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Research paper

Longitudinal change in postoperative right ventricular systolic function in patients undergoing surgery for isolated tricuspid regurgitation

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ABSTRACT

Right ventricular (RV) dysfunction is an indication for tricuspid valve (TV) surgery in patients with severe isolated tricuspid regurgitation (TR). Postoperative RV dysfunction is associated with poor outcome; however, the longitudinal changes in RV function before and after surgery have not been established. We retrospectively analyzed 24 patients who underwent TV surgery for isolated severe TR. For assessing RV systolic function, we measured the RV fractional area change (RVFAC) at baseline, and 1 (immediate) and 4–20 (late) months after surgery. We divided patients into 2 groups according to the RVFAC late after surgery (<35%, post-op. reduced; and ≥35%, post-op. preserved). The mean RVFAC was significantly decreased immediately after surgery compared to baseline (41.5 ± 10.1% vs. 32.2 ± 9.6%; $p < 0.001$). The RVFAC reduction was still observed late after surgery (35.5 ± 7.4%; $p = 0.002$). Of 24 patients, 12 patients (50%) had preserved RV systolic function late after surgery. Although there was no significant difference in the preoperative RVFAC between the 2 groups, the preoperative RV end-systolic area (RVESA) /body surface area (BSA) was significantly less in the post-op. preserved RV systolic function group (13.8 ± 4.3 cm^2/m^2 vs. 8.6 ± 2.6 cm^2/m^2; $p = 0.001$). The optimal cut-off value for the preoperative RVESA/BSA in detecting postoperative preserved RV systolic function was 10.8 cm^2/m^2 (AUC, 0.85; sensitivity, 91.7%; and specificity, 75.0%). In patients undergoing surgery for isolated severe TR, the RVFAC was significantly decreased immediately after surgery and the reduction continued late after surgery. The preoperative RVESA/BSA might be helpful to predict preserved RV function after surgery.

1. Introduction

Tricuspid regurgitation (TR) is a common echocardiographic finding [1]. In the United States, greater than 1.6 million patients have moderate or severe TR [2,3]. TR is caused by valvular abnormalities, such as a flail leaflet (primary TR) or more commonly tricuspid annular dilatation secondary to atrial fibrillation or left ventricular (LV) disease (secondary TR) [4–7]. Although mild TR is usually benign [8], a number of studies have shown that severe TR, which causes right ventricular (RV) dysfunction, is associated with a worse prognosis than previously thought [2,9–13]. In particular, the outcome after tricuspid valve (TV) surgery is poor in patients with preoperative severe RV dysfunction by TR [14–16]. Therefore, recent guidelines recommend TV surgery in patients with severe TR compared to previous guidelines [17–19]. In addition, right-sided heart failure symptoms, progressive RV dilatation, and RV dysfunction are indications for TV surgery according to the recent guidelines [17–19]; however, no clear cut-off value of RV dilatation or function has been established because of limited data regarding the recovery of RV function after surgery. It is therefore essential to perform TV surgery at the time when preserved RV function can be expected postoperatively because postoperative RV dysfunction is associated with a poor prognosis in patients undergoing TV surgery

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[15]. In this study we aimed to elucidate the longitudinal changes in RV systolic function in patients undergoing TV surgery for isolated severe TR. We compared the echocardiographic findings and clinical factors at baseline between patients with reduced and preserved RV systolic function 4–20 months postoperatively.

2. Methods

This study was designed as a retrospective study. We enrolled 27 patients with severe TR who underwent a tricuspid valvuloplasty or TV replacement, and who did not undergo other valve surgery or coronary artery bypass grafting concomitantly at the University of Tokyo Hospital, Sakakibara Heart Institute, Juntendo University Hospital, and Tenri Hospital between July 2008 and September 2016. We excluded patients for the following reasons: other valvular disease (moderate or severe); complicated congenital heart disease, such as congenitally-corrected transposition of the great arteries; cardiac tumors; primary pulmonary hypertension; and insufficient patient data. This observational study was approved by the Institutional Ethics Committee of each institution.

We obtained preoperative clinical information from medical records. Clinical information, including age, height, weight, systolic blood pressure, diastolic blood pressure, heart rate, and New York Heart Association (NYHA) functional class, was obtained. The body mass index was calculated as the body weight in kg divided by height in meters squared. A medical history of hypertension, diabetes mellitus, dyslipidemia, and atrial fibrillation was also obtained from the medical records. In addition, surgical technique, and TV replacement or repair were also obtained from the medical records. Laboratory measurements, including glycosylated hemoglobin A1c, total cholesterol, and creatinine, were performed using standard assays and the estimated glomerular filtration rate was calculated using age, gender, and the serum creatinine concentration.

Two-dimensional echocardiography was performed using commercially available equipment before surgery (within 3 months), immediately after surgery (within 1 month), and late after surgery (4–20 months). Echocardiographic equipment was maintained according to the guidelines of the Japanese Society of Echocardiography [20]. LV and left atrial (LA) chamber assessments were performed according to the guidelines of the American Society of Echocardiography [21]. The severity of TR and other valvular regurgitation was evaluated according to guidelines [22]. The LV ejection fraction was calculated using the Teichholz formula. The maximum LA volume was measured from the apical 4-chamber view and was indexed by body surface area (BSA) to calculate the LA volume index. The RV end-diastolic area (RVEDA), end-systolic area (RVESA), tricuspid annular diameter, and right atrium (RA) area were obtained from the RV-focused apical 4-chamber view, as recommended by the American Society of Echocardiography [21,23]. To assess RV systolic function, the RV fractional area change (RVFAC) was calculated as follows: 100 * (RVEDA – RVESA) / RVEDA. We defined RV systolic dysfunction as a RVFAC <35%, according to the guideline [21]. To elucidate the differences between reduced and preserved RV function late after surgery, we divided patients into 2 groups according to the RVFAC: RVFAC <35% (post-op. reduced RV systolic function); and RVFAC ≥35% (post-op. preserved RV systolic function).

All data, except for days from surgery-to-echocardiography are expressed as the mean ± standard deviation (SD) or the number (%) of patients. Days from surgery-to-echocardiography are expressed as the median with the interquartile range (IQR). We determined the changes in the mean RVFAC, RVEDA, and RVESA by repeated measures single-factor analysis of variance (ANOVA). A paired *t*-test was used to compare the measurements at baseline and after surgery. Continuous variables were grouped by preserved or reduced RV function late after surgery and compared by t-test for preoperative clinical characteristics and echocardiographic parameters. Binary data were compared using chi-squared analysis or Fisher's exact test. Pearson's correlation test was used to determine the correlation between preoperative factors and RVFAC late after surgery. Receiver operating characteristic (ROC) curve analysis was performed to determine the accuracy of the preoperative RVFAC, RVEDA/BSA, and RVESA/BSA in detecting preserved RV systolic function (RVFAC ≥35%) late after surgery and to determine the sensitivities and specificities. A p-value <0.05 was considered statistically significant. All analyses were conducted utilizing SPSS (version 27; SPSS, Inc., Chicago, IL, USA).

3. Results

We enrolled 27 patients who had isolated TV surgery for severe TR (3 patients at the University of Tokyo Hospital, 19 patients at the Sakakibara Heart Institute, 3 patients at Juntendo University Hospital, and 2 patients at Tenri Hospital). We excluded 3 patients who had pathologic changes that may affect postoperative RV function (severe tricuspid stenosis [TS], 1; moderate TR + TS, 1; and poorly-controlled hyperthyroidism, 1). Thus, 24 patients were included in this analysis. None of the patients developed residual severe TR or had heart failure or cardiac death postoperatively during the observation period. There were 17 patients who underwent true isolated TV surgery. Seven patients underwent concomitant heart surgery (left atrial appendage closure, 3; Maze surgery, 3; total arch replacement, 1; and pacemaker implant, 1). The causes of TR were trauma (n = 5), Ebstein anomaly (n = 6), infective endocarditis (n = 2), and TR secondary to annular dilatation (n = 11). Although 2 patients had moderate residual TR postoperatively, the severity of TR was improved to mild or less in the remaining patients. The characteristics of the enrolled patients are summarized in Table 1. Days from surgery-to-echocardiography were 6 (IQR, 5–8) immediately after surgery and 354 (IQR, 226–378) late after surgery.

Table 1
Comparison of preoperative clinical characteristics between the groups.

	Total (n = 24)	Reduced RV systolic function group (n = 12)	Preserved RV systolic function group (n = 12)	p
Age (year)	53 ± 19	53 ± 19	53 ± 20	0.99
Male, n (%)	12 (50%)	6(50%)	6 (50%)	1.00
Body mass index (kg/m^2)	22.6 ± 3.1	23.8 ± 3.0	21.4 ± 2.7	0.05
sBP (mmHg)	122 ± 23	127 ± 26	121 ± 22	0.62
dBP (mmHg)	71 ± 18	74 ± 24	68 ± 8	0.05
Heart rate (bpm)	71 ± 18	74 ± 19	68 ± 17	0.45
NYHA ≥3	5 (21%)	4 (33%)	1 (8%)	0.11
TVR, n (%)	4 (17%)	2(17%)	2 (17%)	1.00
TR cause (primary)	13 (54%)	5 (42%)	8 (67%)	0.22
TR cause (secondary)	11 (46%)	7 (58%)	4 (33%)	
Smoking, n (%)	11 (46%)	6 (50%)	5 (42%)	0.68
Hypertension, n (%)	8 (33%)	6 (50%)	2 (17%)	0.08
Diabetes mellitus, n (%)	1 (4%)	1 (8%)	0 (0%)	0.31
Hyperlipidemia, n (%)	3 (13%)	3 (25%)	0 (0%)	0.06
Atrial fibrillation, n (%)	9 (38%)	6 (50%)	3 (25%)	0.21
Hemoglobin A1c (%)	5.7 ± 0.7	5.8 ± 0.8	5.7 ± 0.4	0.76
Total cholesterol (mg/dl)	174 ± 49	165 ± 51	184 ± 48	0.36
eGFR (ml/min/ 1.73m^2)	79 ± 28	72 ± 23	87 ± 30	0.17

Abbreviations: sBP, systolic blood pressure; dBP, diastolic blood pressure; NYHA, *New York Heart Association* functional classification; TVR, tricuspid valve replacement; TR, tricuspid regurgitation; eGFR, estimated glomerular filtration rate.
Data are shown as the mean ± standard deviation or n (%).

Fig. 1A shows the change in mean RVFAC and Fig. 1B shows the change in RVFAC in each case before and after surgery. There was a significant change in the mean RVFAC after the surgery based on ANOVA ($p < 0.001$). The RVFAC significantly decreased immediately after surgery compared with baseline ($41.5 \pm 10.1\%$ vs. $32.2 \pm 9.6\%$; $p < 0.001$) and slightly improved thereafter; however, a significant reduction in the RVFAC persisted late after surgery compared with baseline ($41.5 \pm 10.1\%$ vs. $35.5 \pm 7.4\%$; $p = 0.002$). As shown in Fig. 1B, there was diversity in the postoperative RVFAC change. Specifically, some patients had excellent recovery of the RVFAC, whereas some patients had a deterioration in the RVFAC. Fig. 2 shows that both the RVEDA/BSA and RVESA/BSA decreased postoperatively ($p < 0.001$ and $p = 0.03$ [ANOVA], respectively). The RVEDA/BSA significantly decreased immediately after surgery (19.2 ± 6.6 cm^2/m^2 vs. 15.1 ± 7.9 cm^2/m^2; $p < 0.001$) and continuously decreased even late after surgery (19.2 ± 6.6 cm^2/m^2 vs. 12.1 ± 2.9 cm^2/m^2; $p < 0.001$). In contrast, a reduction in the RVESA/BSA was not observed immediately after surgery (11.2 ± 4.4 cm^2/m^2 vs. 10.6 ± 7.3 cm^2/m^2; $p = 0.276$) but was observed late after surgery (11.2 ± 4.4 cm^2/m^2 vs. 7.9 ± 2.3 cm^2/m^2; $p < 0.001$). In addition to a decrease in the RVESA/BSA that occurred later, the magnitude of the decrease was also less than the decrease in the RVEDA/BSA.

Of 24 patients, 12 (50.0%) had reduced RV systolic function and 12 (50.0%) had preserved RV systolic function late after surgery. Table 1 shows a comparison of the preoperative clinical characteristics between the 2 groups. There were no significant differences between the groups, including type of TV surgery or cause of TR at baseline, while body mass index and diastolic blood pressure tended to be higher in the post-op. reduced RV systolic function group. A comparison of preoperative echocardiographic parameters between the 2 groups is summarized in Table 2. The preoperative RVFAC in the post-op. preserved RV systolic function group tended to be higher ($p = 0.06$), although there was not a statistically significant difference. Among the other preoperative echocardiographic parameters, RV area, RA area, and tricuspid annular diameter were significantly less in the post-op. preserved RV systolic function group, and the difference in the RVESA/BSA between the 2 groups was particularly notable. The area under the ROC curve (AUC) obtained using the preoperative RVFAC, RVEDA/BSA, and RVESA/BSA cut-off values for detecting preserved FAC (≥35%) late after surgery is shown in Fig. 3. The optimal cut-off values for the preoperative RVFAC, RVEDA/BSA, and RVESA/BSA to detect the post-op. preserved RV systolic function group were 47.5% (AUC, 0.78; sensitivity, 66.7%; and specificity, 91.7%), 23.1 cm^2/m^2 (AUC, 0.78; sensitivity, 100%; and specificity, 58.3%), and 10.8 cm^2/m^2 (AUC, 0.85; sensitivity, 91.7%; and specificity, 75.0%), respectively, and the preoperative RVESA/BSA had a significantly greater AUC than the RVFAC and RVEDA/BSA (both for $p < 0.001$), indicating the importance of assessing the preoperative RV systolic volume. The associations between preoperative RV parameters and the RVFAC late after surgery are shown in Fig. 4. Although the RVFAC late after surgery was significantly associated with the preoperative RVFAC, RVEDA/BSA, and RVESA/BSA, the association with the RVESA/BSA was prominent.

4. Discussion

In this study we examined the change in RV systolic function before and after isolated TV surgery for severe TR. Although a reduction in the RVEDA occurred immediately after surgery and continued thereafter, a reduction in the RVESA did not occur immediately after surgery but occurred late after surgery. Furthermore, the postoperative reduction in the RVESA was small compared to the RVEDA. As a result, the mean RVFAC was reduced immediately after surgery and a reduction in the RVFAC persisted late after surgery. The RVFAC recovered to the normal range in 50% of patients, whereas the other 50% of patients had impaired RV systolic function, even late after surgery. Although there were no significant differences in the preoperative clinical characteristics and the RVFAC between the post-op. reduced and preserved RV systolic function groups, the preoperative RVESA was significantly smaller in the post-op. preserved RV systolic function group, and therefore might be more useful in detecting recovery of RV systolic function than the preoperative RVFAC.

With the growing number of patients with TR, the number that require surgical treatment is also increasing [6]; however, the indication for TV surgery and the optimal timing is controversial, especially in

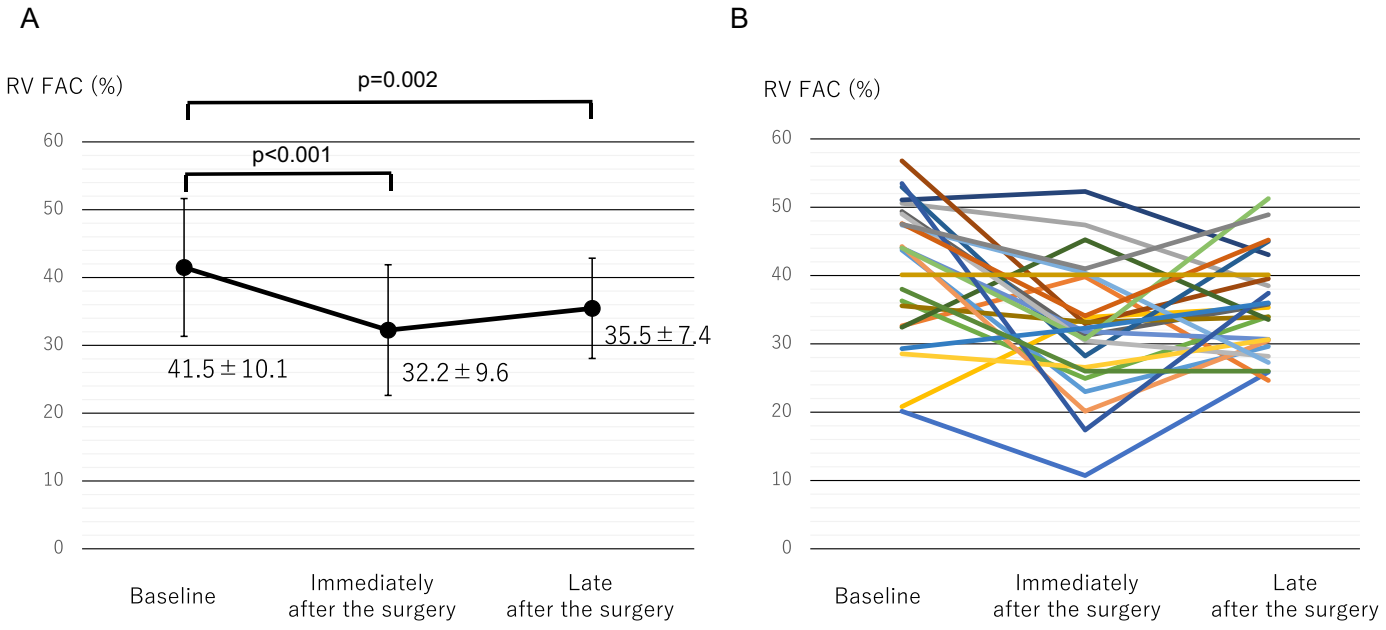

Fig. 1. Change in the RVFAC before and after surgery.
A. The change in the mean RVFAC
The RVFAC was significantly decreased immediately after surgery compared with baseline and was slightly improved late after surgery.
B. The change in the RVFAC in each case
Each line demonstrates a change in the RVFAC for 24 patients.

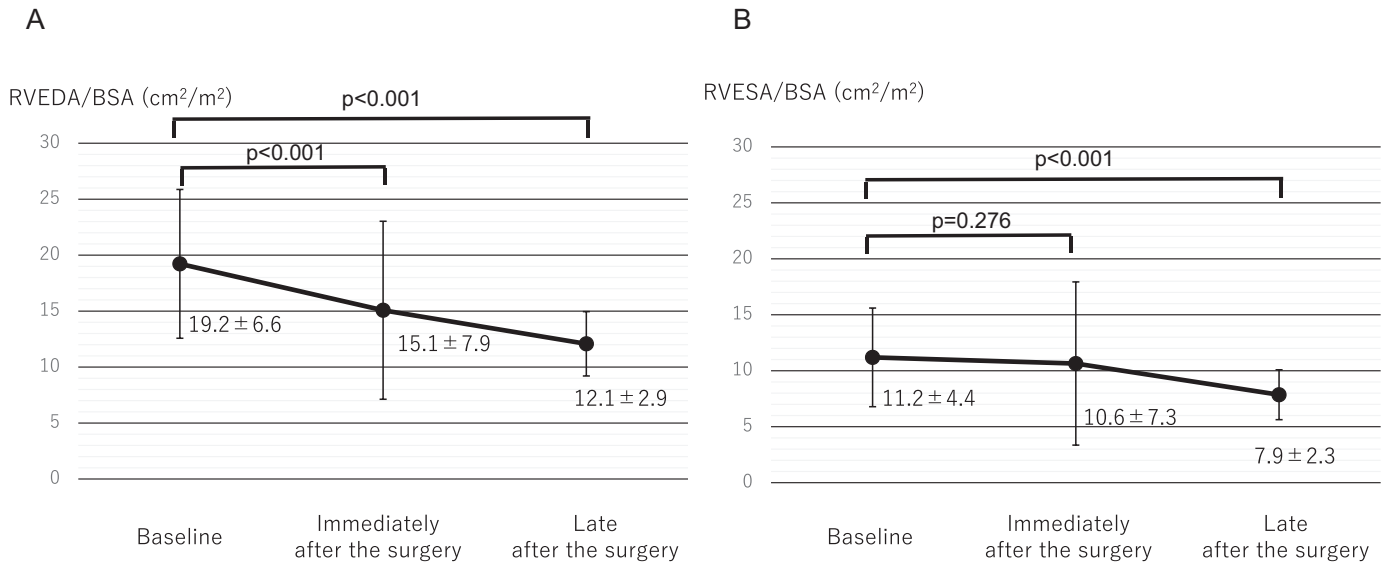

Fig. 2. Changes in the RVEDA (A) and RVESA (B) indexed by BSA before and after surgery.
The RVEDA/BSA significantly decreased immediately after surgery. In contrast, the reduction in the RVESA/BSA occurred late after surgery and was small compared with the RVDSA/BSA.

Table 2
Comparison of preoperative echocardiographic parameters between the groups.

	Total (n = 24)	Reduced RV systolic function group (n = 12)	Preserved RV systolic function group (n = 12)	P
LV ejection fraction (%)	61.1 ± 8.9	59.3 ± 7.6	63.0 ± 10.1	0.33
LA volume index (ml/m^2)	35.7 ± 30.3	41.6 ± 34.2	29.8 ± 26.3	0.40
RVEDA (cm^2)	32.7 ± 13	38.6 ± 12.9	26.8 ± 9.3	0.02*
RVESA (cm^2)	19.1 ± 8.1	23.9 ± 8.0	14.2 ± 4.8	0.002*
RVEDA/BSA (cm^2/m^2)	19.2 ± 6.6	22.4 ± 7.0	16.0 ± 4.6	0.01*
RVESA/BSA (cm^2/m^2)	11.1 ± 4.1	13.8 ± 4.3	8.6 ± 2.6	0.001*
RVFAC (%)	41.5 ± 10.1	37.7 ± 8.5	45.3 ± 10.6	0.06
TA diameter (cm)	4.2 ± 0.8	4.6 ± 0.8	3.9 ± 0.6	0.02*
RA area (cm^2)	36.8 ± 16.1	45 ± 16	29.1 ± 12.5	0.01*
RA area/BSA (cm^2/m^2)	20.6 ± 9.3	25.9 ± 8	17.5 ± 7.2	0.01*

Abbreviations: LV, left ventricular; LA, left atrial; RVEDA, right ventricular end-diastolic area; RVESA, right ventricular end-systolic area; BSA, body surface area; RVFAC, right ventricular fractional area change; TA, tricuspid annulus; RA, right atrial.
Data are shown as the mean ± standard deviation or *n* (%). *Statistically significant difference.

patients with isolated severe TR, due to a lack of clinical evidence. The outcomes after TV surgery are poor in patients who have preoperative severe RV dysfunction resulting from TR [14–16]. In addition, the early postoperative RVFAC has been reported to be a determinant of surgical outcome in patients with isolated TR [15]. Thus, it is essential to perform TV surgery at the time when recovery of RV function can be expected after surgery; however, neither the longitudinal changes in RV systolic function early postoperatively nor the association between pre- and postoperative RV function has been fully understood in patients undergoing surgery for isolated TR. In this study we showed that the RVFAC was reduced immediately after surgery. While the RVFAC recovered to normal in some patients, a reduction in the RVFAC might persist even late after surgery. The reduction in the RVFAC after surgery was similar to the change in LV ejection fraction after surgery for severe mitral regurgitation. Based on a decrease in preload, the LV ejection fraction typically decreases after mitral valve surgery for severe mitral regurgitation [24], thus the preoperative LV ejection fraction may overestimate intrinsic LV systolic function. The TV is an atrioventricular valve, as is the mitral valve. Thus, the RVFAC may also overestimate RV systolic function in patients with significant TR because of regurgitant blood flow to the low-pressure chamber (RA) during systole, as occurs in the LV ejection fraction in patients with severe mitral regurgitation.

A review of previous studies involving mitral valve surgery may provide suggestions for postoperative RV function after TV surgery [25], in agreement with our results. It was reported that LV systolic function decreases immediately after mitral valve surgery and improved late after surgery [25]; however, LV systolic function did not always recover after successful mitral valve surgery. It was concluded that dilatation of the preoperative end-systolic LV dimension is a useful predictor of developing early postoperative LV systolic function following mitral valve surgery because the end-systolic dimension is relatively independent of preload. Like mitral valve surgery, the preoperative RVESA has been reported to be an independent predictor of the postoperative outcome in patients undergoing TV surgery for severe TR [16,26]. In the present study we showed that the preoperative RVESA was smaller in the post-op. preserved RV systolic function group compared to the post-op. reduced RV systolic function group. In addition, the RVFAC late after surgery was more strongly associated with the preoperative RVESA/BSA than the preoperative RVFAC, and the preoperative RVESA/BSA had good sensitivity and specificity for detecting preserved RV systolic function late after surgery. In light of these findings, the RVESA might be a good predictor for recovery of RV systolic function after TV surgery for severe TR because the end-systolic dimension is relatively independent of preload. We reasoned that severe TR based on the dilated RV systolic volume is a surgical indication to prevent persistent postoperative RV dysfunction.

In this study we used the RVFAC as a RV systolic function parameter, which has good correlation with the RV ejection fraction assessed by cardiac MRI [26,27]. Tricuspid annular plane systolic excursion (TAPSE) and the tissue Doppler-derived systolic wave (s') are also widely

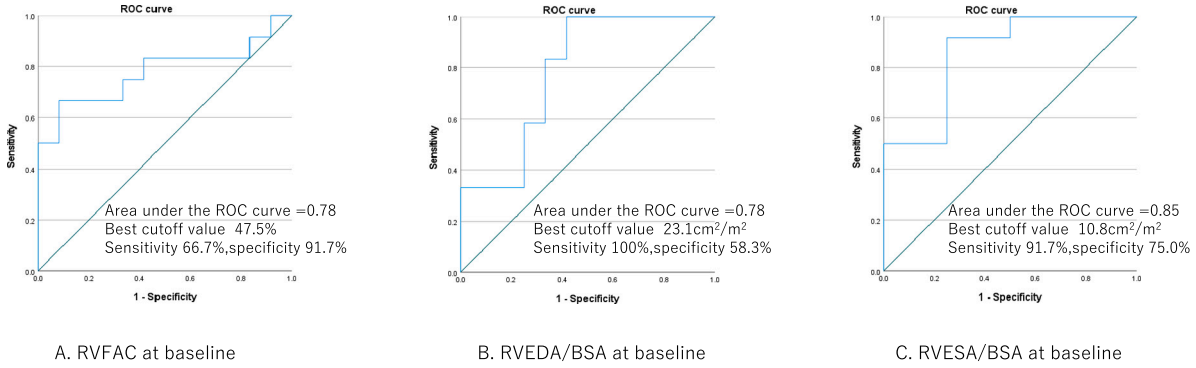

Fig. 3. ROC curve analysis using the preoperative RVFAC, RVEDA/BSA, and RVESA/BSA cut-off values for detecting preserved RVFAC late after surgery. Among the RV parameters, the preoperative RVESA/BSA had the largest area under the ROC curve.

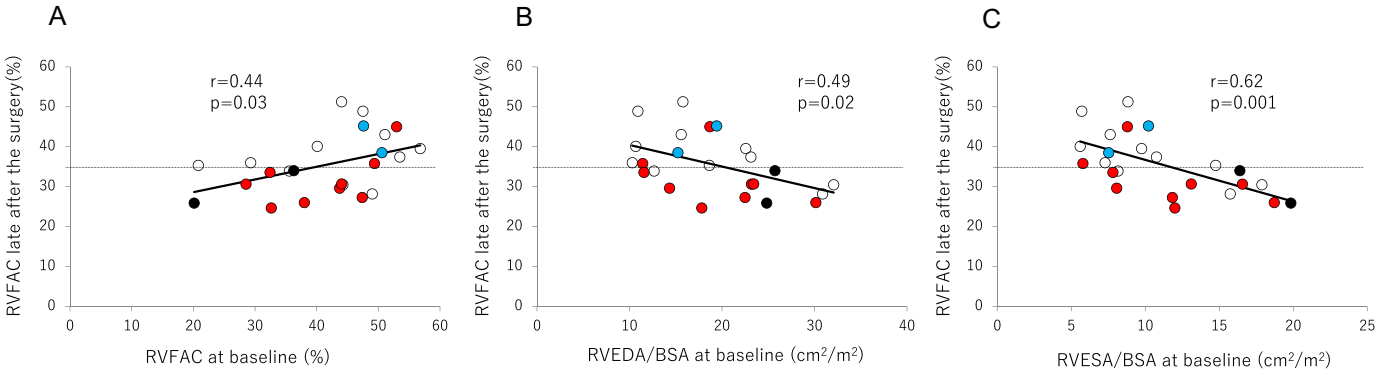

Fig. 4. The associations between preoperative RV parameters and the RVFAC late after surgery.
We showed etiology of individual patient and treatments with different marks. We showed the dots as follows: black dots represented patients with primary TR who underwent TV replacement; blue dots represented patients with secondary TR who underwent TV replacement; white dots represented patients with primary TR who underwent TV plasty; red dots represented patients with secondary TR who underwent TV plasty.
Although the RVFAC late after surgery was significantly associated with all of the preoperative RV parameters, the association with the RVESA/BSA was prominent. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

used as conventional RV systolic function parameters in addition to the RVFAC, and a comprehensive evaluation by multiple RV parameters is considered to be ideal because there are intrinsic limitations for each echocardiographic RV parameter [21,23,28]. In contrast, among patients with severe TR a significant error in the evaluation of RV systolic function by TAPSE and s' has been reported in comparison to the RV ejection fraction by magnetic resonance imaging [29]. Furthermore, TAPSE has been reported to be inaccurate in assessing RV systolic function in patients who previously underwent a tricuspid annuloplasty [30]. Thus, we exploited the RVFAC in the current study; however, recent technological advances provide better assessment of RV function, including RV longitudinal strain, which enables angle-independent assessment of RV function based on 2-dimensional speckle-tracking and 3D echocardiography. Together, such imaging facilitates an accurate assessment of RV volume with complex geometry. These technologies may provide more accurate echocardiographic parameters to predict postoperative RV function in patients undergoing TV surgery for severe TR in corollary studies.

Several limitations of this study should be acknowledged. First, this study included only a small number of patients from four centers because the number of patients undergoing isolated TV surgery for severe TR is limited, although the number of TV surgical procedures is increasing. Second, this was a retrospective study, and 8 patients were transferred to other hospitals after surgery. Therefore, more outcomes than presented in this article were not available. A prospective observational study with a large population is needed to confirm the results and to compare the events between the post-op. preserved RV function group and post-op. reduced RV function group. Third, the RVFAC, which we used in this study, is based on the RV area in the apical 4-chamber view and the RV area, which has intrinsic limitations, may not reflect volume of a RV with complex geometry. Regrettably, we did not measure RV volume either by 3D echocardiography or magnetic resonance imaging that allows accurate measurements of RV volume. A study using 3D echocardiography or magnetic resonance imaging, which allows accurate measurements of RV volume, is expected to overcome this limitation. In addition, we did not measure TAPSE and s' in some patients and these parameters are lacking in our analysis. Because these RV parameters are widely used in routine practice, clinical implications would be more clarified by a further study including these RV parameters. Fourth, we did not consider TR duration in the medical history. A long history of TR may affect postoperative improvement in RV function. Fifth, we did not evaluate medical therapy, which may affect RV function and volume; however, medical therapy for RV systolic function has not been established, thus it is likely difficult to draw definitive conclusions on the effect of medical therapy. On the other hand, the failure to examine the effect of medical therapy including diuretics on the RV volume is a significant limitation of this study. Finally, we did not measure LV volume by the method of disks and stroke volume that represent intravascular and RV volumes in this study. Further study including these parameters is needed to unveil the TV surgical effect on RV function.

In conclusion, we investigated the longitudinal changes in RV systolic function before and after isolated TV surgery for severe TR. We found that RV systolic function significantly decreased immediately

after surgery and might persist even late after surgery. The preoperative RV area was significantly greater in patients with reduced RV systolic function late after surgery. Indeed, the preoperative RVESA/BSA might be helpful in predicting preserved RV function late after surgery. We conclude that a surgical indication for severe TR should be considered before developing significant RV systolic volume dilatation to prevent postoperative RV dysfunction.

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Disclosures

There is no relationship with any industry.

CRediT authorship contribution statement

Megumi Hirokawa: data curation, validation, formal analysis, and writing of the original draft; Masao Daimon: conceptualization, methodology, investigation, writing, review, editing, and supervision; Koki Nakanishi: writing, review, and editing; Keitaro Mahara: data curation, writing, review, and editing; Sakiko Miyazaki: data curation, writing, review, and editing; Makoto Miyake: data curation, writing, review, and editing; Chisato Izumi: data curation, writing, review, and editing; Tomoko Nakao: writing, review, and editing; Norifumi Takeda: writing, review, and editing, Yutaka Yatomi: review, editing, and supervision; Issei Komuro: writing, review, editing, and supervision.

Declaration of competing interest

We have no conflicts of interest in this study.

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