Effects of Left Ventricular Chamber Size and Left Ventricular Diastolic Pressure on Left Atrial Booster Pump Function in Patients with Old Myocardial Infarction

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SUMMARY

We characterized the influence of the size of the left ventricular (LV) chamber (LV diastolic diameter, LVDD) and LV end-diastolic pressure (LVEDP) on left atrial active stroke index (LAASI) in patients with old myocardial infarction (OMI) (n = 118) and controls (n = 25). Patients with OMI were classified into four groups according to the grade of LVDD (≥55 or <55 mm) and LVEDP (≥18 or <18 mmHg). LAASI was determined by multiplying the LV stroke index by the fraction of left atrial active contraction of the velocity-time integral on the pulsed-wave Doppler echocardiogram. LAASI in the “LVDD ≥55 & LVEDP ≥18” group was less than that in the groups with normal LVEDP. LAASI did not differ among the normal LV dimension groups. The combination of LV enlargement and high LVEDP was related to the decrease in LAASI in patients with OMI. (Jpn Heart J 1997; 38: 651–662)

Key words: Left atrial active stroke index, Left ventricular dilatation, Left ventricular end-diastolic pressure, Old myocardial infarction

The diastolic blood flow produced by left atrial active contraction has important implications for maintaining the preload of the left ventricle when left ventricular diastolic compliance is reduced.1 This is known as the booster pump function of the left atrium. Expansion of the infarcted area, hypertrophy of the residual wall, and dilatation of the left ventricle following myocardial infarction may alter the chamber compliance of the post-infarcted left ventricle.2 These pathophysiological changes may affect diastolic filling by the left atrial booster pump function.3–5

The flow velocity profile with the pulsed-wave Doppler echocardiogram is
used in routine clinical practice to evaluate this diastolic filling into the left ventricle. However, this flow velocity is insufficient to estimate the actual changes in blood volume.\textsuperscript{6-8} We thought that quantitative determination of the actual blood volume from the left atrium to the left ventricle by left atrial active contraction, that is, the left atrial active stroke volume, would help to clarify part of the left ventricular diastolic property of the post-infarcted heart.

Our objective was to characterize the influence of the left ventricular enlargement and left ventricular diastolic pressure on left atrial active stroke volume in patients with old myocardial infarction. In this study, actual forward flow due to left atrial active contraction was determined by multiplying left ventricular stroke volume by the fraction of atrial active contraction that was determined from the flow velocity profile with pulsed-wave Doppler echocardiography.

**METHODS**

**Study patients:** A total of 118 patients with an old myocardial infarction were selected from among people who were admitted to the Dokkyo University Hospital between August 1993 and March 1996 (Table I). Patients whose myocardial infarction had occurred at least one month previously were admitted to the study. Patients with acute coronary syndrome, atrial fibrillation, ventricular arrhythmia and regurgitation of the mitral and/or aortic valves of more than a mild degree were excluded. As the normal control group, we evaluated 25 subjects with normal cardiac function and without coronary artery disease.

**M-mode and B-mode echocardiographic examination:** Echocardiographic examinations were conducted immediately before cardiac catheterization. A commercially available echocardiographic system (Toshiba SSH 160A, Tokyo) was used for echocardiographic examination. Patients were in the left lateral decubitus position when recording the echocardiogram. Two-dimensional images were recorded using a 3.75 or 2.5 MHz probe. The M-mode echocardiogram

<table>
<thead>
<tr>
<th>Table I. Clinical Characteristics of 118 Patients with Old Myocardial Infarction and 25 Controls</th>
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<tbody>
<tr>
<td>LVDD (mm)</td>
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<tr>
<td>LVEDP (mmHg)</td>
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<tr>
<td>Number of patients</td>
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<tr>
<td>Infarcted region</td>
</tr>
<tr>
<td>antero-septal</td>
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<tr>
<td>inferior</td>
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<tr>
<td>lateral</td>
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<td>other</td>
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<td>Age (year, mean ± SEM)</td>
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was also recorded with a 3.75 or 2.5 MHz probe on a strip chart at 50 mm/sec. Two-dimensional image, M-mode echocardiogram, electrocardiogram and phonocardiogram were recorded simultaneously.

The left ventricular end-diastolic internal diameter (LVDD) and left ventricular end-systolic internal diameter (LVDS) were determined on either the two-dimensional parasternal long-axis image or M-mode echocardiogram.\(^9\) LVDD was used as the index of left ventricular chamber size. Fractional shortening (%FS), calculated as \(\{(LVDD - LVDS)/LVDD\} \times 100\), was also determined for the systolic parameter. Maximum left atrial anterior-posterior diameter (LAD) was determined. The end-diastolic wall thickness on the infarcted region was also measured on either the two-dimensional parasternal long-axis image or M-mode echocardiogram.

**Doppler echocardiographic examination:** A 2.5 MHz probe was used for pulsed-wave Doppler wave measurements. To record the flow velocity at the left ventricular inflow tract (LVIT), a transducer was placed at the apical window, and the sample volume was positioned at the tip of the mitral valve leaflets\(^8,10,11\) with the cursor line placed parallel to the transmitral flow. Pulsed-wave Doppler velocity tracing, the electrocardiogram and the phonocardiogram were all recorded simultaneously at a paper speed of 50 mm/sec. To record the flow velocity at the left ventricular outflow tract (LVOT), a transducer was also placed at the apical window, and the sample volume was positioned at LVOT 2–3 mm below the aortic valve.

The following parameters from the flow velocity spectrum (average of 5 beats) were determined on the flow velocity profile of the LVIT (Figure 1): 1) the peak velocity of the early diastolic wave (E-wave, cm/sec); 2) the peak velocity of the atrial contraction wave (A-wave, cm/sec); 3) velocity time integral of the E-wave (VTI(E), cm); 4) velocity time integral of the A-wave (VTI(A), cm); 5) deceleration time of the E-wave (msec). In addition, the velocity-time integral of the left ventricular outflow tract (VTI(LVOT)) was determined on the flow velocity profile of the LVOT.

The E-wave to A-wave (E/A) ratio and the ratio of VTI(E) to the VTI(A) (VTI(E)/VTI(A)) were determined. The percentage fraction of VTI(A) to the total of VTI(A) and VTI(E), %A, was also calculated. Left ventricular stroke volume was calculated by multiplying VTI(LVOT) by the cross sectional area of LVOT. This cross-sectional area (cm\(^2\)) of LVOT was obtained by measuring the diameter of the left ventricular outflow tract \(D\) at the level of the sampling point. Calculations were based on the assumption of circular geometry \(3.14*(D/2)^2\).

**Cardiac catheterization:** The left and right heart were catheterized via a femoral approach following the administration of local anesthesia. Cardiac output was measured by the thermodilution method. Intracardiac pressure of the left
Figure 1. Pulsed-wave Doppler velocity tracing at the left ventricular inflow tract and measurements used in this study. E-wave and A-wave = peak velocity of the early diastolic wave and peak velocity of atrial contraction wave, respectively; VTI(E) and VTI(A) = velocity time integral of E-wave and velocity time integral of A-wave, respectively.

Ventricle was measured with a pig-tailed catheter, 7 Fr. (Goodtec P-155BD), that was connected to a pressure transducer (COBE disposable transducer 041–570–504), using the Siemens Cathcor cardiac catheterization analysis system. Zero reference was obtained at the mid-chest level. Left ventricular pressure was recorded on a strip chart at 100 mm/sec. The left ventricular end-diastolic pressure (LVEDP) was determined on this record and used as the index of left ventricular diastolic pressure.

Principle for estimating left atrial active stroke volume: The principle of the estimation of the left atrial active stroke volume is shown in Figure 2. Left atrial active stroke volume refers to the actual forward stroke volume produced by atrial active contraction. In estimating that volume, we assumed that the left ventricular inflow tract area (AREA, cm²) was constant during diastole, and that there was no mitral valve regurgitation.

The inflow volume into the left ventricle during diastole is the same as the outflow volume; that is, the left ventricular stroke volume. This inflow volume is the sum of the inflow volume at early diastolic phase and the inflow volume at
atrial active contraction phase. Blood flow in these two phases is represented as the E-wave and A-wave, respectively, on the flow velocity record at the LVIT on a pulsed-wave Doppler echocardiogram. The inflow volume at each phase is expressed by multiplying the area by VTI(E) or VTI(A), respectively.

Under this assumption, the fraction of inflow volume at the active contraction phase to the total inflow volume during diastole (summation of inflow volume at early diastolic phase and inflow volume at atrial active contraction) is equal to the ratio of VTI(A) to the sum of VTI(E) and VTI(A). The blood flow from the left atrium into the left ventricle by the left atrial active contraction, that is, the left atrial active stroke volume, was determined by multiplying the stroke volume by the ratio of VTI(A) to the VTI(E) + VTI(A). The left atrial active stroke index (LAASI, ml/m²) was determined by dividing the left atrial active stroke volume (ml) by the body surface area (BSA, m²).

**Classification of patients:** The 118 patients with old myocardial infarction were classified into the four groups according to the size of the left ventricular cavity and the left ventricular diastolic pressure; (1) LVDD < 55 mm and
LVEDP < 18 mmHg: Group 1, n = 78; (2) LVDD < 55 mm and LVEDP ≥ 18 mmHg: Group 2, n = 7; (3) LVDD ≥ 55 mm and LVEDP < 18 mmHg: Group 3, n = 24; (4) LVDD ≥ 55 mm and LVEDP ≥ 18 mmHg: Group 4, n = 9 (Table I). There were 25 normal control subjects.

**Statistical methods:** Values are expressed as mean ± standard error of the mean. Comparisons of the indices in each of the five groups of old myocardial infarction were evaluated by one-way analysis of variance (ANOVA). The significance of the difference between the mean values of any groups by ANOVA was analyzed by the Tukey-Kramer test. A level of p < 0.05 was considered statistically significant. Statistical analyses were conducted using the JMP program developed by the SAS Institute (USA).

**RESULTS**

**Clinical characteristics and parameters of hemodynamics, M-mode echocardiogram and Doppler echocardiogram (Table II):** The four groups of patients with OMI and the control group did not differ significantly with respect to age. There was no significant difference in heart rate or LVSP among the four groups of patients with OMI. The mean heart rate and LVSP in the control group did not differ from that observed in the four groups with OMI. The CI in patients with OMI who had a large left ventricular dimension and high LVEDP (group 4) was significantly lower than that in patients with OMI who had a normal left ventricular dimension and normal LVEDP (group 1) and the control group. The CI in the groups with a normal left ventricular dimension (groups 1 & 2) and the group with a large left ventricular dimension and normal LVEDP (group 3) were not decreased, as compared with the control group. The %FS in the four groups with OMI were significantly decreased compared with the control group. The %FS in the groups with a large left ventricular dimension (groups 3 & 4) were also lower than that in the group with a normal left ventricular dimension and normal LVEDP (group 1). There was no significant difference in the %FS between the groups with a large left ventricular dimension (groups 4 & 3). The end-diastolic wall thickness in the infarcted region in patients with OMI who had a large left ventricular dimension and high LVEDP (group 4) was significantly thinner than that in the control group. Deceleration time in this patient in group 4 was also shorter than that in the control group.

**Comparisons of LAASI:** As shown in Figure 3, LAASI in patients with OMI who had a large left ventricular dimension and high LVEDP (group 4) was significantly lower than patients with OMI who had normal LVEDP (groups 1 & 3) and the control group. There was no significant difference in the LAASI between patients with OMI who had high LVEDP (groups 2 & 4). There was
Table II. Hemodynamic, M-mode and Doppler Echocardiographic Parameters in 118 Patients with Old Myocardial Infarction and 25 Controls

<table>
<thead>
<tr>
<th>Parameter</th>
<th>&lt; 55</th>
<th>≥ 55</th>
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<tbody>
<tr>
<td>LVDD (mm)</td>
<td>&lt; 18</td>
<td>≥ 18</td>
</tr>
<tr>
<td>LVEDP [mmHg] number</td>
<td>78</td>
<td>7</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>66.0 ± 1.4</td>
<td>72.3 ± 6.1</td>
</tr>
<tr>
<td>CI (/min/BSA)</td>
<td>2.73 ± 0.07</td>
<td>2.70 ± 0.16</td>
</tr>
<tr>
<td>LAD (mm)</td>
<td>33.8 ± 0.6</td>
<td>32.5 ± 2.4</td>
</tr>
<tr>
<td>LVDD (mm)</td>
<td>48.0 ± 0.5</td>
<td>48.3 ± 1.6</td>
</tr>
<tr>
<td>WTi (mm)</td>
<td>9 ± 0.3</td>
<td>8 ± 0.8</td>
</tr>
<tr>
<td>LVSP (mmHg)</td>
<td>126 ± 2</td>
<td>111 ± 5</td>
</tr>
<tr>
<td>%FS</td>
<td>31.1 ± 1.0</td>
<td>25.1 ± 4.7</td>
</tr>
<tr>
<td>Peak-E (m/sec)</td>
<td>0.60 ± 0.02</td>
<td>0.77 ± 0.07</td>
</tr>
<tr>
<td>Peak-A (m/sec)</td>
<td>0.70 ± 0.02</td>
<td>0.69 ± 0.04</td>
</tr>
<tr>
<td>DT (msec)</td>
<td>170 ± 6</td>
<td>145 ± 10</td>
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</tbody>
</table>

* vs Group 1, ** vs Control
† vs Group 1, Group 2, Control
‡ vs Control
NS = not significant. HR = heart rate; CI = cardiac index; LAD = left atrial diameter between anterior wall and posterior wall; LVDD = left ventricular short axis internal diameter on diastole; WTi = wall thickness on the infarcted region; LVSP = left ventricular systolic peak pressure; %FS = % of fractional shortening; peak-E = peak velocity of early diastolic filling; peak-A = peak velocity of atrial contraction; DT = deceleration time of E-wave.

Figure 3. Comparisons of LAASI in groups with old myocardial infarction and control group. LAASI = left atrial active stroke index.
Figure 4. Comparisons of E/A, VTI(E)/VTI(A) and %A in groups with old myocardial infarction and control group. E/A in Figure 4-A, VTI(E)/VTI(A) in Figure 4-B and %A in Figure 4-C: see "Doppler echocardiographic examination" in the methods.
also no significant difference in this LAASI between the groups with normal left ventricular dimension (groups 1 & 2).

**Comparisons of E/A, VTI(E)/VTI(A) and %A (Figure 4):** The E/A in patients with OMI who had a large left ventricular dimension and high LVEDP (group 4) was significantly lower than the other OMI and control groups. VTI(E)/VTI(A) and %A in group 4 were also significantly lower than patients with OMI who had normal LVEDP (groups 1 & 3) and the control group. However, there were no significant difference in %A between the groups with high LVEDP (groups 2 & 4).

**DISCUSSION**

**Effects of left ventricular chamber size on left atrial booster pump function:** Several studies have reported that following an infarct, there is a progressive dilatation of the left ventricular chamber, which may affect the flow profile at the left ventricular inflow tract. The LAASI in patients with a large left ventricular dimension and high LVEDP (group 4) showed a decrease in the LAASI compared to patients with OMI who had a normal LVEDP (groups 1 and 3 and controls). The LAASI in the groups with a normal left ventricular dimension (groups 1 & 2) remained unchanged, as compared with the control group. There was no significant difference in the LAASI between the groups with high LVEDP (groups 2 & 4). These results indicate that the combination of left ventricular enlargement and increased LVEDP is related to the decrease in forward flow due to left atrial active contraction.

During the course of healing, connective tissue cells enter the myocyte compartment and connect disrupted myocyte fibers. These pathological changes provide the resistance to further stretching of the left ventricular wall. The mechanism responsible for the lowered forward flow by left atrial active contraction in patients with OMI who had a large left ventricular chamber and high LVEDP may be ascribed in part to these pathological changes that may decrease the distensibility of the left ventricular wall. Thus, considering these previous studies, ventricular remodeling after myocardial infarction may be ineffective at increasing left ventricular diastolic compliance in the left atrial active contraction phase.

**Estimation of actual forward stroke volume by atrial active contraction:** Our analysis also showed that E/A, VTI(E)/VTI(A) and %A were decreased in the patients with OMI who had a large left ventricular chamber and high LVEDP (group 4). However, flow velocity patterns determined with pulsed-wave Doppler echocardiography has limitations in the evaluation of left ventricular diastolic properties. This is because factors such as atrial pressure, left ventricular
diastolic pressure, left ventricular compliance, and mitral valve impedance affect the pressure gradient between the left ventricle and the left atrium.\textsuperscript{1,6,8} Kuo and co-workers studied the contribution of atrial contraction to total diastolic filling as observed by Doppler echocardiography from the time-velocity integral of mitral annulus inflow.\textsuperscript{7} They showed that atrial filling fraction varied over a wide range in patients with dilated cardiomyopathy and myocardial infarction. Thus, LAASI, which is quantitatively determined by echocardiographic examination, appears to be a useful index for investigating the pathophysiological relationship between the left atrium and the left ventricle in the left atrial active contraction phase.

Left atrial active stroke volume, which was determined indirectly with echocardiographic examination, has certain limitations for evaluating this parameter. However, this left atrial active stroke volume appears to have clinical potential that deserves further investigation in assessing left ventricular diastolic properties at the left atrial active contraction phase in patients with OMI.

**Study limitations:** While we estimated the forward ejection by the left atrial contraction, this method could not evaluate backward ejection, i.e., the regurgitation from the left atrium to the pulmonary vein. The work of the left atrium should be a sum of the work of the forward flow and the work of the backward flow. Takeuchi and co-authors observed the forward ejection from the left atrium to the left ventricle and the backward ejection from the left atrium to the pulmonary veins in patients with hypertrophic cardiomyopathy, using transesophageal Doppler echocardiography. They showed that an increase in the left atrial afterload may lead to a decrease in left atrial forward ejection and an increase in the left atrial backward ejection.\textsuperscript{21} Our data did not eliminate the effect of the backward flow due to the restriction of left ventricular diastolic filling in the postinfarcted heart.

A heart with myocardial infarction causes a combination of changes in expansion of infarcted region, hypertrophy of the residual non-infarcted myocardium and left ventricular dilatation, which is referred to as ventricular remodeling. We assessed only chamber dilation as the index for these morphological changes. Further investigation is needed to estimate the effects of the other factors on left atrial booster pump function.

**CONCLUSION**

Left atrial booster pump function was assessed by LAASI, which was evaluated by echocardiography. Decreased forward flow by left atrial active contraction was observed in patients with OMI who had left ventricular enlargement and increased left ventricular diastolic pressure. The groups of patients with a
normal chamber size showed no decrease in LAASI, as compared with the control group. The lowered LAASI was attributable in part to the decreased distensibility of the remodeled left ventricle.

ACKNOWLEDGMENT

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