Relation Between Left Coronary Artery Stenosis and Regional Left Ventricular Function

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SUMMARY The effect of stenosis of the left main and proximal anterior descending coronary arteries on left ventricular wall dynamics was investigated in 70 patients with ischemic heart disease by the use of roentgen videometric analysis of left ventricular angiograms. In all patients with ischemic heart disease, mean values for peak rate of systolic wall thickening and diastolic wall thinning were significantly smaller than normal ($P < 0.01$). In patients without infarction, there was no correlation between peak rate of systolic anterior wall thickening and stenosis of the coronary artery supplying it, but there was a significant reduction in peak rate of diastolic wall thinning ($P < 0.01$) in patients with stenosis greater than 90%; this difference was not apparent at any lower degree of stenosis. This population could not be recognized by any other parameter of global or regional ventricular function; thus, diastole is more sensitive to regional left ventricular dysfunction than systole.

THE SIGNIFICANCE OF REDUCTION of coronary blood flow and its relation to the severity of coronary artery stenosis in ischemic heart disease has been difficult to assess, partly because of the inability to quantitate collateral coronary flow, and because the capacity of the coronary flow is generally adequate to maintain normal resting demands in the presence of stenoses up to 85%. Techniques using isotope indicator washout and catheter flow probes provide no information regarding anatomic definition of the coronary artery stenoses or the functional state of the myocardium supplied by stenotic coronary arteries. The relationship between resting coronary blood flow and regional systolic left ventricular function has been established in animals but not in man. Since left ventricular dysfunction may be recognized in diastole before systole, we investigated the effects of stenosis of the left main and proximal left anterior descending (LAD) coronary arteries on global function and on regional systolic thickening and diastolic thinning of the anterior left ventricular wall (LVW). Left ventricular angiograms were analyzed by roentgen videometry in 70 patients with ischemic heart disease (pre-angiographic diagnosis) and in 20 normal patients (post-angiographic diagnosis of normal coronary vessels).

Methods

The left ventricular angiograms of patients who underwent clinically indicated left heart catheterization, biplane left ventricular angiography, and coronary arteriography were analyzed. The patients were divided into two groups. One group consisted of 20 patients investigated for atypical chest pain with normal electrocardiograms, negative treadmill exercise electrocardiograms, and completely normal coronary arteriograms. Twelve were male and eight were female. They ranged in age from 29–64 years, with a mean age of 48 years.

The second group consisted of 70 patients with ischemic heart disease with grade 2–4 angina (Canadian Heart Association classification) and abnormal electrocardiograms during treadmill exercise. Thirteen patients were female and 57 were male. They ranged in age from 37–70 years, with a mean age of 54 years. Twenty-three patients had electrocardiographic
Evidence of transmural myocardial infarction, of which 13 were inferior and 10 anterior. The 70 consecutive patients with ischemic heart disease (seen from August 1974–February 1975) were selected for this study on the basis of the following criteria: 1) There was no evidence of valvular heart disease, congestive heart failure or systemic hypertension. 2) No medication known to influence left ventricular function had been taken within 48 hours of the study. 3) Left ventriculograms technically suitable for videometric analysis were available. 4) The left main and proximal LAD coronary arteries were fully visualized in at least two nearly orthogonal views on the projected coronary arteriogram and permitted accurate measurement of coronary artery stenosis. 5) The left main or LAD coronary artery had a calculated stenosis of at least 50%. Simultaneous biplane left ventriculograms were obtained in the 30° right anterior oblique (RAO) and 60° left anterior oblique (LAO) projections and recorded on cine and videotape. Ventricular opacification was achieved with the use of 50 ml of diatrizoate meglumine (Renografin-76) injected at 15 ml/sec during maintained deep inspiration. Biplane video images were recorded as a single split-screen image at 60 fields/sec, with a simultaneous electrocardiogram, as previously reported. Coronary arteriography was then performed by the Sones or Judkins technique.

Ventricular volume indices (end-systolic and end-diastolic), calculated stroke volume, ejection fraction, and cardiac indices were determined in each patient from biplane left ventriculograms by use of the roentgen videometry system. This operator-interactive video-computer system has been previously described. The volume-computing program was based on Simpson's rule, and only sinus beats were analyzed.

The videometry system provides automatic recognition of the roentgen angiographically outlined endocardial and epicardial surfaces, as indicated by the highlighted endocardial and epicardial ventricular surfaces in the stop-action video display of the left ventricular angiograms (fig. 1). Measurements of LVW thickness were made using the television raster lines and the brightened endocardial and epicardial surface lines (fig. 1), and XY coordinates were generated at the intercepts between the two. Thus, two strings of XY coordinates were obtained, one for the endocardial surface and one for the epicardial surface of the region of midanterior LVW selected for analysis. Wall thickness was then computed as the average of multiple estimates of the shortest distance between epicardial and endocardial XY coordinates in the region analyzed (fig. 1). This distance between endocardial and epicardial brightened surface lines measured along the TV raster lines is equal to wall thickness only if the wall is imaged at right angles to the television raster lines. The greater the angle between the video raster lines and the wall, the larger the segment of wall that must be analyzed (segment indicated by two horizontal enhanced video scan lines) (fig. 1). Consequently, the closer to a right angle the wall is in relation to the television raster lines, the narrower the segment of LVW that can be analyzed and the greater the likelihood that the same location on the wall will be measured throughout the cardiac cycle. For this reason, the video camera was rotated through 45° so that the angle of incidence between the TV raster lines and the anterior LVW on the videotape angiograms in the 30° RAO projection was approximately a right angle.

The region of anterior wall analyzed was the same in all patients, that is, midway between the aorto-left ventricular junction and the apex of the heart. This region was selected because: 1) its blood supply is from the LAD coronary artery, whereas the anterior LVW more distally may derive part of its blood supply from the left circumflex or right coronary artery via the posterior interventricular artery, and 2) using two fixed points — the aorto-left ventricular junction and the apex — the region midway between them could be located accurately. This ensured that the wall analyses were performed on comparable regions of the anterior wall in the 70 hearts. Plots of

**Figure 1.** *Left ventricular wall thickness determination. Stop-action video display of biplane roentgen angiogram with simultaneous epicardial and endocardial border recognition (woman, 59 years old, 50 ml Renografin injection). Magnified view at right.*
continuous anterior LVW dimension were obtained in which minimum and maximum thicknesses were identified by computer, and percentage systolic wall thickening was calculated. In addition, the maximum positive and negative time rates of change of wall thickness were obtained after a running 3-point average smoothing routine was applied to the continuous wall thickness plot, which had 60 data points/sec (that is, one data point every 16.7 msec). The computer program identified the positive and negative peak rates of change of thickness, which represent the peak rates of wall thickening and wall thinning, respectively. The computer program then computed the tangents at these points and superimposed them on the wall thickness plot (fig. 2).

These two parameters together with percentage systolic wall thickening were used to assess regional left ventricular function. The reproducibility of this technique has been reported in previous studies.11

The severity of stenosis of the left main and LAD coronary arteries supplying the anterior LVW was determined by caliper measurements of the diameter of the lumen at the point of most severe stenosis and that of the immediately adjacent normal segment in at least two near-orthogonal views of the projected coronary arteriogram. The luminal cross-sectional area of the stenotic region and that of the immediately adjacent normal coronary segment were then calculated. The severity of the stenosis was expressed as the percentage reduction in cross-sectional area from the normal. Appropriate mathematical incorporation of other factors known to influence vessel resistance (that is, stenosis length and number of less severe stenoses) did not significantly alter assessment of vessel flow resistance and was therefore not used in this study.

Statistical analysis of differences was by the t test.

Results

For both groups, the global parameters of left ventricular function and hemodynamics are shown in table 1 and the regional parameters of left ventricular function are shown in table 2.

Total Left Ventricular Function

Normal Patients

All patients in this group had normal left heart pressures, cardiac indices, and left ventricular and coronary cineangiography (table 1).

Patients With Ischemic Heart Disease

The 70 patients with ischemic heart disease were divided into three subgroups: 1) patients with previous anterior myocardial infarction, 2) patients with inferior infarction, and 3) patients without infarction. Average left ventricular end-diastolic pressure was not significantly different in these subgroups, but was above normal (> 15 mm Hg) in all groups ($P < 0.01$). In patients without infarction, there was no significant reduction in ejection fraction from normal; nor was there any correlation between ejection fraction and severity of coronary stenosis. Ejection fraction was significantly ($P < 0.01$) lower in patients with either anterior or inferior infarction than in those without infarction. In patients without infarction, end-diastolic and end-systolic volume indices were not significantly different from normal, but in patients with either

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**Table 1. Global Measurements of Left Ventricular Function and Hemodynamics (Mean ± SD)**

<table>
<thead>
<tr>
<th>Patients</th>
<th>No.</th>
<th>EDVI (ml/m²)</th>
<th>ESVI (ml/m²)</th>
<th>EF (%)</th>
<th>LVEDP (mm Hg)</th>
<th>CI (l/min/m²)</th>
<th>HR (beats/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>20</td>
<td>79.6 ± 16.3</td>
<td>23.3 ± 6.68</td>
<td>71 ± 7</td>
<td>11.4 ± 2.8</td>
<td>4.64 ± 1.30</td>
<td>75.8 ± 9.2</td>
</tr>
<tr>
<td>Anterior infarction</td>
<td>10</td>
<td>*119.2 ± 40.1</td>
<td>68 ± 36.4</td>
<td>44 ± 13</td>
<td>18.5 ± 7.2</td>
<td>*3.62 ± 1.32</td>
<td>70 ± 7.5</td>
</tr>
<tr>
<td>Inferior infarction</td>
<td>13</td>
<td>*96.7 ± 35.8</td>
<td>48.6 ± 32.5</td>
<td>53 ± 17</td>
<td>18.3 ± 6.2</td>
<td>*3.63 ± 1.32</td>
<td>75.2 ± 14.1</td>
</tr>
<tr>
<td>Stenosis ≥90%</td>
<td>20</td>
<td>84.05 ± 19.5</td>
<td>33.1 ± 16.8</td>
<td>61 ± 15</td>
<td>15.7 ± 6.7</td>
<td>*3.68 ± 0.98</td>
<td>70.8 ± 11.5</td>
</tr>
<tr>
<td>without infarction</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stenosis &lt;90%</td>
<td>27</td>
<td>83.97 ± 22.8</td>
<td>28.6 ± 11.53</td>
<td>62 ± 15</td>
<td>15.1 ± 5.7</td>
<td>*3.82 ± 1.33</td>
<td>69.6 ± 12.4</td>
</tr>
</tbody>
</table>

*P < 0.05.
**P < 0.02.
***P < 0.01.

Differences between other categories within a column are not significant except end-systolic volume index, in which the value for anterior infarction (68 ± 36.4) was significantly different from those of patients without infarction and with either >90% stenosis or <90% stenosis, $P < 0.001$.

Abbreviations: EDVI = end-diastolic volume index; ESVI = end-systolic volume index; EF = ejection fraction; LVEDP = left ventricular end-diastolic pressure; CI = cardiac index; HR = heart rate.
Table 2. Regional Measurements of Left Ventricular Function and Hemodynamics (Mean ± SD)

<table>
<thead>
<tr>
<th>Patients</th>
<th>No.</th>
<th>Peak rate of systolic wall thickening (dTs/dt), mm/sec</th>
<th>Peak rate of diastolic wall thinning (dTd/dt), mm/sec</th>
<th>% systolic wall thickening</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>20</td>
<td>44.9 ± 8.1</td>
<td>55 ± 6.1</td>
<td>77 ± 33</td>
</tr>
<tr>
<td>Anterior infarction</td>
<td>10</td>
<td>*16.4 ± 10.2</td>
<td>*19.2 ± 14.2</td>
<td>*36 ± 14</td>
</tr>
<tr>
<td>Inferior infarction</td>
<td>13</td>
<td>*19.5 ± 10.7</td>
<td>*21.5 ± 11.2</td>
<td>*39 ± 15</td>
</tr>
<tr>
<td>Stenosis ≥90% without infarction</td>
<td>20</td>
<td>*31.6 ± 17.7</td>
<td>*24.9 ± 18.3</td>
<td>150 ± 28</td>
</tr>
<tr>
<td>Stenosis &lt;90% without infarction</td>
<td>27</td>
<td>*32.4 ± 17.2</td>
<td>*39.7 ± 20.9</td>
<td>65 ± 35</td>
</tr>
</tbody>
</table>

*P < 0.01.
**P < 0.001.
Differences between other categories within a column are not significant except where indicated.

Regional Left Ventricular Function

Normal Patients

The mean values and range for peak rate of systolic wall thickening (dTs/dt), peak rate of diastolic wall thinning (dTd/dt) (where dt is 16.7 msec — that is, the time between video fields at 60/sec), and percentage systolic wall thickening are shown in table 2.

Patients With Ischemic Heart Disease

For all three subgroups the anterior regional dTs/dt and dTd/dt were significantly reduced (P < 0.01) from normal (table 2). Mean values for both systolic and diastolic anterior regional wall dynamics and percentage systolic wall thickening were significantly lower in patients with infarction than in those without infarction (P < 0.01) (fig. 3). For patients without infarction, there was no correlation between the severity of left main and LAD coronary artery stenosis and regional anterior systolic left ventricular function (fig. 4). However, patients without infarction with stenosis of the left main or proximal LAD coronary artery greater than 90% showed significant reduction in peak rate of regional diastolic wall thickening compared with those without infarction with stenosis of less than 90% (fig. 5). There were no significant differences in regional diastolic wall function when these patients were examined with respect to other degrees (80%, 70% or less) of coronary stenosis.

Discussion

Increased myocardial oxygen demands are met by the coronary flow reserve, which is the capacity of the coronary circulation for increasing the myocardial blood flow. This reserve ranges from three to seven times basal flow, and it maintains normal resting flow in the presence of coronary stenosis of up to 85%. This capacity for increasing the resting blood flow is abolished by stenoses greater than 88–93%, after which a further increase in the severity of stenosis disproportionately reduces coronary blood flow. For example, an increase in stenosis from 90–95% reduces resting coronary flow by a factor of two. It is not surprising, therefore, that measures of resting coronary blood flow are an insensitive means of assessing the severity of coronary artery disease. The usefulness of measures of resting coronary blood flow are also limited insofar as collateral flow cannot be quantitated, and approximately 75% of patients with stenoses greater than 90% have collaterals. Tennant and Wiggers reported changes in the pattern of contraction in segments of the left ventricle supplied by a coronary artery in which the blood flow had been reduced experimentally, and similar changes...
have been demonstrated in patients with coronary artery disease. More recently, other investigators have shown that LVW dynamics, as measured by percentage systolic wall thickening and velocity of muscle shortening, are sensitive to the degree of reduction of regional myocardial perfusion and the functional status of the myocardium. We therefore examined the coronary arteriograms of 70 patients with ischemic heart disease to examine the relationship between regional myocardial wall dynamics in systole and diastole and the severity of stenosis of the coronary arteries supplying the regions.

In all three subgroups of patients with ischemic heart disease, mean values for peak rates of systolic wall thickening and diastolic wall thinning in the anterior wall were significantly less than normal (table 2). Both systolic and diastolic wall dynamics were still lower ($P < 0.01$) in patients with infarction. Although mean values were significantly different, there was overlapping in the distribution of these two parameters in the infarction and noninfarction groups, so that individual patients with and without infarction could not be separated on the basis of wall dynamics alone. In patients with transmural anterior infarction, the reduced anterior wall dynamics might be explained by myocardial scarring and fibrous replacement of the anterior wall. However, systolic and diastolic dynamics of the anterior LVW in patients with inferior infarction were the same as those in patients with anterior infarction. This may be explained by 1) increased myocardial stiffness due to elevated end-diastolic pressure and chamber volume (table 1), 2) myocardial scarring and fibrosis extending into or adjacent to the anterior wall from the inferior infarction, and 3) the presence of an average stenosis of the left main or LAD coronary artery for these 13 patients of 79%.

In patients without infarction there was no correlation between the peak rate of systolic thickening of the anterior wall and the severity of reduction in cross-sectional area of the coronary arteries supplying it. The lack of correlation between regional systolic function and coronary stenosis parallels the findings of Falsetti et al. that there was no correlation between the degree of coronary stenosis and global left ventricular function. In contrast, there was a correlation between peak rate of diastolic thinning of the anterior wall and severity of coronary stenosis, in that patients with
stenoses greater than 90% had significantly lower values than did those with stenoses less than 90%. This difference in diastolic wall dynamics was not apparent at lower percentages of coronary arterial stenosis. In addition, these patients with stenosis greater than 90% could not be recognized by any other parameter of regional or global function, such as stroke volume, ejection fraction, end-diastolic pressure or systolic wall thickening. The reduction in magnitude of peak rate of diastolic wall thinning in these patients was not due to any difference in cardiac index. Since resting coronary blood flow is normal in patients with stenoses up to 85% and decreases drastically in the presence of stenoses greater than 90%, at which point the perfusion distribution also becomes abnormal, diastolic wall properties may reflect alteration in resting regional coronary blood flow.

Systolic left ventricular abnormalities have long been documented in regions with reduced coronary blood flow. Although systolic parameters have been exhaustively investigated in an attempt to accurately assess cardiac pump function, only recently have diastolic disturbances of ventricular function been recognized. There is evidence that measurements of diastolic function are more sensitive indicators of left ventricular dysfunction than are those of systolic function. This has been shown both in isolated cardiac muscle preparations and in patients with angina, the latter findings having been confirmed in our study. Diastolic relaxation can be radically impaired by both chemical and mechanical interventions in the presence of either no change or even augmentation in systolic function. In patients with angina, changes in left ventricular end-diastolic pressure-volume relations suggest increased ventricular stiffness and reduced compliance as a manifestation of "slowed or incomplete" relaxation or increased stiffness of the passive myocardium, or both.

The rate-governing step in cardiac muscle relaxation appears to be the energy-requiring sequestration of calcium by the sarcoplasmic reticulum, which is known to be sensitive to hypoxia. Katz and Tada have indicated that during chronic ischemia, alterations in high-energy phosphate stores may be responsible for this "failure" in relaxation. Reduction in peak rates of diastolic LVW thinning has not been described in ischemic heart disease previously. One consequence of slowed or incomplete relaxation is that systolic wall tension persists into the early diastolic isovolumic period and may account for the very abnormal inward and outward wall movements resulting in the isovolumic shape changes that are so characteristic of ischemic heart disease. Although systolic wall dynamics distinguished patients with ischemic heart disease from normals, and in addition distinguished the presence or absence of prior infarction, there was no correlation between systolic wall dynamics and severity of stenosis of the coronary arteries in patients without infarction. In diastole, however, there is such a correlation, but only in patients with coronary artery stenosis greater than 90%, in whom resting coronary blood flow is most likely to be reduced.

Acknowledgments

We wish to acknowledge the technical assistance of James L. Fellows and Donald L. Cravath.

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Computed Tomography for Localization and Sizing of Experimental Acute Myocardial Infarcts

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SUMMARY Computed tomography (CT) has been used to quantitate acute myocardial infarct size in isolated, arrested canine hearts. Acute myocardial infarcts were produced in 20 hearts by either left anterior descending (13 dogs) or circumflex coronary artery ligation (seven dogs). Each animal was given iodinated contrast media intravenously immediately before sacrifice 24-72 hours postinfarction. All infarcts greater than 1 g and one of three infarcts 0.5 g or less were detected by CT imaging. Infarct volume determined by CT correlated with gross infarct weight (r = 0.83). CT imaging, however, consistently underestimated infarct volume; understimation was largest in a group of patchy, predominantly subendocardial infarcts. As adequate equipment and techniques for in vivo studies are developed, CT imaging of the heart may become important in clinical evaluation of myocardial infarction.

BODY COMPUTED TOMOGRAPHY (CT) has stimulated interest in the possible use of CT in cardiac diagnosis. Early studies by several groups demonstrated the ability of CT to detect experimental myocardial ischemia and infarction in vitro. The very sensitive capacity of CT to resolve small differences in x-ray attenuation enables identification of areas of ischemia and infarction. However, it has not been clear whether CT is capable of accurate sizing of acute myocardial infarcts. This in vitro study was performed to assess the potential of CT to identify, localize, and quantitate experimental acute myocardial infarcts in isolated canine hearts and thus to help further establish the potential for CT cardiac imaging in vivo.

Materials and Methods

Twenty adult mongrel dogs weighing 15-25 kg were evaluated in this study. These animals were anesthetized with intravenous pentobarbital, intubated and ventilated on a Harvard respirator. Their chests were opened through a left thoracotomy, and either the proximal left anterior descending coronary artery (13 dogs) or the circumflex coronary artery (seven dogs) was permanently ligated. Their chests were closed and the animals were returned to their...
Relation between left coronary artery stenosis and regional left ventricular function.
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