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Right Ventricular Performance Following Valve Repair for Chronic Degenerative Mitral Regurgitation

Running head: Right ventricle and mitral valve repair

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Abstract

Background: Our aim was to assess right ventricular (RV) performance following mitral valve repair using RV focused echocardiography, and to evaluate the influence of elevated pulmonary artery systolic pressure (PASP) on RV recovery.

Methods: Forty consecutive patients undergoing mitral valve repair were prospectively investigated with RV focused echocardiography, including 2-dimensional speckle tracking-derived longitudinal strain and measurement of N-terminal protype-B natriuretic peptide levels performed on the day before surgery and six months postoperatively.

Results: The 30-day mortality was 0%. Overall survival was 97.5±2.5% at 6-month follow-up and the prevalence of postoperative RV dysfunction was 61% (*n*=22). Conventional longitudinal indices presenting RV function decreased significantly following surgery (*n*=36): tricuspid annular plane systolic excursion (mean 24±5 mm vs. mean 15±3 mm, $p<0.001$), systolic peak velocity (mean 14±3 cm/s vs. mean 10±2 cm/s, $p<0.001$), isovolumic acceleration time (mean 2.5±1.0 cm/s^2 vs. mean 2.1±0.7 cm/s^2, $p=0.022$); but the RV free wall, septal, and global strain did not change significantly. Patients with preoperative PASP >50 mmHg showed a significant increase in postoperative RV global strain compared to those without (mean difference 10±30% vs. -17±23%, $p=0.033$).

Conclusions: RV dysfunction was common at 6-month follow-up. Pulmonary hypertension, although reversible following surgery, had a negative effect on RV function. Speckle tracking-derived RV strain may assist in the prioritization of surgical referrals in order to avoid biventricular impairment.

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Introduction

Right ventricular (RV) dysfunction is the result of a complex interaction between the remodeled and enlarged left ventricle, septal performance and pulmonary artery systolic pressure (PASP) with or without degenerative mitral regurgitation (MR) (1-3). Le Tourneau *et al.* have shown that RV dysfunction quickly improves following mitral valve surgery (4) and is not associated with poor survival as demonstrated by Sun *et al.* (5). RV impairment is common in chronic degenerative MR and in combination with reduced left ventricular (LV) ejection fraction is a strong predictor of poor cardiovascular and overall survival (4). Thus, a reliable and systematic RV assessment may identify early signs of RV dysfunction, which may assist in the prioritization of surgical referrals in order to avoid biventricular impairment.

An echocardiographic RV evaluation is often part of the preoperative work-up, but the complex geometry of the right ventricle makes it difficult to assess the performance of this cardiac chamber. In addition, the echocardiographic parameters for assessment of RV dysfunction (2, 6) may be difficult to interpret after cardiothoracic surgery according to the results of previous studies showing postoperative disruption of the native RV contractile pattern (7, 8). The reduction in the RV longitudinal contractile pattern has previously been described with several conventional echocardiographic indices (8), indicating that these observations are not related to a particular method of RV assessment but rather to a more fundamental change in the balance of longitudinal and contractile RV shortening after cardiac surgery (8). Speckle tracking-derived strain is a new modality for assessment of cardiac function (9). Maffessanti *et al.* have indicated the usefulness of this modality in evaluation of RV function following mitral valve repair (10). However, there have been few studies evaluating RV performance with speckle tracking-derived strain following mitral valve repair. Furthermore, neither the incidence of RV dysfunction following mitral valve repair nor the influence of elevated PASP on RV recovery are fully understood.

Our aim was therefore to prospectively assess RV performance following mitral valve repair using RV focused echocardiography and to determine the influence of elevated PASP on RV recovery.

Patients and methods

This prospective study was conducted recruiting 40 consecutive patients in sinus rhythm who were scheduled for elective mitral valve repair between April 2010 and March 2013. Exclusion criteria were: MR of etiologies other than degenerative mitral valve disease; chronic preoperative atrial fibrillation; concomitant aortic, tricuspid, or pulmonary valve stenosis or more than mild regurgitation; concomitant Cox-Maze procedure; previous cardiac surgery; infective valve endocarditis; and emergency surgery. Individual consent was obtained from all study participants and the study protocol had been approved by the ethics committee for clinical research at Lund University, Sweden.

A transthoracic echocardiographic examination was performed on Philips iE33 (Philips, Andover, MA) on the day before surgery and six months postoperatively. Echocardiographic data were obtained by two experienced investigators (CM and AI) and measurements were performed according to established guidelines (11) and blind regarding clinical data. Assessment of RV function was performed using the apical 4-chamber and parasternal RV inflow and outflow tract views. Following quantitative conventional indices were measured: fractional area change, tricuspid annular plane systolic excursion, tissue Doppler-derived tricuspid lateral annular systolic peak velocity, right ventricular index of myocardial performance, and isovolumic acceleration. All echocardiographic parameters were taken into consideration and an overall RV dysfunction was graded qualitatively as normal, mild, moderate, or severe by an experienced cardiologist (CM). PASP was calculated from tricuspid regurgitation maximum pressure gradient using the modified Bernoulli equation, adding an estimate of central venous pressure. Following 2-dimensional speckle tracking-derived RV longitudinal strain was evaluated: global, free wall, and septal. Strain data were analyzed using Philips Q.lab version 9.0 (Philips Healthcare, Best, the Netherlands).

Peroperative myocardial protection was achieved with moderate hypothermia and intermittent combined antegrade and retrograde cold-blood cardioplegia. Transesophageal echocardiography was used to assess valve repair or prosthesis function perioperatively. The invasive hemodynamic evaluation was performed using a 7.5F single-lumen, balloon-tipped, flow-directed thermodilution

fiber-optic pulmonary artery catheter (ICU Medical, Inc., San Clemente, CA) and values were indexed to body surface area.

The NT-proBNP levels were assessed collecting blood samples preoperatively and six months after surgery and using the electrochemiluminiscence immunoassay Elecsys proBNP (Roche Diagnostics, Mannheim, Germany).

Follow-up was 100% complete at 6.3±1.5 months (median 6.1 months, IQR 6.0─7.2), and totaled 18.7 patient-years. One 84-year-old patient succumbed to pneumonia 3.5 months after surgery. One patient declined participation 5 months postoperatively. Two patients had incomplete echocardiographic data. Data obtained six months after surgery included an echocardiographic assessment and NT-pro-BNP measurement.

Categorical variables were expressed as proportions and percentages and continuous variables as mean ± 1 standard deviation. Student's t-test or Mann-Whitney U test was used where appropriate for continuous variables. Categorical data were compared using the chi-squared test, or Fisher's exact test when the expected frequency was less than five. The paired-sample t-test or Wilcoxon test was used where appropriate. Statistical significance was defined as $p<0.05$. Statistical analysis was performed using IBM SPSS software version 21.0 (IBM Corp., Armonk, NY).

Results

The 30-day mortality was 0%. Overall survival was 97.5±2.5% (39/40) at 6-month follow-up. The clinical characteristics of the study population are given in Table 1. The etiology of MR was degenerative in all cases, including prolapse of the posterior leaflet (77.5%, *n*=31), the anterior leaflet (5%, *n*=2), or both leaflets (17.5%, *n*=7). Perioperative transesophageal echocardiography did not reveal any systolic anterior motion, stenosis, paravalvular leakage, or more than mild mitral or tricuspid regurgitation in any of the patients after weaning from extracorporeal circulation. During postoperative follow-up, 6% of the patients (2/36) developed chronic atrial fibrillation. Diuretics were used in 33% (12/36), 75% (27/36) were on beta-blocker treatment, and 56% (20/36) were on ACE inhibitor treatment. At follow-up, 100% of the patients (*n*=36) were in NYHA class I─II.

The echocardiographic data are presented in Table 2. The prevalence of preoperative RV dysfunction was 32% (*n*=12); eight patients had mild dysfunction and four had moderate dysfunction. Patients with preoperative RV dysfunction did not differ significantly from patients with normal RV performance regarding variables indicating left-sided remodeling or presence of pulmonary hypertension: LV ejection fraction (63±13% vs. 66±8%, *p*=0.475), LV end-systolic dimension (39±10 mm vs. 37±6 mm, *p*=0.510), LV end-diastolic dimension (62±9 mm vs. 63±8 mm, *p*=0.597), left atrial volume index (mean 73±17 mL/ m^2 vs. mean 66±21 mL/ m^2, *p*=0.313), and PASP (53±13 mmHg vs. 47±14 mmHg, *p*=0.265).

At 6-month follow-up, the prevalence of RV dysfunction was 61% (*n*=22): 17 patients had mild dysfunction and five had moderate dysfunction (Figure 1). Conventional indices representing RV function, such as tricuspid annular plane systolic excursion, tissue Doppler-derived tricuspid lateral annular systolic peak velocity, and isovolumic acceleration decreased significantly after surgery, whereas the RV free wall, septal, and global strain did not change significantly after surgery (Table 2).

Data were stratified by preoperative PASP dichotomized at 50 mmHg (12) (Table 3 and 4). Preoperative RV performance were significantly impaired in patients with preoperative PASP >50

mmHg compared to those with preoperative PASP ≤50 mmHg: RV free wall strain (-21±6% vs. -27±6%, p=0.005) and global strain (-16±4% vs. -21±6%, p=0.009), fractional area change (31±9% vs. 39±11%, p=0.019), and deceleration time (81±18 ms vs. 103±19 ms, p=0.002). Data representing preoperative left-sided cardiac remodeling did not differ between the two groups (Table 3 and 4).

At 6-month follow-up, patients with preoperative PASP >50 mmHg had a significant decrease in PASP compared to those with preoperative PASP ≤50 mmHg (mean difference -22±12 mmHg vs. -10±8 mmHg, p=0.002). There was a significant increase in postoperative RV global strain in patients with preoperative PASP >50 mmHg compared to those without (mean difference 10±30% vs. -17±23%, p=0.033). The changes in RV strain following surgery are presented in Figures 2 and 3.

Patients with preoperative RV dysfunction had significantly higher mean pulmonary artery pressure than those with normal RV performance (32±10 mmHg vs. 23±9 mmHg, p=0.008), while cardiac index or pulmonary capillary wedge pressure did not differ between the two groups (2.1±0.5 L/min/m^2 vs. 2.1±0.5 L/min/ m^2, p=0.956 and 19±9 mmHg vs. 14±6 mmHg, p=0.077) after induction of general anesthesia.

The mean preoperative NT-proBNP level in patients with RV dysfunction was significantly higher than in those with normal RV performance (2478±3506 ng/L vs. 276±199 ng/L, p<0.001). At 6-month follow-up, the mean NT-proBNP was reduced, but not significantly so, in patients with preoperative RV dysfunction (918±899 ng/L vs. 2478±3506 ng/L, p=0.182). Patients with normal preoperative RV performance showed a significant increase in mean NT-proBNP at 6-month follow-up relative to the mean preoperative level (449±366 ng/L vs. 276±199 ng/L, p=0.001).

The mean NT-proBNP level in patients with preoperative PASP >50 mmHg was significantly higher preoperatively than in those with preoperative PASP ≤50 mmHg (1852±3188 ng/L vs. 364±332 ng/L, p=0.021). In patients with preoperative PASP >50 mmHg, the mean NT-proBNP was reduced, but not significantly so, at 6-month follow-up (552±357 ng/L vs. 1852±3188 ng/L, p=0.173). Patients with

PASP ≤50 mmHg had a significant increase in the mean NT-proBNP at 6-month follow-up (647±756 ng/L vs. 364±332 ng/L, p=0.002).

Comment

In the present study, the prevalence of RV dysfunction - as reflected by conventional echocardiographic indices - increased following mitral valve repair. However, RV performance measured by speckle tracking-derived strain remained unchanged suggesting that quantitative measures of RV function should not rely solely on conventional indices. As supported by invasive hemodynamic data and NT-proBNP measurements, our results suggest that elevated PASP has a negative effect on RV performance, but this is reversible following surgery. Furthermore, in patients with preoperative PASP >50 mmHg, the global strain improved at the 6-month follow-up, while the conventional indices indicated that there was impaired RV performance. The manner in which the RV contracts is altered postoperatively due to the opening of the pericardium and LV unloading following surgery and speckle tracking-derived strain may therefore be used in addition to conventional echocardiographic measures of RV function. Furthermore, preoperative development of secondary pulmonary hypertension, although reversible following surgery, appears to be harmful to the RV; so addressing severe mitral regurgitation should be given priority, especially since biventricular impairment has been shown to be a powerful predictor of both cardiovascular and overall survival (4).

The reduction of conventional echocardiographic longitudinal indices after cardiac surgery, is a well-known phenomenon, although the correlation to reduced RV performance is still controversial (7, 13, 14, 15). This was confirmed by the present study showing a postoperative decrease in conventional longitudinal indices. Different mechanisms have been proposed to explain the loss in RV performance, such as geometric changes in the RV chamber, intraoperative myocardial ischemia, pericardial disruption, influence of cardiopulmonary bypass, and postoperative adherence of the right ventricle to the thoracic wall (8, 16). Thus, RV assessment using conventional echocardiographic indices may inadequately describe postoperative RV performance. Speckle tracking derived-strain represents myocardial deformation and may therefore reflect actual RV function more adequately compare to conventional echocardiography. Previous studies have demonstrated the clinical potential of speckle tracking-derived strain in evaluation of LV function, but only a few studies have focused on its application in RV performance (17). Ternacle *et al.* suggested that reduced preoperative RV global

strain may be a sensitive marker of RV dysfunction that is correlated to postoperative mortality after cardiac surgery (18). Although the preoperative RV global and septal strain was lower than previously reported reference values following mitral valve repair (10), the postoperative strain data in the current study remained unchanged related to baseline in our population. In contrast, a significant postoperative deterioration of the RV free wall and septal strain in patients undergoing mitral valve surgery has been reported by Maffesanti *et al.* (10), although the radial strain remained unchanged. The unchanged strain following mitral valve repair in the present study may be due to several factors such as a relative improvement in RV function through the reversal of PASP following valve repair, or discrepancies in baselines between populations.

RV performance may be influenced by elevated PASP, which is a common finding in degenerative severe chronic MR (19). Grose *et al.* showed that at least part of the RV impairment in patients with elevated PASP was reversible (20), and elevated PASP is often reduced or normalized after mitral valve repair (21). In our study, 42% of the patients had preoperative PASP >50 mmHg, which is in keeping with previous studies (19, 22). Elevated pulmonary artery pressure is not well tolerated by the right ventricle, because of its thin wall and high compliance (13). Thus, an elevation in PASP often results in structural and functional deterioration (23) and decreased RV ejection fraction (20). In the current study, patients with preoperative PASP >50 mmHg had significantly greater right atrial and ventricular chamber dimensions and also impaired RV free wall and global strain compared to those with preoperative PASP ≤50 mmHg. We could not demonstrate any preoperative differences in the left-side dimensions or function between the groups, as assessed echocardiographically. However, invasively measured mean cardiac output was lower than normal in both groups.

As suggested by Le Tourneau *et al.* (4) and as confirmed by our findings, there appears to be a relationship between elevated PASP and RV dysfunction, even though PASP may not be the main determinant of RV performance following mitral valve repair, due to the potential for reversibility. However, other factors such as LV function, including septal performance, may also influence changes in RV function following mitral valve repair (24). This is supported by the findings of Le Tourneau *et*

al. (4) who have shown that biventricular impairment is a powerful predictor of both cardiovascular and overall survival. In addition, RV dysfunction has been recognized as a predictor for survival in patients with ischemic or dilated cardiomyopathy after mitral valve annuloplasty (25). Taken together, these data suggest that biventricular impairment should be strongly avoided, with early referral to mitral valve surgery.

There is accumulating evidence in favor of the usefulness of natriuretic peptides as clinical markers for RV dysfunction in primary pulmonary hypertension (26). However, little is known about the release of natriuretic peptides and RV dysfunction, and PASP in patients with chronic degenerative MR. The present study showed elevated baseline NT-proBNP levels, which were more pronounced in patients with preoperative RV dysfunction or elevated PASP. Following surgery, the NT-proBNP levels were reduced, although not statistically significantly, in patients with preoperative RV dysfunction or elevated PASP. The release pattern of NT-proBNP, as supported by invasive hemodynamic data, suggests that there is a link between elevated PASP and impaired RV performance.

The present study, although prospective, was limited by the lack of a reference standard for RV functional evaluation, such as magnetic resonance imaging or 3-dimensional echocardiography. A larger patient population with RV strain assessment, including radial speckle tracking-derived strain, might have improved the reliability of the statistical inferences.

Despite the excellent early outcome following mitral valve repair, our results suggest that right ventricular dysfunction is common at 6-month follow-up. Pulmonary hypertension, although reversible following surgery, had a negative effect on right ventricular function. A systematic RV assessment including speckle tracking-derived strain may identify early signs of dysfunction and assist in prioritization of surgical referrals in order to avoid the detrimental effects of biventricular impairment.

Acknowledgments

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Table 1. Pre- and perioperative characteristics (*n*=40)

Preoperative data	
Age, years	66±9
Male	28 (70)
BMI, kg/m^2	26±4
eGFR, mL/min/1.73 m^2	84±20
COPD	1 (3)
NYHA class III─IV	22 (55)
Sinus rhythm	40 (100)
Hypertension	24 (60)
Beta-blocker use	15 (38)
ACE inhibitor use	22 (55)
Diuretic use	23 (58)
Diabetes mellitus type II	2 (5)
Logistic EuroSCORE	4.2±2.7
NT-pro-BNP, ng/L	944±2130
Perioperative data	
Repair rate	37 (93)
Type of repair	
- resection plasty	9 (23)
- chordal transfer or neochordae	31 (78)
- annuloplasty	37 (93)
ECC, min	120±43
Cross clamp, min	92±38
Concomitant CABG	5 (13)

Values are mean±standard deviation or number (%).

BMI=body mass index; eGFR=estimated glomerular filtration rate; COPD=chronic obstructive pulmonary disease; NYHA=New York Heart Association; ACE=angiotensin converting enzyme; EuroSCORE=European System for Cardiac Operative Risk Evaluation; NT-pro-BNP=N-terminal protype-brain natriuretic peptide; ECC=extracorporeal circulation; CABG=coronary artery bypass grafting.

Table 2. Echocardiographic data

	Preoperative		Postoperative		*p*-value
	*n**		*n**		
RAVi, mL/m^2	38	33±12	36	32±10	0.549
RVIT, mm	38	40±6	36	40±7	0.659
RVMCd, mm	38	31±6	35	32±6	0.676
FAC, %	38	36±11	35	38±9	0.231
TAPSE, mm	38	24±5	36	15±3	<0.001
S´, cm/s	38	14±3	36	10±2	<0.001
RIMP	37	0.41±0.21	35	0.33±0.11	0.104
Dp/Dt, mmHg/s	34	481±272	34	451±290	0.841
IVA, cm/s^2	36	2.6±1.1	34	2.1±0.7	0.022
RV dysfunction	38		36		
-normal		26 (65)		14 (39)	
-mild		8 (20)		17 (47)	
-moderate		4 (10)		5 (14)	
-severe		0		0	
TR max PG, mmHg	38	43±13	35	28±9	<0.001
Deceleration time, ms	38	94±22	36	97±17	0.634
VCI, mm	37	17±5	36	13±4	0.001
CVP, mmHg	40	6±3	36	5±2	0.023
PASP, mmHg	38	49±13	35	33±10	<0.001
RV longitudinal strain, %					
-free wall	32	-24±7	25	-21±5	0.080
-septal	32	-18±5	25	-16±4	0.329
-global	32	-18±5	25	-17±4	0.190
LAVi, mL/m^2	40	68±21	36	48±17	<0.001
LAd, mm	38	53±7	36	47±7	0.003
IVSd, mm	38	11±2	36	11±2	0.615
LVEDd, mm	40	63±8	36	54±7	<0.001
LVPWd, mm	38	10±2	35	10±2	0.092
LVESd, mm	40	38±7	36	35±9	0.064
LVEF, %	40	65±10	36	56±10	<0.001

Values are mean±standard deviation or number (%). *Patients with data available.

RAVi=right atrial volume index; RVIT=right ventricular inflow tract; RVMCd=right ventricular mid-cavity dimension; FAC=fractional area change; TAPSE=tricuspid annular plane systolic excursion; S´=tissue Doppler-derived tricuspid lateral annular systolic peak velocity; RIMP=right ventricular index of myocardial performance; IVA=isovolumic acceleration; RV=right ventricular; TR max PG=tricuspid regurgitation maximum pressure gradient; VCI=vena cava inferior; CVP=central venous pressure; PASP=pulmonary artery systolic pressure; RV=right ventricular; LAVi=left atrial volume index; LAd=left atrial dimension; IVSd=intraventricular septal dimension; LVEDd=left ventricular end-diastolic dimension; LVPWd=left ventricular posterior wall dimension; LVESd=left ventricular end-systolic dimension; LVEF=left ventricular ejection fraction.

Table 3. Echocardiographic data for patients with preoperative pulmonary artery systolic pressure of more than 50 mmHg

	Preoperative		Postoperative		*p*-value
	*n**		*n**		
RAVi, mL/m^2	16	39±14	15	32±9	0.110
RVIT, mm	16	43±6	15	41±6	0.123
RVMCd, mm	16	33±7	15	33±6	0.842
FAC, %	16	31±9	15	35±8	0.119
TAPSE, mm	16	23±6	15	15±3	<0.001
S´, cm/s	16	14±4	15	10±2	<0.001
RIMP	16	0.48±0.26	15	0.34±0.14	0.081
Dp/Dt, mmHg/s	16	503±340	14	482±203	0.651
IVA, cm/s^2	15	2.8±1.3	14	2.0±0.8	0.041
RV dysfunction	16		15		
-normal		9 (56)		5 (33)	
-mild		4 (25)		8 (53)	
-moderate		3 (19)		2 (13)	
-severe		0		0	
TR max PG, mmHg	16	55±11	14	35±8	<0.001
Deceleration time, ms	16	81±18	15	90±17	0.183
VCI, mm	16	19±5	15	14±4	0.004
CVP, mmHg	16	7±4	15	5±2	0.101
PASP, mmHg	16	62±10	14	40±10	<0.001
RV longitudinal strain, %					
-free wall	13	-21±6	13	-20±3	0.761
-septal	13	-16±5	13	-16±3	0.960
-global	13	-16±4	13	-17±3	0.534
LAVi, mL/m^2	16	68±16	15	47±6	<0.001
LAd, mm	16	53±7	15	47±7	0.047
IVSd, mm	16	12±2	15	11±1	0.571
LVEDd, mm	16	61±8	15	53±8	<0.001
LVPWd, mm	16	10±1	15	11±2	0.127
LVESd, mm	16	37±7	15	34±10	0.312
LVEF, %	16	65±11	15	57±8	0.003

Abbreviations as in Table 2.

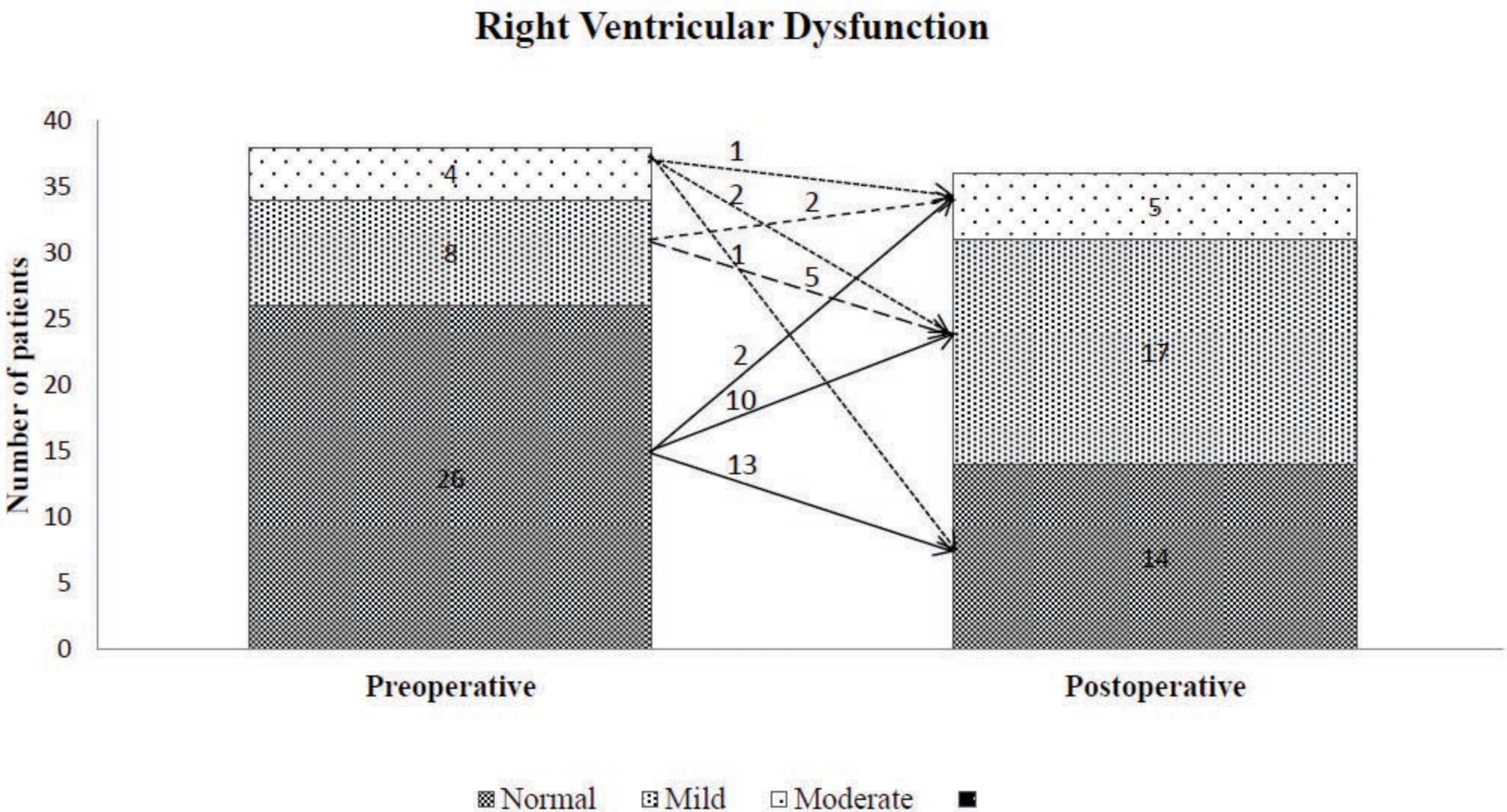
Table 4. Echocardiographic data for patients with preoperative pulmonary artery systolic pressure equal to or less than 50 mmHg

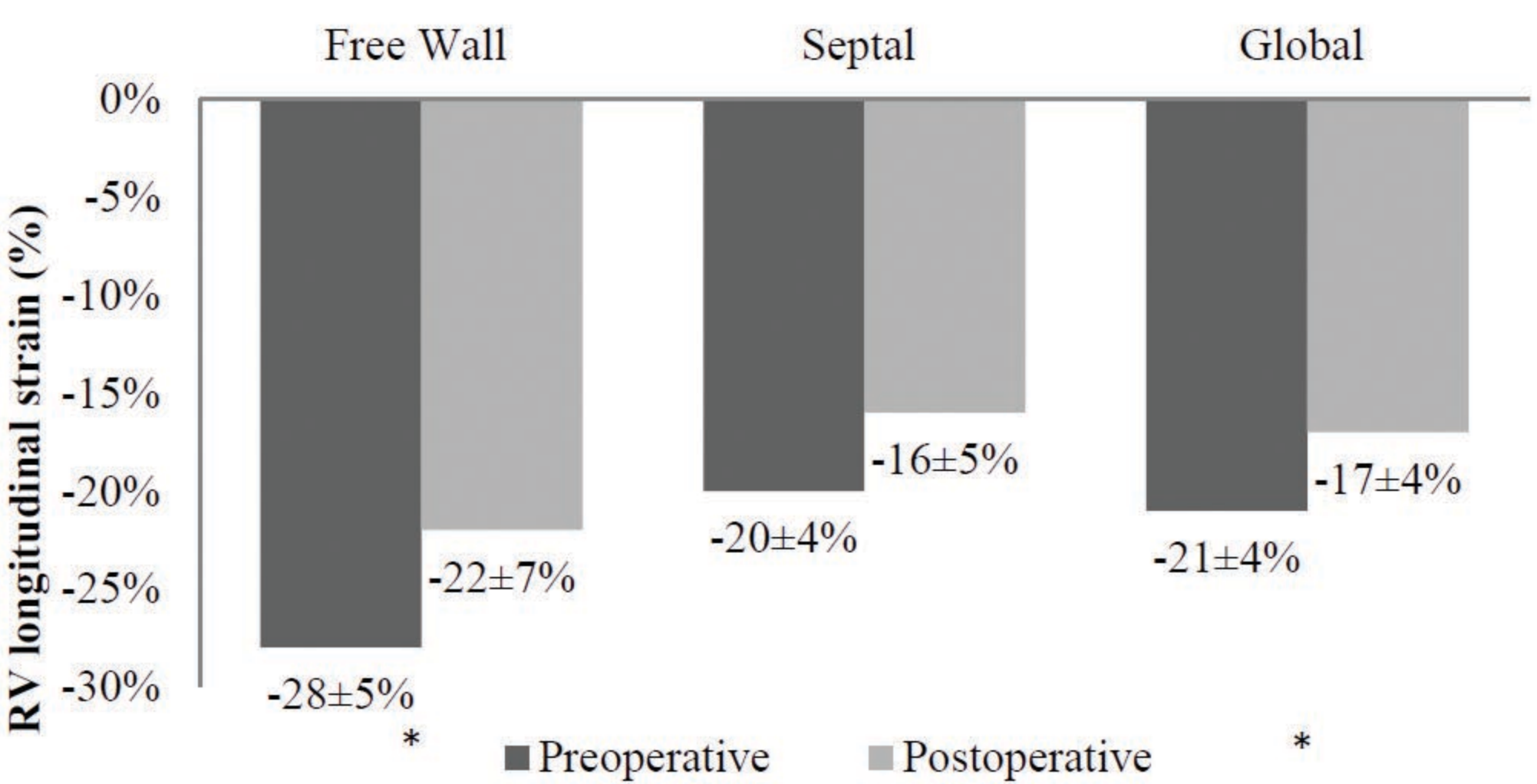
	Preoperative		Postoperative		*p*-value
	*n**		*n**		
RAVi, mL/m^2	21	30±8	20	31±12	0.701
RVIT, mm	21	38±5	20	39±7	0.932
RVMCd, mm	21	30±5	19	31±7	0.503
FAC, %	21	39±11	19	40±10	0.662
TAPSE, mm	21	24±5	20	15±3	<0.001
S´, cm/s	21	14±3	20	10±3	<0.001
RIMP	21	0.36±0.15	19	0.31±0.09	0.321
Dp/Dt, mmHg/s	18	462±203	19	500±424	0.534
IVA, cm/s^2	20	2.4±0.92	19	2.2±0.5	0.258
RV dysfunction	21		20		
-normal		16 (76)		9 (45)	
-mild		4 (19)		8 (40)	
-moderate		1 (5)		3 (15)	
-severe		0		0	
TR max PG, mmHg	22	34±5	20	24±5	<0.001
Deceleration time, ms	21	103±19	20	102±15	0.577
VCI, mm	20	15±4	20	14±4	0.081
CVP, mmHg	22	5±1	20	5±2	0.134
PASP, mmHg	22	39±5	20	28±6	<0.001
RV longitudinal strain, %					
-free wall	17	-27±6	12	-21±7	0.040
-septal	17	-20±6	12	-16±6	0.113
-global	17	-21±6	12	-17±5	0.028
LAVi, mL/m^2	22	71±23	20	49±21	<0.001
LAd, mm	21	52±7	20	48±8	0.048
IVSd, mm	21	11±2	20	11±2	0.067
LVEDd, mm	22	64±8	20	54±7	<0.001
LVPWd, mm	21	10±2	19	10±2	0.387
LVESd, mm	22	39±8	20	36±9	0.170
LVEF, %	22	65±9	20	55±11	<0.001

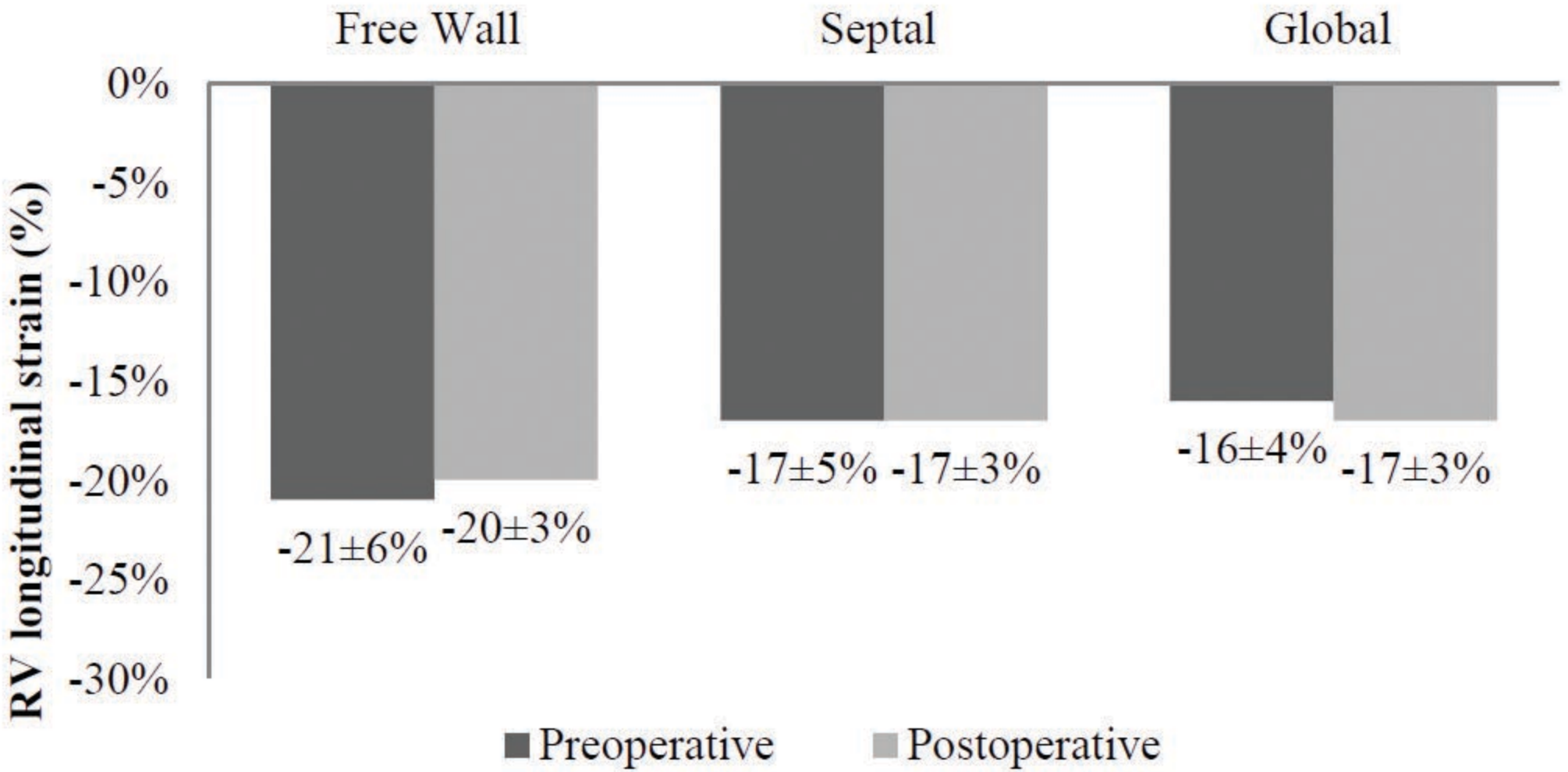
Abbreviations as in Table 2.

Figure legends:

1. Change in right ventricular performance six months after mitral valve repair. Right ventricular dysfunction was assessed as an average of the conventional echocardiographic indices and graded as normal, mild, moderate, or severe.
2. Change in right ventricular longitudinal strain in patients with preoperative pulmonary artery pressure equal to or less than 50 mmHg six months after mitral valve repair (n=10). *p<0.05
3. Change in right ventricular longitudinal strain in patients with preoperative pulmonary artery pressure of more than 50 mmHg six months after mitral valve repair (n=11).


Right Ventricular Dysfunction
Number of patients
40
35
30
25
20
15
10
5
0
4
8
26
1
2
2
1
5
2
10
13
5
17
14
Preoperative
Postoperative
Normal
Mild
Moderate


Free Wall
Septal
Global
0%
-5%
-10%
-15%
-20%
-25%
-30%
RV longitudinal strain (%)
-28±5%
-22±7%
-20±4%
-16±5%
-21±4%
-17±4%
*
*
Preoperative
Postoperative


Free Wall
Septal
Global
0%
-5%
-10%
-15%
-20%
-25%
-30%
RV longitudinal strain (%)
-21±6%
-20±3%
-17±5%
-17±3%
-16±4%
-17±3%
Preoperative
Postoperative