Surgical management of secondary tricuspid valve regurgitation: Annulus, commissure, or leaflet procedure?

Jose L. Navia, MD, a Edward R. Nowicki, MD, MS,a Eugene H. Blackstone, MD,a,b Nicolas A. Brozzi, MD,a Daniel E. Nento, MD,a Fernando A. Atik, MD,a Jeevanantham Rajeswaran, MSc, b A. Marc Gillinov, MD,a Lars G. Svennson, MD, PhD,a and Bruce W. Lytle, MDa

Objectives: Techniques employed today concomitantly with left-sided heart valve surgery address secondary tricuspid valve regurgitation at 3 anatomic levels—annulus, commissure, and leaflet—although success of these alone or in combination in eliminating tricuspid regurgitation is uncertain. Our objective was to assess the comparative effectiveness of these techniques in reducing or eliminating secondary tricuspid regurgitation.

Methods: From 1990 to 2008, 2277 patients underwent tricuspid valve procedures for secondary tricuspid regurgitation concomitantly with mitral (n = 1527, 67%), aortic (n = 180, 7.9%), or combined (n = 570, 25%) valve surgery. These included annulus (flexible prosthesis [n = 1052, 46%]), rigid prosthesis [standard = 387, 3-dimensional = 197; 26%], Peri-Guard annuloplasty [Synovis Surgical Innovations, St Paul, Minn; n = 185, 8.1%], and De Vega suture [n = 129, 5.7%]), commissure (Kay [n = 248, 11%]), and leaflet (edge-to-edge suture [n = 79, 3.5%] ± annulus or commissural) procedures. A total of 4745 postoperative transthoracic echocardiograms in 1965 patients were analyzed.

Results: By 3 months after surgery, only 32% of patients overall had no tricuspid regurgitation. However, by 5 years, this had decreased to 22%, and 3+/4+ tricuspid regurgitation had increased from 11% at 3 months to 17%. Patients with rigid ring annuloplasty alone, either standard or 3-dimensional, had the least increase of 3+/4+ tricuspid regurgitation (to 12% at 5 years) compared with either a commissural or leaflet procedure.

Conclusion: Rigid prosthetic ring annuloplasty, standard or 3-dimensional, provides early and sustained reduction of tricuspid regurgitation secondary to left-sided valve disease without need for an additional leaflet procedure. However, results are imperfect, possibly because other anatomic levels (subvalvular, papillary muscle, and right ventricular) contributing to its pathophysiology are unaddressed. (J Thorac Cardiovasc Surg 2010;139:1473-82)

Supplemental material is available online.

Secondary tricuspid valve regurgitation (TR) is frequent in patients with longstanding left-sided valve disease, particularly in the setting of severe pulmonary hypertension and atrial fibrillation.1 Uncorrected, moderate, or severe TR is associated with progressive heart failure and premature death;2 even if corrected, it is a marker of lower long-term survival. However, the optimal surgical technique to eliminate TR remains challenging.3 Techniques employed today concomitantly with left-sided heart valve surgery address TR at 3 anatomic levels—annulus, commissure, and leaflet—although the degree to which these alone or in combination are successful in sustained elimination of TR is uncertain. Therefore, the objective of this study was to assess comparative effectiveness of techniques addressing these anatomic levels in reducing or eliminating secondary TR.

PATIENTS AND METHODS

Patients

From January 1990 to January 2008, 2277 patients underwent left-sided valve surgery with a concomitant tricuspid valve repair (Table 1). The majority of patients underwent mitral valve procedures, and the predominant etiology was degenerative disease with regurgitation. Nearly half had previously undergone cardiac surgery.

Data were retrieved from the prospective Cardiovascular Information Registry (CVIR) and Echocardiography Database, both of which are approved for use in research by the institutional review board, with patient consent waived.
Anatomic Level of Repair and Procedure Groups

We categorize repair procedures by anatomic level: annulus (flexible and standard or 3-dimensional rigid prosthetic rings, Peri-Guard rings [Synovis Surgical Innovations, St Paul, Minn], and De Vega suture technique), commissure (Kay technique, and leaflet (edge-to-edge technique). These were performed at a single level or double levels (Table 2). From these combinations, we formed 8 groups for analysis: 6 isolated to 1 level (annular: flexible ring [standard and 3-dimensional], rigid ring, Peri-Guard ring, and De Vega technique; commissural: Kay technique) and 2 involving 2 levels (edge-to-edge plus prosthetic annuloplasty,6,7 edge-to-edge plus Kay technique); 6 patients with isolated edge-to-edge technique were not considered.

During the study period, number and variety of tricuspid valve repair procedures fluctuated (Figure 1). Early in the series, procedures were performed almost entirely at the annular level only, predominantly standard rigid prosthetic ring and Peri-Guard annuloplasty. Later, flexible prosthetic rings were introduced along with Kay commissuroplasty. In recent years, rigid rings, both standard and 3-dimensional, have increasingly displaced flexible rings, and edge-to-edge technique has been combined with both annular and commissural level procedures.

Assessment of Postoperative TR

Temporal trend of TR grades after the repair was assessed by transthoracic echocardiography (TTE). TTE was performed routinely before discharge and at the discretion of referring physicians during follow-up. Interpretation of follow-up echocardiograms was obtained at as many time points as available for each patient. A total of 4745 TTE records were available in 1965 patients (86% of the total population) with a median follow-up of 20 days (25th percentile 6 days, 75th percentile 1.4 years, range 1 day to 18 years) (Appendix E1). TR was graded 0 for no regurgitation, 1+ for mild, 2+ for moderate, 3+ for moderately severe, and 4+ for severe. Because of low frequency in grades 3+ and 4+, they were combined into 1 category, 3+/4+.

Temporal pattern of postoperative TR grade was estimated longitudinally using a nonlinear cumulative mixed model. With this model, a number of temporal phases were identified and estimates obtained for the shaping parameters of each phase. The SAS procedure PROC NL MIXED for longitudinal nonlinear cumulative logistic regression8 for repeated measurements was used for this (SAS Institute, Inc, Cary, NC). Because temporal patterns of TR differed for each repair technique, we analyzed these individually.

Risk factors for postoperative TR. Because of the limited capability of PROC NL MIXED to explore multivariable relations, we initially screened the variables listed in Appendix 1 using ordinary multivariable logistic regression (PROC LOGISTIC; SAS, Inc) and the assumption of independence of observations, with liberal entry (P = .2) and stay (P = .12) criteria. This analysis was performed simply to identify possible candidates for the repeated-measurements multivariable model. These candidates and their transformations, if any, were entered at once into the model, then eliminated one by one until all variables remaining had a P value of 0.1 or less.

Other Outcomes

Methods of analysis according to surgical management of TR: long-term tricuspid valve reoperation, survival, and requirement for permanent pacemaker; and estimated right ventricular (RV) systolic pressure and New York Heart Association (NYHA) functional class. This analysis was performed using a nonlinear cumulative mixed model with liberal entry (P = .2) and stay (P = .12). Because of the limited capability of PROC NL MIXED to explore multivariable relations, we initially screened the variables listed in Appendix 1 using ordinary multivariable logistic regression (PROC LOGISTIC; SAS, Inc) and the assumption of independence of observations, with liberal entry (P = .2) and stay (P = .12) criteria. This analysis was performed simply to identify possible candidates for the repeated-measurements multivariable model. These candidates and their transformations, if any, were entered at once into the model, then eliminated one by one until all variables remaining had a P value of 0.1 or less.
TABLE 1. Continued

<table>
<thead>
<tr>
<th>Variable</th>
<th>n*</th>
<th>No. (%) or mean ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Severe</td>
<td>348 (16)</td>
<td></td>
</tr>
<tr>
<td>Atrial fibrillation/flutter</td>
<td>2277</td>
<td>1,549 (68)</td>
</tr>
<tr>
<td>Complete heart block/pacer</td>
<td>2277</td>
<td>335 (15)</td>
</tr>
<tr>
<td>Ventricular arrhythmia</td>
<td>2277</td>
<td>428 (19)</td>
</tr>
<tr>
<td>Previous cardiac operation</td>
<td>2277</td>
<td>1,089 (48)</td>
</tr>
</tbody>
</table>

Noncardiac comorbidity

| PAD                                   | 2275 | 1,001 (44) |
|Stroke                                 | 2277 | 295 (13) |
|Hypertension                           | 2224 | 1,432 (64) |
|COPD                                   | 1984 | 636 (32) |
|Treated diabetes                       | 2256 | 444 (20) |
|Creatinine (mg/dL)                     | 2225 | 1.3 ± 0.94 |
|BUN (mg/dL)                            | 2233 | 27 ± 15 |

Left-sided valve procedure

| Mitral valve alone                    | 1,527 (67) |
|Aortic valve alone                    | 180 (7.9) |
|Aortic + mitral valve                 | 570 (25) |

Concomitant procedure

| CABG                                  | 775 (34) |
|Atrial fibrillation ablation           | 513 (23) |

SD, Standard deviation; NYHA, New York Heart Association; TR, Tricuspid valve regurgitation; n/a, not available; LV, left ventricular; PAD, peripheral arterial disease; COPD, chronic obstructive pulmonary disease; BUN, blood urea nitrogen; CABG, coronary artery bypass grafting. *Number of patients with data available. | Stenosis greater than mild and regurgitation none or mild. | Regurgitation greater than mild and stenosis none or mild. | Stenosis and regurgitation greater than mild. |

Presentation

Simple descriptive statistics were used to summarize the data. Continuous variables are presented as mean ± standard deviation and as 15th, 50th, and 85th percentiles. Categorical data are described using frequencies and percentages. Categorical outcomes were compared using either χ² or Fisher’s exact test and continuous outcomes by the Wilcoxon rank–sum (Kruskal–Wallis) nonparametric test. All analyses were performed using SAS statistical software (SAS version 9.1; SAS, Inc). Uncertainty is expressed by confidence limits equivalent to ±1 standard error (68%).

RESULTS

Postoperative TR, Overall

By 3 months after surgery, only 32% of patients had no TR, 34% had mild (1+) TR, 23% had moderate (2+) TR, and 11% had moderately severe to severe (3+ or 4+) TR (Figure 2). Thereafter, prevalence of 2+ and greater TR slowly increased and 0 TR decreased, such that by 5 years, 22% of patients had no TR, 33% had mild TR, 28% had moderate TR, and 17% 3+ or 4+ TR.

Postoperative TR by Anatomic Procedure Level

The temporal pattern of 3+ or 4+ postoperative TR varied considerably among the 8 procedure groups (Figure 3).

Isolated procedures at the annular level, with the exception of Peri-Guard, had similar patterns of postoperative TR, with an early rapid increase followed by a slower later increase. Of these, patients with either standard or 3-dimensional rigid prosthetic ring annuloplasty had the least increase across time, to 10% and 14% by 5 years, respectively (P = .7), compared with 16% for those receiving flexible prosthetic rings and 24% for those with De Vega annuloplasty. The isolated commissure-level Kay technique was associated with a temporal pattern of TR similar to that of flexible rings, reaching 19% 3+/4+ by 5 years. Data for 2-level procedures involving a leaflet-level procedure (edge-to-edge technique) are limited to 2 years because of their recent introduction, but to date follow different patterns of TR return. When an edge-to-edge procedure was combined with a Kay procedure, initial prevalence of 3+/4+ TR was low (2.4% at 3 months) but then rose to 24% by 2 years. In contrast, the pattern of 3+/4+ TR prevalence after an edge-to-edge technique combined with either rigid or flexible prosthetic annuloplasty followed that of an isolated rigid prosthetic ring, with a prevalence of 8% at 3 months and 8.8% at 2 years.

Risk Factors for Postoperative TR

In addition to surgical management strategies for TR, other factors increasing the risk of recurrent TR included higher grade of preoperative TR, larger tricuspid annuloplasty ring size (larger tricuspid valve), presence of pacemaker leads through the tricuspid valve, mitral valve replacement rather than repair, poor left ventricular (LV) function, and increased LV remodeling (Table 3).
Other Outcomes

Differences observed among the 7 repair groups with respect to postoperative morbidity and mortality did not demonstrate a consistent pattern (Appendix E3). Twenty-nine patients underwent a tricuspid valve reoperation, with freedom from reoperation of 98% at 5 years overall (Appendix E4). Risk of reoperation was similar among procedure groups ($P > .5$; Figure 4). Overall survival was 83%, 64%, and 44% at 1, 5, and 10 years, respectively (Appendix E5). In unadjusted comparison, De Vega, Peri-Guard, and annulus plus leaflet repair groups appeared ($P_{[(\log–rank)]} = .05$) to have lower survival than patients receiving flexible and rigid annuloplasty rings alone (Figure 5).

Permanent pacemakers were present in 363 patients before operation, and 203 additional patients received a pacemaker during follow-up. Freedom from postoperative pacemaker insertion was 90%, 89%, and 87% at 30 days, 1 year, and 5 years, respectively. Patients with an annulus plus leaflet procedure tended ($P = .07$) to have greater pacemaker requirement (Appendix E6), and 18 of 20 without

![FIGURE 1](image1.png)

**FIGURE 1.** Tricuspid valve procedures across time. C-E, Carpentier–Edwards rigid ring; MC$^3$, Edwards MC$^3$ Annuloplasty System 3-dimensional ring.

![FIGURE 2](image2.png)

**FIGURE 2.** Overall temporal trend of postoperative tricuspid regurgitation (TR) grades after tricuspid valve repair. Solid lines represent parametric estimates of percentage of patients (mean effect) in each TR grade. Symbols represent data grouped without regard to repeated measurements within time frame to provide a crude verification of model fit.
preoperative permanent pacemakers (n = 11) and a mitral valve procedure had a surgical approach to the mitral valve through the atrial septum (90%).

Estimated RV systolic pressure changed minimally after operation. Leaflet procedures appeared to have slightly higher pressure than annuloplasty rings alone or a De Vega annuloplasty (Appendix E7).

Among the one third of patients with longitudinal assessment of NYHA functional class, those receiving a Peri-Guard annuloplasty alone appeared to have better functional status ($P = .0002$; Appendix E8).

**DISCUSSION**

**Principal Findings**

Regardless of anatomic level of procedure—annulus, commissure, or leaflet, alone or in combination—we observed early return of TR, and severe TR progressively increased over time. However, the temporal pattern and degree of return of TR varied with procedure. A standard or 3-dimensional rigid prosthetic annuloplasty alone provided the best early and sustained reduction of TR, without need for a procedure at a second level. The disappointing finding that secondary TR is not permanently eliminated by these procedures is consistent with observations of others.\(^9,10\)

**Historical Note**

For many years it was believed that secondary TR disappeared after mitral valve surgery alone,\(^11\) and consequently, it was ignored.\(^12\) Thus, for example, Braunwald, Ross, and Morrow\(^11\) in 1967 recommended conservative nonsurgical management of “functional” TR. In the 1980s it was observed that patients who had undergone successful mitral surgery sometimes returned years later with severe symptomatic TR. When these patients underwent reoperation, mortality was high.\(^13\) More recently, compelling data have shown that surgically untreated secondary TR can persist or even worsen despite correction of the associated left-sided lesion,\(^14\) suggesting that an aggressive approach toward secondary TR is warranted.\(^10,13,15\)

**Pathogenesis of Secondary TR**

Secondary TR associated with left-sided valve disease is thought to be caused by RV dilatation,\(^16-18\) with enlargement and distortion of the tricuspid annulus and tethering of the tricuspid leaflets. This causes rising left atrial pressure that is transmitted through the lungs as pulmonary arterial hypertension, resulting in pressure overload on the RV. TR itself leads to further RV dilatation and dysfunction, more tricuspid valve annular dilation and tethering, and worsening TR. Eventually, the RV fails, causing increased RV diastolic pressure and a shift of the interventricular septum toward the LV. Because of ventricular interdependence, this might compress the LV, causing restricted LV filling and increased LV diastolic and pulmonary artery pressure.\(^19\) Increased left atrial size and pressure might also cause atrial fibrillation, which in turn causes right atrial dilatation leading to further tricuspid annular dilation. Atrial fibrillation has been recognized as an important risk factor for the development of TR in patients with mitral valve disease as well as for the persistence or occurrence of TR after mitral valve surgery or balloon mitral valvotomy.\(^2\) The majority of patients in the study were in atrial fibrillation.
TABLE 3. Risk factors associated with increased likelihood of higher postoperative TR grade

<table>
<thead>
<tr>
<th>Factor</th>
<th>Estimate ± SE</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overall</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Higher grade of preoperative TR</td>
<td>0.46 ± 0.062</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>Female</td>
<td>0.71 ± 0.12</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>Mitral valve replacement</td>
<td>0.40 ± 0.11</td>
<td>.0003</td>
</tr>
<tr>
<td>Early phase</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Surgical management of TR (</td>
<td></td>
<td></td>
</tr>
<tr>
<td>compared with rigid ring)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Flexible ring alone</td>
<td>0.41 ± 0.13</td>
<td>.02</td>
</tr>
<tr>
<td>Peri-Guard alone</td>
<td>0.011 ± 0.35</td>
<td>&gt;.9</td>
</tr>
<tr>
<td>De Vega alone</td>
<td>6.9 ± 3.02</td>
<td>.02</td>
</tr>
<tr>
<td>Kay procedure alone</td>
<td>7.5 ± 3.02</td>
<td>.03</td>
</tr>
<tr>
<td>Annulus + leaflet</td>
<td>−0.47 ± 0.404</td>
<td>.2</td>
</tr>
<tr>
<td>Kay + leaflet</td>
<td>6.7 ± 3.04</td>
<td>.03</td>
</tr>
<tr>
<td>Larger annuloplasty ring size*</td>
<td>5.7 ± 2.7</td>
<td>.03</td>
</tr>
<tr>
<td>Lower LV EF†</td>
<td>−0.78 ± 0.14</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>African American</td>
<td>0.52 ± 0.19</td>
<td>.005</td>
</tr>
<tr>
<td>History of heart failure</td>
<td>0.43 ± 0.14</td>
<td>.002</td>
</tr>
<tr>
<td>Lower body surface area]</td>
<td>1.4 ± 0.78</td>
<td>.07</td>
</tr>
<tr>
<td>Late phase</td>
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<tr>
<td>Surgical management of TR (</td>
<td></td>
<td></td>
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<tr>
<td>compared with rigid ring)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Flexible ring alone</td>
<td>0.72 ± 0.21</td>
<td>.0007</td>
</tr>
<tr>
<td>Peri-Guard alone</td>
<td>2.3 ± 0.40</td>
<td>&lt;.0001</td>
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<tr>
<td>De Vega alone</td>
<td>−0.64 ± 5.6</td>
<td>.9</td>
</tr>
<tr>
<td>Kay procedure alone</td>
<td>−1.7 ± 5.6</td>
<td>.8</td>
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<td>Larger annuloplasty ring size*</td>
<td>−1.7 ± 4.9</td>
<td>.7</td>
</tr>
<tr>
<td>Lower GFR§</td>
<td>0.81 ± 0.21</td>
<td>.0002</td>
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<tr>
<td>Preoperative permanent pacemaker</td>
<td>1.3 ± 0.29</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>At least one coronary artery</td>
<td>0.97 ± 0.22</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>system diseased</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Larger LV mass index</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

TR, Tricuspid regurgitation; SE, standard error; LV EF, left ventricular ejection fraction; GFR, glomerular filtration rate; LV, left ventricle. *Log(annuloplasty ring size), logarithmic transformation. †Log(LV EF), logarithmic transformation. ‡(1/body surface area), inverse transformation. §(60/GFR), inverse transformation. ||(LV mass index/120)², squared transformation.

**Surgical Techniques for Secondary TR**

**Annu1478 lus level.** Tricuspid annular dilatation has long been recognized as a constant feature of secondary TR and thus has been the primary target for intervention. Different surgical methods to treat or prevent secondary TR have been used at the annulus level: prosthetic ring annuloplasty introduced by Carpentier and colleagues21 and suture annuloplasty introduced by De Vega and colleagues.4 Recent long-term studies suggest that ring annuloplasty is more durable than suture annuloplasty. Data from the surgical literature suggest that more than 85% of patients having a ring annuloplasty will be free from 2+ or greater TR 10 years after surgery.3,14

A number of series have reported high recurrence of TR after the De Vega technique, particularly in patients with severe tricuspid annular dilatation or pulmonary hypertension.3,22

A randomized study of 159 patients comparing Carpentier ring annuloplasty with De Vega suture annuloplasty demonstrated a higher recurrence of 2+ or greater TR in the De Vega group at 46-month follow-up (Carpentier 4 of 40, De Vega 14 of 41; P < .0001).22 Recently, 3-dimensional ring annuloplasty has been introduced to address deformation of the tricuspid annulus.23

**Commissural level.** Suture bicuspidization was originally described by Kay, Maselli-Campagna, and Tsuji in 1965. Ghanta and colleagues24 demonstrated that bicuspidization (commissuroplasty) and ring annuloplasty were equally effective and durable at reducing TR up to 3 years postoperatively. Severe TR occurred in 8% of patients early after annuloplasty. At midterm, 69% of patients with ring annuloplasty and 75% of those with bicuspidization commissuroplasty remained free of moderate or greater TR.

**Leaflet level.** Fukuda and colleagues25 suggested that annuloplasty alone may reduce leaflet tethering to a level, but might not suffice for severe leaflet tethering. Thus, on the basis of these insights, we began to perform edge-to-edge leaflet suture combined with prosthetic ring annuloplasty.6,7 Our results demonstrate that inclusion of this leaflet-level technique does not provide additional benefit to rigid ring annuloplasty alone.

**Why Is Permanent Elimination of Secondary TR Difficult?**

Our study demonstrates that current approaches to addressing secondary TR at annular, commissural, and leaflet levels are insufficient to eliminate TR. We speculate that this may be due to imperfect annuloplasty or failure to address other anatomic considerations and levels contributing to its pathophysiology. The normal tricuspid annulus is saddle-shaped, with its highest points in anteroposterior orientation and lowest points in mediolateral orientation. In secondary TR, the annulus becomes dilated, flattened, and circular.17,18 Fukuda and colleagues25 additionally demonstrated an asymmetric reduction in tricuspid annular contraction in patients with secondary TR. This may be important if it is a critical pathophysiologic factor in secondary TR development, in which case the presence of annular dilatation predisposes a patient to future TR. Interestingly, in patients with chronic pulmonary thromboembolic hypertension in whom TR resolved after pulmonary thromboendarterectomy and in patients who had successful mitral balloon valvotomy, there was no change in tricuspid annular dilatation after resolution of pulmonary hypertension. This implies that tricuspid annular dilatation might be irreversible, which may be the cause of late TR in mitral valve disease. Secondary remodeling of the RV leads to conformational change of the trabecular septomarginals, resulting in displacement of the free wall and outflow tract of the RV and the entire subvalvular apparatus, predominantly of the anterior leaflet. These levels—chordae, papillary muscle, and RV wall—are unaddressed by any current surgical technique that targets only annular, commissural, or leaflet levels.

1478 The Journal of Thoracic and Cardiovascular Surgery • June 2010
The high incidence of permanent pacemaker requirement in the edge-to-edge group could be related to the fact that these patients had an incision over the dome of the left atrium to approach the mitral valve. As described by Guiraudon, Ofiesh, and Kaushik, this sacrifices the sinoatrial node artery in 100% of patients and increases the occurrence of early transient nodal rhythm. Kumar and colleagues noted that transient junctional rhythm and conduction abnormalities were common early postoperatively.

Limitations
This is an observational clinical cohort study in which different surgical techniques were used to address secondary TR, introduced at different times, in part because of dissatisfaction with effectiveness, and surgeon preference. As in all previous studies, detailed analyses of RV morphology and function were lacking. Postoperative assessment of TR relied on opportunistic TTEs, which could lead to ascertainment bias and overestimation of prevalence of higher TR grades.

CONCLUSIONS
Rigid prosthetic ring annuloplasty, standard or 3-dimensional, provides early and sustained reduction of TR secondary to left-sided valve disease without need for an additional leaflet procedure. However, results are imperfect, possibly because other anatomic levels (subvalvular, papillary muscle, and RV) contributing to its pathophysiology are unaddressed.

FIGURE 4. Freedom from reoperation (Kaplan–Meier estimates) according to tricuspid valve (TV) procedure. Vertical bars represent 68% confidence limits (equivalent to ±1 standard error).

FIGURE 5. Survival after tricuspid valve repair according to procedure performed. Format is as in Figure 4.
References

APPENDIX 1: Variables Used in Analyses
Preoperative Variables
Demographic: Age (years), gender, race, weight (kg), height (cm), body surface area (m²), body mass index (kg · m⁻²), race
Symptoms: NYHA functional class (I–IV), emergency operation
LV function/structure: Degree of LV dysfunction (1 = none, 2 = mild, 3 = moderate, 4 = severe), ejection fraction (%), previous myocardial infarction, inner diameter in diastole (cm), inner diameter in systole (cm), diastolic volume (mL), systolic volume (mL), diastolic volume index (mL · m⁻²), systolic volume index (mL · m⁻²), fractional shortening, relative wall thickness (wall stress), mass (g), mass index (g · m⁻²), posterior wall thickness (cm), septal thickness (cm)
Left atrial structure: Diameter (cm), volume (mL), volume index (mL · m⁻²)
Valve pathology: Aortic valve regurgitation, aortic valve stenosis, mitral valve regurgitation, mitral valve stenosis, pulmonary valve regurgitation
Coronary anatomy: Left main trunk disease (% stenosis), left anterior descending coronary artery system disease (maximum % stenosis), right coronary artery system disease (maximum % stenosis), left circumflex coronary artery system disease (maximum % stenosis)
RV function: TR, RV systolic pressure (mm Hg)
Other cardiac comorbidity: Atrial fibrillation, hypertension, history of cardiac disease, complete heart block/pacer, preoperative permanent pacemaker, ventricular arrhythmia, endocarditis
Noncardiac comorbidity: Treated diabetes, insulin-treated diabetes, history of peripheral arterial disease, history of smoking, carotid disease, popliteal disease, creatinine (mg · dL⁻¹), blood urea nitrogen (mg · dL⁻¹), bilirubin (mg · dL⁻¹), creatinine clearance, glomerular filtration rate, hematocrit (%)
Experience: Date of operation (years since January 1, 1990)
Procedure: Tricuspid valve (rigid ring [Carpenter–Edwards, McCarthy–Carpenter, IMR ETlogix; Edwards Lifesciences, Irvine, Calif], flexible ring [Cosgrove–Edwards, TAB Tailor; St Jude Medical, Inc, St Paul, Minn], PeriGuard, De Vega, Kay, edge-to-edge [double or triple leaflet with annuloplasty or Kay]), mitral valve repair/replacement,
aortic valve repair/replacement, coronary artery bypass grafting, surgical ablation for atrial fibrillation

Discussion

Dr Farzan Filsoufi (New York, NY). I have a disclosure; I am a speaker for Edwards Lifesciences. First, I congratulate you, Dr Navia, for this important study, which includes a large cohort of patients. I would also like to remind our audience that the first part of this study, from 1990 to 1999, which included 800 patients, was presented by Dr McCarthy at the Eighty-third Annual Meeting of The American Association for Thoracic Surgery and published in this Journal in March 2004.

I think the main conclusions of your work, with which I agree, is that the remodeling annuloplasty with either a Carpentier & Edwards semirigid ring or an MC ring (Edwards Lifesciences) is superior to other techniques such as suture annuloplasty and flexible band annuloplasty, and, as Dr Tirone David has shown, it has been associated with an improved long-term survival. I think the second important conclusion is that the palliative techniques, such as edge-to-edge repair technique, do not play any role in further improving outcome after a remodeling annuloplasty. Finally, although remodeling annuloplasty is superior to other techniques, it is associated with an early failure rate of about 10% to 15% in your study. This has not been our experience at Mount Sinai Medical Center. Therefore, most of my discussion will be directed to our understanding of the mechanism of this early failure.

First, as you know, most patients with functional TR have isolated annular dilatation, type I dysfunction, or restricted leaflet motion, type IIb dysfunction, with associated annular dilatation. Have you observed any difference of outcome, particularly with respect to early or late failure, between these 2 groups of patients, and have you been able to identify any risk factors such as RV dysfunction, enlargement, or pulmonary hypertension for this early failure?

Dr Navia. Thank you for your comments. We have not found any distinctions about this pathologic study in terms of regurgitation. In terms of the treatment, I agree with you; I think the best behaved ring is the rigid ring, and then also the 3-dimensional rings behaved the same. We have not observed any differences of early or late TR between these 2 groups.

Dr Filsoufi. My second question is regarding the ring sizing. What criteria do you use for ring sizing and do you make any difference in patients with type A versus type IIb, particularly with regard to undersizing?

Dr Navia. That is a good question. We do not make any different criteria for sizing between type A and type IIb of the Carpentier classification. We usually undersize the ring. We usually size by the surface area of the septal leaflet and, based on that, we place the ring; the choice of the specific ring depends on the surgeon preference.

Dr Filsoufi. In the article published in 2004 by Patrick McCarthy, which reported your cohort again from 1990 to 1999, the majority of patients who underwent a semirigid annuloplasty had a size 32 mm or 34 mm, and they were not undersized. In the discussion of this same manuscript, Dr McCarthy mentioned that undersizing was commonly performed, at least in patients with cardiomyopathy, during the second part of your study period. Do you have any data comparing patients who have a 2-size remodeling annuloplasty versus an undersized remodeling annuloplasty?

Dr Navia. No, we do not have any data for that.

Dr Filsoufi. My fourth question is this: To prevent the recurrence of early TR, do you use any adjunctive procedures such as anterior leaflet patch extension, as suggested by Gilles Dreyfus?

Dr Navia. This is a very important issue. We do not use any kind of leaflet extension with bovine or autologous pericardium, but I think we need to start thinking about new procedures. I do not think the ring itself is completely effective in reducing TR completely. The other component that we miss here is the ventricle, and we need to start thinking of the subvalvular apparatus and the ventricle geometry and which role they play on secondary TR in this population. I think with just the ring itself, TR can be reduced but not completely eliminated.

Dr Filsoufi. My final question is this: In the presence of recurrent 3þ to 4þ early after the operation, how do you use this information? What is the percentage of patients who had reoperations and what type of procedure do you perform?

Dr Navia. This is a very good question. The rate of 3þ to 4þ TR before discharge is very low, but when it happens, I think these patients need to return to the operating room. If the valve cannot be repaired again, it must be replaced. We usually use a tissue valve. I think it is safer to replace the valve than to send the patient home with the risk that he or she will return with full right heart failure. Many of these patients (eg., 48% of our patients) require a reoperation, and this will be the third one; they have a very high risk of morbidity and mortality—between 30% and 40%. Therefore, I think if you have patients with 3þ to 4þ TR before discharge, you have to return them to the operating room before sending them home.

Dr Norberto De Vega (Malaga, Spain). I congratulate the authors for such a good study. I have some comments and questions for Dr Navia.

Nowadays, we do not have any problem with how to treat the tricuspid valve. We always use a properly performed suture annuloplasty, but we think that it is completely different if it is a rheumatic or a degenerative disease. In patients with rheumatic disease, we do not think there is such a clear-cut difference as we thought in the past among organic and functional regurgitation.

For the past 15 years, we have visually explored every tricuspid valve of our patients with rheumatic mitral disease, and in most of them, even in those without regurgitation at that time, we found some subtle changes in the leaflets that probably progress with the passing of time and are responsible for the TR that appears late after left heart valve surgery. To avoid that, we have proposed to fix the tricuspid annulus in every case to break the vicious circle triggered by any degree of TR and in which annular dilatation plays an essential role.

Do you agree that there is no clear-cut difference, in patients with rheumatic disease, between functional and organic tricuspid disease?

Second, when those patients come back late after mitral surgery with isolated TR, do you have any clue to differentiate patients who are going to benefit by an operation from the ones who will not?

Dr Navia. Thank you for the question, Dr De Vega. These certainly are 2 different patient groups. However, we analyzed your technique in these 2 populations, rheumatic and degenerative. We have more patients with degenerative than rheumatic disease,
and both had the same rate of failure in our experience. Already, 24% of the patients have a recurrent 3+ to 4+ TR at 5 years with the De Vega technique.

Dr Tirone E. David (Toronto, Ontario, Canada). I enjoyed your paper very much. Years ago, your cardiologists published the importance of tethering of the tricuspid leaflets on the outcome of tricuspid annuloplasty. They have been telling us that surgeons are overlooking tethering of the anterior and posterior leaflets, which is not mentioned in your study at all. Aren’t you talking to your cardiologists? Why didn’t you address this issue when you did an annuloplasty? I am saying that because I learned this from your cardiologists, and since we introduced this concept in my operating room, we have not had recurrent TR anymore if you look at tethering before you do annuloplasty.

Dr Navia. That is a very good point. We have not taken a look completely at the tethering effect in all the cases, so we do not have a complete echocardiography result to give the data. Lately, we are starting to get more perspective to see this distance of the tethering and the unbalanced coaptation of the leaflets to be more proactive and try to use new remodeling shaped rings. But I cannot give in this presentation the real number of echocardiograms because we do not have it. Usually, the echo we have mainly is grade of TR. It is very difficult to quantify the leaflet moving and the leaflet coaptation.

Dr David. Are you asking the sonographer before the operation what segment has tethering before you do an annuloplasty?

Dr Navia. Yes, we started doing more and more right after the Yacoub paper, and our papers also showed that the deepness of the coaptation owing to the tethering of the tricuspid leaflet creates more recurrence of TR over time. That is a very important issue. Otherwise, we are looking right now at different concentric changes of the geometry of the RV, mainly the trabecular septomarginalis, that pulls the RV free wall, changes the anterior papillary muscle, and creates anterior leaflet tethering. We are now trying to find new techniques, because we know that the ring alone is insufficient to completely eliminate the TR secondary to left-sided valve. Without question, I cannot agree more with you; we are looking for that.

Dr Richard J. Shemin (Los Angeles, Calif). One of the powers of this study is the large number of patients. However, every surgeon has in his or her armamentarium a whole variety of techniques, and therefore there is bias. Very often you can do a propensity score to try to level the field to be sure that the decision-making of the individual surgeon who has all these techniques at his or her disposal is kind of eliminated. Did you think of doing that?

Dr Navia. That is a reasonable question. That could be the best way to analyze this study. However, unfortunately it is a long procedure of 18 years with different surgeon preferences, so we did only an observational cohort study. The next step would be to directly focus on propensity score analysis.

Dr Patrick M. McCarthy (Chicago, Ill). By way of disclosure, I am the inventor of the Edwards MC ring. Dr Navia, it appeared that about 15% of the patients had a permanent pacemaker before surgery. In the study that we had reported before, that was one of the major risk factors for failure. How do you manage that in this series? Is there a standardized approach now for how to deal with permanent pacing wires?

Dr Navia. Thank you, Dr McCarthy. We found the same situation—15% of the patients had a previous pacemaker. This is one of the high risk factors of recurrent TR, because the pacemaker leads can erode or perforate the leaflet and sometimes become stuck to the leaflet, impeding free leaflet movement and creating TR. We manage this by removing the wires through the tricuspid valve and then placing permanent epicardial leads on the RV. I think that is the best way to eliminate the potential problems of recurrence of TR or cause a negative effect on the tricuspid valve repair technique.

Dr David H. Adams (New York, NY). Dr Navia, can I ask you to speculate on the mechanism of failure with the flexible ring? In the mitral position, we know the intertrigonal distance dilates in heart failure and dilated cardiomyopathy, but I am not aware of any evidence that the septum of the tricuspid annulus dilates in cardiomyopathy. Do you have any idea, any mechanism why you are seeing recurrence of TR with flexible bands?

Dr Navia. I really do not know the intrinsic mechanism of failure of the flexible bands. Perhaps the flexible ring does not have enough strength to maintain the new annular size and does not prevent annular dilation as well as the rigid ring when the RV continues to remodel over time. That could be one of the reasons.
APPENDIX E1. Number of patients with echocardiograms available at and beyond various time points, and number of echocardiograms available for analysis. Black bars = patients; gray bars = echocardiograms.

APPENDIX E2: Method of Analysis for Other Outcomes

In-hospital morbidity and mortality are presumed unadjusted according to surgical management of TR as frequencies and percentages. Tricuspid valve reoperation, all-cause mortality, and requirement for a permanent pacemaker were assessed nonparametrically using the Kaplan–Meier method and parametrically by hazard function decomposition.\textsuperscript{E1} (For additional details, see http://www.clevelandclinic.org/heartcenter/hazard.)

RV systolic pressure was estimated using the method of Berger and colleagues.\textsuperscript{E2} A total of 3154 TTEs were available for 1512 patients (66% of the study group). Temporal trend was estimated by nonlinear mixed modeling and temporal decomposition as described under Methods in the main article.

Temporal trend of NYHA functional status was estimated from 1331 follow-up records available for 751 (33% of study group) patients. Because of low frequency, NYHA classes III and IV were collapsed into a single category for ordinal logistic regression analysis using nonlinear mixed modeling and temporal decomposition.

E-References


APPENDIX E3. In-hospital morbidity and mortality

<table>
<thead>
<tr>
<th>Morbidity</th>
<th>Flexible ring (n = 1052)</th>
<th>Rigid ring (n = 584)</th>
<th>Peri-Guard (n = 185)</th>
<th>De Vega (n = 129)</th>
<th>Kay (n = 248)</th>
<th>Annulus + leaflet (n = 35)</th>
<th>Kay + leaflet (n = 38)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intraop/postop IABP</td>
<td>49 (4.7)</td>
<td>25 (4.3)</td>
<td>7 (3.8)</td>
<td>8 (6.2)</td>
<td>2 (0.81)</td>
<td>3 (8.6)</td>
<td>1 (2.6)</td>
<td>.09</td>
</tr>
<tr>
<td>Return to OR for bleeding</td>
<td>69 (6.6)</td>
<td>54 (9.2)</td>
<td>19 (10)</td>
<td>14 (11)</td>
<td>11 (4.4)</td>
<td>2 (5.7)</td>
<td>3 (7.9)</td>
<td>.08</td>
</tr>
<tr>
<td>Stroke</td>
<td>30 (2.9)</td>
<td>12 (2.1)</td>
<td>1 (0.54)</td>
<td>4 (3.1)</td>
<td>8 (3.2)</td>
<td>1 (2.9)</td>
<td>1 (2.6)</td>
<td>.6</td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>6 (0.57)</td>
<td>5 (0.86)</td>
<td>1 (0.54)</td>
<td>1 (0.78)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>.8</td>
</tr>
<tr>
<td>Renal failure</td>
<td>54 (5.1)</td>
<td>31 (5.3)</td>
<td>9 (4.9)</td>
<td>13 (10)</td>
<td>18 (7.3)</td>
<td>4 (11)</td>
<td>2 (5.3)</td>
<td>.2</td>
</tr>
<tr>
<td>Respiratory insufficiency</td>
<td>143 (14)</td>
<td>95 (16)</td>
<td>39 (21)</td>
<td>31 (24)</td>
<td>33 (13)</td>
<td>9 (26)</td>
<td>6 (16)</td>
<td>.006</td>
</tr>
<tr>
<td>Septicemia/sepsis</td>
<td>61 (5.8)</td>
<td>39 (6.7)</td>
<td>7 (3.8)</td>
<td>20 (16)</td>
<td>17 (6.9)</td>
<td>4 (11)</td>
<td>1 (2.6)</td>
<td>.001</td>
</tr>
<tr>
<td>In-hospital death</td>
<td>52 (4.9)</td>
<td>34 (5.8)</td>
<td>16 (8.6)</td>
<td>18 (14)</td>
<td>12 (4.8)</td>
<td>3 (8.6)</td>
<td>12 (5.3)</td>
<td>.003</td>
</tr>
</tbody>
</table>

Intraop/postop, intraoperative or postoperative, IABP, intraoperative aortic balloon pumping; OR, operating room.

APPENDIX E4. Freedom from reoperation related to tricuspid valve. Each symbol represents a reoperation, vertical bars are 68% confidence limits (equivalent to ±1 standard error), and solid line enclosed within dashed 68% confidence limits is parametric estimate.

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APPENDIX E5. Overall survival after tricuspid valve repair. Format is as in Appendix E4.

APPENDIX E6. Freedom from permanent pacemakers (PPM) after tricuspid valve repair according to procedure performed. Format is as in Figure 4.
APPENDIX E7. Temporal trend of postoperative right ventricular systolic pressure (RVSP) according to procedure group. Format is as in Figure 2.
APPENDIX E8. Temporal trend of postoperative New York Heart Association (NYHA) functional class according to procedure performed. Format is as in Figure 2. A, NYHA class I. B, NYHA class III/IV.