REDUCED EARLY DIASTOLIC EXTENSION IN THE INFARCTED PORTION IN PATIENTS WITH OLD MYOCARDIAL INFARCTION

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To study relaxation characteristics of the infarcted myocardium, cyclic changes in the global left ventricular (LV) volume were measured in 20 patients with old myocardial infarction (OMI) and 17 normals (Normal) and those in the regional segment length were measured in 9 patients with anterior old myocardial infarction (anterior OMI) and 11 normals. The LV volume was calculated by using biplane LV cineangiograms. The regional segment length was calculated by measuring the spatial length between the 2 points of the ramifying branches on the left coronary arteries by using biplane coronary cineangiograms. The LV filling volume before atrial contraction \( (V_R) \) was significantly less in the OMI compared with that in the normals (Normal 38 6 (mean SD) ml/m² vs 30 7 ml/m²: \( p < 0.01 \)), while filling volume by atrial contraction \( (V_a) \) did not significantly differ (Normal 15 4 ml/m² vs OMI 17 5 ml/m²). The lengthening of the segmental wall during diastole before atrial contraction \( (%L_R) \) in the infarcted portion was 5.0 2.9% which was also significantly less than that in the non-infarcted portion (9.6 4.2%). The extent of lengthening by atrial contraction \( (%L_a) \) did not differ between the 2 portions (non-infarcted portion 3.8 1.1% vs infarcted portion 3.5 1.2%). Reduction of \( %L_R \) was speculated to be caused by the incomplete relaxation in the myocardium adjacent to the infarcted portion and stiff myocardium in the infarcted portion. This study suggests that the infarcted myocardium may lead to a reduction of diastolic expansion before atrial contraction.

We studied a group of patients who showed abnormally high left ventricular (LV) end-diastolic pressure but normal LV end-diastolic volume\(^1-3\). Therefore, it was considered that the LV diastolic pressure-volume (or pressure-length) relationship was impaired in those patients. In patients with myocardial infarction, the LV pressure-volume relationship is characterized by an augmented "a" wave of the LV diastolic pressure caused by an increase in LV volume by atrial contraction. Although this characteristic pressure-volume relationship in diastole has been well studied\(^4-6\), little information is available concerning pressure-length curve in the infarcted portion. The aim of the present study is to analyze how the LV volume and lengthening of the segment length of the left ventricle are influenced during early diastole and by atrial contraction in myocardial infarction.

Key words:
Rapid filling
Atrial contraction
Diastolic compliance
Myocardial infarction

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TABLE I  CLINICAL CHARACTERISTICS OF NORMAL SUBJECTS AND PATIENTS WITH MYOCARDIAL INFARCTION

<table>
<thead>
<tr>
<th></th>
<th>Normal</th>
<th>Myocardial infarction</th>
</tr>
</thead>
<tbody>
<tr>
<td>No.</td>
<td>17</td>
<td>20</td>
</tr>
<tr>
<td>Age (y)</td>
<td>45 ± 17</td>
<td>51 ± 11</td>
</tr>
<tr>
<td>(mean ± SD)</td>
<td>Range</td>
<td>16 – 65</td>
</tr>
<tr>
<td>Male</td>
<td>10</td>
<td>17</td>
</tr>
<tr>
<td>Female</td>
<td>7</td>
<td>3</td>
</tr>
<tr>
<td>NYHA</td>
<td>I</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td>II</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td>III</td>
<td>2</td>
</tr>
<tr>
<td>Site of infarction</td>
<td>Anterior</td>
<td>7</td>
</tr>
<tr>
<td></td>
<td>Inferior</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td>Anterior + inferior</td>
<td>4</td>
</tr>
<tr>
<td>Extent of coronary artery disease</td>
<td>1 vessel</td>
<td>10</td>
</tr>
<tr>
<td></td>
<td>2 vessel</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>3 vessel</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>LMT</td>
<td>1</td>
</tr>
</tbody>
</table>

Abbreviations: SD = standard deviations; NYHA = New York Heart Association functional class. Coronary arteriography was not performed in two patients with anterior myocardial infarction.

METHODS

The subjects consisted of 20 patients with old myocardial infarction (OMI) and 17 patients with chest pain who were diagnosed as normal (Normal) by left ventriculogram and coronary cineangiogram (Table I). Nine patients with anterior OMI and 11 patients with chest pain diagnosed as normal underwent biplane left coronary cineangiogram to analyze segmental diastolic filling (Table II). Cardiac catheterization was performed three months later from the onset of acute myocardial infarction. Left ventriculogram was performed in the right and left anterior oblique projection. LV pressure, electrocardiogram and injection signals were recorded on an oscillographic paper (Hewlett-Packard Inc., Type 4588). High-fidelity LV pressure was recorded with a micromanometer-tipped catheter (Millar Instruments Inc., Type PC 481). To adjust the zero-point of a micromanometer-tipped catheter system, precalibrated LV pressure by a micromanometer-tipped catheter was superimposed on LV end-diastolic pressure by a fluid-filled catheter. The lowest point of LV pressure was denoted as $P_{nadir}$ and the pressure at the beginning of the P wave on the electrocardiogram (ECG) as $P_{E_{2C}G}$. 40 ml of 76% Urographin (amidotrizoate sodium meglumine) was injected at a rate of 15 ml/sec to obtain the LV cineangiogram. The cinefilm was obtained at 90 frames/sec and projected on a motion analyzer (Vanguard Inc., Model M-35C, S-13) and the ventricular image was traced with a graph pen (Science Accessories Corporation, GP-3HP). LV volume was calculated with an angioanalyzer system (HP 5693A) programmed according to the biplane area-length method of Dodge et al.7,8 and Rackley et al.9 When ventricular premature contraction occurred during cineangiogram, premature beats and their subsequent beats were excluded from the analysis. LV end-diastolic volume (EDV) was determined as the maximum volume and LV end-systolic volume (ESV) as the minimum volume (Fig. 1). LV volume at $P_{nadir}$ was denoted as $V_{nadir}$, and likewise LV volume at the beginning...
of the P wave on the ECG as \( V_{ECG \ p} \). The LV diastolic filling period was divided into that before atrial contraction and that by atrial contraction. The LV volume was divided into the volume increase before atrial contraction (\( V_R \)) and that by atrial contraction (\( V_a \)).

The biplane left coronary cineangiogram was taken at a film speed of 50 frames/sec with simultaneous LV pressure recordings. The regional segment length was calculated by

\[
L = \sqrt{(X_n - X_m)^2 + (Y_n - Y_m)^2 + (Z_n - Z_m)^2}
\]
measuring the spatial length between the 2 points of the ramifying branch on the left coronary arteries by using biplane coronary cineangiograms. The 3-dimensional coordinates of the points (X, Y, Z) were measured. The segment length (L) between one point (Pn) and the other point (Pm) was calculated as follows (Fig. 2):

\[ L = \sqrt{(Xn-Xm)^2 + (Yn-Ym)^2 + (Zn-Zm)^2} \]

This calculation made it possible to depict the cyclic changes in the shortening and lengthening of the segment length during one cardiac cycle. The following indexes were calculated from the segment length curve:

- \( L_{\text{min}} \): Segment length where the average of three consecutive values shows the minimum value (minimum segment length).
- \( L_{\text{nadir}} \): Segment length at the nadir of LV pressure.
- \( L_{\text{ECG P}} \): Segment length at the onset of the P wave on the ECG.
- \( L_{\text{EDP}} \): Segment length at the LV end-diastolic pressure.
- \( L_{\text{max}} \): Segment length where the average of three consecutive values shows the maximum value.

Segment length in the infarcted portion (anterior wall in the anterior OMI) was evaluated by the electrocardiogram and left ventriculogram, and it was the spatial distance between the bifurcations of coronary artery perfused with the stenotic portion of left anterior descending artery. Segment length in the inferior wall was the spatial distance between the bifurcations of left circumflex coronary artery except the portion of the auriculoventricular groove.

Values are expressed as mean \( \pm \) standard deviation. Comparison among groups was performed by one-way analysis variance. If the F statistics were significant at 0.05 level, t-tests were used to find where the difference existed.
### RESULTS

The phasic changes of LV volume in a normal subject and a patient with OMI are shown in Fig. 1. As a whole, the LV volume increased before P wave ($V_R$) significantly less in the OMI ($30 \pm 7 \text{ ml/m}^2$) than in the normals ($38 \pm 6 \text{ ml/m}^2$). There was no difference in the amount of LV filling by atrial contraction ($V_a$) between the normals and OMI (Fig. 3, Table III). Early extension ($\%L_R$) in a patient with anterior OMI is less than that in a normal subject (Fig. 4, Table IV, V). There was no difference in late extension ($\%L_a$) between the anterior wall in the normal and the anterior wall in the anterior OMI (infarcted portion). The lengthening of segmental wall in diastole before atrial contraction was markedly less in the OMI compared with that in the normals (Fig. 5). The diastolic extension before atrial contraction ($\%L_R$) was reduced to $5.0 \pm 2.9$% only in the infarcted portion in the anterior OMI. In the non-infarcted segment, on the contrary, $\%L_R$ was not less. However, $\%L_a$ in the infarcted segment was equal to that of normals (Fig. 5).

### DISCUSSION

The present study is unique in that the diastolic changes in both infarcted and non-infarcted segments were separately measured by using biplane coronary cineangiograms. The following two findings were obtained. Extent of lengthening before the onset of P wave ($\%L_R$) was significantly less in the infarcted portion than in the non-infarcted or normal portion. Extent of lengthening by atrial contraction ($\%L_a$) in the infarcted portion showed no difference from that in the non-infarcted and normal portion. From present results, LV end-diastolic pressure and nadir pressure were significantly higher in the OMI than in the normals (Fig. 6). This suggests that diastolic pressure-volume relation operates at a higher pressure in the OMI than in the normals. Although the increase of volume by atrial contraction remained the same between the normals and the OMI, the pressure elevation by atrial contraction (pressure elevation from the onset of P wave to end diastole) was greater in the patients with OMI than in the normals. This indicates that the left ventricular wall is stiffer in myocardial infarction. The myocardial lengthening in the infarcted portion by atrial contraction ($\%L_a$) was not significantly different from that of

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### TABLE IV
**DIASTOLIC SEGMENT LENGTH OF THE LEFT VENTRICULAR ANTERIOR AND INFERIOR WALLS IN NORMAL SUBJECTS AND THE PATIENTS WITH ANTERIOR MYOCARDIAL INFARCTION**

<table>
<thead>
<tr>
<th></th>
<th>Diastolic extension</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Total extension (%L)</td>
</tr>
<tr>
<td><strong>L&lt;sub&gt;max&lt;/sub&gt; (mm)</strong></td>
<td><strong>L&lt;sub&gt;nadir&lt;/sub&gt;</strong></td>
</tr>
<tr>
<td><strong>Anterior wall</strong></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>65.7 ± 21.8</td>
</tr>
<tr>
<td>Inferior wall</td>
<td>79.7 ± 21.6</td>
</tr>
<tr>
<td><strong>Anterior myocardial infarction</strong></td>
<td></td>
</tr>
<tr>
<td>Anterior wall (infarcted portion)</td>
<td>63.2 ± 20.5</td>
</tr>
<tr>
<td>Inferior wall (non-infarcted portion)</td>
<td>79.9 ± 25.3</td>
</tr>
</tbody>
</table>

L<sub>nadir</sub> = length at the nadir of LV pressure; L<sub>ECG</sub> P = length at onset of the P wave on the electrocardiogram; L<sub>EDP</sub> = length at the LV end-diastolic pressure; L<sub>max</sub> = maximum length; *p < 0.05; **p < 0.02; ***p < 0.01; SD = standard deviation.

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### TABLE V
**PRESSURE DATA IN NORMAL SUBJECTS AND THE PATIENTS WITH ANTERIOR MYOCARDIAL INFARCTION**

<table>
<thead>
<tr>
<th></th>
<th>Anterior myocardial infarction mean</th>
<th>Normal mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abnormal</td>
<td>+2.2**</td>
<td>+5.1**</td>
</tr>
<tr>
<td>PdAd/PdD</td>
<td>+2.8</td>
<td>+2.6</td>
</tr>
<tr>
<td>PdAd/ECG P</td>
<td>+12.0**</td>
<td>+2.2</td>
</tr>
<tr>
<td>EDP</td>
<td>+3.9</td>
<td>+2.2</td>
</tr>
</tbody>
</table>

**Abbreviations:** See Table III

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ANTERIOR WALL OF THE LEFT VENTRICLE

Fig. 4. Segment length curve of left ventricular anterior wall in a normal subject (left) and in a patient with anterior myocardial infarction (right). The extent of lengthening before the P wave (%L_R) in the patient with anterior myocardial infarction is less compared with that in the normal subject.

Fig. 5. The extent of lengthening in the normals and in the patients with anterior myocardial infarction. %L_R of anterior wall in the patients with anterior myocardial infarction (infarcted portion) is significantly less, as shown of the left side, while %L_R showed no difference among them. SD: standard deviation. We can measure 1 to 3 segments in each case. Accordingly, the number of dots exceeds the number of cases.

wall) in the anterior OMI. Namely, it is suggested that the reduction in extension in the infarcted portion directly influenced early diastolic filling in the OMI.

Although, the increase in LV volume by atrial contraction is the same in both the OMI and
normals, the pressure rise by atrial contraction is significantly larger in the OMI. Two reasons might be cited for this difference. Since the infarcted myocardium is stiff, LV pressure might be elevated more by atrial contraction. Since the blood volume removed from the left atrium to LV during early diastole is reduced in OMI, left atrial (LA) volume at the onset of LA contraction is greater, resulting in greater ejection power of the left atrium, according to Frank-Starling's mechanism.\(^{19,20}\)

In conclusion, this study demonstrated that early (rapid) filling of the left ventricle, rather than atrial filling, is impaired in patients with OMI. We consider that the increased stiffness and incomplete relaxation in the infarcted myocardium are the possible mechanisms that cause

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Reduced Early Diastolic Extension


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