RIGHT VENTRICULAR WALL MOTION DISTURBANCE AND DETERMINANTS OF THE APPEARANCE OF HEMODYNAMIC RIGHT VENTRICULAR INFARCTION

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In order to elucidate the mechanisms of the appearance of hemodynamic right ventricular infarction (RVI), we studied right and left ventriculograms and hemodynamic findings in 52 patients with acute inferior myocardial infarction. Right ventricular wall motion disturbance (RVWMD) was detected in 69% of patients but hemodynamic RVI was observed only in 16%. Among patients with RVWMD, there was no significant difference in right ventricular ejection fraction between those with (group III) and without (group II) hemodynamic RVI, suggesting that right ventricular (RV) systolic dysfunction does not independently produce hemodynamic RVI. Right ventricular end-diastolic volume index was similar in groups II and III in spite of higher mRA in group III. The result suggested that the RV compliance of group III was decreased. Heart rate (HR) was significantly lower in group III than in group II. Not only physiologic pacing but also VVI pacing significantly improved hemodynamics in patients with hemodynamic RVI. A positive correlation between HR and cardiac index was observed (r=0.56, p<0.001) in patients with RVWMD. Decreased RV compliance and bradycardia were considered to be determinants of the appearance of hemodynamic RVI. Volume loading did not improve hemodynamics significantly in patients with hemodynamic RVI.

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USING radionuclide ventriculography or echocardiography, right ventricular wall motion disturbance (RVWMD) was detected in as many as 37–60% of patients with acute inferior myocardial infarction.1–4 However, RVWMD was not always associated with severe hemodynamic impairment of right ventricular infarction (RVI), i.e.,

**Key words:**
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Depressed right ventricular compliance
Bradyarrhythmia
Digital subtraction angiography

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Fig. 1. An example of right and left ventriculograms using digital subtraction angiography in a patient with inferior acute myocardial infarction (AMI) and normal right ventricular (RV) contraction in the 30° right anterior oblique (RAO) projection. Hypokinesis in the inferobasal wall of the left ventricle (LV) was recognized.

Fig. 2. An example of right and left ventriculograms of the same patient as in Fig. 1 acquired in the 50° left anterior oblique (LAO) projection.

the appearance of hemodynamic RVI besides decreased right ventricular ejection fraction. However, the mechanisms of hemodynamic disturbance of RVI are still unclear.

The goal of this study is to elucidate the determining factors of the appearance of right ventricular hemodynamic disturbance in patients with RVWMID. For this purpose, we studied right and left ventriculograms and hemodynamic findings of the same patients with acute inferior myocardial infarction.

METHODS

Patients and study protocol

Subjects were 52 patients with acute inferior myocardial infarction with ST elevation in leads II, III and aVF (35 males and 17 females, average age 65.4 ± 11.2 years) who admitted to Saiseikai Kumamoto Hospital, Kumamoto, between January 1987 and December 1988. After informed consent was obtained, all patients received right and left ventriculography using a digital subtraction angiography (DSA) system on admission, for evaluating wall motion and volume of the right and left ventricles. Four patients with severe pulmonary congestion were excluded from the study because we did not perform ventriculography on admission. After ventriculography, the 52 patient's hemodynamic data were monitored in coronary care unit.

Digital subtraction angiography

Cine mode of right and left ventriculography was performed on admission using DSA system (MODEL DFA-2, HITACHI). The DSA system and radiographic technique were as described previously. The 2 ventriculograms were acquired in the 30° right anterior oblique and the 50° left anterior oblique projections. Regional RVWMID was observed in the inferior and apical wall of the right ventricle (RV). We graded the regional wall motion of RV into 5 categories: normal; mild hypokinesis; severe hypokinesis; akinesis; dyskinesis. Angiographic criteria of RVI were the presence of RVWMID equal to or greater than severe hypokinesis (Figs. 3 and 4). The end-diastolic and end-systolic volume (EDV and ESV) of the RV were calculated using the biplane pyramid method, and those of the left ventricle were calculated using the standard single
plane method\(^{10}\). End-diastolic volume index (EVDI) and ejection fraction (EF) of both ventricles were calculated using the following formulas:

\[
\text{EDVI (ml/m}^2\text{)} = \frac{\text{EDV/BSA}}{100 \times (\text{EDV - ESV})/\text{EDV}}.
\]

\[\text{CI (l/min/m}^2\text{)} = \frac{\text{CO/BSA}}{\text{HR}},\]

\[\text{SVI (ml/beat/m}^2\text{)} = 1000 \times \frac{\text{CI}}{\text{HR}},\]

\[\text{RVSWI (g/m/m}^2\text{)} = \text{SVI} \times (\text{mPA} - \text{mRA}) \times 0.0136\],

\[\text{LVSWI (g/m/m}^2\text{)} = \text{SVI} \times (\text{mBP} - \text{dPA}) \times 0.0136\],

\[\text{TSR (dynes-sec-cm}^{-5}\text{)} = \frac{80 \times (\text{mBP} - \text{mRA})/\text{CO}}{\text{PAR (dynes-sec-cm}^{-5}\text{)} = \frac{80 \times (\text{mPA} - \text{dPA})/\text{CO}}{\text{BSA} = \text{body surface area, mBP}}\]

\text{= mean blood pressure, mRA = mean right atrial pressure, mPA = mean pulmonary arterial pressure, dPA = diastolic pulmonary arterial pressure.}\\

As in a previous study\(^{2}\) mRA of 10 mmHg or greater and a mRA/dPA ratio of 0.8 or greater were chosen for the hemodynamic criteria of RVI.

**Statistical Analysis of Data**

All the data are presented as mean±SD. Analysis of variance and the least-significant difference method or paired t test were used to analyze the differences. In all statistical analyses, p<0.05\(^{11}\) was considered significant.
TABLE I PATIENTS' DATA

<table>
<thead>
<tr>
<th></th>
<th>group I</th>
<th>group II</th>
<th>group III</th>
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<tbody>
<tr>
<td>n</td>
<td>16</td>
<td>28</td>
<td>8</td>
</tr>
<tr>
<td>age</td>
<td>68±19</td>
<td>70±19</td>
<td>67±14</td>
</tr>
<tr>
<td>Male/Female</td>
<td>10/6</td>
<td>20/8</td>
<td>3/5</td>
</tr>
<tr>
<td>CAG (n)</td>
<td>13</td>
<td>25</td>
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Infarct related coronary region

<table>
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<tr>
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<tbody>
<tr>
<td>RCA (proximal to RV)</td>
<td>2-4</td>
<td>8-19</td>
<td>5-7</td>
</tr>
<tr>
<td>(proximal to AM)</td>
<td>2</td>
<td>11</td>
<td>2</td>
</tr>
<tr>
<td>(distal to AM)</td>
<td>7</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>LCX</td>
<td>2</td>
<td>2</td>
<td>0</td>
</tr>
</tbody>
</table>

no. of diseased vessels | 1.7±0.8 | 1.6±0.6 | 2.3±0.5 |

Success of CR (n) (%)

<table>
<thead>
<tr>
<th></th>
<th>7</th>
<th>14</th>
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<tbody>
<tr>
<td>(%)</td>
<td>43.8</td>
<td>50.0</td>
<td>25.0</td>
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</table>

Death before discharge (n) (%)

<table>
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<tr>
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<th>2</th>
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<tbody>
<tr>
<td>(%)</td>
<td>12.5</td>
<td>7.1</td>
<td>25.0</td>
</tr>
</tbody>
</table>

CAG = coronary angiography, RCA = right coronary artery, RV = right ventricular branch, AM = acute marginal branch, LCX = left circumflex coronary artery, no. = number, CR = coronary reperfusion. Asterisks indicate significant differences (*: p<0.05).

TABLE II ANGIOGRAPHIC FINDINGS IN PATIENTS OF 3 GROUPS

<table>
<thead>
<tr>
<th></th>
<th>RVEDVI (ml/m)</th>
<th>RVEF (%)</th>
<th>LVEDVI (ml/m)</th>
<th>LVEF (%)</th>
<th>HR (beats/min)</th>
<th>mBP (mmHg)</th>
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<tbody>
<tr>
<td>group I</td>
<td>n=16</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>66.2±10.5</td>
<td>60.3±6.4</td>
<td>70.0±14.2</td>
<td>58.9±12.2</td>
<td>68±19</td>
<td>92±18</td>
</tr>
<tr>
<td>group II</td>
<td>n=28</td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td></td>
<td>69.8±14.5</td>
<td>48.5±6.7</td>
<td>70.7±19.1</td>
<td>53.1±8.9</td>
<td>70±19</td>
<td>91±18</td>
</tr>
<tr>
<td>group III</td>
<td>n=8</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>70.3±11.9</td>
<td>49.2±3.1</td>
<td>62.7±5.7</td>
<td>59.7±7.9</td>
<td>61±14</td>
<td>93±22</td>
</tr>
</tbody>
</table>

RVEDVI = right ventricular end-diastolic volume index, RVEF = right ventricular ejection fraction, LVEDVI = left ventricular end-diastolic volume index, LVEF = left ventricular ejection fraction, HR = heart rate, mBP = mean blood pressure. Asterisks indicate significant differences compared to group I (**: p<0.001)

RESULTS

(1) Incidences of RVWMD and hemodynamic disturbance of RVI

Examples of right and left ventriculograms using digital subtraction angiography are presented in Fig. 1-4. In Figs. 1 and 2, RV contractions were normal in the right and left anterior oblique projections. In Figs. 3 and 4, akinesis of the inferior and apical RV walls was clearly detected. Bulging of the RV into the left ventricle was also observed in the left anterior oblique projection.

In 52 patients with inferior myocardial infarction, 56 patients (69%) had RVWMD (angiographic RVI). However, hemodynamically diagnosed RVI (hemodynamic RVI) was only seen in 8 patients (16%) within 24 h after admission. According to the angiographic and hemodynamic findings, we divided the 52 patients into 3 groups: patients with normal right ventricular wall motion and with out hemodynamic RVI (group I, 16 patients); those with angio-
<table>
<thead>
<tr>
<th></th>
<th>mBP (mmHg)</th>
<th>mPA (mmHg)</th>
<th>dPA (mmHg)</th>
<th>mRA (mmHg)</th>
<th>HR (beats/min)</th>
<th>CI (l/min/m²)</th>
<th>SVI (ml/beat/m²)</th>
<th>RVSWI (g·m⁻²)</th>
<th>LVSWI (g·m⁻²)</th>
<th>TSR (dynes·sec·cm⁻⁵)</th>
<th>PAR (dynes·sec·cm⁻⁵)</th>
</tr>
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<tbody>
<tr>
<td><strong>group I</strong></td>
<td>92 ± 16</td>
<td>14 ± 5</td>
<td>9 ± 3</td>
<td>4 ± 3</td>
<td>77 ± 15</td>
<td>2.9 ± 0.7</td>
<td>39 ± 9</td>
<td>5.7 ± 3.4</td>
<td>43 ± 12</td>
<td>1705 ± 546</td>
<td>84 ± 75</td>
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<td>n=27</td>
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<tr>
<td><strong>group II</strong></td>
<td>86 ± 15</td>
<td>15 ± 6</td>
<td>10 ± 5</td>
<td>5 ± 3</td>
<td>79 ± 15</td>
<td>2.6 ± 0.7</td>
<td>35 ± 8</td>
<td>4.4 ± 3.1</td>
<td>36 ± 12</td>
<td>1761 ± 666</td>
<td>105 ± 69</td>
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<td>n=62</td>
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<tr>
<td><strong>group III</strong></td>
<td>80 ± 19</td>
<td>16 ± 4</td>
<td>11 ± 3</td>
<td>12 ± 2</td>
<td>66 ± 12</td>
<td>1.6 ± 0.4</td>
<td>25 ± 6</td>
<td>1.3 ± 1.0</td>
<td>25 ± 11</td>
<td>2338 ± 451</td>
<td>187 ± 83</td>
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<td>n=22</td>
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</table>

mBP=mean blood pressure, mPA=mean pulmonary arterial pressure, dPA=diastolic pulmonary arterial pressure, mRA=mean right atrial pressure, HR=heart rate, CI=cardiac index, SVI=stroke volume index, RVSWI=right ventricular stroke work index, LVSWI=left ventricular stroke work index, TSR=total systemic resistance, PAR=pulmonary arterial resistance.

* indicates significant differences (*: p<0.05, **: p<0.001).
RVEF revealed no significant differences between groups II and III. Left ventricular (LV) EDVI in group III was smaller than in groups I and II, but not significantly. LVEF of the 3 groups were similar. HR and mBP during angiography were not significantly different between the 3 groups.

(3) Hemodynamic data of each group

The hemodynamic data of each group obtained within 24 h after angiography are presented in Table III.

None of mBP, mPA and dPA were significantly different between the 3 groups. MRA in group III was significantly higher than in groups I and II (p < 0.001), as a matter of course. HR in group III was significantly lower than in groups I and II (p < 0.05). CI and SVI in group III were significantly lower than in group I and II, respectively (p < 0.001). Those in group II were also lower than in group I (p < 0.05).

(4) Effects of ventricular filling pressure on CI and SVI

In patients with RVWMD (group II+III), CI (y) was plotted as a function of mRA (x). The correlation was significant \( r = -0.38 \), \( p < 0.001 \), \( y = -0.07x + 2.86 \), Fig. 5). Similarly, the relation between mRA (x) and SVI (y) was significant \( r = -0.27 \), \( p < 0.001 \), \( y = -0.62x + 35.72 \). However, the correlation between dPA and CI and that between dPA and SVI were not significant.

In patients with angiographic normal right ventricles, (group I), CI (y) was plotted as a function of dPA (x). The correlation was significant \( r = -0.42 \), \( p < 0.05 \), \( y = 0.09x + 2.16 \), Fig. 5). Similarly the relation between dPA (x) and SVI (y) was significant \( r = 0.38 \), \( p < 0.05 \), \( y = 1.02x + 28.53 \). However, the correlation between mRA and CI and that between mRA and SVI were not significant.

(5) Volume loading in patients with hemodynamic RVI

Volume loading of 800 ml on average was performed in 5 patients (6 trials) in group III. The data before and after therapy are summarized in Table IV. Hemodynamic variables including HR, mRA, dPA, mPA and mBP did not change significantly after volume loading. CI increased in 2 trials and decreased in 4 but did not change significantly. Severe pulmonary congestion appeared in 2 patients after volume loading whereas dPA and mRA did not increase significantly.

(6) Relationship between HR and CI, and the effects of pacing on hemodynamics

CI (y) was plotted as a function of HR (x). In patients with RVWMD the correlation was significant \( r = 0.56 \), \( p < 0.001 \), \( y = 0.02x + 0.51 \), Fig. 6). However, in patients without RVWMD the relation was not significant.

We analyzed the effects of VVI pacing and physiologic (AAI or DDD) pacing on hemodynamic variables in patients with hemodynamic RVI (Table IV). The increased HR by VVI pacing elevated mBP and CI slightly (p < 0.05 and p < 0.01). The physiologic pacing increased mBP and CI considerably (p < 0.01).

DISCUSSION

The elucidate the differences between angiographic and hemodynamic RVI and the mechanisms of the appearance of hemodynamic RVI, we analyzed right and left ventriculograms by a DSA system and the hemodynamic findings in 52 patients with acute inferior myocardial infarction.

Discrepancy between the incidences of angiographic and hemodynamic RVI

Several studies have been reported regarding the diagnosis of RVWMD!−4 In the
studies, RVWMD was detected using radionuclide angiography or echocardiography. The criteria for RVI were the presence of a dilated RV, a RV regional wall motion abnormality, and/or a depressed RVEF. In this study, however, we estimated RV contractions using DSA. The RV volume was calculated by the pyramidal method using the angiograms of right and left anterior oblique projections. As shown in Fig. 1-4, RV and LV wall motion, and RVWMD was clearly recognized in both ventriculograms. The RVWMD was localized in the inferior and apical region of RV, and the wall motion of RV outflow tract was not suppressed. In patients with RVWMD, the occlusion of proximal RCA was frequent. It was considered that the interruption of blood flow of right ventricular branch and acute marginal branch was related to the appearance of RVWMD. Angiographically, RVI was considered to occur in the presence of RVWMD equal to or greater than severe hypokinesis (angiographic RVI). The incidence of angiographic RVI was 69%, higher than in the previous studies! The RVEF of patients with normal RV contraction was 60%, similar to that is previous reports. However, that of patients with RVWMD was 49%, higher than in previous studies!

We chose both mRA of 10 mmHg or greater and a mRA/dPA ratio of 0.8 or greater for the hemodynamic criteria of RVI (hemodynamic RVI). As in the previous studies, the difference between the incidences of angiographic and hemodynamic RVI was recognized in this study. It was considered that the differences were due not only to the difference between the sensitivities of the methods, but to the fact that the methods diagnosed different conditions as RVI, i.e., reduced RV contraction and disturbed hemodynamics.

The role of reduced RVEF in the appearance of hemodynamic RVI

Among patients with reduced RVEF, no significant difference in RVEF was detected between group II without hemodynamic RVI and group III with hemodynamic RVI. Nevertheless, mRA was higher and CI was lower in group III than in group II. Previous studies reported that RV hemodynamic disturbance was not observed in all patients with RVWMD. Experimental RVI did

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not simply lead to hemodynamic disturbance in dogs. The fact that no significant difference in RVEF existed between groups II and III in this study means that patients were seen with and without hemodynamic RVI in spite of reduced RVEF of the same degree. This result suggests that reduced RVEF does not produce hemodynamic RVI independently; additional factors other than RVWMD must be necessary for the appearance of hemodynamic RVI. RVWMD was considered to be a necessary condition of hemodynamic RVI, because all patients with RV hemodynamic disturbance had it.

Mechanisms of low cardiac output in patients with hemodynamic RVI

From the echocardiographic findings and the pattern of right atrial pressure waves, enhanced atrial contraction was reported in some patients with RVI. The lack of enhanced contraction or atrio-ventricular (AV) synchrony led to worsening of the disturbed hemodynamics, suggesting that atrial contraction with AV synchrony was important in hemodynamic compensation in patients with RVI. In this study, bradycardia was considered to be another factor determining the appearance of hemodynamic RVI, because of the lower HR in group III than in group II. Both physiologic pacing and VVI pacing improved hemodynamics in patients with hemodynamic RVI. The correlation between HR and CI was significant in patients with RVWMD. These findings suggest that an HR-dependent compensation was important in patients with RVWMD, as was enhanced atrial contraction with AV synchrony. It is desirable that physiologic pacing should be performed with higher HR to increase the CO in patients with RVI.

In experimental RVI, it was presented that the increased intrapericardial pressure disturbed ventricular filling in dogs? Because the hemodynamic findings in patients with RVI resemble those in patients with constrictive pericarditis, restrictive cardiomyopathy or cardiac tamponade, the increase in intrapericardial pressure of patients with hemodynamic RVI was suggested. In this study, there was no significant difference in RVEDVI between groups II and III, in spite of the extremely higher mRA in group III. This suggests that the RV compliance of group III decreased. Probably the non-compliant pericardium prevented RV from enlarging or dilating acutely over a critical volume level, and CO was maintained low in spite of high filling pressure.

Volume loading was reported in an experimental study to improve the hemodynamics of RVI and in others not to do so. The procedure were also reported to increase the incidence of hemodynamic disturbance of RVI. In our data, transfusion therapy did not change hemodynamics in patients with hemodynamic RVI. Negative correlation coefficients between mRA and CI and between mRA and SVI in patients with RVWMD also suggested that volume loading was not so suitable for RVI. When the intrapericardial pressure is low, volume loading is considered to increase RVEDV and lead to an increase in CO. However, when the intrapericardial pressure is elevated to levels that produce cardiac tamponade, we considered that volume loading can not increase RVEDV. Under these conditions, the increase in CO by the procedure can not be expected. Moreover, we found that severe pulmonary congestion occurred after volume loading therapy in some patients. When volume loading is performed in patients with RVI, one should be careful to maintain the optimal RA pressure and not to produce pulmonary congestion by too much transfusion.

As another factor determining the appearance of hemodynamic RVI, the contribution of LV dysfunction was considered. It was reported that elevated intrapericardial pressure changes LV geometry, decreases LV volume, and produces a low CO condition. In this study, we find no significant difference in LVEDVI between groups II and III although the higher dPA might obscure the smaller LVEDV in group III. But, as shown in Fig. 4, the bulging of the RV into the LV was clearly observed in some patients. It was noted that disturbance of LV dilation by elevated intrapericardial pressure is a mechanism of the appearance of hemodynamic RV.

Conclusion

In patients with inferior infarction and reduced RVEF, there was no significant difference in RVEF between patients with and without hemodynamic RVI. RVEDVI was also similar between the 2 groups in spite of
higher mRA of patients with hemodynamic RVI, suggesting that RV compliance was reduced in patients with hemodynamic RVI. Significantly lower HR was observed in patients with hemodynamic RVI. Reduced RV compliance and bradycardia were considered to be determinants of hemodynamic RVI. The bulging of the RV into the LV was observed in some patients. It was noted that reduced LVEDV was a cause of the appearance of hemodynamic RVI. We would concluded that hemodynamic RVI appears when several compensatory mechanisms to reduce RVEF and pericardial constriction fail to maintain hemodynamics.

REFERENCES


