ESC HEART FAILURE
ESC Heart Failure 2025; **12**: 4451–4462
Published online 28 October 2025 in Wiley Online Library (wileyonlinelibrary.com) **DOI:** 10.1002/ehf2.70001

ORIGINAL ARTICLE

Adaptation of right ventricular function following tricuspid transcatheter edge-to-edge repair

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Abstract

Background The impact of tricuspid transcatheter edge-to-edge repair (T-TEER) on right ventricular (RV) remodelling remains unclear.

Objectives This study focused on characterizing changes in RV ejection fraction (RVEF) following T-TEER and their prognostic implications.

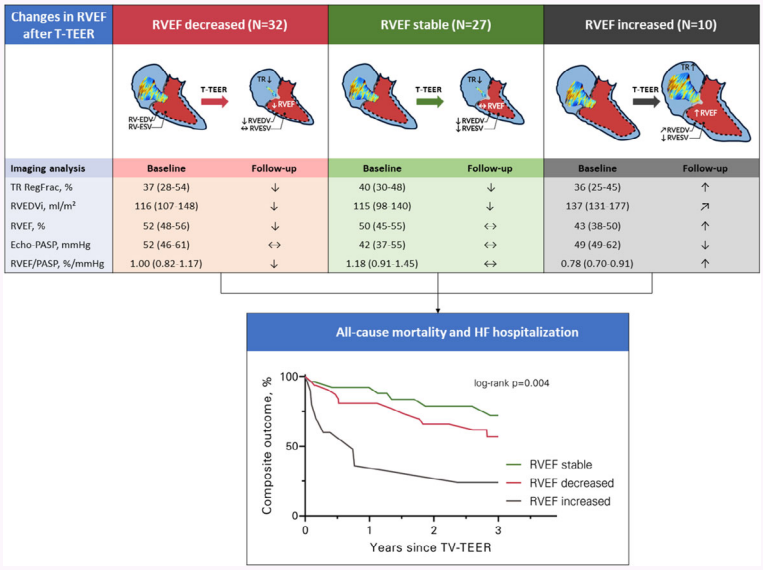
Methods Patients with significant tricuspid regurgitation (TR) who underwent T-TEER and cardiac magnetic resonance (CMR) imaging were included. Follow-up CMR was performed within 1 to 3 months after the procedure. Patients were classified by postprocedural RVEF change: decreased (≤−5%), stable (−4% to 4%) or increased (≥5%). The primary outcome was a composite of all-cause mortality or heart failure hospitalization.

Results The study included 69 patients (median age 78 years; 54% female). RVEF decreased in 32 (46%), was stable in 27 (39%), and increased in 10 (15%). Compared with patients with decreased and stable RVEF, those with increased RVEF had lower baseline RVEF (43% vs. 52% and 50%, respectively, $P = 0.007$) and lower baseline RV to pulmonary artery coupling (0.78 vs. 1.00 and 1.18, $P = 0.045$). Pulmonary artery systolic pressure was lower in patients with stable RVEF (42 mmHg vs. 52 mmHg in decreased and 49 mmHg in increased RVEF group, $P = 0.048$). TR severity was significantly reduced in the decreased and stable RVEF groups ($P < 0.001$ for both) while it worsened in the increased RVEF group ($P = 0.037$). After T-TEER, effective RVEF rose significantly in decreased (30% to 35%) and stable (31% to 39%) groups (both $P < 0.001$) but tended to decline in the increased group (30% to 20%; $P = 0.17$). During a median follow-up of 1016 days, 16 patients died, and 15 were hospitalized for heart failure. Event rates were lowest in the stable RVEF group and highest in the increased RVEF group (log-rank $P = 0.004$).

Conclusions RV response to T-TEER is heterogeneous, mostly influenced by baseline RV function, RV-PA coupling and TR progression. Only a minority of patients exhibited an increase in RVEF post T-TEER, and these patients showed worsening TR and a poorer prognosis.

Graphical Abstract

Right ventricular remodelling following tricuspid transcatheter edge-to-edge repair. After T-TEER, most patients either maintain or decrease their RVEF, while only a minority experience an increase. Patients with increased RVEF were associated with TR worsening and worse prognosis. CMR, cardiac magnetic resonance; HF, heart failure; PASP, pulmonary artery systolic pressure; RVEF, right ventricular ejection fraction; RVEDV, right ventricular end-diastolic volume; RVESV, right ventricular end-systolic volume; T-TEER, tricuspid transcatheter edge-to-edge repair; TR, tricuspid regurgitation.



Keywords cardiac magnetic resonance; right ventricle; right ventricle–pulmonary artery coupling; survival; tricuspid regurgitation; tricuspid transcatheter edge-to-edge repair

Received: 1 July 2025; Revised: 22 September 2025; Accepted: 26 September 2025
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Introduction

Tricuspid Transcatheter Edge-to-Edge Repair (T-TEER) has emerged as a promising therapy for patients with significant tricuspid regurgitation (TR), particularly those at high surgical risk.[1] The TRILUMINATE trial, the first randomized controlled study of T-TEER, demonstrated significant improvements in quality of life compared with optimal medical therapy alone. While no significant differences in mortality were observed, the trial did show a reduction in heart failure hospitalization rates at 3 years of follow-up.[2,3] A key concern with T-TEER involves its haemodynamic impact on right ventricular (RV) function. Acute TR reduction may increase RV afterload by eliminating the low-resistance regurgitant orifice, potentially precipitating or exacerbating RV dysfunction.[4] Previous studies investigating RV function following T-TEER have employed different parameters, imaging modalities and assessment time points, leading to heterogeneous findings.[5–12] Recent evidence using three-dimensional echocardiography suggests that RV remodelling after T-TEER is a dynamic and biphasic process, consisting of an initial phase of rapid volume unloading (reduction in RV end-diastolic volume, RVEDV) and a subsequent phase of structural remodelling (reduction in RV end-systolic volume, RVESV) with gradual recovery of systolic function.[11] However, assessing RV function with echocardiography remains challenging due to the complex RV geometry, especially after T-TEER, and even advanced techniques such as three-dimensional echocardiography are limited by acoustic windows and operator dependency. Cardiac magnetic resonance (CMR) imaging is considered the gold standard for RV assessment, offering superior accuracy and reproducibility. This study aims to characterize the early RV response to T-TEER by employing CMR imaging within 3 months after the procedure and their association with functional and clinical outcomes.

Methods

Study population

Patients who underwent CMR before and 1–3 months after T-TEER for isolated TR of at least moderate severity at the Heart Center Leipzig, University of Leipzig, between June 2016 and July 2022 were retrospectively included in this study. Exclusion criteria for all subjects included poor echocardiographic visualization of the tricuspid valve on screening transoesophageal echocardiography, cardiac implantable electronic device-related TR, any degree of tricuspid stenosis, severe aortic stenosis, tricuspid valve anatomy deemed unsuitable for T-TEER and the generally accepted contraindications to CMR.[5,13] Patients with combined tricuspid and mitral valve repair, inadequate imaging quality or incomplete CMR protocol were excluded from the analysis. Baseline and follow-up clinical and echocardiographic data were collected. N-terminal pro–B-type natriuretic peptide levels and a 6 min walk distance were evaluated. Echocardiographic assessment was performed as described before.[5,14] TR was defined according to current guidelines combining different semi-quantitative and quantitative parameters.[15] An expanded grading scale from Grades 1 to 5 was utilized.[16] The decision to perform TR therapy was based on international guidelines and through heart team consensus. The procedures were conducted using the MitraClip and Triclip (Abbott Cardiovascular, USA) and PASCAL (Edwards Lifesciences, USA) devices. The study conforms to the principles outlined in the Declaration of Helsinki, written informed consent was obtained from all patients and the local ethics committee approved this analysis.

CMR protocol

All patients underwent routine CMR protocol for volumes and flow assessment using a 1.5-Tesla scanner (Intera, Philips Healthcare, Eindhoven, the Netherlands) using a standardized protocol.[5,13] Cine images using retrospectively gated steady-state free precession were acquired in three long-axis (left ventricular 4-, 3-, 2- chamber), and in multiple short-axis views covering the entirety of both ventricles. Aortic and pulmonary flow data were acquired with a flow-sensitive gradient echo sequence. All routine CMR analysis was performed by using the cardiovascular imaging version 42 software (cvi 42, Circle Cardiovascular Imaging, Calgary, Alberta, Canada). Assessment of left ventricular and RV volumes was performed by manually tracing the endocardial border at end-diastole and end-systole. Left ventricular and RVEDV and RVESV were determined using the slice summation method. An example of RV basal contouring in the presence of a TEER device is provided in *Figure* S1. The total stroke volume (SV) was the difference between EDV and ESV, and RV ejection fraction (RVEF) was calculated as SV divided by EDV expressed as a percentage. A threshold of 5% was selected to define clinically meaningful changes in RVEF, as it corresponds to the expected inter-observer variability of RVEF measurements reported in previous studies.[17,18] In addition to RVEF, the effective RVEF was evaluated as a measure of RV global systolic function corrected by TR volume.[19] Its value was calculated according to the formula: pulmonary forward flow/RVEDV. Effective RVSV was calculated as follows: pulmonary forward flow − pulmonary backward flow. Tricuspid regurgitant volume was calculated by subtracting the pulmonary forward flow from the RVSV. Tricuspid regurgitant fraction was calculated as a percentage by dividing the tricuspid regurgitant volume by the RVSV. RV-pulmonary artery (PA) coupling was evaluated using the ratio of RVEF/PA systolic pressure (PASP).[20] Strain analysis was performed using feature tracking analysis offline. RV longitudinal strain was measured from the long-axis 4-chamber view, excluding the septum. Circumferential and radial strain were obtained from the short-axis view, excluding the septum, using at least three different slices representing the basal, midventricular and apical segments of the RV.[13] CMR measurements were indexed to body surface area and denoted with an 'i' (e.g., RVEDVi). CMR analysis was performed blinded to patients' outcomes.

Outcomes and definitions

The procedural success of T-TEER was defined as the successful placement of at least one device and a TR reduction to moderate or less (≤2+). For a more comprehensive evaluation of the RV's response following T-TEER, patients were stratified into three groups based on changes observed in RVEF:

1. Decreased RVEF (RVEF absolute change ≤−5%),
2. Stable RVEF (RVEF absolute change ranging from −4% to 4%),
3. Increased RVEF (RVEF absolute change ≥5%).

The primary outcome was a composite of all-cause mortality and/or heart failure hospitalization at the last available follow-up.

Follow-up examinations

Follow-up examinations, including clinical and echocardiographic assessments, were conducted concurrently with the repeated CMR scans. The evaluation of mortality and heart failure hospitalizations was conducted by reviewing in-hospital data or by contacting the treating general physicians.

Statistical analysis

Continuous data were presented as the median and interquartile range (IQR). Frequencies and proportions were used to present categorical variables. Continuous variables were compared using either the two-sample Wilcoxon rank-sum test (Mann–Whitney test) or the Kruskal–Wallis test, where appropriate. Categorical variables were compared using Fisher's exact test. The Wilcoxon matched-pairs signed-rank test and McNemar's test were used to compare continuous and categorical variables before and after T-TEER, respectively. Spearman's rho was used to assess correlations between non-parametric variables. Linear regression analysis was performed to identify independent predictors of changes in RVEF following T-TEER. Survival analysis was performed using the Cox proportional hazards regression model. Differences in survival among subgroups were analysed with Kaplan–Meier analyses and the log-rank test. A two-sided significance level of $\alpha = 0.05$ was used to indicate statistical significance. In case of missing data, a complete case analysis approach was employed. All statistical analyses were performed using StataSE17 (StataCorp. 2021. Stata Statistical Software: Release 17. College Station, TX, USA: StataCorp LLC.).

Results

Baseline characteristics

A total of 81 patients with isolated TR underwent T-TEER and had both pre- and post-procedural CMR imaging within 1 to 3 months. Twelve patients were excluded due to inadequate image quality in either the pre- or post-procedure CMR scans or lack of flow analysis. Ultimately, the final study population comprised 69 patients for analysis. CMR scans were performed at a median of 1 day (IQR: 1–3 days) prior to the intervention and 43 days (IQR: 34–50 days) after. Based on changes in RVEF from pre- to post-T-TEER, the cohort was divided into three groups: decreased RVEF (n = 32, 46%), stable RVEF (n = 27, 39%) and increased RVEF (n = 10, 15%). In the decreased RVEF group, median RVEF declined from 52% (IQR 48–56) to 43% (IQR 39–46) ($P < 0.001$). In the stable RVEF group, RVEF remained unchanged, from 50% (IQR 45–55) to 50% (IQR 46–55) (P = 0.33). In the increased RVEF group, RVEF improved from 43% (IQR 38–50) to 52% (IQR 48–57) (P = 0.005). Patients exhibited typical features of a T-TEER population, including advanced age, high perioperative risk, impaired functional capacity and a high prevalence of atrial fibrillation. There were no significant differences in clinical baseline characteristics and laboratory values among the groups (*Table* 1). Examining the imaging characteristics, patients with decreased and stable RVEF exhibited similar features in terms of RVEF and RV volumes, whereas patients with increased RVEF were characterized by lower baseline RVEF and higher RV volumes (*Table* 2, *Table* S1). PASP was significantly lower in patients with stable RVEF, both when estimated by echocardiography (P = 0.048) and when measured invasively by right heart catheterization (P = 0.036). As a result, the baseline RV-PA coupling, assessed by the RVEF/PASP ratio, differed significantly among groups (P = 0.017). Patients with increased RVEF had the lowest ratio [RVEF (%)/PASP (mmHg): 0.78], whereas those with stable RVEF exhibited the highest ratio [RVEF (%)/PASP (mmHg): 1.18], indicating more favourable RV-PA coupling. There were no significant differences in tricuspid regurgitant fraction or effective RVEF at baseline among the three groups (P = 0.60 and P = 0.47, respectively), nor in the timeframe of patient inclusion within each of the respective three groups (Figure S2). During the procedure, a total of 133 devices were implanted, with a median of two devices per patient. Among the patients, 62% received MitraClip, 16% received TriClip and 22% received PASCAL. In patients with increased RVEF, the procedural success rate was lowest at 70%, compared with higher success rates of 97% and 93% observed in patients with decreased and stable RVEF, respectively (P = 0.026, Table S2, *Figure* 1).

Changes in RV parameters and haemodynamics after T-TEER

Following T-TEER, the tricuspid regurgitant fraction decreased significantly in both decreased and stable RVEF patients: from 37% (IQR: 28–54) to 14% (IQR: 5–28) in those with decreased RVEF, and from 40% (IQR: 30–48) to 17% (IQR: 12–30) in those with stable RVEF ($P < 0.001$ for both). In contrast, patients with increased RVEF experienced a significant rise in regurgitant fraction, from 36% (IQR: 25–45) to 64% (IQR: 41–66; P = 0.037) (*Table* 3). RVEDVi was significantly reduced following the procedure in patients with decreased or stable RVEF ($P < 0.001$ for both). The change in tricuspid regurgitant fraction showed a strong correlation with the change in RVEDVi (r = 0.71, $P < 0.001$; Figure S3). Despite differing trends in volumetric RVEF, effective RVEF increased significantly in both the decreased and stable RVEF groups: from 30% (IQR: 23–40) to 35% (IQR: 29–44) in the decreased group, and from 31% (IQR: 25–36) to 39% (IQR: 32–47) in the stable group ($P < 0.001$ for both; P = 0.002 for the difference between deltas). In contrast, patients with increased RVEF exhibited a numerical reduction in effective RVEF, from 30% (IQR: 23–31) to 20% (IQR: 18–28), although this was not statistically significant (P = 0.17).

PASP remained stable in patients with decreased and stable RVEF, while a significant reduction was observed in patients with increased RVEF (P = 0.028). Consequently, the RVEF/PASP ratio declined in patients with decreased RVEF, remained stable in those with stable RVEF and increased in

Table 1 Baseline clinical and laboratory characteristics according to type of RV remodelling.

	All patients (n = 69)	RVEF decreased (n = 32)	RVEF stable (n = 27)	RVEF increased (n = 10)	P value
Clinical characteristics					
Age, years	78 (75–82)	78 (74–82)	78 (76–81)	79 (76–82)	0.86
Female, %	37 (54%)	15 (47%)	17 (63%)	5 (50%)	0.46
BMI, kg/m^2	26.5 (23.0–30.5)	26.2 (23.0–30.7)	26.9 (22.7–29.5)	26.9 (23.8–31.2)	0.90
Euroscore II, %	4.63 (2.70–8.16)	4.15 (2.68–7.67)	5.66 (2.70–7.48)	4.31 (2.46–10.16)	0.69
NYHA III–IV, %	54 (78%)	25 (78%)	23 (85%)	6 (60%)	0.29
6MWD, m	289 (171–388)	285 (160–364)	295 (177–393)	314 (203–422)	0.42
Previous PCI, %	13 (19%)	7 (22%)	5 (19%)	1 (10%)	0.92
Previous CABG, %	9 (13%)	3 (9%)	5 (19%)	1 (10%)	0.70
Previous valv. interv., %	17 (25%)	6 (19%)	8 (30%)	3 (30%)	0.60
Lung disease, %	15 (22%)	7 (22%)	5 (19%)	3 (30%)	0.79
Current dialysis, %	2 (3%)	2 (6%)	0 (0%)	0 (0%)	0.63
Atrial fibrillation, %	61 (88%)	29 (91%)	23 (85%)	9 (90%)	0.87
Beta-blockers, %	61 (90%)	29 (91%)	23 (88%)	9 (90%)	1.00
ACE inhibitors/ARB, %	60 (88%)	27 (84%)	23 (88%)	10 (100%)	0.46
Diuretics, %	63 (93%)	30 (94%)	24 (92%)	9 (90%)	1.00
Laboratory tests					
NTproBNP, pg/mL	2,438 (1,479–4,334)	2,255 (1,611–4,355)	2,848 (1,486–3,656)	2,300 (855–5,279)	0.99
Creatinine, mg/dL	1.25 (1.08–1.68)	1.41 (0.99–1.81)	1.21 (1.10–1.50)	1.59 (0.80–1.71)	0.70
Bilirubin, μmol/L	11.9 (8.1–16.3)	10.7 (7.05–14.95)	11.9 (8.0–15.9)	15.1 (11.5–18.3)	0.20
AST, U/L	0.44 (0.38–0.51)	0.43 (0.36–0.53)	0.46 (0.43–0.54)	0.40 (0.34–0.44)	0.06
ALT, U/L	0.31 (0.22–0.41)	0.33 (0.24–0.49)	0.31 (0.25–0.41)	0.22 (0.18–0.29)	0.06
Albumin, g/L	43 (39–46)	43 (39–46)	44 (41–46)	41 (39–46)	0.84

Abbreviations: ACE, angiotensin-converting enzyme; ALT, alanine aminotransferase; ARB, angiotensin receptor blockers; AST, aspartate aminotransferase; BMI, body mass index; CABG, coronary artery bypass graft; n, number of patients; NTproBNP, N-terminal pro-brain natriuretic peptide; NYHA, New York Heart Association; PCI, percutaneous coronary intervention; valv. Interv., valvular intervention; 6MWD, six-minute walking distance.

patients with increased RVEF. Thus, only in patients with increased RVEF did both RVEF and the RVEF/PASP ratio rise, although this was accompanied by a trend towards a reduction in effective RVEF. In line with this, these patients were already characterized by RV-PA uncoupling at baseline, as indicated by lower baseline RVEF and a lower ratio of RVEF/PASP (*Table* 1, *Table* S1).

In linear regression analysis, baseline RVEF was a significant independent predictor of post-procedural change in RVEF (β = −0.339, P = 0.003) (Table S3). Among patients with procedural success, PASP showed a significant inverse association with RVEF change (β = −0.154, P = 0.009) (Table S4).

Outcomes

During a median follow-up period of 1016 days (IQR: 407 to 1095 days), the composite outcome of all-cause mortality and/or heart failure hospitalization was observed in 25 patients (16 deaths and 15 heart failure hospitalizations). Patients with increased RVEF had the worst outcome while outcomes were almost comparable for patients with decreased and stable RVEF (log-rank P = 0.004). This was mainly driven by all-cause mortality (log-rank P = 0.030) but also showed a trend for heart failure events (log-rank P = 0.09) (graphical abstract, *Figure* 2). However, patients with stable RVEF consistently had the lowest event rates. Clinical improvements in New York Heart Association functional class, presence of oedema and the 6 min walking distance were observed in patients with decreased and stable RVEF, but not in those with increased RVEF (*Figure* 3).

In Cox regression analysis, higher baseline RVEF was independently associated with a lower risk of the composite outcome [hazard ratio (HR) per unit increase: 0.94, 95% CI 0.90–0.98, P = 0.007], whereas baseline effective RVEF was not significantly associated (HR 1.01, 95% CI 0.96–1.05, P = 0.75). However, greater increases in effective RVEF after intervention were significantly associated with a reduced risk of the composite outcome (HR 0.91, 95% CI 0.87–0.96, $P < 0.001$; Table S5).

Discussion

This study comprehensively assessed the impact of T-TEER induced haemodynamic changes on the RV and its clinical implications.

The main findings are as follows:

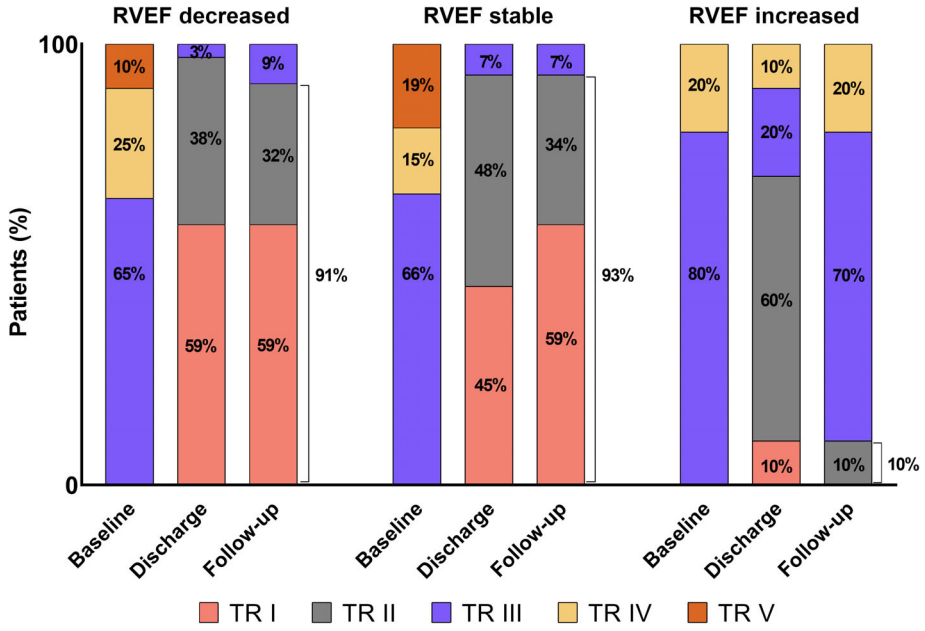
1. There is a heterogenous early response to T-TEER, with some patients showing stable, some decreased and some even increased RVEF within a short-term follow-up;
2. Changes in RVEF following T-TEER are primarily driven by the degree of TR reduction, baseline RVEF and RV-PA coupling;
3. While patients with decreased and stable RVEF exhibit comparable outcomes, patients with increased RVEF show the worst outcomes;

Table 2 Baseline imaging and haemodynamic characteristics according to type of RV remodelling.

	All patients (n = 69)	RVEF decreased (n = 32)	RVEF stable (n = 27)	RVEF increased (n = 10)	P value
Echocardiography					
TR grade					0.59
III	47 (68%)	21 (65%)	18 (66%)	8 (80%)	
IV	14 (20%)	8 (25%)	4 (15%)	2 (20%)	
V	8 (12%)	3 (10%)	5 (19%)	0 (0%)	
TR VC biplane, mm	9 (8–11)	9 (8–11)	10 (8–12)	9 (8–11)	0.73
TR EROA (PISA), mm^2	0.50 (0.40–0.60)	0.50 (0.40–0.60)	0.50 (0.40–0.70)	0.50 (0.40–0.60)	0.74
TR R vol (PISA), mL	47 (37–55)	48 (40–59)	40 (32–56)	44 (36–50)	0.38
PASP, mmHg	49 (42–60)	52 (46–61)	42 (37–55)	49 (49–62)	0.048
Cardiac magnetic resonance					
RVEF, %	50 (44–55)	52 (48–56)	50 (45–55)	43 (38–50)	0.023
Effective RVEF, %	30 (24–36)	30 (23–40)	31 (25–36)	30 (23–31)	0.47
RVEDVi, mL/m^2	120 (105–144)	116 (107–148)	115 (98–140)	137 (131–177)	0.11
RVESVi, mL/m^2	60 (47–78)	61 (47–72)	54 (45–76)	78 (65–106)	0.029
RVSVi, mL/m^2	61 (53–70)	61 (52–74)	59 (52–67)	64 (53–71)	0.49
RA area, cm^2	40 (33–47)	41 (34–46)	37 (33–45)	46 (40–56)	0.14
Effective RVSV, mL	61 (55–84)	66 (57–88)	61 (53–75)	60 (55–84)	0.47
TR RegFrac, %	39 (29–49)	37 (28–54)	40 (30–48)	36 (25–45)	0.60
TR R vol, mL	41 (30–59)	41 (30–68)	43 (34–55)	38 (25–58)	0.62
RVEF/PASP, %/mmHg	1.00 (0.80–1.23)	1.00 (0.82–1.17)	1.18 (0.91–1.45)	0.78 (0.70–0.91)	0.017
Longitudinal strain, %	−19.3 (−22.0 to −16.3)	−19.9 (−22.5 to −18.1)	−18.6 (−21.5 to −16.2)	−17.2 (−19.5 to −15.6)	0.32
Radial strain, %	21.3 (17.3–26.0)	22.3 (19.2–27.1)	20.6 (16.7–24.7)	14.9 (12.4–24.8)	0.17
Circumferential strain, %	−13.9 (−15.8 to −11.6)	−14.5 (−16.0 to −12.2)	−13.2 (−15.5 to −11.5)	−10.7 (−14.8 to −9.1)	0.13
Haemodynamics					
SBP, mmHg	140 (123–153)	146 (128–158)	139 (120–157)	135 (120–144)	0.28
MAP, mmHg	96 (84–109)	96 (90–112)	98 (83–108)	90 (83–99)	0.37
CI, $mL/min/m^2$	2.00 (1.75–2.23)	1.95 (1.70–2.20)	2.00 (1.75–2.23)	2.15 (1.80–2.40)	0.50
RAP mean, mmHg	13 (10–18)	13 (8–19)	14 (10–17)	14 (13–19)	0.53
iPASP, mmHg	46 (39–53)	50 (40–63)	40 (35–49)	51 (46–53)	0.036
PADP, mmHg	17 (14–22)	17 (12–23)	16 (14–19)	21 (17–24)	0.07
Mean PAP	30 (24–34)	31 (24–38)	26 (22–32)	31 (28–34)	0.28
PCWP, mmHg	20 (16–25)	20 (16–23)	20 (14–27)	21 (16–24)	0.77

Abbreviations: EROA, effective regurgitant orifice area; iPASP, invasive pulmonary artery systolic pressure; MAP, mean arterial pressure; n, number of patients; NYHA, New York Heart Association; PADP, pulmonary artery diastolic pressure; PAP, pulmonary artery pressure; PASP, pulmonary artery systolic pressure; PCWP, pulmonary capillary wedge pressure; PISA, proximal isovelocity surface area; RA, right atrium; RAP, right atrial pressure; RegFrac, regurgitant fraction; R Vol, regurgitant volume; RV, right ventricular; RVEDVi, right ventricular end-diastolic volume index; RVESVi, right ventricular end-systolic volume index; RVEF, right ventricular ejection fraction; RVSVi, right ventricular stroke volume index; SBP, systolic blood pressure; TR, tricuspid regurgitation; VC, vena contracta.

Figure 1 Echocardiographic assessment of tricuspid regurgitation severity at baseline, discharge and follow-up. At discharge, patients with decreased and stable RVEF both showed a significant reduction in TR, which was consistently maintained during the follow-up period. In contrast, patients with increased RVEF demonstrated reduced procedural success and a progressive increase in TR severity over the follow-up period. Boxed labels indicate the percentage of subjects with moderate or less TR at the follow-up. RVEF, right ventricular ejection fraction; TR, tricuspid regurgitation.



4. Changes in volumetric RVEF following T-TEER should be interpreted with caution, whereas changes in effective RVEF appear to be a more clinically meaningful marker.

A reduction in RVEF following T-TEER has been reported in multiple studies, without being associated with adverse prognosis, but rather interpreted as a physiological response to changing loading conditions.[5–12]

This pathophysiological framework was contrasted by the different RVEF responses in patients with decreased and stable RVEF. The observed differences in RVEF response were not attributable to variations in the reduction of TR fraction between groups. Instead, they appeared to be linked to baseline conditions, particularly baseline PASP, RV-PA coupling and contractile reserve. Our findings demonstrated that, among patients with procedural success, elevated baseline PASP was significantly associated with a decreased post-procedural RVEF.

Therefore, in patients with higher baseline PASP and impaired RV-PA coupling, device implantation might have led to a more pronounced increase in afterload, which was not adequately compensated by a corresponding increase in contractility due to baseline RV insufficient contractile reserve.[21] Consequently, this subgroup exhibited a decrease in RVEF and effective RVSV.

However, this reduction was not associated with worse outcomes in the present analysis. One possible explanation for this could be that our patient selection process only included individuals who underwent follow-up CMR imaging, specifically selecting patients who demonstrated adaptive mechanisms in the RV, enabling them to tolerate the increased afterload. Patients experiencing acute afterload mismatch may have died between the baseline and follow-up imaging, although acute RV failure is a rare complication following T-TEER.[4] Furthermore, the limited duration of follow-up raises the possibility that individuals with decreased RVEF may experience deteriorating outcomes in the long term. Nevertheless, TR reduction was associated with volume unloading and with improvement in the actual forward RV function, as reflected by the increase in effective RVEF, which may explain their similar clinical outcomes.

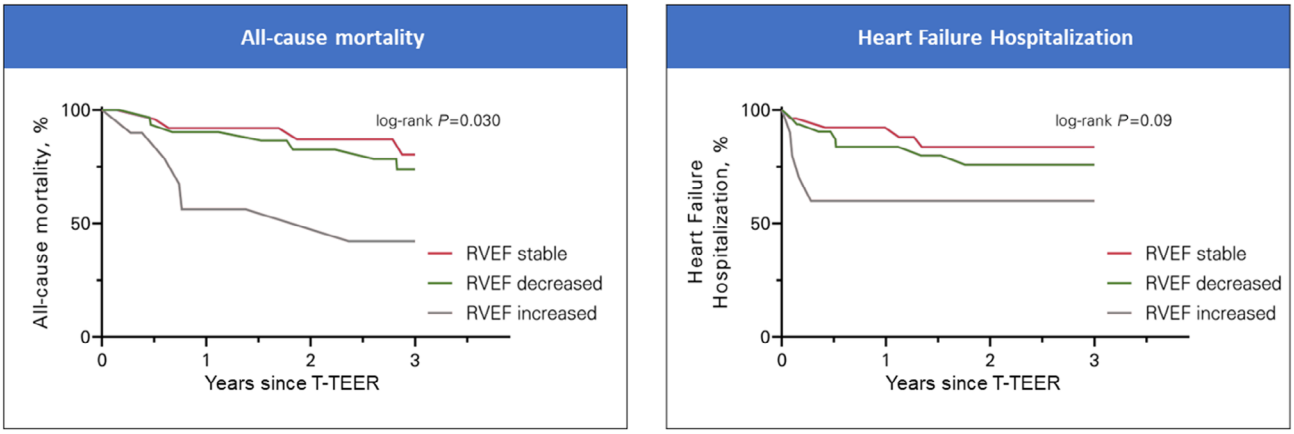
Counterintuitively, patients whose RVEF increased following T-TEER had worse outcomes. These patients had lower baseline RVEF and greater baseline RV-PA uncoupling compared with other groups, and lower baseline RVEF was associated with greater increases in RVEF. It had been demonstrated that patients with RVEF $<$ 45% in the presence of severe TR had a significantly poorer prognosis, likely attributable to pre-existing significant RV dysfunction that was masked by severe TR.[13] Furthermore, these patients exhibited worsening TR, and multiple registries had shown that residual TR was associated with poorer survival and an increased risk of heart failure hospitalization.[22–24] While these cases might have been considered procedural failures, they

Table 3 Imaging Results: Baseline and Follow-up Data according to type of RV remodelling.

	All patients (n = 69)			RVEF decreased (n = 32)			RVEF stable (n = 27)			RVEF increased (n = 10)		
	Baseline	Follow-up	P value	Baseline	Follow-up	P value	Baseline	Follow-up	P value	Baseline	Follow-up	P value
RVEF, %	50 (44–55)	46 (42–52)	<0.001	52 (48–56)	43 (39–46)	<0.001	50 (45–55)	50 (46–55)	0.33	43 (38–50)	52 (48–57)	0.005
Effective RVEF, %	30 (24–36)	35 (28–44)	<0.001	30 (23–40)	35 (29–44)	<0.001	31 (25–36)	39 (32–47)	<0.001	30 (23–31)	20 (18–28)	0.17
RVEDVi, mL/m^2	120 (105–144)	103 (78–134)	<0.001	116 (107–148)	99 (78–118)	<0.001	115 (98–140)	99 (73–121)	<0.001	137 (131–177)	145 (124–169)	0.68
RVESVi, mL/m^2	60 (47–78)	56 (40–71)	<0.001	61 (47–72)	58 (43–70)	0.21	54 (45–76)	46 (33–65)	<0.001	78 (65–106)	71 (59–77)	0.016
RVSVi, mL/m^2	61 (53–70)	44 (38–56)	<0.001	61 (52–74)	38 (33–46)	<0.001	59 (52–67)	50 (40–56)	<0.001	64 (53–71)	72 (65–85)	0.012
RA area, cm^2	40 (33–47)	38 (33–48)	0.39	41 (34–46)	38 (34–45)	0.045	37 (33–45)	36 (32–42)	0.23	46 (40–56)	54 (43–61)	0.016
Effective RVSV, mL	61 (55–84)	61 (51–76)	0.32	66 (57–88)	61 (51–79)	0.004	61 (53–75)	65 (55–76)	0.042	60 (55–84)	54 (42–85)	0.09
TR RegFrac, %	39 (29–49)	18 (9–30)	<0.001	37 (28–54)	14 (5–28)	<0.001	40 (30–48)	17 (12–30)	<0.001	36 (25–45)	64 (41–66)	0.037
TR R Vol, mL	41 (30–59)	16 (6–30)	<0.001	41 (30–68)	10 (3–21)	<0.001	43 (34–55)	13 (9–24)	0.003	38 (25–58)	86 (70–93)	0.028
CI, mL/min/m^2	2.22 (1.81–2.48)	2.31 (1.95–2.75)	0.006	2.24 (1.74–2.61)	2.16 (1.95–2.74)	0.29	2.20 (2.04–2.45)	2.46 (2.00–2.75)	0.004	2.25 (1.91–2.29)	2.08 (1.85–2.83)	0.51
PASP, mmHg	49 (42–60)	48 (39–53)	0.011	52 (46–61)	50 (41–59)	0.14	42 (37–55)	41 (30–51)	0.20	49 (49–62)	44 (41–49)	0.028
RVEF/PASP, %/mmHg	1.00 (0.80–1.23)	1.02 (0.82–1.28)	0.36	1.00 (0.82–1.17)	0.87 (0.66–1.08)	0.019	1.18 (0.91–1.45)	1.16 (1.02–1.60)	0.22	0.78 (0.70–0.91)	1.18 (0.96–1.46)	0.008

Abbreviations: n, number of patients; PASP, pulmonary artery systolic pressure; RA, right atrium; RegFrac, regurgitant fraction; R Vol, regurgitant volume; RV, right ventricular; RVEDVi, right ventricular end-diastolic volume index; RVESVi, right ventricular end-systolic volume index; RVEF, right ventricular ejection fraction; RVSVi, right ventricular stroke volume index; TR, tricuspid regurgitation.

Figure 2 Kaplan–Meier analysis: all-cause mortality and heart failure hospitalization. Patients with increased RVEF exhibited the poorest survival and a tendency towards a higher frequency of HF events. Outcomes for patients with stable and decreased RVEF were almost comparable; however, patients with a stable RVEF consistently showed the lowest event rates. HF, heart failure; RVEF, right ventricular ejection fraction; T-TEER, tricuspid transcatheter edge-to-edge repair.



offered valuable insights into how the RV adapts to altered loading conditions resulting from worsening TR. This scenario resulted in a rise in global RVEF and an increased RVEF/PASP ratio; however, these changes likely represented a maladaptive response rather than a true improvement in RV contractility. Indeed, the observed increase in RVEF did not translate into an actual improvement in effective RVSV or effective RVEF, making them mechanically ineffective. These observations are hypothesis-generating and should be interpreted with caution; however, they are supported by non-invasive haemodynamic estimates as outlined in more detail in the supplementary material (Supporting information methods and results, Tables S6 and S7). In summary, a decrease in RVEF after T-TEER does not necessarily indicate RV dysfunction, but rather reflects the limitations of RVEF, because it does not distinguish between forward and regurgitant flow. In contrast, effective RVEF, which excludes the regurgitant volume from the calculation, provides a more accurate measure of true RV systolic performance following TR correction.

Clinical implications

This study challenges the conventional interpretation of RVEF changes following T-TEER. While impaired baseline RVEF remains a strong predictor of poor prognosis,[13] an increase in RVEF following T-TEER, often assumed to indicate recovery, is associated with worse outcomes.

T-TEER primarily reduces RV preload but also increases afterload. In this context, a reduction in RVEF may reflect a physiological adaptation, particularly in patients with RV insufficient contractile reserve and should not be immediately interpreted as RV dysfunction. In contrast, an increase in RVEF, if accompanied by suboptimal procedural results, likely indicates disease progression and is an early warning sign of adverse outcomes.

Incorporating effective RVEF changes into post-procedural assessment provides a more accurate measure of RV systolic function and may improve therapeutic guidance.

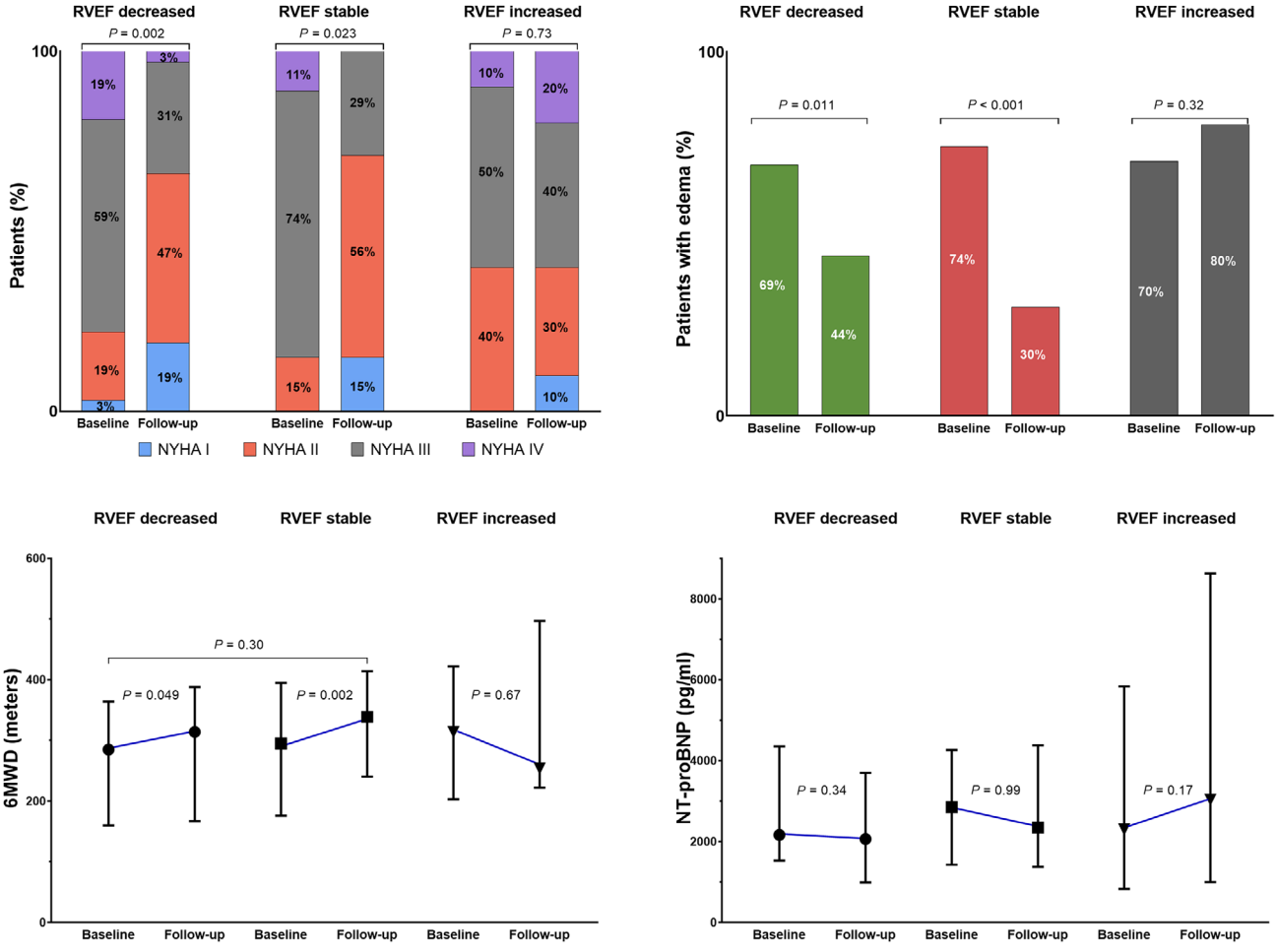
Limitations

This study utilized a retrospective design conducted at a single center, without a control group. The inclusion criteria introduced a potential selection bias as the focus was on patients who had pre- and post-procedural CMR imaging within a specific timeframe. The stratification of patients based on changes in RVEF was performed for exploratory purposes and must be interpreted with caution. The high prevalence of atrial fibrillation may have affected image quality and the accuracy of CMR measurements, potentially influencing the results. Finally, all haemodynamic considerations were derived from non-invasive estimates.

Conclusions

The results of this study provide insights into how to interpret RVEF before and after T-TEER, as well as in assessing procedural success. Contrary to expectations, only a minority experienced an improvement in RVEF, and these patients were associated with worsening TR and the worst prognosis. Further research incorporating invasive haemodynamic measurements before and after the procedure is necessary to validate these findings and gain a more comprehensive understanding of this complex pathophysiology.

Figure 3 Clinical outcomes according to types of RV Remodelling. Changes of (A) NYHA functional class, (B) presence of peripheral edema, (C) 6MWD and (D) NT-proBNP from baseline to follow-up. Data are presented as frequency in Parts (A) and (B) and median with interquartile range in Parts (C) and (D). 6MWD, 6 min walking distance; NYHA, New York Heart Association; NT-proBNP, N-terminal pro-B-type natriuretic peptide; RVEF, right ventricular ejection fraction.



Conflict of interest statement

K.P.R. has received speaker honoraria and consultation fees from Edwards Lifesciences. P. L. has received institutional fees and research grants from Abbott Vascular, Edwards Lifesciences and ReCor, honoraria from Edwards Lifesciences, Abbott Medical, Innoventric, ReCor, Boehringer Ingelheim and Daiichi Sankyo and has stock options with Innoventric. K. P. K. consultant to Edwards Lifesciences. All other authors have reported that they have no relationships relevant to the contents of this paper to disclose.

Acknowledgements

Open Access funding enabled and organized by Projekt DEAL.

Funding

This research received no external funding.

Supporting information

Additional supporting information may be found online in the Supporting Information section at the end of the article.

Table S1. Baseline Characteristics for Different Types of RV Remodelling.
Table S2. Treatment characteristics and procedural outcomes following T-TEER.
Table S3. Linear Regression Analysis of Baseline Predictors of Post-Procedural Changes in RVEF.

Table S4. Linear Regression Analysis of Baseline Predictors of RVEF Change in Patients with Procedural Success.

Table S5. Association of Right Ventricular Functional Parameters with Composite Outcome.

Table S6. Baseline Non-Invasive Hemodynamic Evaluation of RV loading conditions.

Table S7. RV loading conditions: Baseline and Follow-up Data according to type of RV Remodelling.

Table S8. Detailed Procedural and Follow-up Aspects for Patients with increased RVEF.

Figure S1. Example of right ventricular (RV) volume contouring in cardiac magnetic resonance (CMR) at the basal level in a patient with a transcatheter edge-to-edge repair (TEER) device. Reference to the 4-chamber view ensures correct identification and inclusion of basal RV segments.

Figure S2. Number of patients included per year, categorized into the three groups: RVEF decreased, stable, and increased. Abbreviations List. No. - Number of; RVEF – right ventricular ejection fraction.

Figure S3. Correlation between changes in Tricuspid Regurgitant Fraction and RV-EDVi, showing a positive association ($r = 0.71$, $p < 0.001$). Abbreviation List: RV-EDVi – right ventricular end-diastolic volume index.

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