Alterations in the Mitral Flow Velocity Pattern Induced by Acute Myocardial Infarction

— Doppler Findings Before and After Infarction —

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Several studies have demonstrated that myocardial infarction (MI) is likely to alter left ventricular diastolic function. However, it is unclear whether MI per se alters Doppler transmитral flow velocity patterns (TMF) clinically. To investigate how myocardial infarction alters TMF clinically, we assessed serial changes in TMF in 13 patients whose TMF patterns were recorded at a mean of 7 months before and after MI in relation to the size of MI. From TMF, early and atrial filling flow velocities (E and A (m/s), respectively), and the E/A ratio, were measured. From simultaneously recorded two-dimensional echocardiograms, left ventricular dimensions and wall motion score (WMS: sum of 17 segmental scores (dyskinesis=3 to normal=0)) were determined. The patients were divided into two subsets based on the values for WMS in the convalescent stage; 8 patients had small MI (WMS≤10) and 5 patients had large MI (WMS>10). In patients with small MI, although E and the E/A ratio decreased at day-1 compared with pre-MI values (pre-MI vs day-1, E; 0.54±0.12 vs 0.39±0.15, p<0.05, E/A ratio; 0.91±0.23 vs 0.68±0.14, p<0.05), these values increased to levels similar to those observed at pre-MI in the convalescent stage. There were no changes in left ventricular dimensions between pre-MI and in the convalescent period. In patients with large MI, the changes in the TMF patterns varied among patients after MI. In 3 patients with WMS≤25, E and the E/A ratio were decreased in the convalescent stage, compared with pre-MI values. In 2 patients with WMS>25, TMF patterns showed ‘pseudonormalization’ (E/A ratio>1.0) throughout the follow-up period, with a progressive increase in left ventricular dimension. Thus, MI per se does not always produce clear changes in the Doppler TMF pattern in clinical settings, and the size of the MI seems to be a determinant of the TMF pattern after MI.

(Jpn Circ J 1995; 59: 274—283)

The use of Doppler transmитral flow velocity patterns to assess left ventricular diastolic function has been evaluated in several cardiovascular disorders.1−4 Several clinical and experimental studies using Doppler echocardiography have demonstrated that myocardial ischemia and/or infarction introduces left ventricular diastolic filling abnormalities.5−8 Abnormal transmитral flow velocity patterns in patients with myocardial infarction can be explained primarily by the presence of myocardial

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Key words:
Myocardial infarction
Diastolic function
Doppler ultrasound
Reperfusion

(Received May 12, 1994; accepted October 14, 1994)
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fibrosis, which reduces left ventricular distensibility. In addition, possible residual myocardial ischemia may introduce asynchronous and asynergic left ventricular wall motion, which would further impair left ventricular diastolic filling.

In addition to myocardial ischemia and necrosis, several physiologic and pathologic factors, including aging, blood pressure, left ventricular hypertrophy, and loading to the left ventricle should influence Doppler transmural flow velocity patterns, even in patients with myocardial infarction. Since patients with myocardial infarction are usually old and often have multiple risk factors, such as hypertension, diabetes mellitus, and so on, abnormal transmural flow velocity patterns may be attributed to coexistent factors other than myocardial infarction itself. The influence of myocardial infarction on transmural flow velocity patterns can be better understood if serial changes, rather than just a single measurement, are assessed, since many of the aforementioned factors can be neglected when each patient serves as his or her own control.

The purposes of this study were to explore the effect of myocardial infarction per se on the transmural flow velocity pattern and to clarify the relation between the transmural flow velocity pattern and infarct size. In contrast to previous studies, the present study exclusively focused on serial changes in Doppler indices by analyzing patients before and after myocardial infarction, which enabled each patient to serve as his or her own control.

METHODS

This protocol was approved by the Hospital Ethics Committee. Informed consent for Doppler and echocardiographic follow-up studies after infarction was obtained from each patient who participated in this study.

Study Population

From April 1987 to March 1992, 482 patients with their first acute myocardial infarction underwent a two-dimensional echocardiographic and Doppler examinations on the day of infarction in our hospital. From among these patients, we selected 19 consecutive patients who met the following criteria; 1) two-dimensional echocardiographic and Doppler examinations were performed before the infarction in our hospital, and 2) videotape recordings and hard prints were available. The diagnosis of acute myocardial infarction was based on the presence of chest pain for 30 min or longer, characteristic electrocardiographic changes, and a more than 3-fold increase in serum creatine kinase (CK) levels. Six patients were excluded from this study because 1) Doppler examination before infarction was performed during episodes of angina pectoris (3 patients), 2) recurrent ischemic events were observed during a the follow-up period after myocardial infarction (2 patients), or 3) the image quality was inadequate (1 patient). Therefore, this study was conducted in the remaining 13 patients (9 men and 4 women, aged 41 to 72 years, mean 60 years). None of the patients showed atrial fibrillation or significant (≥2+ ) aortic or mitral regurgitation.

The diagnosis before myocardial infarction was stable exertional angina in 11 patients and stable resting angina in 2 patients. Before infarction, diagnostic coronary angiography was performed in 11 of the 13 patients. Significant (>50%) but less than 90% coronary stenosis was observed in one or more major coronary arteries in 10 patients. The diagnosis was anterior wall infarction in 9 patients, posterior wall infarction in 2 patients, the inferior wall infarction in 1 patient, and left main trunk infarction in the remaining patient on the basis of standard electrocardiographic criteria, coronary angiogram, and echocardiographic evaluation of wall motion. Intracoronary thrombolysis or coronary angioplasty was attempted on the day of infarction in 11 patients, and successful coronary reperfusion (residual stenosis of less than 90%) was achieved in 10 of them. In 2 patients (Cases No. 1 and 5: Table I) who did not undergo coronary angiography in the acute stage of myocardial infarction, coronary reflow was observed in the convalescent stage. One patient (Case No. 11: Table I) in whom successful reperfusion was not achieved in the acute stage showed persistent obstruction of the infarct-related artery. Medications during follow-up included nitrates in 13 patients (100%), calcium
<table>
<thead>
<tr>
<th>Case No.</th>
<th>age/sex</th>
<th>Infarct Site</th>
<th>No. of involved vessels</th>
<th>IRA</th>
<th>Tx.</th>
<th>Stenosis</th>
<th>peak CK (IU)</th>
<th>WMS</th>
<th>LVEDP (mmHg)</th>
<th>LVDD (mm)</th>
<th>HR (bpm)</th>
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</thead>
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<tr>
<td>1</td>
<td>69F</td>
<td>A</td>
<td>2</td>
<td>6</td>
<td>Uk iv.</td>
<td>9</td>
<td>*</td>
<td>38</td>
<td>634</td>
<td>0</td>
<td>57</td>
</tr>
<tr>
<td>2</td>
<td>58F</td>
<td>A</td>
<td>3</td>
<td>6</td>
<td>PCTA</td>
<td>38</td>
<td>II (+)</td>
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<td>342</td>
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<td>A</td>
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<td>6</td>
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<td>38</td>
<td>0 (+)</td>
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<td>51</td>
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<td>62</td>
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<tr>
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<td>58M</td>
<td>P</td>
<td>1</td>
<td>3</td>
<td>SVG</td>
<td>Conv. 4</td>
<td>0 (+)</td>
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<td>1091</td>
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<td>6</td>
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<td>805</td>
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<td>1</td>
<td>7</td>
<td>PCTA</td>
<td>62</td>
<td>0 (+)</td>
<td>41</td>
<td>343</td>
<td>0</td>
<td>71</td>
</tr>
<tr>
<td>8</td>
<td>72M</td>
<td>A</td>
<td>1</td>
<td>6</td>
<td>PCTA</td>
<td>30</td>
<td>0 (+)</td>
<td>41</td>
<td>1042</td>
<td>0</td>
<td>85</td>
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<tr>
<td>9</td>
<td>62M</td>
<td>A</td>
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<td>7</td>
<td>PCTA</td>
<td>30</td>
<td>0 (+)</td>
<td>41</td>
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<td>0</td>
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<td>49M</td>
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<td>2</td>
<td>6</td>
<td>PCTA</td>
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<td>0 (+)</td>
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<td>1042</td>
<td>0</td>
<td>85</td>
</tr>
<tr>
<td>11</td>
<td>70F</td>
<td>A</td>
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<td>7</td>
<td>PCTA</td>
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<td>0 (-)</td>
<td>10</td>
<td>1540</td>
<td>0</td>
<td>75</td>
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<td>66F</td>
<td>P</td>
<td>2</td>
<td>11</td>
<td>Conv. 2</td>
<td>III (+)</td>
<td>46</td>
<td>1</td>
<td>1553</td>
<td>1</td>
<td>59</td>
</tr>
<tr>
<td>13</td>
<td>52M</td>
<td>LMT</td>
<td>2</td>
<td>5</td>
<td>PCTA</td>
<td>45</td>
<td>III (+)</td>
<td>40</td>
<td>3500</td>
<td>0</td>
<td>76</td>
</tr>
</tbody>
</table>

Abbreviations: A = anterior wall; Acute = acute period, chronic = chronic period, Conv. = conventional therapy, F = female, HR = heart rate, I = inferior wall, IRA = infract-related artery, LMT = left main trunk, LVDD = left ventricular diastolic/systolic dimension, LVEDP = left ventricular end-diastolic dimension, M = male, P = posterior wall, pre = pre-MI period, PCTA = percutaneous coronary angioplasty, PTCR = percutaneous coronary recanalization, TIMI = thrombolysis in myocardial infarction trial flow grade, SVG = saphenous vein graft, Tx. = therapy, WMS = wall motion score, * = no available data.

Two-dimensional echocardiographic and Doppler examinations were performed 1 to 14 months (mean 7 months) before acute myocardial infarction or in patients who underwent coronary angioplasty and/or percutaneous coronary recanalization and/or in the convalescent stage of myocardial infarction and/or in the convalescent stage of ulcerative angina (23%), patients with acute (25%), and patients with acute myocardial infarction (25%).
videotape. M-mode echocardiograms of the left ventricular septum and posterior wall were recorded immediately below the mitral valve leaflets from the two-dimensional long-axis image, and images were recorded on videotape and as hard copy.

**Cardiac Catheterization**

Coronary angiography and left ventriculography were performed using a brachial approach at a mean of 7 months (range 1 to 18 months) before the onset of infarction in 11 patients. Coronary angiography followed by left ventriculography was performed on the day of infarction in 11 patients using a femoral approach, and was repeated at a mean of 36 days (range 29 to 41 days) after infarction in all of the patients using a brachial approach. Coronary stenosis was evaluated by in terms of % diameter stenosis. In each examination, we measured the left ventricular end-diastolic pressure by carefully positioning a 6-Fr fluid-filled catheter in the mid-left ventricle. In two patients who did not undergo left heart catheterization in the acute stage (Cases No. 1 and 5: Table I), pulmonary capillary wedge pressure was measured instead of left ventricular end-diastolic pressure. In 11 patients, we assessed left ventricular end-diastolic pressure or pulmonary capillary wedge pressure before myocardial infarction, on the day of infarction, and in the convalescent stage.

**Echocardiographic/Doppler Analysis**

Doppler measurements were performed in a random sequence by an observer who was unaware of the patients’ data. Peak early filling velocity (E, m/sec), peak atrial filling velocity (A, m/sec) and the ratio of peak early to atrial filling velocities (E/A ratio) were determined as Doppler indices of left ventricular diastolic filling. The Doppler velocity curve was manually digitized to measure the area under the curve, i.e., the time-velocity integral of the early filling velocity curve (ETVI, m), that of the atrial filling velocity curve (ATVI, m), and the ratio of the early to atrial filling velocity integrals (ETVI/ATVI ratio). All measurements were determined as an average of five consecutive cardiac cycles.

Left ventricular M-mode echocardiographic recordings were used to determine left ventricular end-diastolic and end-systolic dimensions, which were taken as the internal diameter at the onset of the QRS complex and the minimal internal diameter, respectively. Left ventricular wall motion was assessed by dividing the left ventricle into 17 segments (8 segments in the short-axis slice at the levels of the mitral valve and the mid-papillary muscle, and the apical segment in the apical long-axis view). Wall motion in each segment was evaluated by a 4-point scale as follows: 3, akinetic/dyskinetic; 2, severely hypokinetic; 1, hypokinetic; 0, normal. The segmental scores were determined by two independent observers who were unaware of the clinical data and of the results of the other observer. In cases of disagreement, consensus was established by a third observer.

**Reproducibility of Doppler Measurements**

To assess the reproducibility of Doppler measurements, 10 Doppler recordings were randomly selected and Doppler measurements of left ventricular diastolic filling were determined by one observer on two occasions (intraobserver variability). Another observer independently performed the determination for the same 10 recordings (interobserver variability). The observers were unaware of the other’s results. The absolute values of differences between observations were $0.01 \pm 0.04$ m/s and $0.01 \pm 0.03$ m/s (intraobserver and interobserver, respectively) for E, $0.01 \pm 0.02$ m/s and $0.01 \pm 0.02$ m/s for A, $0.06 \pm 0.10$ and $0.05 \pm 0.12$ for the E/A ratio, $0.015 \pm 0.023$ m and $0.016 \pm 0.034$ m for ETVI, $0.015 \pm 0.023$ m and $0.016 \pm 0.024$ m for ATVI, and $0.03 \pm 0.12$ and $0.07 \pm 0.19$ for the ETVI/ATVI ratio.

**Statistical Analysis**

Values are expressed as the mean $\pm$ SD. ANOVA and Scheffe’s test for repeated measures were used to test the significance of the changes after myocardial infarction. Differences were considered significant at a value of $p < 0.05$.

**RESULTS**

The two-dimensional echocardiograms and Doppler echocardiograms were of adequate quality in all examinations in all of the
TABLE III ECHOCARDIOGRAPHIC AND HEMODYNAMIC VALUES IN GROUP 1 PATIENTS

<table>
<thead>
<tr>
<th></th>
<th>pre</th>
<th>acute</th>
<th>chronic</th>
</tr>
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<tbody>
<tr>
<td>WMS</td>
<td>0±1</td>
<td>11±4*</td>
<td>3±3#</td>
</tr>
<tr>
<td>LVDd (mm)</td>
<td>48±3</td>
<td>50±5</td>
<td>49±4</td>
</tr>
<tr>
<td>LVDs (mm)</td>
<td>31±6</td>
<td>35±6</td>
<td>33±5</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>66±9</td>
<td>75±9</td>
<td>71±11</td>
</tr>
<tr>
<td>LVEDP (mmHg)</td>
<td>8±5</td>
<td>13±7*</td>
<td>8±3#</td>
</tr>
</tbody>
</table>

Data were expressed as mean±SD. * = p<0.05 vs pre-MI period, # = p<0.05 vs acute period. Abbreviations are same as Table I.

Patients’ Characteristics

Table I summarizes the patients’ characteristics, coronary angiographic findings, and echocardiographic variables Table II summarizes Doppler variables of transmitral flow in all of the patients. Study patients were classified into two groups based on the wall motion score in the chronic stage. Eight patients with a wall motion score of 10 or less were included Group 1, and the other 5 patients with a relatively large infarct size (wall motion score of greater than 10) were included in Group 2. Peak CK in Group 1 was significantly lower than that in Group 2.

Doppler and Echocardiographic Findings in Group 1

Mean values of left ventricular dimensions and heart rate slightly increased on the day of infarction compared with those before infarction, but these changes were not statistically significant (Table III). In contrast, a significant increase in left ventricular end-diastolic pressure was observed on the day of infarction. These values decreased to levels similar to those observed before infarction in the convalescent stage. The increase in wall motion score on the day of infarction, and its subsequent decrease in the convalescent stage, were both significant.

Fig. 1 illustrates changes in E, A, and the E/A ratio before and after myocardial infarction in Group 1. A significant decrease in E was observed at day-1. Afterwards, E increased to almost the same level as that observed before infarction. In contrast, A
Fig. 1. Serial changes in peak atrial filling flow velocity (A, left panel), peak early filling flow velocity (E, middle panel) and the E/A ratio (right panel) before and after myocardial infarction in Group 1. A did not change during the follow-up period. In contrast, decreases in E and the E/A ratio were observed on the day of the infarction. These variables subsequently increased to levels similar to those observed before infarction. Open circles and bars show mean values and standard deviation, respectively. Abbreviations: E = early diastolic filling flow, A = atrial filling flow, E/A = ratio of peak early to atrial filling flow velocities, Pre = before the onset of myocardial infarction, Acute = day of infarction, Chronic = during the convalescent period. * = p < 0.05

**TABLE IV TIME VELOCITY INTEGRAL VALUES IN GROUP 1 PATIENTS**

<table>
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<th></th>
<th>pre</th>
<th>acute</th>
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<td>ETVI (m)</td>
<td>0.080 ± 0.014</td>
<td>0.055 ± 0.022*</td>
<td>0.07 ± 0.012#</td>
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<tr>
<td>ATVI (m)</td>
<td>0.059 ± 0.014</td>
<td>0.052 ± 0.015*</td>
<td>0.051 ± 0.010*</td>
</tr>
<tr>
<td>ETVI/ATVI</td>
<td>1.4 ± 0.33</td>
<td>1.0 ± 0.24*</td>
<td>1.4 ± 0.25#</td>
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</tbody>
</table>

* = p < 0.05 vs Pre-MI period; # = p < 0.05 vs Acute period. Abbreviations are same as Table I and Table II.

**TABLE V ECHOCARDIOGRAPHIC AND HEMODYNAMIC VALUES IN GROUP 2 PATIENTS**

<table>
<thead>
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<th></th>
<th>Pre ± SD</th>
<th>Acute ± SD</th>
<th>Chronic ± SD</th>
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</thead>
<tbody>
<tr>
<td>WMS</td>
<td>1 ± 2</td>
<td>20 ± 10*</td>
<td>22 ± 8*</td>
</tr>
<tr>
<td>LVDd (mm)</td>
<td>51 ± 1</td>
<td>54 ± 5</td>
<td>63 ± 8*</td>
</tr>
<tr>
<td>LVDs (mm)</td>
<td>34 ± 3</td>
<td>43 ± 6</td>
<td>51 ± 9*</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>74 ± 12</td>
<td>89 ± 15</td>
<td>81 ± 15</td>
</tr>
<tr>
<td>LVEDP (mmHg) (3 cases)</td>
<td>9 ± 4</td>
<td>24 ± 12</td>
<td>21 ± 3</td>
</tr>
</tbody>
</table>

Data were expressed as mean ± SD. * = p < 0.05 vs pre-MI period, # = p < 0.05 vs acute period. Abbreviations are as Table I.

did not show any significant changes during the study period. Consequently, the E/A ratio significantly decreased at day-1, compared with that observed before infarction, and then increased to a level similar to that observed before infarction. Although ATVI decreased after the onset of myocardial infarction, changes similar to those in E and E/A were found in ETVI and the ETVI/ATVI ratio (Table IV). ETVI and the ETVI/ATVI ratio decreased at day-1 of infarction, and then increased to values similar to those found before infarction.

**Doppler and Echocardiographic Findings in Group 2**

Left ventricular dimensions tended to increase on the day of infarction, and increased further during the convalescent period (Table V). Heart rate and left ventricular end-diastolic pressure (3 patients) increased on the day of infarction, but these values decreased slightly in the convalescent stage. A significant increase in the wall motion score was observed on the day of infarction, and there was no subsequent improvement in the convalescent stage.

*Japanese Circulation Journal Vol. 59, May 1995*
The patients in Group 2 showed various changes in the transmirtal flow velocity pattern. In the 2 cases (Cases No.12 and 13: Table I) with the greatest extent of residual asynery (WMS in the convalescent stage of 25 or greater), the E/A ratio was greater than 1 in the convalescent stage of infarction, and remained greater than 1 throughout the chronic convalescent stage, indicating a 'pseudonormalized' pattern (Fig. 2). The other 3 patients with WMS of less than 25 (Cases No. 9, 10, and 11: Table I) showed different patterns. In these patients, the E/A ratio in the convalescent stage tended to be lower than the pre-infarction value (Fig. 3). The 2 former patients exhibited greater wall motion scores in the convalescent stage than the 3 latter patients (mean values, 30 vs 18). The changes in ETVI, ATVI and the ETVI/ATVI ratio were similar to those observed in E, A and the E/A ratio (Table II).

DISCUSSION
Small Myocardial Infarction and the Trans-

mitral Flow Velocity Pattern
In patients with relatively small infarction (WMS of 10 or less), increases in left ventricular dimensions and left ventricular end-diastolic pressure were observed along with impairment of regional wall motion on the day of the infarction. The changes in Doppler indices of left ventricular diastolic function were characterized by decreases in E (ETVI) and the E/A ratio (ETVI/ATVI ratio) compared with the values before infarction. These findings indicate that decreases in left ventricular chamber compliance, and the impairment of left ventricular relaxation caused by acute myocardial infarction, may reduce the transmirtal pressure gradient in the early filling phase, and then disturb filling due to left ventricular suction\textsuperscript{11,13}

In the convalescent stage, regional wall motion showed significant improvement, partially because all of the patients achieved coronary reperfusion. Regarding Doppler variables, ETVI and the ETVI/ATVI ratio increased relative to the values on the day of the infarction. These changes in Doppler
variables reflect an improvement in diastolic filling. In addition, although slight decreases in E and the E/A ratio were measured in the convalescent stage, there were no significant differences between these values pre-infarction and those of in the convalescent stage. These findings indicate that myocardial infarction might not always impair left ventricular diastolic function in the convalescent stage if the infarction is small.

The exact mechanism for the preservation of left ventricular diastolic function after myocardial infarction is unknown. Our data showed that there were no differences between the left ventricular dimensions or end-diastolic pressure before infarction and those in the convalescent stage, indicating that preload (end-diastolic dimension and pressure) and contractility (end-systolic dimension) are not altered, despite the presence of asynergy. Only reserve of left ventricular diastolic function can explain these findings.

Large Myocardial Infarction and the Transmirtal Flow Velocity Pattern

Different results were obtained in patients with large infarction (WMS of greater than 10). In addition, the changes in the transmirtal flow velocity pattern varied among the patients in this group. In patients with moderately large infarction (WMS of greater than 10 but less than or equal to 25, Cases No. 9, 10 and 11: Table I), decreases in E and the E/A ratio were observed in the convalescent stage, compared with the pre-infarction values, indicating that left ventricular diastolic function is impaired due to acute myocardial infarction. Transmirtal flow velocity did not recover to the pattern observed before myocardial infarction, and E and the E/A ratio in the convalescent stage were lower than the respective pre-infarction values. These findings indicate that the impairment of left ventricular diastolic function is not compensated, even in the convalescent stage with moderate-sized myocardial infarction.

On the other hand, transmirtal flow velocity reflected an increase in E and the E/A ratio, and a decrease in A throughout the follow-up period after infarction in the 2 patients who showed a large infarction (WMS of greater than 25). In these patients, the E/A ratio was greater than 1.0, and exhibited an apparently normal waveform regardless of the presumed severely impaired left ventricular function. These patients showed high left ventricular end-diastolic pressure after infarction, so that this pattern was considered “pseudonormalization”. In general, an impairment of diastolic filling causes a decrease in E and an increase in the atrial contribution to filling, which results in an E/A ratio of less than 1.14.15 In patients with congestive heart failure, however, the transmirtal flow velocity pattern is apparently “normal”, with an E/A ratio of greater than 1, and left ventricular diastolic dysfunction may be “masked”. This “pseudonormalized” pattern is a product of “restrictive” left ventricular chamber properties and increased left atrial pressure.16.17 Therefore, our data indicate that the “pseudonormalized” transmirtal flow velocity pattern, especially during the convalescent stage, suggests a large myocardial infarction, which makes the left ventricular chamber “stiff”. As mentioned above, the transmirtal flow velocity pattern develops an abnormal relaxation or “pseudonormalized” pattern only in patients with a relatively large myocardial infarction. Heart rate, left ventricular remodeling, and an increase in filling pressure may also contribute to these changes. In addition, the population examined in this study was so small that the further study is required to precisely determine the effect of the size of the myocardial infarction.

Diastolic Function and Transmirtal Flow Velocity

Various Doppler indices, including E and the E/A ratio, are used to evaluate left ventricular diastolic function in patients with coronary artery disease.6 cardiomyopathy3.18 and hypertension19 Several investigators20 have noted a significant correlation between peak early filling velocity and pressure-derived variables of left ventricular relaxation and early filling fraction. Time-velocity integrals or areas under the velocity curve have also been used in several reports, since they are also correlated with fractional filling as measured by radionuclide angiography.21.22 In this study, we preferred E and the E/A ratio to ETVI and the ETVI/ATVI ratio as estimates of left ventricular diastolic function because they are
much simpler to obtain. Our results indicated that Doppler indices derived from peak velocities and those derived from time-velocity integrals show similar changes after myocardial infarction.

**Limitations**

The most important limitation of this study was the complexity of the diastolic physiology and the inability to control or characterize each factor that could affect it. When interpreting the transmural flow velocity pattern, we should consider several factors, including heart rate, left ventricular relaxation and stiffness, and filling pressure. Since this study was performed retrospectively, we could not assess these parameters in all of the patients, especially before infarction.

Doppler transmural flow velocity patterns before myocardial infarction may have been already been affected by pre-existing coronary stenosis, since coronary stenosis affects the Doppler transmural flow velocity pattern through of chronic ischemia, which results in the asynchronized relaxation of the left ventricle. However, the impairment of left ventricular relaxation largely depends on the degree of coronary artery stenosis. In our patients who underwent coronary angiography before myocardial infarction, the percent diameter stenosis of the infarct-related artery was less than 75%, and the stenosis of the other coronary artery was less than 90% in each patient. In addition, none of the patients showed any signs of myocardial ischemia during echocardiographic examination before myocardial infarction. Only 3 patients showed mild asynergy and none of these asynergies were in this infarcted region. Therefore, coronary stenosis may have only a trivial effect on the pre-infarction transmural flow velocity pattern.

**Implications**

Our data indicate that myocardial infarction per se does not always impair left ventricular diastolic function as assessed by Doppler variables. In patients with relatively small infarction, the transmural velocity pattern was similar to that observed before infarction, without any changes in left ventricular dimensions or filling pressure. In cases with a larger infarct size, the transmural flow velocity showed a pattern different from its own control, but the changes seemed to depend on the size of the infarction. These findings should be taken into consideration when evaluating diastolic function using Doppler variables derived from the mitral flow velocity pattern in patients with myocardial infarction.

**Acknowledgment**

We wish to acknowledge the skillful technical assistance of Yuzo Sakagami and Masakazu Ueda, and the excellent secretarial assistance of Miss Rie Nishizawa.

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