Experimental Myocardial Infarction
XIII. Sequential Changes in Left Ventricular Pressure-Length Relationships in the Acute Phase

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SUMMARY Diastolic pressure-length relationships of an ischemic region of the canine left ventricle were measured over a six-hour period following left anterior descending coronary artery ligation, and their evolution was compared with the extent of systolic aneurysmal bulging. Normalized ischemic segment length excursion, which after coronary artery ligation may be taken as a measure of systolic aneurysmal bulging, increased during the first hour after ligation but thereafter declined toward control values. Concurrently, reciprocal changes were demonstrated in the slope of the end-diastolic pressure-length curves obtained during transient pressure loading of the left ventricle.

These data show that the magnitude of acute systolic aneurysmal bulging following experimental coronary artery ligation is determined not only by loss of contractile function, but also by changes in passive pressure-length relationships of the myocardium. Moreover, the results indicate that development of akinesis in experimental ischemia, heretofore demonstrated only in the chronic phase of infarction, may begin within hours of the onset of myocardial ischemia.

IT HAS LONG BEEN RECOGNIZED that experimental coronary artery occlusion results in prompt systolic aneurysmal bulging in the ischemic segment.1 2 Further observations, made several days after experimental infarction, have demonstrated resolution of the aneurysmal bulge, with development of akinesis.3 These events may bear an important relationship to the degree of overall hemodynamic impairment of the left ventricle,4 and have been thought to explain, at least in part, gradual improvement in function after experimental infarction.5 In the present study, we have examined the time course of changes both in degree of systolic aneurysmal bulging and in regional diastolic pressure-length relationships in the early hours after onset of ischemia.

Methods

Eight adult mongrel dogs weighing 23.1 ± 1.2 kg were anesthetized with intravenous pentobarbital (30 mg/kg). Respiration were maintained by a Bennett Respirotor (Model PR-1), using 40% oxygen in room air. The respirator was adjusted to maintain blood gases and pH in a physiological range. A thoracotomy was performed through the fifth left intercostal space and the heart exposed in a pericardial cradle. The left anterior descending coronary artery was isolated approximately 4 cm below its origin, and a snare occluder placed around it for inducing ischemia (fig. 1). After calibration, two mercury-in-silastic segment length gauges6 were inserted into Teflon holders sutured to the epicardium (fig. 1), one in the area of distribution of the left anterior descending coronary artery, and the other onto the myocardium supplied by the circumflex branch of the left coronary artery (control gauge). The gauges were attached transversely to the ventricular surface, an orientation which has produced consistent morphology of segment length tracings in this laboratory.7 8 Previous studies have demonstrated stable linearity and gain in these gauges.2 The ascending aorta was dissected free from the pulmonary artery, permitting transient aortic constriction for loading of the left ventricle and construction of pressure-length curves, as previously described.

Hemodynamic measurements of aortic and left ventricular pressures were made by an end-hole catheter passed retrogradely from the femoral artery, and by a 6 cm long polyethylene-240 catheter (with a natural frequency of 50 Hz) inserted directly through the apex of the ventricle, respectively. The catheters were attached to Statham P23Db pressure transducers, and recordings were made on a Gould Brush 480 recorder. The zero for pressure measurements was set at mid-chest level. Left ventricular end-diastolic pressure was determined from high gain tracings at the trough of the A wave as previously described.9 After baseline measurements of heart rate, aortic pressure, left ventricular pressure, and phasic segment length tracings, the ascending aorta was gradually constricted over a period of approximately 30 sec by closing an encircling atraumatic clamp (fig. 1), so that left ventricular end-diastolic pressure and length were raised to at least 20 mm Hg (fig. 2). Recordings of aortic occlusion used for analysis were made at rapid paper speeds (50 mm/sec), permitting beat by beat calculation of rising end-diastolic pressure and length over a range of values.

After these baseline measurements were made, myocardial ischemia was induced by occluding the left anterior descending coronary artery. The resulting area of ischemia was small enough so that left ventricular and systemic hemodynamics were minimized affected. Measurements were repeated at 15 min and at one, three and six hours after coronary occlusion. At these intervals the ascending aorta was also transiently constricted and measurements of simultaneous end-diastolic pressure and length were recorded. When supplemental anesthesia was required, it was given after completion of a set of hemodynamic measurements, to allow a period thereafter for stabilization. At the end of the experiments gauges were recalibrated to ensure stable linearity and gain.

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Supported by USPHS Grants HL-14646, HL 71-2498, HE 07299 and American Heart Association Grant 71-1016.
Presented in part at the American College of Cardiology meetings, Houston, Texas, February 1975.
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Received August 18, 1975; revision accepted for publication January 30, 1976.
Calculations

When coronary ligation is produced, the ischemic segment bulges paradoxically during systole instead of shortening. Extent of the aneurysmal bulge was characterized in terms of “muscle lengths,” a normalized value for segment length which we have defined as phasic segment length amplitude/end-diastolic segment length. Normalization is required so that data obtained from gauges of different lengths may be compared.

Pressure-length relationships of the ischemic and control segments were characterized from the slope of the end-diastolic pressure-length curves generated during transient aortic constriction. A linear regression line was fitted through pressure-length points obtained in the range of 5-20 mm Hg, similar to the procedure employed by McCullagh and his colleagues for analyzing left ventricular pressure-volume curves. These data were also analyzed using an exponential curve fit, as employed by other investigators, but the correlation coefficients were not significantly different from those obtained using linear regression equations. Furthermore, calculation and tabulation of a “coefficient of stiffness” based on exponential curve fitting as described by Diamond and coworkers did not alter the conclusions of the study, hence we chose the simpler linear regression analysis for presentation here.

The values obtained from the linear slope of the pressure-length curves at 15 min, one, three and six hours were normalized to the end-diastolic length of the segment at 5 mm Hg at that point in time as shown:

\[
K = \frac{\Delta P}{\Delta L/EDSL \times 100} = \text{mm Hg/}% \Delta \text{in EDSL},
\]

where \(K\) = slope of the pressure-length relationship, \(\Delta P\) = change in end-diastolic pressure, \(\Delta L\) = change in end-diastolic length, and EDSL = end-diastolic length at an end-diastolic pressure of 5 mm Hg.

All data obtained were compared statistically using Student's paired t-test. Data in the text and tables are presented as mean ± SEM.

Results

Hemodynamic changes which occurred as a result of left anterior descending coronary artery ligation were minimal, consisting of a slight but significant rise in left ventricular end-diastolic pressure. There were no significant changes in heart rate, aortic mean pressure, or left ventricular peak sys-
Hemodynamic Data Before and After Left Anterior Descending Coronary Artery Occlusion (Mean ± SEM)

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>15 min</th>
<th>1 hour</th>
<th>3 hours</th>
<th>6 hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate (beats/min)</td>
<td>152 ± 10</td>
<td>150 ± 8</td>
<td>150 ± 6</td>
<td>143 ± 4</td>
<td>128 ± 7</td>
</tr>
<tr>
<td>Aortic mean pressure (mm Hg)</td>
<td>108 ± 6</td>
<td>102 ± 5</td>
<td>100 ± 3</td>
<td>95 ± 3</td>
<td>98 ± 5</td>
</tr>
<tr>
<td>Left ventricular peak systolic pressure (mm Hg)</td>
<td>123 ± 5</td>
<td>117 ± 4</td>
<td>115 ± 5</td>
<td>110 ± 5</td>
<td>110 ± 5</td>
</tr>
<tr>
<td>Left ventricular end-diastolic pressure (mm Hg)</td>
<td>4.3 ± 0.9</td>
<td>7.3 ± 1.3*</td>
<td>6.6 ± 1.0*</td>
<td>6.3 ± 0.8†</td>
<td>5.8 ± 0.7†</td>
</tr>
</tbody>
</table>

*P < 0.01.
†P < 0.02.

After acute coronary artery ligation, an abrupt alteration in the pattern of contraction of the ischemic segment was invariably noted; instead of shortening during ejection the segment lengthened, forming an aneurysmal bulge. The bulge gradually decreased in amplitude over a period of six hours; however, the configuration of the aneurysmal bulge was unaltered, and there was no return of systolic ejection phase shortening of the type noted in control tracings prior to occlusion. In contrast, systolic ejection phase shortening persisted in the control segments throughout the experiment (fig. 3). From a control excursion of 0.094 ± 0.021 muscle lengths the ischemic segment increased to 0.193 ± 0.020 muscle lengths at 15 min (P < 0.001, table 2). The amplitude of this dyskinetic bulge maintained a plateau up to one hour (0.187 ± 0.030 muscle lengths, P < 0.001), after which it showed a decline; at three hours it had decreased to an excursion of 0.153 ± 0.020 muscle lengths (P < 0.02), and by the sixth hour the myocardial segment had an amplitude of 0.127 ± 0.014 muscle lengths (table 2). The uninvolved (control) segment of muscle did not show any significant changes in muscle lengths during the course of the study.

The slope of the end-diastolic pressure-length relationship (fig. 4), and the K values calculated from these slopes (table 3) were also altered in the ischemic myocardial segment. Values of K decreased from a level of 1.712 ± 0.283 to 1.111 ± 0.185 (P < 0.02) after 15 min of coronary occlusion. Subsequently at 60 min the value decreased to 1.148 ± 0.185 (P < 0.01). At three and six hours values of K were 1.304 ± 0.162 (P < 0.02) and 1.473 ± 0.167, respectively, demonstrating a return to the level prior to induction of ischemia (table 3). The uninvolved segment of muscle, which served as control during the experiment, did not show any significant change in the values of K.

The early rise, followed by a decline in amplitude of segmental bulge (muscle lengths), and the early decline, followed by a rise in values of K clearly suggest a relationship between the two measurements. This is also borne out by a point-by-point analysis of all data collected in this experiment from the ischemic segment, following coronary occlusion (fig. 5). A significant inverse relationship between muscle lengths and the values of K is shown.

Change in end-diastolic segment length also occurred in both the ischemic (fig. 4) and control segments. In the ischemic segment end-diastolic segment length increased above the pre-ischemia control by 8.0 ± 1.4%, 7.1 ± 1.6%, 8.0 ± 2.4%, and 9.7 ± 2.5% at 15 min, 60 min, 3 hours and 6 hours, respectively (all P < 0.05; paired differences). Corresponding values for the control gauge were 4.5 ± 1.4%, 3.1 ± 1.5%, 2.3 ± 0.6%, and 2.8 ± 1.4% (all P < 0.05; paired differences).

Discussion

In recent years experimental and clinical evidence has been obtained indicating that both acute and chronic alterations in left ventricular stiffness are noted in myocardial ischemia and infarction. In experimental infarcts studied both in situ and at postmortem examination 3–5 days after coronary ligation in dogs, an increase in stiffness of the ischemic zone, based upon pressure-length measurements, was demonstrated, and this was shown to result in a steeper (stiffer) pressure-volume curve for the entire left ventricle, which could be attributed solely to changes taking place in the ischemic segment. Stiffening of the ischemic segment in these studies was accompanied by...
akinesia, or absence of either appreciable systolic shortening or expansion, as assessed from length gauges sutured to the epicardium of the beating left ventricle in situ. Systolic shortening of epicardial segments of the left ventricle is a well-recognized characteristic of normal myocardium, and systolic bulging is clearly noted to occur in the early phase of ischemia within seconds after onset of coronary occlusion.1,2,6 It follows, therefore, that the changes observed within several days after infarction resemble neither normal nor acutely ischemic myocardium and, in order to be fully understood, require a study both of timing of onset and of mechanism.

In contrast to these findings, other investigators have obtained data suggesting that opposite changes in stiffness may occur in the early phase of experimental myocardial ischemia.11 It has been shown that stiffness of the isolated heart studied one hour after experimental infarction, based upon pressure-volume curves, is decreased compared to the control state. The results of the present experiment support this finding in the intact beating heart, with significant decreases in the slope of the diastolic pressure-length relationship occurring as early as 15 minutes after coronary occlusion. Thus the overall time course of stiffness change in experimental infarction may involve an initial decrease as well as a subsequent increase in stiffness. However, the initial decrease in the slope of the pressure-length relationship after ischemia in intact hearts, as demonstrated in our experiment, is contradictory to results obtained by other investigators,14 who examined deeper layers of myocardium using ultrasonic micrometer gauges. This disparity in results could be due to the different methodology used.

The results of the present investigation further allow an analysis of the time of onset of the delayed increase in the slope of the pressure-length relationship. The lowest values of K were obtained 15 min after coronary occlusion (table 3), and by one hour values had begun to show a return toward control levels. By the end of six hours, values of K did not differ significantly from control (pre-occlusion) values, indicating that the slope of the diastolic pressure-length relationship had returned to normal. These same changes appear to explain the gradual attenuation of the aneurysmal bulge which developed immediately after coronary occlusion (table 2, figs. 3, 5). An alternate explanation for gradual attenuation of the aneurysmal bulge would be a partial return of function to the local segment with

### Table 2. Sequential Change in Muscle Lengths Following Coronary Occlusion

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>15 min</th>
<th>1 hour</th>
<th>3 hours</th>
<th>6 hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0.059</td>
<td>0.230</td>
<td>0.259</td>
<td>0.235</td>
<td>0.170</td>
</tr>
<tr>
<td>2</td>
<td>0.038</td>
<td>0.114</td>
<td>0.092</td>
<td>0.093</td>
<td>0.094</td>
</tr>
<tr>
<td>3</td>
<td>0.062</td>
<td>0.235</td>
<td>0.132</td>
<td>0.160</td>
<td>0.139</td>
</tr>
<tr>
<td>4</td>
<td>0.073</td>
<td>0.195</td>
<td>0.153</td>
<td>0.112</td>
<td>0.079</td>
</tr>
<tr>
<td>5</td>
<td>0.081</td>
<td>0.153</td>
<td>0.160</td>
<td>0.104</td>
<td>0.097</td>
</tr>
<tr>
<td>6</td>
<td>0.070</td>
<td>0.121</td>
<td>0.125</td>
<td>0.113</td>
<td>0.114</td>
</tr>
<tr>
<td>7</td>
<td>0.171</td>
<td>0.243</td>
<td>0.288</td>
<td>0.209</td>
<td>0.196</td>
</tr>
<tr>
<td>8</td>
<td>0.200</td>
<td>0.256</td>
<td>0.286</td>
<td>0.198</td>
<td>0.127</td>
</tr>
<tr>
<td><strong>Mean ± SEM</strong></td>
<td>0.094 ± 0.193</td>
<td>0.187 ± 0.153</td>
<td>0.127 ± 0.021</td>
<td>±0.020</td>
<td>±0.030</td>
</tr>
<tr>
<td><strong>P</strong></td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.020</td>
<td>&gt;0.100</td>
<td></td>
</tr>
</tbody>
</table>

### Table 3. Sequential Change in Slope of the Diastolic Pressure-Length Relationship (K) Following Coronary Occlusion

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>15 min</th>
<th>1 hour</th>
<th>3 hours</th>
<th>6 hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1.333</td>
<td>0.821</td>
<td>0.957</td>
<td>0.676</td>
<td>0.864</td>
</tr>
<tr>
<td>2</td>
<td>2.885</td>
<td>1.545</td>
<td>1.698</td>
<td>1.822</td>
<td>2.163</td>
</tr>
<tr>
<td>3</td>
<td>0.983</td>
<td>0.882</td>
<td>0.718</td>
<td>0.620</td>
<td>0.765</td>
</tr>
<tr>
<td>4</td>
<td>1.743</td>
<td>1.018</td>
<td>1.498</td>
<td>1.538</td>
<td>1.704</td>
</tr>
<tr>
<td>5</td>
<td>1.929</td>
<td>0.829</td>
<td>1.162</td>
<td>1.538</td>
<td>1.862</td>
</tr>
<tr>
<td>6</td>
<td>2.777</td>
<td>2.195</td>
<td>1.950</td>
<td>1.541</td>
<td>1.471</td>
</tr>
<tr>
<td>7</td>
<td>1.122</td>
<td>1.046</td>
<td>0.709</td>
<td>0.969</td>
<td>1.434</td>
</tr>
<tr>
<td>8</td>
<td>0.925</td>
<td>0.531</td>
<td>0.459</td>
<td>0.926</td>
<td>1.522</td>
</tr>
<tr>
<td><strong>Mean ± SEM</strong></td>
<td>1.712 ± 1.111</td>
<td>1.148 ± 1.204</td>
<td>1.473 ± 0.283</td>
<td>±0.185</td>
<td>±0.185</td>
</tr>
<tr>
<td><strong>P</strong></td>
<td>&lt;0.020</td>
<td>&lt;0.010</td>
<td>&lt;0.020</td>
<td>&gt;0.400</td>
<td></td>
</tr>
</tbody>
</table>

**Figure 4.** Linear regression analysis in one experiment, of end-diastolic pressure versus end-diastolic length in a segment of ischemic myocardium prior to and one and six hours following coronary occlusion. The line is shifted to the right and shows a decreased slope at one hour; at six hours the rightward shift persists, but the slope of the line has increased.

**Figure 5.** Linear regression analysis between muscle lengths (ML), a measure of paradoxical bulge, and slope of the pressure-length relationship (K) following ischemia, demonstrating a significant inverse correlation. Plotted points include values obtained for all eight animals over the time course of the experiment (N = 32).
passage of time, enabling the segment to better resist passive stretch. However, this seems unlikely in that there was no return of systolic shortening in the ischemic segment despite a decline in bulge amplitude (fig. 3).

Although prolonged measurements were not carried out, it may be that changes which were first observed two hours after coronary occlusion represent the onset of gradual stiffening which culminates in akinesis several days later. If this is the case, the initial mechanism clearly cannot be attributed to infiltration with granulation tissue, known to occur after several days. The process observed in the early stages may be analogous to the well-known phenomenon of rigor mortis observed in the asphyxiated myocardium; stiffening of the myocardium is known to begin within 40 to 60 min after death. The process may be due to exhaustion of high energy phosphate stores in the ischemic myocardium.

In the present study, increases in end-diastolic segment length were observed in both ischemic and control segments after coronary occlusion, attributable in part to the slight but significant increase in end-diastolic pressure (table 1). However, the incremental increase was greater in the ischemic segment, suggesting a viscous effect upon end-diastolic length. Increases in length localized to the ischemic segment have been observed in an experimental ischemia preparation in which small (1 cm diameter) lesions, not large enough to raise left ventricular end-diastolic pressure, were produced. These observations are consistent with the demonstration of myofibrillar relaxation in electron microscopic views of ischemic myocardial segments subjected to repeated stretching by the intracavity pressure head. In contrast, myocardium which is studied in isolated systems, and therefore not subjected to repeated stretching, may show contracture rather than stress-relaxation.

The observed shift to the right of the end-diastolic pressure-length curves in focal ischemia is therefore consistent with the development of stress-relaxation. The additional alteration in slope of the pressure-length relationship may or may not be directly related to the phenomenon described above, although some authors have hypothesized that "disruption of the myofibrils or supporting elements" might contribute to a stiffness change as well as to stress-relaxation.

It should be emphasized that the changes in slope of the diastolic pressure-length curves demonstrated in this study, though they do affect passive resistance to filling of the ventricle, may not reflect true changes in stiffness of the ischemic myocardial segment. Since radius of curvature and wall thickness of the ischemic segment were not assessed, calculation of true stress-strain relationships within the segment are not possible. Furthermore, correction for thinning of the ventricular wall, known to occur in areas of ischemia, would most likely tend to return the rightward-shifted curves of acutely ischemic segments back toward the control position, although the effect this would have on the slope of pressure-length relationships is unknown.

These altered properties of the myocardium may have implications regarding the mechanical function of the ventricle both in systole and in diastole. In the first few hours after onset of ischemia presumably the decrease in passive resistance to filling of the nonfunctioning ischemic segment permits a degree of aneurysmal bulging exceeding that which would be present had the ventricle retained its original properties. These changes may pose an additional mechanical burden for the remaining normal myocardium by causing sequestration of potential stroke volume into the bulging segment. Furthermore, during diastole the occurrence of stress relaxation and increase in end-diastolic length in the ischemic segment may indicate that the ventricle is operating at an increased volume for a given filling pressure. During the next few hours the amplitude of the bulge decreases, and this would tend to limit sequestration of potential stroke volume.

It has also been demonstrated that patients with chronic coronary artery disease and patients recovering from acute myocardial infarction may show increased left ventricular stiffness when compared with patients having no evidence of cardiac disease. However, degree of stiffening may vary considerably, depending upon the extent of fibrosis of the ischemic segment. Clinical studies very early in the time course of infarction, which might be compared with the observations of the present study, are not yet available. These observations in experimental animals may suggest that a gradual increase in stiffness following myocardial infarction, with the attendant improvement in mechanical advantage for the remaining noninfarcted myocardium, may begin quite early after onset of ischemia, and that the mechanism is unlikely to be due to infiltration of new cellular elements into the infarct. In addition, in the very acute stages of infarction, unanticipated mechanical burdens might be imposed due to an actual decrease in passive resistance to filling of the ischemic zone.

Acknowledgment

We thank Herbert Kayne, Ph.D., and Roger G. Mark, M.D., for their help in statistical evaluation. The technical assistance of Mr. Leon T. Snyder and secretarial help of Ms. Deborah Smith and Ms. Leanne Gitell are gratefully acknowledged.

References

Radionuclide Assessment of Nitroglycerin Influence on Abnormal Left Ventricular Segmental Contraction in Patients with Coronary Heart Disease

ANTONE F. SALEL, M.D., DANIEL S. BERMAN, M.D., GERALD L. DE NARDO, M.D., AND DEAN T. MASON, M.D.

SUMMARY Noninvasive gated cardiac blood pool imaging with technetium-99m autologous erythrocytes was employed to differentiate reversible versus irreversible abnormal ventricular segmental contraction by regional wall and pump responses to sublingual nitroglycerin in 25 patients with chronic coronary heart disease. In 12 patients without ECG infarctions compared to 13 with infarctions, radionuclide images demonstrated significantly greater percent decreases in end-systolic volumes (33.8 ± 6.7 SEM vs 18.7 ± 4.4; P < 0.05) without differences in percent reductions in end-diastolic volumes (13.7 ± 3.9 vs 11.6 ± 6.1; NS) and thereby significantly greater percent increases in ejection fractions (9.3 ± 1.6 vs 4.1 ± 2.0; P < 0.05). In the 22 patients with regional dyssynergy, improvement in disordered pattern and extent of localized dyssynergy following antianginal action of nitroglycerin was related to ECG absence of prior infarction. These observations demonstrate the clinical accuracy of atraumatic scintigraphy in the detection of reversible localized dyssynergy due to myocardial ischemia in coronary heart disease.

SINCE CHRONIC ATHEROSCLEROSIS affecting segmental branches of the coronary arterial system results in regional disorders of ventricular contraction,1,2 considerable investigation has been directed toward the differentiation of localized abnormalities of wall motion due to myocardial ischemia versus necrosis.3,4 However, the methods which have been developed for the identification of ventricular ischemia by demonstration of reversible left ventricular dyssynergy require cardiac catheterization with radiopaque angiography and are limited in clinical application. Therefore, an important need remains for the noninvasive detection and assessment of abnormal ventricular segmental contraction caused by myocardial ischemia, as opposed to infarction, in the management and prognostic evaluation of patients with chronic coronary heart disease.

This study evaluates the ability of technetium-99m autologous red blood cell scintillation camera ventriculography with nitroglycerin to differentiate areas of ventricular dyssynergy due to myocardial ischemia from those consequent to necrosis in a large group of patients with chronic coronary heart disease proven by cardiac catheterization. This technique allows us to measure ventricular volumes at the same time that we are analyzing chamber wall motion.5-11

Methods and Materials

Twenty-five patients, 20 males and five females, ranging in age from 35 to 50 years, with chronic coronary artery disease demonstrated by cardiac catheterization and selective coronary arteriography, were selected for study. Significant coronary artery disease was defined as 75% or greater stenosis of one or more of the three major coronary vessels. Dyssynergy on radiopaque left ventriculography was defined as a localized abnormality of left ventricular contraction...
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_Circulation_. 1976;53:970-975
doi: 10.1161/01.CIR.53.6.970

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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