Echocardiographic Study of the Abnormal Motion of the Posterior Left Ventricular Wall during Angina Pectoris

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SUMMARY
Echocardiographic waves from the posterior left ventricular endocardium were recorded in 30 normal subjects and in nine patients during 13 anginal episodes. At rest the normal maximal systolic endocardial velocity (SEVM) was $6.2 \pm 1.4$ cm/sec, the mean systolic endocardial velocity (SEV) was $4.1 \pm 0.7$ cm/sec, and the systolic endocardial excursion (SEE) was $1.4 \pm 0.3$ cm. The maximal diastolic endocardial velocity (DEV) was $18 \pm 3$ cm/sec, and the mean early diastolic endocardial velocity (DEVM) was $9.4 \pm 1.7$ cm/sec. Exercise in 20 normals caused a significant increase in SEVM, SEV, DEV, and DEVM, but not SEE. In no instance did any of these values fall below the resting levels. The angina patients differed significantly from the normals having at rest a slower DEV (15 \pm 4 cm/sec) ($P < 0.025$) and DEVM (8.4 \pm 0.8 cm/sec) ($P < 0.025$). During exercise, but before angina, there was a significant increase in SEVM and SEV but not SEE, DEV, or DEVM. In no instance did any of these values fall below the resting levels. During angina SEVM and SEV reacted variably and together with SEE were not significantly different from the resting values. In contrast, there was a remarkable slowing of DEV (8.2 \pm 3.2 cm/sec) ($P < 0.001$) and DEVM (5.7 \pm 2.2 cm/sec) ($P < 0.001$). Five minutes after the pain and S-T-segment depression disappeared, the endocardium moved as it did before exercise.

Additional Indexing Words:
Reflected ultrasound  Stress test  Left ventricular posterior wall systolic velocity
Endocardium  Left ventricular posterior wall diastolic velocity

The systolic events in acute myocardial infarction have been studied with reflected ultrasound\textsuperscript{1-3} and found to be clinically useful. One group noted a decrease in maximal posterior wall systolic velocity and posterior wall systolic excursion during the first 36 hours after acute anterior myocardial infarction in eight patients.\textsuperscript{3} Another group found that left ventricular (LV) posterior wall systolic velocity and excursion did decrease before death in one patient, but in nine surviving patients serial measurements were variable during the acute phase of myocardial infarction and demonstrated no trends.\textsuperscript{2}

There have been recent reports of abnormalities in LV compliance in patients with coronary artery disease\textsuperscript{4,5} suggesting that diastolic changes can also occur with ischemia. Since reflected ultrasound is suited to studying both systolic and diastolic events, we applied these technics to study the motion of the posterior LV wall during induced angina pectoris.
Materials and Methods

Normal Subjects

Subjects without clinical evidence of heart disease were examined at rest, during and after exercise in the supine position. Thirty subjects (29 males, one female) were studied at rest. Ten of them held an isometric grip at one-third of their maximum effort for 3 min. Another 10 pedaled an ergometer for 3–5 min at work loads designed to increase the heart rate (HR) without producing respirations so labored that recording of echoes during exercise would be impossible. These two forms of exercise differ in that the isometric handgrip exercise produces a more moderate increase in heart rate and cardiac output but vigorous increases in both systolic and diastolic arterial pressures with little or no change in systemic vascular resistance except in some older subjects in whom it may increase; total systemic vascular resistance decreases markedly with bicycle exercise due to the vasodilatation of the active muscles.

Patients

Nine patients (eight males, one female) with classic angina pectoris were studied. Diagnosis was established by multiple treadmill exercise tests, during which ischemic S-T-segment depression (at least 1 mm of horizontal or downsloping depression of the S-T segment) at low levels of exercise was associated with fully developed anginal distress. Five patients had coronary angiograms prior to this study which confirmed the diagnosis of severe coronary artery disease. They were receiving no cardiac medications other than nitrates which were stopped the day prior to testing. One patient developed angina spontaneously before exercise and was not stressed further. The other eight patients performed isometric handgrip exercise and/or pedaled a bicycle ergometer at increasing work loads until angina of moderately severe intensity was achieved and diagnostic S-T-segment depression was observed. The isometric handgrip exercise was used as an initial form of stress which would not require the patient to move. It produced ischemia in only one person who did not develop pain, but who showed diagnostic S-T-segment depression. After heart rate and ECG had returned to normal, four patients were given a second effort.

Echocardiographic Techniques

The echocardiograms were recorded using an Ekoline 20 echograph* and a 2.25 MHz transducer with 0.5 in. diameter and 10 cm focus.† The ultrasound transducer was placed in the fourth or fifth left intercostal space close to the sternum and first the mitral valve was identified. The beam was then directed laterally and slightly inferiorly to the left, to obtain the echoes simultaneously from the intraventricular septum and posterior left ventricular wall, just below the posterior leaflet of the mitral valve. The transducer was slightly adjusted in this position to obtain the maximum motion of the posterior left ventricular wall. This standardized technic was used on each subject in this study. Identification of the endocardial echo was accomplished first by placing the "gain control" at the minimal level where strong pericardial-epicardial echoes were identified. The gain was then increased and the echoes anterior to the pericardial-epicardial echoes which move as a single unit were identified. The anteriorsmost echo of these myocardial echoes was taken to be the endocardium. It can be clearly distinguished from other more anterior echoes which exhibit no consistent pattern and do not seem to be an integral part of the myocardial echoes. The endocardial echo was also differentiated from the discrete echoes from the posterior chordae tendineae which are frequently recorded just anterior to the endocardial echo. The chordal echoes were identified by their characteristic high intensity, sharpness, and lack of systolic increase in the distance to the myocardial echoes, which show an apparent increase in thickness from end-diastole to end-systole. The echoes were displayed on an oscilloscope in the time-motion mode, and were recorded on polaroid film along with a superimposed electrocardiogram (ECG).

A second ECG was recorded on a standard electrocardiograph machine monitoring the thoracic lead that had been routinely used for that individual during the treadmill tests. Multiple posterior wall echograms of the normal subjects were taken before, during, immediately after, and 5 min after exercise. Similarly, multiple recordings were taken of the angina patients before exercise, during exercise, immediately after exercise, and then 5 min after pain and S-T depression had disappeared.

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*Smith Kline Instruments.
†Aerotech Laboratories.
The recordings of the posterior wall endocardium were analyzed in a similar manner for all groups. A recording was selected from each period on the basis of clarity. Only high-quality photographs were used.

Each complex on the selected films was examined and multiple measurements were made for each phase of endocardial motion and in each case the highest value consistently found was used. Figure 1 shows a diagram of the echocardiographic events of the posterior left ventricular wall during the cardiac cycle. The letters A–F correspond to the periods described by Kraunz and Kennedy and Inoue et al., though they only studied the epicardium. Point A corresponds to the onset of atrial contraction. The line from B to C describes wall motion during isovolumetric contraction. The line from C to D describes the motion during systolic ejection. The period from point D to E represents isovolumetric relaxation. The line from E to F describes the wall motion in early diastole. The total systolic amplitude of the curve was measured to obtain systolic endocardial excursion (SEE) (C–D). Maximal systolic endocardial velocity (SEVM) was determined by drawing a tangent to the steepest point of the systolic limb of the curve and measuring the slope in cm/sec. Maximal diastolic endocardial velocity (DEVM) was determined by drawing a tangent to the steepest point of the diastolic limb of the curve and measuring the slope in cm/sec. The mean systolic endocardial velocity (SEV) was measured as the slope of the line extending from the onset of anterior endocardial displacement to the peak of the echogram (C–D). The mean early diastolic endocardial velocity (DEV) was measured as the slope of the line extending from the peak of the echogram to the end of the rapid posterior motion of the endocardium in early diastole (D–F).

Twelve photographs were randomly selected for analysis of beat-to-beat variation and variation between two observers.

Reproducibility was further checked by comparing the results at two points in time under similar conditions: i.e., during recumbent rest before exercise and after exercise when the heart rate and ECG had returned to the baseline state.

The tests of observer variation and reproducibility were made using Student’s t test for paired variants; all other comparisons were made using Student’s t test for unpaired variants.

**Results**

**Reproducibility of Results**

**Beat-to-beat variation.** The values obtained from two beats on the same photograph for SEVM, SEV, SEE, DEVM, and DEV were not significantly different.

**Observer variation.** The results obtained from the 12 photographs analyzed by the two observers for SEVM, SEV, SEE, DEVM, and DEV were not significantly different.

**Variation with time.** The results obtained before exercise for SEVM, SEV, SEE, DEVM, and DEV were not significantly different from those obtained after exercise at a time when the HR and ECG had returned to the baseline state.

**Normal Subjects.** The normal response of the posterior wall to exercise is demonstrated in figure 2 and the values are summarized in table 1. The range for SEVM, SEV, SEE, DEVM, and DEV for 30 normal subjects at rest was 4.3–10 cm/sec, 3.0–5.8 cm/sec, 1.0–2.0 cm, 13–24 cm/sec, and 7.0–13 cm/sec, respectively.

While handgrip exercise did not change HR or SEE significantly, it did cause a significant rise in SEVM (P<0.005), SEV (P<0.05), DEVM (P<0.005) and DEV (P<0.05). Leg exercise did not change SEE significantly but did cause a significant rise in HR (P<0.01), SEVM (P<0.005), SEV (P<0.005), DEVM (P<0.001), and DEV (P<0.02).

In no instance of either handgrip or leg exercise in normals did SEVM, SEV, SEE, DEVM, or DEV fall below resting values during or immediately after exercise.

**Patients with angina.** A typical response of an angina patient is shown in figure 2 and the values for the group are summarized in table 1. The range for SEVM, SEV, SEE, DEVM, and DEV for nine angina patients before exercise was 5.0–8.5 cm/sec, 3.8–6.2 cm/sec, 1.1–1.9 cm, 11–20 cm/sec, and 7.3–10 cm/sec, respectively. In all instances the induced angina was accompanied by ischemic S-T-segment changes in the ECG.

The angina patients were older than the normals, 61 ± 13 years vs 28 ± 4 years (P<0.001). At rest DEVM and DEV were slightly but significantly slower in the angina patients.
NORMAL BEFORE EXERCISE

NORMAL DURING EXERCISE
than in the resting normals \( (P < 0.025 \text{ for both}) \) while HR, SEVM, SEV, and SEE were not significantly different. One patient had a history of an old diaphragmatic myocardial
infarction; there was no echocardiographic evidence of a posterior wall aneurysm in any of these patients.

We were able to obtain high-quality recordings during exercise but before angina (before pain or S-T-segment depression) in six instances. The HR, SEVM, and SEV were significantly faster than at rest (P < 0.005, P < 0.05, and P < 0.005, respectively) while SEE, DEVVM, and DEV were not significantly different from the resting values. DEVVM increased in five episodes and remained unchanged in one. DEV increased in three episodes and remained unchanged in three. In no instance did SEE, DEVVM, or DEV fall below the resting values.

We were also able to obtain high-quality recordings during seven anginal episodes while the patients were still exercising, during five anginal episodes immediately after exercise was stopped, and during one episode of spontaneous angina.

Since none of the normal values during or immediately after exercise fell below the resting values, we have combined these 13 episodes into one period designated “during angina,” and we have compared these values to the anginal patient’s resting values.

HR was significantly faster during angina (P < 0.005), while SEVM, SEV, and SEE were not significantly different from the resting values. SEVM increased in six episodes (an example is shown in fig. 2), decreased in five, and remained unchanged in two. SEV increased in six episodes, decreased in four, and remained unchanged in three. SEE increased in three instances and remained unchanged in 10.

Of the six episodes studied during exercise but before angina, SEVM increased further with the onset of angina in two instances, did not change in two, decreased to a point still above the resting level in one, and fell below the resting level in one.

A striking difference in early diastolic wall motion was found during angina; there was a marked slowing of DEVVM and DEV (P < 0.01 for both). This slowing occurred in 11 of 13 anginal episodes and is distinctly different from the normal response during or

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### Table 1

**Echocardiographic Measurements**

<table>
<thead>
<tr>
<th>Time of measurement</th>
<th>Heart rate (beats/min)</th>
<th>SEVM (cm/sec)</th>
<th>SEV (cm/sec)</th>
<th>SEE (cm)</th>
<th>DEVVM (cm/sec)</th>
<th>DEV (cm/sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal subjects at rest (N = 30)</td>
<td>64 ± 11</td>
<td>6.2 ± 1.4</td>
<td>4.1 ± 0.7</td>
<td>1.4 ± 0.3</td>
<td>18 ± 3</td>
<td>9.4 ± 1.7</td>
</tr>
<tr>
<td>Normal subjects - handgrip (N = 10)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Before exercise</td>
<td>64 ± 12</td>
<td>5.2 ± 1.0</td>
<td>4.2 ± 0.5</td>
<td>1.2 ± 0.2</td>
<td>19 ± 3</td>
<td>9.9 ± 1.4</td>
</tr>
<tr>
<td>During exercise</td>
<td>71 ± 12</td>
<td>6.9 ± 1.1</td>
<td>4.9 ± 0.8</td>
<td>1.3 ± 0.3</td>
<td>25 ± 5</td>
<td>12 ± 3</td>
</tr>
<tr>
<td>Immediately after exercise</td>
<td>68 ± 13</td>
<td>7.0 ± 1.5</td>
<td>4.8 ± 0.9</td>
<td>1.4 ± 0.3</td>
<td>21 ± 4</td>
<td>11 ± 3</td>
</tr>
<tr>
<td>5 min after exercise</td>
<td>64 ± 9</td>
<td>5.7 ± 0.7</td>
<td>4.3 ± 0.6</td>
<td>1.3 ± 0.1</td>
<td>19 ± 3</td>
<td>9.7 ± 1.2</td>
</tr>
<tr>
<td>Normal subjects - bicycle ergometer (N = 10)</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Before exercise</td>
<td>69 ± 12</td>
<td>6.6 ± 1.0</td>
<td>4.7 ± 0.6</td>
<td>1.4 ± 0.3</td>
<td>17 ± 3</td>
<td>9.3 ± 1.6</td>
</tr>
<tr>
<td>During exercise</td>
<td>87 ± 15</td>
<td>8.7 ± 1.6</td>
<td>5.8 ± 0.9</td>
<td>1.6 ± 0.3</td>
<td>23 ± 3</td>
<td>12 ± 2</td>
</tr>
<tr>
<td>Immediately after exercise</td>
<td>74 ± 14</td>
<td>7.2 ± 1.4</td>
<td>5.0 ± 0.6</td>
<td>1.5 ± 0.3</td>
<td>21 ± 3</td>
<td>10 ± 2</td>
</tr>
<tr>
<td>5 min after exercise</td>
<td>65 ± 12</td>
<td>6.3 ± 0.7</td>
<td>4.5 ± 0.5</td>
<td>1.5 ± 0.3</td>
<td>17 ± 3</td>
<td>9.1 ± 1.4</td>
</tr>
<tr>
<td>Angina patients (N = 9 : 13 anginal episodes)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Before exercise</td>
<td>63 ± 13</td>
<td>6.5 ± 1.3</td>
<td>4.4 ± 0.8</td>
<td>1.4 ± 0.3</td>
<td>15 ± 4</td>
<td>8.4 ± 0.8</td>
</tr>
<tr>
<td>During exercise but before angina</td>
<td>88 ± 15</td>
<td>8.2 ± 1.5</td>
<td>6.3 ± 1.2</td>
<td>1.4 ± 0.2</td>
<td>18 ± 6</td>
<td>10 ± 3</td>
</tr>
<tr>
<td>During angina</td>
<td>90 ± 24</td>
<td>7.2 ± 2.2</td>
<td>5.3 ± 1.6</td>
<td>1.5 ± 0.3</td>
<td>8.2 ± 3.2</td>
<td>5.7 ± 2.2</td>
</tr>
<tr>
<td>5 min after angina</td>
<td>64 ± 11</td>
<td>6.7 ± 1.4</td>
<td>4.7 ± 1.0</td>
<td>1.4 ± 0.3</td>
<td>15 ± 4</td>
<td>8.4 ± 0.9</td>
</tr>
</tbody>
</table>

*All values are mean ± sd.
Abbreviations: See text.
immediately after exercise as is shown graphically for DEVM in figure 3. The two anginal episodes in which DEVM and DEV did not fall below resting values occurred in the same patient. He was not different from the others in age, sex, history, physical signs, or laboratory findings. A coronary arteriogram performed 6 months prior to this study showed a totally occluded right coronary artery, two tight stenoses in the left anterior descending coronary artery, which was diffusely diseased, and a large circumflex system that was virtually free of disease. In all but one instance the values during angina returned to the resting levels with rest alone; one patient required sublingual nitroglycerin.

**Discussion**

This study disclosed that the posterior wall of the heart moved more rapidly during early diastolic relaxation than it did during systolic contraction. This was so at rest in normal subjects as well as in patients with angina pectoris. Exercise significantly increased the rate of systolic motion in both groups. Early diastolic motion became significantly faster

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| ECHOCARDIOGRAPHIC STUDY DURING AP | 911 |

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**Figure 3**

*Open circles and dashed lines = handgrip; closed circles and solid line = bicycle ergometer. Values shown are mean ± sd. Abbreviations: see text.*
with exercise in normals and became faster in the anginal patients although the increase in the anginal patients did not reach statistical significance. In no instance, during or immediately after exercise in the normals or during exercise but before angina in the patients, did SEVM, SEV, SEE, DEV, or DEV fall below the resting values. When the ischemic state manifested by anginal pain and/or S-T-segment depression appeared, there was a marked and highly significant slowing in early diastole in all but one patient, while the rate of systolic movement was variable. This remarkable fall of DEV and DEV below the resting values during angina stands in sharp contrast to the normal response to exercise since the records obtained during angina were taken either during exercise or immediately afterward.

Diamond and Forrester found that patients with coronary artery disease had increased diastolic LV wall stiffness which worsened with the development of acute infarction. These increases in LV wall stiffness were felt unrelated to the level of end-diastolic pressure. Moreover, the depression of contractility secondary to myocardial disease was thought not to account for the increased wall stiffness. It may be that this increased wall stiffness is due at least in part to a failure of normal relaxation during the ischemic state such as we observed in this study.

Relaxation is not a passive process. Langer has estimated that 15% of the total myocardial energy requirements may be needed for pumping Ca⁺⁺ back into the sarcoplasmic reticulum, which is necessary to achieve relaxation during each cardiac cycle. Moreover, he estimates that Na⁺ pumping may require another 2.5%. It is not surprising that ischemia may have profound effects on this period of the cardiac cycle. Evidence has been presented to indicate that contraction and relaxation may be altered in different ways by both mechanical and chemical intervention. The varying systolic responses during the ischemic state seen in these patients could be due to variation in catecholamine responses and/or changes in preload (i.e. venous return) and afterload (i.e. aortic diastolic blood pressure).

At rest the angina patients had a slower DEV and DEV than did the resting normals. This might be an age-related phenomenon or it might be a reflection of a chronic ischemic process. Harrison et al. studied resting individuals (ages 9-97 years who had normal hearts as judged by clinical and electrocardiographic criteria) with simultaneous ECG’s, carotid pulse tracings, and traces of absolute precordial movement (kinetocardiograms). They found that there was a definite slowing of isovolumic relaxation with advancing age. These subjects were not studied during exercise. Bristow, Van Zee, and Judkins studied patients and normals at rest with angiography and measured left ventricular volumes. If we look at their figure 1, it appears that the rate at which the ventricle enlarged in early diastole was slower in two of the three patients with coronary artery disease than it was in the normal subject. We found that while our patients were significantly older than our normal subjects and had a slight but significantly slower DEV and DEV at rest than the normals, the angina patients responded to exercise qualitatively the same as did the normals: i.e. in no instance did SEVM, SEV, SEE, DEV, or DEV fall below the resting values during exercise but before the onset of angina. In contrast, the marked fall in DEV and DEV during angina suggests that it is the ischemic state that is responsible for these changes and not simply an altered response to exercise with aging. Carson and Kanter found that their patients with heart disease of diverse etiology and various ages had a slower rate of posterior wall motion backward at rest than did their normal subjects even after heart failure had been treated. However, it was not possible to distinguish the endocardium from the epicardium in their published echocardiograms.

With the development of a reliable continuous recording system and further refinements in ultrasound equipment, the echocardiographic stress test may find general applica-
tion in the detection and study of ischemic heart disease.

Acknowledgment
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