Correlation Between the Exercise-Induced Increase in Left Ventricular Filling Pressure and the Extent of Ischemic or Infarcted Myocardium

Akitada Ando, M.D., Mitsuhiro Yokota, M.D., Toshikazu Sobue, M.D., Yasuto Nishinaka, M.D., Mitsunori Iwase, M.D., Hiroshi Hayashi, M.D. and Hidehiko Saito, M.D.

We investigated the correlation between left ventricular filling pressure and the extent of ischemic or infarcted myocardium in 39 patients with coronary artery disease; 25 with angina pectoris (group A) and 14 with old myocardial infarction but without overt transient myocardial ischemia (group B). Hemodynamic parameters were measured at rest and during exercise. The extent and severity scores of ischemia or infarct were calculated using thallium-201 ($^{201}$Tl) myocardial single-photon emission computed tomography. In group A, the extent and severity score of ischemia were strongly correlated with pulmonary artery wedge pressure at peak exercise ($r = 0.71, p < 0.001, r = 0.62, p < 0.01$, respectively). In group B, the extent and severity scores of the infarct were significantly correlated with left ventricular ejection fraction ($r = -0.81, p < 0.001, r = -0.77, p < 0.01$, respectively), but were not correlated with pulmonary artery wedge pressure. Since no relationship was found between the extent of infarct and left ventricular filling pressure, dynamic exercise appears to elicit a different compensatory mechanisms in nonischemic myocardium for exercise-induced transient ischemia and in noninfarcted myocardium for old infarction. The compensatory mechanism in patients with old myocardial infarction may be affected by ventricular remodeling. (Jpn Circ J 1995; 59: 705–714)

When a severe coronary stenosis reduces myocardial flow, contractility is reduced in the jeopardized myocardium even if there is no infarction. The resulting regional decrease in contractility influences global cardiac function. Left ventricular filling pressures have been found to increase markedly in patients with angina pectoris during exercise$^1–^5$ and pacing$^6–^10$ in acute ischemia, this increase may result from a compensatory increase in diastolic stretch in the nonischemic myocardium in response to the decreased systolic shortening of the ischemic segment. Furthermore, the increase in left ventricular filling pressure during exercise has been found to be related to the size of the acute ischemic area$^1$. On the other hand, in a report on the mechanism of the increase in left ventricular filling pressure during exercise in old myocardial infarction, pulmonary artery wedge pressure was higher in large infarction than in small infarction. Furthermore, the stroke volume index increased in acute ischemia and in small infarction, but not in large infarction. Since this previous study used an inadequate exercise

Key words:
Left ventricular filling pressure
Extent of ischemia
Extent of infarct
Ventricular remodeling

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First Department of Internal Medicine, Nagoya University School of Medicine and “Cardiovascular Section, Department of Clinical Laboratory Medicine, Nagoya University Hospital, Tsurumai, Showa-ku, Nagoya 466, Japan
Mailing address; Mitsuhiro Yokota, M.D., Cardiovascular Section, Department of Clinical Laboratory Medicine, Nagoya University Hospital, 65 Tsurumai, Showa-ku, Nagoya 466, Japan

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TABLE I ANGIOGRAPHIC FINDINGS

<table>
<thead>
<tr>
<th></th>
<th>Group A (n=25)</th>
<th>Group B (n=14)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. of lesions</td>
<td>No. of patients</td>
</tr>
<tr>
<td>Right coronary artery</td>
<td>10</td>
<td>19</td>
</tr>
<tr>
<td>Left anterior descending artery</td>
<td>12</td>
<td>6</td>
</tr>
<tr>
<td>Left circumflex artery</td>
<td>6</td>
<td>3</td>
</tr>
<tr>
<td>Left main trunk</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Collateral circulation (+)</td>
<td>15</td>
<td>7</td>
</tr>
<tr>
<td>(0)</td>
<td>10</td>
<td>7</td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>76 ± 9</td>
<td>63 ± 13</td>
</tr>
<tr>
<td></td>
<td>(52–87)</td>
<td>(47–76)*</td>
</tr>
<tr>
<td>LVEDVI (mL/m²)</td>
<td>65 ± 13</td>
<td>84 ± 27</td>
</tr>
<tr>
<td></td>
<td>(41–92)</td>
<td>(44–132)*</td>
</tr>
<tr>
<td>LVESVI (mL/m²)</td>
<td>16 ± 10</td>
<td>34 ± 20</td>
</tr>
<tr>
<td></td>
<td>(7–34)</td>
<td>(12–75)*</td>
</tr>
</tbody>
</table>

*p < 0.001; † < 0.02; ‡ < 0.01 vs group A
LVEDVI = left ventricular end-diastolic volume index; LVEF = left ventricular ejection fraction; LVESVI = left ventricular end-systolic volume index.

level of 50 watts, the relation between left ventricular filling pressure and infarct size has not been assessed.

The purpose of the present study was to clarify the relationship between left ventricular filling pressure during exercise and the extent of infarct in patients with old myocardial infarction, and the difference in hemodynamics during exercise between acute transient ischemia and old myocardial infarction.

METHODS

Patients

We studied 39 consecutive patients who underwent exercise 201Tl myocardial single-photon emission computed tomography (SPECT) and cardiac catheterization with hemodynamics measured during supine bicycle ergometer exercise tests. Patients with heart diseases other than coronary artery disease were excluded from the study. Group A consisted of 25 patients (23 men and 2 women; aged 47 to 71 years; mean: 58 years) who had exertional angina pectoris. They had no myocardial infarction by 201Tl SPECT. Group B consisted of 14 men (aged 24 to 68 years; mean: 57 years) with previous myocardial infarctions. Patients in group B had no history of symptomatic anginal attack, exercise-induced ischemic ST depressions on electrocardiograms, or fixed defect in 201Tl SPECT. The average duration from the onset of infarction to the hemodynamic study was 12 months (range 3 months—60 months). The infarcted locations are shown in Table I, and there were no non-Q wave infarctions. None of the patients had unstable angina pectoris or variant angina pectoris, or showed evidence of heart failure before or during the study. Patients with triple-vessel disease were excluded from the study because 201Tl myocardial scintigraphy is less sensitive for detection of true perfusion defects in these patients due to the global reduction in thallium uptake. All patients were in sinus rhythm, and none had echocardiographic evidence of left ventricular hypertrophy. The study was approved by the appropriate institutional review committee. Written informed consent was obtained from all patients prior to their participation in the study.

Cardiac Catheterization

All medications other than sublingual nitroglycerin were withdrawn 3 days before testing. Sublingual nitroglycerin was not used within 12 h before the study. Patients performed tests in the fasting state. A 7-Fr balloon-tipped thermodilution Swan-Ganz catheter (Baxter Healthcare Corp., Edwards Division, Santa Ana, CA, USA) was introduced into a left basilic venous cutdown and advanced to the right pulmonary artery. The position of the catheter was considered satisfactory only if a pulmonary artery wedge pressure of good quality was obtained after balloon inflation. A cannula was percutaneously inserted into the left brachial artery and the systemic arterial blood pressure was recorded simultaneously. A multistage exercise protocol was performed on a supine bicycle ergometer at an initial workload of 25 watts. The workload was increased by 25 watts every 3 min according to a previously described method. The exercise test was halted when symptoms developed, and the patient was unable to continue. A 12-lead electrocardiogram was continuously monitored before, during, and after exercise.
Heart rate, systolic blood pressure, pulmonary artery wedge pressure and cardiac output were recorded at rest and during the last min of each stage of the exercise protocol. Pressure was recorded at a paper speed of 100 mm/sec and was determined by taking the mean value of 2 or more respiratory cycles. Patients who exhibited a large v wave in pulmonary artery wedge pressure, which suggests moderate to severe mitral regurgitation during exercise! were excluded from the analysis. Cardiac output was measured by the thermodilution method. The curve recorded with the thermodilution cardiac output computer (model RI-5DCP, General Scanning, Watertown, Los Angeles, CA, USA) was checked for consistency before the results were considered to reflect cardiac output. The rate-pressure product was calculated as the product of systolic blood pressure and heart rate. Cardiac index (L/min per m²) and stroke volume index (ml/beat per m²) were also determined as follows: cardiac index = cardiac output/body surface area; stroke volume index = cardiac index/heart rate. Selective coronary angiography and left ventriculography were performed by the transfemoral approach (Judkins technique) at least 30 min after the completion of the exercise test. The degree of coronary stenosis and collateral filling of the coronary artery branch were determined by 2 separate observers. A reduction in lumen diameter of 75% or greater in any 1 projection was considered to represent significant stenosis. Left ventricular end-diastolic and end-systolic volumes, and left ventricular ejection fraction were calculated by the biplane area-length method!

201 I Myocardial SPECT

Radionuclide studies were performed within 1 week before or after cardiac catheterization with a gamma camera (GCA-70TOKU, Toshiba, Japan) and a computer (GMS-55A, Toshiba, Tokyo, Japan). One minute before termination of a symptom-limited multistage sitting bicycle ergometer exercise test at an initial workload of 25 watts increased by 25 watts every 3 min, 74 MBq of 201 I was injected from a cannula in the forearm. Initial images were obtained about 5 min after intravenous injection of 201 I and delayed images were obtained about 3 to 4 h after the initial images. The large-field-of-view scintillation camera consisted of dual-headed detectors equipped with low-energy, all purpose, parallel-hole collimators. Sixty projections were obtained at 6-deg sampling steps for 20 sec of imaging time in a 360-deg arc. All projections were stored on a 64 × 64/16-bit matrix. Transaxial tomographs were reconstructed on a 5.3 mm-wide slice by a filtered back projection algorithm using a Butterworth filter, photon absorption correction, and 3 × 3 matrix smoothing. The vertical long- and short-axis view of the left ventricle were extracted from the filtered transaxial tomographs of coordinate transformation using appropriate interpolation. After all of the maximal-count circumferential profiles for each short-axis cut were extracted and normalized to the maximal pixel value for that profile, they were mapped into a 2-dimensional polar representation. To estimate the extent of the ischemic or infarcted area, a comprehensive method was used, as previously described! The method has high reproducibility because the calculations are performed automatically by computer. Normal limits were determined from the distribution profiles established from pooled data obtained in 14 age-matched normal subjects without heart disease and were defined as the curves that represented 2 standard deviations below the mean. These limits were used as the threshold for detection of ischemic or infarcted areas. The extent and severity scores were then calculated for the perfusion defect in the initial image as reported previously! The extent score was determined by calculating the number of points which fell below the corresponding normal lower limits and by expressing this number as a percentage of the total left ventricular points on the corresponding 2-dimensional polar map. The severity score was determined by calculating the difference between the normalized maximal counts and the corresponding normal lower limits, and dividing this difference by the total number of left ventricular points.

Statistical Methods

Data are presented as the mean ± SD. Hemodynamic parameters were compared using the paired or unpaired t-test to evaluate differences between the supine and
TABLE II  HEMODYNAMICS AT REST AND DURING EXERCISE IN GROUPS A AND B

<table>
<thead>
<tr>
<th></th>
<th>Group A</th>
<th>Group B</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>rest</td>
<td>exercise</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>66±13</td>
<td>114±18</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>151±26</td>
<td>189±35</td>
</tr>
<tr>
<td>RPP (beats • mmHg/min)</td>
<td>9958±2747</td>
<td>21336±6387</td>
</tr>
<tr>
<td>PAWP (mmHg)</td>
<td>7±5</td>
<td>23±10</td>
</tr>
<tr>
<td>CI (l/min • m²)</td>
<td>2.8±0.5</td>
<td>5.8±1.5</td>
</tr>
<tr>
<td>SVI (ml/m²)</td>
<td>43±6</td>
<td>50±11</td>
</tr>
</tbody>
</table>

*p<0.02 vs group A  
CI = cardiac index; HR = heart rate; PAWP = pulmonary artery wedge pressure; RPP = rate-pressure product; SBP = systolic blood pressure; SVI = stroke volume index;

Fig.1. Extent or severity score in group A and group B. The extent and severity score scores were significantly higher in group A than in group B.

sitting positions or among groups, respectively. Linear regression analysis was performed to determine correlations. A value of p<0.05 was considered statistically significant.

RESULTS

Angiographic Findings and Hemodynamics During Supine Bicycle Exercise

There were no significant differences between groups in the number of vessels involved and the status of collateral flows (Table I). In group A, left ventricular ejection fraction was normal and there were no localized wall motion abnormalities at rest. The left ventricular ejection fraction was significantly smaller in group B than in group A (p<0.001), and akinesis was detected in the infarcted area. Left ventricular end-diastolic and end-systolic volume indexes were significantly larger in group B than in group A. Exercise durations tended to be shorter in group A (451±141 sec) than in group B (544±125 sec). Exercise duration in group B was not related to the location or extent of myocardial infarction.

Heart rate, systolic blood pressure, and rate-pressure product was were significantly increased at peak supine exercise in both groups (Table II). Heart rate at peak exercise was significantly higher and systolic blood pressure at rest was significantly lower in group B than in group A. There was no difference in the baseline and peak pulmonary artery wedge pressures between groups, although pulmonary artery wedge pressure tended to be higher at peak exercise in group A. Cardiac index increased markedly at peak exercise and stroke volume index increased slightly. The extent score and severity score were significantly higher in group B than in group A (Fig. 1).

In group A, 13 patients were in New York
Heart Association (NYHA) functional class I and 12 were in NYHA II, while 9 were in NYHA I and 5 were in NYHA II in group B. There were no patients in NYHA III–IV. There were no significant differences between the 2 groups.

**Relationship Between Hemodynamics and the Extent of Myocardial Ischemic or Infarct Area**

In group A, the extent score of ischemia was weakly correlated with pulmonary artery wedge pressure at rest ($r = 0.42, p < 0.05$) and the extent and severity scores were strongly correlated with pulmonary artery wedge pressure at peak exercise ($r = 0.71, p < 0.001$, $r = 0.62, p < 0.01$, respectively) (Fig. 2). There was no correlation between other hemodynamic parameters and the extent of the ischemic area. In group B, the extent of infarct was not related to the pulmonary artery wedge pressure (Fig. 3). The extent and severity scores of the infarct were significantly correlated with left ventricular ejection fraction ($r = -0.81, p < 0.001$, $r = -0.77, p < 0.01$, respectively) (Fig. 4) and the extent score was correlated with the left ventricular end-systolic volume index ($r = 0.61, p < 0.05$) at rest. There was no relationship between other hemodynamic...
Fig. 4. Relationship between left ventricular ejection fraction and the extent or severity score (group B). A negative correlation was observed. Extent score: left panel, severity score: right panel.

**TABLE III COMPARISON OF PULMONARY ARTERY WEDGE PRESSURE AND EXTENT OF ISCHEMIA OR INFARCT AT PEAK EXERCISE ACCORDING TO DISEASE CHARACTERISTICS**

<table>
<thead>
<tr>
<th></th>
<th>Group A</th>
<th></th>
<th>Group B</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>PAWP (mmHg)</td>
<td>Ischemic Extent (%)</td>
<td>PAWP (mmHg)</td>
<td>Infarct Extent (%)</td>
</tr>
<tr>
<td>single-vessel disease</td>
<td>22 ± 11</td>
<td>31 ± 17</td>
<td>17 ± 5</td>
<td>48 ± 17</td>
</tr>
<tr>
<td>double-vessel disease</td>
<td>25 ± 5</td>
<td>28 ± 16</td>
<td>20 ± 7</td>
<td>49 ± 26</td>
</tr>
<tr>
<td>RCA lesion</td>
<td>18 ± 13</td>
<td>24 ± 16</td>
<td>18 ± 6</td>
<td>32 ± 11</td>
</tr>
<tr>
<td>LAD lesion</td>
<td>26 ± 9</td>
<td>38 ± 18</td>
<td>16 ± 6</td>
<td>59 ± 10*</td>
</tr>
</tbody>
</table>

*p < 0.01 compared with RCA lesion in group B.
PAWP = pulmonary artery wedge pressure, RCA = right coronary artery, LAD = left anterior descending artery.

**TABLE IV COMPARISON OF CHARACTERISTICS AT END-POINT BETWEEN THE SUPINE AND SITTING POSITIONS**

<table>
<thead>
<tr>
<th></th>
<th>Supine</th>
<th>Sitting</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>ET (seconds)</td>
<td>485 ± 141</td>
<td>562 ± 123</td>
<td>p &lt; 0.001</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>121 ± 20</td>
<td>126 ± 25</td>
<td>p &lt; 0.05</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>182 ± 35</td>
<td>185 ± 29</td>
<td>NS</td>
</tr>
<tr>
<td>RPP (beats - mmHg/min)</td>
<td>22081 ± 5784</td>
<td>23643 ± 6280</td>
<td>NS</td>
</tr>
</tbody>
</table>

ET = exercise time; HR = heart rate; RPP = rate-pressure product; SBP = systolic blood pressure.

parameters and the extent of the infarcted area in group B.

In group A, pulmonary artery wedge pressure at peak exercise and the extent of the ischemic area were not significantly different between patients with single-vessel disease or double-vessel disease (Table III). Pulmonary artery wedge pressure was higher in patients with left anterior descending artery lesions than in patients with right coronary artery lesions, and the myocardial ischemic area was larger in patients with left anterior descending artery lesions than in patients with right coronary artery lesions, but these differences were not significant. In group B, there was no difference in pulmonary artery wedge pressure at peak exercise between patients with single-vessel or
double-vessel disease or between those with involvement of the right coronary artery or the left anterior descending artery. The extent of the infarct in patients with single-vessel disease was similar to that in patients with double-vessel disease, but was larger in left anterior descending artery lesions than in right coronary artery lesions.

**Supine and Sitting Exercise Tests (Table IV)**

Exercise time was significantly shorter in the supine position than in the sitting position. Heart rate in the peak exercise test was significantly lower in the supine position than in the sitting position, but there were no significant differences in systolic blood pressure or rate-pressure product.

**DISCUSSION**

The present study has demonstrated that pulmonary artery wedge pressure in patients with angina pectoris correlated with the extent of the ischemic area during exercise, and pulmonary artery wedge pressure in patients with old myocardial infarction who did not show any clinical evidence of transient myocardial ischemia was not related to the extent of the infarct. No other reports have suggested this relation in patients with old myocardial infarction.

**Change in Left Ventricular Filling and the Extent of the Ischemic or Infarcted Area**

The increase in pulmonary artery wedge pressure from rest to exercise varied among our patients. The correlation between the extent of ischemia and pulmonary artery wedge pressure was weak at rest and strong during exercise. In 1976, Roskamm et al. observed a relationship between the increase in left ventricular end-diastolic pressure and the size of the ischemic area during exercise-induced angina pectoris when the ischemic area was determined by left ventriculography in terms of the size of the hypokinetic or akinetic area. We used 201TI SPECT to measure the extent of ischemia, since it is an easy and reliable method for quantification of the extent of ischemia. Roskamm et al. found that the exercise-induced increase in pulmonary artery wedge pressure increased along with the number of vessels involved. However, we found no difference in the increase in pulmonary artery wedge pressure between patients with single-vessel disease and those with double-vessel disease, possibly because there was no difference in the extent of the ischemic area between single-vessel disease and double-vessel disease. There were no significant differences in pulmonary artery wedge pressure and the extent of the ischemic area between patients with left anterior descending artery lesions and right coronary artery lesions, but pulmonary artery wedge pressure and the ischemic area tended to be higher in patients with left anterior descending artery lesions, since the left anterior descending artery supplies a larger area of the left ventricle than the right coronary artery. Our findings suggest that the extent of the ischemic myocardium, but not the number or site of the involved vessels, is an important determinant of the increase in left ventricular filling pressure.

In old myocardial infarction, the extent of the infarcted area was not related to heart rate, systolic blood pressure, rate-pressure product, pulmonary artery wedge pressure, cardiac index or stroke volume index, but was correlated with left ventricular ejection fraction and the left ventricular end-systolic volume index, which is consistent with previous findings. In the present study, the extent of the infarcted area was correlated with left ventricular ejection fraction, and with the left ventricular end-systolic volume index, but not with the left ventricular end-diastolic volume index. An experimental study has shown that the increase in regional filling pressure in infarcted myocardium is related to ventricular stiffness. Patients with a small or medium-sized infarct area, in the absence of exercise-induced myocardial ischemia, show generally normal hemodynamics during exercise, whereas patients with a large infarction show an abnormal increase in left ventricular filling pressure during exercise, even in the absence of ischemia. We found no relation between pulmonary artery wedge pressure, either at rest or during peak exercise, and the extent of infarct. Since we selected patients who were able to exercise, none of the patients in the study population had a large infarct. In some patients, however, pulmonary artery wedge pressure increased abnormally, regardless of the extent of the infarct.
Possible Differences in Hemodynamic Mechanisms During Exercise in Acute Ischemia and Old Infarction

The mechanism that maintains the stroke volume during exercise may be present in old myocardial infarction as well as in angina pectoris. In angina pectoris, the nonischemic myocardium distends in response to the stiffened ischemic myocardium, resulting in overall expansion of the left ventricle. We found a linear relation between pulmonary artery wedge pressure and the extent of the ischemic area. On the other hand, in old myocardial infarction, the relation between pulmonary artery wedge pressure and the extent of infarct was not linear, even when stroke volume was preserved by a compensatory mechanism. Previous studies of ventricular structure following myocardial infarction have shown changes in the geometry of the left ventricle, referred to as left ventricular remodeling, including left ventricular dilatation and hypertrophy of myocytes in the noninfarcted myocardium.

Left ventricular dilatation has been found to compensate for resting and exercise hemodynamics for up to 4 weeks after infarction, but becomes noncompensatory if the dilatation is progressive. Noninfarcted myocardial tissue develops contractile dysfunction and an increased passive stiffness within 6 weeks of a large myocardial infarction, but functional changes after a smaller myocardial infarction are minimal, and variably increase in the later stage. However, even when dilatation did not occur and the compensatory mechanism was preserved, structural changes occurred after myocardial infarction. Although it was impossible to directly demonstrate remodeling in the present study because of the lack of data on left ventricular volume before infarction, we found that the left ventricular end-diastolic volume index in patients with old myocardial infarction was higher than that in patients with angina pectoris. Furthermore, the fact that the extent of the infarcted area correlates with left ventricular ejection fraction and left ventricular end-systolic volume index may reflect ventricular remodeling. The correlation between the extent of infarct and the end-systolic volume index implies that an infarct leads to an early increase in end-systolic volume and that increased end-systolic wall stress may be the driving mechanism for late left ventricular chamber enlargement. The abnormal increase in pulmonary artery wedge pressure in patients with old infarction may be related to noncompensatory dilatation.

It is possible that the function of the noninfarcted myocardium was relatively well preserved in patients in the present study because none had large infarcts. The lack of a correlation between pulmonary artery wedge pressure and the extent of infarct may indicate variability in the later stage. Left ventricular remodeling appears to maintain stroke volume without increasing the pulmonary artery wedge pressure during exercise if the infarct is not large. With a large infarct, the noninfarcted area is relatively small, and thus the ability to distend the noninfarcted myocardium is limited. Our findings suggest that in patients with old myocardial infarctions, left ventricular dilatation maintains the stroke volume as a compensatory response to infarction. Stretching of the noninfarcted myocardium does not occur during exercise in these patients as it does in the nonischemic myocardium in patients with angina pectoris. Thus, overall left ventricular stiffness during exercise is higher in acute ischemia than in old myocardial infarction when the infarction is small or medium-sized.

Remodelling may occur even if the extent of the infarct is small. The highest propensity to expansion of the infarct is inherent in the anterior location. In our study, anterior infarction was larger than inferior infarction. Moreover, the degree of remodeling is influenced by post-infarction therapy and infarct size. The post-infarction therapy was unknown in the present study. Pfeffer and Braunwald suggested that the relation between pulmonary artery wedge pressure and infarct size during exercise may depend on the type of post-infarction therapy. Collagen content may also be increased by remodeling. Thus, the change in histology may have played a role in the lack of a relation between pulmonary artery wedge pressure and the extent of the infarcted area.

Limitations

Thadani et al. reported that pressures, including pulmonary artery wedge pressure,
differed in sitting and supine exercise. Our study was consistent with their finding, in that symptom-limited exercise in the sitting position lasted longer than that in the supine position and a similar tendency was noted in comparing pulmonary artery wedge pressure in the supine position to that in the sitting position. However, for 201Tl myocardial SPECT in symptom-limited supine and sitting exercise, the extent of ischemia or infarct may be similar between the 2 positions.

Patients in group B had a relatively good ejection fraction even though they had myocardial infarction. A possible bias is that no patients with severe myocardial infarction were included in our study. However, since drugs were withdrawn at least 3 days before our patients performed symptom-limited exercise, they showed fairly good cardiac function. Patients receiving medications may be examined in a future study.

Conclusions
Our study demonstrated that left ventricular filling pressure was correlated with the extent of ischemia during exercise in patients with angina pectoris. No relationship was found between left ventricular filling pressure and the extent of infarct during exercise in patients with old myocardial infarction. Left ventricular filling pressure appeared to be related to a compensatory mechanism in the myocardium in patients with angina pectoris. In patients with infarction, left ventricular filling pressure did not appear to be related to a compensatory mechanism in the myocardium. The interaction between noninfarcted myocardium and infarcted myocardium in old myocardial infarction may be influenced by ventricular remodeling.

REFERENCES


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