Impaired Right Ventricular Filling in Old Myocardial Infarction

Hong-tai Bai, M.D., Kazuhiro Fujitani, M.D., and Hisashi Fukuzaki, M.D.

Summary

To evaluate ventricular filling and interactions between right and left ventricles in patients with old myocardial infarction, right and left ventricular time-volume curves were analyzed from a cineangiographic study of 10 normal subjects (Group 1), 10 patients with old anterior myocardial infarction (Group 2) and 10 patients with old inferior myocardial infarction (Group 3). Volumes of both ventricles were calculated from each frame over an entire cardiac cycle using Simpson's method. From time-volume curves, peak ejection rates, peak filling rates and atrial kick rates were obtained for both ventricles and these parameters were normalized by end-diastolic volume. All patients were in sinus rhythm with heart rates less than 80 beats/min. There were no significant differences among the 3 groups in end-diastolic pressure of both ventricles and mean pulmonary artery pressure. Left ventricular ejection fractions were significantly lower in Groups 2 and 3 than in Group 1 (p<0.001, p<0.005, respectively), although there were no significant differences in end-diastolic volume indexes of either ventricle among the 3 groups. Peak left ventricular ejection rate and peak filling rates of the left and right ventricles were lower in Group 2 than in Group 1 (p<0.01, p<0.05, p<0.01, respectively) and peak filling rate of the right ventricle in Group 2 correlated with the peak filling rate of the left ventricle and left ventricular ejection fraction (r=0.64, r=0.64, respectively). Peak filling rate of the right ventricle in Group 2 correlated inversely with left ventricular peak negative dp/dt (r=−0.72), but no correlation was found between peak filling rate of the right ventricle and left ventricular end-diastolic volume index or mean pulmonary artery pressure. Peak ejection rate of the left ventricle and peak filling rates of both ventricles in Group 3 were lower than in Group 1 (p<0.02, p<0.02, p<0.01, respectively) and no correlation was found between peak filling rates of both ventricles. Wall motion of the right ventricular septal portion was slightly reduced in 5 patients in Group 2. In all patients in Group 3, right ventricular wall motion centering around the right ventricular diaphragmatic portion was re-
duced. These results suggest that in old inferior myocardial infarction, right ventricular wall motion abnormality results in impaired right ventricular filling, whereas in old anterior myocardial infarction, right ventricular filling is reduced indirectly due to impaired left ventricular filling.

Additional Indexing Words:
Ventricular interaction Peak filling rate Right ventricle Filling curve

PARAMETERS of left ventricular diastolic filling such as peak filling rate and filling fractions are useful indicators of diastolic left ventricular function and are frequently reduced in patients with coronary artery disease.1-5) Takeuchi et al previously reported that the impairment of left ventricular filling in patients with coronary artery disease was closely related to the abnormal regional outward wall motion of the left ventricle.5) However, ventricular filling of the right ventricle has not as yet been assessed in patients with old myocardial infarction. Further, the left and right ventricles are anatomically contiguous, and thus mechanically coupled within the pericardium.6,7) Although the interactions between both ventricles have been extensively documented in a variety of experimental animal preparations and clinical investigations,13-27) little work has been done with regard to the effects of the interaction between both ventricles on ventricular filling.

Therefore, to evaluate ventricular filling and interactions between both ventricles in patients with old myocardial infarction, we analyzed time-volume curves of both ventricles from cineangiographic studies.

MATERIALS AND METHODS

Patient populations:
Thirty patients who underwent cardiac catheterization were studied. They were classified as follows:

Group 1 consisted of 10 subjects who served as controls; Group 2 consisted of 10 patients with old anterior myocardial infarction; Group 3 consisted of 10 patients with old inferior myocardial infarction. Each of the patients in Group 1 who complained of atypical chest pain had normal clinical findings, a normal left ventriculogram, coronary arteriogram and normal values for hemodynamic variables at rest and during the supine ergometer exercise test or the ergonovine maleate test. All patients in this study were in sinus rhythm with heart rates of not less than 50 beats/min and not over 80 beats/min; patients with bundle branch block, hypertension and valvular heart disease were excluded from the study. Cineangiographic studies were
Table I. Summary of Clinical and Coronary Angiographic Findings

<table>
<thead>
<tr>
<th></th>
<th>Group 1 (Normal)</th>
<th>Group 2 (Anterior OMI)</th>
<th>Group 3 (Inferior OMI)</th>
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<tbody>
<tr>
<td>Patients (no.)</td>
<td>10</td>
<td>10</td>
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<tr>
<td>Age (years)</td>
<td>47±8</td>
<td>56±8</td>
<td>54±7</td>
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<tr>
<td>Sex</td>
<td>7M, 3F</td>
<td>8M, 2F</td>
<td>9M, 1F</td>
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<tr>
<td>CAD (no. of patients)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1V</td>
<td>0</td>
<td>8</td>
<td>3</td>
</tr>
<tr>
<td>2V</td>
<td>0</td>
<td>2</td>
<td>5</td>
</tr>
<tr>
<td>3V</td>
<td>0</td>
<td>0</td>
<td>2</td>
</tr>
</tbody>
</table>

Abbreviations: OMI = old myocardial infarction; M = male; F = female; CAD = coronary artery disease; V = coronary vessel with luminal stenosis > 50%. Values represent mean ± SD.

performed at least 72 hrs after cessation of treatment with β-blockers or calcium antagonists and at least 12 hrs after cessation of nitroglycerin. Informed consent was obtained from each patient, and no unfavorable complication occurred as a result of the study. The details of the 3 groups are listed in Table I.

Study protocol:
Cardiac catheterization was performed via the femoral approach on patients in the fasting state without sedation. Both left and right ventricular pressures were recorded using a high fidelity micromanometer-tipped catheter (Millar Instruments). The transducers were immersed in a 37°C water bath for 30 min and balanced and calibrated electronically against a mercury manometer in the bath immediately before insertion and after withdrawal of the catheter. Initially, left ventriculography was performed by single-plane 35 mm cineangiography at 60 frames/sec in the right anterior oblique (RAO 30°) projection (Philips Poly-Diagnost C). A bolus of 35 ml of iopamid sodium maglumine (Conraxin-H) was injected at a rate of 12 ml/sec through an 8F pigtail catheter into the left ventricle, with cinefilm exposed at 60 frames/sec. During the cineventriculographic study, high fidelity left ventricular pressure and peak negative dp/dt were recorded during breath-holding at mid-inspiration and calculated by a computer (Philips ACS). Left ventricular pressure during ventriculography and the time of the peak of the R wave on the electrocardiogram were also sampled synchronously with frame exposure and displayed in digital form on the corresponding cinefilm (Cine data, Philips). Thirty minutes after the left ventriculography, to allow for dissipation of the hemodynamic and myocardial depressant effects of the contrast agent, right ventriculography with simultaneous measurements of right ventricular pressure was performed in a similar fashion with biplane 35 mm
cineangiography in the right and left anterior oblique (RAO 30°, LAO 60°) projections. Calibration of each ventriculography was performed using a grid at midchest level. No patient had severe regurgitation of either the mitral or tricuspid valve. Ectopic and postectopic beats were excluded from this study. Coronary angiography was carried out using Jadkins' technique. Significant stenosis was defined as a reduction of more than 50% in luminal diameter at the site of maximal luminal narrowing relative to an apparently normal adjacent arterial segment.

![Graph showing ventricular volume and dV/dt](image)

**Fig. 1.** Typical curves of smoothed ventricular volume and dV/dt calculated from cineangiogram in a normal subject. Lower column shows method of calculating peak ejection rate, peak filling rate and atrial kick rate.

\[
\text{peak ejection rate (PER)} = \frac{\text{peak negative } dV/dt}{EDV}
\]

\[
\text{peak filling rate (PFR)} = \frac{\text{peak positive } dV/dt}{EDV}
\]

\[
\text{atrial kick rate (AKR)} = \frac{\text{positive } dV/dt}{EDV}
\]
Measurements and computations:

Ventricular volumes were measured by Simpson's method. Each ventriculogram was observed and analyzed frame by frame throughout one cardiac cycle within the first three cardiac cycles following commencement of injection. The time-volume curve and its differentiated curve (time-dV/dt curve) for each right and left ventricle were calculated and constructed using a personal computer (SANYO MBC-220) (Fig. 1). A three point moving average filter was applied. End-diastole was defined as the point on the pressure trace at which the derivative of the pressure first exceeds 200 mmHg/sec in the left ventricle and 100 mmHg/sec in the right ventricle, \( ^5, ^{31} \) and all cases coincided with the maximal measured ventricular volume. From the time-dV/dt curve, peak negative dV/dt, peak positive dV/dt and positive dV/dt coinciding with atrial kick were obtained. Each parameter was normalized by end-diastolic volume for each ventricle and was termed peak ejection rate, peak filling rate and atrial kick rate, respectively.

Asynergy of the left ventricle was assessed by percent change of sector area which was obtained as follows (Fig. 2). Each end-diastolic and end-systolic left ventricular silhouette of the right oblique projection was divided into 5 sector areas according to the American Heart Association reporting system \( ^8 \) and evaluated according to the percent change of each sector area. Reduced wall motion was defined as being present when the percent change of each sector area was lower by more than 2 standard deviations than the mean value of a normally contracting left ventricle.

The assessment of right ventricular asynergy was performed so that each end-diastolic and end-systolic silhouette of the right and left anterior oblique

![Fig. 2. Regional wall contraction of the normally contracting left ventricle. The diagram illustrates the percent change of every sector area (mean ±SD) obtained from 14 normal subjects.](image-url)
Fig. 3. Regional wall contraction of the normally contracting right ventricle. The upper panel shows the method of calculating the percent change of 6 sector areas from biplane right cineventriculogram. The lower diagram illustrates the percent change of every sector area (mean ± SD) obtained from 12 normal subjects.

Projections was divided into 3 sector areas and the evaluation based on the percent change of each sector area, i.e., area 1 is the right ventricular antero-septal portion, area 2 is the right ventricular diaphragmatic portion, area 3 is the right ventricular inflow portion, area 4 is the right ventricular septal portion, area 5 is the right ventricular diaphragmatic portion, and area 6 is the right ventricular free wall portion (Fig. 3). Reduced wall motion was considered to be present when the percent change of each sector area was lower by more than 2 standard deviations than the mean value of a normally contracting right ventricle.

Statistical analysis:
Values are expressed as mean ± standard deviation. Differences be-
between groups of patients were analyzed by the unpaired Student's t-test. Linear regression analysis was performed to compare right ventricular peak filling rate and other hemodynamic and volumetric parameters. The significance of differences in the incidence of regional wall motion abnormality and the incidence of regional wall motion abnormality in total regional segmental areas between Groups 2 and 3 were determined by the Chi-square test using Yate's correction if the sample sizes were small. A p value < 0.05 was considered significant.

RESULTS

Comparisons of hemodynamic parameters (Table II):

There were no significant differences in left ventricular end-diastolic pressure and right ventricular end-diastolic pressure among the 3 groups. Furthermore, there were no significant differences in both left and right ventricular systolic pressures, mean pulmonary artery pressure and cardiac index among the 3 groups.

Comparisons of volume measurements (Table III):

There was no significant difference in left ventricular end-diastolic volume index among the 3 groups. Left ventricular end-systolic volume index was $45 \pm 22$ ml/m² in Group 2 and $40 \pm 18$ ml/m² in Group 3, and left ventricular end-systolic volume index was significantly higher in Group 2 than in Group 1 ($p < 0.05$). Left ventricular ejection fraction was $58 \pm 10\%$ in Group 2 and $61 \pm 12\%$ in Group 3, and was significantly lower in these 2 groups than in Group 1 ($p < 0.001$, $p < 0.005$). There were no significant differences in these parameters for the right ventricle among the 3 groups.

| Abbreviations: LVSP = left ventricular systolic pressure; LVEDP = left ventricular end-diastolic pressure; RVSP = right ventricular systolic pressure; RVEDP = right ventricular end-diastolic pressure; PAm = mean pulmonary artery pressure; RAm = mean right atrial pressure; CI = cardiac index. |
| Values represent mean ± SD. |

<table>
<thead>
<tr>
<th></th>
<th>LVSP (mmHg)</th>
<th>LVEDP (mmHg)</th>
<th>RVSP (mmHg)</th>
<th>RVEDP (mmHg)</th>
<th>PAm (mmHg)</th>
<th>RAm (mmHg)</th>
<th>CI (1/min/m²)</th>
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<tbody>
<tr>
<td>Group 1</td>
<td>131±17</td>
<td>15±3</td>
<td>25±3</td>
<td>7±2</td>
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<td>4±2</td>
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<td>Group 2</td>
<td>132±22</td>
<td>17±4</td>
<td>25±4</td>
<td>8±2</td>
<td>15±2</td>
<td>5±2</td>
<td>3.25±0.51</td>
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<tr>
<td>Group 3</td>
<td>134±22</td>
<td>18±6</td>
<td>27±6</td>
<td>8±2</td>
<td>15±1</td>
<td>3±2</td>
<td>3.15±0.66</td>
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Table III. Summary of Volume Measurements

<table>
<thead>
<tr>
<th></th>
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<th>RV</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>EDVI (ml/m²)</td>
<td>ESVI (ml/m²)</td>
</tr>
<tr>
<td>Group 1</td>
<td>103±29</td>
<td>27±13</td>
</tr>
<tr>
<td>Group 2</td>
<td>105±30</td>
<td>45±22*</td>
</tr>
<tr>
<td>Group 3</td>
<td>99±30</td>
<td>40±18</td>
</tr>
</tbody>
</table>

Abbreviations: LV = left ventricle; RV = right ventricle; EDVI = end-diastolic volume index; ESVI = end-systolic volume index; EF = ejection fraction.
Values represent mean±SD.
Group 1 vs Group 2 or Group 3: *p<0.05, †p<0.005, ***p<0.001.

Table IV. Summary of Parameters Derived from Time-Volume Curve

<table>
<thead>
<tr>
<th></th>
<th>LV</th>
<th>RV</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>PER (l/sec)</td>
<td>PFR (l/sec)</td>
</tr>
<tr>
<td>Group 1</td>
<td>3.43±0.54</td>
<td>3.82±0.63</td>
</tr>
<tr>
<td>Group 2</td>
<td>2.74±0.48**</td>
<td>3.01±0.88*</td>
</tr>
<tr>
<td>Group 3</td>
<td>2.79±0.57†</td>
<td>2.85±0.96†</td>
</tr>
</tbody>
</table>

Abbreviations: PER = peak ejection rate; PFR = peak filling rate; AKR = atrial kick rate. Other abbreviations are the same as in Table III. Values represent mean±SD.
Group 1 vs Group 2 or Group 3: *p<0.05, †p<0.02, **p<0.01.

Comparisons of parameters derived from time-volume curve (Table IV):
Peak ejection rate of the left ventricle was 2.74±0.48 l/sec in Group 2 and 2.79±0.57 l/sec in Group 3, and was significantly lower in these 2 groups than in Group 1 (p<0.01, p<0.02). Peak filling rate of the left ventricle was 3.01±0.88 l/sec in Group 2 and 2.85±0.96 l/sec in Group 3 and this parameter was significantly lower in these 2 groups than in Group 1 (p<0.05, p<0.02). There was no significant difference in atrial kick rate of the left ventricle among the 3 groups. Peak filling rate of the right ventricle was 2.17±0.41 l/sec in Group 2 and 2.18±0.41 l/sec in Group 3, and was significantly lower in these 2 groups than in Group 1 (p<0.01, p<0.01), although there were no significant differences in peak ejection rate and atrial kick rate for the right ventricle among the 3 groups.

Relationships of peak filling rate of the right ventricle to left ventricular hemodynamic parameters (Figs. 4 and 5):
In Group 2, peak filling rate of the right ventricle correlated moderately with peak filling rate of the left ventricle, left ventricular ejection fraction and
Fig. 4. Relationships of peak filling rate of the right ventricle (RVPFR) to peak filling rate of the left ventricle (LVPFR), left ventricular ejection fraction (LVEF) and left ventricular peak negative dp/dt (LV peak -dp/dt) in patients with old anterior myocardial infarction (Group 2) and old inferior myocardial infarction (Group 3).

Fig. 5. Relationships of peak filling rate of the right ventricle (RVPFR) to left ventricular end-diastolic volume index (LVEDVI) and mean pulmonary artery pressure (PA mean pressure) in patients with old anterior myocardial infarction (Group 2) and old inferior myocardial infarction (Group 3).
left ventricular peak negative dp/dt ($r = 0.64$, $r = 0.64$, $r = -0.72$), although in Group 3 neither peak filling rate of the left ventricle, left ventricular ejection fraction nor left ventricular peak negative dp/dt correlated with peak filling rate of the right ventricle. The relationships of peak filling rate of the right ventricle to left ventricular end-diastolic volume index and mean pulmonary artery pressure in both Group 2 and Group 3 were not significant.

**Distribution of regional wall motion abnormality in the left ventricle** (Fig. 6):
In Group 2 the abnormal wall motion was mainly observed in area 2 and/or area 3, and in Group 3 abnormal wall motion was mainly observed in area 3 and/or area 4.

**Distribution of regional wall motion abnormality in the right ventricle** (Fig. 7):
In 5 cases in Group 2 right ventricular wall motion centering around the right ventricular septal portion was reduced, i.e., area 1 was reduced in 1 case, area 4 was reduced in 2 cases and areas 4 and 6 were reduced in 1 case. In all cases in Group 3 right ventricular wall motion was reduced in areas 2, 3, 4, 5 and/or 6 (right ventricular diaphragmatic portion, right ventricular inflow portion, right ventricular septal portion, right ventricular diaphragmatic portion and right ventricular free wall portion). The incidence of cases with right ventricular wall motion abnormality and the incidence of right ventricular regional wall motion abnormality in total regional segmental areas (6 segmental areas × 10 cases in Group 2, 6 segmental areas × 10 cases in Group 3) were significantly different between Groups 2 and 3 ($p < 0.05$, $p < 0.01$, respectively).
DISCUSSION

Filling pattern of the left ventricle in old myocardial infarction:

Abnormalities of the diastolic properties of the left ventricle in patients with coronary artery disease have been reported in numerous studies. However, to our knowledge there are little data that have been obtained with a heart rate of less than 80 beats/min. All patients in the present study were in sinus rhythm and heart rates were not over 80 beats/min. Although the ventricular filling pattern is characterized by a biphasic pattern, when early diastolic filling is separated from the late atrial contribution by a slow filling, ventricular filling shows a monophasic pattern when the heart rate is over 80 beats/min. One can detect the effect of atrial contraction on diastolic ventricular volume in most patients with sinus rhythm and rates less than 80 beats/min.

In our present study the peak filling rate of the left ventricle was significantly lower in both Group 2 and Group 3 than in Group 1, and in the former groups reduced left ventricular ejection fraction and left ventricular regional wall motion abnormalities were found. There have been several experimental reports of diminished compliance and impaired relaxation of the left ventricle in myocardial infarction. It is thus suggested that such impaired left ventricular filling in old myocardial infarction might result from the development of myocardial fibrosis, with alterations in the distensibility characteristics of the left ventricle.
Filling pattern of the right ventricle in old myocardial infarction:

Peak filling rates of the right ventricle obtained by time-volume curves were reduced in both Group 2 and Group 3, suggesting that ventricular filling of the right ventricle was impaired in old myocardial infarction.

Clinicopathological correlations of left and right ventricular infarction have been reported. Isner et al demonstrated that right ventricular infarction associated with left ventricular infarction was identified by gross examination at necropsy in 33 (14%) of 236 patients with transmural myocardial infarction. Further, all cases with associated right ventricular infarction were in those patients with posterior left ventricular infarction accompanied by transmural infarction of the posterior ventricular septum. From our study, all cases in Group 3 were observed to have reduced wall motion of the right ventricle centering around the right ventricular diaphragmatic portion. Therefore, in Group 3 reduced wall motion of the right ventricle, suggesting that right ventricular infarction might be associated, would contribute to the impaired right ventricular filling, although hemodynamic parameters in the systolic phase were within normal limits.

The biventricular dynamics during the acute stage of anteroseptal and anterolateral infarction in the porcine heart were compared, and it was demonstrated that the right ventricular changes in anteroseptal infarction were related to direct involvement of the right ventricular free wall and septum rather than secondary to left ventricular alterations. However, we suggest that in Group 2 reduced wall motion centering around the right ventricular septal portion was not the only mechanism of impaired right ventricular filling, because normal wall motion of the right ventricle was found in 5 cases in Group 2. We also demonstrated that in Group 2 peak filling rate of the right ventricle correlated moderately with that of the left ventricle, although in Group 3 no correlation was observed between peak filling rate of the right ventricle and that of the left ventricle. From this point of view, we suggest another mechanism whereby in old anterior myocardial infarction, right ventricular filling is reduced indirectly, probably due to impaired left ventricular filling, i.e., due to ventricular interaction.

The left ventricle and the right ventricle are anatomically contiguous, forming a continuous muscular “syncitium” or syncitium-like structure within the pericardium. Further, both right and left ventricles are connected through the pulmonary circulation. Therefore, the mechanism of ventricular interaction is roughly divided into two mechanisms, i.e., the series circuit through the pulmonary circulation, and the parallel circuit through the interventricular septum and myocardial muscle fiber attachments and the effect of the pericardium.
The effects of afterload on right ventricular performance have been studied by several investigators.\textsuperscript{15\textendash}17\textsuperscript{}) It is known that exercise-induced left ventricular dysfunction in patients with coronary artery disease is the cause of the elevation of pulmonary artery pressure.\textsuperscript{18\textendash} It was demonstrated experimentally that the series ventricular interaction during acute imbalances in biventricular loading, where the output of the right ventricle determines the input of the left, appeared to be more important in determining left ventricular systolic function.\textsuperscript{19\textendash} In the present study, peak filling rate of the right ventricle in Group 2 did not correlate with mean pulmonary artery pressure, although peak filling rate of the right ventricle correlated moderately with peak filling rate of the left ventricle. We suggest from these results that the impairment of right ventricular filling may not be regulated by the impaired left ventricular filling through the pulmonary circulation.

The parallel circuit through the interventricular septum, myocardial muscle fiber attachment and the effect of the pericardium is another important determinant accounting for ventricular interaction. This interaction exists between the ventricles during diastole and systole, and increased distension of either ventricle during filling has been shown to decrease the distensibility of the opposite ventricle, as well as contributing to alterations in ventricular geometry.\textsuperscript{13\textendash}14\textendash}19\textendash}23\textendash) Bemis et al,\textsuperscript{20\textendash} in an experimental study showed changes in left ventricular geometry with a decrease in septum-to-left ventricular free wall distance in isolated, supported canine hearts during increases in right ventricular diastolic filling. There was an increase in left ventricular end-diastolic pressure with increased right ventricular pressures as hemodynamic correlates with the morphologic changes. Furthermore, most investigators agree that ventricular diastolic interaction is much stronger when the pericardium remains intact.\textsuperscript{20\textendash}22\textendash}24\textendash) However, LeWinter et al\textsuperscript{25\textendash} demonstrated that the pericardium stretches in response to chronic cardiac dilatation. The effect of the pericardium may be less significant in chronic overload than in acute overload.

In our study, end-diastolic volume indices of both ventricles in Group 2 were not enlarged and were not significantly different compared with Group 1, and in Group 2 no correlation was observed between peak filling rate of the right ventricle and left ventricular end-diastolic volume index. Further, peak filling rate of the right ventricle in Group 2 correlated with peak filling rate, ejection fraction and peak negative dp/dt of the left ventricle. From these results we speculate that the impaired right ventricular filling in Group 2 is closely related to left ventricular function and this impaired right ventricular filling may be mediated not by distension of the pericardium, but by the parallel circuit through the interventricular septum and/or myocardial muscle
fiber attachment, although in our study it was found that 5 cases in Group 2 had reduced wall motion centering around the right ventricular septal portion.

**Limitation of this study:**

We used Simpson’s method for the calculation of right ventricular volume. This method has proven to be quite accurate when direct right ventricular volume measurements at autopsy were compared to the volumes derived from corresponding biplane right ventricular cineangiograms taken prior to the death of these patients.

The assessment of right ventricular wall motion is difficult and its method has not been established, because the pattern of contraction of the right ventricle is more complex than that of the left ventricle. Ferlinz et al performed an assessment of right ventricular wall motion in patients with old myocardial infarction using biplane right cineventriculogram to analyze seven segmental axes of shortening in each end-systolic and end-diastolic frame. In the left anterior oblique position, the contraction of the right ventricle is mainly composed of the augmentation of the curvature of both right ventricular free wall and septum, and thus precise evaluation such as regional filling is difficult. Accordingly, in this study each end-diastolic and end-systolic silhouette of the right ventricle in the left anterior oblique projection was roughly divided into 3 sector areas (right ventricular septal portion, right ventricular diaphragmatic portion and right ventricular free wall portion) and the evaluation was based on percent change of each sector area.

In conclusion, right ventricular filling was impaired in patients with old myocardial infarction. Further, the impaired ventricular filling of the right ventricle was correlated with that of the left ventricle, not in inferior, but in anterior old myocardial infarction. In old anterior myocardial infarction, the impaired ventricular filling of the left ventricle would induce that of the right ventricle through the interventricular septum, which is the parallel circuit of ventricular interaction. In old inferior myocardial infarction, reduced wall motion of the right ventricle, suggesting that right ventricular infarction might be associated, would play a role in the impaired ventricular filling of the right ventricle.

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