Impact of Transcatheter Pulmonary Valve Replacement on Biventricular Strain and Synchrony Assessed by Cardiac Magnetic Resonance Feature Tracking

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Background—Transcatheter pulmonary valve (TPV) replacement is an emerging therapy intended to restore pulmonary valve function in patients with right ventricular outflow tract conduit dysfunction; the impact of this technique on ventricular strain and synchrony is not known.

Methods and Results—Cardiac magnetic resonance and ECG data acquired at 1 center as part of the US Melody TPV trial were analyzed. Biventricular strain and mechanical synchrony measurements were made based on short-axis and 4-chamber steady-state free precession images using feature tracking software. Post- versus pre-TPV replacement findings were compared for all patients (n=31) and subgroups with predominant pulmonary regurgitation (n=13) or stenosis (n=18). Most patients had tetralogy of Fallot (18/31). After TPV replacement, left ventricular (LV) circumferential strain increased for the whole cohort (P<0.001) and both subgroups (pulmonary regurgitation P=0.01; pulmonary stenosis P=0.02). LV longitudinal strain increased for the whole cohort (P=0.02) and pulmonary regurgitation subgroup (P=0.05); circumferential right ventricular strain increased for the pulmonary stenosis group only (P=0.05). LV longitudinal synchrony improved significantly in the pulmonary regurgitation group (maximum wall delay P=0.03; cross-correlation delay P=0.01). Electric measures of synchrony did not improve.

Conclusions—In patients with right ventricular outflow tract conduit dysfunction, TPV replacement is associated with improved global LV strain, as well as improved right ventricular strain and LV synchrony in subgroups. Given the associations between strain and synchrony and clinical outcomes, these findings support potential long-term benefits of TPV replacement. (Circ Cardiovasc Interv. 2013;6:680-687.)

Key Words: magnetic resonance imaging ■ pulmonary valve ■ tetralogy of Fallot

Patients with chronic pulmonary regurgitation (PR), including those with repaired tetralogy of Fallot (TOF), have adverse long-term outcomes including exercise intolerance, arrhythmia, and death. Efforts to mitigate these outcomes have focused on restoration of normal pulmonary valve function. Traditional methods of pulmonary valve replacement have been surgical. Recently, however, a technique for transcatheter implantation of a stent-mounted bovine jugular venous valve (Melody valve, Medtronic Inc., Minneapolis, MN) in the pulmonary position has been described for the treatment of PR and right ventricular outflow tract (RVOT) obstruction. Early- and midterm results of the US trial of the Melody valve have been encouraging; implantation has been performed with a low burden of morbidity and mortality and has led to a marked and lasting reduction in PR and RVOT obstruction.

Data describing the impact of transcatheter pulmonary valve (TPV) replacement on ventricular mechanics are limited. With few exceptions, echocardiographic studies have reported traditional measures such as ventricular size and global systolic function. Cardiac magnetic resonance (CMR) imaging is frequently used in this patient population to better evaluate RV function and PR given the concerns for poor acoustic windows. CMR data regarding the impact of TPV replacement have also been limited to the assessment of traditional measures of ventricular size and global function.

Speckle-tracking–based strain is a newer index of ventricular performance that measures local myocardial deformation. Strain-based techniques allow for the quantification of both regional myocardial function and measures of ventricular synchrony (eg, segmental difference in time to peak strain). Strain and strain-derived synchrony have been shown to be associated with adverse outcomes in patients with structurally normal hearts as well as those with repaired congenital heart disease; moreover, to this end, they may be more sensitive indicators than ejection fraction (EF). New CMR-based methods for assessing ventricular strain have recently been described. To our knowledge, no CMR-based investigations have reported the impact of TPV replacement on ventricular strain, and there

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are no reports of any kind of its impact on synchrony of the left ventricle (LV) or RV. The goal of the present study was to quantify the impact of TPV replacement on indices of RV and LV strain and synchrony in patients with dysfunctional RVOT conduits using high-quality CMR images.

**Methods**

**Study Design**

This was a retrospective cohort study of patients undergoing placement of a Melody TPV in the setting of a dysfunctional RVOT conduit designed to investigate changes in ventricular mechanics based on CMR images before and after pulmonary valve implantation. This study was performed according to a protocol approved by the Committee for Clinical Investigations at Boston Children’s Hospital, Boston, MA.

**Patients**

Patients included in this study comprised a subset of the cohort enrolled at our center as part of the multicenter US Melody TPV investigational device exemption (IDE) trial. Briefly, the IDE trial was designed to investigate the safety, procedural success, and short-term effectiveness of Melody valve placement in patients with a biventricular circulation and a dysfunctional RVOT conduit. Study protocols and short-term outcomes have been previously reported. As part of their participation in the trial, patients had clinical, ECG, and CMR data collected before and 6±2 months after TPV replacement. Patients were included in the present study if they had high-quality CMR images for analysis both pre- and post-TPV replacement performed between 2007 and 2010. Patients were first analyzed as an entire cohort, and then dichotomized into 2 groups: predominant pulmonary stenosis (PS; RVOT peak systolic ejection gradient ≥40 mm Hg) versus predominant PR (RVOT peak systolic ejection gradient <40 mm Hg).

**Cardiac Magnetic Resonance**

CMR was performed using a 1.5-T scanner (Achieva, Philips Medical Systems, Best, The Netherlands) with radiofrequency coils selected according to body size. All examinations were performed without sedation. Biventricular dimensions and function were measured as previously described. Briefly, ECG-gated breath-hold steady-state free precession imaging was performed in the ventricular short-axis plane with 12 to 14 equidistant slices completely covering both ventricles. Steady-state free precession image data sets were also obtained in the 4-chamber view. Flow measurements were performed in the proximal main pulmonary artery with a retrospectively gated velocity-encoded cine MR pulse sequence during free breathing. CMR data were analyzed using commercially available software packages (QMASS version 6.2 and QFLOW version 4.1, Medis, Leiden, The Netherlands). LV and RV end-diastolic (maximal) and end-systolic (minimal) volumes, stroke volume, and EF and RV end-diastolic mass and mass:volume ratio were measured as previously described. PR fraction was calculated as the retrograde flow in the main pulmonary artery divided by the antegrade flow, expressed as a percentage. The mean reconstructed temporal resolution for the cine steady-state free precession sequences was 28±6 ms.

**Strain**

CMR steady-state free precession image data sets were analyzed using Diogenes software (v. 1.1.02) within the ImageArena VA platform (version 3.0; TomTec Imaging Systems, Unterschleissheim, Germany). Short-axis and 4-chamber views of the RV and LV were imported into the software. Starting at the midpapillary level, the short-axis slice chosen for analysis was the first one in which the RV myocardium was continuous (eg, no infundibular patch was present). In the long axis, the most central slice was selected. Eight to ten points were manually placed along the endocardial surfaces of the RV and LV in the short-axis and 4-chamber views and along the LV epicardium in the short axis. These points were used by the feature tracking software to construct LV and RV longitudinal and circumferential strain (Figure 1A).

Strain versus time was calculated for each of 6 segments defined by these contours. The values of peak strain and time to peak strain were recorded for each segment; segments with dyskinetic motion (eg, positive strain curves) were excluded from time to peak analyses because no meaningful time to peak strain value could be identified. Global strain was calculated as the average of the 6 segments. The SD of the peak strain value for each of the 6 segments in a contour was calculated as an index of global uniformity of strain.

**Synchrony**

For each strain curve, maximum wall delay (latest versus earliest wall; Figure 1B) and SD of time to peak strain for the 6 constituent segments were calculated. In addition, a new technique in synchrony analysis known as cross-correlation was used. Briefly, this method identifies the phase shift between 2 curves by iteratively calculating a series of correlation coefficients between the curves by shifting one curve relative to the other in a stepwise fashion. The shift associated with the maximum correlation coefficient is reported as the cross-correlation delay for those curves (Figure 1C). For each contour, 3 cross-correlation delay values were calculated, 1 for each opposing wall pair; the mean of these 3 values was reported as the intraventricular cross-correlation delay. Interventricular cross-correlation delay was also calculated by comparing the global strain curves for the LV and RV; this was done for both longitudinal and circumferential strain. All cross-correlation values were calculated using a custom-built virtual instrument within LabVIEW version 8.2 (National Instruments, Austin, TX).

**Electrocardiographic Data**

Electrocardiographic measures were made manually on electronic waveforms by 1 author (B.H.) after acquisition from a General Electric Marquette Electronics Mac 5000 platform (Fairfield, CT) and transfer to a customized clinical workstation. Measurements included QRS duration, QRS/QT/JT dispersion (longest–shortest value on 12-lead ECG), and maximum QRS amplitude of the R wave in precordial leads V1 through V6.

**Statistical Analysis**

Continuous data are presented as mean (±SD) or median (minimum–maximum), unless otherwise noted. Comparisons between paired post- and pre-TPV replacement conditions were performed using the Wilcoxon signed-rank test; P values ≤0.05 were considered to be statistically significant. Relationships between continuous baseline characteristics (ECG, CMR, hemodynamic, and strain) and...
changes in LV and RV strain (longitudinal and circumferential) and synchrony (cross-correlation delay and maximum wall delay) were explored with linear regression. Baseline characteristics of groups with improved strain or synchrony were compared with those with worsening of strain or synchrony using the Mann–Whitney U test. For this analysis, given the large number of comparisons made, P values ≤0.001 were considered significant.

Results

Patient Characteristics and Catheterization Data

The study cohort included 31 patients (13 and 18 patients in the PR and PS cohorts, respectively). Patient characteristics and catheterization data are summarized in Table 1 (LV volumes and function could not be calculated for 2 patients in the PS group because of metallic artifacts). Most patients had TOF (n=18; 58%). Conduit types included homograft, bioprosthetic valved, nonvalved tube, and native/augmented RVOT, with sizes between 11 and 26 mm. The median interval from the precatheterization evaluation (including CMR) to TPV implant was 1 day (0–7 months; interquartile range=2 days). The median duration from TPV replacement to follow-up evaluation was 6 months (5–13 months; interquartile range=1 month). Mean body surface area, 1.7±0.3 m², did not change after TPV replacement.

The median peak RVOT gradients before and after TPV replacement were as follows: for the whole cohort, 41 (20–70 mm Hg) to 16 mm Hg (7–37 mm Hg); PR group, 31 (20–39 mm Hg) to 16 mm Hg (8–22 mm Hg); and PS group, 50 (41–70 mm Hg) to 15 mm Hg (7–37 mm Hg).
and circumferential strain were noted in each case, except longitu-
dinal strain in the PS cohort. Among RV strain parameters, a
significant increase was observed in circumferential strain in
the PS cohort. No differences were seen in SD of segmental peak
strain value (uniformity of strain) after TPV replacement for any
of the strain parameters tested in any of the groups.

**Ventricular Synchrony**

Post- versus pre-TPV replacement synchrony measures are
presented in Table 4. In the PR subgroup, TPV replacement was
associated with decreases in both LV longitudinal maximum
wall delay and cross-correlation delay. RV synchrony measures
did not improve significantly. Interventricular cross-correlation
delay did not change significantly after TPV replacement for
circumferential (68±48 versus 62±42 ms; \( P=0.51 \)) or longitu-
dinal (71±131 versus 92±89 ms; \( P=0.37 \)) strain.

**Baseline Factors Associated With Change in Strain and Synchrony**

Modest associations were identified by linear regression
between change in RV circumferential strain versus body
surface area \( (R=0.56; P=0.001) \) and change in LV longitudinal
strain versus systolic pulmonary artery pressure \( (R=0.56; \)
\( P=0.004 \)) before TPV replacement (Figure 2). The correlation
coefficient for all of the other comparisons between baseline
CMR, ECG, hemodynamic, and strain factors and change in
strain and synchrony parameters was <0.5.

Comparison of baseline values of patients with improved
strain with those in whom strain worsened showed that
improved LV longitudinal strain \((n=19)\) was associated with
impaired baseline LV longitudinal strain \((-13±3\% \text{ versus}\
-20±4\% ; P<0.001)\) and that improved RV circumferential
strain \((n=18)\) was associated with lower baseline body surface
area \((1.6±0.3 \text{ versus} 1.9±0.2 \text{ m}^2; P<0.001)\). Among synchrony
parameters, patients with improved LV longitudinal cross-
correlation delay \((n=17)\) had lower baseline RV to pulmonary
artery peak gradient \((38±10 \text{ versus} 54±13 \text{ mmHg}; P=0.001)\),
and those with decreased LV longitudinal wall delay \((n=15)\)
had longer LV longitudinal wall delay at baseline \((507±107
\text{ versus} 247±138 \text{ ms}; P<0.001)\).

**Discussion**

By analyzing cine CMR images before and a median of 6
months after TPV implantation, we found that alleviation of
RVOT conduit dysfunction is associated with an improve-
ment in LV longitudinal and circumferential strain and, in the
cohort of patients with predominant PS, an improvement in
RV circumferential strain. Among patients with primary PR,
LV longitudinal synchrony improved after this procedure.

To date, data describing the impact of TPV replacement on
patients with RVOT conduit dysfunction have been scarce,
inconsistent, and based solely on ultrasound-based meth-
ods.6,23-25 For strain analysis in this cohort, the use of CMR
offers advantages compared with echocardiography. Most
of these patients are adults with poor acoustic windows sec-
ondary to their body size and prior surgical procedures. As
well, the RV can be especially difficult to image reliably in
this population because of its dilation and retrosternal loca-
tion.26,27 CMR is not hindered by these drawbacks, typically
provides high-quality and reproducible imaging data sets, and has become the modality of choice in the assessment of ventricular size and function in many forms of adult congenital heart disease, including TOF. For these reasons, we used a CMR-based approach in this study.

TPV replacement is performed in an effort to mitigate deleterious effects of long-term exposure to chronic RV volume and pressure load such as ventricular dysfunction, arrhythmia, and death. It is well established that pulmonary valve replacement, whether surgical or percutaneous, leads to a marked reduction in RV end-diastolic and end-systolic volumes, PR fraction, and RV stroke volume. These changes were observed in our cohort as well.

In addition to these findings, we used CMR strain analysis to show that all 3 groups had significant improvements in LV strain parameters after TPV replacement. It is possible that these increases in LV strain, if sustained, may provide early evidence of a potential long-term benefit of this therapy. In a recent report, Diller et al showed a strong relationship between impaired LV longitudinal strain and adverse long-term outcomes, including life-threatening arrhythmia and death, in patients with repaired TOF. Other groups have recently shown relationships between reduced longitudinal strain and adverse outcomes in patients with arrhythmia and pressure-loaded hearts. Findings such as these may support targeted efforts to offer TPV replacement to patients with impaired longitudinal strain.

The mechanism of improved LV strain in the study groups likely varied depending on the nature of the RVOT conduit dysfunction (PS versus PR). Increases in longitudinal strain in the PR group exceeded those of the PS group, consistent with the observed inverse relationship between LV longitudinal strain improvement after TPV replacement and pre-procedural systolic pulmonary artery pressure. In all cases, however, it is likely that enhanced LV filling played a role. This mechanism is supported by increases in indexed LV end-diastolic volume and stroke volume among all groups in the absence of a change in heart rate (the absence of statistical significance in most but not all subgroups may plausibly be related to small sample size and loss of power). Lurz et al described improved LV filling associated with relief of RVOT obstruction by TPV replacement, an effect that would likely apply to the PS group in our study. Enhanced LV filling in the PR cohort would be expected from decreased RV volume load and normalization of the diastolic septal configuration.

In this work, we observed an improvement in RV circumferential strain in the PS group but not in the cohort with primary PR. Improvement in the patients with PS almost certainly reflects the load dependence of strain as a measure of contractility. The improvement in circumferential but not longitudinal strain may suggest regional differences of the impact of RV pressure load, with infundibular function more affected than that of the lateral free wall. The absence of

<table>
<thead>
<tr>
<th>Variable</th>
<th>All (n=31)</th>
<th>PR (n=13)</th>
<th>PS (n=18)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Variable</td>
<td>Pre</td>
<td>Post</td>
<td>P Value</td>
</tr>
<tr>
<td>LV EDVi, mL/m²</td>
<td>90±20</td>
<td>95±25</td>
<td>0.05*</td>
</tr>
<tr>
<td>LV ESVi, mL/m²</td>
<td>40±13</td>
<td>41±13</td>
<td>0.60</td>
</tr>
<tr>
<td>LV SVi, mL</td>
<td>50±11</td>
<td>55±15</td>
<td>0.004*</td>
</tr>
<tr>
<td>LV EF, %</td>
<td>56±7</td>
<td>57±7</td>
<td>0.20</td>
</tr>
<tr>
<td>RV EDVi, mL/m²</td>
<td>159±81</td>
<td>128±64</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>RV ESVi, mL/m²</td>
<td>95±80</td>
<td>76±65</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>RV SVi, mL</td>
<td>64±16</td>
<td>52±12</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>Massi, g/mL²</td>
<td>45±20</td>
<td>35±16</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>Mass:volume, g/mL</td>
<td>0.3±0.1</td>
<td>0.3±0.1</td>
<td>0.60</td>
</tr>
<tr>
<td>LV EF, %</td>
<td>47±16</td>
<td>47±16</td>
<td>0.74</td>
</tr>
<tr>
<td>LV PR, %</td>
<td>29±15</td>
<td>1.7±2.2</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>ECG HR, bpm</td>
<td>72±10</td>
<td>71±15</td>
<td>0.89</td>
</tr>
<tr>
<td>ECG QRS dur, ms</td>
<td>142±40</td>
<td>141±38</td>
<td>0.48</td>
</tr>
<tr>
<td>ECG QRS disp, ms</td>
<td>31±11</td>
<td>33±17</td>
<td>0.57</td>
</tr>
<tr>
<td>ECG QT disp, ms</td>
<td>55±35</td>
<td>49±31</td>
<td>0.26</td>
</tr>
<tr>
<td>ECG JT disp, ms</td>
<td>56±34</td>
<td>52±31</td>
<td>0.68</td>
</tr>
<tr>
<td>ECG QRS amp, mV</td>
<td>24±9</td>
<td>19±9</td>
<td>&lt;0.001*</td>
</tr>
</tbody>
</table>

Data are presented as mean±SD; P values reflect results of the Wilcoxon signed-rank test. CMR indicates cardiac magnetic resonance; EDVi, end-diastolic volume index; EF, ejection fraction; ESVi, end-systolic volume index; HR, heart rate; JT disp, JT dispersion; LV, left ventricle; PR, pulmonary regurgitation; PS, pulmonary stenosis; QRS amp, QRS amplitude; QRS disp, QRS dispersion; QRS dur, QRS duration; QT disp, QT dispersion; RV, right ventricle; SVi, stroke volume index; and TPV, transcatheter pulmonary valve.

*Indicates P value ≤0.05.
improvement in the PR group mirrors another report by Lurz et al in which improved RV EF was noted after pulmonary valve implantation in patients with a pressure-loaded, but not volume-loaded, RV. The observed association between lower body surface area and improved RV circumferential strain after TPV replacement lends support to the notion that the capacity of the RV to remodel after TPV replacement may deteriorate with age.

Another important determinant of the efficiency of ventricular performance is contraction synchrony. Patients with dysfunctional RVOT conduits have been shown to exhibit dysynchronous interventricular contraction associated with factors such as baseline RV strain, EF, and end-systolic and diastolic volumes. In a cohort of patients with TOF, LV dysynchrony has been associated with ventricular tachycardia and death. Given these associations, we chose to investigate the impact of TPV replacement on ventricular synchrony. Several measures were investigated given the lack of general consensus in the literature regarding the most appropriate index of ventricular synchrony; as well, we included a new measure known as cross-correlation delay which is potentially more robust than traditional indices. In the PR subgroup, 2 of the 3 measures of LV longitudinal synchrony, including cross-correlation delay, improved after TPV replacement; the third trended toward improved synchrony but did not reach statistical significance. The improvement in synchrony in the PR cohort, but not the PS population, may be related to pronounced displacement of the septum into the LV in diastole in the former group, delaying the re-establishment of the normal round systolic configuration. Although no significant differences in measures of RV synchrony were identified, nonsignificant improvements were noted in almost all measures.

Table 3. Pre- and Post-TPV Replacement Global Strain (%)

<table>
<thead>
<tr>
<th>Strain Value</th>
<th>All (n=31)</th>
<th>PR (n=13)</th>
<th>PS (n=18)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
<td>P Value</td>
</tr>
<tr>
<td>LV Longitudinal</td>
<td>−15±5</td>
<td>−17±4</td>
<td>0.02*</td>
</tr>
<tr>
<td></td>
<td>−18±3</td>
<td>0.03*</td>
<td></td>
</tr>
<tr>
<td>Circumferential</td>
<td>−23±5</td>
<td>−25±5</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td></td>
<td>−26±4</td>
<td>0.02*</td>
<td></td>
</tr>
<tr>
<td>RV Longitudinal</td>
<td>−15±5</td>
<td>−15±5</td>
<td>0.89</td>
</tr>
<tr>
<td></td>
<td>−16±6</td>
<td>−17±6</td>
<td>0.34</td>
</tr>
<tr>
<td>Circumferential</td>
<td>−17±6</td>
<td>−18±7</td>
<td>0.11</td>
</tr>
<tr>
<td></td>
<td>−18±6</td>
<td>−18±7</td>
<td>0.82</td>
</tr>
</tbody>
</table>

Data are presented as mean±SD; P values reflect results of the Wilcoxon signed-rank test. LV indicates left ventricle; PR, pulmonary regurgitation; PS, pulmonary stenosis; RV, right ventricle; and TPV, transcatheter pulmonary valve. *Indicates P value ≤0.05.

Table 4. Pre- and Post-TPV Replacement Dyssynchrony (ms)

<table>
<thead>
<tr>
<th>Variable</th>
<th>All (n=31)</th>
<th>PR (n=13)</th>
<th>PS (n=18)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
<td>P Value</td>
</tr>
<tr>
<td>LV</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Longitudinal</td>
<td>341±241</td>
<td>276±191</td>
<td>0.18</td>
</tr>
<tr>
<td></td>
<td>310±180</td>
<td>210±120</td>
<td>0.04*</td>
</tr>
<tr>
<td>SD (time to peak)</td>
<td>146±76</td>
<td>123±68</td>
<td>0.26</td>
</tr>
<tr>
<td>Cross-correlation delay</td>
<td>218±125</td>
<td>186±148</td>
<td>0.13</td>
</tr>
<tr>
<td>Circumferential</td>
<td>113±99</td>
<td>119±115</td>
<td>0.65</td>
</tr>
<tr>
<td></td>
<td>129±107</td>
<td>125±115</td>
<td>0.70</td>
</tr>
<tr>
<td>SD (time to peak)</td>
<td>43±37</td>
<td>47±48</td>
<td>0.75</td>
</tr>
<tr>
<td>Cross-correlation delay</td>
<td>62±69</td>
<td>60±67</td>
<td>0.91</td>
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<tr>
<td>RV</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Longitudinal</td>
<td>253±189</td>
<td>241±182</td>
<td>0.76</td>
</tr>
<tr>
<td></td>
<td>255±177</td>
<td>295±200</td>
<td>0.96</td>
</tr>
<tr>
<td>SD (time to peak)</td>
<td>109±62</td>
<td>100±61</td>
<td>0.59</td>
</tr>
<tr>
<td>Cross-correlation delay</td>
<td>150±74</td>
<td>138±107</td>
<td>0.37</td>
</tr>
<tr>
<td>Circumferential</td>
<td>186±101</td>
<td>185±108</td>
<td>0.87</td>
</tr>
<tr>
<td></td>
<td>255±177</td>
<td>183±124</td>
<td>0.76</td>
</tr>
<tr>
<td>SD (time to peak)</td>
<td>72±38</td>
<td>71±42</td>
<td>0.86</td>
</tr>
<tr>
<td>Cross-correlation delay</td>
<td>102±93</td>
<td>126±149</td>
<td>0.89</td>
</tr>
</tbody>
</table>

Data are presented as mean±SD; P values reflect results of the Wilcoxon signed-rank test. LV indicates left ventricle; PR, pulmonary regurgitation; PS, pulmonary stenosis; RV, right ventricle; and TPV, transcatheter pulmonary valve. *Indicates P value ≤0.05.
in the PR, but not PS, subgroup. These improvements may reflect increased baseline dyssynchrony in the former group, compared with the latter, perhaps related to a more abnormal diastolic septal position.

Among ECG parameters, a significant decrease in QRS amplitude was observed, likely reflecting RV remodeling associated with a reduction in RV mass or dilation, both of which decreased significantly. Heart rate did not change significantly between the 2 evaluations, neither did QRS duration nor any of the measures of dispersion (QRS, QT, JT). These results are at odds with those of Plymen et al.,37 who suggested improved electric synchrony after TPV replacement based on their reported decreases in QRS duration in the PR subgroup and decreased QTc and QRS/QT/JT dispersion in their whole cohort. These discrepant findings could be attributable to factors such as variation in timing of post-TPV replacement ECG among the studies (6 months in this study versus 1 year in theirs), definitions of subgroups, measurement technique, or small sample sizes. They do not, however, reflect baseline differences in pre-TPV QRS durations in the 2 studies (137 and 142 ms, respectively).

**Limitations**

The temporal resolution of CMR sequences is modest, and higher frame rate acquisitions might lead to changes in the values of the strain parameters reported. These changes, however, would be expected to be small and would apply equally to the pre- and post-TPV CMR evaluations. Intra- and interobserver reproducibility of CMR strain measurements was not investigated. This has been examined in multiple previous publications, however, with acceptable reproducibility repeatedly demonstrated on a global (not segmental) basis.17,38–41

Given the uncertain reproducibility of segmental measurements, these were specifically not reported here; the global LV differences that are noted in this report have been shown to be the most reproducible of the strain measurements. Strain is a load-dependent parameter, and some of the observed changes would be expected to reflect changes in ventricular loading conditions; it is worth noting, however, that there was no significant difference in heart rate post- versus pre-TPV replacement. Finally, small sample sizes reduced the ability of this study to detect differences such as the magnitude of the change in longitudinal versus circumferential strain, especially in subpopulations; strong efforts were made, however, to include data from all eligible patients. The PR and PS subgroups were not pure because most patients with PR also had some degree of RVOT obstruction and most patients with PS had some degree of PR, which may confound the resolution and applicability of findings for those cohorts.

**Conclusions**

TPV implantation in patients with dysfunctional RVOT conduits was associated with improved global longitudinal and circumferential LV strain; subgroups had improved RV circumferential strain (PS cohort) and more synchronous LV longitudinal contraction (PR cohort). Given the association between strain and synchrony and clinical outcomes, these findings suggest a potential long-term beneficial impact of TPV implantation on these patients.

**Disclosures**

Dr McElhinney is a consultant, proctor, and investigator for Medtronic, Inc. The other authors report no conflicts.

**References**


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