An Analysis of the Mechanical Disadvantage of Myocardial Infarction in the Canine Left Ventricle

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SUMMARY An isotropic, initially spherical, membrane model of the infarcted ventricle satisfactorily predicts ventricular function in the infarcted heart when compared to clinical information and available ventricular models of higher complexity. Computations based on finite element solutions of this membrane model yield end-diastolic and end-systolic pressure-volume curves, from which ventricular function curves are calculated, for infarcts of varying size and material properties. These computations indicate a progressive degradation of cardiac performance with increasing infarct size such that normal cardiac outputs can be maintained with Frank-Starling compensation and increased heart rate for acute infarcts no larger than 41% of the ventricular surface. The relationship between infarct stiffness and cardiac function is found to be complex and dependent on both infarct size and end-diastolic pressure, although moderately stiff subacute infarcts are associated with better function than extensible acute infarcts. Also, calculations of extensions and stresses suggest considerable disruption of the border zone contraction pattern, as well as elevated border zone systolic stresses.


IN THIS paper, a theoretical model is presented which predicts the strictly mechanical consequences of a myocardial infarction. In general, infarction may result in an array of mechanical, electrical, metabolic, vascular, and neural disturbances, but our purpose here is to consider only the effects of the abnormal ventricular wall motion which typically occurs in and around the infarcted region. In proceeding to this end, we develop the required analytical tools in three stages. First, we justify, for the present purposes, the use of the simplest model of the heart in diastole: an isotropic spherical membrane of uniform thickness. Next, we modify and extend this model to represent the heart in end-systole, a state in which pressure, volume, and activation are not changing with time. Finally, we extend the model further, in both diastole and end-systole, to include a noncontracting region of altered mechanical properties. This final model is used to predict the impairment of cardiac performance in acute, subacute, and chronic myocardial infarction. The model predicts the effect of infarct size in these states and further indicates derangements in the function of the border zone of viable tissue adjacent to the infarct.

Previous clinical, experimental, and theoretical investigations have not yet established the mechanical processes occurring in the infarcted ventricle. Certainly, taken as a group, patients with ventricular aneurysm (i.e., with abnormally contracting ventricular segments resulting from myocardial infarction) show decreased cardiac output, decreased ejection fraction, and evidence of Frank-Starling compensation by increased end-diastolic volumes and pressures. On the other hand, the role of infarct size and infarct stiffness is still not clear. Experiments with artificially constructed aneurysms in the canine ventricle (Tyson et al., 1962; Austø et al., 1962), although demonstrating that the extensibility of experimental aneurysms (sacs of canine bladder or aorta) was comparable to that of infarcted myocardium, nor established the mechanism by which aneurysm elasticity interferes with heart function. Similarly, previous mathematical models of the infarcted ventricle have been generally inconclusive, since most have been geometrical rather than true mechanical or physical models. For instance, the model presented in Vayo (1966) and Swan et al. (1972), which predicts a decrease in stroke volume in direct proportion to the area of noncontracting infarct, makes the nonphysical assumption that the motion in any ventricular segment is independent of the motion in adjacent segments, an assumption that implies that the infarcted and noninfarcted regions of the ventricle physically separate from one another during contraction. However, a recent model of the diastolic ventricle with a chronic apical aneurysm,
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developed by Janz and Waldron (1978), is a true mechanical model in which deformities and stresses throughout the ventricle are calculated.

The model presented in this paper is also a true mechanical model in which deformations and stresses, in systole as well as diastole, are calculated. The diastolic analysis, although simpler than that employed by Janz and Waldron, may be used to obtain substantially similar results. The systolic analysis of this paper, employing basic ideas from muscle mechanics, enables us to address the problem of how an aneurysm can be expected to change the pumping performance of the heart.

Methods

Experimental Evidence to be Included in the Model

The mechanical properties of heart muscle in diastole and systole have received extensive attention over the years. Our model will depend on the following pieces of experimental evidence.

1. Diastolic Stress-Extension Curves

Resting stress-extension tests in rabbit papillary muscle (Grimm et al., 1970), hamster right and left ventricle (Kane et al., 1976), human left ventricle (Mirskey and Parmley, 1973), rat myocardium (Janz et al., 1976), and canine left ventricle (Rabinowitz, 1978) have shown that all these tissues are well represented by a single empirical form relating the "true" Cauchy stress $\sigma$ (Force/deformed cross-sectional area) and the extension $\lambda$ (final length/initial length at zero stress). This form, also introduced in a different, but related, context by Hill (1969) and Ogden (1972), is:

$$\sigma = \mu_p(\lambda^{k_p} - \lambda^{-k_p/2})$$

where $\mu_p$ and $k_p$ are material constants, $k_p$ being termed the power law exponent. Determination of $\mu_p$ and $k_p$ in different species and by different methods has generally yielded similar values. In rabbit papillary muscle $k_p = 18$ and $\mu_p = 1.75$ mm Hg (Mirskey et al., 1976); and in the human ventricle $k_p = 14.69$ and $\mu_p = 0.64$ mm Hg (Janz and Waldron, 1978). We shall employ here similar data given for the normal canine ventricle by Rabinowitz (1978), where $k_p = 16$ and $\mu_p = 2.0$ mm Hg.

2. End-Systolic Pressure-Volume (P-V) Conditions

Monroe and French (1961) investigated an isolated canine heart preparation in which both preload (end-distolic volume) and afterload (compliance of the chamber receiving the stroke volume) were variable. Even though the conditions of preload and afterload were changed through a wide range, all end-ejection P-V points fell on a single straight line, the same line defined by all of the peak isovolumic P-V points. Thus, the end-systolic volume was dependent only on end-systolic pressure and not on the loading history.

Suga and co-workers have refined this concept further (Suga, 1971; Suga et al., 1973; Suga and Sagawa, 1974; Sagawa, 1978). In their investigations of isolated canine heart preparations (Fig. 1), they have demonstrated that one simple relationship,

$$P_s(t) = E(t)[V(t) - V_d],$$

describes the instantaneous systolic pressure $P_s(t)$ at a particular volume $V(t)$. Here $E(t)$ is the instantaneous P-V ratio and $V_d$ is an empirical constant termed the correction volume. This relation states simply that, at a given instant of time in the systolic period, ventricular pressure is dependent only on ventricular volume, regardless of the loading conditions. By contrast, the time function $E(t)$ was shown to be strongly dependent on the degree of myocardial contractility and thus responsive to inotropic agents. When the heart is maximally activated, i.e., when $E(t) = E_{\text{max}}$, the ventricular P-V relation defined by Equation 2, which Suga and Sagawa termed the end-systolic P-V relation, is found to be nearly identical with the peak isovolumic P-V curve. Further examination of the auxobaric P-V loops also reveals a virtual coincidence between end-systole and end-ejection, suggesting that at end-ejection the ventricle is in a

![Figure 1](https://example.com/figure1.png)

Figure 1: Pressure vs. volume in isolated canine left ventricle. Solid line (and points) show peak isovolumic pressure at several fixed volumes. Also shown are P-V loops for the same ventricle pumping against a range of fixed reservoir pressures. The conclusion is that the end-ejection states fall on the peak isovolumic P-V curve except at the highest pressures and volumes. From Sagawa (1978).
state of isovolumic contraction with zero contraction velocity. As discussed by Sagawa (1978), the superposition of end-systolic P-V curves for isovolumic and ejecting contractions is a matter of approximation. However, this approximation for the canine ventricle involves little error, so that for the present purposes, the $E_{\text{max}}$, peak isovolumic, end-systolic, and end-ejection P-V relations are regarded as identical. We will find this concept useful in suggesting a mathematical model for systole.

3. Shortening of Unloaded Muscle in Systole

Spotnitz et al. (1966) measured sarcomere lengths in intact canine left ventricles which had been rapidly fixed at various instants in the heart cycle. They observed that, although sarcomere lengths at diastolic zero pressure were about 1.87 $\mu$m in the inner layer of the ventricular wall, during systole the sarcomeres could contract to as little as 1.5 to 1.6 $\mu$m. Thus the ratio of uncontracted to contracted length $\lambda_c = 1.87/1.6 = 1.17$, or in the extreme case, $\lambda_c = 1.87/1.5 = 1.25$. A determination of $\lambda_c$ can also be made from the rabbit papillary muscle data of Grimm et al. (1970) which shows $\lambda_c = 1.19$. Taking a value midway between the highest and lowest estimates for $\lambda_c$, we may estimate that $\lambda_c = 1.18$ for a normal contraction. This will be an important parameter for the systolic model.

The Diastolic Model

In this section we will develop a simple model of the diastolic ventricle which directly relates the elastic properties of the myocardium to the diastolic filling curve. By comparing a more complex model with this simpler one, we will be able to demonstrate both the limitations and the usefulness of the simplification. We begin by representing the normal ventricle as a uniform, isotropic, elastic, spherical membrane. Although this geometry is unrealistic, it does allow for uniformity of stress and extension over the sphere's surface. This is consistent with the fact that the architecture of the ventricle is such that variations in ventricular curvature and wall thickness compensate one another so that stress levels tend to be uniform over the ventricular surface (Role et al., 1978). Extensions, and probably stresses, are not uniform across the true ventricular wall, however, as assumed by a membrane model, since myocardial incompressibility requires endocardial extensions to exceed epicardial extensions as the ventricle inflates. We will return to this limitation subsequently.

To be consistent with the observed power-law relation between stress and extension in diastolic myocardium, we first represent such a nonlinear isotropic elastic material by its equivalent strain energy function, which takes the form

$$\Phi = \mu_p \left( \lambda_3^{k_p} + \lambda_1^{k_p} + \lambda_2^{k_p} - 3 \right)$$

(3)

The strain energy $\Phi$ may be understood as a function which relates the stored internal energy in a unit volume of material to the deformation (strain) undergone by that material. Here $\lambda_1$, $\lambda_2$, and $\lambda_3$ are the principal extensions (deformed length/undeformed length), with $\lambda_3 = 1/\lambda_1\lambda_2$, since the material is incompressible. When a material has a strain energy function given by Equation 3, the relation between stress and extension for uniaxial tension is given by Equation 1. From Laplace's law, the relation between pressure and extension in a spherical elastic membrane of uniform thickness with a strain energy function of the form of Equation 3 is (given in Ogden, 1972):

$$P = 2\mu_p h_0 \left( \lambda_3^{k_p-3} - \lambda^{2k_p-3} \right)$$

(4)

where $h_0$ and $R_0$ are a reference (initial) wall thickness and internal radius, respectively, and $\lambda$ is the extension in the membrane surface measured from this reference state. (For a sphere, $\lambda = \lambda_1 = \lambda_2$.)

Since intraventricular volume $V$ is related to the extensions by $V = V_0\lambda^3$, where $V_0$ is the reference, or initial, volume, the pressure-volume relation follows directly from Equation 4:

$$P = 2\mu_p h_0 \left( \frac{k_p-3}{V^3} - \frac{2k_p-3}{V^3} \right)$$

(5)

where $V$ is the normalized volume $V/V_0$.

Because $k_p$ for diastolic myocardium is typically in the neighborhood of $k_p = 16$, the second term in the above expression becomes insignificant for inflations above about 15% by volume, whereas the typical diastolic filling curve entails inflations of at least 100%. Hence, over most of diastolic filling, the following relation holds:

$$P_d = 2\mu_p h_0 \left( \frac{k_p}{V^3} - \frac{3k_p}{V^3} \right).$$

(6)

Therefore, if pressure and volume are plotted against one another on log-log paper, the above expression implies that the slope of the plot at intermediate and higher filling pressures will be equal to $(k_p - 3)/3$. Indeed, Laird (1976) has reported that log-log plots of diastolic pressure-volume data do yield straight lines. Working from the human data of Fester and Samet (1974), Laird found the log-log slope $m = 3.95$. Hence $k_p$ may be calculated for the human ventricle by $k_p = 3m + 3 = 14.85$. It is worth noting that Janz and Waldron (1978), using an entirely different procedure based on a model which fully accounts for variations through the thickness, applied a curve-fitting procedure to the same Fester and Samet data to obtain $k_p = 14.69$. Hence Equation 6, which is based on the spherical membrane model, gives substantially the same result as a procedure based on a more elaborate model for determining $k_p$ from P-V measurements.

The preceding discussion indicates the ease with which the P-V relation can be derived from the
membrane properties. We will next investigate the appropriateness of using the membrane or thin-walled model to characterize the thick-walled ventricle by comparing the membrane model with the thick-walled model proposed by Janz et al. (1976). The latter model employs essentially the same power-law description of diastolic myocardial properties, and assumes the ventricle to be an isotropic sphere. The difference between the two models is that the Janz et al. model fully calculates the stress distribution across the wall. The two models approach one another in the limit of very small wall thicknesses. Here, however, and subsequently we will adopt the value of h_o/R_o = 0.81 estimated by Rabinowitz (1978).

The relationship between pressure and volume predicted by the thick-walled model, converting notation used in Janz et al. to that used in this paper, is

\[ P = -2 \int_{V_o/V_w}^{V_e/V_w} \frac{\mu_o(\lambda^{2k_p} - \lambda^{k_p})}{\lambda(1 - \lambda^2)} \, d\lambda \]  

(7)

where \( V_w \) is wall volume.

For the case of \( h_o/R_o = 0.81, k_p = 16, \) the membrane model overestimates the stiffness of the ventricle. In fact, when the curves are compared numerically by least-squares fitting, it is found that the pressure predicted by the membrane model must be multiplied by 0.253 in order to be consistent with the thick-walled model.

We may restate this result as follows: that a sphere of thickness \( h \), when analyzed by the thick walled model, gives the same P-V curve as a sphere of thickness 0.253\( h \) when analyzed by the membrane method. Furthermore, the relationship between endocardial extension and pressure, as well as the relation between volume and average wall stress, will be identical in the two models as a consequence of the identity of the P-V curves. The value 0.253 does vary somewhat with the value of \( k_p \), but since we will generally restrict future discussion to values of \( k_p \) close to 16, we will assume the factor fixed at 0.253 from now on. In summary, we have described a method by which we may characterize a membrane in such a way that it behaves in the same manner as the thick-walled myocardium. This equivalent membrane is described by the same elastic constants \( \mu_o \) and \( k_p \) as the true myocardium, but has only one-quarter the thickness. This characterization of the equivalent membrane will be useful in the succeeding analysis. Moreover, the validity of this method will be tested in a later section. One immediate application of the equivalent membrane representation is to Equation 5; further substituting for the material parameters gives the P-V relation

\[ P(\text{mm Hg}) = 0.82(\lambda^{13/3} - \lambda^{-55/3}). \]  

(8)

The Systolic Model

In this section we will develop a membrane description of the systolic ventricle. As in the previous diastolic model, the ability to characterize stresses through the wall will be sacrificed in order to develop a simple relationship between systolic pressure and volume. Moreover, the complexity of the model will be reduced to a level adequate to calculate ventricular stroke volume, but not adequate to determine such dynamic quantities as the time course of rising ventricular pressure. The model we begin here attempts to represent the ventricle only at end-ejection, when the contraction velocity is zero.

We now proceed, as in the diastolic case, to derive the end-systolic pressure-volume relationship from the myocardial stress-extension relation. In peak isometric contraction, developed tension is essentially linear with extension within a certain range (Grimm et al., 1970). In terms of the power-law strain energy function, such linear behavior can be described by a single-power strain energy function with \( k_p = 2 \). Since total systolic tension is the sum of resting tension and developed tension, systolic myocardium can now be described by a two-component strain energy function with two sets of power-law terms, those due to \( (k_p = 16) \) resting tension and in addition those due to \( (k_p = 2) \) developed tension. There is, however, an added complication. Resting tension and developed tension do not both fall to zero at the same length; that is, in systole, myocardium assumes a new rest length or zero-stress configuration. If this change in rest length is taken into account and the determination of constants is made (details are given in Appendix I), then the P-V relation at end-systole for the normal ventricle is given by

\[ P_s, \text{total (mm Hg)} = \begin{cases} 0.82(\lambda^{13/3} - \lambda^{-55/3}) & \text{for } \lambda \geq 1 \\ + 246(1.39\lambda^{-1/3} - 0.52\lambda^{-7/3}) & \text{for } \lambda < 1 \end{cases} \]

(9)

Figure 2 shows Equations 8 and 9 plotted as functions of normalized volume. Also shown are two schematic P-V loops for a ventricle assumed to be pumping into a constant-pressure reservoir \( P_s \), representing the aorta. Starting from the end-diastolic point A, pressure rises in isovolumic systole to the onset of ejection at point B. Ejection occurs between points B and C against constant aortic pressure. Isovolumic relaxation proceeds from C to D, and then diastolic filling from D to A completes the cycle.

The stroke volume is the difference between \( V_s \) and \( V_c \), the volumes at end-distole and end-ejection, respectively. As shown schematically by the larger loop, increasing the filling pressure while keeping \( P_s \) fixed increases the stroke volume.

It is perhaps surprising that stroke volume can be calculated in such a simple fashion without apparent consideration of such important matters as electrical propagation through the myocardium, the delay of full muscle activation, the muscle force-velocity relation, and the finite period of myocardial
edly for different degrees of diastolic filling to obtain the diastolic P-V curve, and for different values of end-systolic volume to obtain the systolic P-V curve.

activation. The critical fact is Suga's (1971-1974) observation that, in the healthy heart, the end-ejection points fall on the isovolumic P-V relation (point 2 of the experimental evidence). The conclusion follows that, in the healthy heart, myocardial activation and rates of contraction are sufficiently rapid to allow full ejection within the time limits imposed by the finite activation time. The extension of this assumption to the infarcted heart, as in the next section, must be regarded for the present as conjectural, since it is unproven by any direct experimental evidence.

Model of the Infarcted Heart

In the two previous sections the use of the membrane model to describe the diastolic and systolic P-V relations was demonstrated. An extension of the membrane model to the infarcted ventricle is illustrated schematically in Figure 3. Here, the region below the horizontal line in Figure 3, a and b, is healthy myocardium whose membrane description has been given previously in Equations 8 and 9. The cap-shaped region above the line represents the infarct and therefore has no contractile function. A membrane description of this region is again used, with membrane characteristics consistent with the $\mu_p$ and $k_p$ of infarcted myocardium. Hence, for the purposes of computation, the ventricle is considered as an inhomogeneous, initially spherical membrane, such as that illustrated in Figure 3a. The membrane does not remain spherical; during systole the normal region contracts, causing the infarcted region to bulge, as seen in Figure 3b.

To calculate the deformation in such a ventricle, and hence its P-V relations, it was first necessary to formulate a system of equations describing the physical and geometrical laws governing the deformations. These equations were then solved repeat-
The system of equations employed here, summarized in Appendix II, is based on the membrane formulation for nonlinear elastic membranes (see, for example, Green and Adkins, 1970). The specific, numerically convenient form of these equations is described fully in Needleman (1977). In practice, the equations were solved by a finite element numerical technique on a digital computer. Due to the highly nonlinear nature of this problem, a rather complex solution scheme was employed to ensure both convergence and accuracy. A complete discussion of the relevant numerical methods is given in Needleman (1977) and Rabinowitz (1978).

Here, it is sufficient to note that, in addition to certain internal consistency checks, the finite element method was tested for accuracy against a case in which an exact solution was obtainable, that of the normal ventricle (Equation 9). Errors in wall stress were found to be generally less than 0.1% and no more than 0.28%. In addition, the predicted pressure was exact to 0.001 mm Hg.

Furthermore, Bogen (1977) and Bogen and McMahon (1979) employed a shooting-point method to solve an equivalent, but differently formulated, set of membrane equations. The shooting-point and finite-element methods were used to calculate the end-systolic pressure of a ventricle for an end-diastolic volume of 1.224 when the ventricle included an acute infarct making up 25% of the total wall area. The pressures predicted by the two methods differed by less than 5%. This comparison provided a check on the accuracy of the methods of solution.

Results

Comparison with Janz and Waldron (1978) Model

Janz and Waldron’s (1978) model of the diastolic human heart, including an aneurysmal region, is shown in the inset in Figure 4. This is an axisymmetric finite element representation including realistic geometry and variable wall thickness. The wall material, assumed homogeneous and isotropic, is described by the stress-extension relation Equation 1 with $k_p = 14.69$ and $\mu_p = 0.64$ mm Hg (assuming incompressibility). The broken lines in Figure 4 show Janz and Waldron’s results for the diastolic P-V relation in both the normal ventricle and a ventricle including a fibrous aneurysm making up 41.2% of the wall volume. The lower solid line shows the predictions of our membrane diastolic model (Equation 5) when given the same $\mu_p$ and $k_p$ used by Janz and Waldron, again using a value of $h_0/R_0 = 0.81$ and the scaling factor of 0.253 employed by the membrane model. The upper solid line shows the prediction of our model when a large infarct (41.3%) of wall area is included that has the same parameters specified by Janz and Waldron for the fibrous aneurysm ($\mu_p = 118$, $k_p = 136$). The close agreement between the upper solid and broken lines supports the conclusion that the increased diastolic stiffness due to the presence of an infarct is as well predicted by the initially spherical membrane model as by the Janz and Waldron model.

Ventricular Function in Myocardial Infarction

The ventricular model described above was employed to investigate the effects of varying infarct size and infarct stiffness on cardiac function. These results are given in Figures 5–8 and Table 1. In particular, the role of progressive infarct stiffness in the recovery from myocardial infarction was assessed by applying the model to infarcts with mechanical properties representative of the various stages of myocardial healing and scarification.

Although the mechanical properties of infarcted myocardium may evolve continuously for months in the post-myocardial infarction period, for the purposes of discussion we have identified four particular stages for consideration. The first stage, which is somewhat hypothetical, is termed immediate infarction, and designates the myocardial state in which all contractile function is lost, but in which the passive, or diastolic, length-tension relation is unaltered (i.e., normal). Although it is not clear that all contractile function in the infarct is extinguished before changes in the passive length-tension relation actually begin to occur, the immediate infarction state represents a convenient base line from which to evaluate the effects of changing infarct stiffness.

The second stage is termed acute infarction and designates the myocardial state several hours post-infarction. The accumulated experimental evidence concerning myocardial stiffness in this period indi-
indicates that the mechanical properties are unstable and change over the course of hours, thus making exact specification of mechanical properties difficult. However, it is clear that the infarct stiffness several hours post-infarction is significantly less than infarct stiffness 5–6 hours post-infarction (Pirzada et al., 1976). Thus we have employed values of $\mu_p$ and $k_p$ determined in the acutely (2–4 hours) infarcted canine heart as representative. These values were obtained from Rabinowitz (1978), updated to include data from 14 rather than 9 infarcted canine ventricles.

The third stage is termed subacute infarction and designates the myocardial state 1 week post-infarction. This is a stage of intermediate infarct stiffness, in which edema, cellular infiltration, and perhaps other events have caused the infarct to be less extensible than in the acute infarction stage. Although healing in the canine ventricle progresses more rapidly than in the human ventricle, extensive collagen deposition and fibrosis are not yet dominant features at this point (Mallory et al., 1939; Karsner and Dwyer, 1916). Values of $\mu_p$ and $k_p$ descriptive of subacutely infarcted myocardium were obtained from the experimental canine data of Rabinowitz (1978). (updated).

The final stage is termed chronic infarction and designates the myocardial state months post-infarction in which extensive fibrosis and scarification have occurred. Such an infarct, when complete, with no residual muscle, is virtually inextensible. Values of $\mu_p$ and $k_p$ for the chronic infarct are those obtained from Janz and Waldron's (1978) reanalysis of the human aneurysm data of Parmley et al. (1973). Values of $\mu_p$ and $k_p$ for the four different types of infarct are listed in Table 2.

Using values of $\mu_p$ and $k_p$ appropriate for immediate, acute, subacute, and chronic infarcts, the model was first used to calculate the diastolic and end-systolic P-V relations resulting from such infarcts. Ventricular function curves were then obtained by fixing the end-ejection pressure at 100 mm Hg and by following the procedure outlined in Figure 2 for determining stroke volume as a function of filling pressure. Figure 5 contains the results of these calculations when three sizes of infarct were considered: small infarct = 14.6% of ventricular surface, or diastolic half-angle of 45° ($\theta$ in Fig. 3a); moderate infarct = 25% of surface, angle of 60°; and large infarct = 41.3% of surface, angle of 80°.

Effect of Infarct Size

The relation between infarct size and the depression of the ventricular function curves seen in Figure 5 is clarified in Figure 6 in which stroke volume is plotted directly as a function of infarct size. This figure shows that, when end-diastolic filling pressure is set at 12 mm Hg, the percentage decrease in stroke volume exceeds the percentage of myocardial infarction: that is, for the cases considered here, a 41% infarct reduces function to a level at least as low as 48% of normal and to as little as 28% of normal, depending on the infarct stiffness.

Since the reduction in cardiac function secondary to myocardial infarction ordinarily would be attended by a number of compensating mechanisms operating to restore cardiac output toward normal levels, it is useful here to restrict the discussion and to examine the predicted cardiac output in the infarcted heart when just one effect, the Frank-Starling mechanism, acts as the sole booster of cardiac performance. Figure 5 indicates that larger stroke volumes are obtainable from both the normal and the infarcted heart if diastolic filling pressure is allowed to rise. Maximal stroke volume is achieved in all cases at an end-diastolic pressure of approximately 24 mm Hg, since greater pressures promote the formation of pulmonary edema. A comparison of these maximal stroke volumes in the normal and infarcted heart is given in Figure 7. Here cardiac reserve is defined as the ratio of maximal stroke volume (i.e., at EDP = 24 mm Hg) to nominal “normal” stroke volume in the noninfarcted heart (i.e., at EDP = 8 mm Hg). Thus a cardiac reserve less than 1.0 corresponds to subnormal cardiac performance in spite of maximal diastolic filling. From Figure 7 it is thus apparent that even with Frank-Starling compensation, subnormal cardiac performance will result from infarcts ranging in size from 23% to 29%, depending on infarct stiffness. This of course presumes a constant heart rate, a constant level of ventricular inotropy, and an absence of myocardial hypertrophy.

Compensation by Increased Heart Rate

Since the above results predict the normal cardiac outputs can be maintained with larger infarcts only if other compensating mechanisms are permitted to operate, let us now consider the effect of increased heart rate. If heart rate increases by a factor of 50%, say from 80 to 120 beats/min, and stroke volume remains constant, then cardiac output is similarly increased by 50%. This is indicated in Figure 7, in which cardiac reserve is given for the normal heart rate according to the lefthand vertical scale, but is given for the increased heart rate according to the righthand scale. Here it can be seen that it is possible to maintain normal stroke volume for acute infarcts as large as 41% if both Frank-Starling compensation and tachycardia occur. The ability to sustain still larger infarcts, therefore, would depend upon the development of greater tachycardia, inotropic stimulation of the myocardium, or other compensations.

This result is of some clinical interest. Clinical studies by Page et al. (1971) and Alonso et al. (1973) have shown that, in general, cardiogenic shock in acute myocardial infarction is associated with infarcts with a minimum size of 35–40%. These studies have also indicated that it is the total amount of
Infarct Stiffness

Although the relation between infarct size and ventricular function is fairly clear, with a steady diminution in cardiac performance with larger and larger infarcts of a given stiffness, the relation between infarct stiffness and cardiac performance is not so simply stated. Reference to Figure 5 reveals that the ventricular function curves for infarcts of different stiffness cross over one another, so that the relative levels of ventricular performance de-

![Figure 5](image-url)  
**Figure 5** Ventricular function curves for three sizes of infarct. (a) Infarcts comprising 14.6% of ventricular surface ($\theta = 45^\circ$); (b) 25% infarcts ($\theta = 60^\circ$); (c) 41.3% infarcts ($\theta = 80^\circ$).

![Figure 6](image-url)  
**Figure 6** Ventricular function vs. infarct size. Here stroke volume in the infarcted heart, with EDP = 12 mm Hg, is compared with the stroke volume in the noninfarcted heart at the same filling pressure.

![Figure 7](image-url)  
**Figure 7** Cardiac reserve vs. infarct size. Here, cardiac reserve is defined as the (maximal) stroke volume obtained with a filling pressure of 24 mm Hg, divided by the stroke volume obtained in a noninfarcted ventricle with a normal filling pressure of 8 mm Hg. Under this definition, an infarcted ventricle with a cardiac reserve of 1.0 is just able to produce the normal cardiac output when Frank-Starling compensation is maximal, i.e., when filling pressure is 24 mm Hg. The noninfarcted heart is able to increase stroke volume by a factor of 1.5 as filling pressure rises from 8 to 24 mm Hg. Because increased heart rate increases cardiac output for a given stroke volume, increased heart rate also increases cardiac reserve. Accordingly, the lefthand scale indicates the cardiac reserve for normal heart rate; the righthand scale indicates the cardiac reserve for a 50% increase in heart rate.
pend on infarct stiffness, infarct size, and end-diastolic pressure. For instance, Figure 5 shows that the ventricular function curve belonging to the 15% chronic infarct heart is depressed relative to that of the 15% acute infarct heart for filling pressures greater than 12 mm Hg; however, the ventricular function curve belonging to the 41% chronic infarct heart always lies above that of the 41% acute infarct heart.

The complex relationship between infarct stiffness and cardiac performance is more easily understood by looking directly at the end-systolic and end-diastolic pressure-volume curves in the normal and infarcted heart. Figure 8 gives these curves for the large 41% infarct. Here it can be seen that the end-systolic P-V curves are shifted further to the right with more extensible infarcts, with the chronic infarct being the least shifted and the immediate infarct being the most shifted. This rightward shift implies a greater end-systolic volume for a given end-systolic pressure, and hence a smaller stroke volume for a given end-diastolic volume. Figure 8 also shows that the stiffer infarcts make for less compliant diastolic ventricles, so that a higher filling pressure is necessary to achieve a given end-diastolic volume. Thus the chronically infarcted ventricle is significantly less distensible than the immediately or acutely infarcted ventricle.

Ventricular function depends on both systolic and diastolic events. In the case of the chronic infarct, systolic function is spared relative to the systolic depression seen with more extensible infarcts. However, diastolic filling is so severely compromised that Frank-Starling compensation becomes limited. Maximal diastolic filling in the heart with the large chronic infarct is only about half normal. In contrast to the chronically infarcted ventricle, where the defect is primarily, but not totally, diastolic, the primary defect in the acutely infarcted ventricle is systolic; Figure 8 shows a large rightward shift of the end-systolic P-V relation but a relatively small change in the diastolic P-V relation.

The subacute infarct, with stiffness intermediate to that of the acute and chronic infarcts, similarly shows intermediate end-systolic and end-diastolic P-V relations. The net effect, at high filling pressures, is shown in Figures 5 and 7 in which it is seen that the large subacute infarct is responsible for considerably less cardiac depression than either the acute or chronic infarcts.

Summary of Computations

The computed end-diastolic, end-systolic, and ventricular function curves for the various infarcted hearts are summarized in Table 1. In this table, each computed diastolic filling curve has been compared to the normal filling curve, and the relative compliance of the two curves has been determined by least-squares fitting to the normal curve. Hence a P-V curve with a relative compliance value of 2.0 will have a diastolic pressure one-half that of the normal noninfarcted ventricle at the same volume. Systolic function is simply summarized by the computed values of end-systolic volume in each case, with the usual assumption of an end-systolic pressure of 100 mm Hg. Ventricular function curves are indicated in brief form by recording the calculated stroke volume for two values of end-diastolic pressure: EDP = 12 mm Hg and EDP = 24 mm Hg.

Table 1 also includes summary data for hypothetical infarcts of different material properties. Hypothetical infarcts whose material constant $\mu_p$ is one-half, twice, or eight times the value of $\mu_p$ in the immediate infarct are termed "1/2 $\mu_p" "2 \mu_p" and "8 \mu_p" respectively. Hypothetical infarcts whose material constant $k_p$ is higher or lower than the value of $k_p$ in the immediate infarct are also considered. For the immediate infarct, $k_p = 16$; hence, the hypothetical infarct with $k_p = 20$ is termed "high $k_p" and one with $k_p = 12$ is termed "low $k_p".

Here, as before, stiffer infarcts are associated with less compliant diastolic ventricles and better systolic function.

Steepness of the Systolic P-V Curve

A few additional comments are in order concerning the end-systolic P-V relations. The first is that, besides shifting rightward with increasing infarct extensibility, the end-systolic P-V relation becomes less steep (i.e., $\Delta P/\Delta V$ is smaller); in fact, the P-V relation for the heart with the large acute infarct is approximately one-third as steep as that for the normal or chronically infarcted heart. Thus for a given decrease in end-systolic pressure, the acutely infarcted heart would see a decrease in end-systolic volume three times greater than that seen in the normal or chronically infarcted heart. For this rea-
son, afterload reduction might be expected to have a greater salutary effect in the acutely infarcted heart than in the normal or chronically infarcted heart.

Role of the Border Region in the P-V Relations

The second comment to be made about the end-systolic P-V relation concerns the reason why these curves are shifted more with increasing infarct extensibility. Previous models of dyskinetic ventricular segments (Swan et al., 1972) have argued that the deleterious effect of the extensible infarct is due to actual transfer of blood volume from the remainder of the contracting ventricle into the bulging aneurysmal volume. However, this may not be the entire story. Examination of the distribution of sarcomere extensions predicted by the model of the infarcted heart indicates that the presence of an extensible infarct significantly interferes with normal contraction in the noninfarcted myocardium.

Figure 9a shows the distribution of systolic extensions in the ventricle with a large acute infarct. Here the circumferential extensions adjacent to the infarct are considerably greater than normal and remain elevated throughout a large portion of the myocardium. This effect may be understood intuitively as the tendency of the ballooning infarct to induce a similar but attenuated ballooning in the surrounding viable region.

Calculation of extensions in the chronically infarcted heart, Figure 10, shows that, in this case, again, the presence of the infarct compromises the contraction pattern in the adjacent noninfarcted myocardium in both diastole and systole. In diastole, the stiff infarct constrains the motion of the adjacent myocardium so that the circumferential extensions become subnormal (Fig. 10a). In systole, the stiff infarct again constrains the motion of the adjacent myocardium, but in this instance prevents circumferential contraction, leaving greater than normal extensions at end-systole (Fig. 10b).

Stress in the Border Region

Some comment should also be made here about the systolic stress levels in the adjacent myocardium. Because of the myocardial length-tension relation, greater than normal myocardial extensions in systole would necessarily result in greater than normal systolic stresses, particularly surrounding the extensible infarct, where the largest extensions are observed. Figure 9b shows the calculated stress distribution in the case of the large acute infarct; here the circumferential stress immediately adjacent to the infarct is 3.7 times greater than the stress in the myocardium far away from the infarct. The level of stress amplification was found to be fairly independent of infarct size for infarcts from 15 to 41%, but diminished with increasing infarct stiffness. Thus the stress amplification for immediate, acute, subacute, and chronic infarcts, respectively, was 4.0, 3.7, 3.1, and 2.0.

Stress and extension, however, are local quantities and may not be as well represented by the membrane model as is the overall ventricular stiffness. Generally, in solid mechanics, membrane solutions can give accurate estimates when compared to more complicated full-thickness solutions, except close to edges or discontinuities. Away from stress discontinuities, the membrane solution can be expected to give a reasonable estimate of the thickness averaged stresses and extensions. Hence, in the present example, it is also reasonable to expect that circumferential extensions, and hence systolic stresses, are greater than normal surrounding an extensible infarct, although the exact magnitudes of
lies in the fact that after an acute myocardial infarction there may be a border zone of marginally perfused, ischemic, but noninfarcted tissue surrounding an extensible aneurysm. This border zone is a vital clinical concern since it has the potential of itself progressing toward infarction.

It is generally believed that infarction can occur when myocardial oxygen demand exceeds supply. Therefore, infarction in the border zone becomes more likely when myocardial oxygen demand in that region is increased. Since oxygen consumption is related directly to myocardial stress (Maroko and Braunwald, 1973), stress enhancement in the border zone creates the possibility of infarction in the border zone when the elevated oxygen demand exceeds the marginal oxygen supply. It is worth noting that, although this possibility appears to be much greater for the immediate and acute infarcts, it also remains for the chronic infarct.

In conclusion, the membrane model of the infarcted ventricle represents a first step in the mechanical analysis of myocardial infarction. In spite of its simplifying assumptions it, nevertheless, reveals the dependence of diastolic and end-systolic pressure-volume curves on infarct size and stiffness. Calculation of ventricular function curves underscores the concept that depression in ventricular function may result from either unfavorable systolic or diastolic function, or both. Thus one cannot conclude that the extensible dyskinetic infarct must inevitably be associated with poorer cardiac performance than the stiff akinetic infarct. The model does predict a substantial increase in performance with moderate increases in infarct stiffness, as in the case of the subacute infarct, but also indicates that further increases in infarct stiffness, as in the case of the chronic infarct, may be so detrimental to diastolic function as to counterbalance any further improvements in systolic function. Although these results, of course, must be tempered by recognition of the multitude of physiological phenomena occurring in the post-infarction period, it is still interesting to note areas in which the model agrees with clinical impressions. One such area is the improvement in cardiac performance in the subacute (first-week) period. Since the model indicates that the magnitude of this improvement is dependent on infarct size and end-diastolic pressure, the relative contribution of this effect would be expected to vary from patient to patient. Another area of agreement is the figure of 40% infarction as a rough threshold for cardiogenic shock. The model indicates that Frank-Starling compensation and a 50% increase in heart rate can maintain normal cardiac outputs for acute infarcts up to 41%. According to the model, cardiogenic shock could be avoided in the face of larger infarcts, but only by increased ventricular inotropy and further increases in heart rate.

Other intriguing aspects of the model are the predictions of extension and stress in the noninfarcted and infarcted regions cannot be determined reliably without employing a more complicated model.

The importance of systolic stress amplification lies in the fact that after an acute myocardial infarction there may be a border zone of marginally perfused, ischemic, but noninfarcted tissue surrounding an extensible aneurysm. This border zone is a vital clinical concern since it has the potential of itself progressing toward infarction.

It is generally believed that infarction can occur when myocardial oxygen demand exceeds supply. Therefore, infarction in the border zone becomes more likely when myocardial oxygen demand in that region is increased. Since oxygen consumption...
TABLE 1 Summary of Computations

<table>
<thead>
<tr>
<th>Infarct size</th>
<th>Infarct type</th>
<th>( \mu_p )</th>
<th>( k_p )</th>
<th>( \mu_s )</th>
<th>( k_s )</th>
<th>Stroke volume @EDP = 12 mm Hg</th>
<th>Stroke volume @EDP = 24 mm Hg</th>
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</table>

Diastolic, systolic, and aggregative ventricular function is summarized for three infarct sizes and nine infarct stiffnesses, as well as for the noninfarcted ventricle. Diastolic function is indicated by relative diastolic compliance, a comparison with the normal diastolic filling curve, which is defined as the fraction by which the P-V curve of the noninfarcted ventricle must be scaled along the pressure axis to achieve a least-squares fit with the P-V curve of the infarcted ventricle. Systolic function is indicated by end-systolic volume. Ventricular function is evaluated by indicating stroke volume at two values of end-diastolic pressure: EDP = 12 mm Hg and 24 mm Hg.

noninfarcted myocardium, and it implies that the mechanical disadvantage of the extensible infarct is not only a matter of blood volume shift into the aneurysmal region, but a matter also of mechanically induced contraction derangement in adjacent noninfarcted myocardium.

Appendix I

End-systolic pressure is a consequence of ventricular wall stress, to which there are two components—that due to myocardial resting tension and that due to developed tension. The pressure resulting from resting tension is identical to diastolic pressure (from Eq. 8 substituting for \( v = \lambda^2 \)),

\[
P_d = 0.82 (\lambda_{12}^{-3} - \lambda_{23}^{-3}).
\]  

(I.1)

The pressure resulting from developed tension can similarly be described by the relation

\[
P_s = 2 \mu_p h_s (\lambda_s^{-3} - \lambda_s^{-2k_s^{-3}}), \quad k_s = 2
\]  

(II.2)

where \( \lambda_s \) is the extension with respect to the systolic zero-stress configuration (the new initial configuration resulting from the systolic change in myocardial rest length), \( h_s \) and \( R_s \) are the ventricular thickness and radius of this new reference configuration, respectively, and \( \mu_s \) is the value of the elastic coefficient \( \mu_p \) appropriate for systole. However, it is desirable to give an expression for \( P_s \) in terms of \( \lambda_s \), the extension with respect to the systolic zero-stress configuration. This is accomplished by substituting for \( \lambda_s = \lambda_s \). Here, \( \lambda_s \) is merely the extension with respect to the systolic rest configuration when the ventricle is in the diastolic rest configuration.

<table>
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<th>( k_p )</th>
<th>( \mu_s )</th>
<th>( k_s )</th>
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<td>136</td>
<td>-</td>
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</tbody>
</table>

Parameters for immediate, acute, and subacute infarcts are from Rabinowitz (1978). These values were obtained from the epicardial measurements of normal and infarcted canine ventricles reported in Rabinowitz et al. (1978) and Rabinowitz (1978) (updated), after fitting the data to the thick-walled ventricular model described by Equation 7 here, and in more detail in Janz et al. (1976) and Rabinowitz (1978). Parameters for the chronic infarct are from Janz and Waldron (1978).
ration, i.e., \( \lambda_c = R_o/R_s \). In addition, incompressibility implies \( 4\pi R_o h_o = 4\pi R_s h_s \), and thus \( h_s = h_o \lambda_c^2 \). Substitution for \( \lambda_s, h_s \) and \( R_s \) then gives
\[
P_s = 2\mu h_o \left[ \lambda_c^2 \lambda - \lambda_c^{-2} \right], \quad k_s = 2 \quad (1.3)
\]
or
\[
P_s = 2\mu h_o \left[ \lambda_c^2 \lambda - \lambda_c^{-4} \lambda^{-1} \right]. \quad (1.4)
\]
As explained in the text, \( \lambda_s \) is taken to be 1.18, and \( h_o/R_s \) is taken to be 0.81. The value of \( (2\mu h_o/R_o) \) is chosen so as to make the maximum developed pressure equal to 250 mm Hg, a typical isovolumic peak pressure. This requires that \( \mu_s = 151.9 \) mm Hg.

Now the expression for total pressure \( P_s \) may be written directly with the additional provision that \( P_d \) must be equal to zero for \( \lambda \leq 1 \), since resting tension is also approximately zero for \( \lambda \leq 1 \); therefore
\[
P_s (\text{mmHg}) = 0.82 (\lambda^{12} - \lambda^{-35}) + 246(1.39/\lambda - 0.52\lambda^{-7}) \quad \text{for} \quad \lambda \geq 1
\]
\[
= 246(1.39/\lambda - 0.52\lambda^{-7}) \quad \text{for} \quad \lambda < 1.
\]

### Appendix II

The membrane theory equations employed in the present analysis are approximations to the exact equations of nonlinear elasticity which: (1) neglect variations in extensions and stresses through the thickness and (2) neglect the influence of the normal stress acting through the thickness on the constitutive response of the material. For a sufficiently thin-walled shell, the membrane theory equations are the appropriate limiting case of the exact equations. The applicability of the membrane theory equations to the thick-walled ventricle is by no means obvious. However, as illustrated in Figure 4, it is clear that membrane theory is capable of providing a good representation of the P-V curve of a thick-walled ventricle model. When membrane theory is applied to a thick-walled body, assumption (1) essentially means that the membrane theory quantities are properly interpreted as representing weighted thickness averages. It is perhaps somewhat surprising that assumption (2) does not induce larger errors in the pressure-volume curves than are depicted in Figure 4, since the normal stress acting through the thickness necessarily equals the pressure at the linear surface of the ventricle, which is not negligible compared to the other stresses for a thick-walled body. However, for a highly nonlinear material, there is a rapid decay of the normal stress acting through the thickness; see e.g., Rabinowitz (1978, Fig. IV-2). Hence, although near the inner wall of the ventricle the normal stress acting through the thickness is as large as the averaged wall stresses used in membrane theory, over most of the ventricle wall the normal stress acting through the thickness is much smaller.

The variables of the problem are the three principal extensions \( \lambda_1, \lambda_2, \) and \( \lambda_3 \), describing strain in the longitudinal, circumferential, and thickness directions, and the principal curvature \( k_1 \) and \( k_2 \) describing longitudinal and circumferential curvature in the deformed membrane. A representative point \( G \) in Figure 3a is located a distance \( \eta \) along the surface from the pole, or \( \rho \) units from the axis. This representative point has moved to \( G' \) on the deformed membrane of Figure 3b, where the longitudinal position coordinate has become \( \xi \) and the radial distance to the axis has become \( r \).

For completeness, we list here the governing equations for axisymmetric membranes subject to pressure loading [see, e.g., Green and Adkins (1970)]. There are two nontrivial equilibrium equations
\[
\frac{d}{d\xi} (\mathbf{T}_1 r) = \frac{\mathbf{T}_2}{r} \frac{dr}{d\xi} \quad (II.1)
\]
\[
k_1 T_1 + k_2 T_2 = P \quad (II.2)
\]
where \( T_1 \) and \( T_2 \) are the stress resultants in the longitudinal and circumferential directions, respectively, and \( P \) is the normal pressure difference acting across the membrane.

The curvatures \( k_1 \) and \( k_2 \) are given by
\[
k_1 = -\left[ 1 - \left( \frac{dr}{d\xi} \right)^2 \right]^{-1/2} \frac{d^2 r}{d\xi^2} \quad (II.3)
\]
\[
k_2 = \frac{1}{r} \left[ 1 - \left( \frac{dr}{d\xi} \right)^2 \right]^{-1/2} \quad (II.4)
\]
Deformation is described in terms of the principal extensions \( \lambda_1 \) and \( \lambda_2 \) where
\[
\lambda_1 = \frac{d\xi}{d\eta} \quad (II.5)
\]
\[
\lambda_2 = \frac{r}{\rho} \quad (II.6)
\]
The membrane stresses are defined by
\[
\sigma_1 = \frac{1}{h} T_1 \quad (II.7)
\]
\[
\sigma_2 = \frac{1}{h} T_2 \quad (II.8)
\]
where \( h \) is the current thickness.

The constitutive law employed for the diastolic phase of the analysis has the form
\[
\sigma_i = \mu_p \left( \lambda_i^{\mu_p'} - \Lambda_0 \right) \quad i = 1, 2 \quad (II.9)
\]
where \( \mu_p' = 0.253 \mu_p \) (see discussion in text under The Diastolic Model), \( \Lambda_0 = 1/\lambda_1 \lambda_2 \) and the values of \( \mu_p \) and \( k_p \) for normal and infarcted myocardium are given in Table 2.
In the systolic phase of the analysis, the stress-strain relation for normal myocardium is taken to be

$$\sigma = \mu [\lambda \lambda^T \lambda - (\lambda \lambda^T)^2] + \mu' [\lambda^T - \lambda I]$$

where $$\lambda_i$$ are the principal stretches in the first of Equation II.10 represents the passive component which contributes to the stress only if it is tensile. For the infarcted myocardium, Equation II.9 holds in the systolic as well as the diastolic phase.

The form of the equations, II.1 to II.6, given are those used directly in the shooting method (Bogen, 1977; Bogen and McMahon, 1979) to effect a numerical solution. The finite element numerical scheme employs a system of equations fully equivalent to II.1 to II.6, which are analytically less concise and are written out in Needleman (1977) and Rabinowitz (1978).

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