

A THREE-DIMENSIONAL ANALYTICAL (RHEOLOGICAL) MODEL OF THE HUMAN LEFT VENTRICLE IN PASSIVE-ACTIVE STATES

NONTRAUMATIC DETERMINATION OF THE IN VIVO VALUES OF THE RHEOLOGICAL PARAMETERS

DHANJOO N. GHISTA, ALLAN J. BRADY, *and*
S. RADHAKRISHNAN

From the Biomedical Engineering Program, the Department of Mechanical and Aerospace Engineering, School of Engineering and Applied Science, Washington University, St. Louis, Missouri 63130; the Department of Physiology and Los Angeles County Cardiovascular Research Laboratory, University of California at Los Angeles, Center for the Health Sciences, Los Angeles, California 90024; and the Biomedical Engineering Division, Indian Institute of Technology, Madras 600036, India. Dr. Ghista's present address is the Biomedical Engineering Division, Indian Institute of Technology, Madras 600036, India.

ABSTRACT In this paper a three-dimensional continuum model of a mammalian left ventricle is formulated. The stresses in the model satisfy the conditions of zero stress on the outer (epicardial surface-representing) boundary. The strains of the model are obtained from the actual dynamic geometry measurements (obtained from cineangiocardiology). Since the left ventricular muscle is incompressible, the dilatational strain is zero and hence the (three-dimensional) deviatoric stress components are related to the corresponding strain components by Maxwell and Voigt rheological model analogues of one-dimensional systems; the parameters of the model are series and parallel elastic (SE, PE) elements and the contractile element (CE) (representing the sarcomere). The incorporation of the rheological features of the cardiac muscle into the three-dimensional constitutive equations (for the three-dimensional continuum model of the left ventricle) is a feature of this paper. A procedure is presented to determine the parameters of the constitutive equations (i.e., the SE, PE, and the parameters of the force-velocity relation for the CE) for the left ventricle of a subject from data on the dimensions and chamber pressure of the left ventricle. The values of these parameters characterize the rheology of the left ventricular muscle of the subject. In order to demonstrate clinical application of the analyses, in vivo data of the subjects' left ventricular pressure and dimensions are obtained, and the analyses are applied to the data to determine (for each subject) the values and characteristics of the elastic elements and CEs.

INTRODUCTION

In the last decade the contractile properties of cardiac muscle have received considerable attention, particularly preparations such as excised segments of tissue in which tension, length, and velocity parameters can reasonably be assumed to be represented in one dimension. While much has been learned about the elastic and force-velocity characteristics of isolated heart muscle, the relations between the elasticity of resting muscle, the elasticity of active muscle, and active force development, the anatomical arrangement of these elements has not been clearly established. Nevertheless, it has been fruitful to represent these mechanical properties in terms of the simplest possible analogues. Specifically, these analogues are (Fig. 1) (a) a maxwellian¹ arrangement in which the SE which appears upon excitation of the muscle does not bear resting tension and (b) a Voigt arrangement in which resting tension is borne by both PE and SE elements (arranged in series). The assumptions necessary to characterize these elastic elements and their arrangement with respect to the force generator or CE are that (a) in response to suitable mechanical perturbations, the force generator in the resting state is freely extensible, i.e., it exhibits no elastic or appreciable viscous properties; (b) the active force generator is inextensible and noncompressible in response to the applied perturbations. If the perturbations are limited to less than 0.5/muscle length and occur within 1 ms at room temperature, these assumptions are reasonably valid; and (c) the elastic elements do not bear compressive forces, i.e., they go slack at lengths below zero extension.

Experiments on papillary muscle with small perturbations do not always give a clear resolution of voigt or maxwellian form but usually suggest a more complex

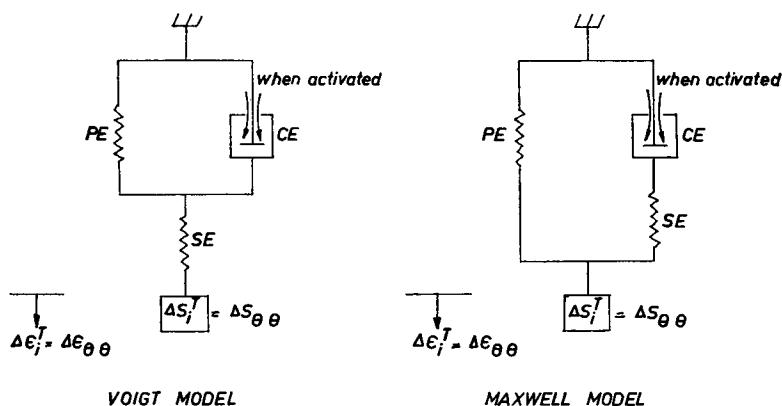


FIGURE 1 Left: Voigt representation of muscle mechanical properties. Right: Maxwell representation. PE, parallel elastic element; SE, series elastic element; CE, contractile element.

¹ The use of the terms Maxwell and Voigt must be qualified as used in this text. The implication here refers only to the arrangement of the components of the models. It is not assumed that the elastic elements are hookian nor that the CE is represented by a newtonian viscosity.

analogue. However, some muscles are fairly well represented in Voigt and others better in Maxwellian form. The purpose of this paper is to analyze a typical three-dimensional muscle element of the intact left ventricular muscle medium and formulate constitutive equations equivalent to those for a one-dimensional system as represented by either a Voigt or a Maxwell model. An analytical procedure is presented to predict the in vivo PE, SE, and CE properties associated with a three-dimensional element (using pressure-volume data from patients) in terms of stress, strain, rate of strain, and time which can in turn be readily determined from routine cine-angiocardiology data with associated cardiac catheterization. In this context a new formulation of rheological relations for a three-dimensional muscle system is established. Also, by assuming various functional relationships between force and velocity, the parameters of the force-velocity relations can be evaluated. The behavior of these parameters gives some insight into the degree to which heart muscle energetics parallel the well-established hyperbolic force-velocity characteristic of skeletal muscle.

NOTATIONS

| | |
|---|--|
| σ, ϵ | Total stress and strain tensors for the continuum model. |
| e | One-third the volume strain. |
| $S_{\theta\theta}; e_{\theta\theta}$ | Deviatric hoop stress and strain for a left ventricular element. |
| S_i^T, ϵ_i^T | Total stress and strain on a rheological model. |
| SE, PE, CE | Series elasticity, parallel elasticity, and contractile element of the rheological model. |
| K^{SE}, K^{PE} | Stiffnesses of the SE and PE elements. |
| K^{EQ} | Equivalent total elasticity of the relaxed myocardium; it is equal to $(K^{SE}K^{PE})/(K^{SE} + K^{PE})$. |
| $S_i^{SE}, S_i^{PE}, S_i^{CE}$ | Instantaneous stresses in the series. |
| $\epsilon_i^{SE}, \epsilon_i^{PE}, \epsilon_i^{CE}$ | Instantaneous strains in the SE. |
| $\epsilon_{i,sh}^{CE}$ | Instantaneous "shortening" (or negative) strain for CE. |
| $\dot{\epsilon}_i^{CE}$ | Instantaneous strain rate for the CE. |
| A, B, H | Left ventricular chamber dimension, as defined in Fig. 3. |
| $\sigma_{rr}, \sigma_{\theta\theta}, \sigma_{zz}$ | Stresses in the equatorial element of the analytical model. |
| K_i^{SE}, K_i^{PE} | Stiffnesses for (nonlinear) SE and PE elements. |
| K^{SE}, K^{PE} | Stiffnesses for (linear) SE and PE elements. |

BASIC EQUATIONS AND ASSUMPTIONS

The three-dimensional left ventricular model (Fig. 2) is similar to that of Ghista and Sandler (1969); the stresses in an element of this model satisfy the boundary conditions that the normal stress on the inner boundary (endocardial surface) equals the measured chamber pressure and that the stress on the outer boundary (epicardial surface) is zero. For the formulation of any elastic system, we need to satisfy (a) equilibrium equations, (b) constitutive equations, and (c) compatibility relations. These relations for the ventricular model are as follows.

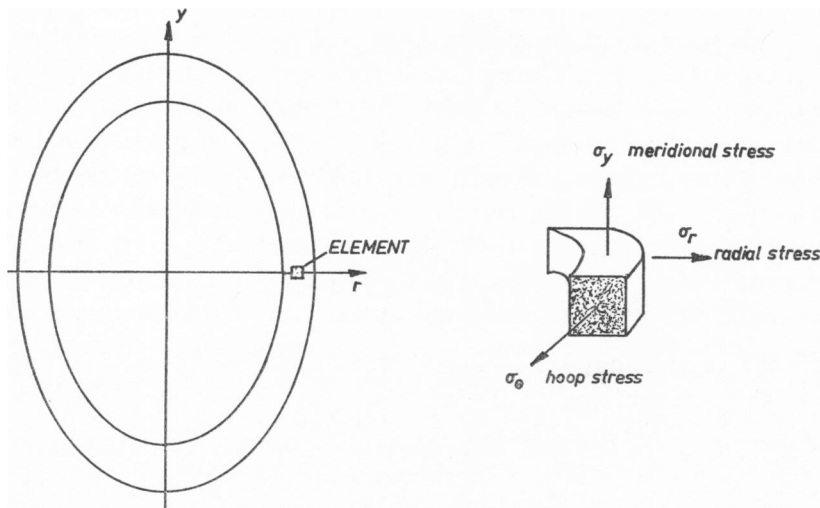


FIGURE 2 Right: Cross-sectional representation of left ventricle. Left: Enlargement of wall element to show stresses acting on the element within the wall.

Equilibrium Equations

The stresses in our left ventricular medium must satisfy equilibrium equations and the boundary conditions specifying zero normal and shear stresses on the outer left ventricular boundary, zero shear stresses on the inner boundary and normal stress at the inner boundary equal to the known chamber pressure. The expressions for the stresses (for an element of the wall [Fig. 2]), satisfying the equilibrium and boundary conditions, are given by Eq. A 3 of the Appendix. These stresses (obtained by employing the Ghista et al. model) are the instantaneous stresses, for an element of the medium, in terms of the instantaneous dimensions and chamber pressure of the left ventricle.

Constitutive Equations

Consider an element within the wall of the model (Fig. 2). For any general element we wish to relate the stress tensor (in cylindrical coordinates)

$$\sigma = \begin{bmatrix} \sigma_{rr} & \sigma_{ry} & \sigma_{r\theta} \\ \sigma_{yr} & \sigma_{yy} & \sigma_{y\theta} \\ \sigma_{\theta r} & \sigma_{\theta y} & \sigma_{\theta\theta} \end{bmatrix}, \quad (1)$$

to the corresponding strain tensor

$$\epsilon = \begin{bmatrix} \epsilon_{rr} & \epsilon_{ry} & \epsilon_{r\theta} \\ \epsilon_{yr} & \epsilon_{yy} & \epsilon_{y\theta} \\ \epsilon_{\theta r} & \epsilon_{\theta y} & \epsilon_{\theta\theta} \end{bmatrix}. \quad (2)$$

If the elements can be so oriented that shear stresses on it are zero then the stress

and strain tensors for the resulting principal elements are

$$\sigma = \begin{bmatrix} \sigma_{rr} & 0 & 0 \\ 0 & \sigma_{yy} & 0 \\ 0 & 0 & \sigma_{\theta\theta} \end{bmatrix}, \quad (3)$$

and

$$\epsilon = \begin{bmatrix} \epsilon_{rr} & 0 & 0 \\ 0 & \epsilon_{yy} & 0 \\ 0 & 0 & \epsilon_{\theta\theta} \end{bmatrix}. \quad (4)$$

The constitutive equations will consist of relations between these two equations by means of three-dimensional rheological models corresponding to the one-dimensional models of Voigt and Maxwell (Brady, 1967). To this end, we will split the stress and strain tensors into dilatational and deviatric components in order to characterize the stress and strain distributions in the model according to whether they produce volume change or a change in the shape of an element of the model. Analytically, (Eqs. 1 and 2) the *dilatational tensors* express the hydrostatic stress and strain parts of the total tensors without the shear components. The *deviatric tensors* express what is left over after the (uniformly distributed) hydrostatic stress and strain components are removed from the total tensors of Eqs. 1 and 2. Thus for the stress tensors in cylindrical coordinates, we have

$$\sigma = \begin{bmatrix} \sigma_{rr} & \sigma_{ry} & \sigma_{r\theta} \\ \sigma_{yr} & \sigma_{yy} & \sigma_{y\theta} \\ \sigma_{\theta r} & \sigma_{\theta y} & \sigma_{\theta\theta} \end{bmatrix} = \begin{bmatrix} S & 0 & 0 \\ 0 & S & 0 \\ 0 & 0 & S \end{bmatrix} \begin{bmatrix} S_{rr} & S_{ry} & S_{r\theta} \\ S_{yr} & S_{yy} & S_{y\theta} \\ S_{\theta r} & S_{\theta y} & S_{\theta\theta} \end{bmatrix}. \quad (5)$$

I II

The first term on the right (I) represents the hydrostatic system with no shear component (uniform pressure in all directions) and the second (II) represents the components of total stress which are left over. In other words, since we have selected

$$S = \frac{\sigma_{rr} + \sigma_{yy} + \sigma_{\theta\theta}}{3}, \quad (6)$$

the second term (II) indicates the deviation of stress from the average stress S and thus is the deviatric tensor.

Now II can be shown, by superposition, to be composed of five simple shear stress systems, i.e.

$$\text{II} = \begin{bmatrix} 0 & S_{ry} & 0 \\ S_{ry} & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} + \begin{bmatrix} 0 & 0 & 0 \\ 0 & 0 & S_{y\theta} \\ 0 & S_{y\theta} & 0 \end{bmatrix} + \begin{bmatrix} 0 & 0 & S_{r\theta} \\ 0 & 0 & 0 \\ S_{r\theta} & 0 & 0 \end{bmatrix} + \begin{bmatrix} S_{rr} & 0 & 0 \\ 0 & S_{rr} & 0 \\ 0 & 0 & 0 \end{bmatrix} + \begin{bmatrix} 0 & 0 & 0 \\ 0 & S_{\theta\theta} & 0 \\ 0 & 0 & S_{\theta\theta} \end{bmatrix}. \quad (7)$$

Where, from Eqs. 5 and 6

$$S_{rr} = \sigma_{rr} - \frac{\sigma_{rr} + \sigma_{yy} + \sigma_{\theta\theta}}{3} = \frac{2\sigma_{rr}}{3} - \frac{\sigma_{yy} + \sigma_{\theta\theta}}{3}, \quad (8)$$

$$S_{yy} = \sigma_{yy} - \frac{(\sigma_{rr} + \sigma_{yy} + \sigma_{\theta\theta})}{3} = \frac{2\sigma_{yy}}{3} - \frac{\sigma_{rr} + \sigma_{\theta\theta}}{3}, \quad (9)$$

$$S_{\theta\theta} = \sigma_{\theta\theta} - \frac{(\sigma_{rr} + \sigma_{yy} + \sigma_{\theta\theta})}{3} = \frac{2\sigma_{\theta\theta}}{3} - \frac{\sigma_{rr} + \sigma_{yy}}{3}. \quad (10)$$

Similarly the strain tensor can be written as

$$\epsilon = \begin{bmatrix} \epsilon_{rr} & \epsilon_{ry} & \epsilon_{r\theta} \\ \epsilon_{yr} & \epsilon_{yy} & \epsilon_{y\theta} \\ \epsilon_{\theta r} & \epsilon_{\theta y} & \epsilon_{\theta\theta} \end{bmatrix} = \begin{bmatrix} e & 0 & 0 \\ 0 & e & 0 \\ 0 & 0 & e \end{bmatrix} + \begin{bmatrix} e_{rr} & e_{ry} & e_{r\theta} \\ e_{yr} & e_{yy} & e_{y\theta} \\ e_{\theta r} & e_{\theta y} & e_{\theta\theta} \end{bmatrix}, \quad (11)$$

III IV

where $e = (\epsilon_{rr} + \epsilon_{yy} + \epsilon_{\theta\theta})^{1/3}$.

Note that III represents a deformation with volume change only (no shear), i.e. the dilatation or compression of the element is uniform in all directions, while IV represents deformation without any change in volume, i.e., a shape change only in which shear is present.

If the system is isotropic then the hydrostatic stress produces only dilatation and deformation with no shear. Thus term I relates to term III for the selected one-dimensional analogue as

$$[S] = [V, \text{ or } M]e, \quad (12)$$

where $[V, \text{ or } M]$ represents the functional relation between stress and strain and which is to be determined for the Voigt (V) or Maxwell (M) representation; Eq. 12 indicates that each stress element S of the term I is related to the corresponding strain element e of term III by means of the relation 12. This functional relationship is expressed by the characteristics of the PE, SE, and CE elements of the Voigt and Maxwell models. For the above relation to represent an in vivo element, S is obtained (by satisfying the equilibrium conditions) from the Ghista-Sandler model; e is obtained from the cineangiocardigraphic data on the dynamic geometry of the left ventricle.

Similarly, each of the five shear stress systems of Eq. 7 gives five analogous strain systems and the relations will be the same for all five pairs, thus,

[each and any component of stress system II, say $S_{\theta\theta}$]

$$= [M, V] [\text{each and any component of strain in system IV, say } e_{\theta\theta}]. \quad (13)$$

Finally, by assuming that the left ventricular muscle medium is incompressible we have the corresponding dilatational (causing volume change) strain tensor equal zero, i.e.,

$$e = 0 \quad (14)$$

Therefore, the general constitutive equation for the in vivo left ventricular muscle element consists of relations (the same for all components) between the components of the deviatoric stress and strain tensors, i.e., between the terms II and IV; thus, say

$$S_{\theta\theta} = [M \text{ or } V]e_{\theta\theta}. \quad (15)$$

Since our analytical model is employed quasistatically, we will relate instantaneous changes in stresses (such as say $\Delta S_{\theta\theta}$) to the corresponding changes in strains ($\Delta e_{\theta\theta}$) by means of rheological characteristics of the Maxwell or Voigt model. On an average, 40 readings of pressure and dimensions are taken during a cardiac cycle, i.e. a cardiac cycle is divided into 40 instants; $\Delta S_{\theta\theta}$ and $\Delta e_{\theta\theta}$ then represent changes in $S_{\theta\theta}$ and $e_{\theta\theta}$ from one instant to another. In this way, we can account for large deformations of the left ventricle (by stepwise adding incremental small strains of the left ventricle, formulated by employing infinitesimal strain theory) as well as for nonlinearity of SE and PE of our Maxwell and Voigt rheological models (by assumption of piecewise linearity over small incremental strains).

Compatibility Equations

In any analysis of a continuum model it must be assured that strains, due to the stress distribution, give continuous and single-valued displacements. In the formulation of our constitutive Eq. 15, the strains are the actual strains of the left ventricle (as obtained from cineangiographic data on the left ventricular geometry); in fact the strains are formulated in terms of the actual displacements (changes in instantaneous geometry) of the left ventricle. In deriving the constitutive relations for the left ventricular muscle medium, we will link the equilibrium and boundary condition satisfying stress distribution, given by Eq. A 1 and A 2 in the Appendix, to this monitored actual in vivo strain state. The model's stress distribution will automatically correspond to a compatible state of strain.

When we apply the above analysis to the actual heart, the stress-strain relations during contraction are not necessarily reflected during relaxation so that contraction and relaxation must be formulated and evaluated separately. In addition, the stress distribution at any instant may be a function of the rate of change of strain as well. These more complex but more real complications will be considered in a subsequent analysis.

MODEL ANALYSIS

In order to determine the nature of the function of V or M for our constitutive Eq. 15, we need to evaluate the deviatoric stress and strain components for a general element of our left ventricular model. Without loss of generality, an equatorial element can be chosen to represent the systems (Fig. 3).

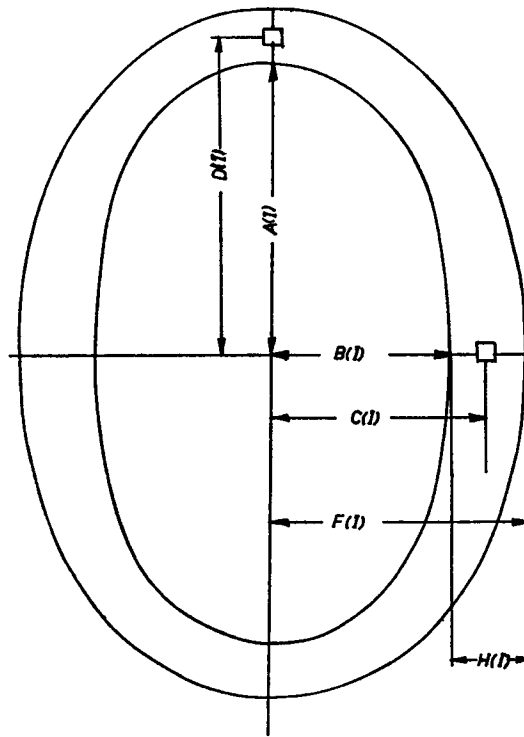


FIGURE 3 Cross section of ventricular wall showing dimensions measured from cineangiographs that are used to determine wall strains.

For the equatorial element, then, the stress tensor is

$$\sigma = \begin{bmatrix} \sigma_{rr} & & \\ & \sigma_{\theta\theta} & \\ & & \sigma_{\theta\theta} \end{bmatrix} = \begin{bmatrix} S & 0 & 0 \\ 0 & S & 0 \\ 0 & 0 & S \end{bmatrix} + \begin{bmatrix} S_{rr} & & \\ & S_{\theta\theta} & \\ & & S_{\theta\theta} \end{bmatrix}. \quad (16)$$

The magnitudes of the above components can be obtained, by means of Eqs. A 3 and A 2, in terms of the instantaneous left ventricular chamber pressure and dimensions; the magnitudes of the components of the changes in stresses ($\Delta\sigma$) at each frame reading corresponding to the associated changes in chamber pressure (Δp) can also be obtained from Eqs. A 3 and A 2; the changes of stress tensor can be written as follows:

$$\Delta\sigma = \begin{bmatrix} \Delta\sigma_{rr} & & \\ & \Delta\sigma_{\theta\theta} & \\ & & \Delta\sigma_{\theta\theta} \end{bmatrix} = \begin{bmatrix} \Delta S & 0 & 0 \\ 0 & \Delta S & 0 \\ 0 & 0 & \Delta S \end{bmatrix} = \begin{bmatrix} \Delta S_{rr} & & \\ & \Delta S_{\theta\theta} & \\ & & \Delta S_{\theta\theta} \end{bmatrix}. \quad (17)$$

The total strain tensor is from Eqs. 4 and 11

$$\epsilon = \begin{bmatrix} \epsilon_{rr} & & \\ & \epsilon_{\nu\nu} & \\ & & \epsilon_{\theta\theta} \end{bmatrix} = \begin{bmatrix} e_{rr} & & \\ & e_{\nu\nu} & \\ & & e_{\theta\theta} \end{bmatrix}. \quad (18)$$

Since the dilatational strain e is zero for the incompressible left ventricular medium. The change in strain tensor ϵ representing instantaneous changes in strains (or incremental strains) of the left ventricle is given by

$$\epsilon = \begin{bmatrix} \Delta\epsilon_{rr} & & \\ & \Delta\epsilon_{\nu\nu} & \\ & & \Delta\epsilon_{\theta\theta} \end{bmatrix} = \begin{bmatrix} \Delta e_{rr} & & \\ & \Delta e_{\nu\nu} & \\ & & \Delta e_{\theta\theta} \end{bmatrix}. \quad (19)$$

The above strain components are obtained in terms of the instantaneous left ventricular chamber instantaneous dimensions as follows (Ghista and Sandler, 1970; Ghista et al., 1970):

$$\Delta\epsilon_{\theta\theta} = \frac{C(I) - C(I-1)}{C(I-1)} = \Delta e_{rr}, \quad (20)$$

where $C(I)$ is an instantaneous dimension (illustrated in Fig. 3), and $C(I-1)$ corresponds to the dimension at a previous instant. $C(I)$ is obtained from cineangiographic data

$$\epsilon_{\nu\nu} = \frac{[C(I) - C(I-1)]C(I-1)}{[D(I-1)]^2} = \Delta e_{\nu\nu}, \quad (21)$$

where the dimension $D = A + (H/2)$ is illustrated in Fig. 3, and

$$\Delta\epsilon_{rr} = \frac{[F(I) - F(I-1)] - [D(I) - D(I-1)]}{H(I)} = \Delta e_{rr}, \quad (22)$$

where the dimensions $H(I)$ and $F(I)$ are illustrated in Fig. 3.

The cyclic variations of the dimensions of the left ventricular chamber are known. Hence the above strains (and the resulting total strain tensor ϵ of Eq. 18) can be calculated from ventricular shape changes at various stages of the cardiac cycle. In order to simplify the subsequent presentation it should be noted that since we have assumed isotropy within the ventricular wall the relations of S_{rr} to e_{rr} , $S_{\nu\nu}$ to $e_{\nu\nu}$, and $S_{\theta\theta}$ to $e_{\theta\theta}$ (and of the changes in stresses to the corresponding changes in strains) will all be the same. Hence, for simplicity the remainder of the analysis will be carried out in terms of $S_{\theta\theta}$ and $e_{\theta\theta}$.

It remains now to relate these total stresses ($S_{\theta\theta}$) and strains ($e_{\theta\theta}$) for the equatorial element of the left ventricle (by stepwise relating $S_{\theta\theta}$ and $e_{\theta\theta}$) by means of a rheological model similar to the one-dimensional Maxwell and Voigt rheological models.

In order to implement and to facilitate the incorporation of the one-dimensional

analogues into the three-dimensional system it has been decided to divide the cardiac cycle into different intervals during which certain combinations of SE, PE, and CE are operational. Thus stage I consists of the interval starting from initiation of filling (or loading) stage. During this interval both $S_{\theta\theta}$ and $e_{\theta\theta}$ increase and the CE is nonfunctional i.e., is slack or freely extensible. Hence the strain $e_{\theta\theta}$ response to loading $S_{\theta\theta}$ is only due to PE (for the Maxwell) or due to both SE and PE (for the Voigt model). This stage ends with the onset of contraction when $e_{\theta\theta}$ starts to decrease.

During stage II, $e_{\theta\theta}$ decreases while $S_{\theta\theta}$ continues to increase. During this stage all the three elements (namely SE, PE, and CE) are operational, i.e., the relation between $S_{\theta\theta}$ and $e_{\theta\theta}$ is due to the participation of SE, PE, and CE. This stage ends when $S_{\theta\theta}$ starts to decrease. At this stage, we assume that a phenomenon analogous to quick-load release takes place. Stage III hence consists of the time interval from end of stage II to the instants of first and second readings after the end of stage II. Study of the readings beyond stage III is not necessary since stage I, II, and III enable (as will be shown) us to calculate the characteristic values of SE, PE, and CE.

Maxwell Analogue

To provide rheological simulation of the left ventricular medium by our Maxwell model, we must have the corresponding components of the stress and strain tensor represent the dynamics of the Maxwell model. Thus we have S_i^T (the total stress at instant i of the rheological model) $\equiv S_{\theta\theta}$, in application to the three-dimensional system; and ϵ_i^T (the total strain at instant i of the rheological model) $= e_{\theta\theta}$. Also correspondingly,

$$\Delta S_i^T = \Delta S_{\theta\theta}; \quad \Delta e_i^T = \Delta e_{\theta\theta}.$$

Now, for the Maxwell analogue the governing equations for all the stages are

$$S_i^T = S_{\theta\theta} = S_i^{PE} + S_i^{SE} = S_i^{PE} + S_i^{CE};$$

$$\Delta S_i^T = \Delta S_{\theta\theta} = \Delta S_i^{PE} + \Delta S_i^{SE} = \Delta S_i^{PE} + \Delta S_i^{CE}, \quad (23)$$

$$\epsilon_i^T = \epsilon_{\theta\theta} = \epsilon_i^{PE} = \epsilon_i^{SE} + \epsilon_i^{CE}; \quad \Delta \epsilon_i^T = \Delta e_{\theta\theta} = \Delta \epsilon_i^{PE} = \Delta \epsilon_i^{SE} + \Delta \epsilon_i^{CE}, \quad (24)$$

$$K_i^{PE} = \Delta S_i^{PE} / \Delta \epsilon_i^{PE}; K_i^{SE} = \Delta S_i^{SE} / \Delta \epsilon_i^{SE}; S_i^{CE} = f_1^M(\epsilon_{i,sh}^{CE}) \quad (25)$$

Stage I. In the diastolic interval CE is considered to be freely extensible (this assumption is readily modified to include certain types of residual active contractile components but these will not be discussed here). We hence have

$$S^{CE} = 0 = S^{SE}, \quad (26)$$

and

$$\Delta_{\theta\theta} = S_i^{\text{PE}}. \quad (27)$$

Also, since CE is freely extensible, $\epsilon_i^{\text{SE}} = 0$, i.e., SE is not extended and

$$\epsilon_{\theta\theta} = \epsilon_i^{\text{PE}}. \quad (28)$$

Now since K_i^{PE} , the stiffness (or elasticity of the PE is defined as $\Delta S_i^{\text{PE}}/\Delta \epsilon_i^{\text{PE}}$, we have from Eqs. 27 and 28,

$$K_i^{\text{PE}} = \Delta S_{\theta\theta}/\Delta \epsilon_{\theta\theta} = d(S_{\theta\theta})/d(\epsilon_{\theta\theta}). \quad (29)$$

Since the change in stress $\Delta S_{\theta\theta}$ with respect to a change in strain $\Delta \epsilon_{\theta\theta}$ is known during the filling phase, the instantaneous stiffness K_i^{PE} can be evaluated. In general, the derivative $d(S_{\theta\theta})/d(\epsilon_{\theta\theta})$ will vary with S_i^{PE} . In fact later on, when our analyses are "clinically" employed by making use of clinical data, the nonlinear characteristics of PE are determined and demonstrated by means of S_i^{PE} vs. ϵ_i^{PE} and K_i^{PE} vs. S^{PE} plots. However, for convenience of computation of K^{SE} and the characteristics of CE, we designate

$$K^{\text{PE}} = [d(S_{\theta\theta})/d(\epsilon_{\theta\theta})] \text{ average}, \quad (30)$$

where the average is taken over the diastolic phase readings. Our general procedure, however, enables us to account for nonlinear SE and PE elements. From here onwards in this paper if we use K^{PE} and K^{SE} instead of K_i^{PE} and K_i^{SE} , respectively, it will imply that PE and SE are taken to be linear and hence their stiffnesses are constants at their average values.

Stage II. In this stage the total strain $\epsilon_{\theta\theta}$ begins to decrease but the total stress $S_{\theta\theta}$ continues to increase. We have

$$\Delta \epsilon_i^T = \Delta \epsilon_{\theta\theta} = \Delta \epsilon_i^{\text{PE}} = \Delta \epsilon_i^{\text{SE}} + \Delta \epsilon_i^{\text{CE}}. \quad (31)$$

Utilizing Eqs. 25, the instantaneous strain change is given by

$$\Delta \epsilon_{\theta\theta} = \Delta S_i^{\text{PE}}/K_i^{\text{PE}} = (\Delta S_i^{\text{SE}}/K_i^{\text{SE}}) - \Delta \epsilon_{i,sh}^{\text{CE}}, \quad (32)$$

where $\Delta \epsilon_{i,sh}^{\text{CE}}$ represents the shortening of CE and K^{SE} is SE elasticity (as yet undetermined).

Now, from the governing equations (Eqs. 23–25) we have

$$\Delta S_i^{\text{SE}} = \Delta S_i^{\text{CE}} = \Delta S_{\theta\theta} - K_i^{\text{PE}}(\Delta \epsilon_{\theta\theta}). \quad (33)$$

Substituting, Eq. 33 in Eq. 32, we get

$$\Delta e_{\theta\theta} = \frac{\Delta S_{\theta\theta} - K_i^{\text{PE}}(\Delta e_{\theta\theta})}{K_i^{\text{SE}}} - \epsilon_{i,sh}^{\text{CE}}, \quad (34)$$

therefore

$$\Delta \epsilon_{i,sh}^{\text{CE}} = (\Delta S_{\theta\theta}/K_i^{\text{SE}}) - (\Delta e_{\theta\theta})[(K_i^{\text{PE}}/K_i^{\text{SE}}) + 1]. \quad (35)$$

Hence the total instantaneous shortening strain of CE is

$$\epsilon_{i,sh}^{\text{CE}} = \sum_i \Delta \epsilon_{i,sh}^{\text{CE}} = \sum_i \{(\Delta S_{\theta\theta}/K_i^{\text{SE}}) - \Delta e_{\theta\theta}[(K_i^{\text{PE}}/K_i^{\text{SE}}) + 1]\}, \quad (36)$$

where the summation indicated is from beginning of stage II to instant i . Strictly speaking, we would sum from beginning of stage I to instant i ; however, since ϵ_i^{CE} is zero during diastolic stage I, its contribution during stage I is zero. Again if SE and PE are considered linear, we can take the \sum sign inside the brace and put $S_{\theta\theta}$ and $e_{\theta\theta}$ for $\sum_i \Delta S_{\theta\theta}$ and $\sum_i \Delta e_{\theta\theta}$, respectively, the summation being from beginning of stage II to instant i during stage II. Thus, for linear element, we have

$$\epsilon_{i,sh}^{\text{CE}} = (S_{\theta\theta}/K^{\text{SE}}) - e_{\theta\theta}[(K^{\text{PE}}/K^{\text{SE}}) + 1]. \quad (37)$$

It is to be noted $e_{\theta\theta}$ will be negative.

Stage III. At this stage, when the value of $S_{\theta\theta}$ drops (corresponding to a quick-load release), the evaluation of K^{SE} depends on our ability to determine a change in $S_{\theta\theta}$ with respect to a change in $e_{\theta\theta}$ at constant CE length. This can be done if it is assumed that in the intervals between measurements of the ventricular dimensions at this stage (during a couple of intervals after the instant at which $S_{\theta\theta}$ starts to decrease) the change in strain $\Delta e_{\theta\theta}$ ($\Delta \epsilon_i^T$) is largely due to excursions of SE and PE; for a sudden jump in stress and hence in stress rate, the CE cannot develop an instantaneous strain, and hence during the prevalent quick-unloading period it is not able to respond. This assumption can have serious limitations and must be checked carefully with data. We then have the situation wherein, during the interval consisting of one or two readings after the start of stage III, the response of the model is essentially due to that of two parallel elastic (SE and PE) elements; hence, the change in stress ($\Delta S_{\theta\theta}$) vs. the change in strain ($\Delta e_{\theta\theta}$) relationship is governed by the stiffnesses of SE and PE acting in parallel. From the governing equations (by putting $\epsilon_i^{\text{CE}} = 0$) we get at an instant i immediately after the start of stage III,

$$K_i^{\text{SE}} + K_i^{\text{PE}} = \Delta S_i^T / \Delta \epsilon_i^T = \Delta S_{\theta\theta} / \Delta e_{\theta\theta}, \quad (38)$$

where i equals the first or at most second reading after the initiation of stage III (or end of stage II), so that $\Delta S_{\theta\theta}$ and $\Delta e_{\theta\theta}$ represent changes in stress and strain during that interval. Now since K_i^{PE} is obtained explicitly by means of Eq. 29 or 30,

we can also obtain K_i^{SE} explicitly from Eq. 38 by substituting the value of K_i^{PE} from Eq. 29 or 30 into Eq. 38.

Force-Velocity Determination. Having determined K^{SE} and K^{PE} , we can now determine the shortening strain rate of CE by differentiating the expression for $\dot{\epsilon}_{i,sh}^{\text{CE}}$ given by Eq. 36. If, however, we assume linear SE and PE elements (for sake of convenience), we get the following expression for $\dot{\epsilon}_{i,sh}$ by differentiating Eq. 37.

$$\dot{\epsilon}_{i,sh}^{\text{CE}} = (1/K^{\text{SE}})\dot{S}_{\theta\theta} - [(K^{\text{PE}}/K^{\text{SE}}) + 1](\dot{e}_{\theta\theta}), \quad (39)$$

where the dot indicates the time derivative of the variable, and $S_{\theta\theta}$, $e_{\theta\theta}$ are formulated by taking summations of their respective instantaneous increments during stage II. For simplicity let $\dot{\epsilon}_{i,sh}^{\text{CE}} = V_i^{\text{CE}}$, i.e., the shortening velocity of the CE at instant i . Now since all the terms on the right-hand side of Eq. 39 are known, we know the variation of $\dot{\epsilon}_{i,sh}^{\text{CE}}$ or V_i^{CE} , the instantaneous shortening velocity of CE. Also, from Eq. 23, we have the expression for the total instantaneous stress in CE given by

$$S_i^{\text{CE}} = S_{\theta\theta} - S_i^{\text{PE}} = S_{\theta\theta} - K^{\text{PE}}e_{\theta\theta}, \quad (40)$$

where, again, $S_{\theta\theta}$ and $e_{\theta\theta}$ represent summations of their respective increments during stage II. Here, too, since all the terms on the right-hand side of the equation are known, we know the variation of S_i^{CE} . A plot of S_i^{CE} vs. V_i^{CE} then can be made and will characterize the functional (as well as pathological) behavior of CE.

Further, a functional relationship can be assumed between the terms S_i^{CE} and V_i^{CE} such as

$$S_i^{\text{CE}} = f_1^{\text{M}}(V_i^{\text{CE}}). \quad (41)$$

Then substituting for S_i^{CE} and V_i^{CE} from Eqs. 39 and 40, we get the relationship

$$S_{\theta\theta} - K^{\text{PE}}(e_{\theta\theta}) = f_1^{\text{M}}\{(\dot{S}_{\theta\theta}/K^{\text{SE}}) - [(K^{\text{PE}}/K^{\text{SE}}) + 1](\dot{e}_{\theta\theta})\}, \quad (42)$$

wherein the left-hand term of the equation as well as the argument of the right-hand side of the equation are known. If now the parametric form of the function f_1^{M} is chosen, then the parameters of the function f_1^{M} can be determined and employed diagnostically. Similarly a parametric relation can be derived if we choose $V_i = f_2^{\text{M}}(S_{\theta\theta}^{\text{CE}})$.

Voigt Analogue

In the Voigt analogue the SE bears resting tension as well as total tension when the muscle contracts and the strains in PE and CE are identical. Thus the governing equations are

$$S_i^T = S_{\theta\theta} = S_i^{SE} = S_i^{PE} + S_i^{CE};$$

$$S_i^T = \Delta S_{\theta\theta} = \Delta S_i^{SE} = \Delta S_i^{PE} + \Delta S_i^{CE}, \quad (43)$$

and

$$\left. \begin{aligned} \epsilon_i^T &= e_{\theta\theta} = \epsilon_i^{PE} + \epsilon_i^{SE} = \epsilon_i^{CE} + \epsilon_i^{SE} \\ \Delta \epsilon_i^T &= \Delta e_{\theta\theta} = \Delta \epsilon_i^{PE} + \Delta \epsilon_i^{SE} = \Delta \epsilon_i^{CE} + \Delta \epsilon_i^{SE} \end{aligned} \right\}. \quad (44)$$

Stage I. At this stage $S_i^{CE} = 0$ since by definition

$$\Delta S_i^{SE} = K_i^{SE} \Delta \epsilon_i^{SE} \text{ and } S_i^{PE} = K_i^{PE} \Delta \epsilon_i^{PE}, \quad (45)$$

we get from Eqs. 43–45

$$\Delta \epsilon_i^T = \Delta e_{\theta\theta} = (\Delta S_{\theta\theta} / K_i^{SE}) + (\Delta S_{\theta\theta} / K_i^{SE}),$$

or

$$(\Delta e_{\theta\theta} / \Delta S_{\theta\theta}) = (1 / K_i^{SE}) + (1 / K_i^{PE}) = 1 / K_i^{EQ}. \quad (46)$$

If we assume linear SE and PE elements,

$$\frac{d(e_{\theta\theta})}{d(S_{\theta\theta})} \text{ averaged over filling phase I} = \frac{1}{K^{SE}} + \frac{1}{K^{PE}} = \frac{1}{K^{EQ}}. \quad (47)$$

Stage II. At the beginning of phase II, $e_{\theta\theta}$ is a maximum and then starts decreasing. Proceeding in the same manner as for the Maxwell analogue, we obtain the following relation, analogous to Eqs. 41 and 42 for the Maxwell analogue:

$$S_i^{CE} = f_1^V (V_i^{CE}) \quad (48)$$

$$\left[\left(1 + \frac{K^{PE}}{K^{SE}} \right) S_{\theta\theta} - K^{PE} (e_{\theta\theta}) \right] = f_1^V \left[\frac{\dot{S}_{\theta\theta}}{K^{SE}} - \dot{e}_{\theta\theta} \right], \quad (49)$$

wherein $S_{\theta\theta} = \sum \Delta S_{\theta\theta}$ during stage II,

Stage III. Since this stage constitutes a quick-load release, CE does not change dimensions appreciably between successive measured cineangiographic frames. As CE remains locked as it was (thereby also preventing PE from responding), during the time interval between start of stage III and one or two successive readings (constituting quick-release period), the response of the model is essentially due to that of a single SE element; hence, during this period, the change in stress ($\Delta S_{\theta\theta}$) vs. change in strain ($\Delta e_{\theta\theta}$) relationship is solely governed by the stiffness of the SE element. Thus,

$$K_i^{SE} = \Delta S_i^T / \Delta \epsilon_i^T = \Delta S_{\theta\theta} / \Delta e_{\theta\theta}, \quad (50)$$

where i equals the first or almost second reading after the start of stage III, so that ΔS_{00} , Δe_{00} represent changes in stress and strain during that interval. Now that K_i^{SE} is determined, we can determine K^{PE} explicitly with the help of Eq. 47.

Force-Velocity Relationship. Now that both K^{SE} and K^{PE} are known, S_i^{CE} and V_i^{CE} can be determined from their corresponding expressions in Eqs. 48 and 49. Hence, S_i^{CE} vs. V_i^{CE} can be plotted to characterize CE; further, the parameters of f_1^V can be determined and employed diagnostically.

DISCUSSION

For the Voigt analogue, a value of K_i^{SE} is obtained from the interval succeeding quick-load release representing stage III. Just before this stage, SE is fully extended and the stress in it (equal to S_{00}) is a maximum. If SE is linear, then K^{SE} represents its elasticity. When the value of K^{SE} is substituted in Eq. 47 for the various instants during stage I, an estimate of possible nonlinear variation of K_i^{PE} (increase of K_i^{PE} with S_i^{CE} , as is typical of biological materials) can be obtained. However, if SE has a nonlinear rheology, the value of K_i^{SE} at stage III represents its hardened (or post-transition) elasticity; in this case, when this value of K_i^{SE} is substituted in Eqs. 47 representative values of K_i^{PE} will not be obtained. In such a case it is preferable to lump both SE and PE together having an equivalent stiffness $K_i^{\text{RQ}} = (K_i^{\text{SE}} K_i^{\text{PE}}) / (K_i^{\text{SE}} + K_i^{\text{PE}})$, representing the total elasticity of the myocardium in relaxed state. In fact, when in the next section we demonstrate the usage of our analyses with on-line subject data on the left ventricular dimensions and pressure, we will determine K_i^{RQ} for the Voigt representation of the left ventricular media and plot it against S_i^{SE} or S_i^{PE} . This parameter can be employed diagnostically; it is expected that the presence of scar tissue from healed infarction will influence the value of K^{RQ} .

For the Maxwell analogue, a nonlinear variation of K_i^{PE} is explicitly obtained from Eq. 29. Now when K_i^{SE} is to be obtained from Eq. 38 at stage III, it is to be noted that at this stage SE is fully extended whereas PE is not. Hence, in the Eq. 38 the initial elasticity value of K_i^{PE} should be substituted, whereupon the derived value of K_i^{SE} will represent hardened (or maximal) elasticity. Summarizing, the Voigt analogue representation provides: (a) the nonlinear variation of K_i^{RQ} , the total elasticity of the relaxed myocardium; (b) the maximal value of K_i^{SE} or the SE elasticity in hardened state; and (c) characterization of CE with the help of S_i^{CE} vs. ϵ_i^{CE} plot. On the other hand, the Maxwell analogue provides: (a) the nonlinear variation of K_i^{PE} ; (b) the maximal value of K_i^{SE} or the SE elasticity in hardened state; and (c) characterization of CE with the help of S_i^{CE} vs. ϵ_i^{CE} plot.

One of the most critical assumptions in this derivation is that the cineangiographic measurements of volume changes during the early part of phase III represent strains associated only with SE and/or PE. If, during this period, an appreciable strain change does occur in the CE during successive measurements of ventricular volume or in some cardiac pathology such as aortic regurgitation then the estimation

of K^{SE} would be incorrect. In this case it would be necessary to calculate K^{SE} and the parameters of the force-velocity relation from successive measurements of ventricular strain as follows.

For the Maxwell model, during phase I, we have the relation given by Eq. 30 for K^{PE} . Now during phase II, we have, starting with reading II + 1, namely the first reading after stage II,

$$\begin{aligned} & \{S_{\theta\theta}[\text{at II} + 2] - K^{PE} e_{\theta\theta}[\text{at II} + 2]\} \\ &= f_1^M \frac{1}{K^{SE}} \frac{\{S_{\theta\theta}[\text{at II} + 2] - S_{\theta\theta}[\text{at II} + 1]\}}{\Delta t} \\ &\quad - \left(1 + \frac{K^{PE}}{K^{SE}}\right) \frac{\{e_{\theta\theta}[\text{at II} + 2] - e_{\theta\theta}[\text{at II} + 1]\}}{\Delta t}, \end{aligned}$$

where the reading [II + 2] implies the second reading after start of stage II, and Δt denotes the time interval between two consecutive cineangiocardigraphic readings. A similar equation can be written for each incremental measurement or reading for as many equations as are necessary to define the total number of CE parameters (in addition to the constant K^{SE}) that are assumed to be contained in the "force-velocity" relation of Eq. 42. The parameters and the constant K^{SE} would be determined from these equations by simultaneous solution with a computer. The same procedure could be used for the remaining phases of the cardiac cycle. A similar approach would also be used for the Voigt analogue except that one more equation would be used since K^{PE} is not determined explicitly as in the case of Maxwell analogue. If the determined parameters are constants for the whole cardiac cycle then the interpretations are simplified; if not then their time dependence would be defined by their instantaneous values. The significance of this analysis is that it begins to establish a more accurate relation for interpretation of cardiac pressure-volume relations in terms of known rheology of the muscle medium.

CLINICAL APPLICATION OF THE ANALYSIS AND INTERPRETATION OF THE RESULTS

The analyses, presented herewith, are now applied to clinically obtained data consisting of the subjects' left ventricular (instant-by-instant) dimensions (obtained by cineangiocardigraphy) and chamber pressure (obtained by cardiac catheterization); in other words, for each subject, with the left ventricular wall, stresses and strains are determined. These are the data employed for the analyses. In applying the analysis to each subject's data, the left ventricular medium is represented (in turns) as a Voigt and a Maxwell model; the values of the elastic element stiffnesses (of K_i^{PQ} for the Voigt model and K_i^{PE} for the Maxwell model) and the stress-strain rate characteristics of the CE are calculated.

The results for three subjects are shown in Figs. 4-6. For each subject, the moni-

| SUBJECT | DISEASE | LVP (mmHg) | L/W at E/D (cm) | L/W at ME (cm) | H=S/D (cm) | K_i^{PE} or K_i^{EQ} $10^5 \times \text{dyn/cm}^2$ | $dS_i^{CE}/d\epsilon_{ish}^{CE}$ $10^3 \times \text{dyn/cm}^2$ per s |
|---------|------------|---------------|--------------------|-------------------|---------------|---|--|
| HG | MS, MI, AI | 117/114 | 13.1/6.4 | 12.26/5.36 | 1.3/1.0 | 4.0/11.0 | 0.25/0.625 |

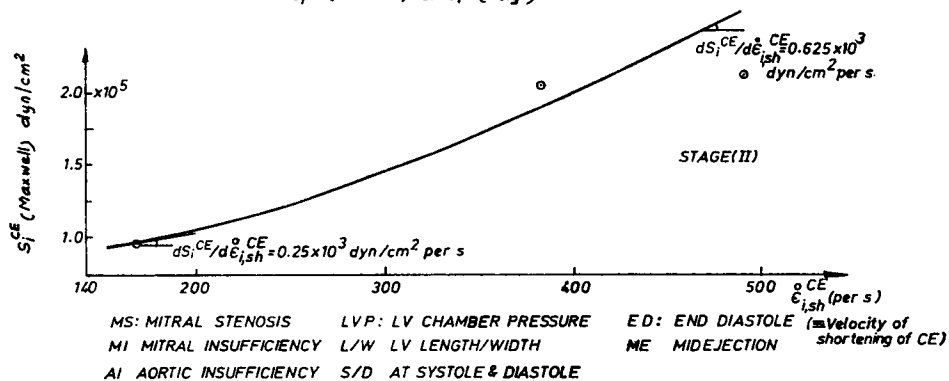
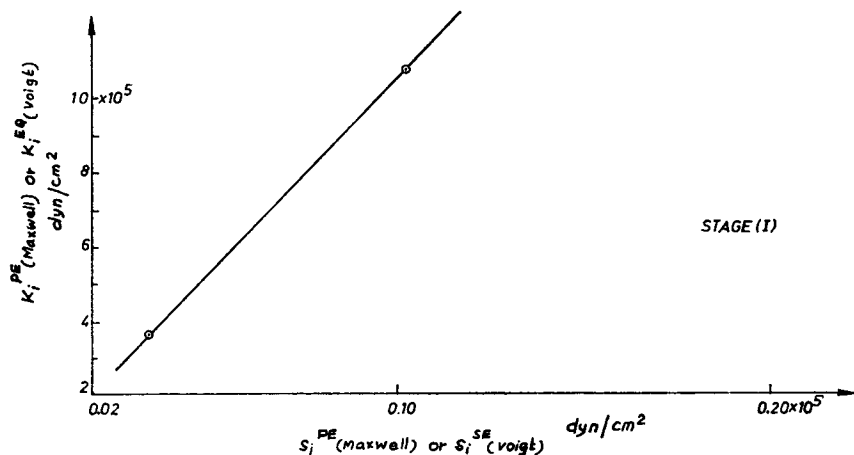
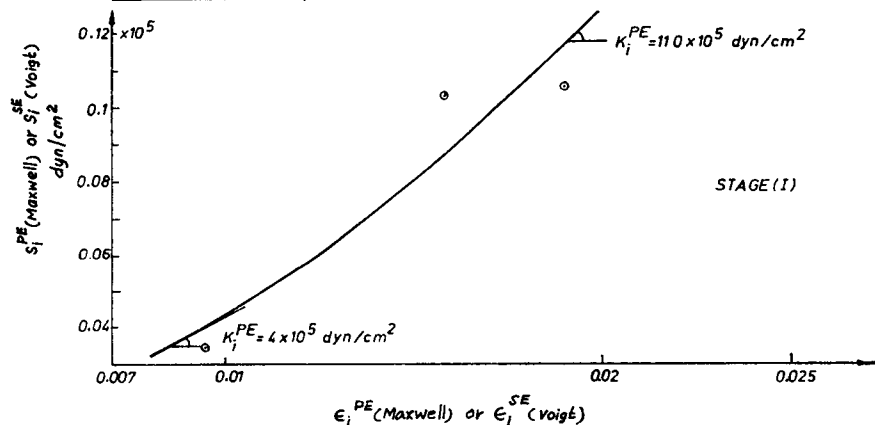


FIGURE 4 Clinical application of the model: determination of left ventricular (LV) chamber muscle's rheological characteristics of subject HG.

| SUBJECT | DISEASE | LVP (mmHg) | L/W at E/D (cm) | L/W at ME (cm) | H = S/D (cm) | K_i^{PE} or K_i^{EQ} $10^5 \times \text{dyn/cm}^2$ | $dS_i^{CE}/d\dot{\epsilon}_{ish}^{CE}$ $10^3 \times \text{dyn/cm}^2 \text{ per s}$ |
|---------|---------|---------------|--------------------|-------------------|-----------------|---|---|
| ML | IMH | 109/12 | 10.5/8.1 | 9.90/7.78 | 0.93/0.76 | 2.0/6.0 | 0.7/7 |

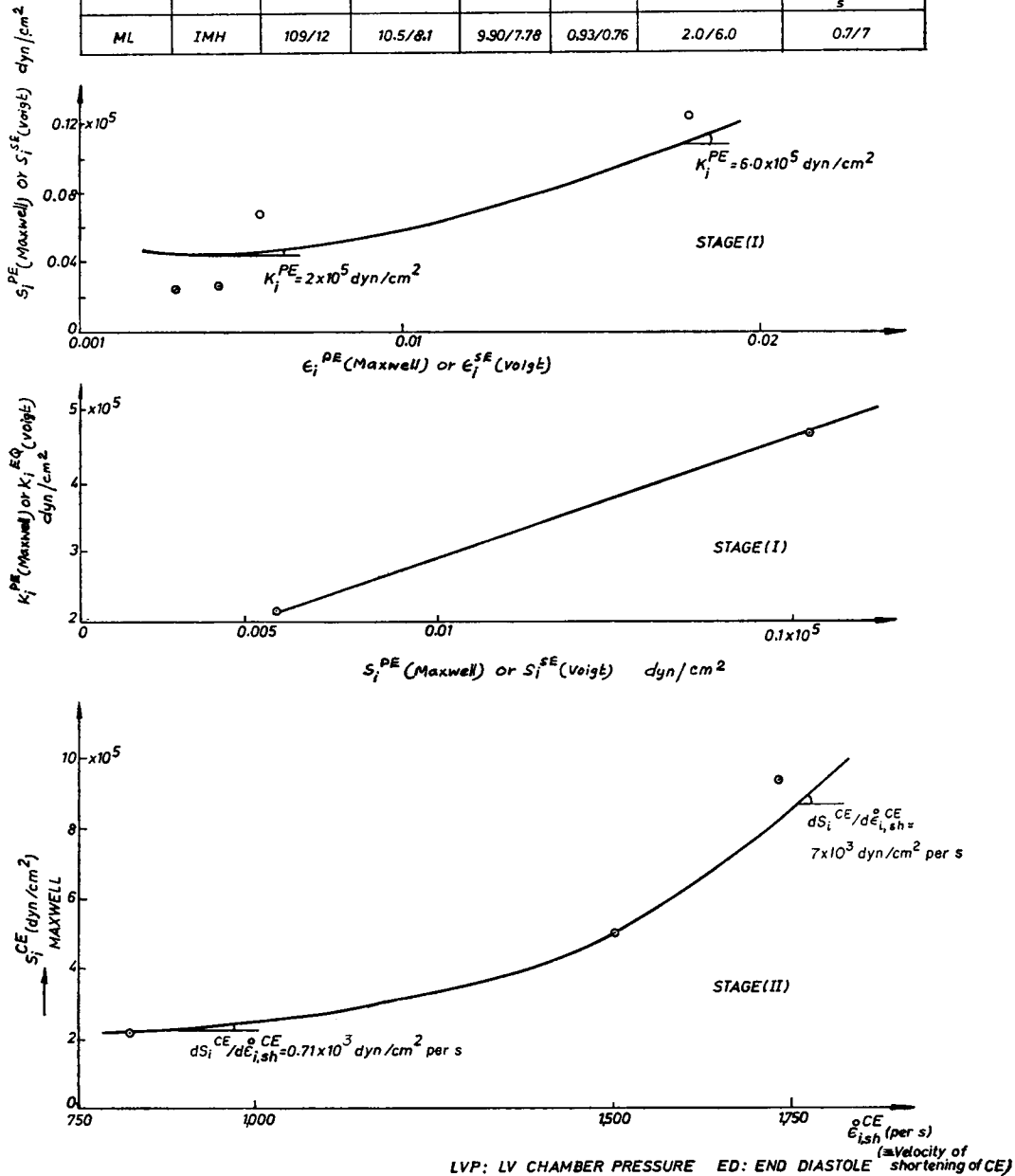


FIGURE 5 Clinical history and calculated left ventricular (LV) muscle's rheological properties of subject ML.

| SUBJECT | DISEASE | LVP (mmHg) | L/W at E/D (cm) | L/W at ME (cm) | H=S/D (cm) | K_i^{PE} or K_i^{EQ} $10^5 \times \text{dyn/cm}^2$ | $dS_i^{CE}/d\dot{e}_{ish}^{CE}$ $10^3 \times \text{dyn/cm}^2 \text{ per s}$ |
|---------|----------|---------------|--------------------|-------------------|---------------|---|--|
| JKK | MS,MI,AI | 145/8 | 10.3/6.2 | 9.42/4.14 | 1.2/0.87 | 7.0 | 0.83 |

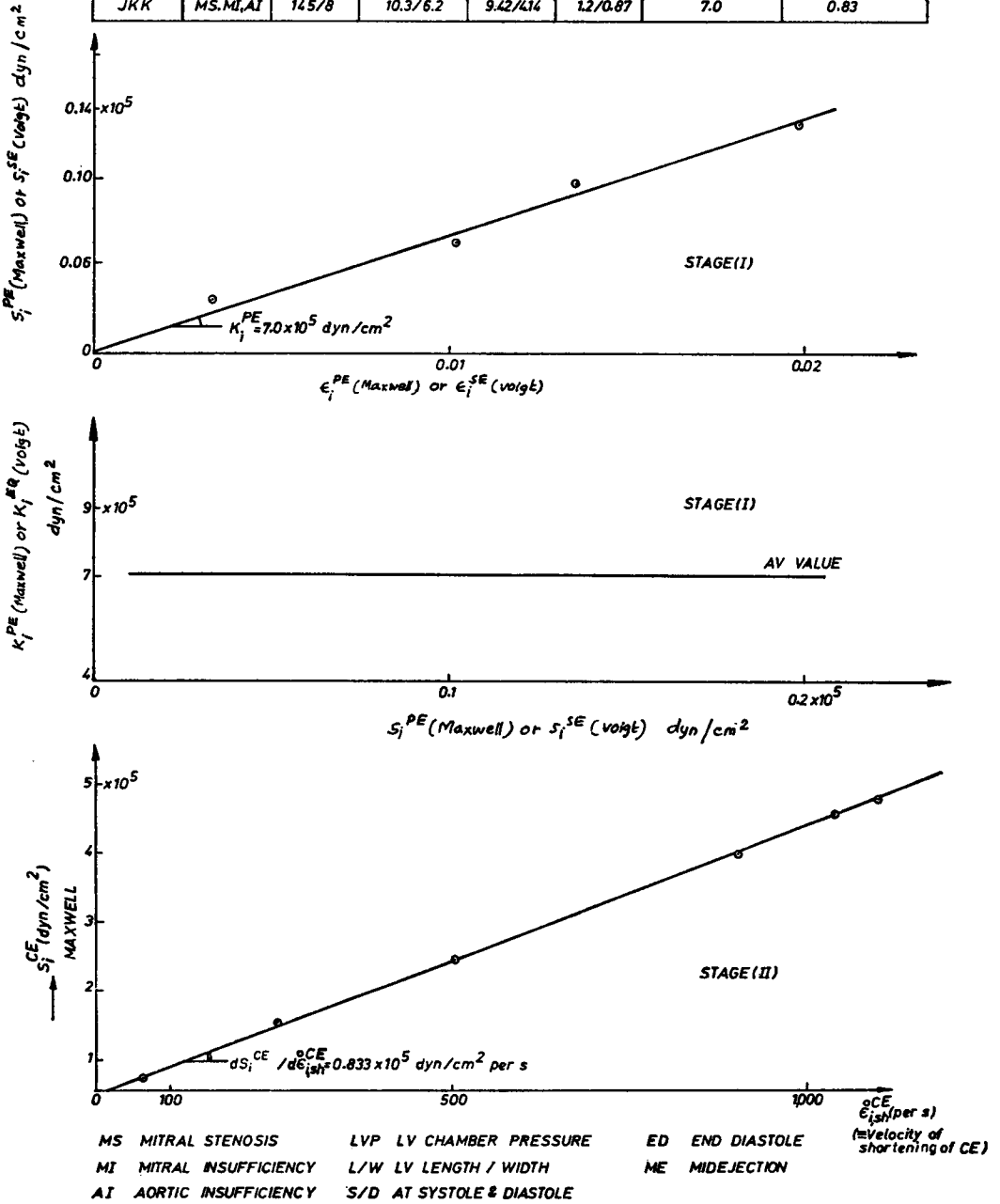


FIGURE 6 Clinical history and calculated left ventricular (LV) muscle's rheological properties of subject JKK.

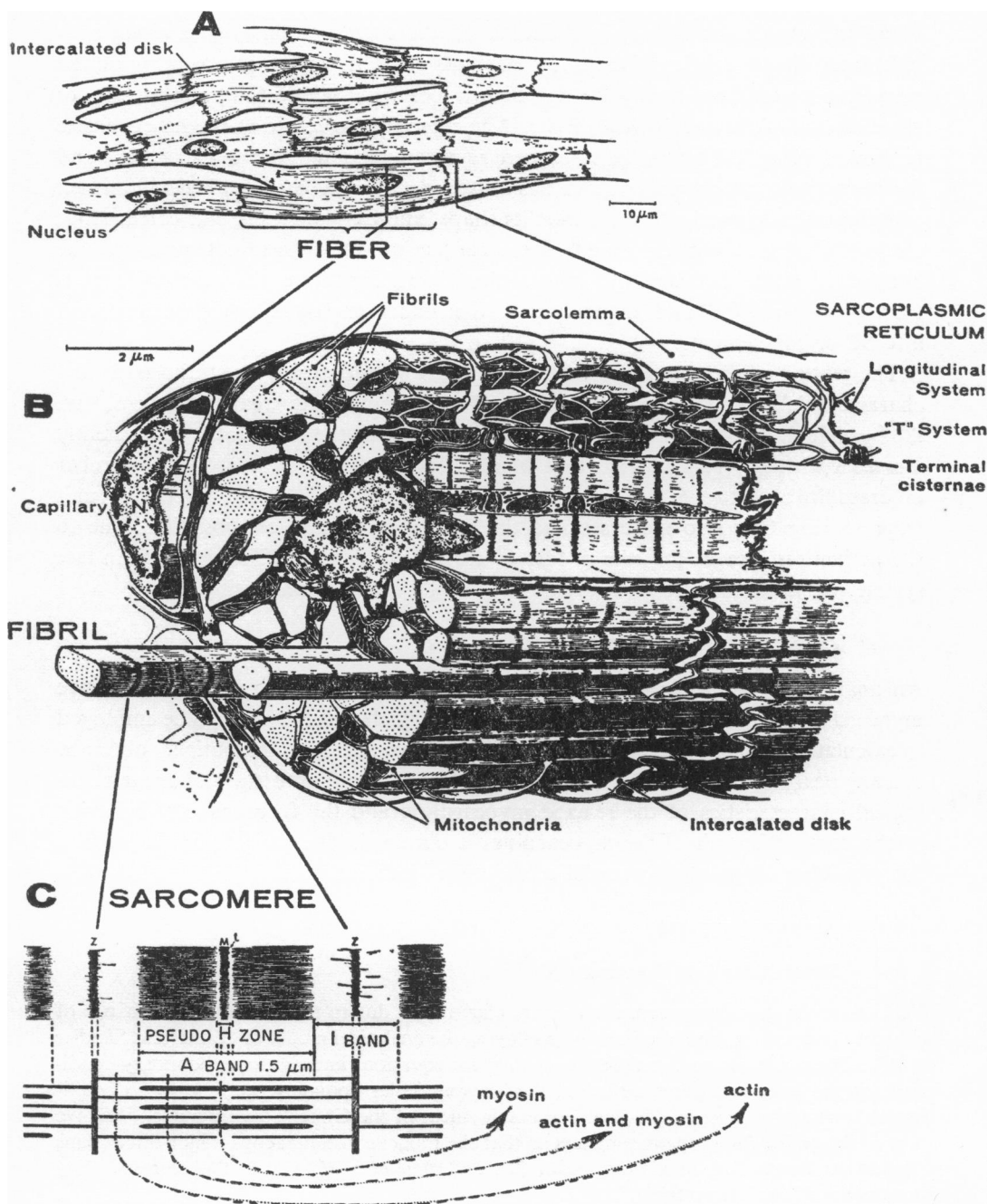


FIGURE 7 Structure of the heart muscle.

tored left ventricular history is indicated on the corresponding figure along with calculated characteristics of the series elements and CE. Before commenting on the results, it is useful to identify the anatomical locations (within the heart muscle) of the elements of the rheological model. A microscopic structure of the heart muscle is shown in Fig. 7. Therein the sarcomere represents the fundamental structural and functional unit of contraction. It is this unit that makes the muscle element contract (develop stress by activation), distort its shape, and do work; this unit corresponds to the CE of a rheological model of the cardiac muscle. The sarcolemma and the connective tissue between the myocardial fibers constitute the elastic elements (namely) SE and PE and, depending on the model employed, could be separated into SE or PE elements.

The plotted nonlinear variations of K^{XQ} and K^{PE} demonstrate the strain-hardening characteristic of the relaxed myocardial medium. A noteworthy feature of the S_i^{CE} vs. $\dot{\epsilon}_i^{CE}$ (= velocity of shortening of CE) plots is that for all the subjects S_i^{CE} consistently increases with $\dot{\epsilon}_i^{CE}$. This suggests that while CE is shortening in stage II, the number of cross-links (between the action and myosin filaments) of the sarcomere unit (representing CE) also increases and the rate of formation of cross-links due to CE activation exceeds the rate of loss of cross-links due to CE shortening; in late systole, we note this trend to reverse and the $\dot{\epsilon}_i^{CE}$ to start decreasing.

CONCLUDING REMARKS

An analysis for determining the rheological parameters of the passive and active myocardium has been presented. The manner in which the analysis is to be employed to calculate the in vivo values of the rheological parameters from routinely obtained cineangiocardigraphic data is demonstrated. An insight into the nonlinear rheological characteristics of the relaxed myocardium and the force-velocity behavior of the contractile unit of the myocardium is obtained. The analysis holds promise for providing diagnostically useful cardiological indices.

APPENDIX I

Stress in the Left Ventricular Model

The stresses in the instantaneous left ventricular model, due to the instantaneous change of chamber pressure, will be obtained by employing the analytical model of Ghista and Sandler (1969). These stresses need satisfy the equilibrium equations and the force boundary conditions on the inner and outer surface of the left ventricular boundary, i.e., they must equilibrate the chamber pressure loading. For employment of the Ghista-Sandler model, we have first to determine the shape parameters so that the model's instantaneous major dimensions (maximum length $2A$, maximum width $2B$, and thickness H) match the corresponding dimensions of the left ventricle.

Shape Parameters of the Model. The analytic model is derived by superposing a line dilatation force system (obtained by distributing point dilatations along a portion of length $2a$ of the Y axis of revolution) of intensity A and uniform hydrostatic stress system of

intensity B . Stress trajectories of the combined system are drawn in a plane containing the axis of revolution, with coordinates $r = r/a$, $y = y/a$. The two trajectories, whose intercept ratios $R_1 = \bar{y}_1/\bar{r}_1$, $R_2 = \bar{y}_2/\bar{r}_2$ on the axis r , y equal the ratios of length to width of the inner and outer surfaces of the left ventricle, are selected to represent the inner and outer boundaries of the geometrically similar model (see Fig. 8). These intercept ratios R_1 , R_2 are referred to as the shape parameters of the model. To make the geometrically similar model match the actual model in size, we select the value of the factor a equal to the ratio of the measured semi-width B of the inner surface of the left ventricular chamber and the r intercept \bar{r}_1 of the inner boundary of the geometrically similar model. At this stage the stresses due to the constituent stress systems of the model are functions of the stress intensity parameters α and β .

The parameters α and β of the two stress systems are obtained by satisfying the boundary conditions that (a) the instantaneous stress on the inner surface of the model must equal the instantaneous change in chamber pressure Δp , as (b) the stress on the outer surface of the model must be zero. Once the intensity parameters α and β are obtained in terms of the data quantities of left ventricular chamber pressure and dimensions, the stresses in the model are also expressed completely in terms of these data quantities.

The Stresses in the Model. The instantaneous stresses in the instantaneous model of the left ventricle (the instantaneous model simulates the left ventricle at an instant during the cardiac cycle) are given in dimensionless cylindrical coordinates (r , y) as follows:

$$\begin{aligned}\sigma_{rr} &= \frac{\alpha}{a^2} \left[-\frac{2}{r^2} \left\{ \frac{(y+1)}{[(y+1)^2 + r^2]^{1/2}} - \frac{(y-1)}{[(y-1)^2 + r^2]^{1/2}} \right\} \right. \\ &\quad \left. + 2 \left\{ \frac{(y-1)}{[(y-1)^2 + r^2]^{3/2}} - \frac{(y+1)}{[(y+1)^2 + r^2]^{3/2}} \right\} \right] + \beta \\ \sigma_{\theta\theta} &= \sigma_{\phi\phi} = \frac{\alpha}{a^2} \left[2r \left\{ \frac{1}{[(y+1)^2 + r^2]^{3/2}} - \frac{1}{[(y-1)^2 + r^2]^{3/2}} \right\} \right] \\ \sigma_{yy} &= \frac{\alpha}{a^2} \left[2 \left\{ \frac{(y+1)}{[(y+1)^2 + r^2]^{3/2}} - \frac{(y-1)}{[(y-1)^2 + r^2]^{3/2}} \right\} \right] + \beta \\ \sigma_{zz} &= \frac{\alpha}{a^2} \left[2 \left\{ \frac{(y+1)}{[(y+1)^2 + r^2]^{1/2}} - \frac{(y-1)}{[(y-1)^2 + r^2]^{1/2}} \right\} \right] + \beta, \quad (A 1)\end{aligned}$$

where $r = r/a$ and $y = y/a$. The determination of the instantaneous value of the size parameter a has been explained earlier in this section. Also in the above Eq. A 1 the parameters α , β are given in terms of the instantaneous values of chamber pressure and dimension as follows:

$$\begin{aligned}\alpha &= \frac{-(\Delta p)}{4 \left\{ \frac{1 + 2(d_2/a)^2}{a^2 [(d_2/a)^2 [(d_2/a)^2 + 1]^{3/2}} - \frac{1 + 2(d_1/a)^2}{(d_1/a)^2 [(d_1/a)^2 + 1]^{3/2}} \right\}} \\ \beta &= \frac{-(\Delta p)(d_1/a)^2 [(d_1/a)^2 + 1]^{3/2} [1 + 2(d_2/a)^2]}{\left\{ \frac{(d_1/a)^2 [1 + 2(d_2/a)^2] [(d_1/a)^2 + 1]^{3/2}}{-(d_2/a)^2 [1 + 2(d_1/a)^2] [(d_2/a)^2 + 1]^{3/2}} \right\}}, \quad (A 2)\end{aligned}$$

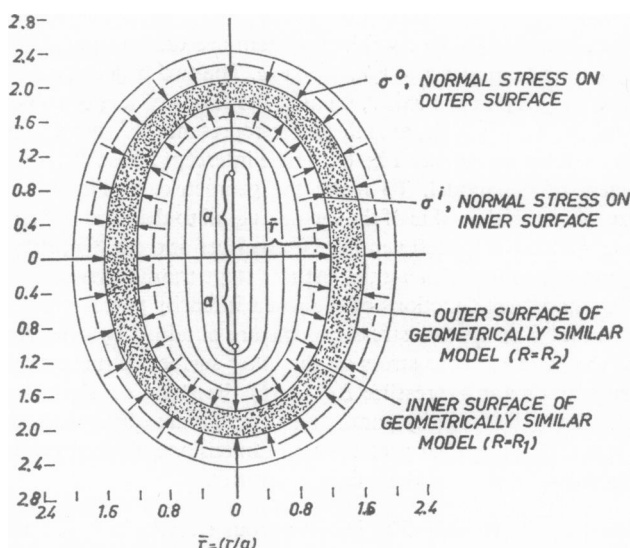


FIGURE 8 Geometrical model of the left ventricle obtained from stress trajectories of the superposed line dilatation and hydrostatic pressure systems.

where d_1 , d_2 are given in terms of the instantaneous semiwidth (B) and wall thickness (F) as follows:

$$d_1 = B; d_2 = F$$

In particular for the central equatorial element shown in Fig. 3, the stresses are given by

$$\left. \begin{aligned} \sigma_{rr} &= \frac{4\alpha}{a^2} \frac{(2\bar{d}_c^2 + 1)}{\bar{d}_c^3(\bar{d}_c^2 + 1)^{3/2}} + \beta, \\ \sigma_{r\theta} &= 0 = \sigma_{\theta r}, \\ \sigma_{\theta\theta} &= \frac{4\alpha}{a^2(\bar{d}_c^2 + 1)^{3/2}} + \beta, \\ \sigma_{\phi\phi} &= \frac{4\alpha}{a^2\bar{d}_c^2(\bar{d}_c^2 + 1)^{1/2}} + \beta, \end{aligned} \right\} \quad (A 3)$$

where $\bar{d}_c = c/2a$. While the above expressions give us the instantaneous stresses, we can also obtain the instantaneous changes in stresses, for an instantaneous change in chamber pressure (Δp) by substituting Δp for p in Eqs. A 2.

The contribution of Dr. Brady was supported by U. S. Public Health Service grant HE 00257-06.

Received for publication 12 February 1971 and in revised form 11 December 1972.

REFERENCES

- GHISTA, D. N., and H. SANDLER. 1969. *J. Biomech.* 2:35.
 BRADY, A. J. 1967. *Physiologist.* 10:75.
 GHISTA, D. N., and H. SANDLER. 1970. *J. Biomech.* 3:161.