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The Role of Right Ventricular Function in Mitral Valve Surgery

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ABSTRACT

Background: An impaired right ventricular function is associated with a poor survival rate in patients with heart failure. Few investigations have analyzed the prognostic value of right ventricular function on the outcomes of mitral valve (MV) surgery. The objectives of this study were to define the effect of right ventricular function on postoperative outcomes after MV repair (MVP) or replacement (MVR).

Methods: From September 2007 to February 2012, 335 consecutive patients underwent MVP or MVR at our institution. Preoperative transthoracic and transesophageal echocardiography (TEE) and postoperative TEE were used to define right ventricular function and MV performance. Preoperative right ventricular function was graded as normal to mild (grade 1-2) or as moderate to severe (grade 3-4). MV or tricuspid valve regurgitation was graded as non-trivial to mild (grade 0-2) or as moderate to severe (grade 3-4) preoperatively and postoperatively. Survival rate was evaluated at 1 year after surgery.

Results: Of the 334 patients in the study, 280 patients showed a normal to a mildly impaired right ventricular function preoperatively (group 1). Fifty-four patients presented with moderate to severe right ventricular dysfunction (group 2). Patients with a compromised right ventricular function were more likely to undergo MVR (28.6% versus 53.7%, P <.001). The mean pulmonary artery pressure was 23.6 mm Hg in group 1 and 34 mm Hg in group 2 (P <.001). The left atrial diameter was 4.6 cm in group 1 and 5.3 cm in group 2 (P <.001). The 2 groups were not different with respect to operative mortality, but the patients in group 2 experienced more transfusion of blood products (588.4 mL versus 1180.6 mL, P <.001), longer intensive care unit stays (83.9 versus 149.6 hours, P <.001), and hospital stays (8.9 versus 12.8 days, P = .005). The rate of postoperative MV regurgitation was significantly higher in group 2 (1.8 versus 14.8%, P <.001). The overall 1-year survival rate was 92.5% in group 1 and 94.5% in group 2 (P = .59).

Conclusions: This study has shown that a dysfunctional preoperative right ventricular function uses more resources and is associated with postoperative MV regurgitation, but it is not associated short- and mid-term mortality after MV surgery.

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INTRODUCTION

The prognostic value of right ventricular (RV) function in valvular heart surgery has been recognized for several years. Previous studies have shown that preoperative RV dysfunction is associated with postoperative mortality [Wencker 2000] and with more postoperative inotropic support [Bolft 1992] after valve surgery. A variety of factors are known to predict outcomes after mitral valve (MV) surgery. The assessment of RV function is complex, however, and there is no consensus regarding a noninvasive method for evaluating RV function. Few investigations have analyzed the prognostic value of RV indices after MV repair (MVP) or replacement (MVR). The main objective of this study was to determine the prognostic value of RV function in MV surgery.

METHODS AND STUDY POPULATION

Patients

The study included 334 consecutive patients who underwent MVR or MVP in our institution between September 2007 and February 2012. Demographics, preoperative comorbidities, intraoperative features, and postoperative outcomes were prospectively collected in the cardiac surgery database in accordance with Society of Thoracic Surgeons (STS) definitions. Postoperative outcomes include complications, resource use, operative mortality, and postoperative MV regurgitation. This study was approved by the Institutional Review Board.

Echocardiographic Assessment

All patients had at least 1 routine preoperative transthoracic echocardiogram, preoperative transesophageal echocardiogram (TEE), and intraoperative TEE. The

Table 1. Preoperative Characteristics of the Groups*

	Grade 1-2 (n = 280)	Grade 3-4 (n = 54)	*P*
Age, y	58.3 ± 13.8	61.6 ± 11.4	.17
BMI, kg/m^2	27.5 ± 6.6	27.6 ± 5	.92
STS score	3.2 ± 6.0	6.3 ± 8.2	<.0001
Diabetes, % (n)	14.6 (41)	16.7 (9)	.70
Hypertension, % (n)	63.9 (179)	64.8 (35)	.90
RF dialysis, % (n)	5 (14)	5.6 (3)	.87
Endocarditis, % (n)	17 (47)	9.3 (5)	.16
Previous MI, % (n)	12.1 (34)	5.6 (3)	.16
AF, % (n)	16.4 (46)	33.3 (18)	.004
Female sex, % (n)	45.7 (128)	46.3 (25)	.94

*Data are presented as the mean ± SD or as (%) n, as indicated. STS indicates Society of Thoracic Surgeons; BMI, body mass index; RF, renal failure; MI, myocardial infarction; AF, atrial fibrillation.

echocardiographic results were reviewed by a cardiologist with specialty training in echocardiography. Also measured were the MV annulus, left atrial (LA) size, left ventricular internal dimension in systole (LVIDs), left ventricular internal dimension in diastole (LVIDd), left ventricular posterior wall dimensions (LVPWD), tricuspid regurgitation maximum velocity (TRmax velocity), tricuspid regurgitation maximum pressure gradient (TRmax PG), and interventricular septal thickness at end-diastole (IVSD).

MV regurgitation and tricuspid valve regurgitation were graded according to the American Society of Echocardiography consensus [Zoghbi 2003]. Preoperative RV function was assessed.

Pulmonary Artery Pressure

In addition to intraoperative TEE, all patients were monitored with a Swan-Ganz pulmonary artery catheter. Preoperative and intraoperative pulmonary artery pressure (PAP) data were obtained from the anesthesia record.

Anesthesia, Cardioprotection, and Surgery

Standard anesthesia, cardiopulmonary bypass (CPB), and myocardial-protection methods were used. All surgeons in our hospital had significant experience with MVR and MVP, and the type of operation chosen was made according to the individual surgeon's preference.

In brief, CPB was established in a standard manner with an extracorporeal circuit between the right atrium and the ascending aorta and in nonpulsatile mode with moderate systemic hypothermia (28°C-32°C). A minimum pump flow of 2.2 to 2.4 L/min per m^2 and a mean arterial pressure of 60 mm Hg were maintained during the course of CPB for all procedures. Cardiac arrest and myocardial protection were obtained by infusion of cold (4°C–6°C) blood cardioplegic solution. MVR and MVP surgeries did not differ with respect to the technique of cardioplegia administration. All of the patients in all procedures received cold intermittent cardioplegia and topical cooling. The composition of 4:1 cardioplegia (4 parts blood diluted with 1 part St Thomas I crystalloid cardioplegia) was administered at 10 to 15 mL/kg at 30-minute intervals via the CPB delivery system. Intraoperative TEE was used routinely in all MV procedures to monitor cardiac function and valve performance. The pathologic diagnosis was acquired from medical records and surgery notes.

Statistics

The results are expressed as the mean ± SD for continuous variables and as a percentage (number of cases) for categorical variables. The 2 groups were compared with the Student t test for continuous variables and with the chi-square test for categorical variables. Univariate analyses were used to find potential predictors of the outcomes of mortality and postoperative MV regurgitation.

One-year overall mortality was assessed by estimating the absolute risk with the Kaplan-Meier method, the lengths of time to a first event for the 2 groups were evaluated with the 2-sample log-rank test, and relative risk was calculated with the Cox proportional hazards model. Patients in the group with a compromised RV function were compared with the patients in the group with a preserved right function. *P* values <.05 were considered statistically significant. All analyses were performed with SAS 9.0 software (SAS Institute, Cary, NC, USA).

RESULTS

We identified 280 patients with a normal to mildly impaired RV function (group 1) and 54 patients with moderate to severe RV dysfunction (group 2). The preoperative demographic and clinical characteristics of the study population are summarized in Table 1. Group 2 patients had a higher incidence of preoperative atrial fibrillation (33.3% versus 16.4%, *P* = .004) and a higher STS mortality score (6.3 versus 3.2, *P* <.001).

Hemodynamic and echocardiographic measurements are summarized in Table 2. Patients with RV dysfunction (group 2) were more likely to have a lower left ventricular ejection

Table 2. Hemodynamic Data and Echocardiography Results*

	Grade 1-2 (n = 280)	Grade 3-4 (n = 54)	*P*
MVR, % (n)	28.8 (80)	53.7 (29)	.01
MVP, % (n)	71.4 (200)	46.3 (25)	.01
Elective surgery, % (n)	60.7 (170)	42.6 (23)	.00
LVEF, % (n)			<.0001
≥35%	96.4 (270)	81.5 (44)	
<35%	3.6 (10)	18.6 (10)	
Mitral stenosis, % (n)	5 (14)	22.2 (12)	<.0001
Mitral insufficiency, % (n)			.001
Mild	3.4 (8)	18.2 (8)	
Moderate to severe	96.6 (226)	81.8 (36)	
Tricuspid insufficiency grade, % (n)			<.001
0-2	97 (227)	85.2 (46)	
3-4	2.1 (6)	14.8 (8)	
CPB time, min	86.7 ± 41.4	103.1 ± 38.7	.01
Cross-clamp time, min	65.7 ± 33.3	76.2 ± 31.3	.03
MV annulus, cm	4.0 ± 0.6	3.9 ± 0.4	.19
MV prolapse, % (n)			.19
Anterior	7.5 (21)	7.4 (4)	
Posterior	26.7 (73)	20.4 (11)	
Both	8.9 (25)	1.9 (1)	
No	57.5 (161)	70.4 (38)	
Chord rupture, % (n)			.234
Anterior	6.4 (18)	7.4 (4)	
Posterior	36.8 (103)	25.9 (14)	
Both	5.4 (15)	1.9 (1)	
No	51.4 (144)	64.8 (35)	
Annulus dilation, % (n)	56.7 (159)	37 (20)	.008
Leaf motion restriction, % (n)	12.1 (34)	7.4 (4)	.316
LVIDs, cm	3.4 ± 0.8	3.6 ± 1.1	.769
LVIDd, cm	5.2 ± 0.7	5 ± 1.3	.601
Left atrial diameter, cm	4.6 ± 0.8	5.3 ± 0.8	<.0001
PAP, mm Hg	23.6 ± 9.4	34 ± 14.7	<.0001
LVPWD, cm	1.1 ± 0.3	1.1 ± 0.2	.951
TRmax velocity, cm · s	286.3 ± 55.5	309.9 ± 62.8	.03
TRmax PG, mm Hg	35.4 ± 14.8	40.4 ± 15.9	.077
IVSD, cm	1.2 ± 0.2	1.2 ± 0.2	.826

*Data are presented as the mean ± SD or as (%) n, as indicated. MVR indicates mitral valve replacement; MVP, mitral valve repair; LVEF, left ventricular ejection fraction; CPB, cardiopulmonary bypass; LVIDs, left ventricular internal dimension in systole; LVIDd, left ventricular internal dimension in diastole; PAP, pulmonary artery pressure; LVPWD, left ventricular posterior wall dimension; TRmax velocity, tricuspid regurgitation maximum velocity; TRmax PG, tricuspid regurgitation maximum pressure gradient; IVSD, interventricular septal thickness at end-diastole.

fraction (LVEF), MV stenosis, and severe tricuspid valve insufficiency preoperatively, and group 2 patients were more likely to undergo MVR rather than MVP, compared with patients with a preserved RV function. Patients with a preserved RV function were more likely to undergo MVP than MVR than patients with RV dysfunction (71.4% versus 46.3%, P <.001).

The 2 groups were significantly different with respect to some of the echocardiographic data. Patients in group 2 had

Table 3. Postoperative Outcomes for the 2 Groups*

	Grade 1-2	Grade 3-4	*P*
Mortality, % (n)	4.6 (13)	3.7 (2)	.76
Stroke, % (n)	1.4 (4)	0	.38
New-onset AF, % (n)	26.8 (75)	24.1 (13)	.68
Postoperative myocardial infarction, % (n)	0.5 (1)	0	1.00
Length of hospital stay, d	8.9 ± 10.8	12.8 ± 13	.01
Prolonged ventilation, % (n)	16.4 (46)	44.4 (24)	<.0001
Renal failure, % (n)	2.5 (7)	7.4 (4)	.06
Total blood transfusion, mL	588.4 ± 895	1180 ± 1636	<.001
Total ICU time, h	83.9	149.55	<.0001
Post-MR (grade 1-2), % (n)	1.8 (5)	14.8 (8)	<.001

*Data are presented as the mean ± SD or as (%) n, as indicated. AF indicates atrial fibrillation; ICU, intensive care unit; Post-MR, postoperative MV regurgitation.

Table 4. Postoperative Pathologic Diagnosis

	Grade 1-2	Grade 3-4	*P*
Degenerative, % (n)	54.3 (152)	51.9 (28)	.74
Endocarditis, % (n)	14.6 (41)	14.8 (8)	.97
Rheumatic, % (n)	4.3 (12)	7.4 (4)	.33

Table 5. Univariate Logistic Regression for Operative Mortality*

Variable	Odds Ratio	95% CI	*P*
Age	1.04	0.99-1.09	.12
Diabetes	8.40	2.54-27.8	.00
Renal failure and dialysis	3.83	0.75-19.5	.11
Infective endocarditis	2.83	0.81-9.83	.10
LVEF	0.97	0.92-1.02	.20
STS mortality score	1.12	1.06-1.12	.00
RV function (grade 1-2)	0.98	0.21-4.62	.98
PAP	1.03	0.99-1.07	.19
LVID	0.49	0.24-1.00	.05
Mitral annulus size	0.09	0.00-1.76	.11
Perfusion time	1.01	1.00-1.02	.01
Cross-clamp time	1.01	1.00-1.03	.02
Post-MR (grade 1-2)	0.23	0.06-0.88	.03

*CI indicates confidence interval; LVEF, left ventricular ejection fraction; STS, Society of Thoracic Surgeons; RV, right ventricle; PAP, pulmonary artery pressure; LVID, left ventricular internal dimension; Post-MR, postoperative mitral valve regurgitation.

larger left atria (5.3 cm versus 4.6 cm, P <.001), a higher preoperative mean PAP (MPAP) (34 mm Hg versus 23.6 mm Hg, P <.001), and a higher TRmax velocity (309.9 cm · s versus 286.3 cm · s, P = .03).

Patients with a compromised RV function experienced longer perfusion times (103.1 minutes versus 86.7 minutes, P = .008) and cross-clamp times (76.0 minutes versus 65.7 minutes, P = .03) than patients with a good RV function.

Outcomes were significantly better for group 1 patients in the univariate analysis (Table 3). Striking differences in postoperative hospital stay, blood transfusion, and prolonged ventilation were observed. Operative mortality rates were similar (4.6% versus 3.7%, P = .761), but patients in group 2 had a significantly increased incidence of postoperative MV regurgitation (14.8% versus 1.8%, P <.001).

Pathologic diagnoses are detailed in Table 4. The 2 groups had similar distributions of degenerative, endocarditis, and rheumatic diseases.

Tests with the univariate logistic model identified the following predictors of operative mortality (Table 5): diabetes (odds ratio [OR], 8.4; 95% confidence interval [CI], 2.54-27.8; P = .000], postoperative MV regurgitation (OR, 0.23; 95% CI, 0.06-0.88; P = .031), and longer perfusion (OR, 1.01; 95% CI, 1.00-1.02; P = .005) and cross-clamp (OR, 1.01; 95% CI, 1.00-1.05; P = .02) times. Patients with a good preoperative RV function (OR, 3.25; 95% CI, 1.46-7.23; P = .004) and without diabetes (OR, 0.42; 95% CI, 0.18-1.00; P = .049) were less likely to experience moderate to severe MV regurgitation and had shorter perfusion (OR, 0.98; 95% CI, 0.98-0.99; P = .000) and cross-clamp (OR, 0.97; 95% CI, 0.97-0.99; P = .000) times during surgery (Table 6). In other words, patients with an impaired RV function, diabetes, and longer perfusion and cross-clamp times were more likely to experience moderate to severe MV regurgitation postoperatively.

Of the 36 deaths, 31 patients died in group 1, and 5 patients died in group 2. Fifteen deaths occurred within 30 days postoperatively, and 21 deaths occurred after surgery until the follow-up date, May 21, 2013.

Table 6. Univariate Logistic Regression for Postoperative Mitral Valve Regurgitation (Grade 1-2)*

Variable	Odds Ratio	95% CI	*P*
RV function (grade 1-2)	3.25	1.46-7.23	.00
Diabetes	0.42	0.18+1.00	.05
Infective endocarditis	0.43	0.19-0.99	.05
Preoperative myocardial infarction	0.44	0.17-1.12	.08
BMI	0.96	0.91-1.01	.13
STS mortality score	0.96	0.92-1.01	.13
PAP	1.00	0.97-1.03	.99
MVR	0.34	0.14-0.82	.02
Perfusion time	0.98	0.98-0.99	.00
Cross-clamp time	0.71	0.45-1.14	.16
LA diameter	0.71	0.45-1.14	.16
RV012	3.35	1.28-8.75	.01

*CI indicates confidence interval; RV, right ventricular; BMI, body mass index; STS, Society of Thoracic Surgeons; PAP, pulmonary artery pressure; MVR, mitral valve replacement; LA, left atrium; RV012, right ventricular function (group 1, n = 280) .

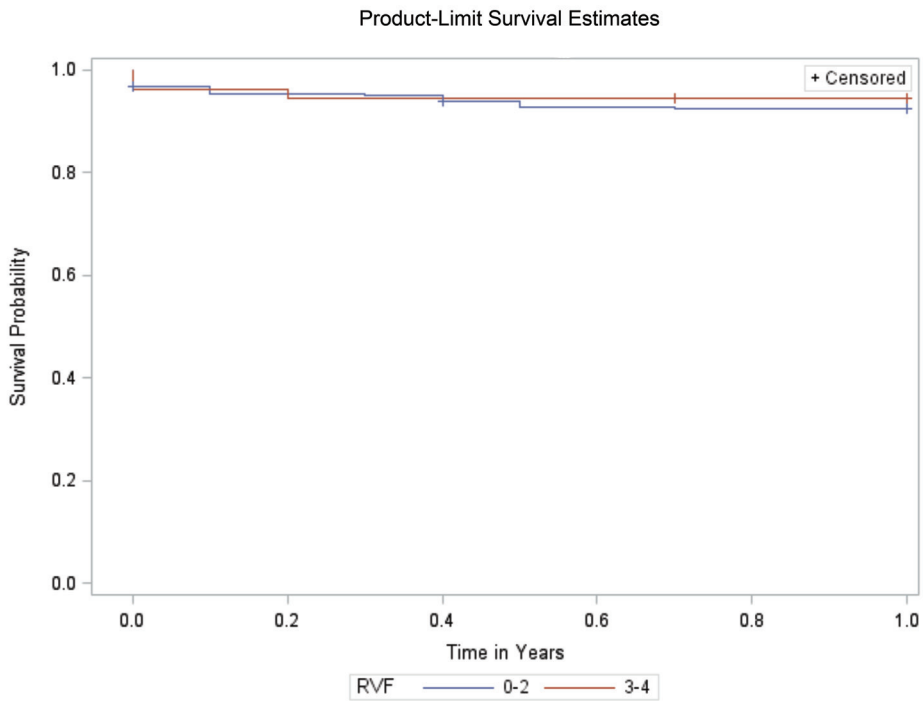
The overall survival probability at 1 year was 92.4% for patients in group 1 and 94.6% for patients in group 2 (*P* = .59; Figure, curve 1).

COMMENTS

The main finding of this study was that preoperative assessment of RV function could improve the risk stratification of patients undergoing MV surgery. The result also supports the conclusion that preoperative RV dysfunction is correlated with worse clinical outcomes.

The evaluation of RV performance is very important for its clinical and prognostic value for several conditions, such as after cardiac surgery. The vulnerability of the RV due to poor RV protection during CPB is widely recognized [Mishra 1998; Ghio 2001]. Although most patients with enough functional reserve are able to tolerate small decreases in RV and LV function, those with significant baseline dysfunction may not be able to tolerate further deterioration.

In nonsurgical patients, previous studies have identified worse outcomes, with RVEF values of <35% [Reichert 1992; De Groote 1998]. The combined morbidity and mortality rate is as high as 47% in patients when damage to RV function has been demonstrated by echocardiography, electrocardiography, or both [Kinch 1994; De Groote 1998; Sakata 2000]. Lansman et al reported that 42 patients who had a preoperative LVEF of <20% and underwent coronary artery



Survival rate at the 1-year follow-up (hazard ratio, 1.24; P = .59). RVF, right ventricular function group (group 1, grade 1-2; group 2, grade 3-4).

bypass grafting exhibited a trend (P = .07) toward a correlation between preoperative RV function and outcome [Lansman 1993]. Despite these studies, current risk assessments do not include RV function as a significant risk factor.

Pinzani et al [1993] showed that preoperative RV failure (as defined by clinical, hemodynamic, or echocardiographic criteria) was related to perioperative mortality in MV and mitral-aortic valve surgery. A multivariate analysis, however, showed that only age was significantly related to perioperative mortality. In a prospective study of 14 patients with severe nonischemic MV regurgitation and at high risk (LVEF 45% or RVEF 20%), Wencker and colleagues [2000] showed that a preoperative RVEF of 20% predicted late postoperative death. Although statistically significant, the value of these results was limited by the small number of events. In aortic stenosis, Boldt and colleagues [1992] demonstrated that RV function was related to the severity of the valvular stenosis and associated with a greater requirement of postoperative inotropic support.

Several factors contribute to RV dysfunction in valvular disease, including pulmonary hypertension (PH), ventricular interdependence, RV myocardial ischemia [Morrison 1983; Borer 1991], and type of valvular disease. Aortic stenosis usually presents with a preserved RV function [Morrison 1984]. In contrast, MV stenosis is an important cause of RV dysfunction and PH. RV function is usually better preserved in MV regurgitation than in MV stenosis [Wroblewski 1981; Borer 1991].

PH frequently complicates RV function and is generally considered an indicator of a poor prognosis [Abramson 1992]. The RV is more vulnerable to an excessive afterload than preload. The pulmonary circulation is a central determinant of RV afterload, and an increase in RV ejection impedance can easily lead to RV failure and tricuspid regurgitation. An increased PAP is coupled with a reduced RV systolic function. RV dysfunction represents a marker of more severe valvular heart disease or concomitant pulmonary disease. In our study of the 2 univariate models, preoperative RV dysfunction is a risk factor for significant postoperative MV regurgitation. Ghio and colleagues [2001] investigated the independent and additive prognostic value of RV systolic function and PAP in patients with chronic heart failure and demonstrated that exceptions to the physiological relationship between the PAP and RVEF may be observed frequently in clinical practice. They suggested that the development of RV dysfunction in response to an increased afterload is time dependent. An alternative explanation is that more favorable RV remodeling could determine a better function in some patients.

A high prevalence of preoperative atrial fibrillation was observed in patients with normal pulmonary pressure and RV dysfunction, a finding leading to the hypothesis that the absence of active atrial contraction could play a major role in the pathogenesis of RV dysfunction.

Infective endocarditis exhibits a trend toward an association with postoperative MV regurgitation, but not with mortality. Diabetes is associated with mortality but not with postoperative MV regurgitation. Perfusion time and cross-clamp time are significantly associated with operative mortality and significant postoperative MV regurgitation. Although other variables were shown to be statistically significant in the univariate logistic model, these results were limited by the small number of events. Because 12 patients experienced significant postoperative MV regurgitation and 12 patients experienced postoperative mortality, we were unable to build 2 multivariate logistic models to define the risk factors for operative mortality and postoperative MV regurgitation. Variables of increased risk are likely to be picked up by multivariate logistic models.

The effectiveness of the surgery was impressive, because the 2 groups did not differ in their 1-year mortality rates. This finding may reflect the fact that the surgery and cardiology staff recognize the deleterious effect of poor RV function and the importance of careful evaluation of the RV prior to MV surgery; some patients with a poor RV function are determined to be inoperable. Once the patient with a poor RV function survives surgery, the reduction in MV regurgitation may allow the RV dysfunction to reverse or stabilize. Onorato et al reported that surgery to treat MV regurgitation could reverse left ventricular remodeling and improve RV function [Onorato 2009], but how the procedure reverses the RV remodeling remains undefined.

Study Limitations

The limitations of this study include its retrospective nature, the single-center experience, and the small number of events. Another limitation is the lack of an independent method for assessing RV function (invasive or nuclear). Echocardiography, however, is a well-documented and simple method for assessing RV function with high sensitivity and specificity, as previous studies have demonstrated.

With regard to the clinical outcomes, the limitations are mainly related to the small number of events and the multifactorial nature of postoperative outcomes. The small number of events prevented us from building multivariate logistic regression models for defining the power of RV function to determine outcomes and identifying other independent risk factors for mortality and significant MV regurgitation.

CONCLUSIONS

Preoperative RV function is associated with postoperative outcomes after MV surgery. The use of noninvasive techniques (echocardiography, magnetic resonance imaging) and studies involving a larger cohort of patients may help standardize future evaluations of RV function for further stratifying overall risk following MV surgery.

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