Right and left ventricular mechanics and interaction late after balloon valvoplasty for pulmonary stenosis

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Aims

This study sought to explore right (RV) and left ventricular (LV) mechanics and ventricular–ventricular interaction in adolescents and young adults late after percutaneous balloon pulmonary valvoplasty (PBPV) for valvar pulmonary stenosis (PS).

Methods and results

Potential late effects of PS despite PBPV on cardiac mechanics have not been well defined. Thirty-one patients aged 20.2 ± 7.6 years were studied at 18 ± 6 years after PBPV. Ventricular myocardial deformation was determined using speckle tracking echocardiography, while RV and LV volumes and ejection fraction as well as LV systolic dyssynchrony index were assessed by three-dimensional echocardiography. The results were compared with those of 30 controls. Pulmonary regurgitation, mostly trivial to mild, was present in 90% (28/31) of patients. Compared with controls, patients had significantly greater RV end-diastolic (P < 0.001), RV end-systolic (P < 0.001), and LV end-systolic (P = 0.04) volumes as well as lower LV ejection fraction (P < 0.001). For deformation, patients had significantly reduced RV longitudinal systolic strain (P = 0.004), decreased LV circumferential systolic strain (P < 0.001), and strain rate (P = 0.001) as well as greater LV mechanical dyssynchrony (P < 0.001). In patients, RV end-diastolic and end-systolic volumes correlated with LV circumferential strain (r = −0.47, P = 0.008 and r = −0.36, P = 0.049, respectively) and dyssynchrony (r = 0.53, P = 0.002 and r = 0.49, P = 0.005, respectively). Patients who had PBPV at age ≤1 year had ventricular deformation indices similar to those who had interventions beyond 1 year.

Conclusion

Impaired RV and LV mechanics and adverse ventricular–ventricular interaction occur in adolescents and young adults late after balloon valvoplasty for isolated PS.

Keywords

Pulmonary stenosis • Percutaneous balloon pulmonary valvoplasty • Ventricular mechanics • Ventricular interaction

Introduction

Percutaneous balloon pulmonary valvoplasty (PBPV) is the treatment of choice for significant valvar pulmonary stenosis (PS). Since its first report of success by Kan et al.,1 this technique has been widely adopted and shown satisfactory long-term outcomes in terms of successful relief of right ventricular (RV) outflow obstruction.2—4 Previous studies have focused primarily on residual or recurrence of PS in infants and children who had undergone the procedure.2—6 In obstructive lesions of the RV outflow including tetralogy of Fallot7,8 and pulmonary atresia with intact ventricular septum,9,10 abnormal RV mechanics and ventricular–ventricular interaction late after surgical intervention have been shown. Potential late effects of PS despite PBPV on RV and left ventricular (LV) mechanics have, however, not been well defined. In this study, we aimed to explore the RV and LV mechanics and RV–LV interaction in adolescents and young adults late after PBPV.

Methods

Subjects

Patients who had undergone PBPV for isolated PS and fulfilled the following criteria were recruited: (i) aged 10 years and above, (ii) at least a 10-year follow-up duration since PBPV, and (iii) residual PS gradient < 30 mmHg. Exclusion criteria included history of systemic-to-pulmonary arterial shunt insertion and RV outflow surgical reconstruction and...
additional cardiac lesions that may cause RV dilation. Balloon valvoplasty
was performed as reported previously, and a standard balloon-to-pulmonary valve annulus ratio of 1.2:1.3 was used in our institution.
The following data were retrieved from the case notes: age at first
and subsequent PBPV, the need for re-intervention, and duration of
follow-up.

Thirty-two patients from our congenital heart database fulfilled the
criteria and were recruited. One patient was pregnant and eventually 31
patients were included in the final analysis. Thirty healthy subjects were
recruited as controls. These included healthy volunteers, subjects identi-
fied in the cardiac clinic with functional murmur, non-specific chest pain,
and palpitation without an organic cause, and healthy siblings of patients.

The body weight and height were measured, and the body surface area
was calculated accordingly. All subjects underwent echocardiographic
assessment as described below. The Institutional Review Board approved
the study, and all adult subjects and parents of minors gave informed
written consent.

Conventional and Doppler echocardiographic
assessments

Echocardiographic assessments were performed using the Vivid 7 ultra-
sound system (GE Vingmed Ultrasound AS, Horten, Norway). The
averages of echocardiographic indices measured from three cardiac
cycles were obtained for statistical analysis.

M-mode assessment from the parasternal short-axis view at the
papillary muscle level was performed to measure LV end-systolic and
end-diastolic dimensions and shortening fraction. Severity of pulmonary
regurgitant (PR) and tricuspid regurgitation was assessed semi-
quantiatively by Doppler colour flow mapping. Pulsed-wave Doppler
examination was performed from the apical four-chamber view to
obtain transmitral and tricuspid early (E) and late (A) diastolic inflow veloc-
ties and E/A ratio. Tissue Doppler echocardiography was performed with
the sample volume positioned at the basal LV and RV free wall–atrioventric-
ular annular junction to obtain the following indices: peak systolic (s),
early (e) and late (a) diastolic myocardial velocity, e/a ratio, and E/e ratio.

Speckle tracking echocardiography

Mechanics of the RV and LV were assessed by two-dimensional speckle
tracking echocardiography (STE) as reported previously. Two-
dimensional echocardiographic recordings were analysed offline us-
ing the two-dimensional strain software (EchoPAC, GE Vingmed
Ultrasound).

Global RV and LV longitudinal systolic strain and systolic and diastolic
strain rates were determined from the apical four-chamber view, while

Figure 1: Representative RV longitudinal and LV circumferential strain curves in a patient and a control subject. Regional volume curves (lower
panel), normalized to individual maximum, demonstrate dyssynchronous contraction of multiple segments in a patient compared with synchronous
contraction in the control subject.
LV circumferential and radial strain and strain rates were assessed from the parasternal short-axis view at the papillary muscle level (Figure 1). RV and LV free walls and ventricular septum were divided, respectively, into three segments (basal, mid, and apical) for quantification of regional longitudinal strain. As the ventricular septum contributes to both RV and LV global function,16 it was included in the assessment of both the RV and LV global strain and strain rate. The LV short axis was divided into six segments (anteroseptal, anterior, lateral, posterior, inferior, and septal) for assessment of regional circumferential and radial strain. Our group has previously reported high reproducibility for measurement of ventricular strain and strain rate by two-dimensional STE.14,17

Real-time three-dimensional echocardiography

Full-volume RV and LV datasets were acquired from the apical four-chamber view using the matrix array transducer and analysed offline by the commercial 4D analysis software (Tomtec Imaging Systems, Unterschleissheim, Germany). The end-systolic and -diastolic volumes and ejection fraction of both ventricles and LV systolic dysynchrony index (SDI) were determined, as reported previously.8 The PR volume in patients was estimated by calculating the difference between RV and LV stroke volumes.

Statistical analysis

Data are expressed as mean ± SD and median with range where appropriate. Absolute values of strain and SR were used to facilitate interpretation and analysis. Ventricular and PR volumes were indexed by body surface area. Differences in demographic, clinical, and echocardiographic parameters between groups were compared using unpaired Student’s t-test and Fisher’s exact test where appropriate. Pearson’s correlation analysis was used to study relationships between PR and RV volumes, and between RV volumes and LV parameters. A P-value of <0.05 was considered statistically significant. All statistical analyses were performed using SPSS version 16.0 (SPSS, Inc., Chicago, IL, USA).

Results

Subjects

The 31 (19 males) patients were studied at a median age of 19 years (range, 10–39 years). They underwent PBPV at a median of 0.7 years (range, 2 days to 12 years) and were followed up for 18 ± 6 years (median 17 years, range, 10–29 years) after catheter intervention. Balloon valvoplasty was performed in the neonatal period in 10, at infancy in 8, and beyond 1 year in 13 patients. Four of the patients required a second PBPV. All the patients were free of cardiac symptoms at the time of study.

The age (20.2 ± 7.6 vs. 19.7 ± 7.6 years, P = 0.75), sex distribution (16/15F vs. 15/15F, P = 0.9), body weight (55 ± 16 vs. 54 ± 9 kg, P = 0.71), and height (162 ± 11 vs. 163 ± 11 cm, P = 0.79) were similar between patients and controls.

RV size and mechanics

Tricuspid regurgitation was mild to moderate in 1, trivial to mild in 25, and absent in 5 patients. Pulmonary regurgitation was present in 90% (28/31) of patients, being trivial to mild, as characterized by thin, small PR jets with a pin-point origin, in 25 and moderate in 3 patients. None of the patients had severe PR.

Table 1 summarizes the RV Doppler and three-dimensional (3D) echocardiographic parameters in patients and controls. Compared with controls, patients had significantly greater tricuspid inflow A velocity (P = 0.001), lower E/A ratio (P = 0.01), and greater E/e ratio (P = 0.01). The tricuspid annular s velocity also tended to be lower in patients than in controls (P = 0.05).

Three-dimensional echocardiography revealed significantly greater RV end-diastolic (P < 0.001) and end-systolic (P < 0.001) volumes in patients than in controls. The RV ejection fraction also tended to be lower in patients than in controls (P = 0.07). In 26% (8/31) of patients, the RV ejection fraction was <50%. In contrast, RV ejection fraction <50% was found only in 10% (3/30) of control subjects (P = 0.11).

The RV global and regional strain parameters in patients and controls are shown in Figure 2. Compared with controls, patients had significantly lower global RV longitudinal systolic strain (25.4 ± 3.8 vs. 27.8 ± 2.2%, P = 0.004), while systolic and diastolic strain rates were similar between the two groups (all P > 0.05). Region analysis showed significantly lower strain in the mid (29.9 ± 6.1 vs. 33.8 ± 4.7%, P = 0.01) and apical (23.2 ± 8.7 vs. 27.8 ± 6.0%, P = 0.02) segments of the RV free wall. There were no significant correlations between RV strain parameters and RV end-systolic or -diastolic volumes (all P > 0.05).

LV mechanics

Table 2 summarizes the LV echocardiographic parameters in patients and controls. The M-mode-derived LV dimensions and shortening fraction were similar between patients and controls (all P > 0.05). Doppler studies showed significantly greater mitral inflow A velocity (P = 0.01) and annular a velocity (P < 0.001), and lower E/A (P = 0.01) and e/a ratios (P < 0.001) in patients than in controls.

Table 1 Right ventricular Doppler and 3D echocardiographic parameters

<table>
<thead>
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<th>Patients (n = 31)</th>
<th>Controls (n = 30)</th>
<th>P-value</th>
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<tr>
<td>Tricuspid inflow pulsed-wave Doppler velocities</td>
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<td>E (cm/s)</td>
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<td>A (cm/s)</td>
<td>38.4 ± 12.4</td>
<td>29.5 ± 7.1</td>
<td>0.001*</td>
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<td>1.7 ± 0.4</td>
<td>2.1 ± 0.5</td>
<td>0.01*</td>
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<td>Tricuspid annular tissue Doppler parameters</td>
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<tr>
<td>s (cm/s)</td>
<td>13.1 ± 2.1</td>
<td>14.1 ± 1.7</td>
<td>0.05</td>
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<td>e (cm/s)</td>
<td>15.4 ± 3.6</td>
<td>16.4 ± 2.4</td>
<td>0.23</td>
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<tr>
<td>a (cm/s)</td>
<td>10.7 ± 3.2</td>
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<td>e/a ratio</td>
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<td>E/e ratio</td>
<td>4.2 ± 1.2</td>
<td>3.6 ± 0.5</td>
<td>0.01*</td>
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<td>3D echocardiographic parameters</td>
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<td>End-diastolic volume (mL/m²)</td>
<td>64.7 ± 14.9</td>
<td>52.9 ± 8.4</td>
<td>&lt;0.001*</td>
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<tr>
<td>End-systolic volume (mL/m²)</td>
<td>30.2 ± 7.1</td>
<td>23.7 ± 4.7</td>
<td>&lt;0.001*</td>
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<td>Ejection fraction (%)</td>
<td>53.1 ± 5.1</td>
<td>55.2 ± 3.6</td>
<td>0.07</td>
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A, late diastolic inflow velocity; a, late diastolic myocardial annular velocity; E, early diastolic inflow velocity; e, early diastolic myocardial annular velocity; s, systolic myocardial annular velocity.

*Statistically significant.
For 3D echocardiographic assessment, patients were found to have significantly greater LV end-systolic volume ($P = 0.04$), lower ejection fraction ($P < 0.001$), and greater SDI ($P < 0.001$) than controls. In 55% (17/31) of patients, the LV ejection fraction was $< 50\%$. In contrast, LV ejection fraction $< 50\%$ was only found in 3.3% (1/30) of control subjects ($P < 0.001$).

The strain parameters of the LV are shown in Figure 3. Patients had significantly lower global systolic strain ($14.1 \pm 3.2$ vs. $17.0 \pm 2.4\%$, $P < 0.001$) and strain rate ($0.85 \pm 0.2/s$ vs. $1.0 \pm 0.2/s$, $P = 0.001$) only in the circumferential dimension, but not in the longitudinal and radial dimensions. Regional analyses revealed a significant reduction in strain of the basal ($18.8 \pm 3.1$ vs. $21.2 \pm 2.3\%$, $P = 0.001$) and mid- ($21.1 \pm 3.0$ vs. $23.0 \pm 2.3\%$, $P = 0.01$) septum from the four-chamber view, and anteroseptal ($19.3 \pm 6.5$ vs. $23.2 \pm 4.0\%$, $P = 0.01$) and anterior ($14.0 \pm 5.4$ vs. $17.5 \pm 4.1\%$, $P = 0.01$) segments from the mid-ventricular short axis.

**Figure 2:** Comparisons of RV longitudinal systolic strain and systolic and diastolic strain rates between patients and controls. SRa, late diastolic strain rate; SRe, early diastolic strain rate; SRs, systolic strain rate. *$P < 0.05$ vs. controls.
The LV ejection fraction correlated negatively with SDI ($r = -0.36, P = 0.045$) and positively with global circumferential strain ($r = 0.37, P = 0.04$) (Figure 4).

**Timing of intervention and ventricular mechanics**

To determine the influence of age at intervention on late ventricular mechanics, echocardiographic parameters of the 18 patients aged 17.1 ± 5.4 years who had undergone PBPV at ≤1 year were compared with the 13 patients aged 24.6 ± 8.1 years who had interventions beyond 1 year of age. Despite the difference in age at the time of study ($P = 0.004$), the two patient subgroups had similar follow-up durations (16.8 ± 5.4 vs. 18.5 ± 6.4 years, $P = 0.45$) and indices of RV and LV mechanics (Table 3).

**RV–LV interaction**

In patients, the PR volume was 15.2 ± 10 mL/m$^2$/beat. For the patient cohort, PR volume correlated positively with both RV end-diastolic ($r = 0.78, P < 0.001$) and -systolic ($r = 0.57, P = 0.001$) volumes (Figure 4).

Among patients, LV global circumferential strain correlated negatively with RV end-diastolic ($r = -0.47, P = 0.008$) and -systolic ($r = -0.36, P = 0.049$) volumes, while LV SDI correlated positively with both RV end-diastolic ($r = 0.53, P = 0.002$) and -systolic ($r = 0.49, P = 0.005$) volumes (Figure 4).

**Discussion**

The present study demonstrates impairment of both RV and LV mechanics and provides evidence of adverse ventricular—ventricular interaction in adolescents and young adults late after balloon valvoplasty for isolated valvar PS. To our knowledge, this is the first study to evaluate comprehensively the mechanical performance of both ventricles in patients with valvar PS followed up for almost three decades after successful catheter intervention.

**Pulmonary regurgitation after balloon valvoplasty**

Our findings of mild PR in the majority and moderate PR in ~10% of patients agree with those reported previously. $^{18-20}$ Given the risk of the use of oversized balloons, $^{21}$ the adoption of a balloon-to-annulus ratio of 1.2–1.3 in our institution has probably prevented the occurrence of severe PR in our patients in the long term. Nonetheless, the RV volume as assessed by 3D echocardiography remains larger in these patients compared with controls. RV dilation has also been found in patients after PBPV using cardiac magnetic resonance (CMR), $^{19}$ although 34% of patients in this study had PR fraction >15% and ~15% had PR fraction >30%. The previous findings may not therefore entirely apply in the present cohort. Notwithstanding the mild residual PR found in our patients, valvar PS results in residual differences in ventricular mechanics even after balloon dilation.

**RV mechanics**

Little is known about long-term RV function in patients with isolated valvar PS after PBPV. In patients studied at a median of 13 years after PBPV, Harrild et al. $^{19}$ found that although RV ejection fraction remains within limits of normal, it has modest correlation with PR fraction. Similarly, we found relatively preserved RV ejection fraction in our patients, although the mean value tended to be lower than that of controls. On the other hand, strain imaging, which is more sensitive in the detection of early subtle myocardial abnormalities, $^{22}$ unveils impairment of RV global systolic strain in patients. Regional analyses further show that the global impaired is contributed by reduced systolic deformation of both the RV free wall and the ventricular septum.

Although reduction in RV strain may be adaptive response to RV dilation, an increase in RV output related to pulmonary with or without tricuspid regurgitation is expected to increase myocardial deformation. The tricuspid annular s velocity tended to be lower in patients than in controls (Table 1). Furthermore, we did not find significant correlations between RV systolic strain and RV volumes. Hence, our findings may reflect subclinical RV systolic dysfunction in patients. While global diastolic strain rates were similar between patients and controls, the greater RV E/e ratio in patients may suggest occurrence of severe PR in our patients in the long term. Nonetheless, the RV volume as assessed by 3D echocardiography remains larger in these patients compared with controls. RV dilation has also been found in patients after PBPV using cardiac magnetic resonance (CMR), $^{19}$ although 34% of patients in this study had PR fraction >15% and ~15% had PR fraction >30%. The previous findings may not therefore entirely apply in the present cohort. Notwithstanding the mild residual PR found in our patients, valvar PS results in residual differences in ventricular mechanics even after balloon dilation.

**Table 2** LV M-mode, Doppler, and 3D echocardiographic parameters

<table>
<thead>
<tr>
<th>Patients (n = 31)</th>
<th>Controls (n = 30)</th>
<th>P-value</th>
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<tr>
<td>M-mode parameters</td>
<td></td>
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<tr>
<td>LVEDD (mm/m²)</td>
<td>27.9 ± 3.7</td>
<td>29.1 ± 2.2</td>
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<tr>
<td>LVESD (mm/m²)</td>
<td>18.0 ± 2.6</td>
<td>18.4 ± 1.6</td>
</tr>
<tr>
<td>Shortening fraction (%)</td>
<td>35.3 ± 3.3</td>
<td>36.7 ± 3.3</td>
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<td>Mitral inflow pulsed-wave Doppler velocities</td>
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<tr>
<td>E (cm/s)</td>
<td>87.7 ± 18.8</td>
<td>90.6 ± 15.8</td>
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<tr>
<td>A (cm/s)</td>
<td>49.6 ± 14.4</td>
<td>41.4 ± 10.0</td>
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<tr>
<td>E/A ratio</td>
<td>1.9 ± 0.6</td>
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<td>Mitral annular tissue Doppler parameters</td>
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<td>s (cm/s)</td>
<td>13.1 ± 2.8</td>
<td>11.9 ± 2.1</td>
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<td>e (cm/s)</td>
<td>17.4 ± 2.8</td>
<td>18.2 ± 2.6</td>
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<td>a (cm/s)</td>
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<td>e/a ratio</td>
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<td>E/e ratio</td>
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<td>3D echocardiographic parameters</td>
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<td>End-diastolic volume (mL/m²)</td>
<td>38.2 ± 8.3</td>
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<tr>
<td>End-systolic volume (mL/m²)</td>
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<td>Ejection fraction (%)</td>
<td>50.1 ± 5.8</td>
<td>56.4 ± 4.0</td>
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<td>Systolic dyssynchrony index (%)</td>
<td>6.9 ± 1.9</td>
<td>4.6 ± 0.8</td>
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LVEDD, left ventricular end-diastolic dimension; LVESD, left ventricular end-systolic dimension, other abbreviations as in Table 1. *Statistically significant.
differences in ventricular mechanics between patients who had interventions at ≤1 year and those beyond. In future studies, it is worthwhile assessing ventricular fibrosis by late gadolinium enhancement CMR in these patients.

**LV mechanics and ventricular–ventricular interaction**

A novel finding of the present study is impairment of LV mechanics as characterized by the significantly lower circumferential systolic strain and strain rate, lower ejection fraction, and greater SDI after PBPV. Furthermore, associations between greater RV volume load and worse LV circumferential strain and mechanical dyssynchrony suggest adverse RV–LV interaction. Preferential involvement of septal segments provides support to the hypothesis that altered septal deformation due to RV dilation, albeit mild, in patients after PBPV may play a contributory role. This mechanism has also been implicated in patients with RV volume overloading after repair of tetralogy of Fallot.5,24,25 While LV twisting mechanics were not assessed in the present study, decreased LV apical rotation and twist in relation

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**Figure 3**: Comparisons of LV systolic longitudinal, radial and circumferential strain and systolic and diastolic strain rates between patients and controls. Ant, anterior; Antsept, anteroseptal; Inf, inferior; Lat, lateral; Post, posterior; Sept, septal, other abbreviations as in Figure 2. *P* < 0.05 vs. controls.
to, respectively, reduced RV apical strain and global strain have been documented in Fallot patients. Hence, the LV twisting mechanics in patients after balloon valvoplasty for PS warrants further studies.

The LV ejection fraction as derived from 3D echocardiography was <50% in about half of our patients. On the other hand, the recent CMR study reported normal LV ejection fraction in patients studied at a median time interval from balloon valvoplasty of ≈13 years. The cause of discrepancy is not entirely clear. However, our patients are older and have been followed up for a longer duration. Underestimation of LV volumes and ejection fraction by 3D echocardiography has, however, been well documented, although the bias in estimation of ejection fraction is smaller. Direct comparisons of absolute values between the two imaging modalities hence require cautious interpretation. Notwithstanding, our patients did exhibit significantly reduced LV ejection fraction compared with age-matched control subjects. Similarity in LV shortening fraction between patients and controls may be related to the limited M-mode assessment of only two LV segments, in contrast to the global evaluation of all LV segments by either 3D echocardiography or CMR.

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Clinical implications
Impairment of exercise capacity has been reported in patients with PS after PBPV. It is worthwhile noting that patients with worse PR were found to have significantly lower peak oxygen consumption. The link between mechanics of the two ventricles and exercise capacity in these patients requires further investigations. The need for pulmonary valve replacement has been reported in patients after surgical pulmonary valvotomy and in those after oversized balloon

Table 3 Ventricular mechanics and age at catheter intervention

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<tr>
<th>Tissue Doppler imaging parameters</th>
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<tr>
<td>s (cm/s)</td>
<td>12.9 ± 2.5</td>
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<td>e (cm/s)</td>
<td>15.8 ± 4.0</td>
<td>15.0 ± 3.0</td>
<td>18.3 ± 2.8</td>
<td>16.0 ± 2.8</td>
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<tr>
<td>a (cm/s)</td>
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<td>11.2 ± 2.9</td>
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Longitudinal deformation

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<tr>
<td>LV SDI (%)</td>
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<tr>
<td>RV EDV (ml/m²)</td>
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<td>LV circumferential strain (%)</td>
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SRa, late diastolic strain rate; SRe, early diastolic strain rate; SRs, systolic strain rate, other abbreviations as in Table 1.

All P > 0.05.
Valvuloplasty. It is reassuring that none of our patients had an estimated RV volume exceeding the recommended criteria for pulmonary valve replacement after repair of tetralogy of Fallot. Nonetheless, serial monitoring of RV volume load of these patients into their late adulthood is needed to provide more light on the need for pulmonary valve replacement in the long term with the contemporary strategy of balloon valvuloplasty. To date, ACC/AHA and ESC guidelines for the management of adults with PS after balloon valvuloplasty have focused on assessment of residual pressure gradient and PR without emphasis on potential LV dysfunction. Our findings of impaired LV mechanics, in addition to RV abnormalities, may perhaps provide a basis for recommending regular surveillance of not only RV, but also LV function, in this patient population.

Limitations
Several limitations to this study warrant comments. First, the cross-sectional nature of the present study does not provide information on the time course of development of impaired ventricular mechanics and prognostic implications. Secondly, the possible underestimation by 3D echocardiography of ventricular volume and ejection fraction has been alluded to earlier. Nonetheless, 3D echocardiographic indices have been found to be reproducible and to correlate strongly with CMR findings. Rendering them useful in the longitudinal follow-up of patients, CMR, however, enables assessment of individual RV components, which may have a differential response to volume load as reported in patients with repaired tetralogy of Fallot. Thirdly, PR was indirectly estimated by the difference in RV and LV stroke volumes as determined by 3D echocardiography. The underestimation of ventricular volumes may potentially be different between the right and left side. The estimation of PR in patients might further be confounded by coexistence of tricuspid regurgitation, although the impact is likely to be small as both one of the patients had either none or minimal tricuspid regurgitation.

Conclusions
Impaired RV and LV mechanics and adverse ventricular–ventricular interaction occur in adolescents and young adults late after balloon valvuloplasty for isolated valvar PS. Further longitudinal studies to determine the prognostic implications of altered ventricular mechanics in these patients are warranted.

Conflict of interest: none declared.

References
Multiple pseudoaneurysms of aortic arch in a patient with Behcet’s disease

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A-24-year-old male was admitted to our clinic because of congestive heart failure. He was diagnosed with Behcet’s disease. Systolic and diastolic murmur was heard in all cardiac areas. Transthoracic echocardiography (five-chamber view) showed aortic regurgitation, mitral regurgitation, a fistula between aorta and left atrium, and dilatation of right aortic sinus (Panel and see Supplementary data online, Video S1A). Full-volume three-dimensional transthoracic echocardiography also revealed dilatation of right aortic sinus (Panel and see Supplementary data online, Video S1B). To clarify this pathology, we performed cardiac computed tomography (CT). Horizontal (Panel C), coronal (Panel D) subvolume maximum intensity projection, and three-dimensional coloured volume rendered (Panel E) CT angiography images displayed multiple pseudoaneurysms in different areas including ascending aorta adjacent to right coronary artery, between aorta and pulmonary artery, brachiocephalic artery, and right common carotid artery.

Aortic pseudoaneurysms most frequently arise from surgical suture lines, but can also result from genetic disorders, infection, or trauma. The underlying pathological mechanism is a weakening of the intima and media of the aorta. Aortic pseudoaneurysms typically grow over time, which can lead to aortic rupture. Therefore, it should be treated surgically at the earliest. We decided to treat him medically because of high surgical mortality. We herein present a case of multiple pseudoaneurysms in an adult patient with Behcet’s disease using multimodality imaging. In the presence of aortic root dilatation, the full spectrum of non-invasive cardiac imaging modalities should be performed in the diagnosis of pseudoaneurysms. Ao, aorta; PA, pulmonary artery; asterisk, pseudoaneurysm; arrow, fistula.

Supplementary data are available at European Heart Journal – Cardiovascular Imaging online.

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