QUANTITATIVE RELATIONSHIPS BETWEEN THALLIUM-201 ESTIMATED MYOCARDIAL INFARCT SIZE AND LEFT VENTRICULAR FUNCTION IN THE ACUTE OR CONVALESCENT PHASE OF THE FIRST ATTACK OF MYOCARDIAL INFARCTION

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Correlations between left ventricular (LV) function and infarct size estimated by computer-assisted thallium (TI)-201 scintigraphy were studied in 16 patients in the acute or convalescent phase of the first attack of transmural myocardial infarction (MI). TI-201 estimation of the infarct size was done using a “corrected” circumferential profile method, by which the total defect score could be obtained. The LV function was evaluated by radionuclide angiography, echocardiography and cardiac catheterization study.

The following results were obtained: 1) A close inverse relationship was found between the defect score and the ejection fraction \( r = -0.649, p < 0.01 \). 2) The linear correlation coefficient was 0.540 \( (p < 0.05) \) between the defect score and the pulmonary arterial end-diastolic pressure and -0.616 \( (p < 0.02) \) between the defect score and the stroke volume index. There was no significant correlation between the defect score and the cardiac index. 3) There was a linear correlation between the defect score and the LV end-diastolic dimension \( r = -0.852, p < 0.001 \). However, there was no relation between the defect score and the left atrial dimension.

When the LV indices were compared between the small (S) and the large (L) defect score group, the L defect group had faster heart rate, larger LV chamber size and the smaller stroke volume index than the S defect group. However, there was no significant difference in the cardiac index between these 2 groups. These results suggest that the LV dilatation in acute or convalescent phase of the first attack of transmural MI is an ominous sign because it was usually accompanied by large infarct size.

The present study also indicates that LV dilatation accompanying a large infarct does not satisfactorily compensate for LV dysfunction by Frank-Starling mechanism, because the stroke volume index decreased in proportion to the infarct size and the cardiac index was maintained by an increase in heart rate in cases with LV dilatation.

Key Words:
Quantitative analysis
TI-201 myocardial images
Acute myocardial infarction
Left ventricular function

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THE procedures which have been used to quantitate myocardial infarct size, such as left ventriculography,1,2 measurement of serum enzymes3-5 and precordial mapping6,7 can estimate the infarct size only indirectly and have
some limitations. On the other hand, thallium (TI)-201 myocardial imaging can disclose directly the anatomical location and extension of a myocardial infarction (MI). Currently, most TI-201 myocardial images are interpreted subjectively by visual identification of the areas having abnormally reduced activity. There have been some reports describing the computer-assisted quantitative analysis of TI-201 myocardial images for evaluating infarct size objectively. We have also reported a computer-assisted method for quantitative analysis of TI-201 myocardial images, using a “correction factor” to correct inherent regional variation of TI-201 radioactivity over the left ventricle (LV). In the present study, we estimated the infarct size of the patients with the first attack of transmural MI using our method (“corrected” circumferential profile method), and evaluated a quantitative relationship between the infarct size and LV function.

MATERIALS AND METHODS

The subjects were classified into 2 groups: One group consisted of 10 healthy young men, ranging in age from 25 to 32 with a mean age of 27.5. All had no previous history or current evidence of cardiovascular disease, and had normal chest X-rays and electrocardiograms. Normal ranges of the “corrected” circumferential profile curves were obtained from this group. The other group included 16 patients with the first attack of transmural MI. The diagnosis of MI was based on the following findings: 1) a typical clinical history of chest pain, 2) electrocardiographic evolution of ST-segment changes compatible with MI, having Q waves of 0.03 sec in duration or the presence of a QS complex on at least 2 of the standard 12 electrocardiographic leads, and 3) a typical rise in serum creatine phosphokinase. During the acute or convalescent period of MI, clinical studies including TI-201 myocardial scintigraphy, cardiac catheterization study using a Swan-Ganz catheter, M-mode echocardiography and radionuclide angiocardiography were performed. Prior to the cardiac catheterization study, none had been treated with diuretics, digitalis, coronary vasodilators or sympathomimetic agents. Drug administration was also stopped at least 12 hours before the non-invasive studies. Patients with arrhythmias, such as atrial fibrillation and atrioventricular conduction
"Corrected" Circumferential Profile Method: TI-201 estimated infarct size was obtained using the "corrected" circumferential profile method which we reported previously. A "corrected" circumferential profile curve for normal controls was drawn in each view from the 10 normal subjects. The LV was outlined visually along the outer edge on the video screen connected to the computer, and divided into equal radial segments by radii (15° arc) constructed from the center of the LV cavity to the outer edge, beginning at 12 o'clock and proceeding clockwise around the outer edge. Each segment was numbered from 1 to 24 in a clockwise rotation. The segments containing the aortic ostium and mitral orifice were excluded from this analysis. Average radioactivity per pixel was obtained in each segment and normalized to the highest segment (=100%). A correction factor for each segment was drawn from the 10 normal subjects. A correction factor was the ratio of 100% to the mean normalized radioactivity in percent in each segment of 10 normal subjects. After correcting the average radioactivity using this factor, and normalized to the highest segment (=100%) in each normal subject, the mean and standard deviation (SD) were calculated from the 10 normal subjects and (100—4SD)% was defined as the normal lower limit (Fig. 1).

In the patients with MI, TI-201 myocardial images were obtained within 17.1 ± 6.7 days (mean ± SD) after the onset of MI. The patient’s "corrected" circumferential profile curve was obtained with the average radioactivity of each segment multiplied by the correction factor and normalized to the highest segment (=100%). This curve was compared with the lower normal limit (Fig. 2). The ratio (%) of the area surrounded by the patient’s profile curve below the lower normal limit and the lower normal limit to the total area below the lower normal limit was obtained at each projection, and the sum of them at 4 projections was called as the total "corrected" defect score.

Cardiac Catheterization Study: All patients except one were examined in the coronary care unit immediately after the admission on average of 1.2 days (range 0 to 4 days) after the onset of MI. Pressure in the right heart and pulmonary artery were obtained using a Swan-Ganz catheter. Cardiac output (CO) was determined with the thermodilution method. Arterial blood pressure was measured by cuff readings. Pulmonary

Disturbance, were excluded from this study.

TI-201 Myocardial Scintigraphy: TI-201 myocardial images were obtained using the standard technique. TI-201 chloride was administered in a dose of 2 mCi when the subjects were supine and at rest. An Anger-type scintillation camera connected to a computer (Scintipac-1200) was equipped with a converging collimator. The camera was closely applied to the chest, and positioned in front of the LV with oscilloscopic monitoring. Images were recorded with a 20% window concentrated on the mercury X-ray peaks (69–80 KeV). Images were obtained at anterior, left anterior oblique (30° and 60°) and left lateral projections 10 min after the administration of TI-201 chloride, and were recorded into a computer and stored on magnetic tape for further analysis. For each view, 400,000 counts were collected in approximately 5 min.

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<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age &amp; Sex</th>
<th>Location of MI</th>
<th>TL201 (defect score)</th>
<th>MABP (mmHg)</th>
<th>HR (beats/min)</th>
<th>CI (L/min/m²)</th>
<th>SVI (ml/beat/m²)</th>
<th>LVSWI (g·m/m²)</th>
<th>TPR1 (dyne·sec·cm⁻⁵·m²)</th>
<th>PAEDP (mmHg)</th>
<th>LVDd index (mm/m²)</th>
<th>LAD index (mm/m²)</th>
<th>LVEF (%)</th>
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<tbody>
<tr>
<td>1</td>
<td>72 M</td>
<td>A</td>
<td>0.44</td>
<td>168/78 (103)</td>
<td>69</td>
<td>2.54</td>
<td>36.8</td>
<td>50.5</td>
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<td>4</td>
<td>32.9</td>
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<td>2</td>
<td>76 F</td>
<td>A</td>
<td>3.78</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>33.3</td>
<td>33.3</td>
<td>51.3</td>
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<td>3</td>
<td>73 F</td>
<td>A</td>
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<td>75</td>
<td>2.74</td>
<td>36.5</td>
<td>40.7</td>
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<td>29.7</td>
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<td>2.46</td>
<td>36.2</td>
<td>31.0</td>
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<td>5</td>
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<td>I</td>
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<td>104/60 (75)</td>
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<td>32.4</td>
<td>2643</td>
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<td>6</td>
<td>70 M</td>
<td>I</td>
<td>8.97</td>
<td>232/120 (157)</td>
<td>68</td>
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<td>82.3</td>
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<td>7</td>
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<td>8</td>
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<td>134/76 (95)</td>
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<td>63 M</td>
<td>I</td>
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<td>2.20</td>
<td>34.4</td>
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<td>A</td>
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<td>2.01</td>
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<td>90/50 (63)</td>
<td>73</td>
<td>2.49</td>
<td>34.0</td>
<td>23.1</td>
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<td>—</td>
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<tr>
<td>13</td>
<td>71 F</td>
<td>A</td>
<td>41.6</td>
<td>110/80 (90)</td>
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<td>62 M</td>
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<td>15</td>
<td>75 F</td>
<td>I</td>
<td>43.7</td>
<td>88/62 (71)</td>
<td>90</td>
<td>2.65</td>
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<tr>
<td>16</td>
<td>67 M</td>
<td>I</td>
<td>47.1</td>
<td>100/74 (83)</td>
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<td>1.48</td>
<td>18.5</td>
<td>16.5</td>
<td>4470</td>
<td>17</td>
<td>49.6</td>
<td>27.0</td>
<td>29.8</td>
</tr>
</tbody>
</table>

**Abbreviations:**
- M = male;  F = female;  MI = myocardial infarction;  A = anterior;  I = inferior;  MABP = mean arterial blood pressure;  HR = heart rate;  CI = cardiac index;  SVI = stroke volume index;  LVSWI = left ventricular stroke work index;  TPR1 = total peripheral resistance index;  PAEDP = pulmonary arterial end-diastolic pressure;  LVDD = left ventricular end-diastolic dimension;  LAD = left atrial dimension;  LVEF = left ventricular ejection fraction obtained by RI angiography
Fig. 3. The relationship between pulmonary arterial end-diastolic pressure (PAEDP) and the TI-201 estimated infarct size (defect score).

Fig. 4. The relationship between the stroke volume index (SI) and the TI-201 estimated infarct size (defect score).

Fig. 5. The relationship between the left ventricular end-diastolic dimension (LVDd) index and the TI-201 estimated infarct size (defect score).

Arterial end-diastolic pressure (PAEDP) was used to determine LV filling pressure. Hemodynamic indices were calculated as follows: mean arterial blood pressure (MABP) = diastolic pressure + (systolic pressure - diastolic pressure)/3 (mmHg); cardiac index (CI) = CO/body surface area (L/min/m²); stroke volume index (SVI) = CI/HR x 1000 (ml/beat/m²), where HR is heart rate (beat/min); left ventricular stroke work index (LVSWI) = SVI x (MABP - PAEDP) x 0.0136 (g·m/m²); and total peripheral resistance index (TPRI) = (MABP - MRAP)/CI x 80 (dyne·sec·cm⁻⁵·m²), where MRAP is mean right atrial pressure in mmHg.

**Echocardiographic Study**: All patients except one underwent an M-mode echocardiographic study within 7.9 ± 4.7 days (mean ± SD) after the onset of MI. M-mode echocardiograms were obtained using a Hitachi EUB-10, a 2.25 MHz 10 cm focused transducer and a strip chart recorder. Measurements of the LV end-diastolic dimension (LVDd) and left atrial dimension (LAD) were made from the M-mode echocardiographic records: 1) LVDd was measured from the endocardial surface of the interventricular septum to the posterior LV wall at the peak of the R wave of electrocardiogram; 2) LAD was measured from the aortic edge of the posterior aortic wall echo to the leading edge of the first strong ultrasonic signal from the left atrial wall at peak anterior aortic motion during systole. LVDd and LAD corrected for body surface area (LVDd index and LAD index) were obtained and these corrected measurements were the subjects of our present study.

**Radionuclide Angiocardiology**: The LV ejection fraction (LVEF) (%) in patients with MI...
TABLE II COMPARISON OF CARDIAC FUNCTION BETWEEN THE SMALL (S) AND THE LARGE (L) DEFECT SCORE GROUP

<table>
<thead>
<tr>
<th></th>
<th>S defect score group</th>
<th>L defect score group</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>TI-201 defect score</td>
<td>9.1 ± 5.84 (9)</td>
<td>35.5 ± 10.8 (7)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>MABP (mmHg)</td>
<td>98.0 ± 26.9 (8)</td>
<td>81.4 ± 11.0 (7)</td>
<td>ns</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>71.5 ± 6.68 (8)</td>
<td>83.7 ± 12.2 (7)</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>CI (L/min/m²)</td>
<td>2.46 ± 0.44 (8)</td>
<td>2.21 ± 0.38 (7)</td>
<td>ns</td>
</tr>
<tr>
<td>SVI (ml/beat/m³)</td>
<td>34.7 ± 6.95 (8)</td>
<td>27.1 ± 6.24 (7)</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>LVSWI (g·m/m³)</td>
<td>42.1 ± 18.5 (8)</td>
<td>24.7 ± 7.71 (7)</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>TPR1 (dyne·sec·cm⁻²·m³)</td>
<td>3270.3 ± 996.6 (8)</td>
<td>3042.4 ± 816.0 (7)</td>
<td>ns</td>
</tr>
<tr>
<td>PAEDP (mmHg)</td>
<td>10.0 ± 4.87 (8)</td>
<td>14.3 ± 4.03 (7)</td>
<td>&lt; 0.1</td>
</tr>
<tr>
<td>LVDD index (mm/m³)</td>
<td>33.9 ± 3.30 (9)</td>
<td>46.6 ± 5.20 (6)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>LAD index (mm/m³)</td>
<td>29.0 ± 6.81 (9)</td>
<td>29.0 ± 4.66 (6)</td>
<td>ns</td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>52.0 ± 17.5 (9)</td>
<td>33.5 ± 13.6 (7)</td>
<td>&lt; 0.05</td>
</tr>
</tbody>
</table>

Numerals in parentheses show the number of cases. ns = not significant. Other abbreviations are the same as in Table I.

was obtained by radionuclide angiocardiology using the first pass technique within 20.9 ± 8.7 days (mean ± SD) after the onset of MI. Twenty mCi of 99m-Tc was injected into an antecubital vein and a gamma camera with a conventional collimator was directed at the patient’s heart from the right anterior 30° projection. The R wave on an electrocardiogram was used as a gate signal and sequential data of 40-msec intervals was acquired in image mode. The sequential data for each 4 beats from the peak time of the right and left low frequency radiogram was selected for multigate processing.

Differences of mean values were evaluated by an analysis of variance and Student's t-test. Correlations were calculated using linear regression analysis.

RESULTS

Table I is a summary of the clinical, hemodynamic and nuclear cardiological data and echocardiographic findings in 16 patients with the first attack of transmural MI. The patients ranged in age from 58 to 83 (mean 71) and included 10 males and 6 females. The locations of abnormal electrocardiographic Q waves were anterior in 8 patients and inferior in 8. The electrocardiographic site of the MI correlated well with the location of MI shown by our "corrected" circumferential profile method. TI-201-estimated infarct size (total "corrected" defect score) ranged from 0.44 to 47.1 (20.6 ± 15.7) (mean ± SD).

Several hemodynamic measurements of intracardiac pressure and flow obtained from a cardiac catheterization study closely correlated with the total "corrected" defect score. The linear correlation coefficient between the total "corrected" defect score and PAEDP was 0.540 (p < 0.05) (Fig. 3), while that between the total "corrected" defect score and the SVI was −0.616 (p < 0.02) (Fig. 4). There was no significant correlation between the total "corrected" defect score and the CI (r = −341, p < 0.2).

As for the internal dimension of the heart measured by an M-mode echocardiogram, there
existed an excellent linear correlation between the total "corrected" defect score and the LVDd index ($r = 0.852$, $p < 0.001$) (Fig. 5). However, there was no relation between the defect score and the LAD index.

The correlation between the total "corrected" defect score and the LVEF obtained from radionuclide angiographic study is shown in Fig. 6. A close inverse relation was found between them ($r = -0.649$, $p < 0.01$).

The LV functional indices were compared between the small total "corrected" defect score group (S defect group, score $< 20$) and the large one (L defect group, score $\geq 20$). Comparison of the results between these 2 groups is summarized in Table II.

The L defect group had a faster HR, a smaller SVI and LVSWI as compared with the S defect group. Differences in mean values of these indices were significant between the 2 groups. There was a tendency for patients in the L defect group to have a higher PAEDP than those in the S defect group. As for the LVEF, a significant difference in mean value was found between the 2 groups.

As for the left-sided cardiac chamber size, although there existed no difference in the LAD index between the 2 groups, the L defect group had a significantly larger LVDd index as compared with the S defect group.

However, there were no significant differences between the 2 groups in the MABP, the CI and the TPRI.

**DISCUSSION**

*Estimation of Infarct Size:* As infarct size is one of the most important determinants of the prognosis of patients with MI²,¹³ its estimation is significant. There have been many reports on the clinical estimation of the infarct size other than TI-201 myocardial imaging, such as left ventriculography¹,² measurements of serum enzymes³⁻⁵ and precordial mapping of electrocardiogram⁶⁻⁷.

TI-201 myocardial imaging can assess the infarct size directly, while the other techniques estimate it by indirect indices. Ventriculography provides information regarding the contractile pattern of the LV. However, it does not provide direct information about regional myocardial blood flow or the distribution of the viable myocardium. Furthermore, this method is highly invasive and is difficult to perform in the acute phase of MI. Enzymatic estimation of the infarct size using creatine phosphokinase has been correlated with biochemical and morphological analysis of the myocardium in experimental¹³ and clinical⁵ assessments of the myocardial necrosis. However, there are certain methodological limitations in particular patients.⁵ Anatomical extension and location of an MI cannot also be evaluated by this procedure.

In contrast to the above-mentioned technique, TI-201 myocardial imaging can directly assess myocardial perfusion under a variety of physical conditions and can evaluate the anatomical location and extension of an MI. However, how to estimate the infarct size both objectively and quantitatively is one of the most important problems. Currently, most TI-201 myocardial images are interpreted subjectively by visual identification of the areas of abnormally reduced activity. Burow et al.⁸ have devised a computer-assisted technique for analyzing TI-201 myocardial images and quantifying the infarct size. We also previously reported a computer-assisted method for quantitative analysis of TI-201 myocardial images (“corrected” circumferential profile method).⁹ Because the TI-201 myocardial image of a normal subject is never homogeneous but has a specific distribution pattern of TI-201 radioactivity over the LV,¹⁰ we developed a correction factor from normal subjects to correct this inherent normal regional variation of TI-201 radioactivity. Both Burow's method and our method can estimate the infarct size objectively. The infarct size obtained by both methods correlates well with a pathologically determined infarct size¹⁴ or with a visually interpreted infarct size obtained from a planar myocardial image.¹⁰

There is a clear tendency for defects on TI-201 myocardial image to decrease in size as the time between the onset of symptoms and the imaging increases.¹⁵ In particular, changes in size of the scintigraphic defects mainly occurred more frequently within 24 hours of an attack of MI than afterwards.¹⁵ During the acute stage of MI, the perfusion defect on TI-201 myocardial image is induced by a combination of myocardial necrosis, ischemia and peri-infarction edema. In order to avoid these changes in defect size on scintigram in the acute phase of MI, we intentionally obtained the images in the convalescent phase of MI, i.e., at least 6 days after the onset of MI. In the convalescent phase, the defect is thought to represent the size of the necrosis of

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the myocardium, which can be generally kept constant except in cases with an extension or a recurrence of MI.

**Infarct Size and Hemodynamic Parameters:** A number of studies have documented that in patients with uncomplicated MI, the CI, the SVI and the LVSWI tend to remain within normal limits; in patients with congestive heart failure, the CI, although being technically still within normal range, is at the lower normal limit, and the SVI and the LVSWI tend to decrease.\(^{16,17}\) Our results were the same as those mentioned above, i.e., although the CI was not different between the L defect group and the S defect group, there existed significant differences in the SVI and the LVSWI. As reported by Scheidt et al.\(^{18}\) the LVSWI seemed to be one of the important prognostic factors, because this factor was intimately related to the infarct size in our present study.

Although some limitations may be exist,\(^{19}\) PAEDP has been used as an index of the left ventricular filling pressure\(^{20}\) and prognostic significance of PAEDP or left ventricular filling pressure has been reported.\(^{16,21,22}\) When we consider the relationship between PAEDP obtained by the initial hemodynamic study and the infarct size estimated in the convalescent phase of MI, there is a definite tendency for having a higher PAEDP in the L defect group than in the S defect group in our present study.

In this study, the TPRI varies over a wide range, and there was no significant difference between patients with large infarct size and those with small one. Hamosh et al.\(^{16}\) have also reported that an increase in peripheral resistance, which might be expected as a compensatory response to a fall in cardiac output, was not uniformly present even in hypertensive patients with shock. It should be recognized that the TPRI is a measure derived from the relation between CO and MABP and, thus, is subject to the clinical limitations of these measurements.\(^{17}\)

**Infarct Size and Cardiac Chamber Size:** There exist many limitations for evaluating LV function using M-mode echocardiography in patients with LV asynergy.\(^{23}\) However, Ratshin et al.\(^{24}\) have suggested that the echocardiographic measurements of the LV end-diastolic dimension and the end-diastolic volume remain accurate even in the presence of asynergy, although the systolic parameters do not. Thus, we studied the effect of the infarct size estimated by the quantitative analysis of a TI-201 myocardial image upon the LV chamber size obtained from echocardiographic records.

Whether MI can cause an enlargement of the heart or not remains controversial. Some reports deny an LV enlargement induced directly by MI.\(^{25-29}\) In experimental study, Hood et al.\(^{27}\) have described that, although the stiffening of the ischemic myocardium and the elevation of LV filling pressure occurred during the first 5 days after MI, it did not signify ventricular dilatation. In clinical study, Broder et al.\(^{28}\) have reported that LVDd was normal on an echocardiogram recorded immediately and 3 weeks after MI.

On the contrary, others have reported the existence of an intimate relationship between the infarct size and the LV volume.\(^{30-32}\) Kostuk et al.\(^{30}\) have found that in many patients the LV became transiently or persistently enlarged on chest roentgenogram after their acute MI. Corya et al.\(^{31}\) have also documented that LV end-diastolic volume on an M-mode echocardiogram correlated with clinical heart failure and increased in 50% of the patients after their first attack of transmural MI. Recently, in experimental study using digital intravenous ventriculography, Gerber et al.\(^{32}\) have reported a significant increase in the LV end-diastolic volume after coronary occlusion. Our present study showed a close relationship between the myocardial infarct size and the LV end-diastolic dimension in patients after their first attack of transmural MI.

It is said that postinfarction cardiomegaly is associated with severe signs of congestive heart failure and a poor prognosis.\(^{31,33,34}\) This finding is compatible with our present results when we consider the interrelations among the infarct size, cardiac enlargement and depressed LV function, that is, a larger LV has a larger infarct size and poor hemodynamic parameters.

Although there have been many reports on the relationship between the infarct size and LV chamber size, few studies have been made on the relation between the infarct size and the left atrial dimension (LAD). The present study suggests that the infarct size did not significantly affect the LAD in the acute or the convalescent phase of the first attack of transmural MI. It has been observed, however, that the M-mode-derived LAD correlated poorly with the normal pulmonary artery wedge pressure, but correlated well with the elevated pulmonary artery wedge pres-

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We also examined the relation of the LAD to PAEDP in our subjects, but there existed no significant correlation between them. This discrepancy may be attributable to a different patient population (i.e., only 5 patients with elevated PAEDP) and/or to our small sample size.

**Infarct Size and LVEF:** The first pass study and equilibrium study by the gate method using a radionuclide are useful non-invasive methods to detect global function and the regional wall motion of the ventricle. The ejection fraction determined by these methods correlated well with that obtained cineangiographically even in the presence of LV asynergy. In this study, we determined the LVEF using the first pass method. As for the relation between the infarct size and the LVEF, our results were in agreement with those of others. That is to say, there was a significant correlation between the infarct size and the LVEF in the patients with the first attack of transmural MI. Therefore, the LVEF is thought to be an important prognostic index because it reflects the infarct size.

**Compensatory Mechanism of the LV in MI:** Compensatory mechanisms of the LV in the acute or the convalescent phase of the first attack of transmural MI are summarized as follows from our present results: Although acute dilatation of the LV is restricted to a limited degree by ventricular compliance, the LV chamber size increases in proportion to the TI-201 estimated infarct size. This dilatation may be a compensatory mechanism to maintain LV performance in accordance with the Frank-Starling principle, that is, an increase of the diastolic fiber length facilitates ventricular contraction and enables the ventricle to eject a greater stroke volume. Furthermore, an elevation of the PAEDP may indicate a mechanism for maintaining the end-diastolic LV volume. However, the compensatory mechanism did not seem to operate well in our present subjects because the SVI and the LSVWI decreased in proportion to the increase of the TI-201 estimated infarct size. The CI was maintained by the increase of HR presumably due to increased plasma catecholamine but not by the increase of SV. In the acute or the convalescent phase of MI, this compensatory mechanism of the heart may fail due to a) a large noncontracting area of the myocardium, b) paradoxical pulsation (dyskinesia) of the infarcted area or c) failure of adequate compensation by increased fiber shortening of the non-infarced myocardium.

### REFERENCES

2. KIMATA S, INOUE Y: Relationship between left ventriculographic findings and clinical symptoms and signs in acute myocardial infarction. *Jpn Circ J* 44: 218, 1980
14. BULKLEY BH, SILVERMAN K, WEISFELDT

changes in left ventricular dimensions and filling pressure in patients after myocardial infarction. Am J Cardiol 33: 363, 1974