

CONTRACTILE FILAMENT STRESS: COMPARISON OF DIFFERENT DISEASE STATES IN MAN¹

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Abstract. Cardiac catheterization data on 39 patients was classified in 5 cardiovascular groups: normal, compensated volume overload, decompensated volume overload, compensated pressure overload, and congestive cardiomyopathy. Both the Lagrangian stress and contractile filament stress for the circumferential axis and the longitudinal axis were computed over a complete cardiac cycle. Contractile filament stress was 24% higher than Lagrangian stress in the circumferential direction, and 43% higher than Lagrangian stress in the longitudinal direction. The percent difference in stress between the contractile filament stress and Lagrangian stress was greatest for patients with pressure overload, and least for patients with compensated volume overload. No significant difference in calculated wall stress was noted between the normal group and the 4 pathological groups. Circumferential velocity of the contractile element occurring at peak stress was plotted as a function of peak contractile filament stress and patients with compensated pressure overload exhibited high values of both velocity and peak stress. Patients with congestive cardiomyopathy showed low values of both velocity and peak stress. Circumferential velocity of the contractile element occurring throughout the cardiac cycle was plotted as a function of both the instantaneous Lagrangian stress and the instantaneous contractile filament stress, resulting in 2 stress-velocity curves for each patient. The value of the maximum velocity extrapolated from either stress-velocity curve was approximately the same, but the maximum stress extrapolated from the contractile filament stress-velocity curve was significantly higher than the maximum stress extrapolated from the Lagrangian stress-velocity curve. The product of peak contractile filament stress in the circumferential direction times heart rate was a clinically useful index of myocardial oxygen consumption, and predicted a lower rate of oxygen consumption than did the product of peak developed stress times heart rate.

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Two types of stress have often been employed to describe the mechanics of ventricular contraction: a *passive* stress, which designates the load placed upon the muscle fibers of the ventricle, and an *active* stress, which designates the force per unit area developed by the myocardial fibers and transmitted to the ventricular wall. In both cases, stress is dependent upon a knowledge of ventricular pressure, geometry and wall thickness, in addition to some assumptions as to the material properties of the ventricle. Stress is important because it affects the velocity of

muscle fiber contraction and the resultant stress-velocity relationship, which characterizes the heart as an active muscle, and provides a quantitative estimation of cardiac performance.

Previous investigators applied stress calculations in a one-dimensional direction, using as a model the isolated muscle fiber which we have previously referred to as the *one-dimensional muscle model* (Grood *et al* 1974). Most investigators have only considered *passive* stress, beginning with Wood (1892) who applied Laplace's Law to the heart. Sandler and Dodge (1963) applied the basic Laplace relation, modified to incorporate wall thickness, to an ellipsoidal left ventricle, but recognized that the application of thin-wall theory to a thick-walled struc-

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ture could only predict an average stress across the wall. Wong and Rautaharju (1968) were the first to actually compute the stress distribution across the wall, employing a thick-walled ellipsoidal left ventricle, but assumed the myocardium was an isotropic, homogeneous material, neglecting bending moments and wall shear forces.

Ghista and Sandler (1969) included visco-elastic properties in a thick-walled ellipsoidal model and accounted for shear forces in the ventricular wall. Mirsky (1969) also performed an analysis which incorporated wall forces (i.e. bending moments and shear stresses) but concluded that their effects were negligible. Gould *et al* (1972) divided the left ventricular wall into 12 shell ring elements (a finite element model) which predicted peak stresses at the apex that were 1.5 times greater than that given by the Ghista and Mirsky models.

Recently, a new generation of models appeared representing ventricular simulations of active stress and predicting the dynamic events of stress development and velocity of shortening of the contractile component as functions of the active state of muscle (Wong 1973, Hannah 1973). Wong recognized that previous models were static and inverted the process of ventricular activity (i.e., wall stress was computed as a function of ventricular pressure) rather than computing pressure as a function of developed wall tension. Both Wong and Hannah employed a ventricular model with multiple shells but the former accounted for fiber orientation.

The primary purpose of our study was to evaluate left ventricular wall stress in various clinical populations with respect to contractile filament stress, which differs from previous approaches to wall stress because it is an *active* stress based upon a *three-dimensional* muscle model. A fundamental understanding of the mechanics of cardiac contraction also depends on the relationship of the generated stress to the velocity of shortening of the myocardial fibers; velocity of fiber contraction being inversely related to the load (or stress) applied. A second purpose of our study was to evaluate the resultant stress-velocity relationship in

various clinical populations, based upon a definition of contractile filament stress. Finally, the product of contractile filament stress in the circumferential direction times heart rate was evaluated as an index of myocardial oxygen consumption.

METHODS AND MATERIALS

Cardiac catheterization data was acquired from a previous study at the State University of New York at Buffalo of 39 patients, 13♂ and 26♀, ages 21 to 64. Forty-five patients were initially included, but 6 were rejected due to incomplete data (Falsetti *et al* 1971a). Patients with localized ventricular disease were also excluded from this study.

GROUPS

NORMAL: Composed of 15 patients who were asymptomatic and were studied to evaluate a cardiac murmur. These patients had no demonstrable abnormality of the left ventricle at cardiac catheterization.

COMPENSATED VOLUME OVERLOAD: Composed of 6 patients who had regurgitation of the mitral or aortic, valve or both, with enlarged left ventricles but no other evidence of clinical congestive heart failure.

DECOMPENSATED VOLUME OVERLOAD: Composed of 9 patients who had aortic or mitral regurgitation, or both, complicated by clinical congestive heart failure (defined as an enlarged heart, gallop rhythm, rales and dyspnea).

COMPENSATED PRESSURE OVERLOAD: Composed of 3 patients. Two patients had aortic stenosis and one had idiopathic hypertrophic subaortic stenosis.

CONGESTIVE CARDIOMYOPATHY: Composed of 6 patients in clinical congestive failure who had high end-diastolic volumes of the left ventricle with low ejection fractions.

Cardiac catheterization and left ventricular angiography were carried out using materials and methods reported by Falsetti and co-workers (1971a) which allowed simultaneous recording of left ventricular pressure with cineangiographic frame exposures. The catheter (KIFA, 1.5 mm I.D. and 100 cm long) was well flushed and connected directly to a Statham P23Db pressure transducer which led into an E for M direct writing recorder. The pressure traces did not exhibit the oscillations observed in very lightly damped catheter systems. The Statham transducer used estimates a natural frequency of 70Hz and a damping ratio of 0.3 for the combined catheter-transducer system.

Left ventricular geometry was previously determined by the one-plane cineangiographic technique (Greene *et al* 1967, Falsetti *et al* 1971b). Measurements were made throughout the cardiac cycle at 60 frames per second. Some scatter appeared in the volumetric data due to the difficulty of making geometric measurements from the cineangiogram frames. Consequently, a filtered 5th-order Fourier series was employed to curve-fit the data points

(Phillips 1977). Ventricular volume (V^*) was assumed to be a prolate spheroid described by the equation:

$$(1) \quad V^* = \pi LM^2/6 f^3$$

where: L =major axis (cm), M =minor axis (cm), and f =magnification factor. The volumes calculated from cineangiograms were corrected for overestimation (which occurred during systole) by use of a regression equation (Greene *et al* 1967):

$$(2) \quad V = 0.848 V^* - 10.24 \text{ ml.}$$

where: V^* =calculated angiographic volume (ml).

STRESS DEFINITIONS AND CALCULATIONS

Both the Lagrangian and contractile filament stresses for the longitudinal and circumferential directions were computed every $16\frac{2}{3}$ msec for a complete cardiac cycle. The Lagrangian stress, referred to as *fiber corrected stress* by Falsetti *et al* (1970) is the force per unit area normalized by some reference areas, and accounts for a changing wall cross-sectional area during systole even though the number of actual muscle fibers remained constant. Contractile filament stress is a type of *active* wall stress and represents the actual ventricular wall stress that is borne by the contractile filaments *per se* (Grood *et al* 1974). Contractile filament stress requires a three-dimensional muscle model in which the radial stress component is identified and coupled to the circumferential and longitudinal stress components. This results in a higher stress than would be predicted by the Lagrangian stress because the three-dimensional binder of water and connective tissue transmits a compressive stress during most of systole in addition to the tensile stresses developed by the contractile fibers.

The following equations were used in which L =major axis of the left ventricle (cm), M =minor axis (cm), W =left ventricular wall thickness (cm), and P =intraventricular pressure (1 mm Hg = $1.333 \text{ K-Dynes/cm}^2$).

Lagrangian stress in the circumferential direction:

$$(3) \quad \sigma_\theta = (\sigma_\theta)^* (\text{Area Ratio})_\theta$$

where $(\sigma_\theta)^*$ =an uncorrected circumferential left ventricular wall stress (Falsetti *et al* 1970):

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

and $(\text{Area Ratio})_\theta$ =the normalization term which corrects $(\sigma_\theta)^*$ for changes in circumferential ventricular cross-sectional wall area (Phillips *et al* 1978):

$$(5) \quad (\text{Area Ratio})_\theta = \frac{(L_d^2/W_d + M_d + L_d^2/M_d + 4W_d/3)(1 + L^2/MW)}{(1 + L^2/M_dW_d)(L^2/W + M + L^2/M + 4W/3)}$$

where L_d =major axis at end-diastole (cm), M_d

=minor axis at end-diastole (cm), and W_d =wall thickness at end-diastole (cm).

Lagrangian stress in the longitudinal direction, σ_ϕ :

$$(6) \quad \sigma_\phi = (\sigma_\phi)^* (\text{Area Ratio})_\phi$$

where $(\sigma_\phi)^*$ =an uncorrected longitudinal left ventricular wall stress, and $(\text{Area Ratio})_\phi$ =the normalization term (Falsetti *et al* 1970):

$$(7) \quad \sigma_\phi = PM^2/4W_d (M_d + W_d)$$

Contractile filament stress in the circumferential direction, $\sigma_{CF\theta}$:

$$(8) \quad \sigma_{CF\theta} = \sigma_\theta + P/2$$

Contractile filament stress in the longitudinal direction, $\sigma_{CF\phi}$:

$$(9) \quad \sigma_{CF\phi} = \sigma_\phi + P/2$$

where $P/2$ is an approximation of the radial stress component (Grood *et al* 1974).

STRESS-VELOCITY CALCULATIONS

The parameters of muscle function were derived from a one-dimensional contractile element (CE) and a series elastic element (SE), each representing the *lumped* values of many individual components of the actual muscle. Contractile filament stress for the circumferential direction of the left ventricle, $(\sigma_{CF\theta})$ was utilized in Equation 10. Lagrangian stress in the circumferential direction (σ_θ) can also be utilized, but the resultant series elastic element velocity (V_{SE}) was virtually identical:

$$(10) \quad V_{SE} = [d(\sigma_{CF\theta})/dt] / [K(\sigma_{CF\theta}) + C]$$

K was assumed to be equal to the stiffness of cat papillary muscle (28.8) after Sonnenblick *et al* (1962). The constant of integration, C , was assumed to be negligible.

Circumferential fiber velocity (V_{CF}) was defined from the hemodynamic measurements:

$$(11) \quad V_{CF} = -(dM/dt + dW/dt) / (M_d + W_d)$$

Contractile element velocity (V_{CE}) was positive for shortening and is defined by:

$$(12) \quad V_{CE} = V_{CF} + V_{SE}$$

The contractile element velocity was normalized for heart rate (\bar{V}_{CE}) by multiplying it by the period of observed cardiac cycle, thus correcting for the positive inotropic effect of heart rate which was observed by Covell *et al* (1967). Normalized contractile element velocity was plotted as a function of contractile filaments stress to define the contractile filament stress-velocity relationship for the left ventricle. Normalized contractile element velocity was plotted as a function of Lagrangian stress. Maximum velocity was obtained by extrapolation of the descending portion of the stress-velocity curve to intercept the velocity axis at zero stress. Maximum stress was obtained by extrapolation of the same descending portion of the curve to intercept the stress axis.

Recently, Burns and Covell (1972) investigated myocardial oxygen consumption during paired isovolumic/isotonic contractions of the intact dog heart. We took their basic data (see their table 4) and evaluated their 11 isovolumic contractions, ignoring the isotonic contractions, since the isovolumic contractions are independent of muscle length or velocity changes. Peak circumferential contractile filament stress (gms/cm²) was defined by adding a radial stress component (σ_r):

$$(13) \quad \sigma_r = (1.36) (\text{Peak Left Ventricular Pressure, mm Hg})/2 \text{ (gms/cm}^2\text{)}$$

to the peak developed stress reported by these authors. The myocardial oxygen consumption per unit mass was then plotted as a function of the product of contractile filament stress times heart rate, and also as a function of the product developed stress times heart rate.

RESULTS AND DISCUSSION

The values of end-diastolic volume, end-systolic volume, and ejection fraction differed slightly from those reported by Falsetti *et al* (1971a), primarily because of differences in curve smoothing techniques (table 1). We employed a 5th order filtered Fourier series to smooth the data, while Falsetti and co-workers used a 5th order polynomial.

The most reliable stress values were for the normal group, which had the largest population (15 patients), and the least reliable values were for the pressure overload group with only 3 patients (table 2). In all cases, the contractile

TABLE 1

Descriptive Hemodynamic Data For 39 Cardiovascular Patients Composing Five Clinical Groups.

Parameter	Normal	Compensated Volume Overload	Decompensated Volume Overload	Compensated Pressure Overload	Congestive Cardiomyopathy
BSA (m ²) ^b	1.69±0.24 ^a	1.69±0.19	1.69±0.16	1.74±0.31	1.84±0.19
EDP (mmHg) ^c	7.7±3.8	3.5±3.0	9.4±7.4	13±3.5	24.3±9.8
PSP (mmHg) ^d	123±30	123±22	134±51	175±16	101±19
EDV (ml) ^e	143±37	213±69	205±77	165±46	293±87
EF ^f	0.69±0.10	0.73±0.08	0.71±0.11	0.76±.006	0.34±0.13

^aValues are shown ± Standard Deviation.

^bBSA = Body Surface Area

^cEDP = End-Diastolic Pressure

^dPSP = Peak Systolic Pressure

^eEDV = End-Diastolic Volume

^fEF = Ejection Fraction

TABLE 2

Group Averages of Peak Lagrangian Stress, Peak Contractile Filament Stress and Percent Change in Stress.

Group	Peak σ_θ	Peak $\sigma_{CF\theta}$	% Change ^e	Peak σ_θ	Peak $\sigma_{CF\theta}$	% Change ^f
Normal	341±111	424±124	25.8±9.8	186±71.1	276±85.7	47.6±16.3
CVO ^a	485±329	560±337	19.8±8.9	296±194	381±202	35.1±14.1
DVO ^b	361±95.2	453±133	24.9±7.5	194±48.6	285±80.6	46.7±12.0
CPO ^c	381±99.8	504±106	33.6±9.1	190±28.0	301±30.8	59.6±11.5
CC ^d	287±54.4	356±61.6	24.8±6.2	187±42.4	252±51.8	35.7±6.7
Total:	362±158	447±170	24.3±7.2	205±95.3	288±107	43.4±12.1

^aCVO = Compensated Volume Overload

^bDVO = Decompensated Volume Overload

^cCPO = Compensated Pressure Overload

^dCC = Congestive Cardiomyopathy

^e $\frac{(\text{Peak } \sigma_{CF\theta}) - (\text{Peak } \sigma_\theta)}{(\text{Peak } \sigma_\theta)} \times 100$

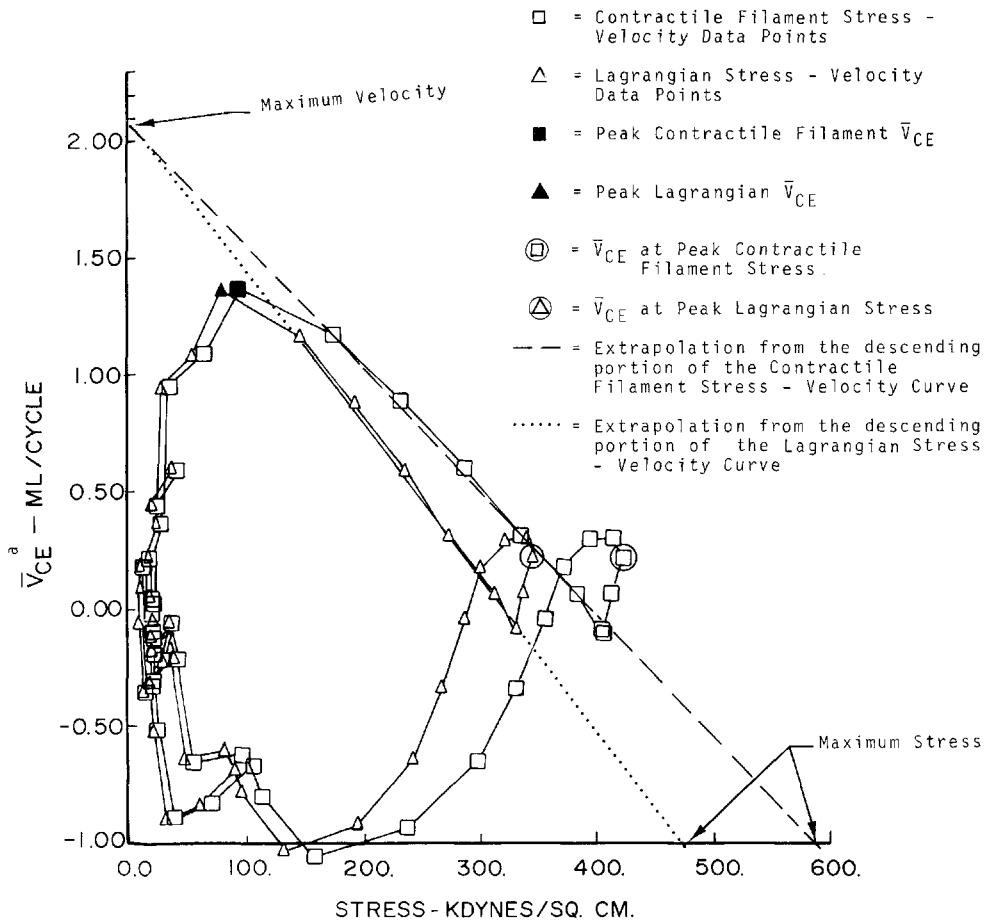
^f $\frac{(\text{Peak } \sigma_{CF\phi}) - (\text{Peak } \sigma_\phi)}{(\text{Peak } \phi)} \times 100$

(Peak σ_θ)

filament stresses were higher than the Lagrangian stresses. Both the Lagrangian stress and the contractile filament stress in the circumferential axis was uniformly higher than in the longitudinal. The average ratio of longitudinal to circumferential stress was .58 for the Lagrangian stress, and .66 for the contractile filament stress.

Paired t-tests between the normal patient group and the other 4 patient groups were performed for each of the 4 stresses. No significant differences were noted between the normal group and each of the pathological groups. This observation was consistent with the results of

Hood *et al* (1968) that the heart tended to hypertrophy appropriately to the pressure and volume demands placed upon it in order to operate in a range of optimal stress values. Our results indicated that the wall stresses for the decompensated volume overload patient group were normalized, whereas they were elevated in the study of Hood *et al* (1968). Since our group consisted of 9 patients, whereas Hood's decompensated volume group overload group consisted of only 3 patients, we feel that in the majority of cases, wall stresses probably do normalize in decompensated volume overload.



^aVelocity of the Contractile Element (Circumferential), normalized for heart rate

FIGURE 1. Contractile filament and Lagrangian stress-velocity relationship for the circumferential axis.

The percent difference of the contractile filament stress over the Lagrangian stress in the circumferential and longitudinal axis for each of the 5 patient groups and the total group was examined (table 2). The percent change of the stress was approximately 43% higher in the longitudinal direction, and approximately 24% higher in the circumferential direction for the total patient population. The percent change of the stress was less than the group average for the compensated volume overload patients, and greater than the group average for the compensated pressure overload patients. With pressure overload, the higher transmural pressures may account for the difference, and with compensated volume overload, a greater degree of myocardial hypertrophy (larger values of wall thickness) may occur than for the decompensated volume overload groups.

Contractile filament stress, as compared to the Lagrangian stress, had various effects on the stress-velocity relationship, as shown in figure 1. Peak con-

tractile filament velocity was equal to peak Lagrangian velocity but occurred at a slightly higher level of stress. Velocity at peak contractile filament stress was equal to velocity at peak Lagrangian stress, but occurred at a significantly higher level of stress. Thus, the descending portion of the contractile filament stress-velocity curve shifted to the right of the Lagrangian stress-velocity curve. The extrapolated value of maximum velocity from each of the two curves was not significantly different, but the extrapolated values of the two maximum stresses were significantly different. This change in maximum stress at a constant maximum velocity implied that the muscle fiber experiencing contractile filament stress was operating at a higher point on the length-tension curve than would be predicted from the Lagrangian stress. If maximum velocity or peak velocity was taken to represent an index of contractility, then the increase in contractile filament stress over Lagrangian stress did not necessarily represent

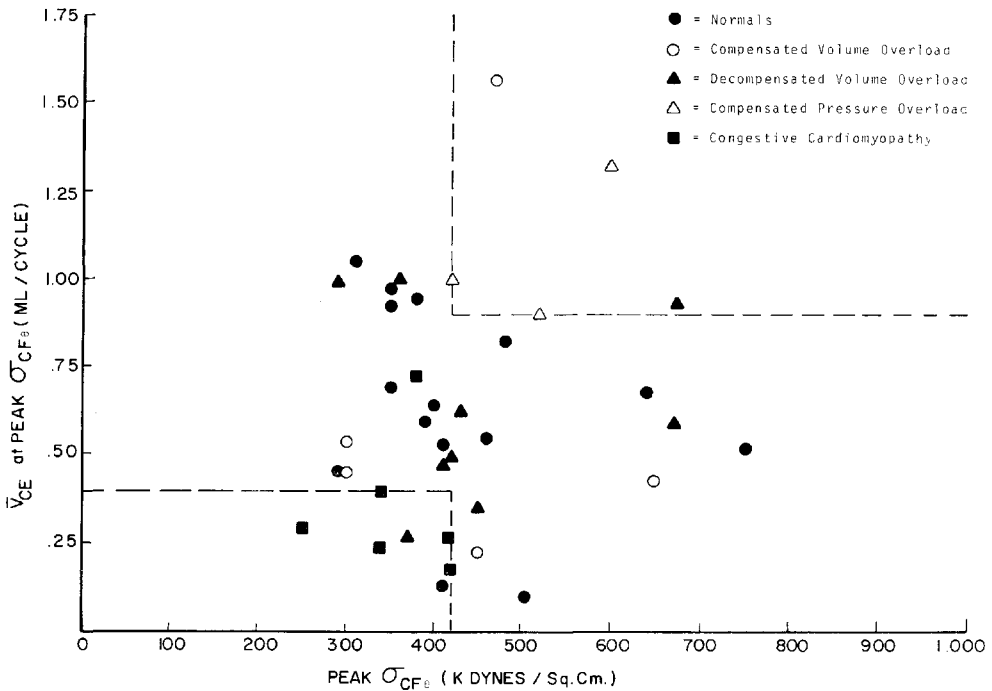


FIGURE 2. Normalized circumferential contractile element velocity (\bar{V}_{CE}) at peak circumferential contractile filament stress (σ_{CF_e}) as a function of peak circumferential contractile filament stress.

an increase in the contractile state of the muscle.

Figure 2 shows velocity of shortening at peak contractile filament stress as a function of peak contractile filament stress for the circumferential direction. Five of the 6 cardiomyopathy patients had low values of contractile element shortening velocities at peak stress (0.40 lengths/cycle and below), and also had lower peak stress levels (420 K Dynes/cm² and below). This observation is consistent with that of Falsetti *et al* (1970), who found that contractile element velocity at peak fiber corrected stress ranged from 0.22 to 0.32 lengths/sec in a group of 4 cardiomyopathy patients. All 3 of the compensated pressure overload patients had high values of contractile element shortening velocities at peak stress (0.90 lengths/cycle and above) and also had higher peak stress levels (420 K Dynes/cm² and above). The difference between these two groups was statistically significant at the 5% level. This distinction between the pressure overload and congestive cardiomyopathy groups was not evident when the Lagrangian stresses were evaluated. The differences in intraventricular pressures, (one half of which represented our approximation of

the radial stress component) can account for the differences. Peak systolic pressure (mmHg) was higher in the pressure overload group (175±16) than normals (123±30), and lower in the cardiomyopathy group (101±19).

Our data suggested that pressure overload may impose significantly higher stress loads on the contractile filaments than does volume overload. This higher stress with pressure overload was not evident when using Eulerian or Lagrangian stress calculations since they do not account for the radial component of stress (approximated as one-half the transmural pressure). The clinical implication may well be to focus on systolic pressure levels (as well as diastolic pressure levels) when treating hypertensive patients. By lowering the overall transmural pressure, the radial stress component is lowered, and the total stress on the myocardial contractile filaments is consequently lowered.

Contractile filament stress may be clinically useful as a predictor of myocardial oxygen consumption. The myocardial oxygen consumption data of Burns and Covell (1972) has been re-examined in this study (fig. 3). Peak contractile filament stress times heart rate correlated well with oxygen consump-

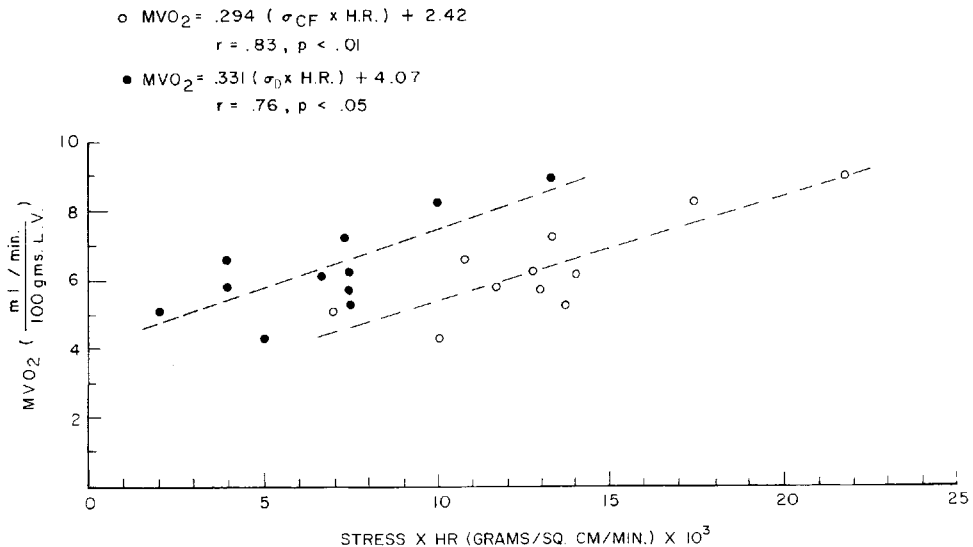


FIGURE 3. Myocardial oxygen consumption (MVO_2) a function of peak circumferential developed stress (σ_p) times heart rate (H.R.) (closed circles) and peak circumferential contractile filament stress (σ_{CF}) times heart rate (open circles) for a series of isovolumic contractions.

tion during isovolumic contractions ($r=0.83$) and was significant at the 1% level. Peak developed stress times heart rate also correlated ($r=0.76$) and was significant at the 5% level. While further studies need to be performed, currently available data indicates that contractile filament stress shifts the oxygen consumption versus stress curve downward and to the right. While our results only provide information about average stresses, they are probably more useful than detailed stress distributions based upon unrealistic assumptions about muscle properties. Furthermore, average stresses are also directly related to tensile forces which can be measured in the open chest preparation (Hefner *et al* 1962).

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