

Changes in right ventricular-pulmonary artery coupling after transcatheter edge-to-edge repair in primary and atrial mitral regurgitation

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ARTICLE INFO

Keywords:

Transcatheter edge-to-edge mitral valve repair
Mitral regurgitation
Right ventricular-pulmonary artery coupling
TAPSE/PASP
Pulmonary hypertension

ABSTRACT

Background: Right ventricular-pulmonary artery (RV-PA) coupling is an established marker of prognosis in mitral regurgitation (MR), but data on its change after transcatheter edge-to-edge mitral valve repair (M-TEER) in primary or atrial MR are limited.

Methods: RV-PA coupling was evaluated in 190 patients undergoing M-TEER for primary or atrial MR before and one month after the procedure. RV-PA coupling was assessed using the ratio of tricuspid annular plane systolic excursion to pulmonary artery systolic pressure (TAPSE/PASP). The primary endpoint was the composite of death or hospitalization for heart failure during follow-up.

Results: One month after M-TEER, RV-PA coupling improved in 78 patients (41.1%), driven by both improved RV systolic function (Δ TAPSE 2.2 ± 4.4 mm) and lower PASP (Δ PASP -14.7 ± 11.9 mmHg). Improvement in TAPSE/PASP was associated with less residual MR and better functional status after the procedure, but not with a lower risk of death or heart failure hospitalization during follow-up. In contrast, preserved RV-PA coupling at one month after M-TEER, defined as TAPSE/PASP >0.33 mm/mmHg, identified a subgroup at lower risk of the primary endpoint (hazard ratio 0.46 [95% confidence interval 0.26–0.81], $p = 0.008$).

Conclusion: M-TEER is associated with improvement in RV-PA coupling in patients with primary or atrial MR. Persistent RV-PA uncoupling identifies a high-risk group for adverse events after the procedure. Future studies in dedicated aetiology-specific cohorts are required to establish the role of serial RV-PA coupling assessment in post-procedural risk stratification after M-TEER.

1. Introduction

Right ventricular-pulmonary artery (RV-PA) coupling defines the relationship between RV contractility and afterload, reflecting the RV's ability to respond to the resistive demands of the pulmonary circulation. [1–3] Although the gold standard evaluation of RV-PA coupling requires technically demanding pressure-volume loop analysis, the ratio of tricuspid annular plane systolic excursion (TAPSE) to pulmonary artery

systolic pressure (PASP) has become a widely accepted non-invasive echocardiographic surrogate that correlates with invasive measurements. [4] Reduced TAPSE/PASP signifies RV-PA uncoupling, wherein sustained hemodynamic stress surpasses the RV's compensatory capacity, resulting in diminished stroke volume and progression toward right heart failure. [2] Accordingly, RV-PA coupling is an important prognostic marker across a range of cardiovascular conditions, including heart failure with both reduced and preserved ejection fraction,

Abbreviations: CI, Confidence interval; HR, Hazard ratio; MR, Mitral regurgitation; M-TEER, Transcatheter edge-to-edge mitral valve repair; OR, Odds ratio; PASP, Pulmonary artery systolic pressure; RV, Right ventricle / right ventricular; RV-PA, Right ventricular-pulmonary artery; TAPSE, Tricuspid annular plane systolic excursion; TR, Tricuspid regurgitation.

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¹ This author takes responsibility for all aspects of the reliability and freedom from bias of the data presented and their discussed interpretation.

<https://doi.org/10.1016/j.ijcard.2026.134953>

Received 22 June 2026; Received in revised form 10 September 2026; Accepted 29 September 2026

Available online 1 October 2026

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Open access funding enabled and organized by Projekt DEAL.

pulmonary hypertension, and valvular heart disease. [1,5–7] Notably, RV-PA uncoupling is a significant predictor of increased mortality and adverse events among patients undergoing transcatheter edge-to-edge mitral valve repair (M-TEER) for ventricular mitral regurgitation (MR), and may be integrated into pre-procedural risk stratification. [8,9] Furthermore, research indicates that RV-PA coupling parameters improve in most patients following M-TEER for ventricular MR, and this improvement has been associated with better outcomes. [10] However, in ventricular MR, impaired RV-PA coupling may reflect not only the consequences of MR itself but also the underlying burden of left ventricular systolic dysfunction and chronic heart failure. Whether similar patterns of RV-PA coupling, post-procedural recovery, and prognostic relevance apply to patients with primary or atrial MR, in whom left ventricular systolic function is typically preserved, remains less well established. Dedicated data in atrial MR are particularly limited. Accordingly, this study aimed to evaluate changes in RV-PA coupling after M-TEER in patients with primary or atrial MR and to determine their association with clinical outcome.

2. Methods

2.1. Study population

From August 2017 to July 2025, patients were consecutively enrolled in a prospective observational study conducted at the University Heart Center Regensburg. The main inclusion criterion was symptomatic moderate-to-severe or severe mitral regurgitation of primary or atrial aetiology treated with M-TEER. MR aetiology was classified by an integrative assessment based on transthoracic and transoesophageal echocardiography. Primary MR was defined by structural abnormalities of the mitral valve apparatus, including leaflet prolapse, flail leaflet, chordal rupture, or degenerative leaflet disease. Atrial MR was defined as significant MR occurring in the setting of predominant left atrial and mitral annular remodelling, with impaired leaflet coaptation despite structurally normal mitral leaflets and chordae and the absence of left ventricular dilatation or systolic dysfunction. The decision to proceed with M-TEER was made on an individual basis by an interdisciplinary Heart Team. M-TEER procedures were performed by experienced interventionalists utilizing either the MitraClip (Abbott Vascular, Menlo Park, USA) or PASCAL system (Edwards Lifesciences, Irvine, USA) under general anaesthesia with guidance from fluoroscopy and three-dimensional transoesophageal echocardiography. Patients with intra-procedural failure to implant a device or incomplete echocardiographic data at baseline or follow-up were excluded from the analysis. The study flow chart is presented in Fig. 1. Informed consent was obtained from all participants. The study was approved by the local ethics committee (approval Z-2017-0862-10).

2.2. Clinical and echocardiographic assessment

Participants underwent a comprehensive clinical and echocardiographic assessment before and one month after M-TEER. MR grading was based on colour and continuous wave Doppler examination in accordance with current guidelines. [11,12] Regurgitation grade was scored from I to IV (I: mild, II: mild-to-moderate, III: moderate-to-severe, IV: severe). Measurement of RV structure and function was performed in accordance with current recommendations. [13] TAPSE was measured using M-Mode in a RV-focused apical 4-chamber view optimized to maintain parallel alignment with the tricuspid annular motion. Right atrial pressure was estimated by measuring inferior vena cava size and collapsibility during normal breathing. PASP was calculated from peak tricuspid regurgitation (TR) velocity using the simplified Bernoulli equation with addition of estimated right atrial pressure. Patients were categorized according to the change in TAPSE/PASP from baseline to follow-up into two groups: responders, those exhibiting improved TAPSE/PASP values; and non-responders, those with unchanged or

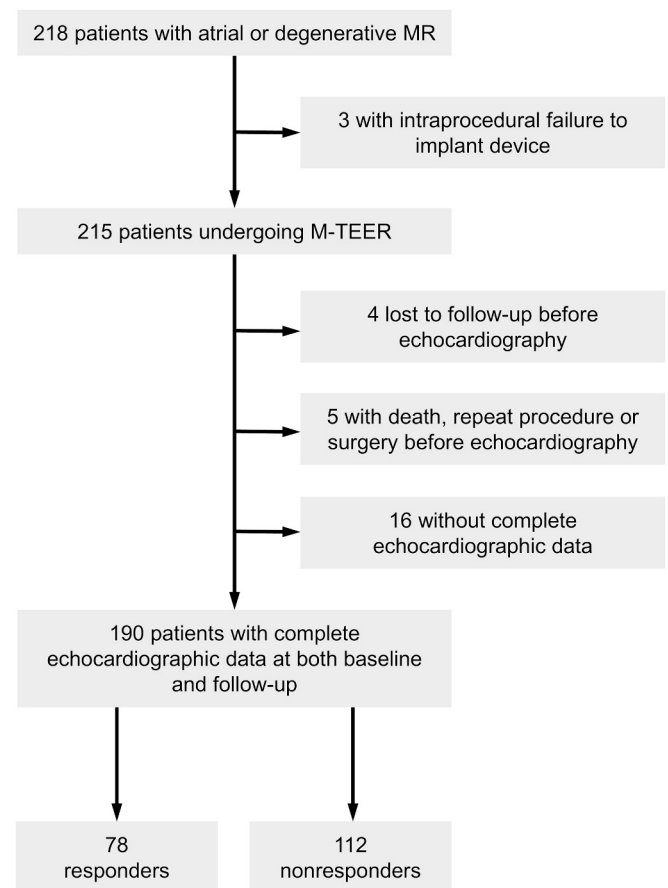


Fig. 1. Study flow chart. MR, mitral regurgitation; M-TEER, transcatheter edge-to-edge mitral valve repair.

deteriorated TAPSE/PASP measures. Reduced RV systolic function was defined as TAPSE <17 mm. RV-PA uncoupling was defined as TAPSE/PASP ≤ 0.33 mm/mmHg. This threshold was selected a priori based on a large meta-analysis of patients undergoing MR intervention, in which TAPSE/PASP ≤ 0.33 mm/mmHg was identified as an optimal cut-off for adverse clinical outcomes. [7] Following the procedure, patients were observed for up to 60 months to assess the incidence of a composite endpoint consisting of death or hospitalization for heart failure.

2.3. Statistical analysis

Continuous variables are reported as mean $\pm$ standard deviation when normally distributed and as median with interquartile range [first quartile-third quartile] when skewed. Categorical variables are presented in numbers and percentages. Differences in unpaired data were assessed using Student's *t*-test for normally distributed data, Mann-Whitney *U* test for ordinal or skewed data, Pearson's chi-squared test for nominal data, and Fisher's exact test for dichotomous data. Differences in paired samples were tested using paired *t*-tests for normally distributed data, Wilcoxon signed-rank tests for skewed or ordinal data, and McNemar's test for dichotomous data. To account for regression to the mean, changes in TAPSE/PASP were adjusted for baseline values using an analysis of covariance approach implemented as a linear regression model. Unstandardized residuals were used to define adjusted changes. Patients exhibiting positive adjusted changes in TAPSE/PASP during follow-up were classified as responders.

To identify predictors of death or hospitalization for heart failure, univariable and multivariable Cox regression models were calculated. Multivariable models were adjusted for predefined clinically relevant covariates selected on the basis of clinical plausibility, including age,

sex, renal function, MR aetiology, TR severity as a marker of advanced right-sided disease, and residual MR grade at discharge to account for procedural success. To evaluate whether the prognostic relevance of RV-PA uncoupling or TAPSE/PASP responder status differed by MR aetiology, Cox regression analyses were repeated separately in patients with primary and atrial MR, and formal interaction terms between MR aetiology and the respective RV-PA coupling variable were tested. Kaplan-Meier analysis was performed to compare the incidence of death or hospitalization for heart failure between groups. The median follow-up period was determined through reverse Kaplan-Meier analysis. A binary logistic regression analysis was conducted to determine predictors of improvement in RV-PA coupling and of preserved RV-PA coupling at follow-up. A two-sided *p*-value of <0.05 was considered statistically significant. All statistical analyses were performed using SPSS Statistics 31 (IBM, Armonk, USA).

3. Results

3.1. Baseline characteristics and procedural outcome

A total of 190 patients were included in the analysis. Baseline characteristics and procedural outcomes are presented in Table 1. The mean age was 78.9 ± 6.3 years. The majority of participants exhibited MR grade IV (73.2%) and significantly decreased functional capacity (66.3% classified as NYHA III or higher). Primary MR was present in 109 patients (57.4%), while atrial MR was observed in 81 patients (42.6%). Concomitant severe TR was identified in 36.3% of the cohort. Reduced right ventricular systolic function, defined by TAPSE <17 mm, was noted in 46 patients (24.2%). Impaired RV-PA coupling, assessed using a TAPSE/PASP ratio ≤ 0.33 mm/mmHg, affected 70 individuals (36.8%). At discharge, 95.8% of patients achieved a reduction of MR to grade I or II.

3.2. Change in RV-PA coupling after M-TEER

At one month after M-TEER, RV-PA coupling improved in 78 (41.1%) patients, who were classified as responders. Clinical and echocardiographic outcomes are presented in Table 2. The responder group exhibited a mean increase in TAPSE/PASP of 0.23 ± 0.14 mm/mmHg, attributable to enhanced RV systolic function (Δ TAPSE 2.2 ± 4.4 mm) and a reduction in PASP (Δ PASP -14.7 ± 11.9 mmHg). None of the responders experienced RV-PA uncoupling at follow-up, and only two (2.6%) demonstrated reduced RV function. Conversely, in the non-responder cohort, 43 patients (38.4%) showed RV-PA uncoupling one month after M-TEER, while 29 (25.9%) had RV systolic dysfunction. Responders also displayed smaller RV diameter, lower left atrial volume, less severe TR, and a trend toward less residual MR at follow-up. No significant differences were observed regarding left ventricular geometry or function. Regarding clinical outcome, responders had better functional status at follow-up, reflected by a lower NYHA class and a longer six-minute walk distance.

3.3. Predictors of RV-PA coupling improvement after M-TEER

Table 1 presents baseline characteristics stratified by responder status one month following M-TEER. Responders were predominantly male, exhibited lower baseline left atrial volume, and demonstrated less severe TR at baseline. Notably, there were no observed differences between responders and non-responders in baseline RV systolic function, RV size, PASP, or RV-PA coupling. Additionally, the groups did not differ in functional capacity, MR aetiology, or MR severity prior to M-TEER. Importantly, responders had less residual MR at discharge, indicating more favourable procedural results. Accordingly, higher residual MR grade was associated with a reduced likelihood of TAPSE/PASP improvement in univariable analysis (odds ratio (OR) per grade 0.57 [95% confidence interval (CI) 0.33–0.98]). Multivariable analysis

Table 1

Baseline characteristics and procedural details of the study population, stratified by improvement of RV-PA coupling following M-TEER.

	All (n = 190)	Responder (n = 78)	Non-responder (n = 112)	<i>p</i> -value
Age, years	78.9 $\pm$ 6.3	79.6 $\pm$ 6.5	78.5 $\pm$ 6.2	0.230
Female sex	86 (45.3)	27 (34.6)	59 (52.7)	0.018
BMI, kg/m ²	25.9 $\pm$ 5.3	25.8 $\pm$ 4.3	25.9 $\pm$ 5.9	0.881
Coronary artery disease	102 (53.7)	47 (60.3)	55 (49.1)	0.141
Atrial fibrillation	135 (71.1)	49 (62.8)	86 (76.8)	0.051
Diabetes mellitus	34 (17.9)	10 (12.8)	24 (21.4)	0.178
GFR, ml/min	51.4 $\pm$ 20.1	52.0 $\pm$ 18.5	50.9 $\pm$ 21.2	0.728
NTproBNP, pg/ml	1799	1525	1999	0.145
	[819–3194]	[683–2997]	[892–3390]	
NYHA functional class				0.639
I	0	0	0	
II	64 (33.7)	29 (37.2)	35 (31.3)	
III	115 (60.5)	43 (55.1)	72 (64.3)	
IV	11 (5.8)	6 (7.7)	5 (4.5)	
Six-minute walk distance, m	286 $\pm$ 105	293 $\pm$ 110	281 $\pm$ 102	0.537
MR aetiology				0.372
Primary	109 (57.4)	48 (61.5)	61 (54.5)	
Atrial	81 (42.6)	30 (38.5)	51 (45.5)	
MR grade				0.309
III	51 (26.8)	24 (30.8)	27 (24.1)	
IV	139 (73.2)	54 (69.2)	85 (75.9)	
LVEF, %	57.1 $\pm$ 7.5	56.3 $\pm$ 8.8	57.7 $\pm$ 6.4	0.185
LV end diastolic diameter, mm	52.0 $\pm$ 7.1	52.3 $\pm$ 6.2	51.8 $\pm$ 7.8	0.625
LA volume index, ml/m ²	82.0 $\pm$ 38.8	72.5 $\pm$ 28.4	88.2 $\pm$ 43.2	0.009
E/e'	14.1 $\pm$ 5.0	14.2 $\pm$ 4.4	13.9 $\pm$ 5.3	0.706
RV basal diameter, mm	39.1 $\pm$ 6.8	38.2 $\pm$ 6.3	39.8 $\pm$ 7.1	0.125
TAPSE, mm	19.6 $\pm$ 4.2	19.8 $\pm$ 4.6	19.4 $\pm$ 3.8	0.456
TAPSE <17 mm	46 (24.2)	20 (25.6)	26 (23.2)	0.733
PASP, mmHg	52.4 $\pm$ 15.5	50.6 $\pm$ 13.8	53.7 $\pm$ 16.5	0.172
TAPSE/PASP	0.42 $\pm$ 0.18	0.42 $\pm$ 0.15	0.41 $\pm$ 0.19	0.628
mm/mmHg				
TAPSE/PASP ≤ 0.33 mm/mmHg	70 (36.8)	24 (30.8)	46 (41.1)	0.170
TR grade				0.002
I	61 (32.1)	36 (46.2)	25 (22.3)	
II	60 (31.6)	20 (25.6)	40 (35.7)	
$\geq$ III	69 (36.3)	22 (28.2)	47 (42.0)	
Number of implanted devices				0.957
1	105 (55.3)	43 (55.1)	62 (55.4)	
2	81 (42.6)	34 (43.6)	47 (42.0)	
3	4 (2.1)	1 (1.3)	3 (2.7)	
Device type				1.000
MitraClip	108 (56.8)	44 (56.4)	64 (57.1)	
PASCAL	82 (43.2)	34 (43.6)	48 (42.9)	
Residual MR grade at discharge				0.037
I	120 (63.2)	56 (71.8)	64 (57.1)	
II	62 (32.6)	20 (25.6)	42 (37.5)	
III	8 (4.2)	2 (2.6)	6 (5.4)	
IV	0	0	0	

Variables are expressed as n (%), mean $\pm$ standard deviation, or median [interquartile range], as appropriate.

BMI, body mass index; GFR, glomerular filtration rate; LA, left atrial; LV, left ventricular; LVEF, left ventricular ejection fraction; MR, mitral regurgitation; M-TEER, transcatheter edge-to-edge mitral valve repair. NYHA, New York Heart Association; PASP, pulmonary artery systolic pressure; RV, right ventricular; RV-PA, right ventricular-pulmonary artery; TAPSE, tricuspid annular plane systolic excursion; TR, tricuspid regurgitation.

Table 2

Clinical and echocardiographic outcomes one month after M-TEER.

	Responder (n = 78)	Non-responder (n = 112)	p-value
NYHA functional class			0.035
I	23 (29.5)	24 (21.4)	
II	41 (52.6)	52 (46.4)	
III	14 (17.9)	36 (32.1)	
IV	0	0	
Six-minute walk distance, m	332 ± 117	290 ± 111	0.019
NTproBNP, pg/ml	1568 [686–3208]	2200 [1038–4036]	0.073
MR grade			0.071
I	51 (65.4)	58 (51.8)	
II	23 (29.5)	46 (41.1)	
III	2 (2.6)	7 (6.3)	
IV	2 (2.6)	1 (0.9)	
MV pressure gradient, mmHg	3.6 ± 1.5	4.2 ± 1.4	0.010
LVEF, %	54.4 ± 8.0	56.3 ± 8.3	0.109
LV end diastolic diameter, mm	50.3 ± 6.7	50.1 ± 7.1	0.896
LA volume index, ml/m ²	63.8 ± 22.9	73.4 ± 36.6	0.043
RV basal diameter, mm	38.0 ± 5.9	40.3 ± 8.2	0.033
TAPSE, mm	22.0 ± 3.8	18.7 ± 3.6	<0.001
ΔTAPSE, mm	2.2 ± 4.4	−0.6 ± 4.1	<0.001
TAPSE <17 mm	2 (2.6)	29 (25.9)	<0.001
PASP, mmHg	35.8 ± 8.7	52.3 ± 15.5	<0.001
ΔPASP, mmHg	−14.7 ± 11.9	−1.4 ± 14.6	<0.001
TAPSE/PASP, mm/mmHg	0.65 ± 0.19	0.38 ± 0.12	<0.001
ΔTAPSE/PASP, mm/mmHg	0.23 ± 0.14	−0.03 ± 0.10	<0.001
TAPSE/PASP ≤0.33 mm/mmHg	0	43 (38.4)	<0.001
TR grade			<0.001
I	49 (62.8)	22 (19.6)	
II	20 (25.6)	45 (40.2)	
≥III	9 (11.5)	45 (40.2)	

Variables are expressed as n (%), mean ± standard deviation, or median [interquartile range], as appropriate.

LA, left atrial; LV, left ventricular; LVEF, left ventricular ejection fraction; MR, mitral regurgitation; MV, mitral valve; M-TEER, transcatheter edge-to-edge mitral valve repair; NYHA, New York Heart Association; PASP, pulmonary artery systolic pressure; RV, right ventricular; TAPSE, tricuspid annular plane systolic excursion; TR, tricuspid regurgitation.

identified female sex (OR 0.50 [95% CI 0.25–0.99], $p = 0.047$) and baseline TR grade (OR per grade 0.62 [95% CI 0.39–0.97], $p = 0.037$) as independent negative predictors of improvement in RV-PA coupling (Supplementary Table 1).

We further examined predictors of preserved RV-PA coupling one month after M-TEER. Patients with follow-up TAPSE/PASP >0.33 mm/mmHg less frequently had atrial fibrillation or diabetes mellitus and showed lower baseline NTproBNP levels and NYHA class, indicating a lower comorbidity burden and better functional status. Lower baseline TR severity was also associated with preserved RV-PA coupling after M-TEER (Supplementary Table 2). However, in multivariable analysis, higher baseline TAPSE/PASP was the only independent predictor of preserved RV-PA coupling at one month (OR per 0.1 mm/mmHg 2.93 [95% CI 1.76–4.89], $p < 0.001$; Supplementary Table 3).

3.4. Impact of RV-PA coupling on long-term outcome after M-TEER

To evaluate long-term outcomes following M-TEER, patients were monitored for the occurrence of a composite endpoint comprising death or hospitalization due to heart failure. The median follow-up duration was 24 months, during which the combined endpoint occurred in 51 (26.8%) participants. The results of the Cox regression analyses are shown in Table 3. A higher TAPSE/PASP ratio at both baseline and one month post-M-TEER was associated with a lower risk of death or heart failure hospitalization. In multivariable analysis adjusting for clinically relevant confounders including age, sex, renal function, MR aetiology, residual MR grade at discharge, and TR severity, TAPSE/PASP remained an independent predictor of the composite endpoint (baseline hazard

Table 3

Prognostic significance of right ventricular function parameters for death or hospitalization for heart failure after M-TEER.

	Univariable		Multivariable [‡]	
	HR (95% CI)	p-value	HR (95% CI)	p-value
TAPSE/PASP at baseline, per 0.1 mm/mmHg	0.71 (0.57–0.88)	0.002	0.72 (0.58–0.90)	0.005
TAPSE/PASP at 1 mon, per 0.1 mm/mmHg	0.77 (0.64–0.92)	0.004	0.82 (0.67–0.99)	0.037
Adjusted ΔTAPSE/PASP, per 0.1 mm/mmHg	0.88 (0.72–1.07)	0.210	0.96 (0.78–1.18)	0.678
TAPSE at baseline, per mm	0.93 (0.86–1.00)	0.042	0.94 (0.87–1.02)	0.115
TAPSE at 1 mon, per mm	0.97 (0.90–1.04)	0.347	0.99 (0.92–1.05)	0.665
PASP at baseline, per 10 mmHg	1.34 (1.13–1.60)	0.001	1.29 (1.07–1.55)	0.007
PASP at 1 mon, per 10 mmHg	1.27 (1.08–1.50)	0.003	1.19 (1.00–1.42)	0.053

CI, confidence interval; HR, hazard ratio; MR, mitral regurgitation; M-TEER, transcatheter edge-to-edge mitral valve repair; PASP, pulmonary artery systolic pressure; TAPSE, tricuspid annular plane systolic excursion.

[‡] Variables included in the multivariable models: age, female gender, atrial MR aetiology, severe TR, GFR <60 ml/min/1.73m², residual MR grade at discharge.

ratio (HR) per 0.1 mm/mmHg 0.72 [95% CI 0.58–0.90], $p = 0.005$; follow-up HR 0.82 [95% CI 0.67–0.99], $p = 0.037$). In contrast, change in TAPSE/PASP from baseline to one month was not associated with a lower risk of death or heart failure hospitalization. Additionally, elevated PASP values at baseline independently predicted adverse clinical outcomes. RV systolic function, assessed by TAPSE alone, did not provide independent prognostic value.

Additionally, patients were dichotomized according to TAPSE/PASP responder status at one month and according to preserved RV-PA coupling, defined as TAPSE/PASP >0.33 mm/mmHg (Fig. 2). Although responders showed a numerically lower rate of death or hospitalization for heart failure than non-responders, this association did not reach statistical significance (HR 0.62 [95% CI 0.34–1.12], $p = 0.111$). In contrast, patients with preserved RV-PA coupling at one month had a lower risk of the primary endpoint than those with persistent RV-PA uncoupling (HR 0.46 [95% CI 0.26–0.81], $p = 0.008$). Analysis of the individual endpoint components suggested that this association was driven predominantly by mortality (HR 0.40 [95% CI 0.18–0.89], $p = 0.024$), whereas the association with heart failure hospitalization was weaker (HR 0.52 [95% CI 0.25–1.07], $p = 0.076$). Similarly, preserved RV-PA coupling at baseline was associated with a lower incidence of the primary endpoint (HR 0.53 [95% CI 0.31–0.92], $p = 0.025$).

3.5. Aetiology-specific exploratory subanalysis

Given the distinct pathophysiology of primary and atrial MR, exploratory analyses were performed separately by MR aetiology to evaluate the findings of the pooled analysis. Baseline characteristics and one-month clinical and echocardiographic outcomes stratified by MR aetiology are shown in Supplementary Tables 2 to 5. TAPSE/PASP responder rates were comparable between subgroups (44.0% in primary MR vs. 37.0% in atrial MR, $p = 0.372$), and the magnitude of TAPSE/PASP improvement was similar across aetiologies. In both subgroups, improvement in TAPSE/PASP at one month remained strongly associated with less severe TR. Among patients with primary MR, responders had a significantly lower mitral valve pressure gradient, whereas this difference was not observed in patients with atrial MR. By contrast, the difference in functional capacity between responders and non-responders was not evident in the primary MR subgroup.

Death or heart failure hospitalization according to TAPSE/PASP response and post-procedural RV-PA coupling

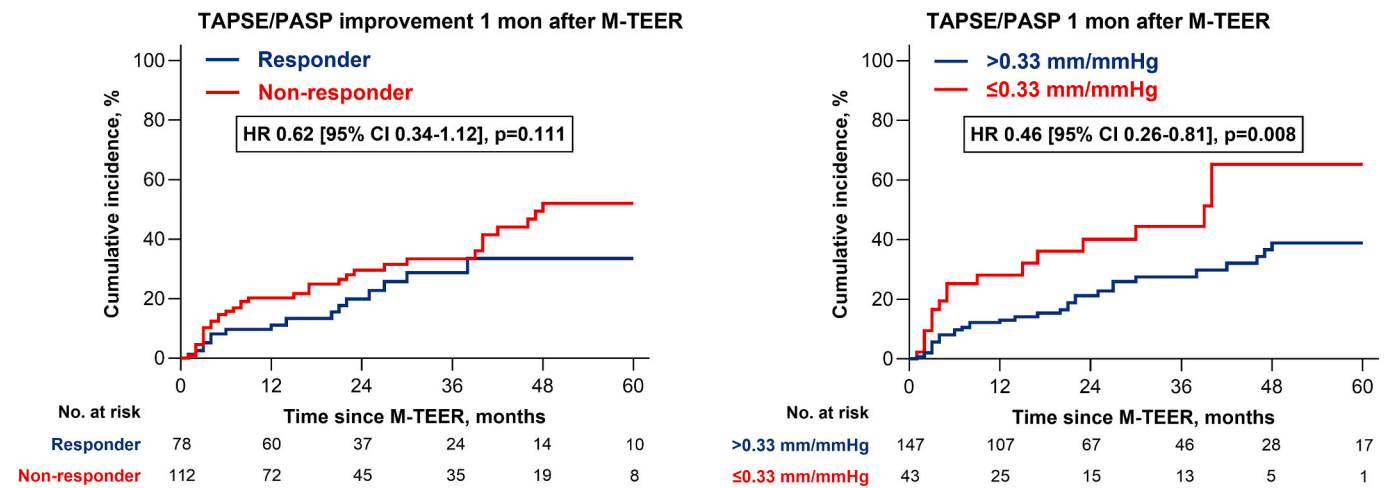


Fig. 2. Death or hospitalization for heart failure after M-TEER according to post-procedural RV-PA uncoupling. Improvement in TAPSE/PASP after M-TEER was not associated with improved long-term outcome. However, preserved RV-PA coupling at follow-up (TAPSE/PASP >0.33 mm/mmHg) was associated with a lower risk of the primary endpoint. CI, confidence interval; HR, hazard ratio; M-TEER, transcatheter edge-to-edge mitral valve repair; PASP, pulmonary artery systolic pressure; RV-PA, right ventricular-pulmonary artery; TAPSE, tricuspid annular plane systolic excursion.

Cox regression analyses were repeated within both aetiological subgroups to assess the prognostic relevance of RV-PA uncoupling and TAPSE/PASP improvement (Table 4). In patients with primary MR, RV-PA uncoupling at baseline and at one month after M-TEER was associated with higher rates of death or hospitalization for heart failure. The association was directionally consistent in patients with atrial MR but did not reach statistical significance. Interaction testing showed no significant interaction between MR aetiology and RV-PA uncoupling, providing no evidence that the prognostic association of RV-PA uncoupling differs by MR aetiology. However, given the limited sample size and event numbers within each subgroup, these findings should be interpreted as exploratory and require validation in larger aetiology-specific cohorts. Consistent with the pooled analysis, TAPSE/PASP responder status was not associated with improved prognosis in either subgroup.

4. Discussion

This study systematically evaluated early changes in RV-PA coupling

Table 4

Prognostic significance of RV-PA coupling for death or hospitalization for heart failure after M-TEER in subgroups stratified by MR aetiology.

	HR (95% CI)	p-value	p-value for interaction
TAPSE/PASP responder			0.664
Primary MR	0.74 (0.32–1.70)	0.473	
Atrial MR	0.56 (0.24–1.32)	0.183	
TAPSE/PASP > 0.33 mm/mmHg at baseline			0.170
Primary MR	0.39 (0.17–0.90)	0.027	
Atrial MR	0.79 (0.37–1.66)	0.528	
TAPSE/PASP > 0.33 mm/mmHg one month after M-TEER			0.663
Primary MR	0.40 (0.16–0.97)	0.043	
Atrial MR	0.54 (0.25–1.16)	0.113	

CI, confidence interval; HR, hazard ratio; MR, mitral regurgitation; M-TEER, transcatheter edge-to-edge mitral valve repair; PASP, pulmonary artery systolic pressure; RV-PA, right ventricular-to-pulmonary artery; TAPSE, tricuspid annular plane systolic excursion.

after M-TEER in a focused cohort of patients with primary or atrial MR. The principal findings are as follows:

- RV-PA uncoupling was prevalent, impacting approximately one third of this patient cohort.
- Short-term improvements in RV-PA coupling were observed in 41.1% of patients and correlated with enhanced functional status post-M-TEER.
- Male sex and less severe TR were baseline predictors for improvement in RV-PA coupling after M-TEER.
- Both baseline and post-M-TEER RV-PA uncoupling were predictive of long-term clinical outcomes.

4.1. Prognostic significance of RV-PA uncoupling in M-TEER

Robust evidence indicates that RV-PA uncoupling prior to M-TEER is linked to a poorer prognosis in patients with ventricular MR. [14] In the pivotal COAPT trial subanalysis, advanced RV-PA uncoupling, characterized by an RV free wall longitudinal strain/PASP ≤0.5%/mmHg, was observed in 70% of participants and independently predicted mortality or heart failure hospitalization at 24 months. This association was significant among patients receiving M-TEER as well as those undergoing medical therapy alone. [8] Similar findings were reported from a large M-TEER registry study evaluating TAPSE/PASP in 707 patients with either ventricular or primary MR. [15] Further studies validated the prognostic value of RV-PA coupling within cohorts exclusively comprising patients with primary MR; specifically, individuals with TAPSE/PASP <0.307 mm/mmHg exhibited more than a two-fold increase in mortality following M-TEER during a two-year follow-up. [16,17]

In a recent meta-analysis, TAPSE/PASP ≤0.33 mm/mmHg was identified as the optimal threshold for assessing elevated risk of adverse outcomes post-M-TEER. [7] Applying this criterion, we were able to confirm these results in our cohort consisting of patients with primary or atrial MR: RV-PA uncoupling at baseline was associated with an almost two-fold increase in death or hospitalization for heart failure after M-TEER, predominantly driven by an increase in mortality. RV-PA uncoupling was observed in approximately one third of our cohort,

consistent with findings from other studies involving patients with ventricular or primary MR. [8,17,18]

While robust evidence supports the prognostic value of RV-PA coupling in primary and ventricular MR, data in atrial MR remain limited. Thus, the inclusion of atrial MR is a relevant aspect of the present analysis. Prior studies of RV-PA coupling in M-TEER have predominantly focused on ventricular MR or have included heterogeneous cohorts combining ventricular and primary MR. In ventricular MR, impaired RV-PA coupling may reflect not only pulmonary vascular and RV consequences of MR, but also the broader substrate of systolic left ventricular dysfunction and chronic heart failure. In atrial MR, by contrast, MR arises from atrial and annular remodelling in the presence of preserved left ventricular systolic function. Therefore, the prognostic and dynamic behaviour of TAPSE/PASP after M-TEER may not be directly extrapolated from ventricular MR cohorts to patients with atrial MR.

In our aetiology-specific exploratory analysis, RV-PA uncoupling was associated with adverse outcome in patients with primary MR, whereas the association was directionally similar but did not reach statistical significance in atrial MR. Importantly, interaction testing provided no evidence that the prognostic association of RV-PA uncoupling differs by MR aetiology. These findings are consistent with a clinically relevant role of RV-PA coupling in both primary and atrial MR; however, the limited sample size and event numbers in the atrial MR subgroup preclude firm conclusions. Given the growing role of M-TEER in atrial MR, [19] larger dedicated cohorts are needed to define the prognostic utility of RV-PA coupling in this population.

4.2. Improvement in RV-PA coupling after M-TEER

Evidence on the dynamics of RV-PA coupling after M-TEER is still evolving and has been derived predominantly from cohorts with ventricular MR. In the multicentre study by Adamo et al., two thirds of 501 patients with ventricular MR demonstrated an improvement in TAPSE/PASP at early follow-up after M-TEER (median 89 days), whereas one third did not; notably, non-responders exhibited not only persistently elevated PASP but also a decline in TAPSE, resulting in further deterioration of the coupling ratio. [10] Similar findings were reported by Shechter et al. in a mixed cohort of 429 patients with primary or ventricular MR, in which 65% exhibited improved RV-PA coupling one month after M-TEER. [15] Our results extend these observations to a population with primary and atrial MR, in whom left ventricular systolic function is preserved. In our cohort, 41.1% of patients met the responder definition at one month, with improvement in RV-PA coupling driven by both a meaningful reduction in PASP and an increase in TAPSE. Importantly, responders were characterized by more favourable procedural outcome in terms of residual MR. This association supports the concept that effective MR reduction promotes pulmonary unloading and facilitates early recovery of RV-pulmonary vascular interaction. Long-term data on RV-PA coupling after M-TEER are available from a study of 129 patients with primary or ventricular MR, approximately one quarter of whom underwent concomitant tricuspid TEER. At one-year follow-up, the authors observed significant improvement in RV-PA coupling, reflected by a mean increase in TAPSE of 3 mm and a mean reduction in PASP of approximately 10 mmHg. [20]

Given the established prognostic value of baseline RV-PA coupling, an important question is whether post-procedural coupling provides additional prognostic information and whether the magnitude of change itself is clinically meaningful. In our study, TAPSE/PASP assessed one month after M-TEER remained an independent predictor of death or heart failure hospitalization, whereas improvement in TAPSE/PASP was associated with better functional status but not with improved long-term outcome. Importantly, this finding is consistent with the study by Shechter et al., who likewise demonstrated that the achieved TAPSE/PASP value at one month, rather than the change in TAPSE/PASP, was associated with clinical outcome after M-TEER. [15] In contrast, Adamo

et al. reported a prognostic association of RV-PA coupling improvement in patients with ventricular MR. [10]

Several mechanisms may explain why the achieved TAPSE/PASP value at one month carried greater prognostic relevance than the magnitude of early change. First, the post-procedural TAPSE/PASP value reflects the patient's actual haemodynamic state after M-TEER, whereas the change in TAPSE/PASP only describes the trajectory from baseline. Patients with severely impaired baseline RV-PA coupling may therefore show a numerically relevant increase in TAPSE/PASP but still remain within an uncoupled, high-risk range. Second, early improvement in TAPSE/PASP may predominantly reflect haemodynamic unloading rather than true reverse remodelling of the right ventricle or pulmonary vasculature. Successful MR reduction can rapidly lower left atrial and pulmonary venous pressures, decreasing PASP and increasing TAPSE/PASP; however, this early pressure unloading does not necessarily imply recovery of RV contractile reserve or reversal of chronic pulmonary vascular remodelling.

Third, persistent RV-PA uncoupling despite apparent improvement may identify patients with advanced or only partially reversible right-sided disease. In such patients, the mitral intervention may reduce pulmonary loading conditions sufficiently to improve TAPSE/PASP numerically, but not enough to restore effective RV-PA coupling. Persistent uncoupling may therefore reflect residual pulmonary vascular disease, ongoing elevation of left-sided filling pressures, relevant TR, or limited RV adaptive reserve. This may explain why short-term improvement in TAPSE/PASP was associated with better functional status, whereas the achieved one-month TAPSE/PASP value more robustly identified patients at increased risk of death or heart failure hospitalization. Taken together, these considerations suggest that early changes in TAPSE/PASP capture the immediate haemodynamic response to M-TEER, while the achieved post-procedural TAPSE/PASP value better reflects the residual burden of RV-pulmonary vascular disease. Therefore, post-procedural coupling status may be more suitable for risk stratification than the magnitude of early change alone. Importantly, baseline TAPSE/PASP was the only independent predictor of preserved RV-PA coupling at follow-up. In addition, patients with preserved post-procedural RV-PA coupling had more favourable baseline functional status and a lower comorbidity burden. These findings suggest that the prognostically relevant coupling status achieved after M-TEER may partly reflect pre-existing RV-PA reserve, alongside the haemodynamic consequences of MR reduction.

Regarding determinants of early recoupling, we identified more severe TR and female sex as independent negative predictors of improvement. Severe TR may indicate advanced right-sided remodelling with volume overload and annular dilatation, a phenotype in which pulmonary unloading after M-TEER alone may be insufficient to restore efficient RV-arterial interaction. Moreover, substantial TR can lead to underestimation of PASP by Doppler methods, potentially obscuring true changes in afterload and thereby limiting the sensitivity of TAPSE/PASP to detect meaningful improvement. These findings align with prior TEER registry data showing that TR severity and RV-PA coupling capture complementary aspects of right-sided disease burden and risk. [14] In addition, several large registries have shown that TR severity at baseline, as well as early TR reduction after M-TEER, carry prognostic information, suggesting that persistent TR may represent an important marker (and potentially modifiable contributor) of residual right-sided load after successful MR reduction. [21,22]

The association between female sex and a lower likelihood of improvement in RV-PA coupling warrants further investigation. Sex-related differences in right heart structure and pulmonary vascular physiology, as well as differing baseline distributions of atrial fibrillation, diastolic dysfunction, and frailty, may influence haemodynamic recovery after valve intervention. Mechanistically, invasive physiology studies in pulmonary arterial hypertension have demonstrated sex-related differences in RV-PA coupling, supporting the concept that biological sex may influence RV adaptation to afterload and the

observed coupling phenotype. [23] Furthermore, reference values and longitudinal trajectories of RV-PA coupling may differ between women and men. In a cohort of 1542 healthy participants, TAPSE/PASP was lower in women than in men and was more strongly associated with functional capacity. [24] This suggests that sex-specific TAPSE/PASP thresholds may be helpful for longitudinal assessment. [25]

4.3. Clinical implications

From a clinical standpoint, indices of RV-PA coupling may provide incremental value beyond established echocardiographic and clinical parameters for risk stratification before and after M-TEER. Consistent with previous reports, [16,17] our data show that impaired baseline RV-PA coupling, assessed by TAPSE/PASP, identifies patients at higher risk of death and heart failure hospitalization despite treatment for atrial or primary MR. Assessing RV-PA coupling during patient selection for M-TEER may therefore support more refined prognostic evaluation and help guide the intensity of post-procedural follow-up. However, RV-PA uncoupling should not be viewed as evidence of futility. In the COAPT trial, impaired RV-PA coupling identified a high-risk phenotype, but the relative benefit of M-TEER over guideline-directed medical therapy was preserved across coupling strata. [8] This suggests that coupling metrics are better used to inform risk communication and individualise care than to withhold treatment.

Beyond procedural selection, serial assessment of RV-PA coupling may be clinically useful in two scenarios. First, in patients with severe MR who are asymptomatic or only mildly symptomatic, emerging RV-PA uncoupling may signal a transition from compensated pulmonary hypertension to limited RV reserve, thereby identifying a subgroup in whom the haemodynamic burden of valvular disease is no longer fully tolerated despite the absence of advanced symptoms. In such cases, worsening coupling may complement conventional clinical and echocardiographic markers and potentially support earlier referral to a Heart Team for comprehensive evaluation when anatomy is favourable and intervention remains technically feasible. Second, our study showed that TAPSE/PASP assessed one month after M-TEER is a significant predictor of death or hospitalization for heart failure. Accordingly, persistent RV-PA uncoupling after M-TEER may identify patients who warrant intensified surveillance for heart failure symptoms, optimisation of volume status and rhythm management, and evaluation of residual contributors to right-sided load such as residual MR, TR progression, or pulmonary vascular disease. In this context, follow-up assessment of RV-PA coupling may help distinguish patients with adequate haemodynamic recovery from those with persistent right-sided vulnerability despite successful mitral intervention, thereby informing the intensity of clinical follow-up and the need for additional diagnostic or therapeutic measures.

4.4. Study limitations

Several limitations should be acknowledged. First, this was a single-centre observational study with a moderate sample size. Accordingly, the findings should be considered hypothesis-generating, particularly with regard to aetiology-specific analyses in atrial and primary MR. Subgroup analyses were limited by smaller patient numbers and event counts and were therefore interpreted as exploratory. Larger multicentre cohorts are required to validate the prognostic value of RV-PA coupling and to determine whether its clinical implications differ by MR aetiology. Second, PASP was derived by Doppler echocardiography rather than invasively. While echocardiographic PASP estimation is established in clinical practice, it is subject to limitations related to TR jet quality and right atrial pressure estimation; [13] therefore, misclassification of coupling status and of short-term changes cannot be excluded. Studies directly comparing invasive and non-invasive assessment indicate a superior prognostic performance of invasively derived RV-PA coupling. [26,27] Nonetheless, the non-invasive measure retains

substantial prognostic value and represents a practical surrogate when right heart catheterization is not feasible. [6,16] Third, we focused on TAPSE-based RV assessment and did not systematically collect additional RV functional indices such as RV fractional area change or RV strain. While these parameters may provide complementary information, TAPSE remains widely used due to its feasibility and reproducibility in routine practice. [13] Finally, echocardiographic follow-up was limited to one month after M-TEER, which precluded analysis of longer-term TAPSE/PASP dynamics. RV reverse remodelling, pulmonary vascular adaptation, and the evolution of TR may occur over longer time horizons; therefore, future studies with serial echocardiographic follow-up at later timepoints are needed to define the temporal course and clinical implications of RV-PA coupling after M-TEER. Nevertheless, the one-month evaluation may serve as an early post-procedural assessment and may therefore be particularly relevant for early risk stratification to guide the intensity of subsequent follow-up after M-TEER.

5. Conclusions

In patients undergoing M-TEER for primary or atrial MR, RV-PA coupling assessed by TAPSE/PASP improved in a substantial proportion of patients at one month. Persistent post-procedural RV-PA uncoupling identified a higher-risk subgroup, whereas early improvement in coupling was associated with better functional status but not with improved long-term outcome. More severe TR, higher residual MR grade and female sex were associated with a lower likelihood of coupling improvement. Future studies in dedicated aetiology-specific cohorts are required to establish the role of serial RV-PA coupling assessment in post-procedural risk stratification after M-TEER.

CRedit authorship contribution statement

Michael Paulus: Writing – review & editing, Writing – original draft, Visualization, Project administration, Investigation, Formal analysis, Data curation, Conceptualization. **Nure Suloja:** Writing – review & editing, Investigation, Data curation. **Franziska Grewe:** Writing – review & editing, Investigation, Data curation. **Moritz Haus:** Writing – review & editing, Investigation, Data curation. **Valeska Bienert:** Writing – review & editing, Investigation, Data curation. **Michael Wester:** Writing – review & editing, Investigation, Data curation. **Christian Schach:** Writing – review & editing, Investigation. **Andreas Luchner:** Writing – review & editing, Supervision, Resources. **Christoph Birner:** Writing – review & editing, Supervision, Resources, Investigation, Conceptualization. **Bernhard Unsöld:** Writing – review & editing, Supervision, Resources, Investigation. **Lars S. Maier:** Writing – review & editing, Supervision, Resources, Project administration. **Kurt Debl:** Writing – review & editing, Supervision, Resources, Project administration, Investigation, Conceptualization. **Christine Meindl:** Writing – review & editing, Writing – original draft, Supervision, Resources, Project administration, Investigation, Conceptualization.

Funding

The work did not receive external funding.

Declaration of competing interest

M. Paulus and C. Meindl have received speaker fees and travel support from Edwards Lifesciences. M. Wester has received travel support from Edwards Lifesciences. C. Birner has received speaker fees from Edwards Lifesciences.

Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.ijcard.2026.134953>.

[org/10.1016/j.ijcard.2026.134953](https://doi.org/10.1016/j.ijcard.2026.134953).

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