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ACTIVE MATERIAL PROPERTIES OF THE MYOCARDIUM: CORRELATION WITH LEFT VENTRICULAR FUNCTION IN MAN[1]

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Abstract. The effects of anisotrophy and nonhomogeneity of the ventricular myocardium as represented by a linear increase in the midwall effective modulus of elasticity were investigated in the present study, specifically as they effect the circumferential stress distribution. Various studies are presented suggesting a linear increase in the effective modulus of elasticity from endocardium to epicardium. In our study, this increase in the effective modulus was constrained by the approximation that stress per unit sarcomere length is constant. We evaluated 12 functionally normal cases and 9 functionally abnormal cases. The stress distribution for the 9 functionally abnormal cases was calculated, first with the normal and secondly with the abnormal variation in the modulus of elasticity. Assuming the myocardium has constant material properties that do not change with functional decomposition, the stress distributions in the first calculations indicated higher stresses through the inner half of the myocardium and lower stresses through the outer half of the myocardium as compared to the second. This finding suggests that the inner fibers are overloaded and the outer fibers are underloaded in left ventricular decompensation. The difference between the first and second stress distributions averaged 26% (range: 11% to 48%). A useful, clinical, and quantitative measure of stress loading of the sarcomeres in functionally normal and abnormal left ventricles is proposed.

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Numerous investigators have applied thin-walled and thick-walled theories to develop models of the myocardial stress distribution. Initially, the law of Laplace (Wood 1892) was used to develop models for the left ventricle using thin-walled theory. The following approximations have been made:

1. the laws of elasticity apply to heart muscle tissue,
2. the myocardium is an isotropic, homogeneous and elastic medium,
3. the left ventricle is a spheroid of constant, thin-wall thickness,
4. ventricular wall stresses and deformations are functions of left ventricular pressure only, and
5. bending moments and shear forces are neglected.

With these approximations, Laplace's law assumes a uniform stress distribution across the wall thickness (Sandler and Dodge 1963).

Recently, investigators have used thick-walled theories to predict nonuniform stress distributions. Wong and Rautaharju (1968) analyzed the stress distributions assuming the heart is a passive, isotropic system. Their analysis applied the above approximations, except the geometry of the heart was modeled as a thick ellipsoidal shell with equal semi-minor axes. This model predicted wall stresses that were highest at the endocardium and lowest at the epicardium.

Streeter and co-authors (1970) developed a similar model but considered the curvature and orientation of the muscle fibers. His model predicted stresses that were highest at the midwall and lowest at the endocardium and epicardium. This method is limited in that empirical fiber curvature data is unavailable for the entire cardiac cycle.

Mirsky (1970) employed a thick-walled, prolate spheroid of constant wall thickness as his model and included relations for various degrees of anisotropy and nonhomogeneity of the myocardium. He assumed the circumferential modulus of elasticity was paraboloid in nature so that his predicted wall stress distribution corresponds with Streeter's predictions based on fiber orientation. Hanna (1973) used a ten-layered spherical myocardiumn as his model.

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The model, based on the force-length-velocity dependence of muscle, predicted the greatest stress at the epicardium and the lowest at the endocardium.

Janz and Waldron (1976) showed that nonhomogeneity of the myocardium is required in order to calculate a constant fiber stress through the myocardial wall. Their model required that the myocardial elastic modulus be lowest at the endocardium and highest at the epicardium. Mirsky (1976) showed in a very simple analysis that since strain (endocardial) is greater than strain (midwall), which in turn is greater than strain (epicardial), and if constant wall stress is assumed, then modulus of elasticity (E) increases from endocardium to epicardium for an incompressible material.

Of the aforementioned models, Mirsky's (1970) appears to be the most useful. Because the myocardium is an anisotropic nonhomogeneous material, the ability to quantify the material properties has a profound influence on the stress distribution through the myocardial wall. Mirsky's equations allow the use of approximate hypothetical modulus of elasticity distributions through the myocardium to establish various stress distributions.

The purpose of our study is to quantify the variation in the active modulus of elasticity and to determine the resultant stress distributions in functionally normal and functionally abnormal left ventricles.

MATHEMATICAL METHODS

Natural, Developed Strain. Natural strain (ϵ_N) has been proposed by Mirsky and Parmley (1974) as a more appropriate strain term for biological materials than Lagrangian strain (ϵ_L), where ϵ_N is defined as:

$$\epsilon_N = \ell n(1+\epsilon_L) \quad (1)$$

where

$$e_L = \ell_D - \ell/\ell_D \quad (2)$$

ℓ_D=end-diastolic equatorial fiber length (cm) and ℓ=instantaneous equatorial fiber length (cm).

Contractile Filament Stress. This study utilized circumferential contractile filament stress ($\sigma CF\theta$) as a modification of Lagrangian stress ($\sigma\theta$). Contractile filament stress accounts for an additional radial stress component (Grood, Phillips, and Mates 1979) and more closely represents the actual ventricular stress borne by the contractile filaments (Phillips and Grood 1978), see amended equation 8):

$$\sigma CF = \sigma_\theta + (P/2)(AR_\theta) \quad (3)$$

Lagrangian stress ($\sigma\theta$)is a circumferential Eulerian stress ($\sigma\theta^*$) normalized by an area ratio (AR_θ).

$$\sigma\theta = \sigma\theta^*(AR_\theta) \quad (4)$$

The circumferential Eulerian stress can be defined in terms of intraventricular pressure (P), instantaneous major axis of the left ventricular cavity (L), minor axis (M), and free-wall thickness (W) after Falsetti *et al* (1970):

$$\sigma\theta^* = PM(2L^2 - M^2)/4W(L^2 + MW) \quad (5)$$

The normalizing factor, the circumferential area ratio, accounts for the changing cross-sectional wall area referenced to the end-diastolic wall area and requires knowledge of the major axis (L_D), end-diastolic minor axis (M_D) and end-diastolic wall thickness (W_D) (Phillips *et al* 1978):

$$AR_\theta = \frac{(L_D^2/W_D + M_D + L_D^2/M_D + 4W_D/3)(1+L^2/MW)}{(L^2/W + M + L^2/M + 4W/3)(1+L_D^2/M_D W_D)} \quad (6)$$

Effective Modulus. The effective modulus ($\bar{E}_A$) is the ratio of stress to strain. Previously (Ghista *et al* 1975), the modulus value approached a large, but finite value during cardiac systole because the strain term reached a maximum. In order for $\bar{E}_A$ to be more realistically defined, the midwall circumferential effective modulus was characterized by a developed stress and an increasing strain during cardiac systole.

$$\bar{E}_A = (\sigma CF_i - \sigma CF_D)/\epsilon_{0.5}. \quad (7)$$

where $\bar{E}_A$=average midwall circumferential effective modulus, kilodynes/cm^2
σCF_i=instantaneous circumferential contractile filament stress, kilodynes/cm^2
σCF_D=end-diastolic circumferential contractile filament stress, kilodynes/cm^2
$\epsilon_{0.5}$=instantaneous midwall natural equatorial strain after equations 1 and 2.

Rationale for Modulus Distribution. It is proposed that myocardial nonhomogeneity is of such a nature that the stress per unit length is constant. Although there is no direct experimental evidence for this assumption, it does represent an "optimal" condition in which all sarcomeres bear an equal amount of the total load. The myocardium is divided at the midwall. The inner wall circumferential fiber length (ℓ_i) is less than the outer wall circumferential fiber length (ℓ_o) as shown in figure 1. Consequently, the outer wall fiber has more sarcomers in series and experiences

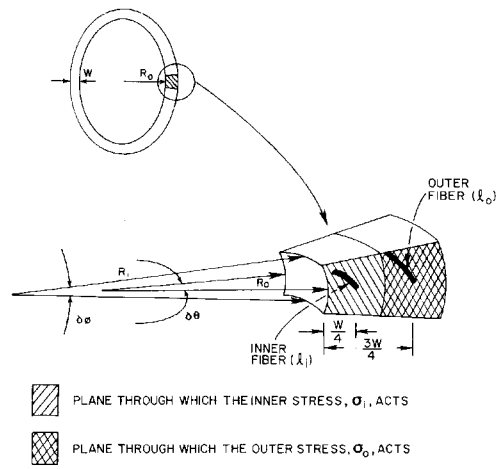

FIGURE 1. Element at the equator of an ellipsoidal left ventricle.

higher stresses than the inner wall fiber (if our hypothesis is correct). Also, if the inner fiber strain is greater than the outer fiber strain (for a circumferentially symmetrical contraction of an incompressible material), these stress-strain relationships combine to yield an increase in the effective modulus from endocardium to epicardium.

Constraint for Optimal Distribution and Definition of $\bar{A}$. The constraint for the optimal stress distribution requires that the stress per unit length of tissue be equally divided through the ventricular wall:

$$(8) \qquad \sigma_i/\ell_i = \sigma_o/\ell_o$$

Cox (1978) proceeded to derive $\bar{E}_\theta$, which is the effective circumferential modulus at X/W fractional wall thickness (in kilodynes per cm^2):

$$(9) \qquad \bar{E}_\theta = \bar{A}\,\bar{E}_A(X/W - .5) + \bar{E}_A$$

where $\bar{A}$=rate of change of $\bar{E}_\theta/\bar{E}_A$ with respect to X/W; where $\bar{E}_A$ has been defined from equation 7 and X/W is the fractional wall thickness as shown in figure 1.

Cox (1978) proceeded to derive an expression for the term, $\bar{A}$:

$$(10) \quad \bar{A} = 4({}^{\epsilon}.25^{\ell}.75 - {}^{\epsilon}.75^{\ell}.25)/({}^{\epsilon}.25^{\ell}.75 + {}^{\epsilon}.75^{\ell}.25)$$

where the subscripts denote fractional wall thickness (see figure 1), ϵ is the circumferential fiber natural strain (from equation 1), ℓ is the circumferential wall fiber length, and both terms (ϵ and ℓ for each fractional wall thickness) have been previously derived (Cox 1978).

Physical Interpretation of $\bar{A}$ and Definition of $\Delta\bar{A}/\Delta\epsilon$. $\bar{A}$ is a proportionality constant defining the rate of change of the effective modulus through the myocardium. If $\bar{A}$ equals zero, the modulus is constant through the myocardium; that is, the myocardium is homogeneous. $\bar{A}$ is a measure of the nonhomogeneity of the myocardium, and to this extent, is a true material property of the ventricular myocardium. However, for a different strain, a different $\bar{E}_A$ is defined, so that $\bar{A}$ is a function of strain (see equation 10). For a linear relationship between $\bar{A}$ and strain:

$$(11) \qquad \Delta\bar{A}/\Delta\epsilon = ({}^{\bar{A}}MAX - {}^{\bar{A}}MIN)/({}^{\epsilon}MAX - {}^{\epsilon}MIN)$$

where $\Delta A/\Delta\epsilon$=rate of change of $\bar{A}$ as a function of strain
${}^{\bar{A}}MAX$=maximum value of $\bar{A}$ (occurring at the end-systolic cineanogiographic frame).
${}^{\bar{A}}MIN$=minimum value of $\bar{A}$ (occurring at the first frame afrer the end-diastolic cineangiographic frame).
${}^{\epsilon}MAX$=maximum value of midwall equatorial strain ($\epsilon_{.5}$), occuring at ${}^{\bar{A}}max$.
${}^{\epsilon}MIN$=minimum value of midwall equatorial strain ($\epsilon_{.5}$), occurring at ${}^{\bar{A}}min$.

Physical Interpretation of $\Delta\bar{A}/\Delta\epsilon$. Mirsky and Parmley (1974) have shown that for papillary muscle, the modulus of elasticity varies with strain: as the strain increases, the modulus of elasticity becomes larger. This however, refers to the passive elastic modulus, as opposed to an "active" elastic modulus. $\bar{A}$, a measure of left ventricular nonhomogeneity, is also a function of strain (equation 10): as the strain increases, the ventricular nonhomogeneity increases. Typically, this behavior is not observed in inert, passive engineering materials; however, since ventricular myocardium is neither inert nor passive but living and active, one can conclude that a $\Delta\bar{A}/\Delta\epsilon$ relationship is a unique characteristic of active biological materials that does not appear to have a counterpart in the domain of inert, passive materials.

Stress Equation. Mirsky (1970) developed stress equations incorporating parameters representative of nonhomogeneity and anisotropy. Our present study considers circumferential stress ($\sigma\theta\theta$) only:

$$(12) \qquad \sigma\theta\theta = A\bar{g}_2(R)R^{-2-1/k} - \Pi$$

where: $\sigma\theta\theta$=Circumferential stress incorporating nonhomogeneity and anisotropy parameters
A=Constant of integration derived from Mirsky's radial force balance equations.
$\bar{g}_2$=Constant used to simplify Mirsky's stress equations
Π=Finite nonzero hydrostatic pressure used in Mirsky's stress equation development
k=Ratio of the two principal radii of curvature of a prolate spheroid

Equation 12 allows one to plot circumferential strain as a function of wall thickness at any given point in the cardiac cycle.

Constant Wall Volume Calculations. Wall thickness (W) was calculated assuming that left ventricular wall volume (${}^{V}wall$) remained constant throughout the cardiac cycle. Values for L, M, and W, obtained from the end-diastolic frame, were used to calculate the control volume.

$$(13) \qquad {}^{V}wall = \pi/6\ [(L+2W)(M+2W)]-LM^2$$

For all subsequent calculations cardiac systole, ${}^{V}wall$ remained constant, L and M were substituted into equation 13 as measured, and W was calculated by regression.

CLINICAL METHODS

Cineangiographic Techniques. The clinical case studies were obtained by cardiac catheterization of the left ventricle performed by Falsetti *et al* (1970). Measurement of the major axis (L), minor axis (M), and free wall thickness (W) were made as per Greene *et al* (1967); we determined that this technique provided sufficient and accurate left ventricle size information. We measured intraventricular pressure simultaneously with L, M, and W, using the cineangiographic techniques described by Falsetti *et al* (1970).

Case Groups. The clinical cases consisted of 21 patients, 10 male and 11 female, between the ages of 21 and 61. No patients had evidence of coronary artery disease, and all ventricles were observed to contract uniformly. The patient population was divided into 4 groups (table 1).

NORMAL (N): Seven patients were asymptomatic and were studied to evaluate a cardiac murmur. No hemodynamic abnormality of the left ventricle was found at the time of cardiac catherization.

COMPENSATED VOLUME OVERLOAD (CVO): Five patients who had regurgitation of the mitral and/or aortic valve and enlarged left ventricles were studied. None of the the patients had signs or symptoms of congestive heart failure.

DECOMPENSATED VOLUME OVERLOAD (DVO): Five patients had regurgitation of the mitral and/or aortic valve, complicated by clinical congestive heart failure (an enlarged heart, gallop rhythm, rales, and dyspnea).

CONGESTIVE CARDIOMYOPATHY (CC): Four patients in congestive heart failure who had large end-diastolic left ventricular volumes and low ejection fractions.

Calculations. The data processing proceeded in 3 phases. First, $\Delta A/\Delta\epsilon$ was calculated for each clinical case from equation 11 by making the appropriate substitutions. Next, σCF was calculated from equation 3 by making the appropriate substitutions. Finally, utilizing the two above variables along with other input variables, $\sigma\theta\theta$ was calcu-

TABLE 1

Descriptive clinical data for 21 patients.

Parameter*	Normal (n=7)**	Compensated Vol. Overload (n=5)	Decompensated Vol. Overload (n=5)	Congestive Cardiomyopathy (n=4)
BSA (m^2)	1.79± 0.23	1.70± 0.21	1.75± 0.19	1.85± 0.24
EDP (mm Hg)	8.7 ± 3.9	2.8 ± 2.0	11.2 ± 7.5	23 ± 9.5
PSP (mm Hg)	121 ±34	121 ±23	137 ±61	112 ± 6
EDV (ml)	159 ±39	224 ±71	242 ±88	258 ±83
EF	0.74± 0.06	0.71± 0.07	0.76± 0.07	0.37± 0.14

*BSA = Body Surface Area, EDP = End-Diastolic Pressure, PSP = Peak Systolic Pressure, EDV = End-Diastolic Volume, EF = Ejection Fraction.

**Number of Patients.

lated from equation 11. A detailed listing of the data processing sequence has been developed (table 1 of Cox 1978).

Data Analysis. The circumferential stress distributions were calculated across the myocardial wall. We calculated the difference between functionally normal and abnormal stress distributions as the area between the two curves (figure 2). This area was approximated by summing the average normal and abnormal stress values at each 0.1 fractional increment of unit wall thickness.

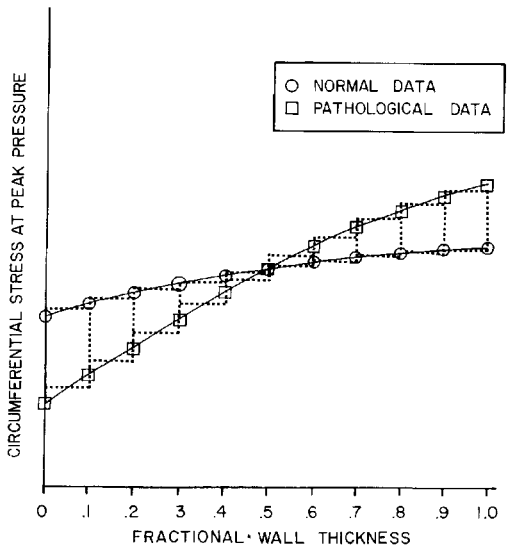

FIGURE 2. Normal and pathological stress distributions as a function of wall thickness illustrating the calculation of efficiency, $\%\Delta\sigma$.

$$\sigma DIFF = 0.1[10/\Sigma/i=i\ (\Delta\sigma_i)] \quad (14)$$

where

$\sigma DIFF$ = normal total difference between stress distributions, kilodynes/cm^2

$\Delta\sigma i$ = individual difference between stress distributions, kilodynes/cm^2

This approximate area was then normalized by the midwall stress value (σMID) and calculated as a percent difference between stress distributions ($\%\Delta\sigma$):

$$\%\Delta\sigma = [\sigma DIFF/(.1)(\sigma_{MID})] \times 100 \quad (15)$$

No difference between the stress distributions is represented by 0%, and increasing percentage values represent increasing differences.

RESULTS AND DISCUSSION

Various approximations have been necessary in order to analyze the active material properties of the left ventricle. First, the ventricle was modeled as a prolate spheroid with constant wall volume. Second, since cineangiographic techniques measure the wall thickness, major axis, and minor axis at discrete points in time, a quasi-steady state analysis was used to calculate the instantaneous circumferential stress through the myocardium at any point in time. Third, the myocardium is a nonhomogeneous material whose inner circumferential wall stress per unit length is equal to the outer circumferential wall stress per unit length. With these approximations and Mirsky's stress equations, circumferential stress through the wall of the heart was calculated for each cineangiographic frame throughout cardiac systole.

Figures 3 and 4 illustrate the midwall effective modulus versus strain for patients E.M. (Normal) and L.R. (DVO), respectively. Typically, the modulus increases very rapidly and then decreases almost exponentially. Normal and CVO cases illustrate very similar effective modulus distributions having similar amplitudes, rates of decay, and strain ranges. DVO and CC cases show distributions similar to one another but different from the Normal and CVO cases.

Table 2 summarizes the data used to determine the characteristic $\Delta\bar{A}/\Delta\epsilon$ value for

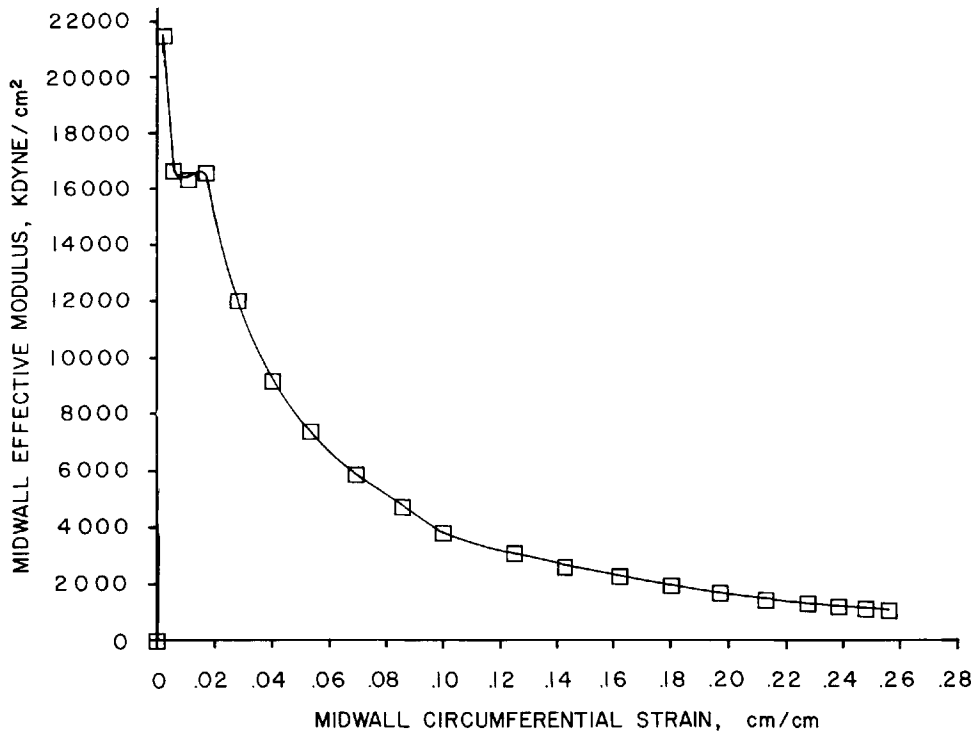

FIGURE 3. The effective modulus distribution as a function of strain for a typical normal case (Patient E.M.)

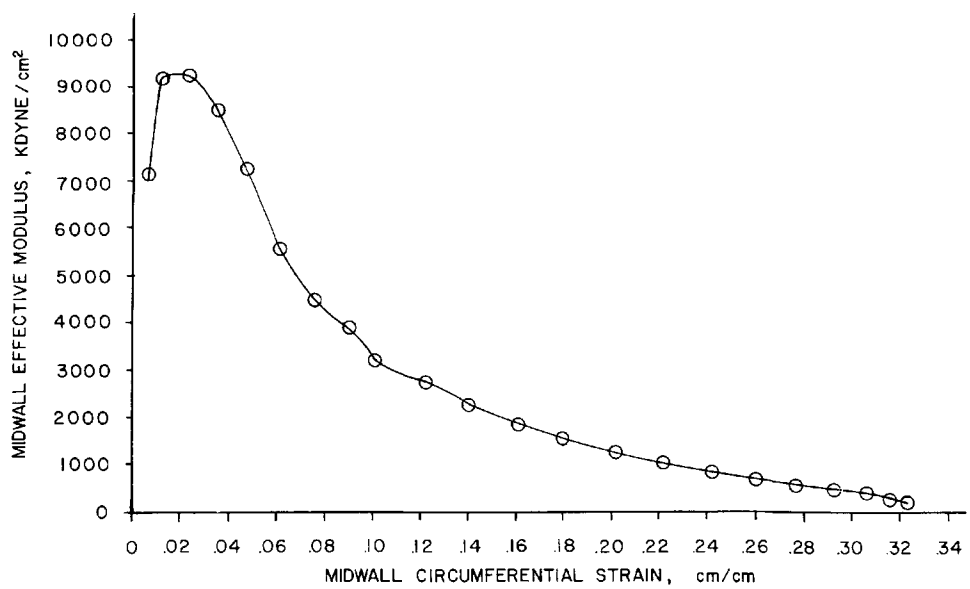

FIGURE 4. The effective modulus distribution as a function of strain for a typical DVO case (Patient L.R.)

TABLE 2

Five material parameters for the 21 cases investigated. Values given as $\bar{X} \pm$ SD.

Functional Group*	Maximum Strain	Minimum Strain	$\bar{A}_{max}$**	$\bar{A}_{min}$+	$\Delta\bar{A}/\Delta\epsilon$++
Normal	0.2662	0.0046	0.47	0.36	0.43
(Normal and CVO)	±0.0311	±0.0042	±0.11	±0.10	±0.20
Abnormal	0.1901	0.0029	0.56	0.40	1.09
(DVO and CC)	±0.0890	±0.0020	±0.24	±0.18	±0.41
Significance	N.S.	N.S.	N.S.	N.S.	$P<.0005$

*Significance of functionally abnormal group compared to the functionally normal group using Welch's t-test: N.S. = no significance. CVO=Compensated Vol. Overload; DVO=Decompensated Vol. Overload; CC=Congestive Cardiomyopathy.

**$\bar{A}_{max}$ = maximum value of $\bar{A}$ occurring at the end-systolic cineanogiographic frame.

+$\bar{A}_{min}$ = minimum value of $\bar{A}$ occurring at end-diastolic frame.

++$\Delta\bar{A}/\Delta\epsilon$ = rate of change of $\bar{A}$ as a function of strain.

each case. A significant difference exists between the mean value of $\Delta\bar{A}/\Delta\epsilon$ for the functionally normal cases (Normal and CVO) and the functionally abnormal cases (DVO and CC), being .43 and 1.09, respectively ($P<.0005$).

It is apparent that the circumferential stress increases from the endocardium to the epicardium (table 3). This is a direct result of the effective modulus distribution varied from the average midwall effective modulus through the wall thickness using the $\bar{A}$ term. The increase in stress between the inner and outer walls of the ventricle is reasonable both biologically and physically. Biologically, contractile filament stress development might be opposed by the collagen fibers that increase in mass through the wall of the heart. An increasing collagen distribution in conjunction with increased contractile units (sarcomeres), in series, constitutes a nonhomogeneity between the inner and outer halves of the myocardium (Buccino 1969).

Physically, the stress for a normal circumferentially symmetrical ventricular contraction should be similar for a fiber located near the inner wall and fiber located near the outer wall. When there is an increase in stress between the inner and outer walls created by increased fiber length (increased numbers of contractile units in series), a difference in stress can only by accounted for by a proportional increase in the effective circumferential modulus between endocardium and epicardium. In order to represent this nonhomogeneous condition, $\bar{E}_\theta$ was allowed to vary based on $\bar{A}$. A physical constraint of the analysis was placed on $\bar{A}$: stress per contractile unit (sarcomere) is constant through the wall of the heart. Since all contractile units are the same

TABLE 3

Three stress and three material parameters calculated at peak pressure ($\bar{X} \pm$ SD).

Functional Group*	Circumferential Stress Endocardium	Midwall	Epicardium	$\bar{A}$**	$\bar{E}_A$+	$\Delta\bar{A}/\Delta\epsilon$++
Normal	272	318	35	0.44	2398	0.43
(Normal and CVO)	±146	±163	±180	±0.11	±1261	—
Abnormal	219	278	321	0.54	2928	1.09
(DVO and CC)	± 89	± 86	± 98	±0.20	±1573	—
Abnormal	236	277	309	0.46	2928	0.43
(DVO and CC)	± 91	± 86	± 95	±0.19	±1573	—
Significance*	N.S.	N.S.	N.S.	N.S.	N.S.	

*Significance of functionally abnormal group compared to the functionally normal group using Welch's t-test: N.S. = no significance. CVO=Compensated Vol. Overload; DVO=Decompensated Vol. Overload; CC=Congestive Cardiomyopathy.

**$\bar{A}$ = rate of change of $\bar{E}_\theta/\bar{E}_A$ with respect to fractional wall thickness.

+$\bar{E}_A$ = average midwall circumferential effective modulus, kilodynes/cm^2.

++$\Delta\bar{A}/\Delta\epsilon$ = rate of change of $\bar{A}$ as a function of strain.

length, the number of contractile units in series increases from endocardium to epicardium. Because of the increasing fiber length, the outer half of the heart wall experiences more stress than the inner half, but the stress per unit length remains constant.

Two conclusions can be drawn from this study:

1. a numerical descriptor, $\Delta\bar{A}/\Delta\epsilon$, had distinctly different values for functionally normal versus functionally abnormal cases, and
2. stress distributions based on the functionally abnormal $\Delta\bar{A}/\Delta\epsilon$ values diverged significantly from stress distributions based upon the functionally normal $\Delta\bar{A}/\Delta\epsilon$ values.

The mean values of $\Delta\bar{A}/\Delta\epsilon$ for the functionally normal and abnormal cases were significantly different, implying that the $\Delta\bar{A}/\Delta\epsilon$ values were representative of two different functional groups.

The stress distribution is optimal for the normal and CVO cases when $\Delta\bar{A}/\Delta\epsilon$ equals 0.43. If the material properties of the CC and DVO cases do not change with clinical decompensation, their $\Delta\bar{A}/\Delta\epsilon$ values would be 0.43 also. Thus, we calculated stress distributions for each functionally abnormal case using the functionally normal $\Delta\bar{A}/\Delta\epsilon$ value. For the DVO and CC groups, however, the individual $\Delta\bar{A}/\Delta\epsilon$ values for the optimal stress distribution were much higher than the functionally normal values, averaging 1.09. By employing this mean $\Delta\bar{A}/\Delta\epsilon$ value for each functionally abnormal case, the optimal circumferential stress distributions were established and compared to the predicted normal stress distributions (when $\Delta\bar{A}/\Delta\epsilon$ equals .43). The optimal stress distributions were not the same as the predicted normal distributions, averaging a 26% difference. Table 4 summarizes the circumferential stress distributions of the 9 functionally abnormal cases for $\Delta\bar{A}/\Delta\epsilon$ values of .43 and 1.09. The $\%\Delta\sigma$ values for the two stress distributions range from 11% to 48% (mean: 26%). These large values indicate that there is a fundamental difference between the functionally normal and functionally abnormal stress distributions.

Figure 5 illustrates the myocardial stress distributions of CC case for both the 1.09 value of $\Delta\bar{A}/\Delta\epsilon$ and the 0.43 value. This decompensated case (R.T.) shows a large difference between the predicted stress distributions for the two $\Delta\bar{A}/\Delta\epsilon$ values.

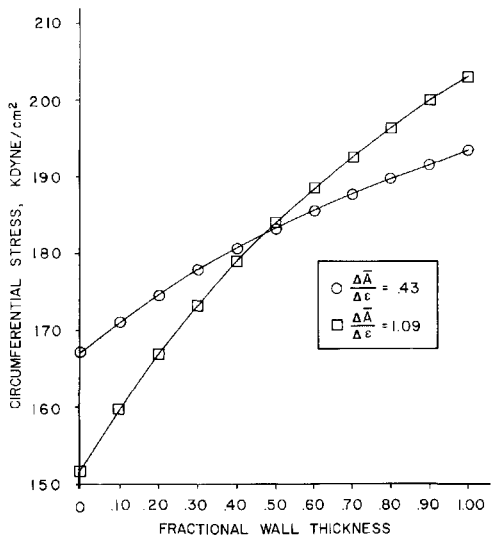

FIGURE 5. Circumferential stress distributions for CC (Patient R.T.)

The difference between the optimal and predicted normal stress distributions (table 4) represents the inefficiency of the functionally abnormal cases compared to the functionally normal cases. Typically, the functionally abnormal cases have optimal stress values higher at the epicardium and lower at the endocardium than the corresponding predicted normal distributions. The endocardial fibers are "overloaded" (they carry a greater than optimal stress per contractile unit) and the epicardial fibers are "underloaded" (they carry a smaller than optimal stress per contractile unit).

This study is significant because the active material properties of the myocardium have been calculated and for the first time correlated with left ventricular function. $\Delta\bar{A}/\Delta\epsilon$ appears to be a unique active material property of ventricular myocardium not observed in passive, inert materials, and its

TABLE 4

Circumferential stress distributions of 9 functionally abnormal cases and the resulting inefficiency of the stress distributions.

Patient	$\Delta\bar{A}/\Delta\epsilon$*	Fractional Wall Thickness				%$\Delta\sigma$**
		.05	.35	.65	.95	
L.R.	0.43	149	236	305	360	33
	1.09	130	231	311	375	
A.S.	0.43	298	299	302	306	16
	1.09	288	297	306	314	
J.N.	0.43	155	159	164	172	48
	1.09	137	155	169	183	
R.K.	0.43	423	445	465	486	26
	1.09	398	439	473	504	
P.L.	0.43	234	262	285	304	32
	1.09	215	257	290	318	
R.F.	0.43	241	266	287	306	26
	1.09	226	262	292	317	
M.S.	0.43	304	327	345	360	11
	1.09	297	325	347	366	
J.F.	0.43	197	225	248	267	13
	1.09	190	223	250	272	
R.T.	0.43	169	179	187	193	32
	1.09	156	176	191	202	

*$\Delta\bar{A}/\Delta\epsilon$ = rate of change of $\bar{A}$ as a function of strain.
**$\Delta\sigma$ = % difference between the stress distributions (see equation 15 and figure 2).

characteristic value appears to distinguish the functionally normal from functionally abnormal left ventricle.

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