Systolic Thickening and Thinning of the Septum and Posterior Wall in Patients with Coronary Artery Disease, Congestive Cardiomyopathy, and Atrial Septal Defect

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SUMMARY Echocardiographic septal and posterior wall thicknesses and the percent change with systole were measured in 146 patients with the following diagnoses: acute myocardial infarction (40), chronic coronary artery disease (49), congestive cardiomyopathy (8), atrial septal defect (20), and no cardiac disease (29). Mean diastolic thicknesses for the groups of patients with coronary artery disease and congestive cardiomyopathy were not significantly different from normal although there were abnormal values for individual patients within each group. Mean diastolic thickness of the septum was greater than normal for the group with atrial septal defect ($P < 0.02$). Wall thinning with systole was associated with acute infarction or ischemia ($P < 0.0001$); decreased thickening (less than normal) commonly occurred in patients with acute myocardial infarction, chronic coronary artery disease, and congestive cardiomyopathy. Patients with atrial septal defect had normal thickening with abnormal motion.

Results of this study show that 1) systolic thinning is indicative of an acute event; 2) abnormal changes in systolic wall thinning occur commonly in patients with coronary artery disease or congestive cardiomyopathy; and 3) abnormal wall motion may occur without abnormal wall thickening, as the echoes of patients with atrial septal defect indicate.

SEGMENTAL ABNORMALITIES of left ventricular wall motion have been demonstrated echocardiographically in patients with angiographically demonstrated coronary artery obstruction $^{1-2}$ and in patients with acute myocardial infarction. $^{3-4}$ Motion in noninfarcted areas is often greater than normal. $^2$ These studies suggest that the abnormal left ventricular echo motion in these patients is related to segmental differences in myocardial fiber shortening. Echo motion may also be affected by other factors such as 1) motion of the entire heart within the chest; 2) unloading during systole; 3) electrical conduction; $^2$ $^4$ 4) relative diastolic volumes of the right and left ventricles; $^7$ $^8$ and 5) cardiac surgery. $^9$ On the basis of experimental data, Ross and Franklin have suggested that dynamic changes in wall thickness might be used to study regional changes in contractile function caused by ischemia. $^{10}$

Because systolic thickening may not be influenced by the variety of factors affecting wall motion, and may therefore be more specifically related to contractility, the present study was undertaken in order 1) to determine whether there are changes in echocardiographic wall thickening in humans with coronary artery disease; 2) to see if these changes might distinguish acute myocardial infarction from chronic coronary artery disease; and 3) to see if changes in systolic thickening are related to changes in wall motion.

Materials and Methods

Patients

The eight groups of patients studied included the following: 29 with no clinical evidence of cardiac disease; 20 with acute anterior myocardial infarction; 20 with acute inferior myocardial infarction; 12 with chronic coronary artery disease and prior myocardial infarction; 28 with chronic coronary artery disease and no history of prior infarction; 8 with congestive cardiomyopathy; 20 with atrial septal defect; and 18 (9 from the normal group and an additional 9 with chronic coronary artery disease) who had echocardiography performed during handgrip stress. All patients except those with acute myocardial infarction had cardiac catheterization to confirm the diagnosis. A diagnosis of chronic coronary artery disease was made when there was 75% or greater narrowing of one or more of the three major coronary arteries. The 29 patients with no clinical evidence of cardiac disease were considered normal.

Echocardiograms

All echocardiograms were obtained with a Smith Kline echograph having a repetition rate of 1000 pulses/sec and utilizing a single element 2.25 mHz transducer, 12 mm in diameter and collimated to 7.5 cm. Sector scans of the left ventricle were obtained from the left sternal border by previously described techniques. $^{11}$ Echocardiograms of patients undergoing cineangiography were obtained the day prior to cardiac catheterization and were recorded on an Electronics-for-Medicine DR8 multichannel strip chart recorder. All patients who underwent handgrip stress studies had normal echocardiographic septal and posterior left ventricular wall motion at rest. The stress for each patient consisted of maintaining 33% of his previously determined max-

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imum voluntary contraction for three minutes or until chest pain occurred. A spring-loaded calibrated handgrip dynamometer was used. The transducer was held in the same place on the chest throughout the control, stress, and recovery periods; and echocardiographic measurements were taken during the control period, at 3 min of sustained handgrip or at the onset of chest pain, and at 1 min after handgrip release.

Patients with acute myocardial infarction were examined echocardiographically within 48 hours of admission to a coronary care unit. Echoes from these patients were recorded on a portable Honeywell #1856 strip chart recorder. A simultaneous electrocardiographic monitoring lead was recorded on each tracing.

Echocardiographic Measurements

Left ventricular measurements were taken at or just below the posterior leaflet of the mitral valve as illustrated in figure 1. The amplitudes of echocardiographic motion from the left septum and left posterior endocardium (during ejection) were measured. In our laboratory the amplitude of left septal motion ranges from 3 to 9 mm (mean 6 mm) and the amplitude of posterior endocardial motion ranges from 9 to 16 mm (mean 13 mm) in normal subjects. Since previous publications the amplitudes of septal and posterior wall motion have been measured in 100 adult patients without angiographic evidence of coronary artery disease and values for the 95% confidence level are now used as normal ranges. Thicknesses of the interventricular septum and posterior left ventricular wall were measured during mid-diastole and at end-systole as shown in figure 1. Septal thickness was measured from the leading edge of the right septal echo to the leading edge of the left septal echo and posterior wall thickness was measured from the leading edge of the endocardial echo to the leading edge of the epicardial echo. Mid-diastole was defined as the interval between the completion of rapid ventricular filling and the onset of atrial filling. End-systole was defined as the point in time when the shortest distance between the left septal and posterior endocardial echoes was reached.

Statistical Analysis

The Student t-test was used to analyze the difference between group means, and in the patients with handgrip stress, to analyze the difference between the means of paired data. Chi square analysis was used to analyze the incidence of systolic wall thinning.

Results

Data from 146 patients in whom both thickness and motion were measurable were analyzed and included in this study. Twenty-nine patients were excluded due to inadequate echocardiograms. Thicknesses of the interventricular septum and left ventricular posterior wall could be measured in 83% of cases (146 of 175) and amplitudes of motion could be measured in 95% of cases (166 of 175).

Ranges and means of the measurements for each group and the statistical significance of the differences are presented in table 1. In addition to these group values, the

![Figure 1](image1.png)

**FIGURE 1.** Normal left ventricular echogram showing when in the cardiac cycle and where on the echogram diastolic and systolic wall thicknesses were measured.

![Figure 2](image2.png)

**FIGURE 2.** Individual values for percent change in septal thickness with systole are shown for patients with coronary artery disease, and group means are compared to normal.
<table>
<thead>
<tr>
<th>Group</th>
<th>No. Pts</th>
<th>IVSd (mm)</th>
<th>PWd (mm)</th>
<th>Systolic Septal Change</th>
<th>Systolic PW Change</th>
<th>No. pts thinning</th>
<th>No. pts thinning</th>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>mm</td>
<td>%</td>
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<td>Normals</td>
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<td>7-12</td>
<td>7-12</td>
<td>+1 to +5</td>
<td>+14 to +57</td>
<td>None</td>
<td>+3 to +11</td>
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<td></td>
<td></td>
<td>9</td>
<td>1.66</td>
<td>+3</td>
<td>+36%</td>
<td>+5</td>
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<td></td>
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<tr>
<td>Acute Infarction</td>
<td>20</td>
<td>5-12</td>
<td>8-14</td>
<td>-2 to +3</td>
<td>-29 to +25</td>
<td>11 (55%)</td>
<td>+1 to +11</td>
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<tr>
<td>Anterior</td>
<td></td>
<td>9</td>
<td>1.97</td>
<td>-0.4</td>
<td>-5%</td>
<td>+7</td>
<td>+10 to +125</td>
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<tr>
<td>Inferior</td>
<td>20</td>
<td>7-17</td>
<td>7-14</td>
<td>+1 to +9</td>
<td>+10 to +115</td>
<td>None</td>
<td>-1 to +9</td>
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<td></td>
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<td>10</td>
<td>2.32</td>
<td>+4</td>
<td>+46%</td>
<td>2.51</td>
<td>-11 to +64</td>
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<td>Coronary Artery Obstruction</td>
<td>12</td>
<td>5-13</td>
<td>8-15</td>
<td>-3 to +2</td>
<td>-29 to +22</td>
<td>1 (8%)</td>
<td>+4 to +9</td>
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<td>Infarction History</td>
<td></td>
<td>8</td>
<td>2.18</td>
<td>+0.4</td>
<td>+4%</td>
<td>+6</td>
<td>+27 to +100</td>
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<td>&lt;0.0001</td>
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<td>No Prior Infarction</td>
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<td>6-13</td>
<td>7-16</td>
<td>0 to +9</td>
<td>0 to +82</td>
<td>None</td>
<td>+2 to +9</td>
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<td>9</td>
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<td>+3</td>
<td>+34%</td>
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<td>+21 to +100</td>
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<td>Myopathy</td>
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<td>0 to +19</td>
<td>None</td>
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<td>8</td>
<td>3.06</td>
<td>+0.4</td>
<td>+5%</td>
<td>+2.3</td>
<td>0 to +63</td>
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<td>&lt;0.0001</td>
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<td>Atrial Septal Defect</td>
<td>20</td>
<td>7-18</td>
<td>8-16</td>
<td>+2 to +6</td>
<td>14 to 30</td>
<td>None</td>
<td>+4 to +8</td>
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<td></td>
<td>11</td>
<td>2.13</td>
<td>+3</td>
<td>+32%</td>
<td>+5</td>
<td>+27 to +88</td>
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<td>&lt;0.02</td>
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<td>NS</td>
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</table>

Abbreviations: IVSd = diastolic thickness of the interventricular septum; PWd = diastolic thickness of the left ventricular posterior wall; sd = standard deviation; NS = P > 0.05.
percent change in thickness between mid-diastole and end-systole for individual patients is shown graphically in figures 2 and 3 for normals and for patients with coronary artery disease, and in figure 4 for normals and for patients with atrial septal defect or cardiomyopathy.

**Acute Myocardial Infarction**

*Anterior*

Eighteen of 20 patients (90%) had abnormally decreased septal thickening; 19 (95%) had abnormally decreased septal motion; and 18 patients (90%) had both septal thickening and abnormal wall motion. Systolic septal thinning was recorded in 11 patients (55%) of whom six had paradoxical motion and five had complete absence of motion. An example of septal thinning is shown in figure 5. Although mean posterior wall thickening was significantly greater than normal in this group (63% versus 47%), there was considerable overlap and only two patients had exaggerated (above the normal range) posterior wall thickening.

*Inferior*

Thirteen of the 20 patients in this group (65%) had abnormally decreased posterior wall thickening; 18 (90%) had abnormally decreased posterior wall motion and 13 (65%) had both posterior wall thickening and abnormal motion. Systolic thinning of the posterior wall was recorded in one patient. Exaggerated septal thickening was recorded in four patients (20%) while 13 (65%) had exaggerated normal septal motion. Both exaggerated septal thickening and motion were present in three patients (15%).

**Chronic Coronary Artery Disease**

Thirty-seven of the 40 patients (93%) with angiographic evidence of significant coronary artery obstruction had significant coronary artery disease involving the left anterior descending coronary artery and 23 (58%) had associated significant disease in the right and/or left circumflex coronary artery. Twelve patients had a history of myocardial infarction, with abnormal Q waves in the anterior electrocardiographic leads in nine and in the inferior leads in three.

*With Previous Myocardial Infarction*

All 12 patients with previous infarction had abnormal septal motion with the motion paradoxical in five, absent in
three, and decreased in four. Abnormally decreased septal thickening with systole was observed in eight patients (67%) and one additional patient (8%) showed septal thinning with systole. Two patients (17%) had exaggerated posterior wall thickening and one patient (8%) had exaggerated motion.

**Without Previous Infarction**

Abnormal septal motion was observed in ten of 28 (36%) patients in this group with five (18%) having decreased septal thickening. Four patients (14%) showed abnormally decreased posterior wall thickening although no one had abnormally decreased posterior wall motion.

**Handgrip Stress**

At three minutes of sustained handgrip stress, mean septal thickening for each group (normal and coronary artery disease) was significantly less than during the control period, but there was no significant difference in response between the two groups. Only two patients developed abnormal systolic thickening with stress. One patient with coronary artery disease and normal septal thickening at rest developed septal thinning during stress when he developed chest pain, and another patient with coronary artery disease had a decrease in septal thickening to an abnormal value during stress.

**Cardiomyopathy**

Septal motion and thickening were both decreased in five patients. Posterior wall motion was decreased in six and posterior wall thickening was decreased in two patients.

**Discussion**

The most interesting finding in this study was that, with two exceptions, systolic thinning occurred only in patients with acute infarction. One exception was a patient with coronary artery disease who was having chest pain produced by handgrip stress. The other exception was a patient who had significant coronary artery disease at cardiac catheterization, which was performed because she was having frequent prolonged episodes of angina pectoris. Thickening which was decreased, or less than normal, was seen in patients with acute and chronic coronary artery disease and in patients with cardiomyopathy and is less specific. While the presence of systolic wall thinning was indicative of an acute ischemic event in this study, its absence did not rule out the presence of acute infarction as only 12 of the 40 acute infarction patients had this finding.

More direct ultrasonic techniques have been used to study changes in systolic wall thickening which occur during ischemia in experimental animals. It has been shown that decreases in systolic wall thickening which occur with ischemia are proportional to decreases in segment shortening. Systolic thinning occurred in the ischemic myocardial segments which bulged during systole and increased thickening was seen in control segments.

Among patients in our study abnormalities of motion occurred with greater frequency than abnormalities of thickening, although abnormally decreased thickening with normal motion was occasionally observed. We detected abnormal motion with normal thickening in six of the 40 patients with acute myocardial infarction and in nine of the 40 patients with chronic coronary artery disease. Perhaps in those patients the decreased motion was not directly due to decreased contractile function in that area.

All 20 patients with atrial septal defect had abnormal septal motion with normal systolic thickening of the septum as illustrated in figure 6. This was expected because the paradoxical septal motion in atrial septal defect is thought to be due to right ventricular volume overload and not due to abnormal contractility of the septum.

Exaggerated septal thickening was seen in only three of the 13 acute inferior myocardial infarction patients who had exaggerated septal motion. This suggests that the exaggerated septal motion in many of these cases may be due to a mechanism other than contractility such as an unloading effect during systole related to a dyskinetic segment in another area of the ventricle.

Because echocardiographic measurements of thickening

![Figure 6. Normal septal thickening with systole in a patient with atrial septal defect even though there is paradoxical septal motion and a dilated right ventricle.](image-url)
were frequently normal, even in the presence of severe coronary artery obstruction, we tried, unsuccessfully, to reveal abnormalities by handgrip stress. We chose this stress because it is noninvasive and does not interfere with the echocardiographic examination. Lack of success was not surprising since it has been shown that handgrip stress is not a sensitive means of producing angina and that myocardial blood flow during handgrip stress is not significantly different from normal in patients with coronary artery disease who do not develop angina during the stress.  

Although group mean diastolic thicknesses for the groups of patients with coronary heart disease were not significantly different from normal, there were values outside the normal range for individual patients. Previous reports suggest possible reasons for these abnormalities. A decrease in thickness occurs within seconds after ischemia is produced in open-chest pigs; progressive thinning of ischemic segments over a period of weeks has been shown to occur in chronic dogs, suggesting tissue loss; and thin dense septal echoes have been found to be associated with the presence of septal scarring at surgery or autopsy in patients with coronary artery disease.

Increased thickness could be due to compensatory hypertrophy of normal myocardium in patients with chronic coronary artery disease. Three of the four patients with atrial septal defect and increased septal thickness had pulmonary hypertension at cardiac catheterization.

In summary, this study, in addition to experimental studies performed by Ross and Franklin and by Theroux et al., indicates that echocardiographic measurements of left ventricular wall thicknesses and the changes which occur during systole will be useful in evaluating the performance of myocardial segments in human subjects.

References

Systolic thickening and thinning of the septum and posterior wall in patients with coronary artery disease, congestive cardiomyopathy, and atrial septal defect.
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