THE RESPONSE OF LEFT VENTRICULAR REGIONAL FUNCTION
TO AFTERLOAD STRESS IN PATIENTS WITH OLD
MYOCARDIAL INFARCTION AND
VENTRICULAR ANEURYSM

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The functional response of the left ventricle with scar to increased afterload, was examined in 15 patients with old myocardial infarction and left ventricular aneurysm (OMI). Interventional cine left ventriculography during elevating left ventricular pressure with methoxamine. Wall motion was assessed by the radial and the centerline method. Augmented afterload didn’t change ejection fraction in patients with OMI, but normalized wall motion (Z) increased in the aneurysmal region and decreased in the remote region in both methods. In the remote region in patients with OMI, afterload stress shortened left ventricular pressure-radial length (P-L) loops along length axis, and reduced percent systolic radial shortening (SS).

In the aneurysmal region, P-L loops showed systolic elongation of length at rest and the slope of end-diastolic point to end-systolic point became steeper with increased afterload, resulting in a decrease of aneurysmal expansion.

In summary, with increasing afterload, wall motion decreased in non-infarcted regions and increased in aneurysmal regions, in left ventricles with aneurysm. This mechanism may be interpreted as afterload-induced shifts of P-L loops in each region.

HYPERTENSION and atherosclerotic coronary arterial obstruction frequently coexist in patients, in whom elevation of arterial pressure raises left ventricular wall tension and myocardial metabolic requirements. This leads to an aggravation of myocardial ischemia. On the other hand, myocardial function may be improved by decreasing coronary perfusion pressure, coronary blood flow and collateral flow. Thus, the augmentation of systemic blood pressure seems to act on the myocardium both favorably and unfavorably.

Many investigators have examined the response of left ventricular function to afterload stress in clinical and experimental studies. Kerber et al. studied the effect of methoxamine-induced high blood pressure on segmental dyskinesis during acute myocardial ischemia, and they found a reduction of aneurysmal bulging. Roan et al. showed that phenylephrine-induced systemic arterial hypertension had no effect on the extent of paradoxical systolic

Key words: LV regional function Afterload stress Old myocardial infarction Ventricle aneurysm.

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wall thinning in acute ischemic myocardium after proximal occlusion of the left anterior descending coronary artery in dogs. However, there are few clinical reports concerning the effects of elevated afterload on regional function in old myocardial infarction. Therefore, we investigated the effect of increased afterload on left ventricular regional function in patients with old myocardial infarction and left ventricular aneurysm (OMI).

**METHODS**

**Patients**

Fifteen patients, 11 men and 4 women, were included in this study population. Their age averaged 58.2 years (range 41 to 72). They had suffered from a myocardial infarction, defined by having a history of typical chest pain in association with elevation of serum creatine kinase and characteristic electrocardiographic changes. Nine patients had anterior infarction which resulted from a totally or subtotally occluded left anterior descending coronary artery, and the other 6 patients had inferior infarction with total or subtotal occlusion of the right coronary artery as documented by coronary arteriography. The time period after the onset of acute myocardial infarction up to the time of this study in these patients was a mean of 6.7 months (range 2.5 to 24 months). Five patients with chest pain but normal coronary arteriograms and left ventriculograms served as controls. Thirteen other patients (10 men and 3 women) examined because of atypical chest pain but having no coronary, congenital, or valvular diseases, were used as normal subjects for quantitative left ventricular wall motion analysis (These 13 patients were not evaluated by the afterload stress test). None was taking any diuretics, chronic nitrate or beta-blocking medication at the time of this study. All patients gave informed consent.

**Cardiac catheterization and cine left ventriculography (LVG)**

Cardiac catheterization was carried out via the femoral approach. After routine right heart catheterization, left heart catheterization was performed with a 7F pig-tail catheter (Cordis Corp., Miami, Fla). Left ventricular pressure was measured with a fluid-filled system and Statham P23Db transducer with the reference of mid-chest level. In 5 patients, a high-fidelity micromanometer-tipped angiocatheter (Mikro-tip, model SPC-474A, Millar Instruments, Houston, Tex) was used for LVG. LVG was achieved by injecting 34 to 40 ml of contrast medium (metrizamide for the control LVG) through the catheter at a rate of 12 or 13 ml/sec and recording on 35 mm cine film at 50 frames/sec.

TABLE 1  PATIENT DATA DURING CONTROL AND METHOXAMINE INFUSION

<table>
<thead>
<tr>
<th>Pt NO.</th>
<th>Age</th>
<th>Sex</th>
<th>NoVD</th>
<th>OcV</th>
<th>MI Site</th>
<th>LVSP (mmHg)</th>
<th>LVEDP (mmHg)</th>
<th>EDVI (ml/m²)</th>
<th>SVI (ml/m²)</th>
<th>EF (%)</th>
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<td>54</td>
<td>M</td>
<td>1</td>
<td>F</td>
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<td>16</td>
<td>106.1</td>
<td>57.4</td>
<td>54.1</td>
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<td>Ant.</td>
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<td>127.5</td>
<td>45.9</td>
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<td>2</td>
<td>LAD</td>
<td>Ant.</td>
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<td>52.9</td>
</tr>
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<td>LAD</td>
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<td>8</td>
<td>107.0</td>
<td>47.3</td>
<td>44.2</td>
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<td>LAD</td>
<td>Ant.</td>
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<td>9</td>
<td>82.8</td>
<td>53.4</td>
<td>64.5</td>
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<td>6</td>
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<td>LAD</td>
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<td>137</td>
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<td>102.5</td>
<td>52.9</td>
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<td>LAD</td>
<td>Ant.</td>
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<td>120.9</td>
<td>52.9</td>
<td>43.8</td>
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<td>8</td>
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<td>LAD</td>
<td>Ant.</td>
<td>109</td>
<td>14</td>
<td>126.1</td>
<td>60.8</td>
<td>48.2</td>
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<td>9</td>
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<td>RCA</td>
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<td>19</td>
<td>100.1</td>
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<td>58.1</td>
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<td>128.4</td>
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<tr>
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<td>60.6</td>
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<td>14</td>
<td>70</td>
<td>F</td>
<td>1</td>
<td>RCA</td>
<td>Inf.</td>
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<td>44.1</td>
<td>50.3</td>
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<td>F</td>
<td>3</td>
<td>RCA</td>
<td>Inf.</td>
<td>133</td>
<td>18</td>
<td>118.8</td>
<td>57.4</td>
<td>48.3</td>
</tr>
</tbody>
</table>

Control mean: 133.2 15.2 106.6 52.4 49.9
Control SE: 5.4 1.6 6.4 2.8 2.0

Methoxamine mean: 173.4 21.4 116.7 56.8 50.1
Methoxamine SE: 5.6 1.9 7.5 3.0 2.6

p value: <0.01 <0.01 <0.01 <0.05 NS

Abbreviations: NoVD = number of vessel diseased; OcV = occluded or subtotally occluded vessel; MI = myocardial infarction; LVSP = left ventricular systolic pressure; LVEDP = left ventricular end-diastolic pressure; EDVI = left ventricular end-diastolic volume index; SVI = stroke volume index; EF = ejection fraction; LAD = left anterior descending artery; RCA = right coronary artery; C = control period; M = methoxamine infusion; Ant. = anterior wall; Inf. = inferior wall.

When the micromanometer-tipped catheter was used for LVG, left ventricular pressure, electrocardiogram, cineangiographic frame markers, and an injection marker were simultaneously recorded at a paper speed of 250 mm/sec with a multichannel optical recording system (Mingograph 804, Siemens-Elema). Left ventricular volumes and ejection fractions were calculated by the area-length method of Dodge et al.21 Ventriculograms of sinus beats were traced at end-systole and end-diastole. The boundaries of two left ventricular silhouettes were traced manually by an observer unaware of interventions. The ventricular silhouette with the largest total area immediately before mitral
TABLE II  HEMODYNAMIC AND VOLUME CHANGES IN THE CONTROL PATIENTS (n=5)

<table>
<thead>
<tr>
<th></th>
<th>HR (bpm)</th>
<th>LVSP (mmHg)</th>
<th>LVEDP (mmHg)</th>
<th>EDVI (ml/m²)</th>
<th>SVI (ml/m²)</th>
<th>EF (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>control state</td>
<td></td>
<td></td>
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<tr>
<td>mean ± SE</td>
<td>65 ± 4</td>
<td>126 ± 8</td>
<td>11 ± 3</td>
<td>100 ± 4</td>
<td>58 ± 6</td>
<td>56 ± 5</td>
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<tr>
<td>methoxamine</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>mean ± SE</td>
<td>60 ± 5</td>
<td>175 ± 7</td>
<td>20 ± 4</td>
<td>115 ± 3</td>
<td>69 ± 3</td>
<td>61 ± 4</td>
</tr>
<tr>
<td>p value</td>
<td>NS</td>
<td>&lt;0.01</td>
<td>&lt;0.05</td>
<td>&lt;0.05</td>
<td>&lt;0.05</td>
<td>NS</td>
</tr>
</tbody>
</table>

HR=heart rate; LVSP=left ventricular systolic pressure; LVEDP=left ventricular end-diastolic pressure; EDVI=left ventricular end-diastolic volume index; SVI=stroke volume index; EF=ejection fraction.

Fig.2. The changes of wall motion in control patients (n=5) by methoxamine infusion. Open circles show wall motion in the baseline, and closed circles shows wall motion during methoxamine infusion. (left panel: radial method) Percent radial shortening and Z value are shown. The numbers 1, 13, and 23 mean the radius of anterobasal, apical and posterobasal portions respectively in the left ventricle (Fig. 1-A). (right panel: centerline method) Shortening fraction and Z value of all chords 1 through 100 are shown (Fig. 1-B). In both methods, methoxamine did not depress the regional systolic function in normal myocardium.

valve closure was traced for end-diastole. The end-systolic silhouette was defined as that which had the smallest area before mitral valve opening.

Protocol
First, a simultaneous biplane LVG was performed in the 30 degrees right anterior oblique (RAO) and 60 degrees left anterior oblique (LAO) projections during the control state. Approximately 20 min after the first LVG, when left ventricular pressure had returned to the base line, methoxamine (0.5—1.5 mg/min) was drip-infused to keep elevating the left ventricular systolic pressure by 40 mmHg. Then the second LVG was carried out at the steady state. At the time of the second LVG, heart rate was kept at

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Fig. 3. A typical change of regional shortening (upper panel) and Z value (lower panel) in a patient (patient 2 in Table II) by radial method. Open circles show wall motion during control period. Closed circles show wall motion during methoxamine infusion. Shaded area is normal range of the shortening, (mean ± 2SD) based on measurements in the normal control group (n=13). During methoxamine infusion, wall motion in the aneurysmal region increased (aneurysmal dyskinetic motion decreased), in contrast to a reduction in the remote region.

was considered a significant coronary stenosis.

Quantitative left ventricular wall motion analysis

We used the radial method22, 23 and the centerline methods24-28 for quantitative left ventricular wall motion analysis in 30 degrees RAO view of LVG. In the radial method, the major axis in the RAO view was defined as the line from the apex to the midpoint of the aortic valve plane. The end-diastolic and end-systolic frames were superimposed along the major axis and at its center (Fig. 1-A). Lines (radii) were drawn from the center of the axis to the endocardial margin at 12 degrees intervals. The line from the center to the midpoint of the aortic valve served as zero reference. The left ventricular wall motion was evaluated by 23 radii (36 to 300 degrees) which corresponded to the ventricular muscular portion. Radial length at end-diastole (EDRL) and at end-systole (ESRL) were measured, and percent systolic radial shortening (SS) was calculated as

$$SS = \frac{EDRL - ESRL}{EDRL} \times 100\%$$

In the centerline method24-28 wall motion was measured along 100 equidistant chords perpendicular to a centerline drawn between the end-diastolic and the end-systolic contours and numbered clockwise from the anterior aortic valve (Fig. 1-B). To normalize for heart size, the motion at each chord was divided by the end-diastolic perimeter to yield a dimensionless shortening fraction (SF). Chords 1 to 10 and 81 to 100 were excluded because of high variability in these regions27.

Since the extent of regional wall motion varies among the regions of the left ventricle (ex. anterior vs posterior wall), we normalized SS by calculating Z values in all radial segments according to the following formula22, 23

$$Zr = \frac{SSr - Mr}{SDr}$$

SSr is SS in a radius, r, for the 15 patients with OMI or the 5 control patients. Mr is the mean SS for the 13 normal subjects in a radius, r. SDr is the standard deviation for

the control value by right atrial pacing to prevent reflex bradycardia provoked by methoxamine infusion. We kept the distance from the X-ray tube to the subject constant on the first and second LVG. A 50 millimeter length lead bar was used for calibration. Diaphragm movement was excluded by keeping maximum inspiration during both LVGs. No patient developed chest pain or other symptoms, and no arrhythmia or ST changes on electrocardiogram occurred during this study. After the protocol, each patient had selective coronary angiography by Judkins' technique. Seventy-five percent or more of coronary artery diameter narrowing...
SS for the 13 normals in a radius, r. In other words, \( Z_r \) represents the number of standard deviations by which the measurement SSr varies above or below the mean of the 13 normal subjects at that radius, r. In the same way, the normalized motion or shortening fraction (SF_i) of each chord i was converted into units of standard deviations (SD_i) from the normal group mean Mi^25–28:

\[
Z_i = \frac{SF_i - Mi}{SD_i}
\]

A more negative \( Z \) value indicates that the regional systolic function is worse.

In all 15 patients with OMI, anterior or inferior aneurysm was detected in 30 degrees RAO projection of the first LVG. We visually determined the aneurysmal region, which exhibited either paradoxical systolic expansion or apparent lack of motion (akinesia), by superimposing end-systolic frame on end-diastolic frame. We defined the region neighboring the aneurysmal region as the adjacent region, which contained 3 radii (or 9 chords), at both anterior and inferior sides from aneurysmal radii. The remote region included the remaining radii with the exception of 2 radii (or 6 chords) neighboring the adjacent region.

In the radial method, we calculated SS, \( Z \) value and EDRL in all radii, and compared 3 radii in each region (the most hypokinetic 3 radii in the aneurysm region; the most hyperkinetic 3 radii in the remote region; the median 3 radii in the adjacent region), before and during methoxamine infusion.

In the centerline method, in analyzing serial studies, the most hypokinetic 5 chords in the aneurysm regions in the 2 studies were compared. The most hyperkinetic 5 chords in the remote regions were also compared. This comparison was done, because changes in ventricular shape might cause renumbering of chords because of relative changes in centerline length so that regions might have different chord numbers when restudied.

*Japanese Circulation Journal Vol. 55, December 1991*
Pressure-length relationships

Sequential ventricular frames (RAO) were superimposed on the end-diastolic frame throughout 1 cardiac cycle, and 23 radial lines were drawn from the center of major axis to the endocardial margins in each frame (Fig. 1-A). The aneurysmal segment was defined as the radius which had the greatest negative Z value among aneurysmal radii at the first LVG. In the same way, the remote (non-infarcted) segment was defined as a radius which had the highest Z value among remote radii at the first LVG. The radial length of each frame was measured, and sets of radial length and the corresponding left ventricular pressure were plotted on the pressure-length plane with a non-computerized method.

STATISTICS

All data were expressed as mean±SE. Paired dimensional data were analyzed with the paired t test. Unpaired dimensional data were analyzed using unpaired t test. A p value of less than 0.05 was considered significant.

RESULTS

Hemodynamic and ventricular volume changes in patients with left ventricular aneurysm and in control patients

Hemodynamic variables before and after methoxamine infusion in patients with OMI are shown in Table I. From resting state to the state of methoxamine infusion, left ventricular systolic pressure, end-diastolic pressure, end-diastolic volume index, and stroke volume index increased significantly (133.2±5.4 to 173.4±5.6 mmHg; p<0.01, 15.2 ± 1.6 to 21.4±1.9 mmHg; p<0.01, 106.6 ± 6.4 to 116.7±7.5 ml/m²; p<0.01, and 52.4 ± 2.8 to 56.8±3.0 ml/m²; p<0.05,
respectively). No significant changes were found in heart rate and ejection fraction (64.8 ± 2.2 to 63.4 ± 2.4 beats/min, and 49.9 ± 2.0 to 50.1 ± 2.6%; ns, respectively).

Hemodynamic and ventricular volume changes in control patients were shown in Table II. There were significant increases in left ventricular systolic pressure, end-diastolic pressure, end-diastolic volume index, and stroke volume index (126.2 ± 8.0 to 175.3 ± 6.5 mmHg; p < 0.01, 11.2 ± 2.5 to 20.1 ± 4.0 mmHg; p < 0.05, 100.2 ± 4.0 to 115.3 ± 3.4 ml/m²; p < 0.05, and 58.4 ± 6.2 to 69.1 ± 3.1 ml/m²; p < 0.05, respectively). Heart rate decreased slightly, and ejection fraction increased slightly, but neither of these changes were significant.

**Regional function in control patients**

Changes of SS and Z value in control patients were summarized in Fig. 2. Methoxamine infusion increased SS (SF) and Z in inferior wall slightly, but not significantly. In the anterior wall, methoxamine did not change the regional systolic function. Thus, methoxamine did not depress the regional function in normal myocardium.

**Regional function in aneurysmal, adjacent and remote regions in patients with left ventricular aneurysm**

A representative change of SS and Z value evaluated by the radial method in a patient with anterior old myocardial infarction with left ventricular aneurysm is shown in Fig. 3. Augmenting the afterload caused a reduction of wall motion in the remote region and an increase in wall motion in the aneurysmal region. This phenomenon was recognized in the centerline method (Fig. 4). In Fig. 5, the changes of SS, Z value and EDRL in patients with OMI are summarized using the radial method. After methoxamine was infused, Z value increased in the aneurysmal region (-4.82 ± 0.18 to -3.88 ± 0.16; p < 0.01) and decreased in the remote region (0.36 ± 0.15 to -0.62 ± 0.13; p < 0.01). There was no significant change in the adjacent region (-1.80 ± 0.22 to -2.02 ± 0.24; ns). SS increased in the aneurysmal region (-3.51 ± 1.12 to 2.42 ± 1.06%; p < 0.01) and decreased in the remote region (46.56 ± 1.40 to 36.2 ± 1.32%; p < 0.01). There was no significant difference in changes of EDRL in these regions (aneurysmal: 38.2 ± 0.8 to 38.5 ± 0.8, adjacent: 31.4 ± 0.9 to 32.0 ± 0.9, and remote region: 34.1 ± 0.9 to 34.4 ± 1.0 mm; ns). In Fig. 6, the changes of SF and Z value were evaluated in the centerline method in the patients with OMI. SF and Z increased in the aneurysmal region (SF; -0.73 ± 0.11 to 0.04 ± 0.11; p < 0.01 and Z; -5.08 ± 0.09 to -4.15 ± 0.12; p < 0.01), and decreased in the remote region (SF; 5.12 ± 0.18 to 3.96 ± 0.20; p < 0.01 and Z; -0.35 ± 0.15 to -1.21 ± 0.12; p < 0.01). In the adjacent region, SF and Z did not change significantly (SF; 1.55 ± 0.11 to 1.42 ± 0.13 and Z; -2.48 ± 0.08 to -2.60 ± 0.08). In this study, 9 patients had

Fig. 7. Changes of Z values (radial method) in aneurysmal (An), adjacent (Ad) and remote (Re) regions in single vessel group (SVD: left panel) and multivessel group (MVD: right panel) with methoxamine infusion. Afterload stress reduced Z value in remote region and increased Z value in aneurysmal region in both groups. However, in the adjacent region, a different responses to afterload stress was found between the 2 groups.

Fig. 8. Representative pressure-radial length (P-L) loops in remote non-infarcted region (left panel) and in aneurysmal region (right panel) in a patient (patient 2 in Table I). Open circles show P-L relationship at the control state, and closed circles during methoxamine infusion (see text). LVP: left ventricular pressure. ES: end-systole, ED: end-diastole.
an isolated occluded coronary artery (single vessel group; SVD), and 6 patients had 2 or 3 diseased vessels (multivessel group; MVD) (Table I). Fig. 7 showed changes of Z value (by the radial method) in each region in both SVD and MVD with methoxamine infusion. After methoxamine, Z decreased in the remote region in 2 groups (SVD; 0.69±0.21 to −0.54±0.21; p<0.01 and MVD; −0.15±0.15 to −0.73±0.11; p<0.01). Z increased in the aneurysmal region in 2 groups (SVD; −4.97±0.23 to −3.98±0.21; P<0.01 and MVD; −4.59±0.29 to −3.72±0.25; p<0.01). In the adjacent region, Z decreased in SVD (−2.32±0.20 to 2.81±0.23; p<0.01). However, there was no significant difference in Z value in MVD (−1.84±0.18 to −1.74±0.25; ns).

Pressure-length relationship in both aneurysmal and remote regions.

The left ventricular pressure-radial length (P-L) loops was studied in 5 patients. Fig. 8 shows representative P-L loops obtained from patients before and after methoxamine infusion. In the aneurysmal radius, the P-L loop at the control state was warped indicating that aneurysm plays no part in ejecting blood and that wall motion of aneurysm is a passive phenomenon. The slope of end-diastolic point to end-systolic point after methoxamine infusion was steeper than that before methoxamine infusion, which indicated a reduction of aneurysmal expansion. In 5 cases, the slopes during methoxamine infusion were steeper than during the control state (102.5±65.0 to 159.8±73.0 mmHg/mm; p<0.05). The P-L loop of the remote radius showed rectangular configuration at the basal state. With augmented afterload, the loop showed a deformation which was expanded along the pressure-axis and shortened along the length-axis, leading to a reduction of systolic radial shortening.

DISCUSSION

In the present study we demonstrated that increasing afterload reduced wall motion of the remote, noninfarcted region and increased that of the aneurysmal region. In addition, the P-L loops in the aneurysmal region increased its slope of end-diastole to end-systole and deformed in the noninfarcted region in those patients with left ventricular aneurysm by methoxamine infusion. We used the pure alpha-adrenergic drug, methoxamine, to increase blood pressure. Heart rate was unchanged during the study.

Mechanisms of increased wall motion in aneurysmal region after afterload increase

The most plausible explanation for reduced aneurysmal dyskinetic motion by increased afterload would be that, once methoxamine is infused, the aneurysmal region commences functioning on the steeper portion of the exponential curve of its length-tension relationship. It has been reported that asynery determined by cine-LVG can be an indicator of fibrosis within the ventricle. So, in this study, the akinetic or dyskinetic region in LVG may consist mainly of fibrotic scar tissue, which is considered to be completely noncontractile and to have passive length-tension characteristics. It has been demonstrated that chronic ventricular aneurysm functions on the nearly exponential curve in its length-tension relationship. In the present study, we observed that, in the P-L loops of aneurysm, the slope of end-diastolic point to end-systolic point became steeper by afterload stress. That is, aneurysm came to move in the stiffer, less compliant, portion. As consequence, aneurysmal dyskinetic motion was reduced.

Alternatively, the mechanism of increased wall motion (or decreased aneurysmal expansion) in the aneurysmal region may be explained by mechanical intraventricular interaction between infarcted and noninfarcted myocardium. The aneurysmal region, which is noncontractile, moves passively, and its motion seems to be partially determined by the motion of the noninfarcted region within the ventricle. Considering the infarct ventricle as a two component spherical model, consisting of the non-infarcted component and the aneurysmal component, when the motion of the non-infarcted myocardium decreases with increased afterload, the systolic expansion of the aneurysm may decrease through intraventricular interaction.

Mechanism of decreased wall motion in remote (noninfarcted) region by afterload stress

Many investigators in canine
models of acute myocardial ischemia, have shown that ejection phase indices of regional function, such as segment shortening or wall thickening, were reduced in the nonischemic region with elevated afterload. The mechanisms of this phenomenon are still unclear. This phenomenon was observed in patients at the chronic stage of myocardial infarction.

To explain reduced wall motion with increased afterload, the concept of the force-velocity-length relation of myocardial fiber shortening could be applied to in vivo noninfarcted myocardium. That is, if regional preload (EDRL) in noninfarcted segments is not allowed to undergo compensatory augmentation for a sudden increase in the afterload, wall motion of noninfarcted region may be decreased by afterload elevation. When the left ventricular remodeling occurs in noninfarcted myocardium as compensatory volume overload hypertrophy after acute myocardial infarction, the myocardium suffers from a "pathologic" hypertrophy late after the onset of myocardial infarction, even if initially it is a "physiologic" hypertrophy. The myocardium with pathologic hypertrophy may produce stress-induced dysfuncti

As another explanation, altered relationship between myocardial oxygen supply and demand may be considered in the noninfarcted region due to afterload stress. Left ventricular wall tension, oxygen consumption and coronary arterial flow increase as the systemic arterial pressure increased. Since the resultant effect of the increased arterial pressure is so variable, it is possible that increased aortic pressure produces the disproportion between regional wall stress and regional blood flow in noninfarcted region in our study.

Study limitation

We used the radial method, in which deformation of left ventricles might influence our regional wall motion analysis, to evaluate left ventricular regional wall motion. We determined apical point and major axis carefully in this method, and excluded the data of 1 patient which showed large dislocation of axis by the intervention and the data of 1 patient in which the apical point was unclear. We analyzed regional wall motion in all subjects with the centerline method to validate our results analyzed with the radial method. The same results were obtained by both methods, and we consider that the methodology has not influenced our conclusions.

We think contrast media used in the first LVG may influence left ventricular function at the time of the second LVG to a minimum. We performed the second LVG at the time when left ventricular pressure returned to base-line. A nonionic contrast agent, metrizamide, in the first LVG was used for its less depressive effect on cardiac performance.

We used an alpha-adrenergic agent, methoxamine hydrochloride, for augmentation of afterload. The existence of alpha-1-adrenoceptors mediating positive inotropic effect in the heart of various mammalian species is well established. However, this positive inotropic effect is less than that produced by beta-adrenoceptor-stimulating agents in many clinical cases except the patients with chronic beta-blocker therapy and hypothyroidism. So, in our study, methoxamine-induced positive inotropic action may be almost negligible.

We investigated the acute effect of increasing afterload in the patients with OMI and ventricular aneurysm, and clarified that they maintained homeostasis of left ventricular function under the condition with a sudden increase in afterload. We did not examine left ventricular function in a longer period of afterload stress, but chronic hypertension involved increasing left ventricular wall stress. We could speculate that this increased regional wall stress might lead to decreased contractility in the non-infarcted segment and infarct expansion in the infarcted segment, which could be responsible for late heart failure.

To conclude, we demonstrated that wall motion of the noninfarcted region decreased and that wall motion of the infarcted region increased (that is, paradoxical motion of aneurysm was decreased), in left ventricles of patients with old myocardial infarction and left ventricular aneurysm by afterload stress. This phenomenon may be explained by the transformation of P-L loops in noninfarcted and aneurysmal regions. Even though increased afterload produced such antipodal effects on each region, the left
ventricular global function remained the same.

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