Analysis of Systolic Bulging
Mechanical Characteristics of Acutely Ischemic Myocardium in the Conscious Dog

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SUMMARY. To determine the mechanical factors affecting regional segmental motion after acute coronary occlusion, we studied seven conscious dogs, instrumented with sonomicrometers. Loading conditions were changed by the withdrawal of 500 ml of blood and the transfusion of 800 ml of blood. To express segmental motion, percent systolic shortening, percent systolic elongation, and early diastolic shortening were calculated. Blood withdrawal decreased left ventricular preload, increased percent systolic elongation (from 6.9 ± 3.1% to 9.9 ± 3.5%) and early diastolic shortening (12.9 ± 5.3% to 16.6 ± 5.3%), and decreased percent systolic shortening. Blood transfusion increased left ventricular preload, decreased percent systolic elongation (to 5.2 ± 1.8%) and early diastolic shortening (8.8 ± 2.9%), and increased percent systolic shortening. Manipulation of loading did not change regional myocardial blood flow. In acutely ischemic myocardium, the tension-length loop showed an exponential upstroke during isovolumic systole and a nearly superimposed exponential downstroke during the isovolumic relaxation phase after systole, compatible with essentially passive movement as seen with an elastic material. The changes in loading conditions affected the tension-length curve to a very minor extent. The uniformity of the curve and its exponential shape explain the load-dependency of systolic bulding and segmental motion. It is concluded that systolic bulging depends on the change in the preload tension due to the compliant portion of tension-length curve, and that shortening of ischemic myocardium during the isovolumic relaxation phase is a completely passive phenomenon. (Circ Res 58: 209-217, 1986)

SYSTOLIC bulging following total coronary artery occlusion has been recognized since 1935 (Tennant and Wiggers, 1935). There has been, however, no clear definition of the determinants and implications of systolic bulging, although, recently, it has been demonstrated to be an isovolumic phase phenomenon (Herman et al., 1967; Ellings et al., 1977; Lew et al., 1985) occurring during moderate ischemia with graded coronary occlusion (Akaishi et al., 1985b). After complete coronary occlusion, prominent systolic bulging or akinetic segment motion without bulging can occur. These variations in ischemic segment motion have been considered to be related to the passive, elastic property of acutely ischemic myocardium (Hood et al., 1970; Forrester et al., 1972; Pirzada et al., 1976). However, this asynchronous motion of the ischemic myocardium has not been completely characterized. Herman et al. (1967) suggested that delay in electrical activation or excitation may result in asynchronous contraction in the ischemic zone. Tyberg et al. (1969) studied systolic elongation in papillary muscle in vitro, and related paradoxical motion to differences in the time course of contraction and relaxation between ischemic and normal muscle. Since paradoxical motion of ischemic myocardium is not a true mirror image of normal contraction, the pattern of systolic bulging cannot be explained by simple interaction between two regions, as previously proposed (Tyberg et al., 1974; Kumada et al., 1979). Thus, the quantitative relationship of systolic bulging to blood flow or hemodynamics remains unclear.

The specific questions examined in this study are: (1) is segmental motion in severely ischemic tissue entirely passive, or is there an active component?, and (2) what are the determinants of segmental motion throughout the cardiac cycle in severely ischemic tissue? These questions are explored by analyzing myocardial segment motion and the tension-length relation in acutely ischemic myocardium in conscious dogs under varying left ventricular (LV) loading conditions.

Methods

Instrumentation

Using sterile technique, seven mongrel dogs weighing 22-35 kg underwent left thoracotomy in the 5th intercostal space under sodium pentobarbital anesthesia (25 mg/kg body weight, iv). The pericardium was opened, the left
anterior descending coronary artery (LAD) was dissected free after its first diagonal branch, and a hydraulic cuff or loop-type snare occluder was placed loosely around the vessel. Pairs of ultrasonic crystals were inserted in both the normally perfused area of the lateral wall and in a zone to be rendered ischemic in the center of the LAD-perfused area. One crystal pair at each site was implanted subendocardially in a circumferential plane for measurement of segment length (Weintraub et al., 1981). In three dogs, a pair of ultrasonic crystals was implanted on the anterior and posterior endocardial surfaces through an incision in the LV apex to measure the LV anterior-posterior minor axis internal diameter, as described previously (Akaishi et al., 1985a). Pacing electrodes were sutured to the left atrial appendage. A 7F vinyl catheter was placed into the left atrium for injection of radioactive microspheres. The pericardium was left open, and the catheter was maintained throughout the experiment.

Three to 7 days after the operation, studies were performed while the dogs were sedated with morphine sulfate (30 mg, im) and diazepam (10 mg, im) and were lying quietly on their right sides. Two catheter-tipped microtransducers (Millar Instruments) were placed into the left ventricle and the aortic arch at the level of the valve cusps through bilateral femoral arteriotomies performed under local anesthesia. A catheter was placed into a carotid artery to withdraw blood during injection of radioactive microspheres.

Protocol

In five of seven dogs, 15-μm radioactive microspheres were used to measure myocardial blood flow. After the first set of microspheres had been injected and control hemodynamics and sonomicrometry data were recorded, complete coronary occlusion was produced with the occluder and was maintained throughout the experiment. Fifteen minutes after coronary occlusion, the second set of microspheres was injected while the measurements were repeated. Then, 500 ml of blood were withdrawn slowly from the venous catheter. Five minutes after the completion of blood withdrawal, when hemodynamic parameters had stabilized, the third injection of microspheres was made, and measurements were repeated. Next, 800 ml of blood (previously withdrawn, supplemented by 300 ml of warm, pH-corrected blood from a donor animal) were transfused over 30 minutes. The last set of microspheres was injected, and recordings were performed 5 minutes after the transfusion was completed. In two dogs, phenylephrine (10 mg, iv) was injected to elevate aortic pressure, and recordings were repeated.

Tissue Preparation

After the experiment, the heart was removed, frozen for 24–72 hours, and then was cut while still partially frozen. Transmural myocardial samples were taken from the remote nonischemic zone and from the ischemic zone in the center of the LAD-perfused area. The samples were divided into subendocardial, mid-myocardial, and subepicardial thirds. Myocardial blood flow was determined in each sample by standard techniques (Weintraub et al., 1981).

Measurement of Segment Length and Shortening

End-diastole was defined as the time of abrupt change of LV dP/dt during diastole; this always occurred after the a-wave, if seen. The onset of ejection was defined as the time when LV pressure surpassed aortic cusp pressure during systole. End systole was defined as the time that LV pressure fell below the diastolic notch of the aortic cusp pressure. Myocardial segment lengths were measured at end diastole (EDL) and end systole (ESL). In addition, during systolic bulging produced by coronary occlusion, the maximum length (Lmax) and minimum length (Lmin) were measured. All lengths were normalized by assuming EDL in the control state to be 10 mm. To describe segmental motion systolic shortening (%SS), systolic bulging (%bulging), total segment length, and early diastolic recoil (%recoil) were defined as follows:

\[
\text{%SS} = \frac{\text{EDL} - \text{ESL}}{\text{EDL}} \times 100(\%)\]

\[
\text{%bulging} = \frac{\text{Lmax} - \text{EDL}}{\text{EDL}} \times 100(\%)\]

\[
\text{%recoil} = \frac{\text{ESL} - \text{Lmin}}{\text{EDL}} \times 100(\%)\]

Since systolic bulging is an isovolumic phase phenomenon (Lew et al., 1985; Akaishi et al., 1985b), the time at %bulge (at \(L_{\text{max}}\)) will be close to, but not necessarily exactly the same as, the time of onset of ejection.

Estimation of relative change in regional tension was performed. According to Laplace's law for a simple spherical model, tension (T) may be expressed as:

\[
T = \frac{P \times R}{2} \quad (1)
\]

where P is the transmural pressure (disregarding extracardiac pressure) and R is the radius of the left ventricle. Since the major portion of the left ventricle is the non-ischemic zone, the instantaneous R may be estimated from the equation:

\[
R = \frac{L_{\text{n}}}{\omega} \quad (2)
\]

where \(L_{\text{n}}\) is nonischemic segment length and \(\omega\) is the center angle in radians for the arc of \(L_{\text{n}}\). The three dogs with implanted diameter crystals showed the following linear regression equation describing the relation between diameter (D) and \(L_{\text{n}}\) (in mm):

\[
D = 4.39 \times L_{\text{n}} + 3.43
\]

(mean \(D = 44.8\) mm, \(r = 0.94, P < 0.001\)).

Between 1000 and 1500 points were compared in each dog. The Y intercept was only 8% of the mean diameter value. Because \(L_{\text{n}}\) is linearly related to D, \(L_{\text{n}}\) may be used as an index of D in calculations to estimate T. Therefore, using Equations 1 and 2, we derived an index of tension, as follows:

\[
T = \frac{P \times L_{\text{n}}}{2 \times \omega} \quad (3)
\]

If \(\omega\) is assumed to be constant throughout the cardiac cycle and after coronary occlusion, the relative value of tension in the ischemic zone may be expressed as the product of \(P\) and \(L_{\text{n}}\). Since the segment length was normalized to an EDL of 10 mm and \(\omega\) depends on the size of the heart, T reflects tension in arbitrary units. It must
be recognized that this is a first order approximation of
tension, given that the center angle may not be constant,
and that there may be error in determining the correct
diameter. Nonetheless, this calculation primarily reflects
the fact that load on the segment decreases during systole
as the heart becomes smaller.

**Digitization of Data**

Segment lengths, internal diameter, and pressure were
digitized with a digitizing tablet (Bitpad One, Summa-
graphics) and entered into a computer (Dec Vax 11/750).
The spatial resolution of the tablet is less than 0.1 mm
long, and the area is less than 0.01 mm². Measurement
error by duplicate manual tracing is maximally 0.2%. We
utilized locally developed software to perform data
smoothing, with a proportional 3-points method, and to
perform linear interpolation. From a 1-second strip chart
record, about 800 points were digitized by hand tracing.
These 800 points were smoothed and interpolated into
1000 points at 1-msec intervals. The product of P and L™
length loops were created with an X-Y plotter (Hewlett-
Packard). To describe these curves, exponential fitting
(Minsky, 1976) was performed by the least squares
method.

**Statistical Analysis**

All data are presented as mean ± s. For these statistical
determinations, analysis of variance and linear regression
analysis were performed, using BMDP statistical software
(Dixon et al., 1983).

**Results**

**Hemodynamics**

Hemodynamic data are displayed in Table 1. Heart rate increased after coronary occlusion, in-
creased further with blood withdrawal, and de-
creased with transfusion. During blood trans-
fusion, left atrial pacing was used to maintain the heart rate,
at least at the level recorded after coronary occlu-
sion, but before blood withdrawal. Aortic and LV
systolic pressure decreased with blood withdrawal,
and increased with blood transfusion. Aortic dia-
stolic pressure did not change significantly with
changes in loading. LV end-diastolic pressure in-
creased after occlusion, decreased after blood with-
drawal, and increased after transfusion. LV dp/dt
did not change significantly after coronary occlusion
or with changes in loading conditions. In the two
dogs that were given phenylephrine while the heart
rate was maintained by atrial pacing (mean of 106
beats/min), aortic systolic pressure increased to a
mean of 215 mm Hg, and LV end-diastolic pressure
increased to a mean of 42 mm Hg.

**Myocardial Blood Flow (Table 2)**

In the control state, there was no significant dif-
ference in myocardial blood flow in any transmural
layer between the ischemic and the nonischemic
regions. Coronary occlusion resulted in decreased
blood flow in all layers of the ischemic zone. No
significant changes in ischemic zone flow were ob-
erved after blood withdrawal or blood transfusion.
In the nonischemic region, there was no significant
change in blood flow after occlusion, blood with-
drawal, or blood transfusion.

**Regional Myocardial Function**

Figure 1 shows representative tracings of LV
dp/dt, ischemic zone segment length, nonischemic
segment length, LV internal diameter, and LV pres-
sure recorded at high and low gain. Data are shown
in the control state and after coronary occlusion,and
then after blood withdrawal and after blood trans-
fusion. As seen from the tracing of ischemic segment
length during coronary occlusion, the motion of the
acutely ischemic myocardium was characterized by
lengthening (bulging) in early systole, slowly de-
creasing length in mid-systole, rapidly decreasing length
(recoil) in early diastole, and slowly increasing length
in the remainder of diastole. In the nonis-
chemic zone, the EDL increased and showed in-
creased shortening (Akaishi et al., 1985b), and
chamber diameter showed increased end-diastolic
dimension and decreased shortening (Akaishi et al.,
1985a) after coronary occlusion. With blood with-

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Hemodynamics</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Control (n = 7)</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>109 ± 17</td>
</tr>
<tr>
<td>AoSP (mm Hg)</td>
<td>131 ± 26</td>
</tr>
<tr>
<td>AoDP (mm Hg)</td>
<td>101 ± 26</td>
</tr>
<tr>
<td>LVSP (mm Hg)</td>
<td>131 ± 26</td>
</tr>
<tr>
<td>LVEDP (mm Hg)</td>
<td>10 ± 4</td>
</tr>
<tr>
<td>LVdP/dt (mm Hg/sec)</td>
<td>2914 ± 649</td>
</tr>
</tbody>
</table>

Abbreviations: AoDP = aortic diastolic pressure; AoSP = aortic systolic pressure; HR = heart rate;
LVdP/dt = first derivative of left ventricular pressure; LVEDP = left ventricular end-diastolic pressure;
LVSP = left ventricular systolic pressure.
* P < 0.03 vs. control.
† P < 0.05 vs. control.
‡ P < 0.02 vs. control.
§ P < 0.05 vs. blood withdrawal.
TABLE 2

Myocardial Blood Flow

<table>
<thead>
<tr>
<th></th>
<th>Control (n = 5)</th>
<th>Occlusion (n = 5)</th>
<th>Blood withdrawal (n = 5)</th>
<th>Blood transfusion (n = 5)</th>
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</thead>
<tbody>
<tr>
<td><strong>Ischemic region</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subendo</td>
<td>1.01 ± 0.46</td>
<td>0.07 ± 0.04*</td>
<td>0.09 ± 0.05*</td>
<td>0.11 ± 0.11*</td>
</tr>
<tr>
<td>Mid</td>
<td>0.85 ± 0.30</td>
<td>0.04 ± 0.03*</td>
<td>0.05 ± 0.02*</td>
<td>0.03 ± 0.03*</td>
</tr>
<tr>
<td>Subepi</td>
<td>0.84 ± 0.26</td>
<td>0.10 ± 0.05*</td>
<td>0.13 ± 0.05*</td>
<td>0.11 ± 0.08*</td>
</tr>
<tr>
<td>Transmural</td>
<td>0.89 ± 0.32</td>
<td>0.07 ± 0.02*</td>
<td>0.09 ± 0.03*</td>
<td>0.09 ± 0.07*</td>
</tr>
<tr>
<td><strong>Nonischemic region</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subendo</td>
<td>1.37 ± 0.69</td>
<td>1.40 ± 0.50</td>
<td>1.37 ± 0.39</td>
<td>1.81 ± 0.63</td>
</tr>
<tr>
<td>Mid</td>
<td>1.29 ± 0.61</td>
<td>1.33 ± 0.44</td>
<td>1.18 ± 0.42</td>
<td>1.78 ± 0.35</td>
</tr>
<tr>
<td>Subepi</td>
<td>1.02 ± 0.53</td>
<td>1.16 ± 0.41</td>
<td>1.29 ± 0.70</td>
<td>1.50 ± 0.33</td>
</tr>
<tr>
<td>Transmural</td>
<td>1.21 ± 0.59</td>
<td>1.28 ± 0.44</td>
<td>1.27 ± 0.48</td>
<td>1.67 ± 0.35</td>
</tr>
</tbody>
</table>

Abbreviations: Subendo = subendocardial blood flow (ml/min per g); Subepi = subepicardial blood flow (ml/min per g); Mid = mid-myocardial blood flow (ml/min per g).

*P < 0.002 vs. control.

drawal after coronary occlusion, the ischemic segment showed a greater amplitude of motion, with both greater systolic bulging and greater recoil, whereas nonischemic segment length and internal diameter decreased. With blood transfusion, the ischemic segment showed a smaller amplitude of motion, with less systolic bulging and less recoil, whereas LV end-diastolic pressure increased, and nonischemic segment length and internal diameter increased. Nonischemic segment length and internal diameter showed corresponding changes with loading.

The summarized myocardial function data are presented in Table 3. In the ischemic region, coronary occlusion increased EDL, blood withdrawal decreased EDL, and transfusion increased EDL. After occlusion, total percent systolic shortening %SS changed to a negative value as a result of the systolic elongation. Systolic bulging increased after blood withdrawal, and then decreased after blood transfusion. Similarly, diastolic recoil increased after blood withdrawal (P < 0.05), and decreased after transfusion (P < 0.002).

The EDL of the ischemic region increased after coronary occlusion and was always greater than the EDL in the nonischemic region at any loading state (P < 0.007). Furthermore, these lengths correlated with each other (Fig. 2) after coronary occlusion, with a slope of almost 1.0. Thus, after coronary occlusion, ischemic EDL was always larger than nonischemic EDL, but the change in EDL that resulted from changing loading conditions was the same in both regions.

Thus, variations in systolic bulging were clearly affected by loading. The change in systolic bulging and the change in EDL were closely but negatively

**FIGURE 1.** Representative tracings of ischemic and nonischemic segment length, diameter, and left ventricular pressure. From left to right, four panels are presented. The first is control, followed by three panels after coronary occlusion. The second panel is after occlusion, the third is after blood withdrawal, and the last is after transfusion.
correlated, despite variation of heart rate and aortic pressure \((r = -0.91, P < 0.0001)\) (Fig. 3). Thus, as EDL rises, systolic bulging is diminished.

**Tension-Length Relationships in the Ischemic Region**

Figure 4 illustrates tension-length loops in the ischemic region of four dogs, drawn by computer graphics from data recordings made under varying loading conditions. During the control state, the loop was rectangular and counterclockwise. From end diastole to early systole, there was initially an increase in tension without change in length (isovolumic systolic phase), then a decrease in length with little change in tension (ejection phase), followed by a decrease in tension with no change in length (isovolumic relaxation phase), and, finally, by an increase in length while the tension slowly increased.

After coronary occlusion, the loop shifted toward the right, and showed an exponential upstroke during isovolumic systole and a nearly superimposed exponential downstroke during the isovolumic relaxation phase. From end diastole to early systole, there was initially an increase in tension with increase in length (isovolumic systolic phase), then little change in length and tension during the ejection phase, followed by a decrease in tension with decrease in length (isovolumic relaxation phase), and, finally, by an increase in length while the tension slowly increased. There was, therefore, no isometric phase in the ischemic zone during either isovolumic systole or isovolumic relaxation in the left ventricle as a whole. There was relatively unchanged length during the ejection phase when tension changed little. The minimum length oc-
curred at the time of minimum tension, or the left lower corner of the tension-length curves. In spite of changes in preload, afterload, and heart rate, the tension-length curve described a relatively constant upsloping and downsloping exponential function in each dog.

Although coronary occlusion caused a large shift to the right of the tension-length relation, interventions after coronary occlusion which changed the size of the heart caused slight parallel shifts of the tension-length relation (Fig. 4). Blood withdrawal caused a slight shift to the left; blood transfusion and phenylephrine caused a slight shift to the right.

Table 4 presents the exponential equations describing the average relationship of tension to ischemic segment length derived under varying loading conditions in all dogs. Figure 5 displays these average tension-length relationships graphically. The curves representing the data obtained after coronary occlusion, blood withdrawal, blood transfusion, and phenylephrine infusion were not substantially different, although the ranges of tension and segment length utilized in generating the curves were different.

**Table 4**

<table>
<thead>
<tr>
<th>Tension-Length Curves</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood Occlusion</td>
</tr>
<tr>
<td>Blood withdrawal</td>
</tr>
<tr>
<td>Blood transfusion</td>
</tr>
<tr>
<td>Phenylenephine infusion</td>
</tr>
</tbody>
</table>

| a  | 1.15 ± 4.7 | 1.02 ± 4.9 | 1.25 ± 4.9 | 1.42       |
| b  | -9.9 ± 4.8 | -8.4 ± 4.8 | -11.6 ± 5.6 | -14.6     |
| r  | 0.95 ± 0.04 | 0.96 ± 0.02 | 0.95 ± 0.02 | 0.99      |

The parameters a and b (given as mean ± s.e.) may be used to calculate the tension index (T) at any segment length in the ischemic zone at each state from the exponential equation:

\[ T = e^{a + b(L - L_0)} \]

where \( L_0 \) is the instantaneous length of the ischemic segment and \( L_0 \) is the length at preocclusion end diastole. r is the correlation coefficient.

**Discussion**

Tennant and Wiggers (1935) first documented the development of a systolic bulge after coronary occlusion. Tyberg et al. (1974) and Theroux et al. (1974) described the shortening and lengthening behavior of acutely ischemic myocardium as holosystolic expansion. Nakamura et al. (1980) described changes in the pattern of myocardial shortening resulting from reduction of regional coronary blood flow. Recently, systolic function in ischemic tissue has been characterized as bulging during the isovolumic phase and variable shortening during the ejec- tion phase following total coronary occlusion (Lew et al., 1985) or graded partial coronary occlusion (Akaishi et al., 1985b). The present study demonstrates in conscious dogs that, whereas the segmental motion pattern of acutely ischemic myocardium is more complicated than simple paradoxical expansion, the underlying determinants of ischemic segment motion are straightforward.

After complete coronary artery occlusion followed by manipulation of preload, systolic bulging varied inversely with ischemic EDL. After coronary occlusion, the tension-length relationship of ischemic myocardium during systole and diastole can be described as a single exponential equation. The correlation of %bulging with EDL occurs because volume change sets the ischemic myocardial segment length at a higher or lower portion of the curve with different slope. At high EDL, the tension-length
Changes in loading conditions did not affect the myocardial function (Hood et al., 1970; Parmley et al., 1973; Pirzada et al., 1976). Previous investigators have hypothesized that akinesis is essentially different from dyskinesis, or paradoxical systolic expansion; dyskinesis has been considered to indicate a state of compliance higher than akinesis (Hood et al., 1979).

The present study suggests that increasing preload may change dyskinesis to akinesis, and that decreasing preload may result in dyskinesis even if myocardial mechanical properties, i.e., the tension-length curve, are not changed. In an echocardiographic study, Kerber and Abboud (1975) noted complementary data. After coronary occlusion, dogs were given methoxamine, phenylephrine, or noradrenaline. Of these three, only methoxamine caused an increase in left ventricular end-diastolic pressure and end-diastolic diameter, and only methoxamine caused a decrease in systolic bulging. Thus, it is essential, when analyzing segmental wall motion over time after coronary occlusion, to take loading into account.

The potential decrement in left ventricular function due to systolic bulging may be gleaned from close scrutiny of the work of Bogen et al. (1980). In this theoretical study, an isotropic, initially spherical membrane model of the heart before and after infarction was used. Stroke volume was predicted over a wide range of left ventricular end-diastolic pressure for infarcts of various sizes. A difference was predicted between the immediate stage (loss of contractile function) and the acute stage (increase in myocardial extensibility) which may be attributable to paradoxical bulging. From Figure 5 of that paper, the percent fall in stroke volume from the immediate to the acute stage will depend on left ventricular end-diastolic pressure LVEDP and the size of the infarct, but does not appear likely to exceed 10%.

In the present study, the relative change in tension was estimated to characterize segmental motion. Although segment shortening might vary, depending on fiber orientation (Freeman et al., 1985) and depth in the wall (Weintrab et al., 1981), the ventricular wall contracts as a unit. This segmental wall motion is determined by the tension against which a segment contracts as opposed to the stress on a particular muscle layer. Tension, unlike stress, neglects direction and thickness. Moreover, multiple assumptions are necessary to calculate regional stress. These assumptions may not be valid, especially in a regionally ischemic ventricle (Mirsy, 1979). It is necessary to note that the calculation of tension presented here is an approximation which depends on several assumptions noted in Methods. Since tension could not be calculated exactly, changes of the index of tension were estimated to characterize ischemic segment motion behavior. In the present analysis, dynamic factors such as viscosity or inertia were of little importance, since the minimum length of the ischemic segment occurred at the nadir of LV pressure or tension. Moreover, a good correlation between the observed data and calculated data obtained with a simple elastic model justified these simplifications within one cardiac

After complete occlusion, the ischemic myocardium shows passive motion, determined for the most part by regional tension. The exponential tension-length curve throughout the cardiac cycle is that which would be expected of an elastic material. Changes in loading conditions did not affect the curve substantially, although the increase in the size of the left ventricle tended to shift the curve toward the right in a parallel fashion, probably due to creep, or stretching at constant higher tension over the course of each cardiac cycle. The creep occurring with changes in loading is smaller than the creep after coronary occlusion; i.e., EDL is longer in the ischemic region than in the nonischemic zone, due to stretching of the ischemic region by the systolic tension developed in the nonischemic myocardium (Forrester et al., 1972; Tyberg et al., 1974; Edwards et al., 1981). The relative uniformity of the curve despite varying hemodynamics provides the mechanism by which changes in preload alter the magnitude of systolic bulging. Thus, the uniform tension-length relationship resulted in the apparently complicated segmental motion in ischemic myocardium and in the apparently paradoxical observation of load dependency of systolic bulging. This observation, while apparently a fundamental one, is qualitative, and it is not possible to predict the tension-length curve for all infarcts. The shape of this curve may depend on the size of the ischemic zone, whether it is nearer the apex or the base of the heart, and on particular properties of the myocardium in each case, such as whether there is any fibrosis in the area from previous ischemia or if the myocardium is hypertrophied.

Although shortening during isovolumic relaxation in the ischemic myocardium (recoil) has been speculated to be due in part to persistent contractile activity (Gaash et al., 1985), this study demonstrates that this shortening, like bulging, is a completely passive phenomenon, at least in severely ischemic myocardium. The magnitude of recoil is load dependent, however.

After complete coronary artery occlusion, changing loading conditions did not affect myocardial flow in the ischemic zone. Since myocardial blood flow was unchanged by hemodynamic changes after complete coronary occlusion, the load dependency of systolic bulging cannot be attributed to changes in regional myocardial blood flow. Systolic bulging occurred primarily during the isovolumic period, confirming our previous observation (Akaishi et al., 1981) and that of the Lew et al. (1985).

The effect of systolic bulging on LV function has been considered to be a potential adverse factor for LV function (Hood et al., 1970; Parmley et al., 1973; Pirzada et al., 1976). Previous investigators have hypothesized that akinesis is essentially different from dyskinesis, or paradoxical systolic expansion; dyskinesis has been considered to indicate a state of compliance higher than akinesis (Hood et al., 1979). The present study suggests that increasing preload may change dyskinesis to akinesis, and that decreasing preload may result in dyskinesis even if myocardial mechanical properties, i.e., the tension-length curve, are not changed. In an echocardiographic study, Kerber and Abboud (1975) noted complementary data. After coronary occlusion, dogs were given methoxamine, phenylephrine, or noradrenaline. Of these three, only methoxamine caused an increase in left ventricular end-diastolic pressure and end-diastolic diameter, and only methoxamine caused a decrease in systolic bulging. Thus, it is essential, when analyzing segmental wall motion over time after coronary occlusion, to take loading into account.

The potential decrement in left ventricular function due to systolic bulging may be gleaned from close scrutiny of the work of Bogen et al. (1980). In this theoretical study, an isotropic, initially spherical membrane model of the heart before and after infarction was used. Stroke volume was predicted over a wide range of left ventricular end-diastolic pressure for infarcts of various sizes. A difference was predicted between the immediate stage (loss of contractile function) and the acute stage (increase in myocardial extensibility) which may be attributable to paradoxical bulging. From Figure 5 of that paper, the percent fall in stroke volume from the immediate to the acute stage will depend on left ventricular end-diastolic pressure LVEDP and the size of the infarct, but does not appear likely to exceed 10%.

In the present study, the relative change in tension was estimated to characterize segmental motion. Although segment shortening might vary, depending on fiber orientation (Freeman et al., 1985) and depth in the wall (Weintrab et al., 1981), the ventricular wall contracts as a unit. This segmental wall motion is determined by the tension against which a segment contracts as opposed to the stress on a particular muscle layer. Tension, unlike stress, neglects direction and thickness. Moreover, multiple assumptions are necessary to calculate regional stress. These assumptions may not be valid, especially in a regionally ischemic ventricle (Mirsy, 1979). It is necessary to note that the calculation of tension presented here is an approximation which depends on several assumptions noted in Methods. Since tension could not be calculated exactly, changes of the index of tension were estimated to characterize ischemic segment motion behavior. In the present analysis, dynamic factors such as viscosity or inertia were of little importance, since the minimum length of the ischemic segment occurred at the nadir of LV pressure or tension. Moreover, a good correlation between the observed data and calculated data obtained with a simple elastic model justified these simplifications within one cardiac
cycle. Thus, the pattern of motion of the ischemic myocardial segment may be explained by the change in regional tension. It should be noted that the pressure-length loops of Lew et al. (1985) are similar in shape to the tension-length loops presented here. The ischemic loops formed a nearly uniform upstroke and downstroke with a small figure-of-eight pattern. There was no positive work in diastole. Thus, the manner of presentation of the data as tension-length or pressure-length does not change the overall interpretation of the data.

This entirely passive motion can be applied to only the most severely ischemic segments. Hypokinetic segments may have more complex motion, determined by both passive and active features. Furthermore, as noted in recent work by Paulus et al. (1985), there are fundamental differences in the properties of myocardium that is ischemic due to increased oxygen demand, as opposed to decreased supply, i.e., after coronary occlusion. It should also be noted that the behavior of an ischemic segment may change over time as it scars and becomes less compliant (Hood et al., 1970; Theroux et al., 1977).

Alternatively, there may be return of function due to survival of an epicardial rim that begins to contract actively, over time, although this would be more common in a reperfusion model (Theroux et al., 1976; Braunwald and Klomer, 1982). Thus, the present observations are most relevant to severe ischemia following acute coronary occlusion. In this setting, however, the mechanical properties of acutely ischemic myocardium observed here are fundamental to any analysis of regional wall motion abnormalities.

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