JACC: CARDIOVASCULAR IMAGING VOL. 19, NO. 7, 2026

PUBLISHED BY ELSEVIER

ORIGINAL RESEARCH

Redefining Right Ventricular Function

Incremental Prognostic Utility of Effective RVEF on CMR in Functional Tricuspid Regurgitation—A Multicenter Validation Study

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ABSTRACT

BACKGROUND Right ventricular ejection fraction (RVEF) is a known predictor of adverse outcomes; however, its prognostic value diminishes in tricuspid regurgitation (TR).

OBJECTIVES This study aims to assess whether effective right ventricular ejection fraction (eRVEF) offers a more physiologic assessment of RV function and improves risk stratification in patients with TR.

METHODS The derivation cohort comprised 453 consecutive patients with at least moderate functional TR (regurgitant fraction ≥30% or volume ≥30 mL) on cardiac magnetic resonance (CMR). eRVEF was calculated as the ratio of forward volume to RV end-diastolic volume. The eRVEF threshold (≤25%) was derived based on all-cause mortality data. Clinical data were collected from standardized questionnaires at the time of CMR and supplemented with electronic health records; the primary outcome was all-cause mortality. External validation was performed in 2 independent cohorts, totaling 316 patients using identical inclusion criteria.

RESULTS In the derivation cohort, impaired eRVEF was associated with more advanced biventricular remodeling, worse biventricular function, and greater burden of late gadolinium enhancement ($P < 0.05$ for all), which was paralleled by higher TR volume and fraction (both $P < 0.05$). Over a median follow-up period of 2.7 years (Q1-Q3: 0.6-6.6 years), 20% of the patients died; mortality was higher in patients with impaired versus preserved eRVEF (28% vs 12%; HR: 1.72 [95% CI: 1.16-2.54]; $P = 0.007$). After adjusting for known TR risk markers including age, RV size, TR severity, conventional RVEF, and clinical markers of right-sided congestion, eRVEF remained independently predictive of mortality (HR: 0.49 [95% CI: 0.24-0.97]; $P = 0.042$). Adding eRVEF to a model inclusive of RVEF improved mortality prediction (chi-square from 30.6 to 37.0; $P = 0.011$) whereas adding RVEF to eRVEF did not (chi-square from 35.4 to 37.0; $P = 0.199$). External validation confirmed the prognostic significance of eRVEF ≤25% in both cohorts (HR: 2.66-2.86; both $P < 0.05$).

CONCLUSIONS eRVEF independently predicts mortality in TR and provides incremental prognostic value over conventional prognostic markers. (JACC Cardiovasc Imaging. 2026;19:810-821) © 2026 by the American College of Cardiology Foundation.

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Javier Sanz, MD, served as Guest Editor for this paper.

ISSN 1936-878X/$36.00 https://doi.org/10.1016/j.jcmg.2026.02.018

Tricuspid regurgitation (TR) is increasingly recognized as a significant public health concern, with advanced (≥ moderate) TR affecting >1.6 million people in the United States alone.[1] Advanced TR is associated with substantial morbidity, primarily caused by progressive right ventricular (RV) dysfunction and an increased risk of right heart failure.[2-5] Accurate assessment of RV function plays a key role in managing TR,[6] but conventional measures of RV function, including right ventricular ejection fraction (RVEF), are limited by their inability to differentiate between forward stroke volume and regurgitant flow. In severe TR, RVEF may appear normal despite significant impairment in forward cardiac output caused by the pseudo-normalization effect of regurgitant volume.[7,8] Consequently, although RVEF has been identified as an important predictor of adverse outcomes in TR,[3] its reliability is diminished in the presence of significant regurgitant volume, highlighting the need for alternative measures that more accurately reflect RV function in this setting.

To address this limitation, effective right ventricular ejection fraction (eRVEF), calculated as the ratio of forward stroke volume to RV end-diastolic volume, has emerged as a promising prognostic marker in TR.[3,8] Unlike conventional RVEF, eRVEF accounts for the volume of blood effectively ejected into the pulmonary circulation, thereby providing a more accurate reflection of RV function in the presence of significant regurgitant flow. Cardiac magnetic resonance (CMR), which accurately quantifies ventricular and regurgitant volumes,[9,10] is ideally suited to assess eRVEF due to its high precision in measuring forward stroke volume and differentiating it from regurgitant volume.

The need for improved RV functional assessment is particularly timely given the recent emergence of transcatheter therapies for TR. Recent clinical trials have demonstrated promising results with transcatheter edge-to-edge repair[11,12] and transcatheter tricuspid valve replacement,[13] offering lower-risk alternatives to surgical intervention for patients with severe TR. As these percutaneous interventions become more widely available, identifying patients who would derive the greatest benefit and determining the optimal timing for intervention have become increasingly important.

In this study, we aimed to: 1) establish a generalizable threshold for CMR-derived eRVEF to stratify mortality risk; 2) compare the relative prognostic utility of eRVEF and conventional RVEF; and 3) test whether eRVEF provides incremental risk prediction beyond established markers of adverse outcomes in TR, including conventional RVEF and TR severity.

METHODS

POPULATION. This multicenter study consisted of a derivation cohort and 2 external validation cohorts. The derivation cohort included 453 consecutive patients who underwent CMR at Weill Cornell Medicine, NewYork-Presbyterian Hospital between July 2005 and June 2024 and had at least moderate native tricuspid valve regurgitation (regurgitant volume ≥30 mL and/or fraction ≥30%) (**Figure 1**).[9,14] External validation was performed in 2 independent cohorts: patients from Houston Methodist DeBakey Heart and Vascular Center (n = 239) and Duke University Medical Center (n = 77), using identical inclusion criteria. For patients with multiple examinations across all sites, the initial CMR study demonstrating at least moderate TR was used for study purposes.

In all patients, CMR examination data were retrieved from institutional databases and reviewed by experienced investigators with >5 years of CMR experience. Patients with prior tricuspid valve repair or replacement and congenital or primary tricuspid pathologies (prolapse, rheumatic, leaflet perforation) were excluded. Clinical indices were attained via standardized questionnaires at time of CMR, supplemented by institutional database queries. The Institutional Review Boards at all participating sites provided approval for this study, including waiver of informed consent for the use of preexisting imaging and clinical data as analyzed for research purposes.

CMR. Image Acquisition. CMR was performed using 1.5-T or 3.0-T scanners. Cine-CMR imaging was performed using a steady-state free precession pulse sequence in contiguous left ventricular (LV) short-axis and long-axis orientations (2-, 3-, and 4-chamber views). Gadolinium-based contrast agents

ABBREVIATIONS AND ACRONYMS

CMR = cardiac magnetic resonance

eRVEF = effective right ventricular ejection fraction

LGE = late gadolinium enhancement

LV = left ventricular

LVEDV = left ventricular end-diastolic volume

RV = right ventricular

RVEDV = right ventricular end-diastolic volume

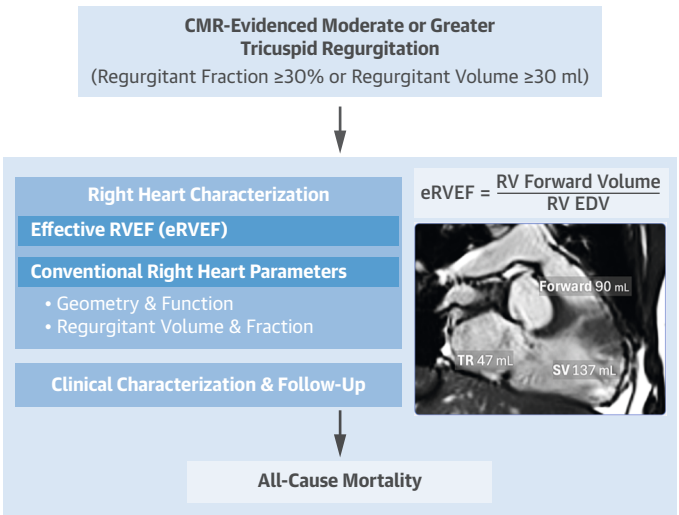
RVEF = right ventricular ejection fraction

TR = tricuspid regurgitation

The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the Author Center.

Manuscript received September 23, 2025; revised manuscript received February 3, 2026, accepted February 19, 2026.

FIGURE 1 Study Design



Overall study design illustrating study population, CMR analyses inclusive of eRVEF quantification, and clinical follow-up. CMR = cardiac magnetic resonance; EDV = end-diastolic volume; eRVEF = effective right ventricular ejection fraction; RV = right ventricular; RVEF = right ventricular ejection fraction; SV = stroke volume; TR = tricuspid regurgitation.

(total dose: 0.15-0.20 mmol/kg) were administered, and late gadolinium enhancement (LGE)-CMR imaging was performed ~10 minutes after contrast injection using orientations matched to cine-CMR.

Image Analysis. LV and RV chamber size and systolic function were quantified volumetrically using cine-CMR, with end-diastolic volume and end-systolic volume measured by endocardial border planimetry on contiguous short-axis images. Prior studies from our group demonstrated the high reproducibility of these methods for both LV and RV analyses.[15-17] In the derivation cohort, TR volume was calculated by subtracting the pulmonary artery forward flow from the RV stroke volume when phase-contrast imaging of the pulmonic valve was available (n = 279) (61.6%) (**Central Illustration**). If the appropriate data were unavailable and in the absence of significant mitral or aortic regurgitation, TR volume was computed by subtracting the LV stroke volume from the RV stroke volume (n = 174) (38.4%), in accordance with consensus guidelines and prior research.[9,18]

In the validation cohort, at Houston Methodist, tricuspid regurgitant volume was calculated as differential pulmonic forward and RV stroke volume in all patients, as previously reported.[19] At Duke University, all patients had TR quantified via differential RV-LV stroke volumes.[9] This methodological approach across sites allowed assessment of eRVEF robustness across different TR quantification approaches.

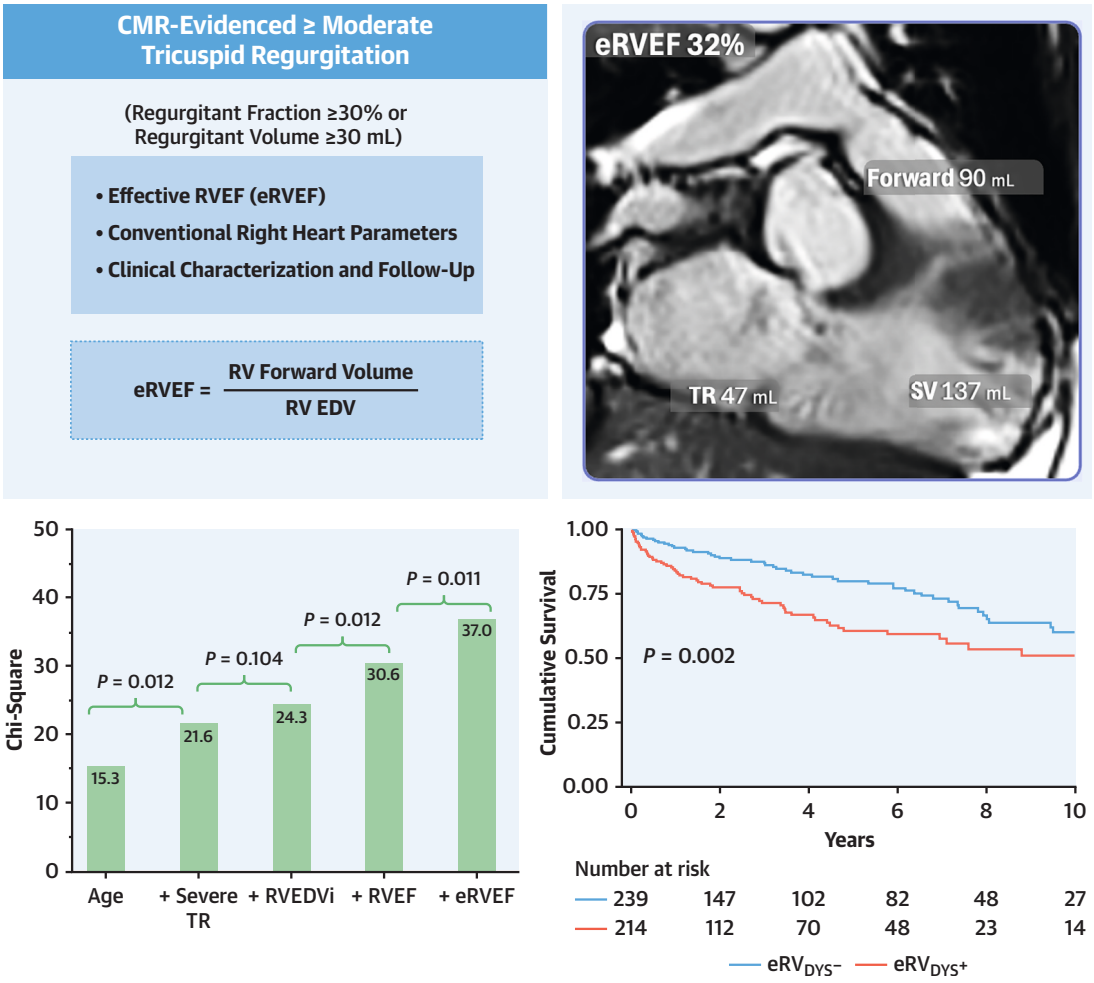
eRVEF was calculated as the ratio of RV forward stroke volume and right ventricular end-diastolic volume (RVEDV), where RV forward stroke volume accounts for the volume overload by excluding TR volume from the total RV volume.[20] LGE was evaluated using an established semiquantitative method.[21,22] The conventional RVEF threshold of 50% was used to define impaired RVEF in accordance with prior published reports.[22-24]

CLINICAL OUTCOMES. The primary outcome was all-cause mortality at follow-up. Mortality was ascertained via the Social Security Death Index and supplemented with electronic medical records. As established in the 2014 American College of Cardiology/American Heart Association Cardiovascular Endpoints Data Standards, heart failure hospitalization events were defined as any hospitalization with a primary diagnosis of heart failure where the patient has a length of stay of at least 24 hours, symptoms or objective evidence of new or worsening heart failure, and initiation or intensification of heart failure therapies as documented in the hospital course or discharge summary by the admitting physician.[25]

STATISTICAL ANALYSIS. Continuous variables are presented as mean ± SD whereas categorical data are expressed as frequencies and proportions. To assess the relationship between eRVEF and all-cause mortality, Cox proportional hazard models were constructed and adjusted for relevant covariates. The covariates were selected based on clinical rationale and prior studies demonstrating their prognostic relevance in TR.[17,19,24,26-29] The proportional hazard assumption for each model was tested with Schoenfeld residuals.[30] For each model, the incremental predictive power was assessed according to increments in the global chi-square values.

The optimal cutoff point for eRVEF was determined by using a receiver-operating characteristic analysis that was performed for all-cause mortality based on a univariable logistic regression model, with the threshold defined as the value that maximized the Youden index. The log rank test was used to compare outcomes between the groups. For external validation, the prespecified eRVEF was applied to both validation cohorts. Univariable analyses were then conducted within each cohort to determine whether the prognostic association between eRVEF ≤25% and mortality was reproducible in independent data sets. All analyses were performed using Stata 18 software (StataCorp LP).

CENTRAL ILLUSTRATION **eRVEF Is an Independent Marker of Increased Mortality Risk in TR**



Zhang RS, et al. JACC Cardiovasc Imaging. 2026;19(7):810–821.

Study population of 769 patients with moderate or greater TR undergoing comprehensive analysis including eRVEF quantification (top). Kaplan-Meier survival curve demonstrating impaired eRVEF (≤25%) to stratify all-cause mortality risk (bottom, right) and stepwise increase in global chi-square values demonstrating the incremental prognostic value of eRVEF (bottom, left). CMR = cardiac magnetic resonance; DYS = dysfunction; EDV = end-diastolic volume; eRVEF = effective right ventricular ejection fraction; RV = right ventricular; RVEDVi = right ventricular end-diastolic volume index; RVEF = right ventricular ejection fraction; SV = stroke volume; TR = tricuspid regurgitation.

RESULTS

DERIVATION COHORT. eRVEF Threshold. The derivation cohort included 453 patients with CMR-quantified moderate or greater TR: 282 patients (62.3%) with moderate TR and 171 (37.7%) severe TR. Concomitant left-sided cardiac disease was common, with moderate or greater mitral regurgitation present in 164 patients (36.2%), moderate or greater aortic regurgitation in 12 patients (2.6%), and moderate or greater aortic stenosis in 14 patients (3.1%).

TABLE 1 Clinical Characteristics

	Overall (N = 453)	eRVEF >25% (n = 239)	eRVEF ≤25% (n = 214)	*P* Value
Age, y	71.0 ± 15.7	72.6 ± 15.3	69.3 ± 16.0	**0.02**
Male	242 (53.4)	124 (51.9)	118 (55.1)	0.49
BSA, m^2	1.9 ± 0.3	1.9 ± 0.3	1.9 ± 0.3	0.45
Cardiovascular risk factors				
Diabetes mellitus	105 (23.3)	56 (23.5)	49 (23.0)	0.90
Hypertension	298 (65.8)	151 (63.2)	147 (68.7)	0.22
Hyperlipidemia	244 (54.1)	128 (53.8)	116 (54.5)	0.89
History of AF	188 (41.6)	96 (40.2)	92 (43.2)	0.51
Prior MI	85 (18.8)	42 (17.6)	43 (20.2)	0.48
COPD	50 (11.1)	33 (13.9)	17 (8.0)	**0.047**
Prior CVA/TIA	53 (11.8)	34 (14.3)	19 (8.9)	0.077
Cardiovascular medications				
Aspirin	179 (40.0)	95 (40.4)	84 (39.6)	0.86
Statin	214 (47.9)	117 (49.8)	97 (45.8)	0.39
Beta-blocker	258 (57.7)	144 (61.3)	114 (53.8)	0.11
ACEI/ARB	170 (38.0)	88 (37.4)	82 (38.7)	0.79
Aldosterone antagonist	89 (19.9)	39 (16.6)	50 (23.6)	0.07
Loop diuretic	191 (42.7)	100 (42.6)	91 (42.9)	0.94
SGLT2 inhibitor	15 (3.4)	7 (3.0)	8 (3.8)	0.63
Sacubitril-valsartan	21 (4.7)	10 (4.3)	11 (5.2)	0.64
Laboratory values				
Serum albumin, g/dL	3.5 ± 0.7	3.7 ± 0.7	3.3 ± 0.7	**<0.001**
Total bilirubin, mg/dL	1.0 ± 0.7	0.9 ± 0.5	1.2 ± 0.8	**<0.001**
AST, IU/L	45.4 ± 116.8	36.4 ± 67.1	54.5 ± 150.8	0.12
ALT, IU/L	49.1 ± 144.8	39.4 ± 84.7	58.9 ± 186.4	0.18
ALP, IU/L	106.9 ± 75.4	94.3 ± 61.4	119.4 ± 85.6	**<0.001**
eGFR, mL/min/1.73 m^2	69.4 ± 24.2	73.0 ± 22.3	65.8 ± 25.6	**0.003**
eGFR <30, mL/min/1.73 m^2	22 (5.5)	9 (4.5)	13 (6.5)	0.36
WBC, 10^9/L	7.3 ± 5.1	7.5 ± 6.5	7.0 ± 2.9	0.38
Hemoglobin, g/dL	12.5 ± 2.3	12.6 ± 2.2	12.4 ± 2.5	0.55
Platelet count, 10^9/L	213.3 ± 89.3	219.8 ± 92.7	206.8 ± 85.5	0.15

Values are n (%) or mean ± SD, unless otherwise indicated. **Bold** indicates statistical significance.

ACEI = angiotensin-converting enzyme inhibitor; AF = atrial fibrillation; ALP = alkaline phosphatase; ALT = alanine transaminase; ARB = angiotensin receptor blocker; AST = aspartate transaminase; BSA = body surface area; COPD = chronic obstructive pulmonary disease; CVA = cerebrovascular accident; eGFR = estimated glomerular filtration rate; eRVEF = effective right ventricular ejection fraction; MI = myocardial infarction; SGLT2 = sodium-glucose cotransporter 2; TIA = transient ischemic attack; WBC = white blood cell count.

To identify the optimal eRVEF threshold for predicting all-cause mortality, Harrell's C-statistic was performed. An eRVEF cutoff of ≤25% demonstrated the highest prognostic discrimination for all-cause mortality and was therefore selected for subsequent analyses. A total of 214 (47.2%) patients had an eRVEF ≤25%, significantly higher than that of impaired conventional RVEF (ie, RVEF < 50%) (36.4%).

As shown in Table 1, baseline characteristics were broadly similar between the groups except for lower serum albumin (3.3 ± 0.7 vs 3.7 ± 0.7 g/dL; $P < 0.001$), higher total bilirubin (1.2 ± 0.8 vs 0.9 ± 0.5 mg/dL; $P < 0.001$), and higher alkaline phosphatase (119.4 ± 85.6 vs 94.3 ± 61.4 IU/L; $P < 0.001$) in the eRVEF ≤ 25% group. Additionally, patients with impaired eRVEF had lower eGFR (65.8 ± 25.6 vs 73.0 ± 22.3 mL/min/1.73 m^2; $P = 0.003$). No significant differences were observed in age, sex, body surface area, or other cardiovascular risk factors, including diabetes, hypertension, or prior myocardial infarction.

A total of 32 patients (7.1%) underwent tricuspid valve intervention after CMR, including 24 (75%) who had surgical tricuspid repair, 3 (9.4%) who underwent transcatheter repair, and 5 (15.6%) who had surgical valve replacement. There were no differences in the frequency of tricuspid valve intervention after CMR between the 2 groups (7.5% vs 6.8%; $P = 0.81$). Finally, a total of 21% of the patients (n = 94) had prior left-sided valve surgery.

eRVEF and Chamber Remodeling. Impaired eRVEF (≤25%) was associated with more advanced

TABLE 2 Imaging Characteristics

	Overall (N = 453)	eRVEF >25% (n = 239)	eRVEF ≤25% (n = 214)	*P* Value
LV geometry/function				
LVEDV index, mL/m^2	87.7 ± 39.2	80.5 ± 28.7	95.8 ± 47.0	**<0.001**
LVESV index, mL/m^2	50.5 ± 38.2	37.5 ± 23.9	65.0 ± 45.3	**<0.001**
LV dilation[a]	127 (28.0)	45 (18.8)	82 (38.3)	**<0.001**
LV mass, g/m^2	70.3 ± 25.6	68.0 ± 23.9	72.9 ± 27.1	**0.048**
LVEF, %	48.0 ± 18.8	56.2 ± 14.8	38.9 ± 18.7	**<0.001**
LV dysfunction, <50%	213 (47.0)	66 (27.6)	147 (68.7)	**<0.001**
LGE, % LV	4.8 ± 8.8	3.4 ± 7.2	6.4 ± 10.1	**<0.001**
RV geometry/function				
RVEDV index, mL/m^2	118.0 ± 39.5	103.3 ± 30.4	134.4 ± 41.9	**<0.001**
RVESV index, mL/m^2	67.7 ± 32.9	49.6 ± 20.5	87.8 ± 32.4	**<0.001**
RV dilation[b]	272 (60.0)	103 (43.1)	169 (79.0)	**<0.001**
RVEF, %	44.8 ± 12.3	53.1 ± 8.2	35.5 ± 9.1	**<0.001**
RV dysfunction, RVEF <50%	165 (36.4)	85 (35.6)	203 (94.9)	**<0.001**
eRVEF, %	26.0 ± 9.5	33.4 ± 5.8	17.7 ± 4.6	**<0.001**
Tricuspid regurgitation				
Regurgitant volume, mL	39.7 ± 18.0	36.3 ± 11.3	43.4 ± 22.8	**<0.001**
Regurgitant fraction, %	42.7 ± 10.9	36.9 ± 7.2	49.0 ± 10.8	**<0.001**
Regurgitant fraction, >50%[21]	118 (26.0)	16 (6.7)	102 (47.7)	**<0.001**
Regurgitant volume, ≥45 mL[21]	120 (26.8)	52 (22.0)	68 (32.1)	**0.012**
Atrial geometry				
Right atrial area, cm^2	31.2 ± 11.1	29.6 ± 10.0	33.0 ± 12.1	**0.001**
Left atrial area, cm^2	29.0 ± 11.5	28.5 ± 12.0	29.5 ± 10.9	0.35

Values are n (%) or mean ± SD, unless otherwise indicated. **Bold** indicates statistical significance. [a]LVEDV index >108 mL/m^2 for men; LVEDV index >96 mL/m^2 for women.[37] [b]RVEDV index >109 mL/m^2 for men; RVEDV index >97 mL/m^2 for women.[37]

LGE = late gadolinium enhancement; LV = left ventricular; LVEDV = left ventricular end-diastolic volume; LVEF = left ventricular ejection fraction; RV = right ventricular; RVEDV = right ventricular end-diastolic volume; RVEF = right ventricular ejection fraction.

biventricular remodeling (Table 2). Patients with impaired eRVEF had significantly larger left ventricular end-diastolic volume (LVEDV) index (95.8 ± 47.0 vs 80.5 ± 28.7 mL/m^2; $P < 0.001$), a higher prevalence of LV dilation (38.3% vs 18.8%; $P < 0.001$), lower left ventricular ejection fraction (38.9 ± 18.7% vs 56.2 ± 14.8%; $P < 0.001$), and greater extent of LGE (6.4 ± 10.1% vs 3.4 ± 7.2%; $P < 0.001$). Consistent with this pattern, greater remodeling was also observed for the RV, with larger RVEDV index (134.4 ± 41.9 vs 103.3 ± 30.4 mL/m^2; $P < 0.001$), higher rates of RV dilation (79.0% vs 43.1%; $P < 0.001$), and lower RVEF (35.5 ± 9.1% vs 53.1 ± 8.2%; $P < 0.001$).

TR severity was also greater in patients with eRVEF ≤25%, with significantly higher regurgitant volume (43.4 ± 22.8 vs 36.3 ± 11.3 mL; $P < 0.001$) and regurgitant fraction (49.0 ± 10.8% vs 36.9 ± 7.2%; $P < 0.001$) among those with impaired eRVEF. We found that eRVEF correlated strongly and inversely with TR severity ($r = -0.67$; $P < 0.001$) whereas conventional RVEF showed only a weak inverse correlation ($r = -0.22$; $P < 0.001$), highlighting the limited sensitivity of RVEF in reflecting TR burden. There was a strong correlation between conventional RVEF and eRVEF ($r = 0.86$; $P < 0.001$).

eRVEF and Mortality. The median follow-up time was 2.7 years (Q1-Q3: 0.6-6.6 years). At 3 years, the all-cause mortality was 20%, with significantly higher mortality among patients with impaired eRVEF compared with those with preserved eRVEF (28% vs 12%; $P < 0.001$). In univariable analysis, eRVEF was significantly associated with all-cause mortality (HR: 0.70 [95% CI: 0.58-0.85]; $P < 0.001$) (Table 3). When dichotomized, the patients with impaired eRVEF had a nearly 2-fold increased risk of mortality (HR: 1.72 [95% CI: 1.16-2.54]; $P = 0.007$) (Figure 2). Conventional RVEF also showed a significant, though weaker, association with mortality (HR: 0.98 [95% CI: 0.97-1.00]; $P = 0.013$), among several other imaging and clinical parameters (Table 3).

To assess the independent prognostic value of eRVEF, multivariable Cox regression analysis was performed; eRVEF remained a strong independent predictor of all-cause mortality (HR: 0.49 [95% CI: 0.24-0.97]; $P = 0.042$ [every 10% increase]), even after adjusting for established prognostic markers in

TABLE 3 Univariable and Multivariable Cox Proportional Hazards Analysis of Predictors for All-Cause Mortality

	Univariable Analysis		Multivariable Analysis	
	HR (95% CI)	*P* Value	HR (95% CI)	*P* Value
Demographics and clinical variables				
Age, >70 y	1.91 (1.30-2.80)	**0.001**	1.92 (1.25-2.95)	**0.003**
Male	0.82 (0.58-1.17)	0.28	0.80 (0.54-1.18)	0.26
Diabetes	0.91 (0.59-1.38)	0.65		
Hyperlipidemia	0.94 (0.65-1.37)	0.72		
Chronic kidney disease	1.09 (0.58-2.04)	0.77		
History of atrial fibrillation	0.98 (0.67-1.43)	0.93		
Prior myocardial infarction	1.05 (0.60-1.84)	0.07		
History of CVA	1.05 (0.60-1.84)	0.85		
Imaging parameters				
RV geometry and function				
RVEF, %[a]	0.98 (0.97-1.00)	**0.013**	1.39 (0.86-2.24)	0.18
RV dysfunction, EF <50%	1.54 (1.04-2.26)	**0.028**		
RVEDV index, mL/m^2	1.00 (0.99-1.01)	0.29	1.00 (0.99-1.01)	0.31
eRVEF, %[a]	0.70 (0.58-0.85)	**<0.001**	0.49 (0.24-0.97)	**0.042**
eRVEF, ≤25%	1.72 (1.16-2.54)	**0.007**		
TR regurgitant fraction, >50%	1.53 (1.04-2.27)	**0.03**	1.03 (0.44-1.80)	0.75
LV geometry and function				
LVEF, %	0.99 (0.98-1.00)	0.44		
LVEDV index, mL/m^2	0.99 (0.99-1.00)	0.08		
LV mass, g/m^2	1.00 (0.99-1.00)	0.50		
Laboratory parameters				
Albumin, g/dL	0.49 (0.38-0.63)	**<0.001**	0.63 (0.46-0.87)	**0.005**
Total bilirubin, mg/dL	1.02 (0.77-1.37)	0.87	0.88 (0.65-1.19)	0.41
eGFR <30, mL/min/1.73 m^2	3.79 (1.96-7.32)	**<0.001**	2.62 (1.26-5.43)	**0.010**
Hemoglobin, g/dL	0.81 (0.75-0.88)	**<0.001**	0.92 (0.83-1.02)	0.12
Platelet count, 10^9/L	1.00 (0.99-1.01)	0.05	1.00 (0.99-1.01)	0.58

Bold indicates statistical significance. [a]For every 10% increase.
CVA = cerebral vascular accident; TR = tricuspid regurgitation; other abbreviations as in Tables 1 and 2.

TR including age, RVEDV index, RVEF, TR severity, and other clinical markers of right sided congestion (GFR <30 mL/min/1.73 m^2, albumin, total bilirubin). The incremental prognostic value of eRVEF was further demonstrated through sequential improvements in model performance, illustrated by the rising chi-square values with each added prognostic factor (Figure 3).

Starting from a baseline model including age (chi-square = 15.3), the addition of TR severity (regurgitant fraction ≥50%) significantly improved the model, increasing the chi-square to 21.6 ($P = 0.012$). Subsequent incorporation of RVEDV index yielded further incremental improvement, raising the chi-square to 24.3 ($P = 0.10$). Importantly, the addition of eRVEF improved the prognostic performance of the model, increasing the chi-square to 35.4 ($P = 0.001$). However, further addition of conventional RVEF did not provide a significant incremental benefit, with only a modest increase in chi-square to 37.0 ($P = 0.20$). Conversely, when conventional RVEF was introduced before eRVEF (Figure 3A), eRVEF improved the model's performance (chi-square increase from 30.6 to 37.0; $P = 0.011$) over RVEF.

To evaluate whether temporal changes in CMR acquisition influenced outcomes, the patients were stratified by decade of CMR examination. Decade of imaging was not associated with mortality (HR: 1.04 [95% CI: 0.75-1.44]; $P = 0.79$), indicating no significant temporal heterogeneity in the prognostic performance of eRVEF.

Collectively, these findings demonstrate that eRVEF provides independent and incremental prognostic value beyond established parameters whereas conventional RVEF offers no significant incremental value once eRVEF is included.

Survival Stratification by eRVEF and RVEF. Kaplan-Meier survival analysis, stratified by RV chamber dilation and impaired eRVEF (≤25%), demonstrated a stepwise increase in mortality risk across the 4 predefined patient groups (log-rank $P = 0.001$) (Figure 4A). Patients with both RV dilation and

impaired eRVEF had the highest mortality risk. Conversely, patients with normal RV size and preserved eRVEF demonstrated the most favorable survival outcomes. Patients with isolated RV dilation but preserved eRVEF and those with impaired eRVEF but normal RV size had intermediate mortality risk, emphasizing the additive prognostic value of impaired eRVEF.

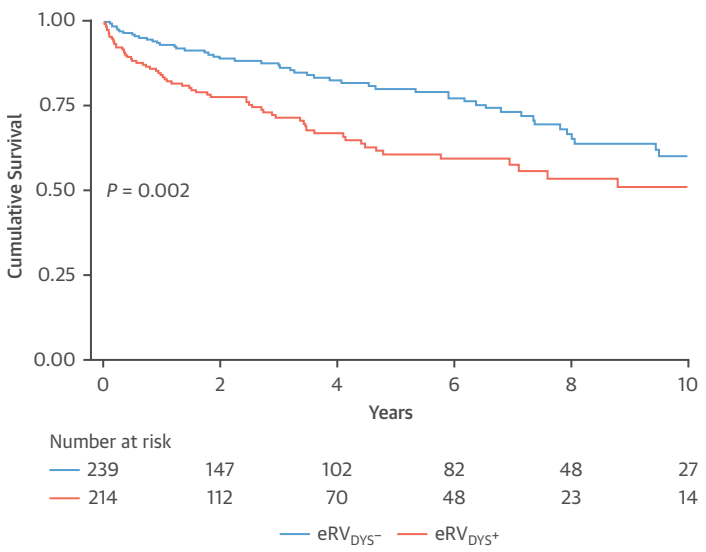
Similarly, Kaplan-Meier analysis stratified by the presence or absence of RV dysfunction (RVEF <50%) and impaired eRVEF (≤25%) revealed a clear stepwise increase in mortality risk (log-rank $P = 0.02$) (Figure 4B). As shown, patients with both RV dysfunction and impaired eRVEF had the highest mortality whereas those with preserved RVEF and preserved eRVEF demonstrated the best outcomes. Patients with isolated RV dysfunction but preserved eRVEF and those with preserved RVEF but impaired eRVEF had intermediate survival, further highlighting the incremental prognostic significance of eRVEF beyond conventional RV function and structural parameters.

eRVEF and Heart Failure Hospitalizations. Heart failure hospitalizations occurred in 21% of the cohort at 3 years, with significantly higher rates in patients with eRVEF ≤25% compared with those with eRVEF >25% (32.6% vs 10.5%; HR: 2.78 [95% CI: 1.86-4.15]; $P < 0.001$). In a multivariable analysis adjusting for the same variables for mortality, eRVEF was a strong predictor of HF hospitalizations (HR: 0.43 [95% CI: 0.21-0.89]; $P = 0.02$, every 10% increase) (Supplemental Table 1).

VALIDATION COHORT. The Houston Methodist validation cohort included 239 patients with moderate or greater TR (age 59.3 ± 14.7 years; TR fraction 41.9 ± 11.0%; eRVEF 25.5 ± 10.6%). Using the prespecified eRVEF threshold of ≤25%, 120 patients (50.0%) were classified as having impaired eRVEF. As shown in Supplemental Table 2, 24% of the patients died at 3 years. Three-year mortality was significantly higher with impaired eRVEF (≤25%) compared with those with preserved eRVEF (46% vs 12%; $P < 0.001$). During a median follow-up of 2.6 years (Q1-Q3: 1.7-4.3 years), an impaired eRVEF was associated with a significantly increased risk of mortality (HR: 2.66 [95% CI: 1.53-4.61]; $P < 0.001$).

The Duke University validation cohort comprised 77 patients (age 62.5 ± 16.4 years; TR fraction 39.7 ± 9.5%; eRVEF 48.5 ± 14.7%), among whom 37 patients (48.1%) had impaired eRVEF (≤25%). At 3 years, the overall mortality of the cohort was 17.7%, and mortality was significantly higher in patients with impaired versus preserved eRVEF (27.5% vs 11.8%; $P < 0.001$). During a follow-up period of 3.1 years (Q1-Q3: 1.8-4.8 years), an impaired eRVEF was similarly associated with an increased risk of mortality (HR: 2.86 [95% CI: 1.03-7.89]; $P = 0.04$).

FIGURE 2 Kaplan-Meier Survival Curve Stratified by eRVEF

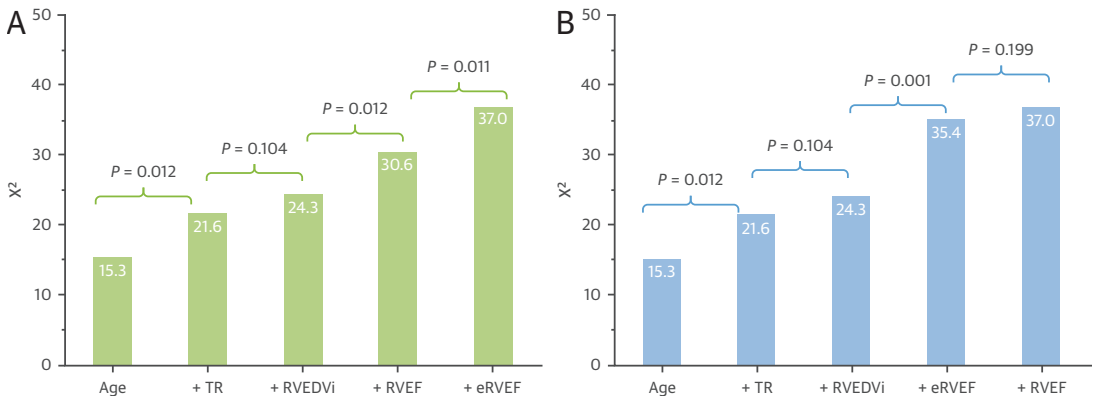


Kaplan-Meier survival curves comparing patients with vs without impaired eRVEF ($eRV_{DYS}+$ vs $eRV_{DYS}–$), demonstrating lower survival in the impaired eRVEF group. DYS = dysfunction; other abbreviations as in Figure 1.

DISCUSSION

In this multicenter study of 769 patients with advanced TR, we demonstrate that CMR-derived eRVEF is a robust, independent predictor of all-cause mortality. To our knowledge, this is the largest CMR-based cohort to date evaluating the prognostic value of eRVEF. A threshold of eRVEF ≤25% identified patients with nearly a 2-fold increased risk of death, likely representing a critical point in disease progression where compensatory mechanisms are exhausted and intrinsic RV contractile dysfunction can be identified. Importantly, eRVEF added incremental prognostic value beyond established clinical markers, including age, TR severity, and conventional RVEF, highlighting its ability to comprehensively risk stratify this population. The consistency of results across 3 centers with different patient populations, imaging protocols, and health care systems strengthens the evidence for eRVEF as a reliable prognostic marker. Importantly,

FIGURE 3 Incremental Prognostic Value of eRVEF Over Conventional RVEF for All-Cause Mortality



(A) Stepwise increases in global chi-square values for mortality prediction demonstrating eRVEF confer prognostic information beyond RVEF. (B) Conversely, introducing eRVEF first eliminates additional prognostic utility from RVEF, highlighting eRVEF's unique clinical value. RVEDVi = right ventricular end-diastolic volume index; other abbreviations as in Figure 1.

the prognostic value of eRVEF was maintained regardless of TR quantification methodology, with validation demonstrated across both phase-contrast imaging and differential stroke volume approaches, supporting the clinical robustness of this finding.

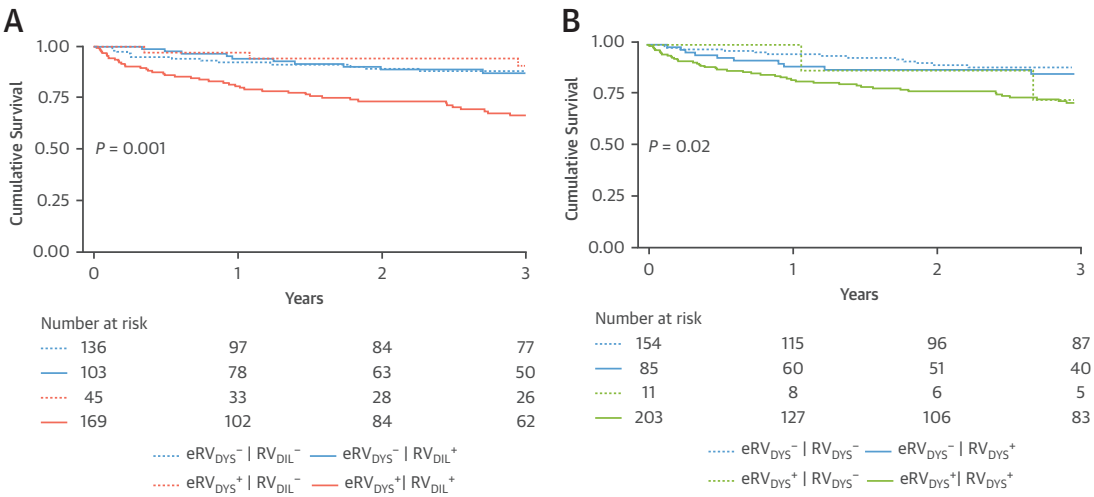
Our findings build upon the prior work by Hinojar et al[31] that first identified an association between CMR-evidenced reduced eRVEF and adverse outcomes in a cohort of 75 patients with severe TR. However, the small sample size limited the statistical power and precluded adjustment for confounders. By contrast, our larger cohort enabled robust multivariable analysis, confirming eRVEF as an independent prognostic marker and establishing a prognostic threshold (≤25%) for risk stratification.

Furthermore, we extend insights from Stolz et al[32] who recently demonstrated biphasic right ventricular reverse remodeling after transcatheter edge-to-edge repair using 3-dimensional echocardiography. Although Stolz et al[32] observed 3-dimensional echocardiography-derived eRVEF improvements after the procedure, they did not link these changes to survival or clinical endpoints. Nonetheless, their study offers valuable data on longitudinal changes in eRVEF, suggesting that eRVEF effectively captures meaningful shifts in right ventricular function. By contrast, conventional RVEF remained largely static in their cohort, reflecting its limitations in distinguishing hemodynamic unloading (ie, reduced regurgitant volume) from functional recovery. Consistent with this physiologic limitation, our analysis revealed only a weak association between TR severity and RVEF, highlighting RVEF's limited utility in tracking disease progression and identifying high-risk patients. Collectively, the findings from Stolz et al[32] and our present study reinforce eRVEF's prognostic importance and clinical relevance by unmasking pseudo-normalized RVEF and identifying high-risk patients.

It is important to recognize that eRVEF should be interpreted as a comprehensive, flow-based marker that integrates multiple physiologic components rather than a pure measure of myocardial contractility. Specifically, eRVEF incorporates intrinsic RV myocardial performance, ventricular preload conditions, pulmonary vascular afterload, and the hemodynamic burden of TR. Unlike conventional RVEF, which can be pseudo-normalized by large regurgitant volumes, eRVEF specifically quantifies the proportion of ventricular stroke volume that contributes to forward cardiac output, making it a more clinically relevant measure of effective cardiac performance in the setting of significant regurgitation.

Building on this, we show patients with impaired eRVEF to demonstrate more advanced biventricular remodeling, with increased chamber volumes, reduced EF, and higher burden of LGE. These findings are consistent with the known systemic effect of TR, where chronic RV volume overload impairs LV filling through interventricular dependence and

FIGURE 4 Kaplan-Meier Survival Curves Stratified by eRVEF and Conventional RV Parameters



Kaplan-Meier curves demonstrating cumulative survival stratified by conventional and effective RV parameters. (A) This Kaplan-Meier curve similarly illustrates that eRVEF impairment has stronger prognostic implications than RV dilation alone, with combined abnormalities conferring the highest risk. (B) This Kaplan-Meier curve shows that isolated eRVEF impairment ($eRV_{DYS}+/RV_{DYS}-$) carries greater mortality risk than isolated conventional RVEF impairment ($eRV_{DYS}-/RV_{DYS}+$), with the worst outcomes observed when both parameters are abnormal. DIL = dilation; other abbreviations as in Figures 1 and 2.

pericardial constraint, which can impact both left and right heart function.[33,34] These findings position eRVEF as a comprehensive marker, integrating both valvular regurgitation burden and associated biventricular remodeling. Finally, CMR offers precise quantification of regurgitant volume,[19] biventricular volumes,[35,36] and forward flow,[19] positioning it as the reference standard for quantifying eRVEF. The robustness of eRVEF across different TR quantification approaches, both phase-contrast and differential stroke volume methods, further supports its clinical utility and broad applicability in diverse imaging protocols.

STUDY LIMITATIONS. First, the retrospective study design limits assessment of how changes in medical therapy or subsequent interventions during follow-up could have influenced outcomes. However, we stratified patients by decade of CMR acquisition and found no association between imaging period and mortality, suggesting that temporal variation in imaging techniques or clinical practice did not influence the prognostic performance of eRVEF. Second, although we validated our findings in 2 external cohorts, larger prospective multicenter studies would further establish the clinical utility of eRVEF. Third, the retrospective design precluded systematic correlation of CMR-derived TR parameters with concurrent echocardiographic assessments, limiting our ability to categorize patients according to established echocardiographic TR severity grades.

Fourth, TR quantification methods varied across cohorts, with the derivation cohort using phase-contrast imaging or differential stroke volumes while Houston Methodist used phase-contrast imaging exclusively and Duke University used differential stroke volumes exclusively. Despite these methodological differences, eRVEF ≤25% demonstrated remarkably consistent prognostic associations across all cohorts (HR: 1.72-2.86), supporting the reliability and generalizability of eRVEF threshold across TR quantification approaches.

Finally, our cohort included patients with functional TR from multiple etiologies. Detailed stratification of the association between eRVEF and outcomes by specific TR etiology was limited by overlapping mechanisms and small subgroup sizes. Future studies are needed to delineate the prognostic impact of eRVEF across distinct TR etiologies and to

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explore its association with other clinically relevant endpoints, such as cardiovascular mortality and quality of life, to further establish its utility in routine clinical decision-making.

CONCLUSIONS

In this large multicenter study with external validation, by integrating TR severity, intrinsic RV function, and chamber remodeling, eRVEF demonstrates superior prognostic value compared to conventional TR risk markers. An eRVEF threshold of ≤25% identifies patients at increased risk of mortality and may help guide clinical decision-making regarding the timing and appropriateness of intervention. The prognostic value of eRVEF was maintained across TR quantification methodology, with validation cohorts demonstrating consistent prognostic value across both phase-contrast imaging and differential stroke volume approaches, supporting the clinical robustness of this finding. Future prospective studies are needed to determine whether eRVEF-guided therapeutic strategies improve outcomes in this high-risk population and whether tricuspid valve interventions can favorably modify eRVEF and associated mortality risk.

ACKNOWLEDGMENTS The authors thank Lily Jin, BS, and Giorgia Falco, MD, for their administrative and technical support.

FUNDING SUPPORT AND AUTHOR DISCLOSURES

This study was supported by funding from the National Institutes of Health/National Heart, Lung, and Blood National Institutes of Health/NHLBI K23HL140092 (to Dr J. Kim). Dr Khalique has served as a consultant to Siemens, Edwards, Philips, and Abbott. All other authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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PERSPECTIVES

COMPETENCY IN MEDICAL KNOWLEDGE: Comprehensive assessment of RV function in tricuspid regurgitation is essential for accurate risk stratification. Conventional RVEF may appear preserved in the setting of advanced tricuspid regurgitation because it reflects total stroke volume, including regurgitant volume, rather than true forward flow. Echocardiographic parameters, including tricuspid annular plane systolic excursion, are also limited in this setting because they primarily assess longitudinal function and not global RV performance. This study demonstrates that eRVEF, which quantifies the proportion of stroke volume contributing to forward pulmonary flow, provides a more physiologic and clinically meaningful marker of RV performance. Incorporating eRVEF into routine CMR interpretation allows for a more nuanced and accurate measure of RV function, thereby improving prognostic assessment and informing potential management strategies, which are of particular interest given the rapidly evolving landscape of transcatheter tricuspid therapies.

TRANSLATIONAL OUTLOOK: Accurate evaluation of RV performance in tricuspid regurgitation remains a major unmet need, particularly as transcatheter tricuspid interventions rapidly expand. This study demonstrates that eRVEF, a physiologic marker of forward stroke volume, provides incremental prognostic value compared with conventional measures. Before these findings can be incorporated into routine clinical practice, additional prospective studies are needed to clarify how eRVEF should inform patient selection and timing of emerging tricuspid regurgitation therapies and to determine whether improvements in eRVEF after intervention translate into meaningful clinical benefit.

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KEY WORDS ejection fraction, magnetic resonance imaging, right ventricle, tricuspid regurgitation

APPENDIX For supplemental tables, please see the online version of this paper.