2\pi R} \frac{2\pi R_0}{2k^{1/2} P^{1/2}} \frac{\partial P}{\partial t} = \]  

\[ \frac{R_0}{2R} \frac{\partial P}{\partial t} = \left( \frac{V_0^{1/2}}{V_0 + \Delta V} \right)^{1/2} \]

\[ \frac{1}{2k^{1/2} P^{1/2}} \frac{\partial P}{\partial t} \]  

(14a)

where \( V_0 \) is the resting volume enclosed by the ellipsoidal surface containing the muscle fiber in question, and \( \Delta V \) is the difference between \( V_0 \) and the volume of the ellipsoid at the instant at which equation 14a applies. Note that the resting volume, \( V_0 \), is a function of \( R_0 \) which is the co-ordinate of the resting positions of the muscle fibers and, thus, is independent of time. In contrast the volume increment, \( \Delta V \), is only a function of time since as discussed previously the incompressibility of muscle tissue requires that all concentric ellipsoidal shells comprising the heart muscle must traverse identical increments of volume during any time period. Therefore, \( \Delta V \) will be identical from one ellipsoidal shell to the other. Consequently, \( \Delta V \) is independent of \( R_0 \) and is only a function of time.

To obtain the spatial average of the time rate of spring elongation, \( v_s \), equation 14a should be integrated with respect to \( R_0 \) across the wall thickness from the endocardial to the epicardial surface as was done in calculating the spatial average of the time rate of wall shortening, \( v \). In the case of equation 14a this cannot be done analytically in any simple way. In view of the preliminary nature of these investigations certain further simplifying assumptions will be used.

As discussed earlier we are interested in calculating \( v_s \) so that we may evaluate the shortening velocity of the contractile elements themselves, \( v_c \) (\textit{vide infra} equation 19). Except during periods of rapidly changing tension, \( v_s \) will tend to be small as can be seen from equation 14a. Consequently, approximations in estimating \( v_s \) will not be as serious as for example those in estimating \( v \) (equation 7). For the present, therefore, we shall use the following approximations: 1) replace \( \Delta V \) with its temporal average, 2) replace \( V_s \) with its spatial average so that

\[ \frac{V_s^{1/2}}{(V_0 + \Delta V)^{1/2}} = k_s \]  

(15)

and 3) use the instantaneous spatial average tension (equation 6) for \( P \). Then equation 14a becomes
where

\[ K = \frac{k_2}{2k^{\frac{1}{3}}} \]  

With these assumptions the spatial average rate of spring elongation per unit circumference is equal to equation 14b, i.e.,

\[ v_s = \bar{v}_s \]  

Substituting equation 6 for \( P \) in equation 14b and carrying out the indicated differentiation gives

\[ P_L = \frac{dP}{dt} = \frac{K}{p^{\frac{1}{2}}} \left[ \frac{V^{\frac{1}{2}} - (V - W)^{\frac{1}{2}}}{(V - W)^{\frac{1}{2}}} \right]^{\frac{1}{2}} \]

\[ \left[ 2V^{\frac{1}{2}}(V - W) - \frac{dp}{dt} - p Q \left( V^{\frac{1}{2}} - (V - W)^{\frac{1}{2}} \right) \right] \]

\[ 2V^{\frac{1}{2}}(V - W)^{\frac{1}{2}} \left[ V^{\frac{1}{2}} - (V - W)^{\frac{1}{2}} \right] \]

where \( \bar{v}_s \) is the spring elongating velocity per unit circumference, \( K \) is a series elasticity coefficient, \( V \) is the external volume of the left ventricle, \( W \) is the left ventricular muscle volume, \( p \) is the intraventricular pressure, and \( Q \) is the ventricular ejection rate.

**Average Shortening Velocity of Contractile Elements (\( v_s \))**

The average shortening velocity per unit circumference of the contractile elements, \( v_s \), will be the sum of the shortening velocity per unit circumference of the wall, \( \bar{v}_s \) (equation 11) and the elongating velocity per unit circumference of the series elastic component, \( \bar{v}_s \) (equation 18). Thus

\[ v_s = \bar{v}_s + \bar{v} \]

This equation represents the shortening velocity per unit circumference of the contractile elements themselves, and is the velocity that would correspond to the "velocity" in Hill's 3 force-velocity relationship.

Although the approximations chosen for the derivation of equation 18 were used as a matter of convenience for this preliminary study, it should be noted that except for the assumptions of an ellipsoidal ventricular shape, a fixed systolic major semiaxis, and a circumferential muscle fiber orientation, all of the equations through 14a are precise. Therefore, if the distribution of fiber tension, pressure, and shortening velocities of the contractile elements in the heart wall become of sufficient interest, it is possible to evaluate these distributions numerically if adequate boundary conditions are available and digital computer techniques are used. For example, the shortening velocity, \( v_s \), of any contractile element at a time, \( t \), located a distance, \( R \), from the center of the heart is given by

\[ v_s = v + v_s = (eq. 7) + (eq. 14a) = f(R,t,P) \]

If the exact relationship of \( P \) to \( v_s \) in the intact heart can be determined by studies such as those of Hill for skeletal muscle, it will be possible to express \( v_s \) also as another function of tension and dimensions:

\[ v_s = g(R,P) \]

Then with proper boundary conditions such as information about \( Q, p, V_0 \) etc. it should be possible to evaluate numerically equations 20a and 20b simultaneously with a digital computer to give tension, \( P \), as a function of time, \( t \), and position in the heart wall, \( R \), i.e.,

\[ P = h(R,t) \]

Then, substituting this solution back through equations 20b and 20a, the distribution of \( v_s \) and \( v_e \) as functions of time and fiber location may be obtained. Moreover, since the radial tension or pressure existing between the muscle fibers is rather simply related to the curvature of the fibers and to the tangential tension, \( P \), the distribution of pressure in the heart wall could also be computed from equation 20c. The distribution of pressure in the heart wall has been a subject of great interest particularly to those studying the factors determining coronary vascular blood flow resistance.

**Calculation of the Series Elasticity Coefficient (\( K \))**

The numerical evaluation of the series elasticity coefficient, \( K \), in equation 18 cannot be approached directly. However, an indirect estimation is possible. Consider equation 19. Just before the valves open for ejection, \( \bar{v} \) is approximately zero (except for the geometric rearrangements of fibers during isovolumetric contraction) and the mean short-
ening velocity of the contractile elements, $v_c$, is about equal to the mean elongating velocity of the series elastic component, $v_s$. At this instant one knows $p$, $\frac{dp}{dt}$, $V$ and $W$.

If one knew $Q$ and $\frac{dQ}{dt}$ which is equal to $v_c$, also at this instant, it would be possible to solve numerically equation 18 for $K$. Study of many ventricular ejection curves reveals that the peak ejection occurs within a few hundredths of a second after the valves open. During the brief period between valve opening and peak ejection rate the volume ($V$) and pressure ($p$) change relatively little. Therefore, it could be argued that if the tension-velocity-length relationships do in fact govern the behavior of the contractile elements, then the contractile elements will be shortening with approximately the same velocity just after the valves open as just before. Moreover, by the time of peak ejection the rate of change of pressure $\frac{dp}{dt}$ has greatly decreased. Consequently, the rate of elastic elongation is small and most of the shortening velocity of the wall represents that of the contractile elements themselves. With the foregoing reasoning it has been assumed that the peak ejection rate just after valve opening may be substituted into equation 11 to calculate the shortening velocity of the wall at this instant which is about equal to the shortening velocity of the contractile elements just prior to valve opening. As explained, these elements should be shortening with about the same velocity as that just prior to valve opening since neither volume (length) nor pressure (tension) have changed greatly. Consequently, peak ejection $Q$ and $v_c$ calculated from equation 11 ($v_c = v_s$) could be substituted into equation 18 along with $V$, $W$, $p$, and $\frac{dp}{dt}$ just before valve opening to calculate an approximate value for $K$. The constant $K$ will then represent the elasticity coefficient of a bar of muscle of unit cross sectional area and unit length in accordance with equation 14b. Although in theory the value of $K$ is determined only by the series elasticity, in actual practice this value is probably influenced by many factors most notable of which would be the geometric rearrangements of the heart muscle that occur during isovolumetric contraction. It has been found, however, that calculations of $K$ under wide varieties of pressure and flow conditions have shown moderately large random variation but no systematic trends.

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