RIGHT VENTRICULAR FUNCTION IN PATIENTS WITH MITRAL STENOSIS

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Measurements of the right ventricular volume were performed using biplane cineangiocardiography on 17 patients with mitral stenosis, ranging in age from 34 to 62. The right ventricular end-diastolic volume index ranged from 69 to 130 ml/m² (99 ± 18, mean ± SDM), and the right ventricular ejection fraction ranged from 0.33 to 0.69 (0.51 ± 0.08). There was an inverse relationship between the right ventricular end-diastolic volume index and the right ventricular ejection fraction (r = -0.52, p < 0.05). The cardiac index measured by the dye-dilution method ranged from 1.61 to 4.40 L/min/m² (2.56 ± 0.72) and correlated with the right ventricular ejection fraction (r = 0.78, p < 0.001). The right ventricular end-diastolic volume was larger in 11 patients with tricuspid regurgitation than those without (p < 0.05). It was larger in 11 patients with atrial fibrillation than those with sinus rhythm (p < 0.05).

The right ventricular ejection fraction was lower in 8 patients with low cardiac output (0.47 ± 0.08) than those with normal cardiac output (0.47 ± 0.08 vs 0.55 ± 0.07, p < 0.05). It was lower in 11 patients with atrial fibrillation than those with a sinus rhythm (0.48 ± 0.07 vs 0.57 ± 0.09, p < 0.05). The right ventricular ejection fraction was also lower in 7 patients with re-stenosis of the mitral valve following a commissurotomy than those without a previous operation (0.46 ± 0.08 vs 0.55 ± 0.07, p < 0.02).

The left ventricular ejection fraction was lower in 8 patients with low cardiac output than those with a normal one (0.49 ± 0.09 vs 0.56 ± 0.04, p < 0.05).

These results indicate that the right ventricular function is abnormally depressed in patients with mitral stenosis associated with low cardiac output.

MITRAL stenosis induces an afterload on the right ventricle, and the right ventricle, therefore, must eject the same amount of blood against the increased afterload. Although the left ventricular function in mitral stenosis has been reported to be reduced,1-2 little is known of the right ventricular function in this disease. Wroblewski et al. have reported that the right ventricle maintains a normal size and a normal ejection fraction in mitral stenosis with moderate pulmonary hypertension.3 Winzelberg et al. have reported that the right ventricular ejection fraction was below normal in mitral stenosis with right ventricular hypertension.4 As the right ventricular dysfunction and tricuspid regurgitation, which occurred secondary to mitral stenosis, may reduce cardiac output, it is important to assess

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<th>RAm (mmHg)</th>
<th>LA-LVEDP (mmHg)</th>
<th>RVEDVI (ml/m²)</th>
<th>RVEF</th>
<th>LVEDVI (ml/m²)</th>
<th>LVVF</th>
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Abbreviations: Pre.op = previous operation for mitral stenosis; TR = tricuspid regurgitation; CI = cardiac index (L/min/m²); PAP = pulmonary artery pressure (mean); RAm = right atrial mean pressure; LA-LVEDP = left atrial to left ventricular end-diastolic pressure gradient; RVEDVI = right ventricular end-diastolic volume index; RVEF = right ventricular ejection fraction; LVEDVI = left ventricular end-diastolic volume index; LVVF = left ventricular ejection fraction; PVR = pulmonary vascular resistance; HR = heart rate; F = female; M = male; NSR = normal sinus rhythm; AF = atrial fibrillation.
TABLE II  LEFT VENTRICULAR VOLUME, PULMONARY VASCULAR RESISTANCE
AND LEFT ATRIAL TO LEFT VENTRICULAR END-DIASTOLIC PRESSURE GRADIENT

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<th>CI &gt; 2.50</th>
<th>CI &lt; 2.50</th>
<th>p</th>
<th>pre.op (−)</th>
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<td>LVEDVI (ml/m²)</td>
<td>93 ± 12</td>
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<td>ns</td>
<td>91 ± 14</td>
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<td>LVEF</td>
<td>0.56 ± 0.04</td>
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<td>PVR (dynes·sec·cm⁻⁵)</td>
<td>361 ± 235</td>
<td>431 ± 406</td>
<td>ns</td>
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<td>L.A.-LVEDP (mmHg)</td>
<td>11 ± 8</td>
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<td>10 ± 7</td>
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Data is expressed as mean ± SD.

Abbreviations: CI = cardiac index (L/min/m²); p = probability; pre.op = previous operation for mitral stenosis; LVEDVI = left ventricular end-diastolic volume index; LVEF = left ventricular ejection fraction; PVR = pulmonary vascular resistance; LA-LVEDP = left atrial to left ventricular end-diastolic pressure gradient; ns = not significant

Fig. 1. Relationship between the right ventricular ejection fraction (RVEF) and the right ventricular end-diastolic volume index (RVEDVI). There was an inverse relationship between them (r = −0.52, p < 0.05).

![Graph](image)

Fig. 2. Relationship between the cardiac index (CI) and the right ventricular ejection fraction (RVEF). They correlated closely (r = 0.78, p < 0.001).

![Graph](image)

the right ventricular function in this disease. The purpose of the present study is to assess the characteristics of mitral stenosis with reference to the right ventricular volume and function.

MATERIALS AND METHODS

Seventeen patients with mitral stenosis admitted to Osaka University Hospital were studied. Eight patients had a functional capacity of class II according to the New York Heart Association and 9 were in class III. None of the patients had clinical evidence of right ventricular failure (edema, hepatomegaly, ascites and right ventricular third sound). There were 5 men and 12 women, ranging in age from 34 to 62 (44 ± 8, mean ± SD). Patients with significant coronary arterial disease were excluded. No patient had had right or left ventricular infarction. All patients were divided into 2 groups: Group 1 consisted of 9 patients with a cardiac index of over 2.50 L/min/m² and Group 2 of 8 patients with a cardiac index of less than that. There were 5 patients with trivial mitral regurgitation and 4 others with trivial aortic regurgitation. There were 7 patients with recurrent mitral stenosis, who had undergone closed mitral commissurotomy, but had not had surgical treatment of the tricuspid valve, and the period from the operation to the present study in these 7 patients ranged from 11 to 25 years (18 ± 6). Ten patients

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had not undergone mitral valve surgery. In the 7 patients who underwent surgery had slight mitral regurgitation. The average age of these 7 was 42, and that in the other 10 who did not undergo surgery was 45. Eleven patients had an atrial fibrillation and 6 sinus rhythm at the time of study.

Left and right heart catheterization data
including right and left ventricular volume was obtained from all 17 patients. Pressures were recorded prior to cineangiography using an NIH catheter connected to a P23Db Statham transducer with a zero reference at the mid-chest level. Left atrial pressure was directly obtained using the transseptal procedure (Brockenbrough method) in all patients. Cardiac output was determined by the dye-dilution method in all patients.

Right and left ventriculograms were obtained by biplane cineangiography at 60 exposures/sec. Contrast medium, 0.8 ml/kg, was injected into the left and the right ventricle.
respectively. The left ventricular volume was calculated from the films obtained at a right anterior oblique projection, using the following regression equation: \( V_t = 0.89 \, V_c + 9.0 \) (ml)\(^2\). Right ventricular volume was calculated from the biplane films obtained at anterior-posterior and lateral views, using the following regression equation: \( V_t = 0.76 \, V_c - 0.2 \) (ml)\(^2\), where \( V_t \) and \( V_c \) are the true and the calculated ventricular volume, respectively. Volume analyses for the right and left ventricles were based on the area-length method. Simultaneous recordings of an electrocardiogram were made during the ventriculographic studies. All ectopic and post-ectopic beats were excluded from the analysis.

In patients with atrial fibrillation, right and left ventricular end-diastolic and end-systolic volumes were obtained from multiple beats with a preceding R to R interval of more than 400 msec\(^7\) and were averaged, respectively. Respective volumes ranging from 90 to 110% of the averages were used as the data in each patient with atrial fibrillation. Eleven patients had tricuspid regurgitation of different degrees. No patient had tricuspid stenosis.

RESULTS

All data is listed in Table I. There was a significant inverse relationship between the right ventricular end-diastolic volume index and the right ventricular ejection fraction \((r = -0.52, p < 0.05)\) (Fig. 1). Cardiac index correlated well with the right ventricular ejection fraction \((r = 0.78, p < 0.001)\) (Fig. 2).

The right ventricular end-diastolic volume indices were 96 ± 21 ml/m\(^2\) in Group 1 and 103 ± 13 in Group 2. The right ventricular ejection fractions were 0.55 ± 0.07 in Group 1 and 0.47 ± 0.08 in Group 2, and this difference was statistically significant \((p < 0.05)\) (Fig. 3).

The cardiac index was 2.11 ± 0.43 L/min/m\(^2\) in recurrent patients and 2.88 ± 0.72 in patients with no previous operation \((p < 0.05)\). The right ventricular end-diastolic volume indices in these 2 patient groups were 104 ± 17 and 96 ± 18 ml/m\(^2\), respectively, and the right ventricular ejection fractions were 0.46 ± 0.08 and 0.55 ± 0.07, respectively \((p < 0.02)\) (Fig. 4).

The cardiac index was 2.81 ± 0.95 L/min/m\(^2\) in 6 patients with sinus rhythm and 2.42 ± 0.56 in 11 with atrial fibrillation (Fig. 5). The right ventricular end-diastolic volume index was 87 ± 13 ml/m\(^2\) in sinus rhythm and 106 ± 16 in atrial fibrillation \((p < 0.05)\); the right ventricular ejection fractions were 0.57 ± 0.09 and 0.48 ± 0.07, respectively \((p < 0.05)\) (Fig. 5).

There was no significant difference in the cardiac index between 11 patients with tricuspid regurgitation and 6 with no incompetence. The right ventricular end-diastolic volume index was 108 ± 14 ml/m\(^2\) in patients with tricuspid regurgitation and 85 ± 13 in those with no incompetence \((p < 0.05)\); the right ventricular ejection fractions were 0.49 ± 0.10 and 0.56 ± 0.04, respectively (Fig. 6).

The left ventricular end-diastolic volume index was 93 ± 12 ml/m\(^2\) in Group 1 and 89 ± 13 in Group 2. The left ventricular ejection fractions were 0.56 ± 0.04 in Group 1 and 0.49 ± 0.09 in Group 2 \((p < 0.05)\); the left ventricular end-diastolic volume indices were 91 ± 14 ml/m\(^2\) in patients with a previous surgery and 91 ± 11 in those with no surgery. The left ventricular ejection fraction was 0.55 ± 0.04 in patients with no previous surgery and 0.49 ± 0.09 in those with recurrence after their surgery (ns) (Table II).

DISCUSSION

The right ventricular end-diastolic volume increases in patients with tricuspid regurgitation and in those with atrial fibrillation. Eight of 11 patients with atrial fibrillation were associated with tricuspid regurgitation. The loss of atrial contraction, as observed in atrial fibrillation, reduces ventricular end-diastolic pressure and volume, ultimately causing an impairment of myocardial performance\(^8\). Tricuspid regurgitation is probably a major factor to increase right ventricular volume. There was an inverse relationship between the right ventricular end-diastolic volume and the right ventricular ejection fraction. The right ventricular ejection fraction in patients with tricuspid regurgitation was the same as that in those with no incompetence. Therefore, tricuspid regurgitation of a high degree suggests a depression of right ventricular function. The right ventricular ejection fraction was low in patients with low cardiac output and correlated well with the cardiac index, although many patients had tricuspid regurgitation of different degrees. This indicates that forward flow is maintained normally in patients with normal right ventricular pump function. In patients with an increased volume of the right ventricle, the forward flow decreased, and the ejection fraction of the right ventricle reduced. Thus, the right
ventricular function was abnormally depressed in patients with low cardiac output.

In this disease, the left ventricle does not have an additional hemodynamic burden. If the low left ventricular ejection fraction is due to a decreased inflow into the left ventricle, the right ventricular function would be the main factor in maintaining cardiac output adequately. In the present study cardiac output did not correlate with the pulmonary vascular resistance or the left atrial to left ventricular end-diastolic pressure gradient, but it correlated with the right ventricular ejection fraction. Seven of our patients had a left atrial to left ventricular end-diastolic pressure gradient of more than 10 mmHg and 10 had pulmonary hypertension with a pulmonary arterial mean pressure of more than 25 mmHg. Therefore, our study included data on the right ventricular function in patients with mild or moderate mitral stenosis.

The right ventricular ejection fraction did not correlate with the right ventricular systolic pressure or the pulmonary vascular resistance. These findings were different from those in the previous report and our study does not indicate that pulmonary hypertension is a factor in decreasing the right ventricular ejection fraction. Kirschbaum et al. have reported that the improvement of the right ventricular ejection fraction following mitral valve replacement is not influenced by the presence and/or by the level of pulmonary hypertension. We could not clarify factors involved in the mechanism which depress right ventricular function in patients with low cardiac output, nor did we determine which comes first, low cardiac output or depressed right ventricular function. Kirschbaum et al. have also shown that the right ventricular ejection fraction increased after mitral valve replacement and they suggested that a reduction of afterload for the right ventricle improves right ventricular function. However, it is doubtful whether or not the right ventricular function in patients with tricuspid regurgitation and a low level of afterload is improved after mitral valve repair or valve replacement.

The right ventricular ejection fraction was significantly lower in patients who suffered from restenosis after closed mitral commissurotomy. Halperin et al. have reported that the left ventricular ejection fraction in patients with mitral stenosis decreases in patients after mitral commissurotomy. In the present study, the left ventricular ejection fraction was low, but not significantly so, in patients with restenosis of the mitral valve. Therefore, this decrease of the right ventricular pump function in recurrent patients may be related to the duration of mitral valve disease. Right ventricular dysfunction may also be induced by rheumatic inflammatory changes in its active phase.

The left ventricular end-diastolic volume was normal in all patients and the left ventricular ejection fraction was reduced in patients with a low cardiac output of less than 2.50 L/min/m². The presence of an abnormal left ventricular function in patients with mitral stenosis has been reported previously and it may be induced by inflammatory changes of myocarditis or fibrosis of the papillary muscle. The reduced left ventricular ejection fraction is probably due to a decreased inflow to the left ventricle. Mitral stenosis causes a hemodynamic burden on the right ventricle. This disease is an acquired heart disease, and the left ventricle can grow to the normal adult size.

REFERENCES

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