Immediate impact of successful percutaneous mitral valve commissurotomy on right ventricular function

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Aims Mitral stenosis (MS) affects right ventricular (RV) function as a result of myocardial and haemodynamic factors. Although the long-term effects of mitral commissurotomy are well known, the aim of this study was to evaluate the immediate impact of percutaneous mitral commissurotomy (PTMC) on RV function in patients with MS.

Methods and results Twelve female patients (mean age 29 ± 7 years) with isolated rheumatic MS, all in sinus rhythm, were studied before and 24–48 h after PTMC. Multiple parameters of global and longitudinal RV function were assessed by conventional and tissue Doppler imaging echocardiography. Immediately following PTMC, mitral valve area increased from 0.91 ± 0.29 cm² to 1.86 ± 0.43 cm² (P < 0.0001) and RV outflow tract fractional shortening (RVOTfs) increased from 57 ± 15% to 72 ± 12% (P = 0.002). There was a significant decrease in systolic pulmonary artery pressure from 46.4 ± 32.1 mmHg to 29.1 ± 13.4 mmHg (P = 0.02), in the RV Tei index from 0.44 ± 0.025 to 0.29 ± 0.17 (P = 0.021), in myocardial acceleration during isovolumic contraction (IVA) at the lateral tricuspid annulus from 0.36 ± 0.11 m/s² to 0.25 ± 0.07 m/s² (P = 0.023), and in isovolumic contraction velocities at the lateral tricuspid annulus from 11.03 ± 3.37 cm/s to 8.50 ± 2.04 cm/s (P = 0.034). In contrast, tissue Doppler velocities at the septal tricuspid annulus remained unchanged. The RV Tei index correlated with systolic pulmonary artery pressure before but not after PTMC (r = 0.70, P = 0.01, and r = 0.270, P = 0.053).

Conclusion Immediately after successful PTMC, significant decrease in RV contractility as assessed by IVA was observed whereas other parameters of infundibular and global RV function as assessed by RVOTfs and Tei index showed significant improvement. These discordant results may be related to the relative insensitivity of currently available echocardiography parameters of RV function that are not completely immune to loading conditions. Further work using larger numbers of patients is needed to confirm our findings and to assess their utility in patient follow-up and management.

Keywords Mitral stenosis; Echocardiography; Doppler tissue imaging; Right ventricular function; Percutaneous mitral commissurotomy

Introduction

Abnormalities of right ventricular function (RVF) play an important role in the development of clinical symptoms and the overall prognosis of patients with mitral stenosis (MS).1 RVF may be affected either by the rheumatic process directly2 or through haemodynamic changes due to pulmonary vascular alterations.3 Haemodynamic and radionuclide studies4,5 have demonstrated long-term improvement in RV function after percutaneous balloon mitral valvuloplasty (PTMC). However, few studies have examined the immediate impact of mitral valvuloplasty on echocardiographic markers of RV systolic and diastolic function.1,6,7 The purpose of this study was to assess the immediate effect of PTMC on RV function using two-dimensional and Doppler echocardiographic indices.

Methods

Population

Twenty-one patients presented to our institution with MS between 20 April and 20 July 2006. Of these, 12 females ranging in age

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from 22 to 36 years (mean 29 years) with pure MS were treated with PTMC. None had atrial fibrillation, systemic hypertension, diabetes mellitus, more than mild mitral or aortic regurgitation and/or aortic stenosis, New York Heart Association functional class IV, or previous aortic or mitral valve surgery. Indications for PTMC were New York Heart class \( \geq II \), \( \leq IV \), planimetered MVA \(< 1.5 \text{ cm}^2 \), mitral regurgitation \( \leq 2^+ \), suitable valve morphology, and absence of concomitant cardiovascular disease requiring surgical correction. Of the nine other patients admitted in the same period for MS, four patients had valve morphology not suitable for PTMC, one patient had severe tricuspid regurgitation (TR), which required open-heart surgery, and four patients had atrial fibrillation. The control group consisted of nine age-matched healthy women who ranged in age from 25 to 37 years (mean age 31 years). None had ECG or echocardiographic evidence of structural or functional cardiovascular disease.

Echocardiographic measurements

Two-dimensional (2D) echocardiography and Doppler studies were performed before and 24–48 h after PTMC. All studies were obtained using a Philips® Sonos 5500 ultrasonographic machine equipped with a 3.5 MHz transducer. The same experienced echocardiographer (A.D.) performed all measurements using the recommendations of the American Society of Echocardiography.\(^8\) Mitral valve area (MVA) was determined by planimetry in every patient. The peak and mean mitral valve transannular pressure gradients and late filling velocities were measured using the Bernoulli principle from continuous wave Doppler recordings through the centre of mitral inflow. The Wilkins score\(^9\) was used to judge mitral leaflet mobility, valvar and subvalvar thickening, and calcification. Twenty four to 48 h after mitral balloon dilatation, MVA was again determined by planimetry. Systolic pulmonary artery pressure was derived from the tricuspid regurgitant jet peak velocity using the modified Bernoulli equation (peak gradient \(4V^2\), where \(V\) is the maximal velocity of the tricuspid regurgitant jet). We further assumed a right atrial mean pressure of 10 mmHg in patients, based on the absence of inferior vena cava dilation greater than 20 mm, and 5 mmHg in controls. From the parasternal short-axis view at the level of the aortic root, the RV outflow tract diameters at end-diastole and end-systole were measured. RV outflow tract fractional shortening (RVOTf) was calculated using the formula RVOTf = \([\text{RVOTd}-\text{RVOTs}]/\text{RVOTd}\)\(^{10}\) where \(d\) and \(s\) represent end-diastolic and end-systolic dimensions. The tricuspid annular plane systolic excursion (TAPSE) was determined by the difference in the displacement of the RV base during systole and diastole.\(^11\) RV diastolic and end-systolic areas were measured from the apical four-chamber view to calculate RV fractional area change (RVFAC).\(^12\) With the same views, the RV ejection fraction was calculated using Simpson's Rule.\(^13\) The Tei index of RV myocardial performance was calculated as the time between tricuspid valve closure to tricuspid valve opening, divided by the RV ejection time, determined by pulsed Doppler.\(^14\) We did not measure regional RV Tei index from DTI (Doppler tissue imaging).

Pulsed wave DTI was obtained by activating the machine's Doppler tissue imaging function with gains adjusted to eliminate transvalvular flow velocities and minimize noise. A 3.5 mm sample volume was placed at the septal and lateral side of the tricuspid annulus. Peak myocardial velocities during systole, early, and late diastole together with the isovolumic contraction time were measured at a sweep speed of 100 mm/s. Myocardial acceleration during isovolumic contraction (IVAC) was measured by dividing myocardial velocity during isovolumic contraction by the time interval from onset of the myocardial velocity during isovolumic contraction to the time at peak velocity of this wave.\(^15\) The final values of all parameters were obtained after averaging over three cardiac cycles. All measurements were made by a single observer (A.D.), blinded to the patient's identity and to pre-, and post-PTMC status.

Intra-observer variability of RV Tei, RVFAC, RVOTf and IVA was tested by repeated measurements of the same beats on the same occasion in eight consecutive recordings. The mean percentage error was then derived from the difference between these two sets of measurements divided by the mean of the two observations.

Percutaneous mitral commissurotomy

All patients underwent PTMC by the antegrade transeptal approach using an Inoue balloon and a stepwise dilatation strategy.\(^16\) The nominal balloon diameter was decided according to the height of the patient \([\text{height (cm)/10} = \text{balloon diameter}]\).\(^17\) Echocardiography was done at the end of the procedure to assess for perforation and to look for an atrial left-to-right shunt using colour flow Doppler. Successful PTMC was defined as post-valvuloplasty valve area \(> 1.5 \text{ cm}^2\) with no more than 2+ mitral regurgitation.

Statistical analysis

Data were expressed as mean \(\pm SD\). The normal distribution of our population was checked by Shapiro–Wilks test.\(^18\) Analysis employed the Student's \(t\)-test for paired data to determine the significance of differences before and after PTMC. The differences between patients with MS and healthy subjects were identified using an unpaired Student's \(t\)-test (after checking for normality and homogeneity of variance assumptions). To show the relationship between the variables in the patient groups, Pearson correlation analysis was performed. A \(P\)-value \(< 0.05\) was considered statistically significant. The statistical package SPSS 11.5 (version 11.5, SPSS Inc., Chicago, IL, USA) was employed.

Results

Nine patients were in New York Heart Association class II and three were in class III before PTMC; the mean score of Wilkins was 7.9 \(\pm 1.2\); by planimetry the mean MVA was 0.91 \(\pm 0.29 \text{ cm}^2\); and the mean value of pulmonary systolic arterial pressure \((SPAP) = 46.4 \pm 32.1\). Four patients had SPAP more than 40 mmHg: 44, 52, 67, and 141 mmHg. PTMC was successfully completed in all 12 patients. There was no evidence of cardiac perforation, significant left-to-right atrial shunting, or more than 2+ MR post PTMC. The heart rate remained stable at 72.4 \(\pm 7.2\) bpm before and 75.1 \(\pm 13.2\) bpm after PTMC \((P = 0.13)\). Comparison of pre- and post-PTMC echocardiographic measurements and DTI velocities are shown in Tables 1, 2, and 3. After PTMC, the mean transmural gradient decreased from 16.4 \(\pm 8.8\) mmHg to 5 \(\pm 1.5\) mmHg \((P = 0.001)\), left ventricle \((LV)\) end-diastolic diameter and LV ejection fraction remained stable whereas LV end-systolic diameter increased \((P = 0.001)\). Ten patients had measurable peak TR gradients before PTMC and eight patients after. TR was judged visually grade I in eight patients before and six patients after PTMC. Two patients had grade II TR that remained at the same grade after PTMC. No patient had more than grade II TR. The RVOTf increased \((P = 0.002)\) and the RV Tei index significantly decreased \((P = 0.02)\) (Figure 1). The isovolumic acceleration and contraction velocities obtained at the lateral tricuspid annulus decreased \((P = 0.02, P = 0.03;\) respectively) (Figures 2 and 3), and the RV ejection fraction, RVFAC, excursion of the tricuspid annular plane and Doppler myocardial velocities at the septal annulus remained stable (Tables 2 and 3). Only RV Tei index was found to correlate directly to systolic pulmonary artery pressure before but not after PTMC \((r = 0.70, P = 0.01; r = 0.270, P = 0.053)\).
Reanalysing the data by omitting the patient presenting with SPAP at 141 mmHg did not change the results.

The intra-observer variabilities were as follows: RV Tei index = 3.5 ± 2.2%; RV FAC = 6.8 ± 4.4%; RVOTTs = 4.4 ± 4.6%; and IVA = 4.2 ± 3.4%.

Discussion

The quantitative echocardiographic assessment of RV function is difficult because of the ventricle’s complex trapezoidal anatomy. A wide variety of techniques have been proposed, but none is currently considered the gold standard. In practice, clinicians largely rely on two modalities: two-dimensional echocardiography and DTI echocardiography. From the first modality, ejection fraction (EF), fractional area change (FAC), and TAPSE are considered to reflect global systolic function, and the Tei index reflects both RV systolic and diastolic function, and RVOTTs reflects RV infundibular function. However, all these parameters may vary with load conditions and contractile state.

DTI echocardiography allows quantitative assessment of longitudinal RV function by means of direct measurement of systolic myocardial velocities and by measuring the velocities during the isovolumic contraction period. Vogel et al. have shown in an animal model that isovolumic acceleration is load and shape independent and that IVA correlates positively with global LV and RV contractility. In a study of humans, Kjaergaard et al. showed that RV IVA was unaffected by at least moderate levels of augmented volume and pressure loading. Therefore, it appears that IVA is likely the most trustworthy parameter of RV function. However, in the absence of a gold standard, we decided to utilize multiple parameters to study RV functional changes before and after PTMC. To our knowledge, this is the first study to use tissue Doppler echocardiography to assess RV functional changes in patients with MS acutely after PTMC.

Our results suggest that patients with MS have depressed global and regional RV function compared with normal subjects, and RVOTTs, RV outflow tract fractional shortening, TAPSE, tricuspid annular plane systolic excursion.

Table 1: Echocardiographic data before and immediately after percutaneous mitral commissurotomy (PTMC) and in controls

<table>
<thead>
<tr>
<th></th>
<th>Controls</th>
<th>Before</th>
<th>After</th>
<th>P-valueb</th>
</tr>
</thead>
<tbody>
<tr>
<td>LA antero-posterior diameter (mm)</td>
<td>35 ± 3</td>
<td>47 ± 5.2</td>
<td>41.3 ± 5.1</td>
<td>0.003</td>
</tr>
<tr>
<td>LVEDD (mm)</td>
<td>NS</td>
<td>48.5 ± 2.4</td>
<td>47.9 ± 6.6</td>
<td>49.8 ± 7.7</td>
</tr>
<tr>
<td>LVESD (mm)</td>
<td>NS</td>
<td>28.2 ± 3.4</td>
<td>31.2 ± 6.5</td>
<td>34.1 ± 6.1</td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>NS</td>
<td>71.9 ± 8.4</td>
<td>72.0 ± 8.4</td>
<td>63.3 ± 11.1</td>
</tr>
<tr>
<td>RV diastolic diameter (mm)</td>
<td>NS</td>
<td>23.2 ± 2.80</td>
<td>22.6 ± 3.5</td>
<td>22.2 ± 2.2</td>
</tr>
<tr>
<td>MVA (mm³) [Planimetry]</td>
<td>-</td>
<td>0.91 ± 0.29</td>
<td>1.86 ± 0.43</td>
<td>0.0001</td>
</tr>
<tr>
<td>MVA (mm³) [PHT]</td>
<td>-</td>
<td>0.97 ± 0.38</td>
<td>1.51 ± 0.26</td>
<td>0.0001</td>
</tr>
<tr>
<td>Mean transmitral gradient (mmHg)</td>
<td>-</td>
<td>16.4 ± 8.8</td>
<td>5 ± 1.5</td>
<td>0.001</td>
</tr>
<tr>
<td>Maximum transmitral gradient (mmHg)</td>
<td>-</td>
<td>24.6 ± 11.1</td>
<td>9.7 ± 3.1</td>
<td>0.0001</td>
</tr>
<tr>
<td>PAP (mmHg)</td>
<td>0.01</td>
<td>18.5 ± 1.3</td>
<td>46.4 ± 32.1</td>
<td>29.1 ± 13.4</td>
</tr>
<tr>
<td>Tricuspid inflow</td>
<td>E (cm/s)</td>
<td>NS</td>
<td>60 ± 18</td>
<td>48 ± 10</td>
</tr>
<tr>
<td></td>
<td>A (cm/s)</td>
<td>NS</td>
<td>42 ± 11</td>
<td>47 ± 27</td>
</tr>
<tr>
<td></td>
<td>E/A</td>
<td>NS</td>
<td>1.50 ± 0.48</td>
<td>1.23 ± 0.40</td>
</tr>
</tbody>
</table>

LA, left atrium; LV, left ventricle; MVA, mitral valve area; LVEDD, LV end-diastolic diameter; LVESD, LV end-systolic diameter; LVEF, LV ejection fraction; PAP, systolic pulmonary artery pressure.

aComparison of patients before PTMC and normal subjects.
bComparison of pre- and post PTMC.

Reanalysing the data by omitting the patient presenting with SPAP at 141 mmHg did not change the results.

The intra-observer variabilities were as follows: RV Tei index = 3.5 ± 2.2%; RV FAC = 6.8 ± 4.4%; RVOTTs = 4.4 ± 4.6%; and IVA = 4.2 ± 3.4%.

Discussion

The quantitative echocardiographic assessment of RV function is difficult because of the ventricle’s complex trapezoidal anatomy. A wide variety of techniques have been proposed, but none is currently considered the gold standard. In practice, clinicians largely rely on two modalities: two-dimensional echocardiography and DTI echocardiography. From the first modality, ejection fraction (EF), fractional area change (FAC), and TAPSE are considered to reflect global systolic function, the Tei index reflects both RV systolic and diastolic function, and RVOTTs reflects RV infundibular function. However, all these parameters may vary with load conditions and contractile state.

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Our results suggest that patients with MS have depressed global and regional RV function compared with normal subjects. In the absence of a gold standard, we decided to utilize multiple parameters to study RV functional changes before and after PTMC. To our knowledge, this is the first study to use tissue Doppler echocardiography to assess RV functional changes in patients with MS acutely after PTMC.

Some authors speculate that the rheumatic pathologic process may directly affect the myocardium to cause dys-function. Malhotra et al., in a histo-morphological study of cases of rheumatic heart disease, found that intramyocardial branches of coronary vessels were involved in a form of active rheumatic vasculitis or inactive lesions characterized by medial hypertrophy and replacement fibrosis. They speculated that these changes might affect myocardial function. And the passive increase in left atrial...
pressure and reactive changes in pulmonary arteriolar resistance may lead to increased RV afterload and RV failure. The decrease in RV Tei index and peak pulmonary artery systolic pressure together with the increase in RVOTfs immediately post-PTMC suggest that RV outflow tract systolic function improved as a result of an acute decrease in RV afterload. This is concordant with the study by Borges et al. who demonstrated an improvement in Tei index after vasodilatator therapy in patients with chronic pulmonary hypertension. This is also consistent with the work of Lindqvist et al. who demonstrated that the RVOTfs is sensitive to variation in pulmonary arterial pressure. Hence, the increase in RVOTfs after PTMC may be related to decrease in afterload and not necessarily to improvement in RV function. In our study, we found that there was a positive correlation between Tei index and pulmonary artery systolic pressure before but not after PTMC. The lack of correlation after PTMC may be because of the decrease in RV afterload changing the contractile function of the RV as has been demonstrated by Grignola et al. and/or because the small number of patients creates insufficient power to demonstrate a positive correlation.

The FAC and EF of the RV did not change after PTMC although there was a positive trend in the data ($P = 0.27$ and $P = 0.24$), which might have reached statistical significance with a larger number of study patients. The absence of significant change in FAC and EF following PTMC may reflect that these are measures of change in the RV inflow, not the outflow or reflect simply that these two parameters may be afterload independent.

### Table 3

<table>
<thead>
<tr>
<th>Tricuspid annulus</th>
<th>$P^a$</th>
<th>Controls</th>
<th>Before</th>
<th>After</th>
<th>$P^b$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Septal</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IVp (cm/s)</td>
<td>NS</td>
<td>6.24 ± 1.87</td>
<td>6.24 ± 1.38</td>
<td>6.41 ± 2.15</td>
<td>NS</td>
</tr>
<tr>
<td>Sr (cm/s)</td>
<td>0.007</td>
<td>8.54 ± 1.20</td>
<td>6.43 ± 1.81</td>
<td>6.18 ± 1.52</td>
<td>NS</td>
</tr>
<tr>
<td>Er (cm/s)</td>
<td>0.02</td>
<td>11.01 ± 2.91</td>
<td>6.83 ± 3.24</td>
<td>7.21 ± 2.09</td>
<td>NS</td>
</tr>
<tr>
<td>Ar (cm/s)</td>
<td>0.025</td>
<td>9.68 ± 2.51</td>
<td>6.36 ± 2.03</td>
<td>5.41 ± 1.30</td>
<td>NS</td>
</tr>
<tr>
<td>$E_r/A_r$</td>
<td>NS</td>
<td>1.23 ± 0.52</td>
<td>1.17 ± 0.53</td>
<td>1.40 ± 0.46</td>
<td>NS</td>
</tr>
<tr>
<td>IVA (m/s$^2$)</td>
<td>NS</td>
<td>0.21 ± 0.08</td>
<td>0.19 ± 0.05</td>
<td>0.18 ± 0.06</td>
<td>NS</td>
</tr>
<tr>
<td>Lateral</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IVp (cm/s)</td>
<td>0.02</td>
<td>15.25 ± 4.97</td>
<td>11.03 ± 3.37</td>
<td>8.50 ± 2.04</td>
<td>0.03</td>
</tr>
<tr>
<td>Sr (cm/s)</td>
<td>0.02</td>
<td>14.58 ± 2.07</td>
<td>11.69 ± 2.37</td>
<td>11.25 ± 3.14</td>
<td>NS</td>
</tr>
<tr>
<td>Er (cm/s)</td>
<td>NS</td>
<td>10.76 ± 3.50</td>
<td>11.12 ± 3.85</td>
<td>11.72 ± 4.31</td>
<td>NS</td>
</tr>
<tr>
<td>Ar (cm/s)</td>
<td>0.04</td>
<td>16.70 ± 3.46</td>
<td>13.08 ± 4.09</td>
<td>11.63 ± 3.35</td>
<td>NS</td>
</tr>
<tr>
<td>$E_r/A_r$</td>
<td>NS</td>
<td>0.68 ± 0.32</td>
<td>0.95 ± 0.50</td>
<td>1.09 ± 0.35</td>
<td>NS</td>
</tr>
<tr>
<td>IVA (m/s$^2$)</td>
<td>NS</td>
<td>0.40 ± 0.15</td>
<td>0.36 ± 0.11</td>
<td>0.25 ± 0.07</td>
<td>0.02</td>
</tr>
</tbody>
</table>

$A_r$, late diastolic velocity; $E_r$, early diastolic velocity; IVA, myocardial acceleration during isovolumic contraction; IVp, isovolumic contraction velocity; Sr, systolic velocity during ejection period.

$^a$Comparison of patients before PTMC and normal subjects.

$^b$Comparison of patients pre- and post PTMC.

![Figure 1](image1.png)  
**Figure 1** Changes in right ventricular Tei index before and after percutaneous mitral commissurotomy (PTMC). Difference assessed by paired $t$-test. Mean observations before and after PTMC as horizontal line ± 1SD.

![Figure 2](image2.png)  
**Figure 2** Changes in myocardial acceleration during isovolumic contraction (IVA) at lateral side of the tricuspid annulus before and after percutaneous mitral commissurotomy (PTMC). Difference assessed by paired $t$-test. Mean observations before and after PTMC as horizontal line ± 1SD.
Figure 3 Changes in isovolumic contraction velocities (IVC) at lateral side of the tricuspid annulus before and after percutaneous mitral commissurotomy (PTMC). Difference assessed by paired t-test. Mean observations before and after PTMC as horizontal line ± 1SD.

We also noted that myocardial acceleration during isovolumic contraction and isovolumic velocities on the lateral side of the tricuspid annulus decreased after PTMC. This decrease immediately after PTMC, may actually reflect the acute decrease in RV afterload with its consequence on contractile function of the RV as has been demonstrated by Grignola et al. Thus, we believe that changes in IVA reflect most probably changes in contractile state rather than afterload dependency. Also, one could suspect the influence of sympathetic tone in the reduction of IVA after PTMC; however, in our study the heart rate remained stable after the procedure. The reason why systolic velocities during ejection period at the lateral tricuspid annulus remained stable after PTMC, whereas IVA and isovolumic contraction velocities decreased may be related to the fact that parameters illustrating isovolumic contraction period may be more sensitive markers of myocardial function than systolic velocities during the ejection period. In fact, Edvardsen et al. demonstrated that disturbances of myocardial motion occur predominantly during the isovolumic phases. Therefore, we believe that the decrease in IVA and isovolumic contraction velocities at the lateral side of the tricuspid annulus may indicate a reduction in global RV contractility following PTMC. In effect, Lysegeen et al. demonstrated that myocardial acceleration is more representative of global ventricular contractility than regional contractility. The opposite response of the IVA and the Tei index may be because the Tei index is more sensitive to loading conditions than IVA and, hence, its improvement is not necessarily related to RV function improvement.

The significance of reduced RV function as assessed by IVA, immediately after PTMC, remains unclear and follow-up studies with larger numbers of patients are needed to assess whether this finding has any prognostic implications. However, our finding of a decrease in RV contractility, as assessed by IVA, may be clinically useful in prompting further diagnostic evaluation for patients with MS.

Finally, even though our study was not designed to examine LV function, we noted that mean LVEF of our patients and normal controls was supranormal possibly because we selected only young patients and young controls with sinus rhythm. After PTMC the ejection fraction showed no significant change implying that the overall LV function was unchanged.

The low intra-observer variability from remeasurements of RV Tei index, FAC, RVOTfs, and IVA reflect acceptable reproducibility in our measurements.

Limitations

The main limitation of our study is the small number of patients examined. This was due to the exclusion criteria, which aimed to limit the number of confounding factors that might interfere with RVF. Nevertheless, we report several significant observations.

Conclusion

Immediately after successful PTMC, significant decrease in RV contractility as assessed by IVA was observed, whereas other parameters of infundibular and global RV function as assessed by RVOTfs and Tei index showed significant improvement. These discordant results may be related to relative insensitivity of currently utilized echocardiography parameters of RV function, which may not be completely immune to loading conditions. Further work using larger numbers of patients is needed to confirm our findings and to assess their utility in patient follow-up and management.

Conflict of interest: none declared.

References