The effect of baseline pulmonary artery pressure on right ventricular functions after mitral balloon valvuloplasty for rheumatic mitral stenosis: a tissue Doppler imaging study

Romatizmal mitral darlığı olan hastalarda bazal pulmoner arter basınçının perkütan mitral balon valvüloplasti sonrası sağ ventrikül fonksiyonlarına etkisi: Doku Doppler görüntüleme çalışması

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Objectives: We evaluated the effect of baseline pulmonary artery pressure (PAP) on right ventricular functions after percutaneous mitral balloon valvuloplasty (PMBV) for rheumatic mitral stenosis (MS).

Study design: The study included 56 patients (15 males, 41 females; mean age 35 years) who underwent PMBV for isolated rheumatic MS. The patients were divided into two groups according to the baseline median systolic pulmonary artery pressure (PAP ≥40 mmHg, n=33; PAP <40 mmHg, n=23) measured before PMBV by echocardiography. Right ventricular function was assessed by pulse wave tissue imaging and the Tei index. Assessments were repeated 48 hours and three months after PMBV.

Results: The peak systolic (S) velocity of the lateral tricuspid annulus did not differ between the two groups at baseline. In patients with pulmonary artery hypertension (PAH), it showed a slight increase at 48 hours, but fell behind the baseline at three months. In patients without PAH, it showed a significant increase at 48 hours and remained unchanged at three months. Peak late diastolic (A) velocities were significantly higher at all times in patients without PAH. Patients with PAH had a significantly higher E/A ratio both at baseline and at 48 hours; however, at three months, this difference disappeared. Patients with PAH had higher isovolumic relaxation time (IVRT) at baseline and 48 hours; however, final IVRT was lower than the baseline only in patients with PAH. Isovolumic contraction time showed a steady but insignificant increase in both groups over three months. E-wave deceleration time showed a significant increase and contraction time showed a slight increase over three months only in patients with PAH. The baseline Tei index was higher in patients with PAH (p=0.004). Changes in the Tei index over time were not significant.

Conclusion: Our findings suggest that, if PMBV is performed before the development of PAH, it may be more effective in the improvement of right ventricular longitudinal functions in patients with rheumatic MS.

Keywords: Balloon dilatation; echocardiography/methods; mitral valve stenosis/therapy; hypertension, pulmonary; rheumatic heart disease/therapy; ventricular function, right.

Amac: Romatizmal mitral darlığı (MD) olan hastalarda başlayan pulmoner arter basınçının (PAB) perkütan mitral balon valvüloplasti (PMBV) sonrası sağ ventrikül fonksiyonlarına etkisi değerlendirilmiştir.

Çalışma planı: Çalışmaya romatizmal MD nedeniyle PMBV uygulanan 56 hasta (15 erkek, 41 kadın; ort. yaş 35) alınmıştır. Hastalar PMBV öncesinde ekokardiyoğrafi ile ölçülen bazal ortanca sistolik pulmoner arter basınçına göre iki gruba (PAB ≥40 mmHg, n=33; PAB <40 mmHg, n=23) ayrılmıştır. Sağ ventrikül fonksiyonlarını pulse wave doku Doppler ekokardiyoğrafi ve Tei indeksi ile değerlendirilmiştir. Tüm ölçümler PMBV’den 48 saat ve üç ay sonra tekrarlanmıştır.

Bulgular: İşlem öncesinde iki gruba benzer olan trıkuspid lateral annulusun tepe sistolik (S) hızı, pulmoner arter hiper-tansiyonu (PAH) olan grupta ilk 48 saatte hafif artış gösterdi, üçncü ayda ise başlangıç değerinin gerisine düştü; PAH olmayan grupta ise ilk 48 saatte anlamlı artış göstermek bu dönemde üçncü ayda kadar korudu. Tepe geç diyastolik (A) hızı PAH olmayan grupta üç oranı daha yüksekti. İzvolümik relaksasyon zamanı PAH’li grupta başlangıçta ve 48. saatte anlamlı derecede iken, üçncü ayda bu fark kaybıldı. İzvolümik kontraksiyon zamanı PAH’li grupta başlangıçta ve 48. saatte daha yüksekti, son değerlendirme sadece bu grupta başlangıç değerinin gerisine düştü. İzvolümik kontraksiyon zamanı, üçncü aya kadar her iki grupta da anlamlı olmayan artış gösterdi. Sadece PAH’li grupta olmak üzere, üçncü aya kadar E dalgası deselerasyon zamanında anlamlı, kon-traksiyon zamanında hafif artış görüldü. Başlangıçta PAH’li grupta anlamlı derecede yüksek (p=0.004) olan Tei indeksi derece değişiklikler gruplarda anlamlı farklılıkları göstermedi.

Sonuç: Bulgularımız, romatizmal MD’li hastalarda PMBV’nin PAH gelişiminden önce uygulanması halinde, sağ ventrikül longitudinal fonksiyonlarında daha fazla düzelve sağlayıcı-Leceğini göstermektedir.

Anahtar sözü: Balon dilatasyonu; ekokardiyoğrafi/yöntem; mitral kapak darlığı/tedavî; hiper-tansiyon/pulmoner; romatoid kalıp hastalığı/tedavî; ventrikül fonksiyonu, sağ.
Rheumatic mitral stenosis (MS) may impair right ventricular (RV) function due to passive increase in left atrial pressure and reactive changes in pulmonary arteriolar vasculature leading to RV overload.

The effect of successful percutaneous mitral balloon valvuloplasty (PMBV) on global RV systolic and diastolic functions in patients with rheumatic MS has not been well-defined. Radionuclide ventriculography and magnetic resonance imaging (MRI), the most frequently used methods for the evaluation of RV function, are time consuming, relatively expensive, and not appropriate for bedside use. Pulsed Doppler tissue imaging (DTI) is a unique method of measuring systolic and diastolic velocities of annular motion. It is also a potentially noninvasive and appropriate technique for bedside use and less expensive than radionuclide ventriculography or MRI. Previously, a Doppler-derived index (Tei index) combining systolic and diastolic time intervals was proposed by Tei. It is defined as the sum of isovolumic contraction and relaxation time divided by the ejection time and considered to be simple, reproducible, and independent of heart rate and blood pressure. Being a combined measurement of systolic and diastolic myocardial performance, the Tei index is thought to be more valuable because it represents overall cardiac function rather than systolic or diastolic functions alone in both ventricles. It provides a measure of global cardiac function.

Our literature search did not reveal any published data as to whether baseline pulmonary artery pressure (PAP) influenced RV systolic and diastolic functions following PMBV as assessed by DTI-derived Tei index. This study was designed to evaluate the effect of PMBV on global RV systolic and diastolic functions assessed by DTI and DTI-derived Tei index and to determine whether the effect of PMBV was influenced by the presence or absence of preprocedural pulmonary artery hypertension (PAH).

**PATIENTS AND METHODS**

The study included 56 consecutive patients (15 males, 41 females; mean age 35 years; range 19 to 68 years) who underwent PMBV for isolated rheumatic MS between April 2003 and March 2005. Baseline systolic pulmonary artery pressures were measured before PMBV by echocardiography. The patients were divided into two groups according to the median pulmonary artery pressure (PAP ≥40 mmHg, n=33; PAP <40 mmHg, n=23). The study was carried out in accordance with the Declaration of Helsinki (1964) and current revisions of the Good Clinical Practice guidelines of the European Commission, and all patients gave informed consent.

All the patients were in sinus rhythm and had class II or III functional capacity according to the New York Heart Association classification.

Patients with any of the following were excluded from the study: moderate or severe mitral regurgitation (MR), moderate or severe aortic regurgitation or stenosis, tricuspid stenosis, previous aorta or mitral valve operation or valvuloplasty, clinical, electrocardiographic or angiographic evidence for coronary artery disease, diabetes mellitus, systemic hypertension, wall motion abnormality, bundle branch block, evidence for rheumatic disease activity in the preceding six months, and severe mitral annular calcification.

Percutaneous mitral balloon valvuloplasty was performed using the Inoue balloon technique and without any complications.

**Echocardiography.** Echocardiographic examination was made by an experienced echocardiographer at baseline, and 48 hours and three months after PMBV. All patients were examined in the left lateral decubitus position by M-mode, two-dimensional, Doppler and DTI echocardiography with the use of an Aloka SSD-5500 echocardiography device (Aloka Holding Europe AG, Switzerland) and a 3.5 MHz transducer. A continuous one-lead electrocardiogram was recorded during the procedure. Mitral valve area was calculated with the pressure half-time method. Peak and mean diastolic transmitral gradients were measured by continuous-wave Doppler echocardiography. Estimated valve areas were verified by using the area trace method in all patients. Mitral regurgitation was categorized as absent, mild, moderate, or severe using a combination of qualitative and quantitative parameters. Tricuspid regurgitation (TR) jet flow was assessed from the apical, subcostal, and parasternal views. Systolic pulmonary artery pressure (PAP) was measured with continuous-wave Doppler. The maximum peak TR velocity (V) recorded from any view was used to determine right ventricular systolic pressure with the simplified Bernoulli equation (PAP = 4V² + RAP). To estimate right atrial pressure, inferior vena cava diameter was measured from the subxiphoid long-axis view. Right atrial pressure was estimated using the caval respiratory index as described by Kircher et al. Pulmonary artery systolic pressure was estimated from the sum of the transtricuspid
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Gradient and right atrial pressure. Left atrial diameter was calculated from the parasternal long-axis view during M-mode echocardiography.

Doppler tissue imaging. In the apical four-chamber view, the DTI cursor was placed at the tricuspid annulus of the RV free wall. The filter settings were kept low (50 Hz), gains were adjusted at the minimal optimal level to minimize noise and eliminate the signal produced by the transmitral flows, and a 3-mm sample volume was used. A Doppler velocity range of -20 to 20 cm/sec was selected. Maximum systolic and diastolic velocities were measured online at a sweep speed of 50 mm/sec. Special attention was paid to align the pulsed-wave cursor so that the Doppler angle of incidence was as close to 0° as possible to the direction of motion of the mitral and tricuspid annulus.

Three major velocities were recorded: the positive systolic velocity when the tricuspid ring moved toward the cardiac apex, and two negative diastolic velocities when the mitral and tricuspid annulus moved toward the base away from the apex. Systolic (S), early diastolic (E), and late diastolic (A) maximum velocities, isovolumic contraction time (IVCT), ejection time (ET), isovolumic relaxation time (IVRT), and E-wave deceleration time (DT) were measured by DTI from the sites of the mitral annulus as demonstrated in Fig. 1. Systolic indexes of DTI included myocardial peak velocity of S, IVCT (from the onset of ECG QRS to the beginning of S), and ET (from the beginning to the end of S-wave). Diastolic indexes included E and A peak velocities, E/A ratio, DT and IVRT which was measured as the time interval occurring between the end of S and the onset of E-wave. The DTI-derived Tei index was obtained from the tricuspid annulus as the sum of IVCT and IVRT divided by ET. Five cardiac cycles were measured and averaged.

Reproducibility. Intraobserver variability was assessed in 10 patients by repeating the measurements on two occasions (1 to 10 days apart) under the same basal conditions. To test the interobserver variability, the measurements were made offline from video recordings by a second observer who was blinded to the results of the first examination. Error was calculated as the difference between the two measurements divided by the mean of the measurements.

Intraobserver and interobserver variabilities for conventional DTI-derived parameters of the tricuspid lateral annulus ranged from 3.1% to 5.4%. The corresponding figures for the Tei index were 3.2±2.6% and 4.4±1.8%, respectively.

Statistical analysis. All data were processed using SPSS for Windows 11.5 statistical software package and were expressed as mean ± standard deviation or percentage. The observed changes after valvuloplasty were analyzed for statistical significance using the 2x2 analysis of variance for repeated measures and “Bonferroni adjustment” was made for multiple comparisons. Echocardiographic parameters of the two groups were compared. P values of less than 0.05 were considered significant.

RESULTS

During baseline echocardiographic evaluations, minimal MR was found in 48 patients. After PMBV, minimal MR developed in seven patients who had no MR before. In six patients with minimal MR at baseline, the degree of MR increased to moderate. Of these, two patients were found to have minimal MR again at three months.

Echocardiographic and DTI findings after PMBV. Echocardiographic and DTI findings obtained before and after PMBV (at 48 hours and 3 months) are presented in Table 1. At 48 hours after PMBV, the mean mitral valve area increased to 1.9±0.2 cm² (p<0.0001), the mean mitral valve gradient decreased to 4.2±3.0 mmHg (p<0.0001), and left atrial diameter became smaller (p<0.05) compared with the baseline values. Aside from S-wave peak velocity which showed a
There was no difference between the two groups with regard to the baseline peak S velocity. Patients with PAH exhibited a slight increase in peak S velocity at 48 hours, but at three months, it fell behind the baseline value. In contrast, patients without PAH showed a different course. Peak S velocity showed a significant increase at 48 hours and remained unchanged at three months.

Compared to the patients with PAH, peak A velocities were significantly higher at all times in patients without PAH (p<0.05); however, they remained similar within the groups at baseline, 48 hours, and three months.

Patients with PAH had a significantly higher E/A ratio both at baseline and at 48 hours compared to the patients without PAH.

**Table 1. Echocardiographic variables before and after valvuloplasty**

<table>
<thead>
<tr>
<th></th>
<th>Before valvuloplasty</th>
<th>48 hours</th>
<th>3 months</th>
<th><strong>p</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Mitral valve area (cm²)</td>
<td>1.1±0.2</td>
<td>1.9±0.2*</td>
<td>1.8±0.2*</td>
<td>0.048</td>
</tr>
<tr>
<td>Mitral valve gradient (mmHg)</td>
<td>11.2±1.8</td>
<td>4.2±3.0*</td>
<td>6.3±4.8*</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Left atrial diameter (cm)</td>
<td>4.5±0.5</td>
<td>4.0±0.6*</td>
<td>3.9±0.7*</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Pulmonary artery systolic pressure (mmHg)</td>
<td>34.5±8.6</td>
<td>25.5±9.2*</td>
<td>29.5±9.5</td>
<td>0.048</td>
</tr>
<tr>
<td>Peak systolic (S) tricuspid annular velocity (cm/sec)</td>
<td>11.0±2.8</td>
<td>13.0±2.2</td>
<td>12.7±2.5</td>
<td>0.495</td>
</tr>
<tr>
<td>Peak early (E) diastolic tricuspid annular velocity (cm/sec)</td>
<td>12.1±4.1</td>
<td>13.8±6.4</td>
<td>12.0±2.4</td>
<td>0.392</td>
</tr>
<tr>
<td>Peak late (A) diastolic tricuspid annular velocity (cm/sec)</td>
<td>13.6±3.9</td>
<td>13.1±3.8</td>
<td>13.1±5.6</td>
<td>0.900</td>
</tr>
<tr>
<td>Isovolumic contraction time (msec)</td>
<td>92.8±24.3</td>
<td>89.6±27.8</td>
<td>99.0±20.2</td>
<td>0.725</td>
</tr>
<tr>
<td>Contraction time (ET) (msec)</td>
<td>296.3±43.5</td>
<td>279.6±30.6</td>
<td>288.1±33.0</td>
<td>0.605</td>
</tr>
<tr>
<td>Isovolumic relaxation time (msec)</td>
<td>74.3±39.1</td>
<td>76.1±38.4</td>
<td>67.7±34.8</td>
<td>0.510</td>
</tr>
<tr>
<td>E-wave deceleration time (msec)</td>
<td>87.5±40.0</td>
<td>93.0±21.6</td>
<td>107.1±43.9</td>
<td>0.167</td>
</tr>
<tr>
<td>E/A ratio</td>
<td>0.9±0.5</td>
<td>1.1±0.6</td>
<td>1.0±0.4</td>
<td>0.193</td>
</tr>
<tr>
<td>Tei index</td>
<td>0.57±0.21</td>
<td>0.60±0.25</td>
<td>0.59±0.24</td>
<td>0.568</td>
</tr>
</tbody>
</table>

**Table 2. Echocardiographic variables before (baseline) and after valvuloplasty in patients with or without baseline pulmonary hypertension**

<table>
<thead>
<tr>
<th></th>
<th>Pulmonary hypertension (+) (n=33)</th>
<th>Pulmonary hypertension (–) (n=23)</th>
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<tbody>
<tr>
<td></td>
<td>Baseline 48 hours 3 months</td>
<td>Baseline 48 hours 3 months</td>
</tr>
<tr>
<td>Echocardiographic variables</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pulmonary artery pressure (mmHg)</td>
<td>45.2±4.2 35.0±7.2* 35.6±8.7*</td>
<td>33.3±8.2 24.4±9.1* 28.8±9.9**</td>
</tr>
<tr>
<td>Mitral valve area (cm²)</td>
<td>1.0±0.1 1.8±0.4* 1.8±0.2*</td>
<td>1.0±0.1 1.9±0.2* 1.8±0.2*</td>
</tr>
<tr>
<td>Mitral valve gradient (mmHg)</td>
<td>13.0±1.4 3.2±1.7* 3.0±1.4*</td>
<td>10.9±1.7 4.4±3.2* 7.1±4.7*</td>
</tr>
<tr>
<td>Pulsed-wave Doppler tissue imaging</td>
<td>Tricuspid annular velocity (cm/sec)</td>
<td></td>
</tr>
<tr>
<td>Peak systolic (S)</td>
<td>11.5±4.9 12.0±1.4 10.5±0.7</td>
<td>10.9±2.6 13.3±2.3* 13.2±2.5*</td>
</tr>
<tr>
<td>Peak early diastolic (E)</td>
<td>13.5±0.7 15.0±4.2 12.5±0.7</td>
<td>11.9±4.4 13.6±6.9 12.3±2.5</td>
</tr>
<tr>
<td>Peak late diastolic (A)</td>
<td>10.0±5.6 9.5±3.5 10.5±3.7</td>
<td>14.3±3.4 13.9±3.5 13.7±6.0</td>
</tr>
<tr>
<td>E/A ratio</td>
<td>1.6±0.9 1.7±1.1 1.0±0.1**</td>
<td>0.8±0.3 1.0±0.4 1.0±0.4</td>
</tr>
<tr>
<td>Isovolumic contraction time (msec)</td>
<td>103.3±7.7 104.5±45.9 117.5±19.2</td>
<td>89.3±37.9 90.0±33.1 95.3±19.2</td>
</tr>
<tr>
<td>Contraction time (ET) (msec)</td>
<td>287.0±36.7 280.5±42.7 309.0±35.5</td>
<td>298.2±56.8 279.5±27.6 284.6±34.9</td>
</tr>
<tr>
<td>Isovolumic relaxation time (msec)</td>
<td>97.0±7.21 91.5±37.4 59.0±21.2**</td>
<td>56.2±32.0 84.4±38.9 69.5±37.6</td>
</tr>
<tr>
<td>E-wave deceleration time (msec)</td>
<td>55.5±16.0 104.5±19.0* 120.0±15.5*</td>
<td>93.9±35.9 90.8±22.2 104.6±37.8</td>
</tr>
<tr>
<td>Tei index</td>
<td>0.72±0.37 0.77±0.52 0.57±0.19</td>
<td>0.49±0.21 0.63±0.25 0.60±0.26</td>
</tr>
</tbody>
</table>

*p<0.05: Comparison with baseline within each group; **p<0.0001: Comparison between measurements at 48 hours and at 3 months.
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without PAH (p<0.05); however, at three months, the difference between the two groups disappeared.

Baseline and 48-hour IVRT values of the patients with PAH were higher than those of patients without PAH. However, final IVRT was lower than the baseline only in patients with PAH. In contrast, IVCT showed a steady increase from the baseline over three months in both groups without exhibiting significant differences within each group or between the two groups. E-wave deceleration time increased significantly over three months only in patients with PAH, whereas it remained similar in patients without PAH. Contraction time was slightly increased from baseline at three months only in patients with PAH.

The baseline Tei index was higher in patients with PAH (p=0.004). In this group, it decreased below the baseline at three months, whereas remained higher than the baseline in patients without PAH, but none of these changes were significant.

DISCUSSION

The RV function is an important determinant of clinical symptoms, exercise capacity, and survival in patients with MS.[7] Radionuclide and hemodynamic studies demonstrated that RV ejection fraction decreased in MS due to increased RV afterload and improved immediately after PMBV. Improvement in RV ejection fraction after valvuloplasty was thought to be directly related to the increase in stroke volume, which was shown to be inversely correlated with RV ejection fraction before valvuloplasty.[8] The studies which evaluated the effect of PMBV on RV functions often used catheter- or radionuclide-based measurements.[9,10]

Increased pulmonary wedge pressure as a result of MS is associated with a rise in pulmonary artery pressure, increased afterload, and consequently, a drop in RV ejection fraction. Although a rapid improvement has been reported in RV systolic function following relief of mitral valve obstruction,[9] it has also been shown that stroke volume response to exercise remains abnormal for variable durations following PMBV.[14]

There is no clear data as to whether the effect of PMBV on RV functions is influenced by the pre-procedural PAH existence. Meluzin et al.[2] demonstrated the utility of DTI of tricuspid annular motion for non-invasive evaluation of RV systolic function. Several studies have been published regarding the use of the Tei index[3,15,16] and pulsed DTI to identify patients with impaired systolic[16-18] and diastolic[13,16-22] function. The Doppler-derived Tei index, a combined measurement of systolic and diastolic myocardial performance, is a reproducible method that can be easily obtained and does not require heart rate and blood pressure correction. The index is thought to be more representative of overall cardiac function than systolic or diastolic function alone in both ventricles, and thus, may provide a measure of global cardiac function. Besides, the Tei index was demonstrated to correlate with parameters of RV function obtained by right heart catheterization.[23]

The Tei index encompasses important energy-dependent periods of systolic contraction, ejection and diastolic relaxation. The use of Doppler offers the possibility of high feasibility in patients with poor image quality and accurate characterization of function in the context of complex chamber geometry. Calculation of the Tei index is based on measurement of Doppler-derived time intervals including IVCT, IVRT, and ET. In the case of systolic dysfunction, IVCT increases and RV ET decreases. Most abnormalities of diastolic function are manifested by an abnormally slow rate of pressure decline. These changes are accompanied by prolongation of IVRT. As global myocardial dysfunction progresses, myocardial performance index increases due to changes in all three time interval components used for its calculation.[17]

The Tei index is not greatly influenced by changes in heart rate, preload, blood pressure, afterload, RV pressure and dilatation, or tricuspid regurgitation in the clinical setting.[3,24-28] A previous study reported that, despite a rapid fall in pulmonary artery pressures concomitant with a significant increase in mitral valve area following PMBV, RV global function did not show a significant improvement in the immediate postprocedural period.[29] It was also shown that the Doppler index of global RV function indicated a substantial improvement over a mean period of one year, with 65% of patients attaining normal values.

To our knowledge, there is no information about the effect of baseline PAP on RV systolic and diastolic functions following PMBV, estimated by TDI and the Tei index, in patients with isolated MS. In this respect, our findings correlated well with the aforementioned study suggesting the persistence of subtle residual RV dysfunction in a significant proportion of patients, especially in those with baseline PAH.[29]

It was noticeable that, in the intermediate period, a change in IVRT was seen only in patients with baseline PAH, and an increase in S velocity occurred only in patients without PAH. The decline in PAP after the procedure was attributable to decreased afterload in
patients with baseline PAH, while patients without PAH exhibited increases in systolic function parameters even though their afterload values seemed to be much lower. After PMBV, we observed a change in the RV S-wave velocity only in patients without PAH, which might be due to a more protected RV function in this particular group. Another confounding factor might be the shortness of the follow-up period, which was only three months. Similar RV changes might be expected within a longer follow-up period in patients with baseline PAH, but this needs to be clarified by further studies.

It has been reported that pulmonary pressure usually normalizes within six months, but may stay elevated for more than two years in some patients. In our study, although improvement in systolic function was more significant in patients without PAH, there was no significant difference in the Tei index during three-month follow-up. Evaluation at three months showed that the Tei index of patients with baseline PAH decreased to values similar to those of patients without baseline PAH. Although the range of PAP values was relatively narrow, it was noted that PAP was still relatively higher in the intermediate follow-up period in patients with baseline PAH compared to patients without baseline PAH, but the difference was not statistically significant.

The changes observed in the PAH group after PMBV, such as decreases in the E/A and IVRT, and prolongation of DT were suggestive of an improvement in diastolic function. Our interpretation was that a switch from a pseudonormalization pattern occurred to a relaxation abnormality after PMBV. Based on this observation, we may expect an increased S-wave velocity at a longer follow-up.

On the other hand, significant increase in longitudinal movement of the tricuspid annulus after the procedure may also be related to improvement in functional restriction of the mitral valve apparatus after valvuloplasty. As the left and right ventricles are related to each other anatomically and functionally, it may be speculated that any change in left ventricular function will also lead to a change in RV long-axis systolic and diastolic function. These findings are not surprising when close anatomical connection is considered between both ventricles. In a previous study, we showed that mitral annular S-wave velocity was increased after PMBV.

The abnormal IVRT detected in patients with baseline PAH before PMBV may indicate afterload-independent subtle abnormalities of RV function in this patient group. This might be the result of partial involvement of RV in the process of myocardial fibrosis, which was previously reported in patients with MS. Besides, a rigid mitral valve apparatus may also affect RV diastolic function because of close interaction between the right and left ventricles.

While tricuspid annular motion primarily reflects the function of longitudinal myocardial fibers, RV ejection fraction or global function are influenced by both the contractility of longitudinal and concentric myocardial fibers. Thus, damage to RV myocardium may affect annular velocities and the global functions in different ways.

Limitations. The narrow range of PAP values is an important limitation to our study. We did not measure the RV ejection fraction or volumes. It is difficult to assume that the change in the Doppler index is primarily dependent upon the improvement in RV global function and, secondarily, upon RV systolic function. Further studies with large patient groups and long-term follow-up are needed for the clinical effects of these changes on RV functions.

Additionally, in the present study, the degree of tricuspid regurgitation was not determined quantitatively. It might have been more severe in the PAH group, which would affect the RV systolic and diastolic functions.

Calculation of mitral valve area by the pressure half-time method is the easiest way, but has some limitations if it is applied immediately after PMBV. In order to improve accuracy, we also measured valve area by using the area trace method in all the patients.

In conclusion, our findings suggest that, in the intermediate follow-up period after successful PMBV, the longitudinal systolic wave velocities are increased in patients without PAH, while the Tei index showing the global functions improve in patients with PAH and reach to similar levels as in patients without PAH. If PMBV is performed before the development of PAH, it may be more effective in the preservation of RV longitudinal functions in patients with pure rheumatic MS.

REFERENCES

2. Meluzin J, Spinarova L, Bakala J, Toman J, Krejci J,


