

# Management of Moderate Functional Tricuspid Valve Regurgitation at the Time of Pulmonary Valve Replacement: Is Concomitant Tricuspid Valve Repair Necessary?

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**Abstract** Congenital heart defects with a component of pulmonary stenosis are often palliated in childhood by disrupting the pulmonary valve. Patients often undergo subsequent pulmonary valve replacement (PVR) to protect the heart from the consequences of pulmonary regurgitation. In the presence of associated moderate functional tricuspid valve (TV) regurgitation, it is unclear what factors contribute to persistent TV regurgitation following PVR. In particular, it is unknown whether PVR alone will reduce the right ventricular (RV) size and restore TV function or whether concomitant TV annuloplasty is required as well. Thirty-five patients were analyzed. Each patient underwent initial palliation of congenital pulmonary stenosis or tetralogy of Fallot, underwent subsequent PVR between 2002 and 2008, and had at least moderate TV regurgitation at the time of valve replacement. Serial echocardiograms were analyzed. Pulmonary and TV regurgitation, along with RV dilation and dysfunction, were scored (0, none; 1, mild; 2, moderate; 3, severe). RV volume and area were also calculated. Potential risk factors for persistent postoperative TV regurgitation were evaluated. One month

following PVR, there was a significant reduction in pulmonary valve regurgitation (mean, 3 vs. 0.39;  $P < 0.0001$ ) and TV regurgitation (mean, 2.33 vs. 1.3;  $P < 0.0001$ ). There were also significant reductions in RV dilation, volume, and area. There were no significant further improvements in any of the parameters at 1 and 3 years. There was no difference in the degree of TV regurgitation postoperatively between those patients who underwent PVR alone and those who underwent concomitant tricuspid annuloplasty (mean, 1.29 vs. 1.31;  $P = 0.81$ ). We conclude that following PVR, improvement in TV regurgitation and RV size occurs primarily in the first postoperative month. TV function improved to a similar degree with or without annuloplasty.

**Keywords** Tricuspid valve regurgitation · Pulmonary valve replacement

Congenital heart defects having a component of pulmonary stenosis are often palliated in childhood by disrupting the pulmonary valve. This arrangement can provide an excellent quality of life as these children grow and enter young adulthood. Unfortunately, the pulmonary insufficiency that is created by disrupting the pulmonary valve results in volume overload of the right heart. This may eventually lead to right ventricular dilation [3]. Further complications include right heart failure, ventricular arrhythmias, and functional tricuspid valve (TV) regurgitation. Placement of a competent pulmonary valve at a subsequent operation is a means to eliminate the volume overload and prevent these further sequelae.

In the presence of associated functional tricuspid regurgitation (TR), the etiology is felt to be dilation of the right heart, stretching of the TV annulus, and subsequent

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failure of leaflet coaptation. Following pulmonary valve replacement (PVR), it is unclear what factors contribute to persistent postoperative TV regurgitation. In particular, it is unknown whether PVR alone will reduce the right ventricular (RV) size and restore TV annular size and function or whether concomitant TV annuloplasty is required as well.

## Methods

After internal review board approval and waiver of consent were obtained, patients who underwent PVR were reviewed. They were included in the analysis if they had a congenital diagnosis of pulmonary stenosis or tetralogy of Fallot (TOF), underwent PVR between 2002 and 2008, and had at least moderate TV regurgitation at the time of valve replacement.

### Population Description

Thirty-five patients met the criteria. Patients had an initial diagnosis of TOF (26 pts) and congenital pulmonary stenosis (9 pts). Although all patients underwent prior pulmonary valve disruption, the exact type of operation was difficult to obtain from old records. The median age at initial pulmonary valve disruption was 2.5 years (range, 0–39 years) and the median interval to subsequent replacement was 22.5 years (range, 0–43 years). In all patients, this was their first operation following the valve-disrupting procedure.

### Operative Description

The resultant age at PVR was 30.5 years (range, 2–64 years). All patients had severe pulmonary valve regurgitation by preoperative echocardiography. Twenty-eight patients received bioprosthetic valves, six patients received bioprosthetic valved conduits between the right ventricle and the pulmonary artery, and one patient received a mechanical valve. Conduits were used primarily for heavily calcified RV outflow tracts where suturing a bioprosthetic valve would be difficult. Although all patients had at least moderate TV regurgitation, only 16 of 35 (46%) underwent concomitant TV annuloplasty. Thirteen were suture annuloplasties and three were ring annuloplasties. A total of 25 concomitant procedures were performed in 20 patients.

The operations were performed by several congenital heart surgeons. The decision to perform tricuspid valvuloplasty was primarily based on surgeon preference. Influential factors likely included the absolute degree of TR, whether cardioplegic arrest would be required for

adequate repair, ease of sternal re-entry and right atrial dissection, and ease of bicaval dissection and cannulation.

### Imaging

Preoperative and postoperative echocardiograms were reviewed. The degree of pulmonary regurgitation, TR, RV dilation, and RV dysfunction were scored (0, none; 1, mild; 2, moderate; 3, severe). The RV volume and area were also calculated.

Echocardiography was performed by a single sonographer dedicated to the adult congenital program. TV regurgitation was assessed by the effective regurgitant orifice. An orifice less than 0.2 cm<sup>2</sup> was considered mild; 0.2 to 0.4 cm<sup>2</sup>, moderate; and greater than 0.4 cm<sup>2</sup>, severe. RV volume was determined from the apical four-chamber view by using a single plane by the method of discs, and area was determined by tracing the right ventricle at end diastole. Normal values for RV end-diastolic volume and area are approximately 30 cm<sup>3</sup> and 20 cm<sup>2</sup>, respectively. The degree of dilation was based on the values obtained from volume and area measurement.

Cardiovascular magnetic resonance (CMR) imaging was recently introduced into the routine preoperative evaluation of these patients. This strategy was adopted prior to the study period, such that most patients in this series underwent preoperative CMR. However, postoperative CMR is not routine, and the number of patients who underwent postoperative imaging was too low to allow for meaningful preoperative and postoperative comparison.

### Statistics

The Wilcoxon and Mann–Whitney rank sum tests were used for comparisons. A regression analysis was performed to identify risk factors for increased postoperative TV regurgitation. Because criteria for normalcy in the outcome variable were not initially met, the variable was transformed using a logarithmic transformation. Normalcy was subsequently tested and verified. A *P*-value < 0.05 was considered significant.

## Results

### Serial Evaluations for the Entire Population

Summary echocardiographic results over a 3-year period are shown in Table 1. There were significant reductions in both pulmonary valve regurgitation (mean, 3 vs. 0.39; *P* < 0.0001) and TV regurgitation (mean, 2.33 vs. 1.3; *P* < 0.0001) between the preoperative and the 1-month postoperative echocardiograms. There were also significant

**Table 1** Summary of echocardiographic findings (mean ± SD): 0, none; 1, mild; 2, moderate; 3, severe

	Preoperative	Postoperative		
		1 mo	1 yr	3 yr
Pulmonary valve regurgitation	3 ± 0	0.39 ± 0.69*	0.47 ± 0.81	0.82 ± 0.91
Tricuspid valve regurgitation	2.33 ± 0.44	1.3 ± 0.62*	1.53 ± 0.67	1.46 ± 0.54
RV dilation	2.25 ± 0.69	1.61 ± 0.79*	1.3 ± 0.77	1.29 ± 0.96
RV volume (cm <sup>3</sup> )	110.42 ± 42.57	66.3 ± 38.43*	65.33 ± 50.36	73 ± 37.99
RV area (cm <sup>2</sup> )	37.91 ± 8.18	27.2 ± 9.21*	25.33 ± 11.37	28.67 ± 10.95
RV dysfunction	1.64 ± 0.94	1.45 ± 0.95	1.47 ± 0.92	1.18 ± 1.01

Note. RV, right ventricle

\* Significant reduction between the preoperative and the 1-month postoperative parameters

reductions in RV size 1 month postoperatively based on RV dilation (mean, 2.25 vs. 1.61;  $P = 0.0002$ ), RV volume (mean, 100 vs. 66 cm<sup>3</sup>;  $P = 0.008$ ), and RV area (mean, 37 vs. 27 cm<sup>2</sup>;  $P = 0.015$ ). There was no significant further improvement in any of the parameters at 1 and 3 years.

Serial Evaluations for Separate Cohorts

Patients were divided into two groups. Table 2 shows the preoperative and 1-month postoperative echocardiographic parameters for those patients who underwent PVR alone and those who underwent concomitant TV annuloplasty. Again, there were significant reductions in pulmonary valve regurgitation, TV regurgitation, and RV dilation in each group.

Looking specifically at TV regurgitation, there was no difference in the resultant degree of TR 1-month postoperatively between those patients who underwent PVR alone and those who underwent concomitant TV annuloplasty (mean, 1.29 vs. 1.31;  $P = 0.81$ ). Even for the patients with severe TR preoperatively, the resultant TR 1-month postoperatively appeared similar between the nonannuloplasty group (mean = 1;  $n = 1$ ) and the annuloplasty group (mean = 1.3;  $n = 8$ ) (too low for statistical analysis).

Risk Factor Analysis (Table 3)

Initial diagnosis of TOF, higher degree of preoperative TV regurgitation, increased preoperative RV dilation, and not performing a concomitant TV annuloplasty were evaluated as potential risk factor for increased 1-month postoperative TV regurgitation. None were identified as risk factors in a multivariate analysis. Although the postoperative TR in patients with TOF was higher than in those with congenital pulmonary stenosis (mean, 1.4 vs. 0.89), it did not reach significance.

Outcomes

No patients have required subsequent pulmonary or TV surgery. There were no major surgical morbidities, but there were two surgical mortalities.

The first patient was a 48 year old with multiple medical problems including obesity, hypertension, cerebrovascular accident, elevated liver transaminases, and gastrointestinal bleeding. She underwent concomitant classic Glenn take-down (due to arteriovenous malformations and desaturations) with complex reconstruction. On postoperative day 12, she had an episode of massive pulmonary hemorrhage with cardiopulmonary arrest.

**Table 2** Echocardiographic comparisons (mean ± SD)

	PVR alone			PVR/TV annuloplasty		
	Preoperative	1 mo postoperative	<i>P</i> -value	Preoperative	1 mo postoperative	<i>P</i> -value
For at least moderate TR	<i>(n = 19)</i>			<i>(n = 16)</i>		
Pulmonary regurgitation	3.00 ± 0	0.41 ± 0.87	<b>&lt;0.0001</b>	3.00 ± 0	0.37 ± 0.47	<b>&lt;0.0001</b>
Tricuspid regurgitation	2.08 ± 0.26	1.29 ± 0.5	<b>0.0002</b>	2.63 ± 0.43	1.31 ± 0.75	<b>0.0002</b>
RV dilation	2.24 ± 0.56	1.53 ± 0.82	<b>0.002</b>	2.27 ± 0.86	1.70 ± 0.77	<b>0.012</b>
RV dysfunction	1.42 ± 0.83	1.03 ± 0.88	0.105	1.93 ± 1.04	1.9 ± 0.83	0.966
For severe TR	<i>(n = 1)</i>			<i>(n = 8)</i>		
Tricuspid regurgitation	3.00 ± 0	1.00 ± 0		3.00 ± 0	1.31 ± 0.88	

Note. PVR, pulmonary valve replacement; TV, tricuspid valve; RV, right ventricle, TR, TV regurgitation. Significant *P*-values in boldface

**Table 3** Risk factor analysis for increased postoperative tricuspid valve (TV) regurgitation (TR)

Characteristic	<i>N</i>	1 mo postoperative TR (mean ± SD)	<i>P</i> -value (multivariate)
Congenital diagnosis of tetralogy of Fallot			
Yes	24	1.46 ± 0.0666	0.06
No	9	0.89 ± 0.22	
Increased degree of preoperative TR	33		0.64
Increased degree of preoperative RV dilation	33		0.75
Concomitant TV annuloplasty			
No	17	1.29 ± 0.50	0.54
Yes	16	1.31 ± 0.75	

Note. RV, right ventricular

The second patient was a 63 year old who underwent concomitant three-vessel coronary artery bypass grafting. He developed respiratory insufficiency, ARDS, and rapid progression to multisystem organ. Care was withdrawn on postoperative day 7.

## Discussion

### Evaluation

All of our patients with pulmonary regurgitation underwent clinical history, physical examination, electrocardiogram (EKG), and echocardiogram. In addition, potential surgical candidates over 40 years of age underwent cardiac catheterization for coronary angiography. While these are all useful tools in evaluating these patients, studies have suggested that CMR imaging has become the gold standard imaging modality for the periodic evaluation and follow-up of patients with pulmonary regurgitation [2]. Unlike echocardiography, CMR works independently of geometric assumptions for evaluation of RV volume and function and provides essential and complementary data facilitating management and prognosis for these patients [2].

Currently, all patients undergo preoperative CMR imaging to analyze the right ventricle, pulmonary arteries, and sternal relationships for operative planning. However, we do not routinely obtain postoperative CMR, and the number of patients with preoperative and serial postoperative CMR images in this series was too low to allow for meaningful comparison.

### Indications for Surgery

Although the optimal timing of surgery remains a challenge and decisions need to be made on an individual basis, PVR should be performed in these patients prior to the development of irreversible ventricular dysfunction. Some

programs have set very specific recommendations for PVR in this patient population [3]. These include repaired TOF or similar physiology with moderate or severe pulmonary regurgitation (regurgitant fraction >25% measured by CMR) and two or more of the following criteria: RV end-diastolic volume index of  $\geq 160$  ml/m<sup>2</sup>, RV end-systolic volume index of  $\geq 70$  ml/m<sup>2</sup>, left ventricular end-diastolic volume index of  $\leq 65$  ml/m<sup>2</sup>, RV ejection fraction  $\leq 45\%$ , RV outflow tract aneurysm, and clinical criteria such as exercise intolerance, symptoms and signs of heart failure, cardiac medications, syncope, and sustained ventricular tachycardia [3]. The presence of other hemodynamically significant lesions such as moderate to severe TR, residual atrial or ventricular septal defects, and severe aortic insufficiency also triggered referral for surgery in patients with moderate or severe pulmonary regurgitation [3].

We have been relatively aggressive at recommending PVR in these patients, and our criteria follow those mentioned above.

### Whether to Operate on the Tricuspid Valve

In the presence of associated functional TR, is concomitant TV annuloplasty necessary? It is unclear whether PVR alone will reduce the RV size and restore TV annular size and function or whether concomitant TV annuloplasty is required as well.

Isolated TV disease is rare, and TV surgery is usually performed as a concomitant reconstruction procedure in addition to the correction of other cardiac pathologies [4]. Functional TR may be a consequence of left-sided valve diseases. One study showed that the correction of left-sided valve diseases without concomitant repair of functional TR was associated with significant late morbidity and mortality [8]. This occurred on account of progressive RV dysfunction and increasing need for reoperation.

Functional TR can also occur after repair of right-sided obstructive lesions. In another study, DeVega tricuspid

annuloplasty was performed for moderate or severe TR during correction of other heart defects, 88% of which were PVR or RV-to-pulmonary artery conduit replacement [5]. Early postrepair echocardiography quantified TR as absent or mild (34 patients; 81%), mild to moderate (4 patients), moderate (3 patients), and severe (1 patient). They concluded that DeVega tricuspid annuloplasty safely provided excellent relief of TR, usually in children undergoing PVR or conduit replacement. Unfortunately, this study did not have a nonannuloplasty control group for comparison.

In our study, there were no specific criteria used to select which patients underwent concomitant tricuspid annuloplasty. For those with at least moderate preoperative TR (grade  $\geq 2$ ), 19 underwent PVR alone and 16 underwent concomitant tricuspid annuloplasty. The resultant degree of TR at 1 month was similar between groups (mean, 1.29 vs. 1.31;  $P = 0.81$ ). Although the number was too small for statistical analysis, even those patients with severe preoperative TR (grade = 3) appeared to have a similar degree of TR at month. It appears that functional TR improves similarly following PVR with or without annuloplasty.

In a multivariate analysis, no risk factors were identified for increased TV regurgitation. During ventricular septal defect closure, the TV is at risk of injury, particularly when the septal leaflet is temporarily taken down to improve exposure [10]. However, most studies report only trivial or mild postoperative TV regurgitation, regardless of technique [1, 10]. Nonetheless, in patients with TOF, PVR may not improve TR if its etiology relates to prior VSD closure. In our series, although the postoperative TR in patients with TOF was higher than in those with congenital pulmonary stenosis (mean, 1.4 vs. 0.89), initial congenital diagnosis of TOF did not reach significance for increased postoperative TR. Other studies have shown the degree of preoperative TR and RV hypertension as risk factors for persistent TR following annuloplasty [5, 6, 9]. In our series, none of our other potential risk factors evaluated (higher degree of preoperative TV regurgitation, increased preoperative RV dilation, and not performing concomitant TV annuloplasty) significantly affected postoperative TR.

### Choice of Operation

Several studies have been performed comparing TV repair and replacement. One study compared 93 replacements (72 biologic, 21 mechanical) and 222 repairs [7]. In the replacement cohort, survival was 85% at 1 year, 79% at 5 years, and 49% at 10 years. In the repair cohort, survival rates were similar: 80% at 1 year, 72% at 5 years, and 66% at 10 years ( $P = 0.66$  vs. replacement). Another compared 310 repairs (74.5%) and 106 replacements (25.5%) [4]. Forty-five patients (10.8%) required a TV reoperation after

7.7  $\pm$  5.1 years. Freedom from TV reoperation 10 years after TV repair and replacement was 83%  $\pm$  3.6% and 79%  $\pm$  6.1%, respectively ( $P = 0.092$ ). From these and other studies, it appears that survival is similar and that the incidence of reoperation is low with no significant difference when the TV has been repaired or replaced.

Several techniques are available to correct functional TR. These include stitch annuloplasty (semicircular De Vega annuloplasty and lateral Kay annuloplasty), novel techniques such as edge-to-edge or clover technique and suture bicuspidization technique, flexible and rigid prosthetic rings, and flexible prosthetic bands [8]. One study compared the durability of TV annuloplasty techniques and identified risk factors for repair failure [6]. Seven hundred ninety patients who underwent TV annuloplasty for functional regurgitation using four techniques: Carpentier-Edwards semirigid ring, Cosgrove-Edwards flexible band, De Vega procedure, and customized semicircular Peri-Guard annuloplasty. Tricuspid regurgitation 1 week after annuloplasty was 3+ or 4+ in 14% of patients. Regurgitation severity was stable across time with the Carpentier-Edwards ring ( $P = 0.7$ ), increased slowly with the Cosgrove-Edwards band ( $P = 0.05$ ), and rose more rapidly with the De Vega ( $P = 0.002$ ) and Peri-Guard ( $P = 0.0009$ ) procedures. Risk factors for worsening regurgitation included higher preoperative regurgitation grade, poor left ventricular function, permanent pacemaker, and repair type other than ring annuloplasty. Tricuspid reoperation was rare (3% at 8 years). They concluded that TV annuloplasty did not consistently eliminate functional regurgitation, and across time regurgitation increased importantly after Peri-Guard and De Vega annuloplasties.

In our 16 patients who underwent TV annuloplasty, 13 were suture annuloplasties (De Vega) and 3 were ring annuloplasties. Although the numbers were too low to allow for comparison between techniques, overall the TR improved significantly at 1 month postoperatively. Importantly, the degree of TR remained stable without progression at 1 and 3 years.

### Conclusion

Following PVR, improvement in TV regurgitation and RV size occurs primarily in the first postoperative month. Initial diagnosis of TOF, higher degree of preoperative TV regurgitation, increased preoperative RV dilation, and not performing a concomitant TV annuloplasty were not identified as risk factors for increased postoperative TV regurgitation. TV function improves to a similar degree with or without TV annuloplasty.

## References

1. Bol-Raap G, Weerheim J, Kappetein AP, Witsenburg M, Bogers AJ (2003) Follow-up after surgical closure of congenital ventricular septal defect. *Eur J Cardiothorac Surg* 24(4):511–515
2. Bouzas B, Kilner P, Gatzoulis M (2005) Pulmonary regurgitation: not a benign lesion. *Eur Heart J* 26(5):433–439
3. Geva T (2006) Indications and timing of pulmonary valve replacement after tetralogy of Fallot repair. *Semin Thorac Cardiovasc Surg Pediatr Card Surg Ann* 9:11–22
4. Guenther T, Noebauer C, Mazzitelli D, Busch R, Tassani-Prell P, Lange R (2008) Tricuspid valve surgery: a thirty-year assessment of early and late outcome. *Eur J Cardiothorac Surg* 34(2):402–409
5. Kanter KR, Doelling NR, Fyfe DA, Sharma S, Tam VK (2001) De Vega tricuspid annuloplasty for tricuspid regurgitation in children. *Ann Thorac Surg* 72(4):1344–1348
6. McCarthy PM, Bhudia SK, Rajeswaran J, Hoercher KJ, Lytle BW, Cosgrove DM, Blackstone EH (2004) Tricuspid valve repair: durability and risk factors for failure. *J Thorac Cardiovasc Surg* 127(3):674–685
7. Moraca RJ, Moon MR, Lawton JS, Guthrie TJ, Aubuchon KA, Moazami N, Pasque MK, Damiano RJ Jr (2009) Outcomes of tricuspid valve repair and replacement: a propensity analysis. *Ann Thorac Surg* 87(1):83–88
8. Raja SG, Dreyfus GD (2009) Surgery for functional tricuspid regurgitation: current techniques, outcomes and emerging concepts. *Expert Rev Cardiovasc Ther* 7(1):73–84
9. Tatebe S, Miyamura H, Watanabe H, Sugawara M, Eguchi S (1995) Closure of isolated ventricular septal defect with detachment of the tricuspid valve. *J Card Surg* 10(5):564–568
10. Zhao J, Li J, Wei X, Zhao B, Sun W (2003) Tricuspid valve detachment in closure of congenital ventricular septal defect. *Tex Heart Inst J* 30(1):38–41