Clinical Investigation

Left Ventricular Function Improves after Pulmonary Valve Replacement

in Patients with Previous Right Ventricular Outflow Tract Reconstruction and Biventricular Dysfunction

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Congenital heart defects that have a component of right ventricular outflow tract obstruction, such as tetralogy of Fallot, are frequently palliated in childhood by disruption of the pulmonary valve. Although this can provide an initial improvement in quality of life, these patients are often left with severe pulmonary valve insufficiency. Over time, this insufficiency can lead to enlargement of the right ventricle and to the deterioration of right ventricular systolic and diastolic function. Pulmonary valve replacement in these patients decreases right ventricular volume overload and improves right ventricular performance. To date, few studies have examined the effects of pulmonary valve replacement on left ventricular function in patients with biventricular dysfunction. We sought to perform such an evaluation.

Records of adult patients who had undergone pulmonary valve replacement from January 2003 through November 2006 were analyzed retrospectively. We reviewed preoperative and postoperative echocardiograms and calculated left ventricular function in 38 patients.

*In the entire cohort, the mean left ventricular ejection fraction increased by a mean of 0.07 after pulmonary valve replacement, which was a statistically significant change (*P *<0.01). In patients with preoperative ejection fractions of less than 0.50, mean ejection fractions increased by 0.10.*

We conclude that pulmonary valve replacement in patients with biventricular dysfunction arising from severe pulmonary insufficiency and right ventricular enlargement can improve left ventricular function. Prospective studies are needed to verify this finding. ***(Tex Heart Inst J 2011;38(3):234-7)***

Key words: *Adult; cardiac output; cardiac volume; heart valve prosthesis; hemodynamics; pulmonary valve insufficiency/complications/surgery; reoperation; retrospective studies; stroke volume; tetralogy of Fallot/surgery; time factors; treatment outcome; ventricular dysfunction, left; ventricular dysfunction, right*

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Congenital heart defects that have a component of right ventricular (RV) outflow tract obstruction, such as tetralogy of Fallot, are frequently palliated in childhood by disruption of the pulmonary valve. Although this can provide an initial improvement in quality of life, these patients are often left with severe pulmonary valve insufficiency. Over time, this insufficiency can lead to enlargement of the RV[1,2] and to worsening RV systolic and diastolic function.[3] Pulmonary valve replacement (PVR) in these patients has been shown to decrease RV volume overload and improve RV performance.[4] Although the optimal timing of PVR has yet to be determined, the improvements in RV health are well documented.

Much has been written about the RV after PVR, but data regarding the effect of PVR on the left ventricle (LV) are relatively few. Previous studies have suggested that there is an interaction between right and left ventricular function and that RV dysfunction can result in an unfavorable RV–LV interaction.[5]

Conversely, because of this known interaction, improvement in RV performance after PVR could translate into a favorable effect on the LV in patients who have preoperative biventricular dysfunction.

Patients and Methods

After approval from the institutional review board, we retrospectively identified adult patients from our surgical database who had undergone PVR from January 2003 through November 2006, late after repair of tetralogy of Fallot or other surgery for pulmonary valve disruption. Sixty patients were initially identified. Pre-

operative and postoperative echocardiograms were reviewed; 22 patients were excluded due to inability to obtain preoperative echocardiograms (3 patients), poor echocardiographic images that made measurement of the LV dimensions unreliable (18), and intraoperative death (1).

Patient Population

Thirty-eight patients were included in the final analysis. Data on these patients' preoperative characteristics appear in Table I. There were 28 females and 10 males. Twenty-six patients had a diagnosis of tetralogy of Fallot, and 12 patients had a diagnosis of pulmonary stenosis. The median age at the time of pulmonary valve disruption for the entire study population was 4 years, ranging from less than 1 year to 56 years. The mean age at the time of pulmonary valve disruption was 6.6 ± 10.6 years. Thirty-six patients had surgical disruption of the pulmonary valve at an age of less than 18 years. One patient required pulmonary valve disruption surgery a second time because of restenosis. The median age at PVR for the entire study population was 34 years, and the mean age was 33.1 ± 13.2 years. Six patients in our study underwent a second PVR, and in those cases we followed the results of that procedure.

Echocardiographic Measurements

Transthoracic echocardiograms were reviewed after our final identification of patients who had undergone PVR late after repair of tetralogy of Fallot or other surgery for pulmonary valve disruption. We chose as the preoperative echocardiogram the one performed closest to the time of surgery. The median time from PVR surgery to the postoperative echocardiogram was 55 days (range, 5–840 d). Data from the preoperative and postoperative echocardiograms were obtained by retrospective analysis. Left ventricular ejection fractions (LVEFs) were calculated by means of Simpson's method, using both the 2- and 4-chamber views. For those patients with 2-chamber views of insufficient quality to allow for accurate measurements, LVEFs were calculated using only the 4-chamber view in both the preoperative and postoperative studies.

Statistical Analysis

Preoperative and postoperative results were compared via a simple Student paired *t* test using Microsoft Excel 2007. A *P* value of less than 0.05 was considered statistically significant.

Results

For the entire study population, there was a significant increase in LVEF between the preoperative echocardiogram (mean, 0.44 ± 0.13) and the postoperative echocardiogram (mean, 0.51 ± 0.11) (P <0.01). The mean increase was 0.07 (Fig. 1). Twenty-six patients had a preoperative LVEF of less than 0.50. For these patients, there was also a significant postoperative increase in the LVEF (mean, 0.48 ± 0.11), compared with the preoperative LVEF (mean, 0.38 ± 0.10) (P <0.01). The mean increase was 0.10. Thirty-three of the 38 patients (87%) had an improvement in LV function after PVR.

Two patients underwent pulmonary valve disruption surgery as adults, at ages 40 and 56 years. After PVR, the 1st patient had a slight increase in LVEF of 0.03. The 2nd patient had a decrease in LVEF of 0.04.

All told, 5 patients showed a slight decline in LVEF after PVR. The LVEF was reduced by 0.04 in 3 patients, 0.03 in 1, and 0.02 in 1. Despite these reductions, the postoperative LVEF was greater than 0.50 in 4 of the 5 patients.

TABLE I. Preoperative Characteristics of the Study Population (38 Patients)

Characteristic	Value
Female	28 (74)
Male	10 (26)
Tetralogy of Fallot	26 (68)
Pulmonary stenosis	12 (32)
Median age at PV disruption, yr	4
Mean age at PV disruption, yr	6.6 ± 10.6
Median age at PVR, yr	34
Mean age at PVR, yr	33.1 ± 13.2
Treated with β-blockers, n	17
Treated with β-blockers and ACEI, n	5
Mean preoperative QRS duration, ms	136.4 ± 34.6
Mean preoperative LVEF	0.44 ± 0.13
Mean postoperative LVEF	0.51 ± 0.11

ACEI = angiotensin-converting enzyme inhibitors; LVEF = left ventricular ejection fraction; PV = pulmonary valve; PVR = pulmonary valve replacement

Values are presented as number and percentage or as mean ± SD, unless otherwise stated.

Preoperative Use of Heart-Failure Medications

Twenty-one patients were not taking cardiovascular medications before surgery. The mean LVEF in these 21 patients increased by 0.07, from 0.47 to 0.54. Seventeen patients were taking a β-blocker preoperatively. In the β-blocker group, the mean LVEF increased by 0.07, from 0.41 to 0.48. Five patients in the β-blocker group were also treated preoperatively with angiotensin-converting enzyme inhibitors. Their mean LVEF increased by 0.05, from 0.40 to 0.45. The improvements in LVEF in patients taking β-blockers and those taking

both β-blockers and angiotensin-converting enzyme inhibitors preoperatively were not significantly different from those of patients who were taking no cardiovascular medications ($P=0.4$ and $P=0.3$, respectively).

Discussion

Repair of tetralogy of Fallot in early childhood has low operative mortality rates and excellent long-term results.[2,6] Many investigators have examined in depth the causal relationships between disruption of the pulmonary valve annulus and subsequent pulmonary insufficiency, deleterious effects on the RV, and increased risk of sudden death.[7-10] Improvement in RV function after PVR has also been well documented in the medical literature.[11,12] It has been suggested that the reduced volume load on the RV after PVR results in the rapid improvement of RV systolic function and in a more gradual recovery of RV diastolic function.[3] Our study provides evidence of improvement in LV systolic function after PVR in patients with a history of pulmonary valve disruption surgery.

Overall, in our study, patients had a mean increase in LVEF of 0.07 (Fig. 1). Those patients who started with an LVEF of less than 0.50 experienced a greater mean increase of 0.10.

The exact cause of this favorable result is unclear. One likely cause is improved RV–LV interaction resulting from a decrease in RV volume. An unfavorable RV–LV interaction has been shown in patients with repaired tetralogy of Fallot.[5] Another possibility is improved synchrony accompanied by a decrease in QRS duration.

Medications

The use of heart-failure medications preoperatively did not correlate with significant postoperative improvement in LVEF.

Ventricular Interaction

The interdependence of the RV and LV is well accepted. Previous studies have suggested that RV dysfunction can result in an unfavorable RV–LV interaction, which leads ultimately to LV dysfunction in the preoperative period.[5] Because of the same interaction, improvement in RV performance after PVR could have a favorable effect on the LV in patients who are experiencing biventricular dysfunction.

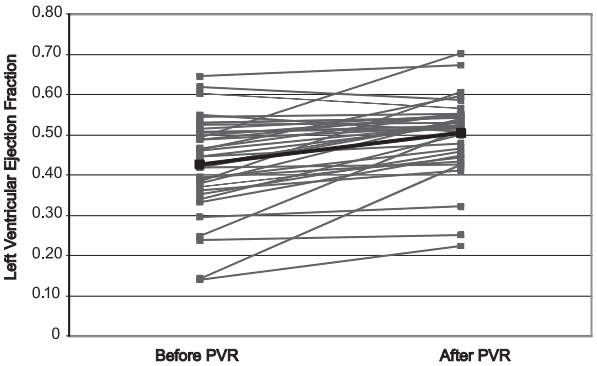

Fig. 1 *Mean left ventricular ejection fraction change before and after pulmonary valve replacement surgery (PVR).*

Other mechanical mechanisms of decreased LV function late after pulmonary valve disruption to palliate tetralogy of Fallot or pulmonary stenosis can include abnormalities of septal motion due to patching of a ventricular septal defect or to septal fibrosis or myocardial injury in association with the initial repair. These abnormalities are not likely to improve after PVR. Improvement of LV function after PVR most probably involves improvement in the unfavorable interaction between a volume-overloaded, dilated RV and the LV.

Results for the 2 patients in our study who underwent pulmonary valve disruption at older ages did not show a meaningful trend toward improvement in LVEF. Undergoing pulmonary valve disruption and PVR therapy late in life raises the possibility of irreversible ventricular dysfunction. Although the optimal time for PVR in patients who have a RV that is beginning to dilate has not yet been established, it is probable that the passage of a certain period of time diminishes the improvement that can be expected in LV function consequent to improved RV–LV interaction.

Limitations

A major limitation of our study is the retrospective nature of the data collection. High-quality echocardiographic 2-chamber views were not available for all patients in our population. This necessitated the use of single-plane (4-chamber) images for analysis. While the absolute measurements of LV end-diastolic and end-systolic volumes might be affected by this limitation, changes in LVEF over time can still be evaluated. Clearly, better volumetric analysis, such as that available with the use of cardiovascular magnetic resonance, would have been ideal. Many of the patients in our study, however, received care before cardiovascular magnetic resonance was readily available.

Another possible limitation of this study lies in our placing patients with tetralogy of Fallot in the same category as those with pulmonary stenosis. Tetralogy of Fallot is of course a different disease entity, which raises other pathophysiologic concerns. In comparing the effects of pulmonary valve disruption, however, we believe that similarities between the 2 groups of patients can be used to evaluate the effects of PVR on LV systolic function.

Conclusion

Despite some limitations, this study clearly shows that LV function can improve, dramatically at times, after PVR in patients who manifest biventricular dysfunc-

tion some years after surgery for pulmonary valve disruption. Prospective studies are necessary to evaluate this finding and to identify those features that are associated with minimal or no improvement in LV function and those that are associated with favorable outcomes.

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