Comparison of Results of Tricuspid Valve Repair Versus Replacement for Severe Functional Tricuspid Regurgitation

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The optimal decision regarding whether to repair or replace the tricuspid valve (TV) remains controversial in patients with very severe functional tricuspid regurgitation (TR). We sought to compare clinical outcomes of TV repair versus replacement for very severe TR associated with severe TV tethering. We included 96 consecutive patients (20 men, 58 ± 11 years of age) who had both severe tethering of TV and very severe functional TR and consequently underwent TV surgery during left-sided valve surgery. TV repair was performed on 79 patients (repair group), whereas 17 patients underwent TV replacement (replacement group). The primary end point of the study was defined as the composite of operative mortality, cardiac death, repeat TV surgery, and hospitalization due to congestive heart failure during follow-up. The 2 groups had similar baseline clinical, echocardiographic, and operative characteristics, but operative mortality was significantly higher in the replacement group than in the repair group ($p = 0.008$). During a median follow-up of 87 months, 19 patients (24%) in the repair group and 8 (47%) in the replacement group attained the composite end point, and TV replacement was independently associated with end points in the Cox proportional hazards analysis after adjustment with propensity score (hazard ratio 4.033, 95% CI 1.470 to 11.071; $p = 0.007$). In conclusion, compared with TV repair, replacement was associated with higher operative mortality and worse long-term clinical outcomes in patients with very severe functional TR. Repair should be the preferred surgical option even for severe TR associated with more advanced tethering and right ventricular dilatation. **(Am J Cardiol 2017;119:905−910)**

Functional tricuspid regurgitation (TR) is the most common cause of TR and negatively impacts functional class and survival.[1−3] Current guidelines recommend tricuspid valve (TV) surgery for patients with severe TR undergoing left-sided valve surgery because TR does not reliably resolve after treatment of left-sided valve lesions.[4,5] Generally, TV repair is preferable to replacement because it can be accomplished without significantly increasing ischemic time and surgical risks,[4,5] and functional TR can usually be effectively managed with tricuspid annuloplasty.[1,6,7] However, tricuspid annuloplasty alone does not address leaflet malcoaptation resulting from severe leaflet tethering due to progressive RV dilation and dysfunction,[8] and severe TV tethering is associated with residual or recurrent functional TR and poor clinical outcomes in patients undergoing tricuspid annuloplasty.[9−11] Thus, TV replacement has been considered for severe TR associated with more advanced tethering and RV dilatation,[9] but it may be associated with higher perioperative mortality.[12] Because no studies have compared TV repair and replacement in patients with very severe functional TR, we sought to compare the clinical outcomes of TV repair versus replacement using our prospectively collected registry data of patients with very severe TR.

Methods

The study population was a part of the Asan Valve Registry[13,14] and included consecutive patients who had both severe tethering of TV and very severe functional TR and consequently underwent TV surgery during left-sided valve surgery between 2000 and 2012. Case report forms, including patient demographics, clinical presentation, and echocardiographic data, were stored in an electronic database.[13,14] Clinical and echocardiographic follow-up data of study patients were collected annually and entered into the database. All patients satisfied the following criteria for severe tethering of TV and very severe functional TR: (1) total failure of coaptation of normal TV leaflets due to severe tethering by RV dilatation; (2) tethering area $>$ 1.6 cm^2; (3) jet area >10 cm^2; (4) vena contracta width >1.0 cm; and (5) systolic flow reversal in the hepatic vein (Figure 1). Patients with organic TV disease, including rheumatic involvement of leaflets, prolapse of leaflets, ruptured chordae, or Ebstein anomaly; and those who had previous TV operations were excluded. The decision to

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See page 909 for disclosure information.

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0002-9149/17/$ - see front matter
http://dx.doi.org/10.1016/j.amjcard.2016.11.071

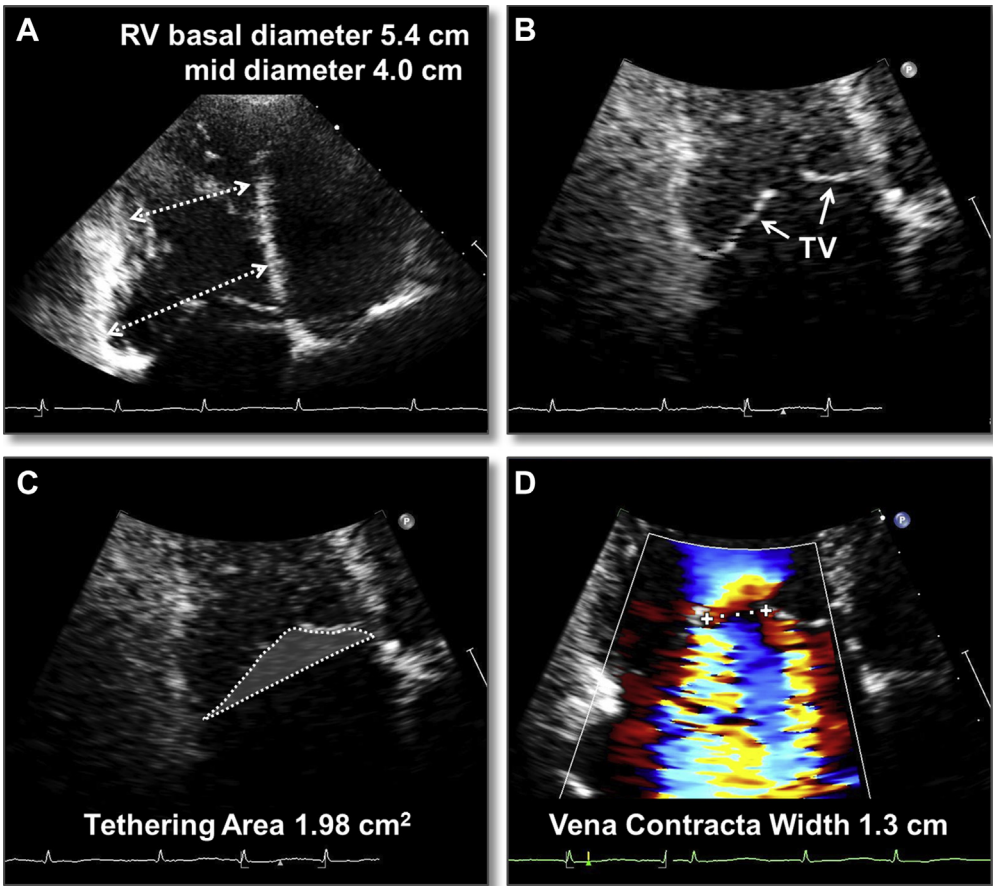

Figure 1. Very severe functional TR on echocardiography. Total failure of coaptation of TV leaflets due to severe tethering by RV dilatation was observed *(A* and *B)*. A tethering area *(C)* was obtained by tracing the area between the leaflets and the tricuspid annulus, and a vena contracta width *(D)* was measured on color flow imaging.

perform TV repair or replacement was at the surgeon's discretion. Informed consent was obtained from each patient, and the study protocol was approved by the institutional review board at our institution. Echocardiographic evaluation was performed before surgery, before discharge of patients after surgery and annually during follow-up. RV size and TR were evaluated in the adjusted 4-chamber view to focus particularly on the RV.[15] The tricuspid annular dimension was measured at the time of maximal TV opening from the insertion of septal leaflet to the insertion of anterior leaflet.[16] The tethering area was obtained by measuring the area between the atrial surface of the leaflets and the tricuspid annular plane at end-systole.[9]

Procedures were performed with the use of standard cardiopulmonary bypass. After left-sided valve surgery, TV repair, or replacement was performed according to the attending surgeon's preference. In the repair group (n = 79), TV repairs were performed using ring annuloplasty (n = 61) or the De Vega annuloplasty (n = 18). In the replacement group (n = 17), TV replacements were performed with mechanical (n = 12) or bioprosthetic valves (n = 5). Intraoperative transesophageal echocardiography was routinely performed during surgical procedures.[14]

All the study patients regularly visited their attending physicians at 3-month intervals. Data were collected until December 2015. Operative mortality was defined as any death that occurred during the initial hospitalization or within 30 days of the surgery. Follow-up information was complete for 95 patients (99%), and for the patient lost to follow-up, dates and causes of death were obtained from the Korean National Registry of Vital Statistics. The primary end point of the study was defined as the composite of operative mortality, cardiac death, repeat TV surgery, and hospitalization due to congestive heart failure (CHF) during follow-up, and the secondary end point was death from any cause.[14]

Categorical variables are presented as numbers and percentages and were compared using the Fisher's exact test. Continuous variables are expressed as mean ± SD and were compared using the Student *t* test or Mann-Whitney *U* test as appropriate. Event-free survival curves were constructed with Kaplan-Meier estimates and compared using the log-rank test. Unadjusted Cox proportional hazards models were used to examine the association of baseline characteristics with all-cause mortality and primary end points (Supplementary Tables 1 and 2). Covariates considered for inclusion in the models included all variables presented in baseline clinical, echocardiographic, and operative findings. To ascertain whether tethering area or RV end-systolic dimension is independently associated with all-cause mortality, hazard ratio (HR) and 95% CI were calculated with the Cox proportional hazards model adjusted for age and Society of Thoracic

Table 1
Baseline characteristics of patients who underwent tricuspid valve repair and those who underwent tricuspid valve replacement

Variable	Tricuspid valve		P-value
	Repair (n=79)	Replacement (n=17)	
Age (years)	58 ± 11	58 ± 11	0.10
Male	16 (20%)	4 (24%)	0.75
Body surface area (m^2)	1.55 ± 0.17	1.61 ± 0.12	0.18
NYHA class III or IV	25 (32%)	2 (12%)	0.14
Diabetes mellitus	5 (6%)	1 (6%)	1.00
Hypertension	8 (10%)	1 (6%)	1.00
Atrial fibrillation	63 (80%)	14 (82%)	1.00
Renal failure	5 (6%)	0 (0%)	0.58
Hemoglobin (g/dL)	11.8 ± 2.1	11.5 ± 1.7	0.60
Creatinine (mg/dL)	1.1 ± 0.9	0.9 ± 0.2	0.29
Albumin (g/dL)	3.7 ± 0.5	3.7 ± 0.5	0.57
Society of Thoracic Surgeons score (%)	3.1 ± 3.1	2.3 ± 1.4	0.31
LV ejection fraction (%)	54 ± 10	59 ± 7	0.10
RV basal end-diastolic dimension (mm)	51 ± 8	53 ± 7	0.21
RV basal end-systolic dimension (mm)	41 ± 7	43 ± 6	0.27
RV mid end-diastolic dimension (mm)	44 ± 8	47 ± 6	0.11
RV mid end-systolic dimension (mm)	35 ± 7	37 ± 6	0.30
RV end-diastolic area (cm^2)	26 ± 8	29 ± 6	0.14
RV end-systolic area (cm^2)	17 ± 6	18 ± 4	0.42
RV fractional area change (%)	34 ± 10	36 ± 8	0.40
Tricuspid annulus diameter (mm)	44 ± 6	48 ± 8	0.044
Tethering area (cm^2)	2.5 ± 0.8	2.6 ± 0.8	0.61
Vena contracta width (mm)	14 ± 3	16 ± 3	0.09
Peak tricuspid regurgitation velocity (m/sec)	3.2 ± 0.7	2.9 ± 0.6	0.21

LV = left ventricle; NYHA = New York Heart Association; RV = right ventricle.

Surgeons (STS) score, which is an important predictor of outcomes.[17] In addition, a propensity score−adjusted analysis was carried out to reduce the effect of treatment selection bias and potential confounding.[18] The propensity scores were estimated without regard to outcome variables, using multiple logistic regression analysis. Using a full nonparsimonious model which included 26 clinically relevant covariates listed in Tables 1 and 2, the propensity score for type of TV surgery was calculated. The individual propensity score was incorporated into the Cox proportional hazards model as a covariate to calculate the propensity score-adjusted HR of TV replacement. We also adjusted for differences in baseline characteristics using weighted Cox proportional hazards regression models with inverse probability-of-treatment weighting method,[19] with weights for patients receiving TV replacement being the inverse of (1−propensity score) and weights for patients receiving TV repair being the inverse of propensity score. All reported p values were 2 sided, and a value of $p < 0.05$ was considered statistically significant. SAS software, version 9.1 (SAS Institute, Inc., Cary, North Carolina), was used for statistical analyses.

Results

Baseline clinical and echocardiographic characteristics of the TV repair and replacement groups were compared in Table 1. The both groups had similar baseline characteristics, but the tricuspid annular dimension was significantly larger in the TV replacement group. Table 2 shows operative characteristics and a total of 17 patients (18%) had a previous history of left-sided valve surgery. The 2 groups were similar in terms of most aspects, but the TV replacement group tended to have longer cardiopulmonary bypass time and higher frequency of redo valve surgery. Although severe residual TR was observed in 2 patients on intraoperative transesophageal echocardiography after TV repair, additional procedures were not performed based on the attending surgeon's decision.

Table 2
Comparison of operative characteristics between patients who underwent tricuspid valve repair and those who had tricuspid valve replacement

Variable	Tricuspid valve		P-value
	Repair (n = 79)	Replacement (n = 17)	
Redo cardiac surgery	11 (14%)	6 (35%)	0.07
Cardiopulmonary bypass time (min)	172 ± 75	204 ± 63	0.10
Cross-clamp time (min)	107 ± 51	119 ± 43	0.40
Concomitant procedure			
Mitral valve repair	14 (18%)	4 (24%)	0.73
Mitral valve replacement	50 (63%)	9 (53%)	0.38
Aortic valve replacement	4 (5%)	3 (18%)	0.10
Dual valve replacement	11 (14%)	1 (6%)	0.38
Coronary artery bypass graft	5 (6%)	0 (0%)	0.58
Maze procedure	32 (41%)	6 (35%)	0.79
Residual severe tricuspid regurgitation*	2 (3%)	0 (0%)	1.00

* Observed on intraoperative transesophageal echocardiography performed immediately after valve surgery.

There were 4 deaths (5%) in the TV repair group and 5 deaths (29%) in the TV replacement group within 30 days of the surgery or before hospital discharge ($p = 0.008$). The causes of operative mortality in the TV repair group were postoperative bleeding in 2 patients, sepsis in 1, sudden death in 1, whereas the causes of operative mortality in the TV replacement group were acute RV failure (n = 3), postoperative bleeding (n = 1) and pneumonia (n = 1). The median follow-up was 8.0 years (interquartile range [IQR] 3.1 to 10.5 years) in the TV repair group and 5.4 years (IQR 3.3 to 12.5 years) in the TV replacement group ($p = 0.685$). During follow-up, there were 10 cardiac and 8 noncardiac deaths in the TV repair group, and 1 cardiac death in the TV replacement group. The estimated actuarial 10-year survival rates were 71 ± 6% in the TV repair group and 65 ± 12% in the TV replacement group ($p = 0.298$; Figure 2). The causes of noncardiac deaths were pneumonia (n = 3), stroke (n = 3), and gastrointestinal bleeding (n = 1) in the TV repair group. The causes of cardiac deaths in the TV repair group were CHF in 7 patients, sudden cardiac death in 2, and acute myocardial infarction in 1, whereas one patient in the TV replacement group died of complications related to cardiac transplantation performed after TV surgery. In the TV repair group, 3 patients underwent repeat TV surgery and 2 patients with recurrence of severe TR required hospitalization for CHF without cardiac mortality or redo TV surgery. In the TV replacement group, 2 patients were urgently hospitalized for CHF; one patient

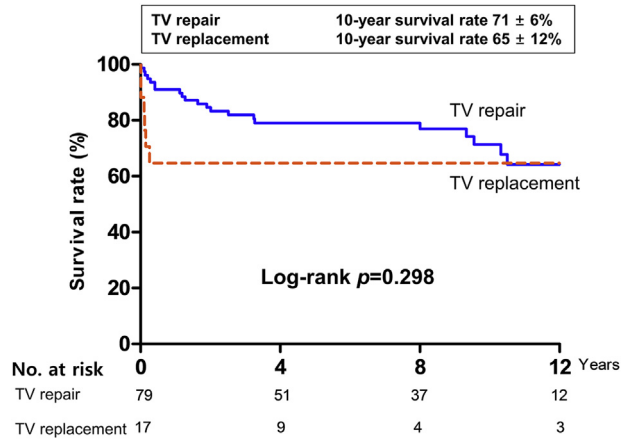

Figure 2. Kaplan-Meier estimates of survival according to treatment groups.

received thrombolytic therapy for thrombotic occlusion of mechanical prosthetic TV, and the other underwent redo aortic valve surgery due to severe obstruction of prosthetic aortic valve by pannus formation. Thus, 19 patients (24%) in the TV repair group and 8 (47%) in the TV replacement group attained the composite end point (Table 3), and the estimated actuarial 10-year event-free survival rate was significantly higher in the TV repair group (75 ± 6%) than in the TV replacement group (43 ± 2%; p = 0.019; Figure 3).

Using unadjusted Cox proportional hazards analysis, we found that old age, male gender, diabetes, renal failure, anemia, higher serum creatinine and lower albumin levels, higher STS score, RV diastolic and systolic dimensions, and tethering area were associated with overall mortality, but redo surgery and surgical times were not associated with mortality. Tethering area (HR 1.647, 95% CI 1.133 to 2.395; p = 0.009) and RV basal end-systolic dimension (HR 1.085, 95% CI 1.029 to 1.143; p = 0.002) were independently associated with mortality in the Cox proportional hazards analysis adjusted for age and STS score (Supplementary Table 1). Baseline correlates of composite end point were age, diabetes, renal failure, serum creatinine level, higher STS score, RV systolic dimensions, tricuspid annulus diameter, and TV replacement on unadjusted Cox proportional hazards analysis (Supplementary Table 2). TV replacement was independently associated with end points in propensity score−adjusted Cox proportional hazards analyses (HR 4.033, 95% CI 1.470 to 11.071; p = 0.007). The risk of end point was also significantly greater in the TV replacement group after adjustment with inverse probability-of-treatment weighting method (Table 4).

Among 75 patients who survived surgery in the repair group, persistent severe TR was observed in 2 patients (3%) on immediate postoperative echocardiography. During a median echocardiographic follow-up of 5.0 years (IQR 2.3 to 8.8 years), progression to severe TR occurred in 15 patients (20%). In the repair group, actuarially estimated survival rates were significantly higher in 58 patients without residual or recurrent severe TR than those in 17 patients who had such TR (78 ± 7% vs 63 ± 12% at 10 years, p = 0.024). The estimated actuarial 10-year

Table 3
Primary end points

Outcome	Repair (n=79)	Replacement (n=17)
Primary end point	19 (24.1%)	8 (47.1%)
Operative mortality	4 (5.1%)	5 (29.4%)
Late cardiac mortality	10 (12.6%)	1 (5.9%)
Tricuspid valve repeat surgery	3 (3.8%)	0 (0%)
Heart failure hospitalization	2 (2.6%)	2 (11.8%)

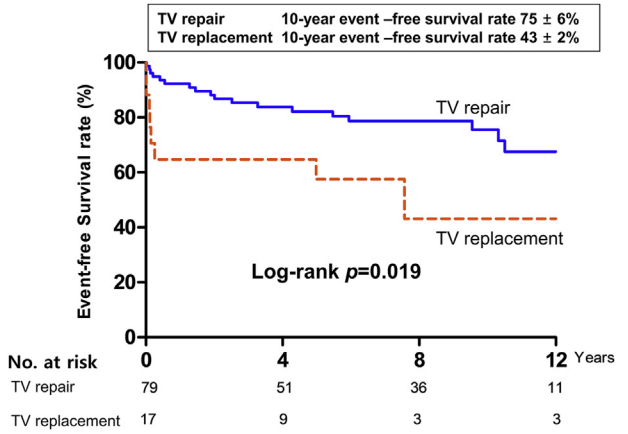

Figure 3. Kaplan-Meier estimates of event-free survival according to treatment groups.

event-free survival rate was also significantly higher in patients without residual or recurrent severe TR (88 ± 5%) than that in those with severe TR (51 ± 12%) during follow-up (p <0.001).

Discussion

This study demonstrates that in patients with very severe functional TR associated with severe tethering of TV, TV replacement combined with left-sided valve surgery is associated with a higher operative mortality and poorer late clinical outcomes than TV repair. Practice guidelines recommend TV repair for patients with moderate TR undergoing left-sided valve disease.[4,5] However, the guidelines do not specify whether to repair or replace the TV for severe TR at the time of left-sided valve surgery[4,5] because there is no evidence showing which of TV surgical interventions is superior.[20,21] As a general principle, valve repair is preferable to replacement for patients with significant TR undergoing left-sided valve disease[4,5] because tricuspid annuloplasty adds little time and complexity to valve surgery.[22] However, tricuspid annuloplasty has been associated with a high rate of recurrent TR and adverse events in patients with tethering of TV in addition to enlarged TV annulus.[9,11] Previous clinical studies have shown discrepant results regarding whether surgical outcome of severe TR is affected by type of surgery.[12,20,21] Similar to the results of the present study, operative mortality (22%) of TV replacement in the study by Singh et al[12] was significantly higher than that (4%) of repair, and they speculated that TV replacement is associated with low output syndrome and RV dysfunction

Table 4
Hazard ratios for clinical outcomes of tricuspid valve replacement compared with tricuspid valve repair

Model	All-cause Death		Primary End-point	
	HR (95% CI)	P-value	HR (95% CI)	P-value
Crude	1.608 (0.651-3.972)	0.304	2.595 (1.131-5.952)	0.024
Multivariable adjusted*	2.244 (0.872-5.774)	0.094	3.600 (1.486-8.722)	0.005
Propensity score adjusted	2.499 (0.878-7.118)	0.086	4.033 (1.470-11.071)	0.007
IPTW method	1.916 (0.590-6.230)	0.280	2.917 (1.049-8.115)	0.040

CI = confidence interval; HR = hazard ratio; IPTW = inverse probability-of-treatment weighting method.
* Adjusted for age and STS score.

secondary to a rigid TV prosthesis. In contrast to our study, Moraca et al[23] reported that operative mortality was similar for TV replacement and repair in their propensity-matched cohort. Differences in operative mortalities of TV repair, causes, and severity of TR may explain these conflicting results. Patients with secondary functional TR or isolated organic TR were analyzed together,[20,23] and the operative mortality (18%) of TV repair in the study by Moraca et al was much higher than that (5%) of the present study. The present study shows that the operative risk of TV replacement is greater than potential benefits of preventing TR recurrences even in patients with severe tethering of TV and high risk of recurrence of TR. Thus, TV repair should be considered a preferred treatment option for patients with left-sided valve disease and severe functional TR.

In the present study of very severe TR secondary to left-sided valve disease, we noted a high operative mortality of 9% and a poor event-free survival. In contrast, operative mortality rates of left-sided valve disease are as low as 1% to 2% in many cardiac centers.[24] This poor surgical outcome may be related to late performance of surgery after RV dysfunction might already be irreversible.[22] Because the timing of surgical intervention for severe functional TR is determined according to the surgical indications for left-sided valve disease without considering RV systolic dysfunction,[4,5] surgical correction of severe TR is often performed after development of severe tethering and marked dilation of RV, which were independent predictors of mortality in the present study. We found that both tethering area and RV end-systolic dimension were reliable markers for alteration of RV geometry and systolic dysfunction, which were closely related to recurrence of TR,[10] operative mortality and late outcomes.[11] Because evaluation of RV systolic function is difficult due to its complex geometry and because ejection fraction or fractional area change overestimates RV contractility in the presence of TR,[4] serial assessments of RV dimensions and volumes would be more useful measures to determine optimal timing of TV surgery. Recently, Yiu et al[11] also demonstrated that RV dimensions and tethering area were important preoperative measures associated with adverse events. Although current guidelines consider TV surgery only for severe primary TR in patients with deterioration of RV dilatation and/or systolic dysfunction,[4,5] TV surgery should be also considered for severe functional TR when tethering area or RV end-systolic dimension significantly increases during serial imaging follow-up.

In the present study, outcomes of TV repair were also suboptimal with a high rate of residual and recurrent severe TR, which was associated with adverse events and mortality. Earlier tricuspid annuloplasty performed before alteration of the RV geometry and excessive TV tethering occurs, as well as refinement of repair technique may further improve clinical outcomes.[8,11,21] Ring annuloplasty is generally the preferred surgical approach for significant functional TR, and in addition, if significant leaflet tethering is present, augmentation of the anterior tricuspid leaflet may be necessary.[8,21]

Only patients with very severe functional TR confirmed by strict criteria were consecutively enrolled in our prospectively designed registry to minimize biases and heterogeneity in study patients. Nonetheless, comparison of surgical options was subject to the limitations inherent to nonrandomized assignment and such limitations may have significantly affected our results due to selection bias and unmeasured confounders. To control for the inherent biases related to treatment selection and heterogeneity in baseline factors, we performed propensity score-adjusted and inverse probability-of-treatment weighting analyses, which consistently showed that TV repair was superior to replacement. It is currently accepted that TV annuloplasty with a ring is more durable than suture repair,[7] but 23% of patients in the repair group underwent suture annuloplasty, which might have affected a rate of recurrence of TR. As surgery was mainly indicated for severe left-sided valve disease in the present study, only 2-dimensional echocardiographic parameters were used for assessing RV geometry, and tricuspid annular plane systolic excursion, RV strain, right atrial pressure, or the presence of sleep apnea was not evaluated. Further study using cardiac magnetic resonance imaging[21] or 3-dimensional echocardiographic parameters with a precise geometric analysis[25] will be needed to optimize surgical timing.

Disclosures

The authors have no conflicts of interest to disclose.

Supplementary Data

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.amjcard.2016.11.071.

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