Pulmonary Valve Replacement in Young Children after Repair of Typical Tetralogy of Fallot

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Background. Residual lesions after tetralogy of Fallot (TOF) repair, especially pulmonary regurgitation (PR), bring RV dysfunction, exercise intolerance, and increased risk of cardiac death. This study presents our experience of pulmonary valve replacement (PVR) after TOF repair.

Methods and Results. A total of 540 patients underwent total correction of typical TOF at Seoul National University Children's Hospital from 1988 to 1999. Of these 540, 14 underwent PVR after a mean interval of 2.31 ± 1.56 years. Mean age at PVR was 4.21 ± 2.92 years. All patients had moderate to severe PR and cardiomegaly, and 9 had moderate to severe tricuspid regurgitation (TR). Additional procedures performed at the time of PVR were: pulmonary artery angioplasty (7), VSD repair (5), tricuspid valve repair (4), relief of infundubular stenosis (2), pacemaker insertion (2), and cryoablation of RV outflow tract (1). Results: Mean follow up period was 2.29 years. Twelve are in New York Heart Association (NYHA) class I; none are in class II. Mean cardiothoracic ratio decreased from 0.69 ± 0.08 to 0.59 ± 0.07 ($p = 0.002$). There was no significant change in mean QRS duration (139 ± 26 ms to 144 ± 23 ms, $p = 0.799$). Eight patients showed mild PR and 2 moderate PR. The RV end-diastolic diameter/body surface area (mm/m^2) ratio decreased in 2 and remained unchanged in 8.

Conclusions. In children who have undergone repair of TOF, early correction of PR by PVR, in addition to corrective surgery for branch pulmonary artery stenosis or VSD, is necessary to preserve RV function.
(***Korean J Cardiovasc Dis*** 2001;2:132-138)

Key Words: Tetralogy of fallot; Pulmonary valve insufficiency; Surgery

INTRODUCTION

Good long-term survival and quality of life after repair of tetralogy of Fallot (TOF) is well documented.[1)2)] As more patients survive into adulthood after initial repair, an increasing number of patients are undergoing reoperations due to late complications. These complications include pulmonary regurgitation(PR) or stenosis, residual right ventricular (RV) outflow tract obstruction, residual ventricular septal defect, aortic valve regurgitation and tricuspid regurgitation(TR).[1)2)]

Although PR after repair of TOF is generally considered benign, persistent PR can lead to progressive RV dilatation and failure, poor exercise tolerance, and a highly

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Received August 4, 2001; Accepted September 12, 2001
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increased risk for ventricular arrhythmias and sudden death.[3-5] Generally, pulmonary valve replacement (PVR) for symptomatic PR has lead to improvement in both clinical status and RV function,[5] possibly due to the reversible nature of RV dysfunction. However, the optimal timing for PVR remains to be established. The purpose of this review is to evaluate the results of our experience of PVR after repair of TOF.

MATERIALS AND METHODS

We reviewed the hospital records to obtain the clinical status of patients before PVR, examine the indications for PVR, and evaluate the subsequent clinical course after PVR. The surgical data were reviewed for details of initial palliative and total procedures, and of PVR itself. The total number of patients that underwent repair of typical TOF at Seoul National University Children's Hospital from 1988 to 1999 was 540, and 14 of whom subsequently underwent PVR between 1990 and May 2001. Demographic data are shown in Table 1. The patient population consisted of 10 boys and 4 girls, with age ranging from 21 months to 13 years (mean 4.21±2.92 years) at the time of PVR. PVR was performed 9 to 82 months (mean 27.7±18.7 months) after initial repair. At the time of initial repair, 7 patients also underwent transannular patch insertion, 3 pulmonary valvotomy, and 10 infundibular muscle resection to relieve RV outflow tract obstruction (RVOTO). Five patients (35%) had received a Blalock-Taussig shunt as a palliation prior to the initial repair. Previously stable patients underwent PVR due to the presence of associated lesions such as branch pulmonary artery stenosis or VSD, in addition to PR (Table 2). Patients who had PVR at the time of initial total repair, or within 1 month postoperatively, were excluded. Cardiothoracic ratios before and after PVR were measured from chest x-rays. Twelve-lead electrocardiograms (ECG) and Holter recordings were reviewed to check QRS durations and clinical arrhythmias both before and after PVR. Preoperative and postoperative echocardiogram recordings were also reviewed, although only 10 were available for the postoperative group. PR and TR were graded as mild, moderate or severe. Right ventricular end diastolic diameter (RVEDD) dimensions were measured at apical four chamber views according to the standards of Foale et al,[7] and were expressed in millimeters/body surface area (mm/m^2). Cardiac catheterization was performed on all patients before surgery to evaluate pulmonary artery anatomy, the degree of RV dysfunction, and the size of residual intracardiac shunts. All patients underwent associated procedures at the time of PVR (Table 3); branch pulmonary artery angioplasty in 7, residual VSD repair in 5, tricuspid valve repair in 4, relief of infundibular stenosis in 2, pacemaker insertion in 2, and cryoablation of RV outflow tract in 1 patient, respectively.

Statistical methods

We analysed the data using SPSS for Windows. Continuous variables are presented as mean±standard deviation. Comparisons were evaluated by the Wilcoxon signed ranks test. A p value of less than 0.05 was considered significant.

Table 1. Demographic data of 14 patients undergoing PVR after TOF repair

Variable	(n=14)
Male gender	10 (71%)
BT shunt before TOF repair	5 (35%)
Mean age at TOF repair (years)	1.89±1.74
Mean age at PVR (years)	4.21±2.92
Mean time from TOF repair to PVR (years)	2.31±1.56
Re-PVR	1 (7%)
Time from PVR to re-PVR(years)	2.8

PVR: pulmonary valve replacement, TOF: tetralogy of Fallot, BT shunt: Blalock-Taussig shunt

Table 2. Major hemodynamic indications for reoperation

Major lesion		
Mainly PR	3*	21%
PR + residual VSD	4	29%
PR + branch PA stenosis	6	43%
PR + residual VSD + branch PA stenosis	1	7%

PR: pulmonary regurgitation, VSD: ventricular septal defect, PA: pulmonary artery, *: represents number

Table 3. Associated procedures performed at the time of PVR

PA angioplasty	7	50%
Residual VSD closure	5	36%
Tricuspid valve repair	4	29%
Relief of infundibular stenosis	2	14%
Pacemaker insertion	2	14%
Ventricular cryoablation	1	7%

PVR: pulmonary valve replacement, PA: pulmonary artery, VSD: ventricular septal defect, *: represents number

RESULTS

Chest X-rays showed cardiomegaly in all patients before PVR (CT ratio 0.69±0.08). Two patients had complete AV block, and one demonstrated sustained ventricular tachycardia. The stages according to the preoperative New York Heart Association (NYHA) classification were class I in 13 and class II in the other. Mean RVEDD/BSA before PVR was 65.2±17.9 mm/m², mean cardiothoracic ratio was 0.69±0.08, and mean QRS duration was 139±26 ms. The degrees of PR and TR decreased in severity after PVR (Table 4). Even though all patients showed at least moderate degrees of PR, the same three conditions of clinical arrhythmias before PVR remained unchanged after PVR. Thus, the severity of PR did not correlate with the incidence of arrhythmias. Residual lesions were noticed in all patients: ventricular septal defect (5), and branch pulmonary artery stenosis (7) were noted at the time of PVR.

Table 4. Degrees of PR and TR before and after PVR

Echocardiographic Variables	Pre-PVR (n=14)	Post-PVR (n=10)
PR		
Mild	0 (0%)	8 (80%)
Moderate	12 (86%)	2 (20%)
Severe	2 (14%)	0 (0%)
TR		
Mild	5 (36%)	9 (90%)
Moderate	5 (36%)	1 (10%)
Severe	4 (28%)	0 (0%)

PVR: pulmonary valve replacement, PR: pulmonary regurgitation, TR: tricuspid regurgitation

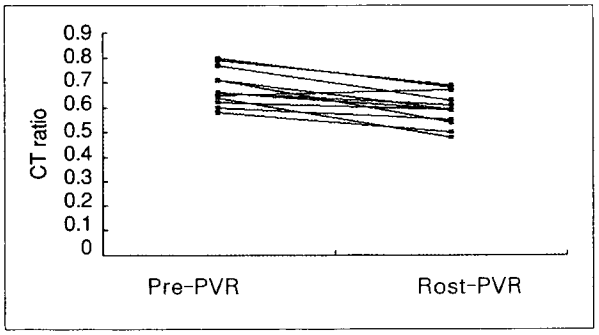

Figure 1. Reduction of cardiothoracic ratio(measured on chest X-ray) after pulmonary valve replacement for moderate or severe pulmonary regurgitation. CT ratio: cardiothoracic ratio, Pre-PVR: before pulmonary valve replacement, Post-PVR: after pulmonary valve replacement.

Pulmonary valve replacement(PVR)

PVR was performed at the mean age of 4.21±2.92 years (range 21 months to 13 years). Five patients received aortic homografts (size ranging from 20 to 23 mm), 3 received polystan valved conduits (size ranging from 18 to 20 mm), 2 received Carpentier-Edwards valved conduits (size ranging from 20 to 23 mm), 2 received Hancock valves (size ranging from 23 to 25 mm), and 2 had monocusp valves inserted.

Chest X-rays

After PVR, cardiothoracic ratio decreased in all patients, from 0.69±0.08 before PVR to 0.59±0.07 after PVR (p = 0.002) (Figure 1).

Electrocardiogram

The mean QRS duration increased from 139±26 ms to 144±23 ms after PVR, but the change was not statistically significant (p = 0.799).

2-D echocardiography

Postoperative echocardiographic data was available in 10 patients only. Transthoracic echocardiography, performed at a mean follow-up of 2.29 years after PVR, showed a decrease in the severity of both PR and TR (Table 4). RVEDD/BSA showed a decrease in 2 patients, and no significant change in 8 patients (p = 0.575) (Figure 2).

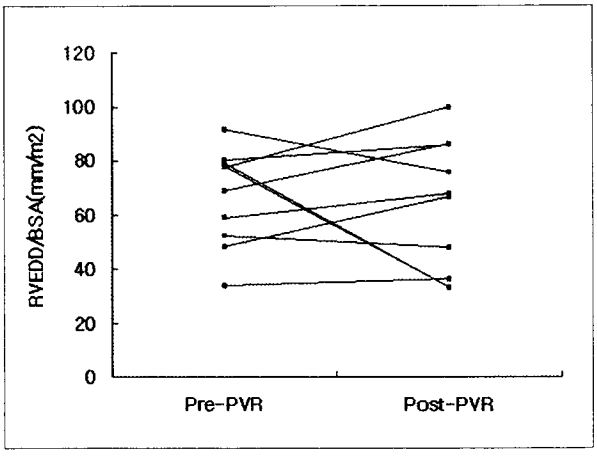

Figure 2. Right ventricular end-diastolic volume/body surface area, measured at rest, before and after PVR. PVR: palmonary valve replacement, RVEDD/BSA: right ventricular end diastolic diameter/body surface area, Pre-PVR: before pulmonary valve replacement, Post-PVR: after pulmonary valve replacement.

Table 5. Hemodynamic results from cardiac catheteriza-tion before PVR

Classification	Hemodynamic results	Patients (n=14)
Excellent	RV systolic pressure < 40 mmHg, and RV-PA systolic gradient < 20 mmHg, and No ventricular shunt	1
Good	RV systolic pressure 40-60 mmHg, or RV-PA systolic gradient 20–40 mmHg, or Small L → R ventricular shunt (Qp:Qs<1.5:1)	3
Satisfactory	RV systolic pressure 60–70 mmHg, or RV-PA systolic gradient < 50 mmHg, or Small L → R ventricular shunt (Qp:Qs < 1.5:1)	3
Unsatisfactory	RV systolic pressure > 70 mmHg, or RV-PA systolic gradient > 50 mmHg, or L → R ventricular shunt (Qp:Qs > 1.5:1)	7

PVR: pulmonary valve replacement, RV: right ventricle, PA: pulmonary artery, L → R: left to right, Qp: palmonary flow in L/min/m^2, Qs: systemic flow in L/min/m^2

Table 6. NYHA class before and after PVR

NYHA class	Before PVR (n=14)	After PVR (2 patients lost)
Class I	13	12
Class II	1	none

NYHA: New York Heart Association, PVR: pulmonary valve replacement

Cardiac catheterization

RV systolic pressure, RV-pulmonary artery (PA) systolic gradient, and Qp:Qs ratio was obtained prior to PVR (Table 5). The results were classified into 4 categories; excellent, good, satisfactory, and unsatisfactory. Seven patients were classified as unsatisfactory prior to PVR.

Survival and follow up

There was no early perioperative mortality. One patient suffered acute renal failure at early postoperative period, and received methylprednisolone therapy. Follow up data on two patients was unobtainable The remaining 12 patients were all in NYHA class I (Table 6). At the latest follow-up, there was no case of late death. Dyspnea was evident in one patient prior to PVR, but this disappeared after PVR. One patient exhibited sustained ventricular tachycardia before PVR and received ventricular cryoablation at the time of PVR. He suffered another episode of sustained ventricular tachycardia 2 years after PVR, and is currently on mexiletine. One patient underwent replacement of valved conduit due to conduit obstruction at 32 months after PVR.

DISCUSSION

Over 40 years have passed since the first successful corrective operation for TOF was performed, and many patients have survived into adulthood.[1)2)8-10)] As long term survival and quality of life have improved, the number of long term complications has increased proportionately.[11-14)] Some of these complications were due to the limitations of surgical techniques employed at the time of initial repair. In the past, surgeons inserted transannular patches at the time of repair in order to relieve RVOTO. Although still performed, the use of this procedure is less frequent than before because such transannular patches have commonly resulted in PR after repair of TOF.[13)] A large outflow patch in the RVOT can decrease effective stroke volume by expanding paradoxically during systole and diastole, causing obstruction. It can also lead to volume overload due to the return of retained blood to the right ventricle.[12)] Eight of our patients (57%) had had transannular patches inserted into their RVOT at the time of initial repair, and this may have been the cause of their PR and consequent RV volume overload (RVVO).

Early adverse effects of PR can go unnoticed if PR is solely evaluated on the presence of clinical symptoms. Burnell et al[15)] demonstrated that dogs with experimentally induced PR developed an increase in RVEDD and a decrease in cardiac output and ejection fraction. Long-term PR has been reported to cause right ventricular dysfunction well before overt clinical symptoms develop.[16)] The abnormal characteristics of surgically repaired RV, such as conduction disturbances, resection of muscle during repair, and hypertrophy, may add to the RV dysfunction. In the present study of 14 patients, all patients showed at least moderate degrees of PR and cardiomegaly on chest x-ray in the absence of any clinical symptoms. This is consistent with the finding that the evaluation of symptoms alone is not enough to detect RV dysfunction after repair of TOF.[12)17)]

If not corrected in time, PR can lead to RV dilatation, exercise intolerance, arrhythmias, and an increased incidence of sudden death.[4-6)16)17)] Therefore, management of PR is needed to preserve RV function before irreversible damage is done to the myocardium. The timely perfor-

mance of PVR has been reported by many researchers to bring both clinical and objective hemodynamic improvement in RV function.[18-21] In a study of 11 patients, Bove et al[16] showed the reductions in RV volume by the decrease in cardiothoracic ratios and RVEDD. In their review of 49 patients with valveless repair of RVOTO, Conte et al.[18] also reported reductions in cardiothoracic ratio and echocardiographic measurements of right ventricular/left ventricular end diastolic dimension. Ilbawi et al.[12,21] identified a significant reduction of RV end systolic volume in angiographic data. They also stressed the need for PVR within 2 years after the onset of hemodynamic deterioration to prevent long term deterioration in RV function.

Isolated PR after TOF repair is not a common finding, and the presence of lesions that further increase the RV afterload such as peripheral pulmonary artery stenosis could aggravate RV dilatation in addition to PR. In the additional presence of such lesions, PR is even less tolerated, and early PVR is especially important to preserve RV function.[12] Peripheral pulmonary artery stenosis can be easily identified in many TOF patients because they often show hypoplastic pulmonary vascularity. Previous palliative shunts also contribute to the distortion of pulmonary arteries. Residual obstructions of the pulmonary vascular bed may persist after surgical repair, and in coexistence with PR, the resulting RV hypertension combined with RVVO accelerates RV dysfunction. The adverse effects of ventricular hypertension in addition to volume overload have been demonstrated in several studies. Ruzyllo et al[11] stated that PR is poorly tolerated when combined with distal obstruction. The relatively short time interval occurring between initial TOF repair and PVR in our cases was possibly due to the poor tolerance of even a moderate degree of PR in the presence of distal obstruction.

Residual lesions such as VSD or TR both increase RV preload, and in addition to the already existing RVVO, further aggravate RV dysfunction. Five of our patients (35%) had residual shunts from VSD leaks, and underwent VSD patch leak repair at the time of PVR. TR secondary to RV dilatation also contributes to further RV dilatation, allowing a vicious cycle to be formed.[16,22]

In our study of 14 patients, one patient with dyspnea prior to PVR experienced an improvement in her symptoms after the operation. Prior to PVR, she had shown AV block on ECG, a moderate degree of PR on echocardiography, and an RV to pulmonary artery gradient of greater than 50 mmHg. All patients manifested significant reductions in cardiothoracic ratios after PVR, but only two showed a significant reduction in their RVEDD/BSA ratio. This discrepancy in data may be due to the method of RV measurement. Measuring RV volume is difficult because of its complex structure, and RVEDDs obtained from apical four chamber views could be oversimplifying. Helbing et al[24] states that even abnormally shaped RV can be measured accurately with multisection cine MRI. Transthoracic 2-D echocardiography could be used for the initial screening of patients requiring further evaluation by MRI.

Previous reports on PVR focused on patients older than 15 years as their study groups,[16,18-20,23] and showed reductions in their RV volumes. Ilbawi et al[21] reported that PVR within 2 years of the detection of hemodynamic deterioration is necessary to reverse the deleterious effects of PR on RV function. Patients who had PVR more than 2 years after the detection of hemodynamic deterioration did not show any evidence of improvement in RV function or exercise tolerance.[12,21]

We believe that the findings of our study deserve special attention because of the high percentage of subjects with associated branch pulmonary artery stenosis (50%)and VSD (35%) in addition to PR. In contrast, previously reported studies on PVR after TOF repair focused on patients with PR as their main lesion. This difference between the subjects of the study groups may explain the difference between the results of our study and those of the others.[16,18-20] Our patients underwent PVR at a younger age than those of the previous studies because of their rapid RV function deterioration due to the presence of associated lesions in addition to PR, as indicated by the relatively short time interval (2.31 ± 1.56 years) between initial repair and PVR.

The mean QRS duration did not change significantly after PVR. One patient received RV cryoablation at the time of PVR. He had undergone total correction for TOF at 10 months of age, following which he had suffered sustained ventricular tachycardia which required cardioversion 17 months after the initial repair. RV dysfunction was detected 3 months later, at which time the patient underwent PVR at age 30 months. However, he showed no significant reduction in RVEDD/BSA or QRSD after PVR. Persistent RV dysfunction has been shown to be associated with an increased risk of ventricular arrhythmias.[4] In the case of our patient, persistent RV dilatation probably caused recurrent episodes of ar-

rhythmia.

One patient underwent reoperation for conduit obstruction 34 months after initial PVR. At initial PVR he had not exhibited any associated branch pulmonary artery stenosis or VSD, and had had a pacemaker inserted for AV block. He underwent PVR with a Polystan valved conduit at the age of 4 yrs 11months, and the only risk factor associated with conduit obstruction may be his relatively young age at the time of PVR.[25] He showed a reduction in RVEDD/ BSA as well as cardiothoracic ratio after his initial PVR. Bielefeld et al[26] stated in their study of 223 children that reoperative homograft RVOT reconstruction demonstrates good midterm event-free survival with low morbidity and mortality.

Study limitations

Neither exercise testing nor VO_2 measurement were performed on our patients, which prevented an investigation by measurement of maximal exercise capacity into whether there was any objective improvement or deterioration in RV function. A further limitation arose from the method of RV volume measurement as the RVEDD/BSA ratio measured from apical 4 chamber view may not be an accurate indicator of true RV dimension. Radionuclide angiogram (RNA) would have yielded more information on postoperative RV function, including RV ejection fraction. RNA for evaluation of RV ejection fraction and RV volume has gained acceptance and is being applied in centers for the detection and quantification of impaired RV function and dilation.[12,27] In addition, magnetic resonance imaging has recently gained employment in an increasing number of centers for the assessment of ventricular function.[24]

The present study was also limited by the PVR performed on the subjects not being the primary operation, but rather an associated procedure to the main procedure for the correction of branch pulmonary artery stenosis or residual VSD leak.

CONCLUSION

It is our finding that PR after repair of TOF, when associated with lesions such as peripheral pulmonary artery stenosis or VSD, can lead to earlier deterioration in RV function. PVR performed concurrently with corrective surgery for associated lesions can preserve RV function if it is done before irreversible damage is suffered by the myocardium.

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