Time Course of Passive Elasticity of Myocardial Tissue Following Experimental Infarction in Rabbits and Its Relation to Mechanical Dysfunction

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SUMMARY Given the substantial reserve of normal myocardium, its inability to sustain life in the presence of 30-50% necrosis of the left ventricle (LV) seems a paradox. It is known that dyskinesia of the infarcted area probably plays a dominant role in initiating failure after an infarction. To study this problem, a well defined experimental infarction was produced by cryogenic means in 58 rabbits, and the animals were allowed to recover. Groups of rabbits were killed 4 hours and 1, 2, 5, or 10 days following infarction. As quickly as possible (within 4 minutes) a sample specimen from the infarcted area was removed from the LV and subjected to a force-elongation test while being bathed in Ringer solution at 37°C equilibrated with 95% O₂-5% CO₂. The data were interpreted assuming an exponential stress-strain law with constants K and C. Mean values of K of 10.6 ± 0.94 (SEM) were found for the noninfarcted control group, whereas, rather surprisingly, no significant trend in K over 10 days was found in the infarcted group. Mean values of K ± SEM for the postinfarction groups were as follows: 4 hours, 9.51 ± 0.63; 1 day, 10.54 ± 1.13; 2 days, 13.15 ± 2.28; 5 days, 11.59 ± 1.36; and over 10 days, 12.93 ± 1.02. The functional implications were estimated with a simple model of the shortening required of the viable muscle during the isovolumic phase. It was found that contractile reserve fell rapidly with increasing infarct size, reaching zero for a 60% infarct when K = 10. With K greater than 100, there was no appreciable reduction in reserve. With a constant infarct size, variation in reserve with the afterload-preload ratio was found to be logarithmic.

IT IS GENERALLY recognized that dyskinesia of infarcted myocardium represents an important consideration in relation to cardiac dysfunction secondary to acute myocardial infarction. It is therefore rather surprising that so little seems to be known of the passive elastic properties of infarcted myocardium following infarction. Reasoning backwards from measurement on intact hearts, for example diastolic compliance, is difficult due to the analytic problems posed by the complex geometry, and by regional and spatial inhomogeneities, as well as the nonlinear elastic properties of biological tissue. Previously published studies of "compliance changes" in the period following an infarction do not permit one to draw an unequivocal conclusion. This study represents an attempt to measure directly the time course of change in passive elasticity in the period following experimental infarction of myocardial infarction in the rabbit and, second, to interpret those results in the light of the possible functional consequences.

Methods

EXPERIMENTAL DESIGN

A total of 58 New Zealand rabbits, between 4 and 5 months old, were divided into eight groups. In five groups (n = 42), cryogenic infarction of limited size was induced in the anterolateral wall of the left ventricle and the rabbits were allowed to recover. Following a randomized schedule, the various groups were anesthetized at 4 hours, 1 day (22-26 hours), 2, 5, and 10 days after infarction, the beating heart was removed, and a mid-wall specimen from the infarct was removed and as quickly as possible subjected to a force elongation test. An additional noninfarcted group (n = 7) served as a control. The noninfarcted control group was subdivided into a group in which the specimen was subjected to the force elongation test as soon as possible, and a group in which the test was intentionally delayed by 20 minutes in order to gain an impression of any dependence of elastic properties on the elapsed time after removal from the living animal. During this time the specimen remained immersed in saline at room temperature. An additional six rabbits were used to investigate the effect of freezing time on the depth of the infarct, and the variability of fiber orientation in the specimen was examined microscopically in specimens from three rabbits.

SURGICAL PROTOCOL

First Procedure

Four- to five-month-old New Zealand rabbits weighing from 3 to 4 kg were preseeded with intramuscular fluani-sone phentanylcitrate (Hypnorm), 0.5 ml, intubated, and allowed to breathe a 95% O₂-5% CO₂ mixture supplemented with 30% N₂O and halothane (3%). After ensuring that an acceptable level of anesthesia had been

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achieved curare, 2 mg, iv, was given and the free antero-
lateral wall of the left ventricle was exposed via an incision
in the fourth left intercostal space using sterile technique.
The experimental infarct was made cryogenically by use
of a variant of the technique suggested by Schaper. A
special freezing tool consisting of an aluminium block
measuring 8 × 10 × 10 mm was mounted on a brass rod
(25 cm long, 7 mm in diameter) and fitted with a wooden
handle. The aluminium block had rounded corners, a
highly polished surface over which a thin (approximately
0.05 mm) layer of Avcothane-511 was applied to impede
any tendency of the tool to stick the epicardium. This
tool was allowed to equilibrate with liquid nitrogen and
applied to the lateral wall of the left ventricle. The tool
was applied in the transverse direction with the most
anterior portion of the tool approximately 3 mm lateral
from the left anterior descending coronary artery and the
8-mm dimension orientated in the base-apex direction.
The tool was held in place without moving for 60 seconds
(except for the group in which freezing time was a
variable). Just prior to application of the freezing tool, a
prophylactic dose of 2% lidocaine-HCl (0.2 ml, iv, plus
approximately 0.3 ml topically) was administered. After
removal of the tool, the pericardium was carefully closed
with from 3 to 5 interrupted 5-0 Prolene sutures. The
thoracotomy was closed and the rabbit allowed to recover.
The duration of the surgical procedure was 30 minutes
(range, 20-45 minutes). Later on the day of surgery,
each rabbit was given a phrophylactic dose of oxytetracy-
cline (50 mg) as well as 0.25 μg of methadone. It should
be noted that, in the view of those responsible for the
care of the rabbits, the procedure was exceptionally well
tolerated and the rabbits did not appear to be in any
distress. In the postoperative period, water and food were
available ad libitum.

Total mortality was 20% and with the exception of two
rabbits was wholly confined to the first 15 minutes follow-
ing the infarction. The postoperative course was uncom-
plicated.

Second Procedure

In accordance with the schedule, rabbits were anesthe-
tized with pentobarbital sodium (60 mg, iv), the chest
was opened with a midline sternotomy, and the beating
heart was excised as quickly as possible and placed in a
dissection dish filled with saline at room temperature.

PREPARATION OF SPECIMEN

A midwall specimen was excised as quickly as possible
with a special jig and two scalpels taped together. Care
was taken to ensure that the orientation of the lengthwise
direction was inclined approximately 20° from the local
transverse plane of the ventricle. The net result of this
was a specimen of the midwall of the left ventricle
approximately 1–2 mm square and 14–18 mm long. The
abutting ventricular muscle was placed in a triphenyltetra-
zolium-chloride (TTC) solution. A special hook was made

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up by an amount proportional to the error. This implies that the zero stress slope constant $C$ would be sensitive to this difficult to determine offset. The nonlinear constant, $K$, on the other hand, for small values of strain, does not show this sensitivity. These considerations have led us to exclude the measurements of $C$ from this study. The pooled data were culled and retained as part of the study provided all of the following conditions were satisfied.

1. The retained specimen and all abutting pieces were (in the case of infarcted specimens) uniformly and completely infarcted. Judgment was made on the basis of macroscopic examination of TTC stain reaction, as well as histological examination.

2. The force-length curve from the stretch following that used in the data analysis was essentially the same as the one analyzed, thus suggesting that no irreversible changes occurred during the elongation.

3. All calibrations and controls were in order and the curves were of good quality and length (more than 50 data points).

**Results**

Infarcts were studied by the TTC stain in a group of six rabbits in order to assess the dependence of depth of penetration of the infarct zone on the time during which the freezing tool was held in place. With a contact time of 60 seconds, the depth obtained was 5 mm with a total wall thickness ranging from 5 to 6 mm. A 40-second exposure resulted in an infarct depth of 4 mm. A fatty epicardium was found to halve the infarct depth and therefore all such rabbits (3) were excluded from the study. A freezing time of 60 seconds was selected as a standard for the rest of the study. This resulted in a nearly transmural infarction with only a thin endocardial layer spared, presumably by conduction warming from the left ventricular cavity. The epicardial area of the infarct was larger than the tool by approximately 3 mm in each direction. The resulting specimens from three non-infarcted hearts, prepared as described above, were examined microscopically to ascertain whether the fiber orientation was reasonably parallel to the lengthwise axis of the specimen. In the three random samples, this was true to within $\pm 15$ degrees. Since the error in stress would be proportional to the cosine of this angle ($0.97$), this source of error was considered more than acceptable. The least squares linear fit shown in Figure 1B has an estimated slope $K/l_0$ of 11.12 cm$^{-1}$. The value of zero stress reference length $l_0$ derived by the objective averaging procedure described above was 1.50 cm, giving a value of $K$ of 16.68 for this experiment. By comparison with Figure 1A, an extrapolated estimate of $l_0$ would be approximately 1.45 cm or 3% lower. Up to and including 10 days, we found histological evidence of myofibrillar degeneration, leukocyte infiltration, and proliferative reactions consistent with the age of the infarct. The results are presented in Figure 2 with each data point representing one rabbit. The mean value of each group is indicated by the shading. The noninfarcted control group is subdivided further into two groups arranged vertically and consisting of three and four data points; these correspond to fresh (3-4 minute) and 20-minute delayed samples. As can be seen, no obvious difference developed during a 20-minute delay in room temperature saline. The mean value of $K$ for the group of noninfarcted samples pooled together was 10.60 $\pm$ 0.94 (SEM). Mean values of $K$ $\pm$ SEM for the other groups were as follows: 4 hours, 9.51 $\pm$ 0.63; 1 day, 10.54 $\pm$ 1.13; 2 days, 13.15 $\pm$ 2.28; 5
cardiac muscle fails to reveal any structures obviously intended to support loads in a direction transverse to the group was 10.6 at a temperature of 37°C. Pinto and Fung7 used right ventricular papillary muscles from rabbits and found a value of 11.4. Given the scatter in our sources of artifact in this study are deviations in muscle and in any case the values are not demonstrable different. The implication of the above findings, one should attempt to exclude the possibility that the data are distorted due to technique artifacts and/or faulty experimental design. The most likely attempt to draw a conclusion and to assess the potential artifacts prompted the in vitro design of this study.

Aside from straightforward technical errors, which we have made every attempt to exclude, the most likely reason for the disparities between the values of the passive elastic constant K is in many of the groups rather large, objective examination of the data provides no basis whatsoever for suspecting the results to be in error.

**Discussion**

Referring to the data presented in Figure 2, one is compelled to make the following observations. First, within each experimental group there is substantial variation. Second, over a period of 10 days after the induction of a cryogenic experimental infarction in rabbits, no obvious trend in elastic constant K was detected. Before attempting to draw a conclusion and to assess the potential implications of the above findings, one should attempt to exclude the possibility that the data are distorted due to technical artifacts and/or faulty experimental design. The mean value of K obtained for in noninfarcted control group was 10.6 at a temperature of 37°C. Pinto and Fung7 used right ventricular papillary muscles from rabbits and found a value of 11.4. Given the scatter in our measurements, this agreement can be considered good, and in any case the values are not demonstrable different. Aside from straightforward technical errors, which we have made every attempt to exclude, the most likely sources of artifact in this study are deviations in muscle fiber orientation from the lengthwise axis of the specimen and failure of the technique employed for fixing the ends of the specimen in test rig. Histological examination of cardiac muscle fails to reveal any structures obviously intended to support loads in a direction transverse to the fibers. Thus it might be expected that myocardium would be more elastic in the transverse direction, although we do not know of any measurement which either supports or disproves this hypothesis. In such a circumstance it can be postulated that the errors would vary with the cosine of the angle of inclination. A 10% variation in stress would thus admit an angular range of ±25 degrees, which seems unlikely. Making a rigid attachment to a highly elastic specimen requires consideration. Pinto and Fung7 have argued that a high level of compression makes the ends much stiffer. As a consequence of increased stiffness, as stress increases the high prestress at the attachment points makes them stiff, compared with the relatively low stress levels employed in this study.

The basic design of the experiment entails the in vitro measurement of elastic properties, and one must also consider this as a potential source of artifact. It is generally accepted that following death the denaturation of proteins, etc., eventually leads to a stiffening of biological tissues, in particular skeletal muscle, as suggested by the ancient term rigor mortis. On the other hand, it is our impression that a fresh dead heart is more flaccid than the normal beating heart during diastole. We are unaware of any quantitative studies which permit the objective assessment of these conflicting subjective observations. Clearly the elapsed time from death could be expected to play a role. To examine this effect, we can compare two subsets of data. The normal noninfarcted group was subdivided into two groups. In the first, the data were recorded as quickly as possible after removal of the beating heart from the anesthetized rabbit. In the second group, an intentional delay of 20 minutes was introduced, during which time the specimen was immersed in a stationary normal saline bath at room temperature. As shown in Figure 2, there were no obvious differences between the two groups. This suggests that if there are artifacts due to the in vitro design of the experiment, they occur either on a time scale shorter than a few minutes or are very slow (compared with 20 minutes) in development. It must be conceded that such artifact cannot be absolutely excluded, but the very difficulty of inferring elastic properties of muscle from intact heart measurements prompted the in vitro design of this study.

The method chosen for inducing the infarction is without question very artificial and, species differences aside, extreme caution is required in extrapolating these findings to the clinical situation. The argument that persuaded us to use the cryogenic technique was primarily the production of a homogeneously infarcted area of well defined extent. Moreover, the infarct could be made large enough to gain a specimen of practical size while at the same time maintaining an acceptably low operative mortality. The infarcted area after thawing (approximately 15 seconds) was clearly dyskinetic and cyanotic. Sections taken at 4 hours after infarction show gross interstitial hemorrhage, cellular evidence of necrosis, and a lack of staining by TTC. The wall thickness in the infarcted region at 10 days was estimated to be some 30% less than the surrounding normal myocardium. Thus, whereas the cause of the infarction was clearly artificial, there is nothing to suggest that the healing process was fundamentally differ-
ent. One is left with, at first glance, the rather surprising conclusion that, over a period of time up to 10 days following an experimental infarction, the nonlinear elastic constant K does not differ importantly from that of normal noninfarcted myocardium. In Figure 2 there are a few data points which differ substantially from the mean values of the groups to which they belong. A reexamination of the original data in those cases provides no basis whatsoever for suspecting the result. Rather it suggests that other factors not controlled in this study may play a role. In a recent paper on the intact heart following experimental infarction produced by ligation of a coronary artery branch, Vokonas et al. have shown that end-diastolic length increases rapidly (within 1 hour) and is accompanied by dyskinetic systolic bulging. After a period of 6 hours, the dyskinetic stretching of the infarcted segment is reduced while the end-diastolic length remains essentially the same. They have interpreted this finding as indicating an increased stiffening. It is interesting to consider whether their findings of increased "stiffness" can be compatible with our finding of no important change in nonlinear elastic constant K. At first glance one is tempted to say one of the two findings must be in error. However, the transformation of a stress-strain relationship into a pressure-length loop involves a number of factors in addition to the elasticity constant K. The zero stress slope C, which is often neglected, effective zero stress length l₀, and geometry in particular as expressed by radius of curvature and wall thickness may be changing, and it is difficult to separate the effects of such changes from those consequent to a change in the intrinsic elastic constant K. Looking at Figure 6 of Vokonas et al., it may be possible to postulate that, between 1 and 6 hours, edema has decreased the ratio between radius of curvature and wall thickness, thus tending to shift the two curves along the same stress-strain curve. Attempts to do this by multiplying the 6-hour pressure values by a constant reducing factor were not especially convincing. However, by expressing the length as a function of its maximum and replotted their results, one obtains an almost identical loop, consistent with postulating that the unstressed length l₀ has changed. These results suggest that a creep and/or stress relaxation response to a repeated cyclic stress history may well be an important consideration.

POSSIBLE CLINICAL IMPLICATIONS

As always with experimental preparations, extreme caution is required when extrapolating results to the clinical situation. Not only must one appreciate potential species differences, but there is absolutely no pretense that the cryogenic infarct in any way simulates the still unclear origins of an infarct encountered in man. At the same time it must be stated that the healing process as well as the resultant elastic constant, K, are not obviously at variance with what little is known for man. The remarkable inability of man to withstand an infarction some 40% of the ventricle, in the presence of substantial contractile reserve of the remaining normal myocardium, requires consideration. The importance of dyskinesia of the infarcted segment has been assessed with the use of a model by both Parmley et al. and Swan. They have computed the mechanical disadvantage of both compliant and nondistensible infarcts of varying size. The results of their model calculations are important in developing a greater insight into the role of dyskinesia in cardiac decompensation following a large myocardial infarction. The only intellectual criticism of their model is that it requires the assumption of both end-diastolic and end-systolic stress, and, moreover, that the normal muscle contracts so as to displace two-thirds of its subtended volume. This results in a contraction geometry difficult to visualize and quantitatively suspect. At the same time it must be conceded that, if one wishes to obtain a result comparable with clinical data, such as ejection fraction, a number of assumptions are required. As the goal was improved, insight and not quantitative comparison with intact heart data, such an approach can be regarded as prudent. We have retained the same goal but sought a model which might be easier to visualize and yet illustrate qualitatively the effects of variations in the value of the nonlinear elastic constant K. The model consists of an assumed geometry for the left ventricle, in this case a circular cylinder. A transverse cross-section of such a "heart" consists of an annular ring, and it is further assumed that both the normal and infarcted segments consist of homogenous "pie-slices" with a sharp line of demarcation. The percent infarct is then defined as the end-diastolic fractional circumference subtended by infarcted tissue divided by the total circumference. During the isovolumic contraction phase of such a "heart," the cross-sectional inside diameter remains constant as the normal muscle contracts, stretching the passive infarcted segment. As a result of this process, the pressure rises from its left ventricular end-diastolic value to aortic diastolic pressure. One can then calculate the fractional shortening of the remaining normal muscle required to attain this pressure. The detailed derivation of the relevant equations and the method of solution are given in the Appendix. There are several approaches one could take in attempting to characterize the contractile properties of the remaining normal myocardium. Each of these will present its own set of uncertainties. Despite our present incomplete understanding of the contractile properties of cardiac muscle, it can be safely assumed that there is an absolute upper limit on the percent shortening attainable. If we subtract the shortening used up in dyskinetic stretching of the infarcted segment from this upper limit, we obtain the fractional shortening which is "available" for actually pumping blood. This difference we have defined as the "shortening reserve." Moreover, we have assumed a constant value of the absolute maximum fractional shortening of 40%, when working against physiologically normal load. This would correspond to an ejection fraction of 0.64 for the cylindrical geometry with no infarct. The reader who chooses to take issue with our choice of 0.4 as a normal maximum can mentally shift the resultant curves up or down accordingly. The results are shown in Figure 3, where the shortening reserve was calculated as a function of the percent infarction, with the elastic constant K as a parameter. The results are, of course, also dependent upon both the end-diastolic left
ventricular pressure and aortic diastolic pressure. As can be seen, the shortening reserve is seriously compromised when $K$ is in the range of 10 to 15 and the infarcted area comprises approximately 40% of the ventricle. Under these conditions, the percent shortening required of the normal muscle to passively distend the infarcted segment varies from 10% to 16% of the end-diastolic length. Logically enough, as the infarct size increases the contractile reserve falls with an ever increasing rate. As is evident from the figure, the elastic constant $K$ of the infarcted tissue would have to be substantially higher in order to mitigate these effects of dyskinesia. The variation with the passive constant $K$ appears to be roughly logarithmic, and in order to attain a negligible compromise in function, $K$ would have to be of the order of 100. As is evident from the equations presented in the Appendix, these results are also dependent on the ratio of the aortic diastolic pressure to the left ventricular end-diastolic pressure. To elucidate the sensitivity of reduced contractile reserve to changes in that pressure ratio, a series of calculations was performed with a constant end-diastolic ventricular pressure of 10 mm Hg and a 40% infarct size. With a $K$ of 10, reducing the aortic diastolic pressure from 100 mm Hg to 60 mm Hg lowers the dyskinetic shortening of the normal muscle from approximately 17% to 13%, and constitutes a reduction of nearly 25% in wasted “effort.” This seemingly modest improvement, one could speculate, may represent an important consideration in determining the effectiveness of such interventions as afterload lowering drugs and intraaortic balloon pumping.

**Appendix**

**MATHEMATICAL MODEL OF THE EFFECT OF ISOVOLUMIC DYSKINETIC STRETCHING OF INFARCTED SEGMENT ON LV CONTRACTILE RESERVE**

We assume: (1) thin-walled cylindrical geometry, (2) that during isovolumic contraction up to pressure $P_s$ from $P_d$, the radius remains constant, and (3) infarcted segment is a homogeneous pie-shaped wedge with well defined edge. From (2) above, the circumferences in diastole and in systole are equal.

$$\Gamma = \gamma_{nd} + \gamma_{ns} = \gamma_{nd} + \gamma_{ns}$$  \hspace{1cm} (1)

where $\gamma_{nd}$ and $\gamma_{ns}$ refer to the partial circumferences of the normal and infarcted tissue, respectively. The subscripts, $d$ and $s$, refer to end-diastolic and end isovolumic systole, respectively. The infarcted fraction of the left ventricle is defined as

$$f = \gamma_{ns}/\Gamma.$$  \hspace{1cm} (2)

The fractional shortening of the normal muscle is defined as

$$S = 1 - (\gamma_{nd}/\gamma_{ns}).$$  \hspace{1cm} (3)

Combining Equations 1 and 2, and substituting into Equation 3 and rearranging, we get

$$\gamma_{ns} = (1 - f)(1 - S)\Gamma.$$  \hspace{1cm} (3a)

The Lagrangian strain of the infarcted segment at end isovolumic systole is

$$\epsilon_s = (\gamma_{ns}/\gamma_{ns}) - 1$$  \hspace{1cm} (4)

and end diastole is

$$\epsilon_d = (\gamma_{nd}/\gamma_{nd}) - 1$$  \hspace{1cm} (5)

where the subscript $o$ refers to the unstressed state. Let us further assume that the constitutive relation $d\sigma/d\epsilon = K\epsilon + C$ can be reduced to $d\sigma/d\epsilon = K\epsilon$, which can be directly integrated between a diastolic and a systolic stress to give

$$\frac{1}{K}\ln\left(\frac{\sigma}{\sigma_d}\right) = \epsilon_s - \epsilon_d = (\gamma_{ns} - \gamma_{ns})/\gamma_{ns}.$$  \hspace{1cm} (6)

Under assumptions (1) and (2), the stress is given by the Young-La Place law

$$\sigma = PR/t$$  \hspace{1cm} (7)

where $t$ is the wall thickness. Since the radius $R$ is a constant substituting Equation 7 into 6, we get

$$\gamma_{ns} = \frac{K}{P_s} \ln \left(\frac{P_{ls}}{P_{ld}}\right) = \gamma_{ns}.$$  \hspace{1cm} (8)

Combining Equation 8 with 2 and noting that for the thin-walled cylinder, $t_0a = \gamma_{ns}$, we get

$$\gamma_{ns} = \frac{K}{P_s} \ln \left(\frac{P_{ls}}{P_{ld}}\right) + f\Gamma.$$  \hspace{1cm} (9)

and from Equations 1 and 3a,

$$\gamma_{ns} = \Gamma - \gamma_{ns} = \Gamma - (1 - f)(1 - S)\Gamma = (f + S - fS)\Gamma.$$  \hspace{1cm} (10)

Moreover,

$$\gamma_{ns} = 1 - S + \frac{S}{f}\Gamma.$$  \hspace{1cm} (11)
Substituting Equation 11 into 9, equating 9 and 10, and rearranging, we get

\[ \left( \frac{\gamma_f}{\gamma_{inf}} \right)^b \frac{f}{K} \ln \left( \frac{P_s/P_d}{f} \right) - fS = 0 \]  

(12)

If we further assume that \( \gamma_f/\gamma_{inf} = 1 \) or that \( K \) can be regarded as an effective \( K \) multiplied by prestrain, we finally get

\[ \left( \frac{f}{K} \right) \ln \left( \frac{P_s}{P_d} \right) - fS = 0 \]  

(13)

For any value of \( K \) and the systolic-diastolic pressure ratio, \( P_s/P_d \), we can numerically solve Equation 13 for the dyskinetic shortening \( S \) as a function of the size of the infarct, \( f \). This was done using the “roots of \( f(x) = 0 \)” program supplied with the Hewlett-Packard 9830 table calculator and the results presented graphically in the form of a “contractile reserve” = 0.4 - \( S \). This is equivalent to assuming that the ultimate shortening of the normal muscle against a load was 40%, this number being rather arbitrarily chosen to correspond to an ejection fraction of 0.64 for a cylindrical heart.

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