Objectives: In this study, we compare the follow-up results of two types of tricuspid valve repair and review the results of no repair in moderately severe tricuspid regurgitation (TR) along with mitral valve replacement or mitral and aortic valve replacement.

Patients and Methods: This prospective follow-up study, carried out on 106 consecutive patients, was conducted at the Department of Cardiac Surgery, Punjab Institute of Cardiology, Lahore, from September 1994 through August 2008. The patients were divided into three groups: suture bicuspidization, 36 (34%); modified De Vega's repair, 47 (44.3%); and no repair, 23 (21.7%). Freedom from moderate TR was compared among these groups by the Kaplan-Meier method.

Results: In the suture bicuspidization group, freedom from TR$^2$ was 97.2%, 77.8%, and 39.6% at 30 days and 7 and 14 years after surgery, respectively. In the modified De Vega group, it was 100% at the end of 7 years. In the no-repair group, it was 91.3%, 91.3%, and 61.6% at 30 days and 7 and 14 years after surgery, respectively. Overall in-hospital mortality was 3 (2.8%), with 4 late deaths after discharge. Preoperative right ventricular dysfunction, preoperative TR severity, preoperative mitral regurgitation, preoperative ejection fraction, and remnant TR following surgery were significant independent predictors of TR recurrence.

Conclusion: Repair by suture bicuspidization fails to prevent further annular dilatation and has no comparable results. Modified De Vega's repair is safe with superior results compared to other groups at the end of 7 years. Patients without significant annular dilatation in the no-repair group improved during follow-up once their left-sided valve lesion was corrected. (Ann Thorac Cardiovasc Surg 2010; 16: 417–425)

Key words: rheumatic heart disease, tricuspid regurgitation, tricuspid valve repair, suture bicuspidization, modified De Vega's repair

Introduction

Rheumatic heart disease (RHD) has a prevalence of 5.7 in 1000 in Pakistan. Half of the patients with rheumatic mitral valve disease requiring mitral valve replacement (MVR) have tricuspid regurgitation (TR) $\geq 2+$ (TR $\geq 2+$). TR resulting from left-sided valvular heart disease is usually functional secondary to chronic mitral valve disease, which causes pulmonary hypertension and right ventricular (RV) volume overload. Furthermore, increasing grades of TR lead to RV dilation and dysfunction and elevated right atrial pressure.
Organic tricuspid valve (TV) disease requires TV replacement, whereas functional or secondary TR can be corrected with TV repair. Functional TR was initially managed by a conservative approach, i.e., no repair. Uncorrected tricuspid valve disease, especially severe TR, and dilated annulus with concomitant mitral valve lesion have poor prognoses with high mortality and increased morbidity.

Suture bicuspidization is a simple technique that has been used to repair moderate to severe TR since 1965. De Vega’s semicircular annuloplasty has superseded it because of its better results. The selection of an appropriate repair has paramount importance in the management of functional TR. Although ring annuloplasty is a more durable valve repair, suture annuloplasty is the most commonly used technique. Modified De Vega’s repair is still advocated because of its good results.

Dealing with TR is a challenge because very few definitive clinical trials and guidelines for management are available, especially in patients with rheumatic fever.

Issues that need to be resolved are type of repair, when to repair, and whether it should be a simple repair or one with a supporting ring; questions must also be answered about patients with mild to moderate annular dilatation and moderate TR with normal valve pathology.

This study was designed to compare the follow-up results of two types of tricuspid valve repair (TVR) and the review results of no repair in moderately severe TR, along with MVR or mitral and aortic valve replacement (AVR).

We changed from the suture bicuspidization group to the modified De Vega’s group because in our suture bicuspidization group at 7 years postrepair, we had recurrences that we felt were due to a progressive dilatation of the anterior annulus of tricuspid valve.

**Materials and Methods**

From September 1994 through August 2008, prospective follow-up with clinical assessment, international normalized ratio (INR) measurement, and echocardiography were carried out on 106 consecutive patients having moderately severe TR undergoing mechanical heart valve replacement for rheumatic heart disease by the same surgeon. Follow-up ranged from 0.3 to 13.83 years (mean, 5.13 ± 4.06 years; median, 5.33). It was defined as immediate postoperative, midterm, and long term, i.e., the end of 30 days and of 7 years and 14 years, respectively. The patients were divided into 3 groups: group 1, suture bicuspidization, 36 patients; group 2, modified De Vega’s repair, 47; group 3, no repair, 23. The demographic details and diagnoses are given in Table 1. The mean age of these patients was 27.1 ± 9.2 years, which was similar in all the groups. The females totaled 56 (52.8%) and the males 50 (47.2%). Twenty-three (21.7%) of the 106 patients were in New York Heart Association (NYHA) functional classes III-IV. Severe pulmonary hypertension, with mean pulmonary artery systolic pressure (PASP) of 78.6 ± 20.7 mmHg, was present in groups 1 and 2. In group 3, the mean PASP was 84.4 ± 19.8 mmHg. Overall, 75 (70.8%) patients had TR4+ when undergoing surgery, and the remaining 31 (29.2%) had TR3+. TR4+ was present mostly in patients of suture bicuspidization and modified De Vega’s repair; patients in the no-repair group had TR3+. Atrial fibrillation, which was more frequently associated with 24 (66.7%) patients in the suture bicuspidization group, was present in 65 (61.3%) patients.

Prior to surgery, all patients underwent transthoracic and Doppler echocardiography with a view to assessing the severity of TR. For an assessment its severity, Doppler echocardiography was graded on a scale from 1+ to 4+ (1+ = mild, 2+ moderate, 3+ moderate to severe, and 4+ severe). Annular diameter was considered normal if it was 40 mm from anterosetal commissure to the anteroposterior commissure. If annular dilatation was ≥ 70 mm, the valve was repaired regardless of the TR severity of TR. Perioperative transesophageal echo was not used because we agree with Filsoufi et al. about the use of this modality to assess the severity of TR. The anesthetic causes a reduction in systemic vascular resistance as a result of vasodilatation; this will bring on a reduction in afterload that can cause an underestimation of TR. We agree that patients with severe TR > 3+ and annular dilatation should undergo TV repair. The issue is in patients with moderately severe TR without significant annular dilatation of TV.

A median sternotomy was performed. All patients undergoing TV repair were operated on with the use of standard cardiopulmonary bypass with systemic hypothermia and local hypothermia with topical ice slush. Aortic and bicaval cannulation was performed with snares. Before cannulation, the TV was assessed digitally for the extent of TR and coaptation of leaflets on the finger.

A standard right atrial approach was performed, and a tricuspid valve was assessed. We measured by ruler the tricuspid annulus from anterior septal commissure to anteroposterior commissure, and if the dimension was ≥ 70 mm, tricuspid valve repair was done.
Kays (suture bicuspidization), we plicated the posterior leaflet, using two 2-0 Ethibond double pledget mattress sutures (Ethibond Excel 2-0 sutures, Johnson & Johnson Intl., USA) from the anteroposterior commissure to posterior septal commissure along the posterior annulus. This was performed until June 2001.

From June 2001 we changed to the modified De Vega’s repair, using a 2/0 25-mm Ethibond suture from the anteroseptal to the posteroseptal commissure with autologous pericardial pledgets between each entry site into the annulus. This is then tied down on a 29-mm valve sizer. Initially we used a 20-ml syringe, as Ghanta et al. did. Following this, the TV is tested by instilling normal saline into RV, using a 50-mm bulb syringe to assess the repair. If it is found to be adequate, the right atrium (RA) was closed. The left ventricles was deaerated, and the patient was weaned from cardiopulmonary bypass (CPB) in the routine manner. After weaning, we digitally assessed the competence of TV via the RA appendage.

Patients were maintained at an INR of 2.5–3.5. All patients were assessed by two dimensional and color Doppler echocardiography (Toshiba 6000 Power Vision, Toshiba, Japan) preoperatively and postoperatively in the intensive care unit, and prior to discharge. Follow-up echocardiography was performed at 3 months and then yearly, unless otherwise indicated.

The primary endpoint was freedom from moderate or TR2+, and secondary endpoints were mortality (early and late) and prosthetic-valve related complications. A recurrence of TR was considered significant if there was evidence of TR > 2+. Early mortality was death within 30 days postoperatively or during the same hospital admission. Postoperative complications were defined according to criteria set by Edmunds et al.

### Table 1. Pre-, per-, and postoperative variables of the study population

<table>
<thead>
<tr>
<th>Variables</th>
<th>Suture bicuspidization n=36</th>
<th>Modified De Vega repair n=47</th>
<th>No repair n=23</th>
<th>Total n=106</th>
</tr>
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<tbody>
<tr>
<td>Age (mean years)</td>
<td>26.92 ± 8.78</td>
<td>27.1 ± 8.5</td>
<td>27.6 ± 11.4</td>
<td>27.1 ± 9.2</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>16 (44.4%)</td>
<td>24 (51.1%)</td>
<td>10 (43.5%)</td>
<td>50 (47.2%)</td>
</tr>
<tr>
<td>Female</td>
<td>20 (56.4%)</td>
<td>23 (48.9%)</td>
<td>13 (56.5%)</td>
<td>56 (52.8%)</td>
</tr>
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<td>NYHA Class</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>II</td>
<td>27 (75%)</td>
<td>33 (70.2%)</td>
<td>20 (87%)</td>
<td>80 (75.5%)</td>
</tr>
<tr>
<td>III</td>
<td>8 (22.2%)</td>
<td>12 (25.5%)</td>
<td>3 (13%)</td>
<td>23 (21.7%)</td>
</tr>
<tr>
<td>IV</td>
<td>1 (2.8%)</td>
<td>2 (4.3%)</td>
<td>0</td>
<td>3 (2.8%)</td>
</tr>
<tr>
<td>PA systolic pressure mean (mmHg)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TR grade</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TR3+</td>
<td>8 (22.2%)</td>
<td>3 (6.4%)</td>
<td>20 (87%)</td>
<td>31 (29.2%)</td>
</tr>
<tr>
<td>TR4+</td>
<td>28 (77.8%)</td>
<td>44 (93.6%)</td>
<td>3 (13%)</td>
<td>75 (70.8%)</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>24 (66.7%)</td>
<td>28 (60.9%)</td>
<td>13 (56.5%)</td>
<td>65 (61.3%)</td>
</tr>
<tr>
<td>Aortic cross-clamp time (mins)</td>
<td>57 ± 13.6</td>
<td>65.6 ± 29.6</td>
<td>43.5 ± 14.2</td>
<td>57.9 ± 23.8</td>
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<tr>
<td>CPB time (mins)</td>
<td>88.8 ± 38</td>
<td>92.1 ± 35.5</td>
<td>64.8 ± 19.8</td>
<td>85.1 ± 35.2</td>
</tr>
<tr>
<td>Operation done</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MVR</td>
<td>0</td>
<td>0</td>
<td>22 (95.7%)</td>
<td>22 (20.8%)</td>
</tr>
<tr>
<td>MVR + TV Repair</td>
<td>35 (97.2%)</td>
<td>28 (59.6%)</td>
<td>0</td>
<td>63 (59.4%)</td>
</tr>
<tr>
<td>MVR + AVR + TV Repair</td>
<td>1 (2.5%)</td>
<td>16 (34%)</td>
<td>0</td>
<td>17 (16%)</td>
</tr>
<tr>
<td>MVR + ASD + TV Repair</td>
<td>0</td>
<td>1 (2.1%)</td>
<td>0</td>
<td>1 (0.9%)</td>
</tr>
<tr>
<td>Redo MVR + TV Repair</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1 (0.9%)</td>
</tr>
<tr>
<td>Redo MVR + AVR + TV Repair</td>
<td>0</td>
<td>1 (2.1%)</td>
<td>0</td>
<td>1 (0.9%)</td>
</tr>
<tr>
<td>Mortality</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IN-HOSPITAL</td>
<td>2 (5.6%)</td>
<td>1 (2.1%)</td>
<td>0</td>
<td>3 (2.8%)</td>
</tr>
<tr>
<td>LATE</td>
<td>3 (8.3%)</td>
<td>0</td>
<td>1 (4.3%)</td>
<td>4 (3.8%)</td>
</tr>
<tr>
<td>Incomplete follow-up</td>
<td>7 (19.4%)</td>
<td>1 (4%)</td>
<td>0</td>
<td>8 (7.5%)</td>
</tr>
</tbody>
</table>

AVR, aortic valve replacement; ASD, atrial septal defect; CPB, cardiopulmonary bypass; MVR, mitral valve replacement; NYHA, New York Heart Association; PA, pulmonary artery; TR, tricuspid regurgitation; TV, tricuspid valve.
Statistical Analysis

The data were analyzed using the Statistical Package for Social Sciences, version 14.0 for Windows (SPSS, Inc., Chicago, IL, USA). Categorical variables were expressed as percentages and continuous variables as mean ± standard deviation. Freedom from moderate 2+ TR was compared between the 3 groups by the Kaplan-Meier method. Events were defined as death and valve-related complications. Independent predictors of TR recurrence were determined by means of Cox regression analysis. Backward elimination of nonsignificant determinants was employed in analyses. Significance was assumed for p < 0.05.

The variables entered in the multivariable model to determine the significant independent predictors of recurrent TR were age, gender, preoperative NYHA class, preoperative PASP, preoperative RV dysfunction, preoperative TR, TV repair type, preoperative mitral regurgitation (MR), preoperative ejection fraction (EF), and postoperative PASP.

Results

Mean aortic cross-clamp time of the three groups was 57.9 ± 23.8 minutes. It was longer in the modified De Vega repair group, 65.6 ± 29.6 minutes, compared to the suture bicuspidization group, 57 ± 13.6 minutes, and the no-repair group, 43.5 ± 14.2 minutes, because there were 16 (34%) triple valve procedures in the former, and patients mostly had isolated mitral valve replacement in the latter group. Mean cardiopulmonary bypass time was 85.1 ± 35.2 minutes (Table 1). All patients had prosthetic valve replacement at the mitral position, 63 (59.4%) had concomitant TV repair, and 22 (20.8%) had isolated MVR. Overall, 17 (16%) patients had triple valve surgery: 1 (2.5%) in the suture bicuspidization group and 16 (34%) in the modified De Vega group.

The main outcome measure was postoperative TR grade and progression of TR to > 2+.

Figure 1 has been drawn to follow suture bicuspidization patients according to TR severity. We have followed each patient according to TR severity over the entire study period. Preoperatively, 8 (22.2%) patients had TR3+, and 28 (77.7%) had TR4+. Postoperative improvement to TR0-1+ was immediately observed in 29 (80.5%) patients; deterioration to TR4+ was immediately observed in 1 (2.7%) immediately postoperatively, in 3 (8.3%) at midterm, and in 8 (22.2%) at the end of the study period (Fig. 1).

In the De Vega modified group, 2 (4.3%) patients had TR2+ in the immediate postoperative period, and both improved to TR0-1+ at midterm. Except for 1 patient who had incomplete follow-up, the remaining 46 (97.9%) patients had TR0-1+ at the end of midterm.

In the no-repair group, 20 (87%) patients had TR3+ preoperatively. In the same group, 21 (91.3%) had improvement to TR0-1+, and 2 (8.7%) had TR2+ at 30 days. The same status was maintained until midterm. One late death occurred at 8 years of follow-up because of a cerebrovascular accident. At the end of the follow-up, 19 (82.6%) had TR0-1+, 2 (8.7%) had TR2+, and 1 (4.3%) had TR3+.
Figure 2 shows a Kaplan-Meier analysis of freedom from moderate or TR2+. In the suture bicuspidization group, freedom from TR2+ was 97.2%, 77.8%, and 39.6% at 30 days, 7 years, and 14 years, respectively. In the modified De Vega group, freedom from TR2+ was 100% at the end of 7 years of follow-up. In the no-repair group, freedom from TR2+ was 91.3%, 91.3%, and 61.6% at 30 days, 7 years, and 14 years, respectively.

Overall in-hospital mortality was 3 (2.8%). Among them, two patients were in the suture bicuspidization group. One patient had refractory ventricular fibrillation and died on the 2nd postoperative day; the other had cardiac arrest secondary to low cardiac output and expired on the 5th postoperative day. The third patient in the modified De Vega group had triple-valve surgery and died on the 3rd postoperative day as a result of low cardiac output syndrome. There were 4 late deaths, 3 (8.3%) in the suture bicuspidization group and 1 (4.3%) in the no repair group. Two patients in the suture bicuspidization group had cerebrovascular accidents, and 1 had miliary tuberculosis. All 3 died within 5 years of operation, and there was no TR recurrence in these patients (Table 1). Eight (7.5%) patients had incomplete follow-ups, 7 (19.4%) in the suture bicuspidization group and 1 (4%) in the modified De Vega group.

Two patients underwent redo surgery because the aortic valve developed severe regurgitation warranting an AVR. They each had an MVR with a suture bicuspidization of the tricuspid valve and developed severe TR postoperatively. Both had intact Kay’s repair with dilatation of the anterior tricuspid annulus leading to TR4+; one patient was 7 years postoperative and the other was 9. In both, a modified De Vega repair was performed with uneventful recovery. The 6 remaining patients have TR4+ and are being managed with aggressive diuretic therapy.

Significant independent predictors of recurrent TR, derived according to the Cox proportional hazards model, were the presence of preoperative RV dysfunction, preoperative TR severity, preoperative MR, preoperative EF, and remnant TR just after surgery. In our study, preoperative PASP and postoperative PASP had nonsignificant association with TR recurrence (Table 2). However, preoperative and follow-up pulmonary artery systolic pressure in patients with and without recurrent TR in the 3 groups was identified. Patients having recurrence of TR in the no-repair group were observed to have higher preoperative and follow-up PASP, but in those with suture bicuspidization, preoperative PASP in TR recurrence patients was lower than in those having no recurrence. However, follow-up PASP was higher in patients having recurrence of TR than in those having no recurrence.
Discussion

Tricuspid valve disease with associated left-sided lesions, such as mitral or aortic valve disease, is associated with high mortality and increased risk of events. Tricuspid valve regurgitation secondary to left-heart pathology is mostly functional; thus correcting the left heart lesion leads to a removal of impetus for progressive regurgitation. In selected cases, having $\text{TR} \geq 2+$ without annular dilatation correction of mitral valve lesion leads to a decrease in TR severity and consequent improvement in morbidity and mortality.\(^1\)

In the current study, we prospectively followed up patients of moderately severe functional TR undergoing suture bicuspidization and modified De Vega repair. We compared both groups for TR recurrence and observed patients in the no repair group for recurrence of TR $> 2+$.

In our study, patients undergoing modified De Vega repair had least recurrence of TR. Our results are in accordance with previous studies.\(^2\)\(^\text{19-22}\) Bernal and colleagues\(^7\) reported 232 patients, most having RHD, undergoing De Vega annuloplasty followed for a mean duration of 6.8 years. At the end of the study, TR had disappeared or was mild in 95.5% of the patients. Bernal et al. concluded that suture annuloplasty was effective and reliable for treating TR because of left-sided valve lesions. In our study, none of our patients undergoing modified De Vega repair had a recurrence of TR at the end of 7 years of follow-up. There was no in-hospital or late mortality; however, 1 patient was lost during the follow-up. Bernal et al.\(^7\) observed an in-hospital mortality of 8.1% with a late mortality of 23.3%. At 12 years, the actual survival rate was 50.5% ± 6.1%, and the actual freedom from reoperation was 75.7% ± 7.3%. None of our patients in this group required a reoperation.

Morishita et al.\(^19\) performed a De Vega tricuspid annuloplasty for functional TR in 408 patients; 14 (3.4%) experienced early deaths and 63 (15.4%) late deaths. A favorable outcome with regard to TR recurrence was noted at 15 years with a 91.6% freedom from reoperation. The actual survival rate at 15 years after operation was 74%.\(^19\)

Aoyagi et al.\(^20\) reported a modified De Vega annuloplasty in 321 patients. The actual survival rate of freedom from reoperation on the tricuspid valve was 97.6%. It was further reported that surgical experience with the modified De Vega annuloplasty as a method of first choice found it to be a simple and reliable technique that resulted in a reduction of the severity of functional TR in 88.4% of patients.\(^20\) Similar results have been reported by Peltola et al.,\(^21\) which are comparable to ours.

In our series, we performed the modified De Vega repair (i.e., with pledgets between every suture) because this technique has been reported to have a lower risk of suture dehiscence and recurrence of TR.\(^17\) Tang et al.\(^17\) and McCarthy et al.\(^5\) compared the De Vega repair with ring annuloplasty and showed the placement of an annuloplasty ring in patients undergoing tricuspid valve repair to be associated with improved event-free survival. We
performed the modified De Vega repair instead of implanting a prosthetic ring or band because for a correction of functional TR in patients undergoing left-sided valve surgery, it is simpler, quicker, and inexpensive with fewer complications.2)

Suture bicuspidization was performed for the correction of functional TR in the initial days of our study. Ghanta et al.16) have reported 157 patients undergoing suture bicuspidization of the tricuspid valve. At 3 years postoperatively, TR was zero to mild in 75%, moderate in 11%, moderate to severe in 6%, and severe in 8%. These results were similar to patients undergoing ring annuloplasty. Ghanta and colleagues16) concluded that bicuspidization and ring annuloplasty both produce an effective and durable repair after 3 years of operation. Suture bicuspidization has proved to be as effective as ring annuloplasty in the midterm postoperative period. Nakano et al.23) have also reported a favorable long-term outcome as a result of suture bicuspidization for functional TR.22) In our series, freedom from TR2+ was 77.7% at midterm and 39.6% at long term. The failures at midterm and long term were due to the dilatation of the anterior leaflet, which was not plicated or supported. We feel that in whomever the tricuspid valve annulus is dilated beyond 70 mm, the dilatation will not regress with the correction of the left-sided heart valve lesion, and whatever reduction in TR was noted in the early postoperative to midterm was probably a result of the reduction in annular size secondary to posterior annular plication. We feel that because of persistent chronic rheumatic activity and ongoing rheumatic process, the annular dilatation continues over the years, and thus the recurrence of TR. In cases of this kind, a repair needs to be carried out, and annuloplasty is required to address the anterior annulus of TV. We also agree with Filsoufi et al.14) on the drawbacks of suture bicuspidization.

In the current study, 23 patients having functional TR had no repair. We observed freedom from TR2+ in 91.3%, 91.3%, and 61.6% immediately postoperative, at midterm, and at the end of follow-up, respectively, in patients in whom a conservative approach was used, i.e., no repair performed. This treatment option was reserved only for patients with either TR3+ or TR4+ and no significant annular dilatation. TR was secondary to mitral valve disease and increased pulmonary hypertension. In this group, functional TR was conservatively managed without any repair of the tricuspid valve.6) Recently ACC/AHA guidelines23) have suggested that functional TR without annular dilatation or significant pulmonary hypertension does not require repair.23) The incidence of recurrent TR immediately after surgery and during the midterm follow-up period was much less in the no-repair group compared to Kay’s annuloplasty group. The reason for this was a nondilated TV annulus in the no-repair group compared to a dilated TV annulus in Kay’s group. In our study, patients in the no-repair group had moderately severe TR without significant annular dilatation, i.e., < 70 mm or less than 2 times normal measured by transthoracic echo. Our study population had rheumatic etiology with severe pulmonary hypertension at the time of left-sided valve surgery. We have observed that patients who had a significant drop in pulmonary artery (PA) pressure after the correction of left-sided lesions showed significant reduction in TR grade and freedom from TR recurrence because there was no previous involvement of tricuspid annulus. However, those patients who had a nonsignificant drop in PA pressure had TR recurrence on long-term follow-up. The reason for this recurrence could be a failure of decrease in postoperative PA, which leads to progressive dilatation of RV and annular dilatation of the tricuspid valve leading to TR. These findings are supported by Fukuda et al.,24) who observed that an increase in the severity of pulmonary hypertension caused worse functional TR during midterm follow-up after TV annuloplasty.24) We believe it is not a risk factor in the repair group, whereas in the no-repair group there was significance of the pulmonary hypertension in patients with TR (4 patients). In this group, preoperative mean PASP was 93.3 ± 19.3; compared to PA pressures in groups 1 and 2, there was a significant difference of preoperative mean PA systolic pressures. The most probable reason is because patients having systemic pulmonary arterial pressures have a significant decrease in PA pressures, compared to those having suprasystemic PA pressures, because of elevated pulmonary vascular resistance that remains elevated even after the correction of left-sided valve lesions.25) Sadeghi et al.26) have reported that a reduction of RV pressure after thromboendarterectomy in patients having chronic pulmonary hypertension is associated with a reduction in functional TR. Therefore in our population, patients having functional TR improved, though deterioration in TR grade occurred only in those patients having some organic involvement of TV as a result of ongoing rheumatic process. It has also been observed that patients undergoing percutaneous transluminal mitral commissurotomy (PTMC) for mitral stenosis have a reduction of pulmonary hypertension and TR regression. Thus we believe that when the left-heart lesions are corrected, pulmonary hypertension and TR regresses.27) Dreyfus et
al.\textsuperscript{15} have advocated repairing all cases of moderate TR because they believe that annular dilatation is an ongoing process regardless of the severity of TR. We tend to disagree with this and believe that without significant tricuspid annular dilatation, once the left-sided lesion is corrected RV after load decreases, and then mild to moderate dilatation regresses as the right ventricular diameter returns to normal. Once it is greater than twice the normal size, it does not significantly decrease to decrease the TR, and a TV repair is imperative. We agree with Filsoufi et al.\textsuperscript{11} that if an organic lesion is present in the TV, it should be repaired, and if this is mild to moderate annular dilatation (i.e., $<2$ times normal), it can be left because it will correct itself once the left-sided lesion is corrected.

We do not agree with Boyaci et al.\textsuperscript{28}, who have demonstrated that preoperative significant functional TR without repair may persist regardless of any reduction in pulmonary artery pressure or the hemodynamic outcome of mitral valve surgery. In our study, we prospectively followed up patients undergoing TV repair and no repair and have observed that long-term results, especially the recurrence of TR, are similar in the two categories.

Moraca et al.\textsuperscript{29} reported that surgical treatment of tricuspid valve disease, regardless of the operative approach, is associated with significant morbidity and mortality. Using cohort-matched propensity analysis, the authors were unable to identify any beneficial outcomes favoring tricuspid valve repair over replacement. Thus they have recommended considering tricuspid valve replacement for patients in whom there is a reasonable chance for recurrence of regurgitation after repair. We have performed valve repair instead of valve replacement with adequate long-term results.

In our experience with tricuspid valve surgery for functional TR, we have observed that patients of functional TR having no significant annular dilatation can be left without any intervention, and those having annular dilatation can be optimally treated by modified De Vega repair because it is a simple and inexpensive technique with good long-term results.

**Conclusion**

Suture bicuspidization fails to prevent further annular dilatation and does not have comparable results. Modified De Vega repair is safe with superior results in comparison with other groups at the end of 7 years. Patients without significant annular dilatation in the no-repair group improved during follow-up once their left-sided valve lesion was corrected.

**Study Limitations**

Our study has several limitations. First, in the presence of latest annuloplasty rings for TV repair, we still advocate the modified De Vega repair. The reason for this is that because the population undergoing valve replacement for rheumatic heart disease belongs to a poor socioeconomic class and cannot afford the costly annuloplasty ring, which has been largely supported by American and Western authors. Second, a significant number of our patients in the suture bicuspidization group had incomplete follow-up, which could have potential confounding effects on the poor outcome in this group. We have advocated the superiority of the De Vega repair; however, we believe that further follow-up is required to draw this conclusion.

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