JACC: CARDIOVASCULAR INTERVENTIONS
VOL. 19, NO. 6, 2026
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PUBLISHED BY ELSEVIER

FOCUS ON TRICUSPID INTERVENTIONS

ORIGINAL RESEARCH: STRUCTURAL

Management and Outcome of Failed Transcatheter Tricuspid Edge-to-Edge Repair

Insights From the FATE International Registry

Federico De Marco, MD, PhD,[a] Gianmaria Calamita, MD,[b] Valentina Ardizzone, MD,[a] Mirjam Gauri Winkel, MD,[c] Mohammad Kassar, MD,[c] Fabien Praz, MD,[c] Nicolas M. Van Mieghem, MD,[d] Joris F.W. Ooms, MD,[d] Muhammed Gerçek, MD,[e,f] Felix Rudolph, MD,[e,f] Marianna Adamo, MD,[g,h] Augustin Coisne, MD,[i] Jérémie Arlanda, MD,[i] Philipp Lurz, MD,[j] Rodrigo Estévez-Loureiro, MD,[k] Neil P. Fam, MD,[l] Joachim Schofer, MD,[m] Christina Brinkmann, MD,[m] Marina Urena, MD,[n] Gregory Ducrocq, MD,[n] Jan-Malte Sinning, MD,[o] Tobias Geisler, MD,[p] Alessia Azzano, MD,[q] Elvis Brscic, MD,[q] Antonella Millin, MD,[r] Azeem Latib, MD,[r] Maurizio Taramasso, MD,[s,t] Antonio Popolo Rubbio, MD,[u] Francesco Bedogni, MD,[u] Stephanie Brunner, MD,[v] Stefan Toggweiler, MD,[v] Martin J. Swaans, MD,[w] Leo Timmers, MD,[w] Francesco Giannini, MD,[x] Alfonso Ielasi, MD,[x] Omar Oliva, MD,[y] Nicolas Dumonteil, MD,[y] Didier Tchetchè, MD,[y] Dabit Arzamendi, MD,[z] Giulia Maisero, MD,[aa] Giuseppe Tarantini, MD,[aa] Gabriele Crimi, MD,[bb] Italo Porto, MD,[bb] Fausto Castriota, MD,[cc] Luca Ferri, MD,[dd] Matteo Montorfano, MD,[dd] Alessandro Sticchi, MD, PhD,[ee] Maria Lopez-Laporte, MD,[ff] David Messika-Zeitoun, MD,[ff] Marino Labinaz, MD,[ff] Peter Lüdike, MD,[gg] Horst Sievert, MD,[hh] Giulia Antonelli, MD,[b] Fabio Fazzari, MD,[a] Cristina Ferrari, MD,[a] Antonio Mangieri, MD[b]

ABSTRACT

BACKGROUND Tricuspid regurgitation (TR) is linked to significant morbidity and mortality. Tricuspid transcatheter edge-to-edge repair (T-TEER) provides a less invasive option for high-risk patients, but real-world data on device failure mechanisms and outcomes remain limited.

OBJECTIVES The aim of this study was to investigate the incidence, management, and outcomes of T-TEER device-related failures due to loss of leaflet insertion, single-leaflet device attachment, or device embolization, offering insights into these adverse events.

METHODS The retrospective, multicenter FATE (Failed Tricuspid Transcatheter Edge-to-Edge) registry identified all device-related failures associated with moderate or greater residual or recurrent TR among 2,278 consecutive T-TEER procedures performed at 31 centers from 2017 to 2024. Failure mechanisms were classified by multiparametric echocardiography. Coprimary endpoints were the incidence and management of device-related failure and the composite of death or heart failure rehospitalization.

RESULTS Among 2,278 T-TEER procedures, 123 device-related failures (5.4%) were identified (loss of leaflet insertion, 24%; single-leaflet device attachment, 75%; embolization, 1%), mostly diagnosed early and associated with severe or greater TR in 73%. Management was medical therapy in 54% and reintervention in 46%. Reintervention achieved greater TR reduction and right atrial reverse remodeling than medical therapy, but over a median follow-up period of 255 days, the composite of death or heart failure rehospitalization remained frequent (38.2%) and did not differ between strategies. Higher TRI-SCORE, acute kidney injury, and greater discharge TR severity independently predicted adverse outcomes.

CONCLUSIONS Device-related failure after T-TEER is uncommon but clinically relevant. Reintervention achieves greater TR reduction and right atrial reverse remodeling than medical therapy, but events remain similar between both strategies. (JACC Cardiovasc Interv. 2026;19:726-736) © 2026 by the American College of Cardiology Foundation.

ISSN 1936-8798/$36.00
https://doi.org/10.1016/j.jcin.2026.01.285

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Severe tricuspid regurgitation (TR) presents a significant clinical challenge, being associated with considerable morbidity and mortality. It affects approximately 3% to 6% of the general population, rising to 10% to 23% in patients with left-sided valve disease and heart failure (HF).[1] Severe isolated TR is linked to a 5-year mortality or HF hospitalization rate of 40%.[2] Secondary TR, which accounts for 85% of all TR cases, is the most common form. Patients with atrial secondary TR tend to have a better prognosis compared with those with ventricular secondary TR.[3] Transcatheter tricuspid valve interventions (TTVIs) have emerged as an effective therapeutic option for patients with TR who are at high surgical risk.[4]

Tricuspid transcatheter edge-to-edge repair (T-TEER) is a safe, effective, and minimally invasive treatment option for severe TR in patients at high surgical risk.[5-8] The TRILUMINATE (TRILUMINATE Study With Abbott Transcatheter Clip Repair System in Patients With Moderate or Greater TR), CLASP TR (Edwards PASCAL Transcatheter Valve Repair System in Tricuspid Regurgitation Early Feasibility Study), and Tri.Fr (Multicentric Randomized Evaluation of Tricuspid Valve Percutaneous Repair System [Clip for the Tricuspid Valve] in the Treatment of Severe Secondary Tricuspid Disorders) studies demonstrated the safety and efficacy of both the TriClip (Abbott Cardiovascular) and PASCAL (Edwards Lifesciences) devices, showing sustained reductions in TR severity to moderate or less at 1- and 3-year follow-up.[9] These studies also reported significant reductions of HF symptoms, as assessed by NYHA functional class, and in quality of life, evaluated using the Kansas City Cardiomyopathy Questionnaire, at 3 years compared with baseline and with patients treated with optimal medical therapy (MT).

However, residual or recurrent TR following transcatheter edge-to-edge repair remains a clinical concern. A substantial proportion of patients experience significant TR recurrence after the procedure, often due to progression of the underlying disease or device-related leaflet issues such as loss of leaflet insertion (LLI) or single-leaflet device attachment (SLDA), reported in 7% of cases in the TRILUMINATE study, 4.6% in the CLASP TR trial, and 3.8% in the bRIGHT (An Observational Real-World Study Evaluating Severe Tricuspid Regurgitation Patients Treated With the Abbott TriClip™ Device) registry.[10]

These complications may lead to both acute and delayed TR recurrence, potentially affecting long-term clinical outcomes. Currently, data on the incidence and clinical impact of these postprocedural complications are limited. The aim of this study was

ABBREVIATIONS AND ACRONYMS

2D = 2-dimensional

3D = 3-dimensional

HF = heart failure

LLI = loss of leaflet insertion

MT = medical therapy

RAVi = right atrial volume index

SLDA = single-leaflet device attachment

TEE = transesophageal echocardiography

TR = tricuspid regurgitation

TTE = transthoracic echocardiography

T-TEER = tricuspid transcatheter edge-to-edge repair

TTVI = transcatheter tricuspid valve intervention

From [a]Centro Cardiologico Monzino IRCCS, Milan, Italy; [b]IRCCS Humanitas Research Hospital, Rozzano, Milan, Italy; [c]Department of Cardiology, Inselspital, Bern University Hospital, University of Bern, Bern, Switzerland; [d]Department of Cardiology, Thoraxcenter, Erasmus University Medical Center, Rotterdam, the Netherlands; [e]Clinic for General and Interventional Cardiology/Angiology, Herz- und Diabeteszentrum Nordrhein-Westfalen, Ruhr-Universität Bochum, Bad Oeynhausen, Germany; [f]Herz- und Diabeteszentrum Nordrhein-Westfalen, Medizinische Fakultät OWL (Universität Bielefeld), Bad Oeynhausen, Germany; [g]University of Brescia, Brescia, Italy; [h]Department of Cardiology, ASST Spedali Civili, Brescia, Italy; [i]University Lille, Inserm, CHU Lille, Institut Pasteur de Lille, Lille, France; [j]Department of Cardiology, Universitätsmedizin Johannes Gutenberg-University, Mainz, Germany; [k]Department of Interventional Cardiology, University Hospital Alvaro Cunqueiro, Vigo, Spain; [l]St. Michael's Hospital, University of Toronto, Toronto, Ontario, Canada; [m]MVZ Department of Structural Heart Disease at St. Georg, Hamburg, Germany; [n]Assistance Publique-Hôpitaux de Paris, Bichat Hospital, Paris, France; [o]Department of Cardiology, St. Vincent Hospital Cologne, Cologne, Germany; [p]Department of Cardiology and Angiology, Eberhard Karls University of Tübingen, Tübingen, Germany; [q]Cardiovascular Department Humanitas Gavazzeni Hospital, Bergamo, Italy; [r]Montefiore Einstein Center for Heart and Vascular Care, Montefiore Medical Center, New York, New York, USA; [s]Herzzentrum Hirslanden Zürich, Zürich, Switzerland; [t]University of Zürich, Zürich, Switzerland; [u]IRCCS Policlinico San Donato, Milan, Italy; [v]Heart Center Lucerne, Luzerner Kantonsspital, Lucerne, Switzerland; [w]Department of Cardiology, St. Antonius Hospital, Nieuwegein, the Netherlands; [x]Department of Interventional Cardiology, IRCCS Galeazzi Sant'Ambrogio Hospital, Milan, Italy; [y]Clinique Pasteur Toulouse, Toulouse, France; [z]Department of Cardiology, Hospital Universitari de la Santa Creu i Sant Pau, IIB Sant Pau, Barcelona, Spain; [aa]Department of Cardiac, Thoracic and Vascular Sciences and Public Health, University of Padova, Padova, Italy; [bb]Cardiovascular Disease Unit, IRCCS Ospedale Policlinico San Martino, Genoa, Italy; [cc]Interventional Cardiology Unit, Maria Cecilia Hospital, Cotignola, Italy; [dd]Interventional Cardiology Unit, IRCCS San Raffaele Scientific Institute, Milan, Italy; [ee]University of Pisa, Pisa, Italy; [ff]Division of Cardiology, University of Ottawa Heart Institute, Ottawa, Ontario, Canada; [gg]Department of Internal Medicine/Cardiology and Intensive Care at Marienhospital Osnabrück of the Niels-Stensen Clinics, Osnabrück, Germany; and the [hh]CardioVascular Center Frankfurt, Frankfurt am Main, Germany.

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 29, 2025; revised manuscript received January 8, 2026, accepted January 14, 2026.

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JACC: CARDIOVASCULAR INTERVENTIONS VOL. 19, NO. 6, 2026
MARCH 23, 2026:726–736

to present real-world data on the incidence, mechanisms, management, and outcomes of T-TEER device-related failure.

METHODS

STUDY DESIGN. The FATE (Failed Tricuspid Transcatheter Edge-to-Edge) registry is an investigator-initiated, multicenter, retrospective, observational study. All consecutive T-TEER procedures performed between September 2017 and November 2024 at 31 high-volume centers were screened. Among these, all patients who developed device-related failure were included in the FATE registry cohort (Supplemental Figure 1). The study evaluated the incidence, predictors, and management of device-related failure in a real-world setting.

The study was conducted in accordance with the Declaration of Helsinki. The protocol was approved by the Institutional Review Board or ethics committee at each participating center, and all patients provided written informed consent according to local regulations.

POPULATION. Patients were eligible if they were symptomatic and had at least moderate residual or recurrent TR attributed to device-related T-TEER failure. Device-related failure was defined as the presence of LLI, SLDA, or device embolization occurring during the index procedure, the index hospitalization, or after discharge. Patients in whom such index intraprocedural complications were successfully corrected during the index procedure with TR less than moderate were not enrolled in the registry. By design, the registry focused on device-related mechanisms of failure; patients in whom recurrent or progressive TR was predominantly attributed to progression of the underlying atrial or ventricular disease, annular dilation, or other non-device-related mechanisms in the absence of LLI, SLDA, or embolization were not included in the study (Supplemental Table 1).

TR severity and device-related mechanisms of failure were assessed using 2-dimensional (2D) and 3-dimensional (3D) transthoracic echocardiography (TTE) and/or transesophageal echocardiography (TEE) using a multiparametric approach that integrated quantitative, semiquantitative, qualitative, and anatomical findings, in accordance with guidelines from the American Society of Echocardiography and the European Association of Echocardiography.[11,12]

The 3 device-related mechanisms of T-TEER failure were defined as follows[13] (Supplemental Figure 2):

1. LLI and/or leaflet damage: The device remains attached to both target leaflets; however, the depth and extent of leaflet insertion into the device arms are reduced in at least 1 leaflet and/or a leaflet injury is present. Leaflet injury was defined as a structural defect involving the treated leaflet(s), including
   - tear (disruption of leaflet integrity reaching the leaflet edge),
   - perforation (disruption of leaflet integrity not reaching the leaflet edge), and
   - shape distortion (marked leaflet deformation impairing coaptation).

   On color Doppler, the resultant regurgitant jet typically originates from the device-leaflet interface and is often parallel to the device, with a flow convergence zone located between the clip and the leaflet tip.
2. SLDA: Complete loss of connection between the device and 1 leaflet, with the device remaining attached to the other leaflet only. SLDA was diagnosed when the device was clearly seen to be grasping a single leaflet on 2D or 3D imaging, with a large eccentric regurgitant jet originating from the nongrasped commissure.
3. Device embolization: Complete loss of contact between the device and both leaflets, with displacement of the device from the intended tricuspid position to the right atrium, right ventricle, or pulmonary artery. In all such cases, severe TR was present.

In cases in which the mechanism of postprocedural TR remained uncertain on both 2D and 3D TTE and TEE, contrast-enhanced cardiac computed tomography was used at the discretion of the local heart team.[14] Multimodal imaging assessments, interpretations, and final diagnosis of the mechanism of failure were performed locally at each center on the basis of the predefined criteria described later.

PROCEDURE. Patients treated with T-TEER using commercially available tricuspid devices or off-label use of mitral transcatheter edge-to-edge repair systems on the tricuspid valve were eligible for inclusion in the registry. All T-TEER procedures were performed guided by TEE and fluoroscopy. The number and type of devices implanted were left to the operator's discretion, on the basis of intraprocedural assessment of tricuspid valve.

After failure diagnosis, the decision between reintervention and continued MT for postprocedural device-related failure was made on a case-by-case basis by the heart team, considering a combination

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of clinical symptoms, anatomical findings, and functional assessments. Additionally, candidacy for surgery or further TTVI was evaluated according to individual comorbidities, frailty scores, and patient preference.

OUTCOMES AND ENDPOINT DEFINITIONS. The prespecified coprimary endpoints were: 1) the incidence, classification, and management of device-related failure mechanisms after T-TEER; and 2) the coprimary clinical endpoint, a composite of all-cause mortality or HF rehospitalization after failure diagnosis in patients managed with reintervention vs MT alone.

All clinical events were defined according to the Tricuspid Valve Academic Research Consortium guidelines.[15] HF rehospitalization was any unplanned admission for HF symptoms requiring intravenous diuretic agents, vasodilators, or inotropes.

FOLLOW-UP. Patients were scheduled for follow-up according to each center's protocol and guideline recommendations. Follow-up assessments included a structured clinical evaluation, laboratory testing, and TTE; TEE was performed when clinically indicated or when image quality on TTE was insufficient to confidently characterize leaflet-device interaction, according to local practice.

Follow-up events were ascertained through clinic visits, structured telephone interviews, review of medical records, and linkage with regional and national registries when available.

STATISTICAL ANALYSIS. Categorical variables are reported as numbers and percentages and continuous variables as mean ± SD or median (Q1-Q3). Group comparisons used the chi-square or Fisher exact test for categorical variables and Student's *t*-test, the Mann-Whitney *U* test, or analysis of variance for continuous variables, after checking normality using the Shapiro-Wilk test. *P* values <0.05 were considered to indicate statistical significance. Time-to-event analyses were performed using Cox proportional hazards models with days from hospital discharge after management of failed T-TEER to the composite endpoint (all-cause death or HF rehospitalization) as the time scale. Covariates were selected a priori on the basis of clinical relevance and a directed acyclic graph informed by previous studies of severe TR and T-TEER. The final prognostic multivariable model included age, sex, TRI-SCORE, acute kidney injury, discharge TR severity, ratio of tricuspid annular plane systolic excursion to systolic pulmonary artery pressure as an index of right ventricular-pulmonary arterial coupling at the time of diagnosis, and the management strategy (MT vs reintervention). To account for heterogeneity among the 31 participating centers, robust SEs clustered by center were used. The proportional hazards assumption was assessed using Schoenfeld residuals, which did not indicate any relevant violation. The incidence of procedure failure was calculated for each center as the number of failures divided by the total number of T-TEER procedures performed per year, and the average incidence across centers was then derived. All analyses were performed using R version 3.5.3 (R Foundation for Statistical Computing).

RESULTS

CLINICAL AND ECHOCARDIOGRAPHIC CHARACTERISTICS. Among 2,278 consecutive T-TEER procedures performed between September 2017 and November 2024 across the 31 contributing centers, 123 cases of T-TEER failure (5.4%; 95% CI: 4.5%-6.4%) were observed: 30 cases due to LLI (24.4%), 92 due to SLDA (74.8%), and 1 device embolization.

Baseline clinical characteristics of the cohort of patients who experienced T-TEER failure are summarized in **Table 1** and Supplemental Table 2. Patients were elderly (mean age 78.1 ± 7.3 years), female (49.6%), and high risk (mean Society of Thoracic Surgeons risk score 9.1 ± 0.5, mean TRI-SCORE 4.9 ± 2.0). Atrial fibrillation was present in 92.6% and permanent transvalvular device leads in 23.6%. Most patients were symptomatic (65.0% in NYHA functional class III or IV), with signs of right HF in 48.0% and HF hospitalization in the previous year in 60.9%. All patients underwent index T-TEER for severe, torrential, or massive TR (**Figure 1**). The underlying etiology was functional in 77.2%, primary in 8.9%, and mixed in 13.8%; the mean left ventricular ejection fraction was 52.7% ± 11.2%. Preprocedural echocardiography showed a mean coaptation gap of 7.3 ± 3 mm, with a median effective regurgitant orifice area of 0.65 cm^2 and a vena contracta of 11.1 ± 4 mm.

PROCEDURAL FEATURES. In patients with LLI or SLDA, a median of 2 T-TEER devices were implanted during index procedure, mostly TriClip devices (71.5%; Abbott Structural Heart). Intracardiac echocardiographic guidance was added to TEE in 17.1% of index procedures. Postprocedural transvalvular gradients were low (mean 2.5 ± 0.75 mm Hg; median 3.0 mm Hg; Q1-Q3: 2.5-3.0 mm Hg), whereas residual severe or greater TR persisted in 31.7% of patients,

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MARCH 23, 2026:726–736

TABLE 1 Baseline Characteristics According to Medical Therapy or Redo Intervention

	Total Population (N = 123)	Medical Management (n = 67)	Redo Intervention (n = 56)	*P* Value
Female	61 (49.6)	39 (58.2)	22 (39.3)	0.037
Age, y	78.1 ± 7.3	78.3 ± 8.0	78.0 ± 6.5	0.479
BMI, kg/m^2	25.5 ± 5.2	25.4 ± 6.0	25.7 ± 4.0	0.195
STS score	9.1 ± 0.5	9.5 ± 0.7	8.5 ± 0 .8	0.249
TRI-SCORE	4.9 ± 2.0	4.8 ± 1.9	5.1 ± 2.1	0.483
Hypertension	90 (73.1)	53 (79.1)	37 (66.0)	0.104
Diabetes	33 (26.8)	18 (26.8)	15 (26.8)	0.992
Prior CABG	17 (13.9)	11 (16.6)	6 (10.7)	0.344
Prior PCI	31 (25.2)	14 (20.9)	17 (30.3)	0.229
Previous MI	20 (16.2)	11 (16.4)	9 (16.0)	0.578
Previous valve intervention	42 (34.1)	22 (32.8)	20 (35.7)	0.737
Previous CRT, CRT-D, ICD, or PPM	29 (23.5)	14 (20.9)	15 (26.7)	0.443
History of AF	114 (92.6)	63 (94.0)	51 (91.0)	0.530
eGFR, mL/min/m^2	46 ± 18.8	48.9 ± 18.4	42.5 ± 18.7	0.054
Previous stroke/TIA	17 (13.8)	10 (14.9)	7 (12.5)	0.700
COPD	27 (22)	12 (17.9)	15 (26.8)	0.236
Liver disease	13 (10.5)	7 (10.4)	6 (10.7)	0.962
NYHA functional class				0.699
II	31 (25.2)	19 (28.3)	12 (21.4)	
III	80 (65.0)	42 (62.7)	38 (67.8)	
IV	12 (9.7)	6 (8.9)	6 (10.7)	
Previous HF hospitalization	97 (78.8)	53 (54.6)	44 (45.3)	0.943
RHF signs	59 (47.9)	30 (44.7)	29 (51.8)	0.438
Echocardiographic pre-index procedure parameters				
TR grade before index procedure				
3	43 (34.9)	27 (40.3)	16 (28.5)	0.160
4	40 (32.5)	23 (34.3)	17 (30.3)	
5	40 (32.5)	17 (25.3)	23 (41.0)	
EROA, cm^2	0.74 ± 0.31	0.74 ± 0.3	0.74 ± 0.3	0.961
Vena contracta, mm	11.1 ± 4.0	10.9 ± 3.85	11.3 ± 4.3	0.875
Septolateral TV annular EDD, mm	45.0 ± 5.8	45.0 ± 6.3	45.0 ± 5.4	0.994
RAVi, mL/m^2	69.2 ± 33.1	77.2 ± 28.7	59.7 ± 37.0	0.012
RV FAC, %	36.1 ± 7.8	36.7 ± 6.5	35.5 ± 9.2	0.425
TAPSE, mm	17.3 ± 4.7	17.7 ± 4.6	16.8 ± 4.7	0.263
sPAP, mm Hg	40.9 ± 12.0	41.2 ± 12.8	40.5 ± 11.0	0.823
Moderate to severe MR	55 (44.7)	27 (49.9)	28 (50.1)	0.281
LVEF, %	52 ± 11.2	54.5 ± 10.3	50.4 ± 11.9	0.048

Values are n (%) or mean ± SD.

AF = atrial fibrillation; BMI = body mass index; CABG = coronary artery bypass grafting; CRT = cardiac resynchronization therapy; CRT-D = cardiac resynchronization therapy defibrillator; EDD = end-diastolic diameter; eGFR = estimated glomerular filtration rate; EROA = effective regurgitant orifice area; FAC = fractional area change; HF = heart failure; ICD = implantable cardioverter-defibrillator; LVEF = left ventricular ejection fraction; MI = myocardial infarction; MR = mitral regurgitation; PCI = percutaneous coronary intervention; PPM = permanent pacemaker; RAVi = right atrial volume index; RHF = right heart failure; RV = right ventricular; sPAP = systolic pulmonary artery pressure; STS = Society of Thoracic Surgeons; TAPSE = tricuspid annular plane systolic excursion; TIA = transient ischemic attack; TR = tricuspid regurgitation; TV = tricuspid valve.

including those in whom failure was already evident during the index procedure.

FAILURE CHARACTERISTICS. Most failures were diagnosed early, with a median time to diagnosis of 5 days (Q1-Q3: 0.7-71 days): 35.8% intraprocedurally and 29.2% during the index hospitalization. The remaining 34.1% were identified at follow-up, with a mean time to diagnosis of 66.2 ± 17.5 days from the index procedure. Overall, LLI accounted for 30 of 123 (24.4%) and SLDA for 92 of 123 (74.8%) failures, while device embolization occurred in 1 case (0.8%). Approximately one-half of patients in each group were managed medically, and one-half underwent redo intervention; the embolization case was treated by snaring of the device followed by MT. Failure modes did not differ across TR etiologies. At the time of diagnosis, TR was moderate in 21 of 123 cases (17.1%), severe in 53 of 123 (43.1%), massive in 19 of 123 (15.4%), and torrential in 19 of 123 (15.4%). The detailed distribution of leaflet involvement for LLI and SLDA is reported in **Table 2**.

MANAGEMENT. Overall, 67 of 123 patients (54.5%) received MT, and 56 of 123 (45.5%) underwent reintervention. Among reinterventions (Supplemental Table 3), 31 of 56 patients (55.4%) underwent redo T-TEER, 4 of 56 (7.1%) TTVR, 9 of 56 (16.1%) caval valve implantation, and 12 of 56 (21.4%) surgery (1 repair, 11 replacements). No bailout surgery was required after redo T-TEER.

OUTCOME IN HOSPITAL AND FOLLOW-UP. In-hospital cardiovascular mortality occurred in 4 of 123 patients (3.2%); 2 (1.6%) had stroke and 5 (4.1%) experienced major or life-threatening Valve Academic Research Consortium bleeding. Stage 2 to 4 acute kidney injury developed in 14 of 123 patients (11.4%), 6 of 67 (9.0%) in the MT group and 8 of 56 (14.3%) in the reintervention group; no acute kidney injury occurred after TTVR. Worsening HF during hospitalization was similar with MT and redo intervention (16 of 67 [24.6%] vs 10 of 56 [17.8%]; $P = 0.2$) (**Table 3**).

At discharge, 64 patients had severe or greater TR: 46 candidates for MT (71.8%) vs 18 who underwent redo interventions (28.1%) ($P = 0.001$).

At a median follow-up of 255 days (Q1-Q3: 109-425 days), the composite outcome of death or HF rehospitalization did not differ between MT (n = 24 [35.8%]) and reintervention (n = 23 [41.1%]) ($P = 0.3$). Within the reintervention group, events occurred in 15 patients (48.4%) after redo T-TEER, 4 (44.4%) after caval valve implantation, and 4 (33.3%) after surgery, whereas no deaths or HF rehospitalizations were observed after TTVR. Supplemental Figures 3 and 4 shows the Kaplan-Meier curves for the composite endpoint. Severe or greater TR persisted in 54 MT patients (70%) and 22 reintervention patients (30%) ($P = 0.001$) (**Figure 1**, **Table 4**). The

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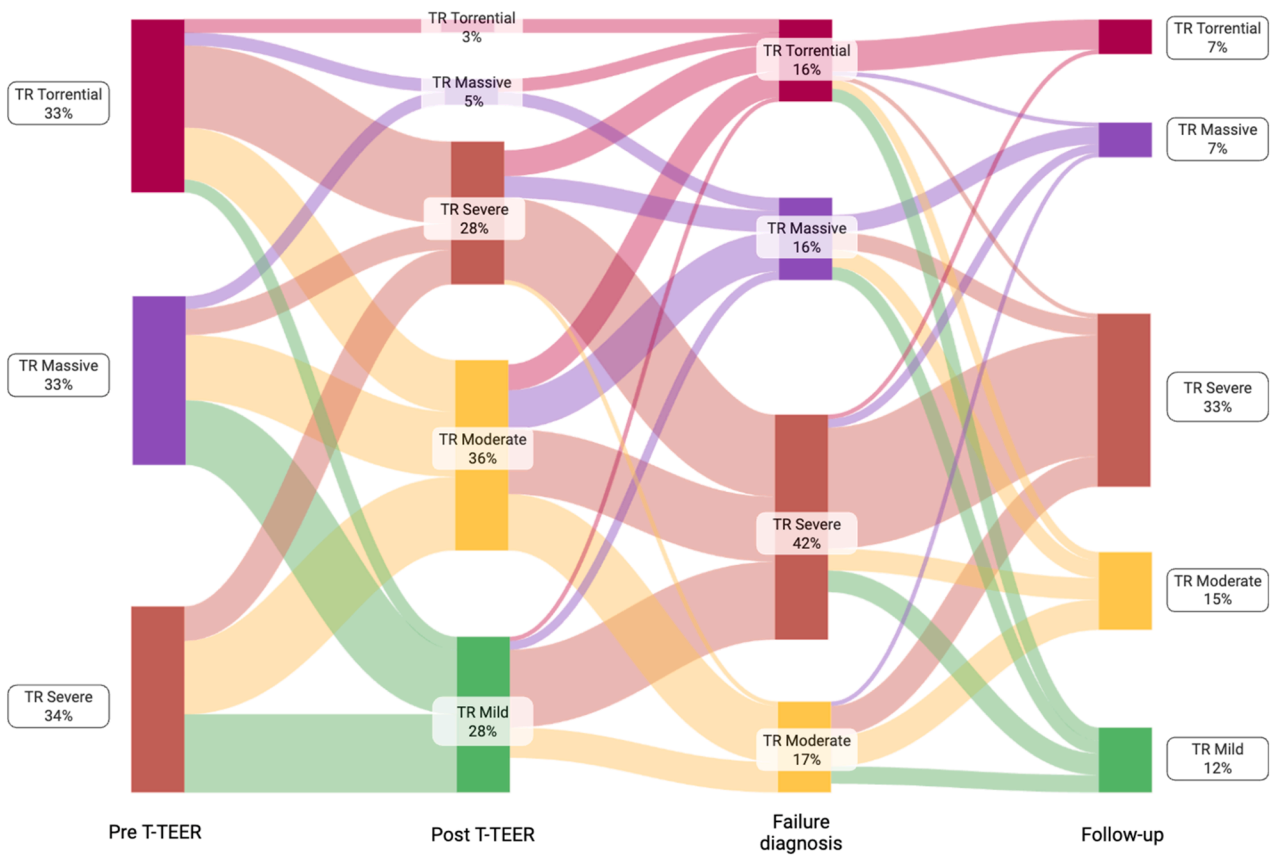
FIGURE 1 Evolution of TR



Tricuspid regurgitation (TR) before index procedure and at the time of failure diagnosis and residual tricuspid regurgitation after redo intervention or first rehospitalization. T-TEER = tricuspid transcatheter edge-to-edge repair.

reintervention group showed a significant reduction in right atrial volume index (RAVi) (59.7 ± 37.0 mL/m^2 vs 51.8 ± 28.0 mL/m2; ΔRAVi = −7.9 mL/m^2; P = 0.007), whereas no relevant improvement was observed in the MT group (77.2 ± 28.7 mL/m^2 vs 74.9 ± 35.0 mL/m^2; ΔRAVi = −2.5 mL/m^2; P = 0.5).

PREDICTORS OF COMPOSITE ENDPOINT DEATH AND REHOSPITALIZATION FOR HF. In multivariable Cox regression (Table 4), higher TRI-SCORE, acute kidney injury, and greater discharge TR severity were independently associated with the composite endpoint. Specifically, each 1-point increase in TRI-SCORE was associated with a 17% higher risk for death or rehospitalization (HR: 1.17; 95% CI: 1.03-1.33; P = 0.016), acute kidney injury conferred a more than 3-fold higher risk (HR: 3.68; 95% CI: 1.91-7.09; P < 0.001), and TR at discharge was also associated with worse outcome (HR: 1.25 per grade increase; 95% CI: 1.03-1.51; P = 0.026). Baseline ratio of tricuspid annular plane systolic excursion to systolic pulmonary artery pressure showed a nonsignificant trend toward a protective effect (HR: 0.12; 95% CI: 0.01-2.51; P = 0.174), and the reintervention strategy was associated with a better benefit than MT alone, although this difference was not statistically significant (HR: 0.70; 95% CI: 0.36-1.29; P = 0.243).

DISCUSSION

To our knowledge, the FATE registry represents the largest real-world report of T-TEER failure and the first to delineate real-world management pathways and outcomes according to the mechanisms of failure. The principal findings can be summarized as follows (Central Illustration).

First, among 2,278 T-TEER procedures performed at 31 high-volume centers, the incidence of early

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TABLE 2 Procedural Characteristics and Results According to Management

	Total Population (N = 123)	Medical Management (n = 67)	Redo Intervention (n = 56)	*P* Value
Device number	2 ± 0.5	2.01 ± 0.60	2 ± 0.73	0.449
Device model				0.561
TriClip	88 (72.7)	50 (76.9)	38 (67.8)	
PASCAL	18 (14.9)	8 (12.3)	10 (17.8)	
MitraClip	15 (12.4)	7 (10.7)	8 (14.3)	
TR grade after index procedure				0.359
1	36 (28.0)	18 (27.2)	18 (29.1)	
2	44 (36.1)	24 (36.3)	19 (34.5)	
3	33 (27.1)	19 (28.8)	14 (25.4)	
4	6 (5.0)	2 (3.0)	4 (7.2)	
5	3 (2.5)	3 (4.5)	0	
Mean gradient after index procedure, mm Hg	2.5 ± 0.7	2.6 ± 0.6	2.5 ± 0.8	0.710
LLI	30 (24.4)	18 (26.9)	12 (21.4)	0.475
Position of LLI				0.167
Anterior leaflet	16 (13.1)	8 (12.0)	8 (14.3)	
Posterior leaflet	7 (5.7)	6 (9.0)	1 (1.8)	
Septal leaflet	7 (5.7)	3 (5.0)	4 (5.4)	
SLDA	92 (74.8)	53 (79.1)	40 (71.4)	
Position of SLDA				0.471
Anterior leaflet	42 (34.1)	25 (37.3)	17 (30.3)	
Posterior leaflet	15 (12.2)	10 (15.0)	5 (9.0)	
Septal leaflet	35 (28.4)	18 (26.0)	17 (30.3)	
Device embolization		1 (0.8)	NA	
TR grade after failure				0.359
2	21 (17.1)	13 (22.4)	8 (14.8)	
3	53 (43.1)	31 (53.4)	22 (40.7)	
4	19 (15.4)	6 (10.3)	13 (24.0)	
5	19 (15.4)	8 (13.7)	11 (20.3)	
Mean gradient at failure time, mm Hg	2.9 ± 0.7	2.93 ± 0.7	2.9 ± 0.7	0.096
TAPSE/sPAP	0.45 ± 0.1	0.45 ± 0.1	0.46 ± 0.1	0.900

Values are mean ± SD or n (%).

LLI = loss of leaflet insertion; NA = not applicable; SLDA = single-leaflet device attachment; other abbreviations as in Table 1.

device-related failure was 5.4%, due predominantly to SLDA (92 of 123 [74.8%]) and LLI (30 of 123 [24.4%]); intraprocedural device embolization was rare (1 of 123 [0.8%]). At the time of failure diagnosis, residual device-related TR was substantial, with 73.0% of patients graded severe or greater. The composite endpoint of death and/or HF rehospitalization occurred in 38.2% of patients during follow-up.

Second, compared with MT alone, redo intervention achieved a greater reduction in TR and RAVi. However, about one-third of patients in the redo strategy group (mainly those undergoing redo T-TEER and caval valve implantation) still had severe TR, and this did not translate into better outcomes, as mortality and the composite of death and/or HF rehospitalization were similar between groups.

Third, TRI-SCORE, acute kidney injury, and TR grading at discharge independently predicted the composite endpoint of death and/or rehospitalization.

Cohort failure rates were comparable with the TRILUMINATE trial (7%) and slightly higher than in CLASP TR (4.6%) and bRIGHT (3.8%), likely because the registry explicitly reports LLI and reflects real-world heterogeneity. Failures were most frequently identified during the index hospitalization (35.7% intraprocedural, 29.2% in hospital), while 34.1% were first detected at follow-up (mean 66 ± 17.5 days), underscoring the importance of standardized pre-discharge imaging and a constant postprocedural surveillance window during the first year after the procedure.

In this failure cohort, adverse outcomes probably reflect the interplay between incomplete TR reduction and advanced right heart disease. Prior registries have shown that residual TR ≥3+ is associated with limited right ventricular reverse remodeling and worse clinical outcomes; our event rate (38.2%) is consistent with this signal.[16] Patients with device-related failure showed markers of advanced right-sided disease, notably right atrial enlargement, which has been reported as an independent predictor of mortality and HF hospitalization in previous studies.[17]

Reintervention after T-TEER failure had a clear impact on valvular hemodynamic status: two-thirds of redo patients achieved moderate or less TR at discharge and maintained this trend at follow-up, whereas significant TR (≥3+) persisted in 70% of patients managed medically. In parallel, the reduction in TR was accompanied by a significant decrease in RAVi after redo interventions (59.7 ± 37.7 to 51.9 ± 28.3 mL/m^2; ΔRAVi = −7.9 mL/m^2; P = 0.007), suggesting early reverse remodeling of the right heart. Recent literature on TTVI has reported that correction of TR could induce right heart reverse remodeling, supporting the concept that structural right atrial remodeling may represent an important marker of clinical benefit at follow-up.[5-8,18]

However, despite a numerically lower risk for death or HF rehospitalization with redo intervention (HR: 0.70), we did not observe a statistically significant difference in the composite endpoint, likely because of the limited sample size and follow-up. In line with randomized data showing that TTVI mainly alleviates symptoms and improves health status rather than hard outcomes at 1 year, our findings suggest that the primary treatment goal after T-TEER failure could be to achieve and maintain moderate or less TR together with atrial remodeling.

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TABLE 3 In-Hospital Outcomes, Discharge, and Follow-Up According to Management

	Total Population (N = 123)	Medical Management (n = 67)	Redo Intervention (n = 56)	*P* Value
In-hospital stay				
Stroke/TIA	2 (1.6)	2 (3.0)	0	0.291
AKI stages 2-4	14 (11.4)	6 (9.1)	8 (14.3)	0.270
Heart failure	26 (21.1)	16 (24.6)	10 (17.8)	0.249
Discharge mean tricuspid gradient, mm Hg	3.5 ± 0.8	3.45 ± 0.8	3.66 ± 1.0	0.787
Discharge TR severe to massive	64 (52.0)	46 (71.2)	18 (28.1)	0.001
RAVi, mL/m^2	66.1 ± 35.2	74.9 ± 35.0	51.8 ± 28.0	0.009
sPAP, mm Hg	37.5± 10.9	38.3 ± 11.8	36.5 ± 9.9	0.534
Discharge LVEF, %	53.6 ± 10.1	54.4 ± 8.4	52.8 ± 11.0	0.496
Outcome follow-up				
Composite of death and HF rehospitalization	47 (38.2)	24 (35.8)	23 (41.1)	0.340
Mean tricuspid gradient, mmHg	3.72 ± 1.0	3.6 ± 0.8	3.8 ± 1.1	0.433
TR more than severe	77 (62.6)	54 (70.1)	23 (29.8)	0.001
LVEF, %	53.7 ± 10.4	56.1 ± 8.9	50.9 ± 11.4	0.020
sPAP, mm Hg	38.4 ± 11.0	39.4 ± 11.7	37.2 ± 10.1	0.423
Any unsuccessful redo	5 (4.1)	NA	5 (8.9)	

Values are mean ± SD or n (%).
AKI = acute kidney injury; other abbreviations as in Tables 1 and 2.

The definitive prognostic impact of this strategy will require confirmation in larger, longer term studies.[5-8,18]

This cohort represents patients with advanced and anatomically complex tricuspid disease, in whom a heavy clinical burden is compounded by unfavorable anatomy and further aggravated by device-related failure. Therapeutic options are limited, and prognosis is largely dictated by disease stage. In this high-risk, anatomically complex population, reintervention was able to produce better TR reduction and early right atrial reverse remodeling, but this did not translate into a clear survival or HF rehospitalization benefit over a median of 8 months. These findings support an individualized, risk- and anatomy-driven strategy and highlight the need for prospective studies on earlier escalation to TTVR or surgery in selected phenotypes.

STUDY LIMITATIONS. First, FATE is a retrospective, observational registry in which the choice between reintervention and MT alone was made by local heart teams on the basis of clinical status, anatomical feasibility, and perceived procedural risk. Moreover, although this is the largest cohort of T-TEER failures reported to date, the overall sample size and the number of patients in each reintervention subgroup were modest. Taken together, these factors make the comparison between management strategies highly susceptible to confounding by indication and residual bias and limit the statistical power to detect differences in hard outcomes; therefore, the analyses should be regarded as descriptive and hypothesis generating.

Second, by design we enrolled only patients with at least moderate to severe residual or recurrent TR in whom a device-related mechanism of failure (LLI, SLDA, or device embolization) was documented. Patients with recurrent TR due mainly to progression of atrial or ventricular remodeling, annular dilatation, or other non-device-related mechanisms, as well as those with intraprocedural leaflet complications that were fully corrected before discharge, were not captured. Therefore, the registry does not provide a comprehensive estimate of all mechanisms of TR recurrence after T-TEER, and the observed incidence of leaflet injury likely underestimates the true burden of postprocedural leaflet abnormalities.

TABLE 4 Independent Predictors of Composite Outcome Death And/or HF Hospitalization

	HR	95% CI	*P* Value
Age	1.01	0.97-1.05	0.522
Female	1.2	0.65-2.30	0.500
TRI-SCORE	1.17	1.03-1.33	0.016
RV-PA coupling[a]	0.20	0.01-2.5	0.174
Reintervention	0.70	0.3-1.2	0.240
AKI	3.90	1.9-7.0	<0.001
Discharge TR	1.24	1.1-1.5	0.020

[a]TAPSE/sPAP ratio as an index of RV-PA coupling.
PA = pulmonary arterial; other abbreviations as in Tables 1-3.

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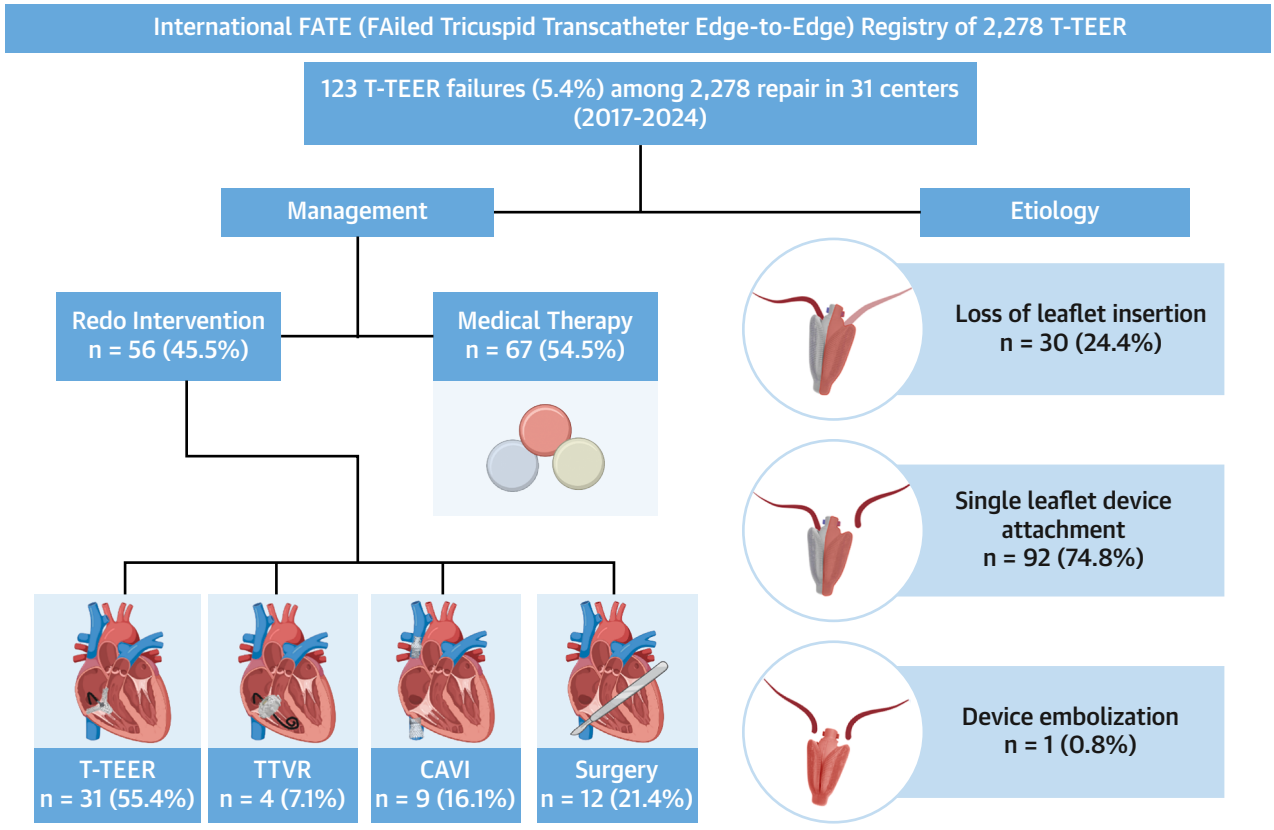
JACC: CARDIOVASCULAR INTERVENTIONS VOL. 19, NO. 6, 2026
MARCH 23, 2026:726–736

CENTRAL ILLUSTRATION **Failed Tricuspid Transcatheter Procedures**



- In this large real-world registry, device-related failure after T-TEER is relatively uncommon (5.4%), but clinically meaningful, marked by high residual TR (48%) and high rate of death and/or HF rehospitalization (38.2%) at 255 days (IQR: 109-425 days) of follow-up.
- Redo intervention achieves better TR reduction (32.7% vs 74.2%) and a significant RAVI reduction (Δ-7.9 mL/m^2; P = 0.007) compared with medical therapy, while death and/or HF rehospitalization remain similar between strategies.
- Independent predictors of death/HF rehospitalization: higher TRISCORE (HR: 1.17 per point), postprocedural AKI (HR: 3.68), and greater discharge TR severity (HR: 1.25 per grade).

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AKI = acute kidney injury; CAVI = caval valve implantation; FATE = Failed Tricuspid Transcatheter Edge-to-Edge; RAVI = right atrial volume index; T-TEER = tricuspid transcatheter edge-to-edge repair; TR = tricuspid regurgitation; TTVR = transcatheter tricuspid valve replacement.

Third, all participating centers were high-volume T-TEER programs using different devices, imaging protocols, and management strategies, and no prespecified treatment algorithm or imaging core laboratory was used. This heterogeneity may have led to misclassification of failure modes and variability in TR grading and right heart reverse remodeling.

Fourth, the study did not incorporate the Kansas City Cardiomyopathy Questionnaire score, which precluded an analysis of quality of life in the cohort. This point is important, as in randomized trials against MT, T-TEER has shown an improvement in quality of life and a reduction in HF hospitalizations and has not yet been shown to decrease mortality.

Fifth, follow-up was of mid-term duration (median 255 days) and was based on site-reported events. Late device-related failures, very late

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reverse remodeling, and long-term differences between management strategies may not have been fully reported. In addition, our findings originate from experienced centers and may not be generalizable to lower volume programs or different health care settings.

In view of these limitations, the present results should be considered exploratory and hypothesis generating and warrant confirmation in prospective, ideally randomized, studies with standardized imaging protocols, adjudication of failure mechanisms and longer follow-up.

CONCLUSIONS

In the largest real-world series to date, device-related failure after T-TEER was relatively uncommon, but clinically meaningful, marked by high residual TR and a high rate of death and/or HF rehospitalization at follow-up. Reintervention produced greater TR reduction and right atrial reverse remodeling than MT but did not clearly improve survival or HF rehospitalization. Outcomes were driven largely by global risk, postprocedural acute kidney injury, and discharge TR severity, supporting a hypothesis-generating interpretation.

FUNDING SUPPORT AND AUTHOR DISCLOSURES

Dr De Marco is a proctor and consultant for Abbott Laboratories and Edwards Lifesciences; and has received speaker fees from Abbott Laboratories and Edwards Lifesciences. Dr Mangieri has received speaker fees from Abbott Laboratories and Edwards Lifesciences. All other authors have reported that they have no relationships relevant to the contents of this paper to disclose.

ADDRESS FOR CORRESPONDENCE: Dr Federico De Marco, IRCCS Centro Cardiologico Monzino, Via Parea 4, 20100 Milan, Italy. E-mail: federico.demarco@gmail.com.

PERSPECTIVES

WHAT IS KNOWN? Severe TR is associated with high morbidity and mortality, and T-TEER has emerged as a key option for high-risk patients. However, device-related failure mechanisms such as LLI and SLDA, and their optimal management, are poorly characterized in real-world practice.

WHAT IS NEW? In this large multicenter registry, device-related failure after T-TEER occurred in 5.4% of procedures, was due predominantly to SLDA, and was usually associated with severe or greater residual TR. Reintervention, compared with MT alone, produced greater TR reduction and right atrial reverse remodeling but did not improve survival or decrease HF rehospitalization, reflecting a high-risk population in which higher TRI-SCORE, acute kidney injury, and greater discharge TR severity independently predicted adverse outcomes.

WHAT IS NEXT? Prospective, ideally randomized studies with standardized imaging and longer follow-up are needed to define management algorithms for device-related failure, including timely escalation to surgery or TTVR in complex cases. Future work should determine whether strategies that reliably achieve and maintain moderate or less TR and sustained right atrial reverse remodeling can translate into durable improvements in hard outcomes and quality of life.

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KEY WORDS LLI, SLDA, TEER failure, T-TEER, TTVI

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

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