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A Contemporary Look at the Landscape of Treatment of Tricuspid Regurgitation
A Review

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IMPORTANCE Untreated severe tricuspid regurgitation carries a poor prognosis. We aim to provide a contemporary review of the anatomy, clinical manifestations, and diagnostic and management strategies, including medical, surgical and transcatheter options. By synthesizing current knowledge, this review seeks to equip clinicians with the insights necessary to navigate the complexities of TR treatment.

OBSERVATIONS Tricuspid regurgitation is predominantly secondary to annular dilation and leaflet tethering but can also be associated with cardiac implantable electronic device leads and primary leaflet pathologies. Isolated tricuspid valve surgery is infrequently performed, especially in high surgical risk patients, prompting the emergence of transcatheter treatment options. These advancements are complemented by significant strides in multimodality imaging, including three-dimensional echocardiography, computed tomography, and magnetic resonance imaging, which enhance diagnostic accuracy and procedural planning.

CONCLUSIONS AND RELEVANCE The effective management of tricuspid regurgitation necessitates a multidisciplinary approach, integrating input from interventional cardiology, cardiac surgery, heart failure cardiology, imaging, and electrophysiology. Surgical and transcatheter interventions such as tricuspid transcatheter-edge-to-edge repair and transcatheter tricuspid valve replacement have demonstrated favorable early clinical and functional outcomes, but ongoing research is necessary to refine patient selection and improve treatment decision-making. Individualizing treatment plans to optimize health outcomes and quality of life for patients with tricuspid regurgitation is paramount.

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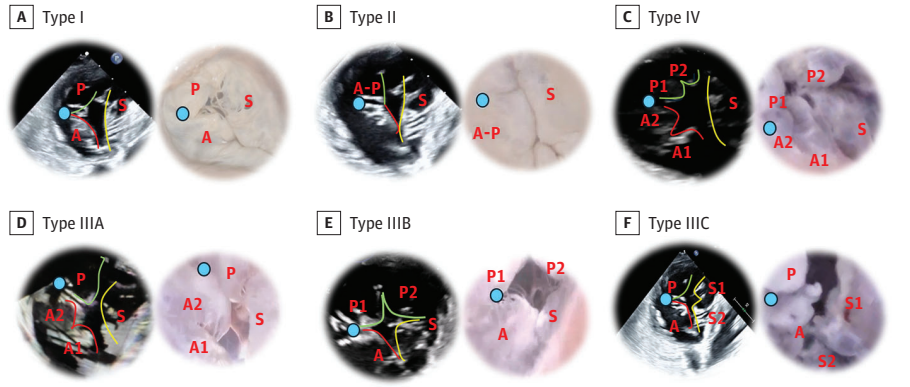
Untreated severe tricuspid valve regurgitation (TR) carries a poor prognosis.[1] The Framingham Heart Study reported a TR prevalence of up to 5.6% in women and 1.5% in men older than 70 years,[2] with higher rates in older age groups. While TR has multiple causes, secondary TR from annular dilation and leaflet tethering remains predominant.[3] Severity assessment depends on multiple factors, including cardiac loading conditions and imaging modality.[4,5] Advances in multimodality imaging, such as 3-dimensional transthoracic echocardiography (TTE), transesophageal echocardiography (TEE), computed tomography (CT), and cardiac magnetic resonance imaging, have improved the understanding of tricuspid valve (TV) anatomy and right ventricular (RV) pathology.[5-7] Treatment options have also advanced, including medical management, surgery, and transcatheter interventions. In this review, we aim to provide a contemporary look at the landscape of TR treatment for general cardiologists from a multidisciplinary perspective.

TV Anatomy

The TV regulates blood flow from the right atrium to the RV and is the largest cardiac valve.[8,9] The valve has 4 our main components: the annulus, leaflets, chordae tendineae, and papillary muscles. The annulus is a dynamic D-shaped fibrous ring that anchors the leaflets and can vary in area by up to 30% during the cardiac cycle.[8] Typically, the TV has 3 leaflets, anterior, posterior, and septal, but leaflet segment numbers can vary significantly. Classification includes type I (3 leaflets), type II (2 leaflets), type III (4 leaflets), and type IV (>4 leaflets), complicating imaging and intervention planning (**Figure 1**).[10] The surrounding anatomy, including vena cavae orientation, right coronary artery proximity, and atrioventricular node location, influences intervention strategies and necessitates meticulous preprocedural planning to avoid complications.[9]

Figure 1. Classification of Tricuspid Valve Leaflet Morphology[10]



The tricuspid valve is now classified into 4 different morphologies: type I (3 leaflets), type II (2 leaflets), type III (4 leaflets), and type IV (more than 4 leaflets). The severity of tricuspid regurgitation is proposed to be 5-graded: mild, moderate, severe, massive, and torrential based on different echocardiographic parameters. A indicates anterior leaflet; P, posterior leaflet; S, septal leaflet; the blue dot, the papillary muscle.

Table 1. Mechanisms of Tricuspid Regurgitation (TR)

		Secondary		CIED related	
	Primary	Atrial[a]	Ventricular[a]	CIED related	CIED associated
Pathology	Lack of leaflet coaptation due to leaflet or chordal abnormality	Right atrial or tricuspid annulus dilation	Right ventricular enlargement and/or dysfunction	TR induced by the CIED lead	Presence of CIED lead without interfering with valvular apparatus
Etiologies	Congenital (Ebstein), endocarditis, carcinoid, degenerative, rheumatic, traumatic, postendomyocardial biopsy, postradiation	Chronic atrial fibrillation/flutter, heart failure with preserved ejection fraction	Left ventricular dysfunction, left-sided valvular disease, pulmonary hypertension, right ventricular infarction, right ventricular cardiomyopathy	Leaflet impingement, perforation or avulsion; valvular or subvalvular adhesions or restriction	CIEDs present as a bystander; TR is related to other mechanism, including pacing-related right ventricular dyssynchrony and dysfunction
Features	Restricted or excessive leaflet mobility or leaflet perforation	Marked tricuspid annular and right atrium dilation; right ventricle volume is typically normal (except in late stages)	Marked tricuspid leaflet tethering and annular dilation, right ventricle and atrium are dilated	Lead directly interfering tricuspid valve apparatus, tricuspid annulus, right ventricle and atrium can be dilated if chronic	No interaction of lead with the tricuspid valve apparatus

Abbreviation: CIED, cardiac implantable electronic device.

[a] Atrial and ventricular secondary TR often coexist.

Clinical Presentation and Classification of Tricuspid Regurgitation

Patients with severe TR typically present with clinical signs of volume overload (edema and ascites), decreased cardiac output (fatigue, exercise intolerance, and dyspnea), arrhythmias, and end-organ damage (cardiorenal and cardiohepatic syndromes).[4] The often subtle nature of early symptoms and signs frequently delays diagnosis. During clinical assessment, it is important to recognize the symptoms of decreased cardiac output. If symptoms are refractory to initial medical therapy or if end-organ damage develops, early referral for potential intervention is warranted.[11] Besides, preintervention staging of renal and liver dysfunction is essential in advanced TR, as it impacts procedural risk and helps guide the intensity of interventions.

TR is broadly classified as primary, secondary, or cardiac implantable electronic device (CIED) related (**Table 1**).[12] Primary TR involves structural TV changes (myxomatous degeneration, congenital anomalies, rheumatic disease, carcinoid, or endocarditis) and is less common than secondary TR. Secondary TR (functional) is subcategorized into atrial and ventricular secondary TR, both of which result in annular dilation and a variable degree of leaflet tethering.[3,4,12] Atrial secondary TR typically results from right atrial dilation due to chronic atrial arrhythmias[4] and occurs without significant RV remodeling. It is also seen in patients without atrial fibrillation who have elevated filling pressures in the setting of heart failure (HF) with preserved ejection fraction.[13] Ventricular secondary TR is a consequence of RV dilation from primary RV disease, prior RV infarct, pulmonary hypertension, left ventricular dysfunction, or left-sided valvular heart disease and has a less favorable prognosis.[14]

CIED-related TR is expected to increase alongside an aging population and significant rise in CIED implantation.[15] TR in the presence of a CIED lead is classified as CIED-associated TR (TR not directly caused by the lead) and CIED-related TR (TR directly caused by lead placement).[16] This classification is crucial to determine the optimal treatment strategy.

Due to the complexity of diagnosis and management, the 2025 European guidelines for valvular heart disease recommend a careful multidisciplinary heart team evaluation of all patients with severe TR prior to intervention (class Ic).[11]

Table 2. Imaging Modalities for Management of Tricuspid Regurgitation (TR)

Modality	Role in management of TR	Strengths	Weaknesses
TTE (2D/3D)	• Primary modality to assess tricuspid valve and right heart size and function, and estimate pulmonary artery pressures	• Noninvasive, more readily available • Near-field imaging of the tricuspid valve allows 2D and 3D imaging • Rarely can be used to guide procedures	• Acoustic window dependent on patient factors (ie, body habitus), operator expertise • If used to guide procedures, simultaneous fluoroscopy should not be used
TEE (2D/3D)	• Quantitation of TR • Determining anatomic suitability of repair vs replacement depending on individual anatomy • Intraprocedural guidance for TTVI	• Multilevel imaging of the tricuspid valve • 3D reconstruction and rendering possible with high temporal/spatial resolution • Essential for TTVI	• Semi-invasive, requires sedation/general anesthesia if prolonged use with risk of tissue injury • Off-axis imaging of the annular plane and further distance to the probe resulting in suboptimal Doppler quantification • Prone to shadowing artifact from implanted cardiac prosthesis
3D intracardiac echocardiogram	• Intraprocedural guidance for TTVI	• Close to TV and potential to avoid shadowing artifact to image TV from cardiac prosthesis	• Narrow field of view • 3D resolution inferior to TEE • Cost
Computed tomography	• Preprocedural planning for TTVR, and assessment of RV size and function	• High resolution of cardiac structures • Allows detail procedural planning from vascular access to entry to RV	• Early data suggesting cutoffs for quantifying TR severity by AROA on dual-source CT • Requires specific contrast protocols to optimal imaging of right heart • Lower temporal resolution compared to echocardiography
Cardiac magnetic resonance imaging	• Quantitative assessment of RV volumes, TR severity and RV function	• High spatial resolution allowing delineation of complex tissue structures • Allows for quantification of tricuspid regurgitant volume and regurgitant fraction • Can quantify RV impairment and fibrotic remodeling	• Not readily available • Requires operator expertise

Abbreviations: 2D, 2-dimensional; 3D, 3-dimensional; AROA, anatomic regurgitant orifice area; CT, computed tomography; RV, right ventricle; TEE, transesophageal echocardiography.; TTE, transthoracic echocardiography; TTVI, transcatheter tricuspid valve interventions; TTVR, transcatheter tricuspid valve replacement; TV, tricuspid valve.

Imaging Modalities to Evaluate TV Anatomy, Pathology, and Treatment Options

Comprehensive assessment of TV anatomy, pathology, and treatment options requires a multimodal approach to imaging that includes TTE, TEE, cardiac CT, and magnetic resonance imaging (**Table 2**).[5] Imaging is central to the evaluation of TR severity and etiology, identification of complex valvular anatomy, right heart size and function, assessment of venous congestion, and estimation of pulmonary pressures. These assessments may be integral to predicting device efficacy and thus determining device choice.

While TTE is the first-line modality for assessing TR and cardiac function, limitations such as dependence on acoustic windows, operator expertise and the shortcomings of current guideline-recommended assessment methods, may underestimate TR severity.[17] Three-dimensional color Doppler TTE measures vena contracta area without geometric assumptions,[18] but acquiring high-resolution images can be challenging. Current 2025 European guidelines[11] use the different proximal isovelocity surface area and 3-dimensional vena contracta area cutoffs originally proposed by Hahn and Zamorano[19] ($\geq$40 mm^2 and $\geq$75 mm^2, respectively), recognizing the consistent underestimation of TR severity by proximal isovelocity surface area.[20] New volumetric methods of quantifying regurgitant volume may provide an additional mean to assess TR severity.[5] Three-dimensional TEE provides enhanced TV visualization and is crucial for procedural guidance.[21] Additionally, 3-dimensional intracardiac echocardiography can support TEE in various transcatheter TV interventions.[22] Cardiac CT offers high-resolution anatomic detail, allowing precise annular measurements, the visualization of surrounding structures,[23] and preprocedural planning for transcatheter interventions. Cardiac magnetic resonance imaging is supplementary to echocardiographic assessment of TR severity by its ability to more precisely measure RV volumes and function, regurgitant TV volume, and strain.[24] Invasive hemodynamics directly measure right heart pressures, differentiate pulmonary hypertension types, and identify pulmonary vascular remodeling, which can inform treatment strategies and optimize outcomes by addressing reversible vasoconstriction before intervention,[25] and the 2025 European guidelines recommend right heart catheterization in all candidates for transcatheter interventions.[11] Comprehensive RV assessment—utilizing both advanced imaging and hemodynamic metrics—is crucial for risk stratification and predicting postintervention outcomes.[5,26]

Recognizing that TR severity varies with loading conditions underscores the importance of assessing TR in a euvolemic state. Classification of TR severity has now expanded to 5 grades (massive and torrential beyond severe)[19] which has not only proved to have prognostic importance in medically treated patients, but also in patients undergoing transcatheter therapies. Registries,[27,28] early feasibility studies,[29] and randomized clinical trials[30] have shown lower efficacy of the tricuspid transcatheter edge-to-edge repair (T-

TEER) devices with massive and torrential disease. Thus severity of TR as well as multisegment morphologies,[10] may influence treatment strategy (Figure 1).[5]

Treatment Options

Medical

Currently, there are no class I recommendations for the medical management of TR.[31,32] Medical therapy primarily focuses on symptom control, addressing volume overload with diuretics and there is a high degree of variability in managing secondary TR including inotropes, inodilators, pulmonary vasodilators, and neurohormonal therapy depending on the clinical context.[31] Managing atrial arrhythmias can sometimes reduce TR severity, but the effect is variable and dependent on atrial size, arrhythmia duration, and durability of sinus rhythm.[33] Since HF with preserved or reduced ejection fraction frequently coexists with secondary TR, optimal medical therapy (OMT) indicated in HF including sodium-glucose cotransporter 2 inhibitors should be used. Studies suggest that RV reverse remodeling, improvement in RV function, and reduction in pulmonary artery pressures occur following initiation of sodium-glucose cotransporter 2 inhibitors in patients with HF with reduced ejection fraction.[34,35] Whether OMT improves TR severity needs further investigation.[13] However, medical therapy alone does not improve symptomatic TR, as shown in the control arms of the Trial to Evaluate Cardiovascular Outcomes in Patients Treated with the Tricuspid Valve Repair System Pivotal (TRILUMINATE) and EVOQUE Transcatheter Tricuspid Valve Replacement: Pivotal Clinical Investigation of Safety and Clinical Efficacy Using a Novel Device (TRISCEND II) randomized clinical trial.[30,36] Nonetheless, maintaining OMT after intervention is essential to confer sustained clinical improvement.[37]

Surgical: Repair vs Replacement and Outcomes

Current guidelines give a class I recommendation for concomitant TV surgery for either primary or secondary severe TR, during left-sided valvular surgery.[11,31] A randomized clinical trial in patients with moderate TR or mild TR with annular dilation demonstrated that TV repair at the time of MV surgery for degenerative MR reduced the risk of TR progression, though this outcome was limited to patients with moderate TR at baseline.[33] Nonetheless, there is evidence that mild TR is associated with progression of the disease and the 2025 European guidelines give IIa recommendations for concomitant TV repair in the setting of moderate TR, and a IIb recommendation for mild TR with annular dilatation.[11] The 2025 European guidelines also give a class I indication for isolated TV surgery for severe, symptomatic primary TR without severe RV dysfunction or severe pulmonary hypertension.[11]

Surgical decisions, including repair vs replacement and outcomes, are complex. A comprehensive review on advances in TV surgery is available.[38] Minimally invasive[39] and robotic techniques[40] continue to be explored. Importantly, there are no studies demonstrating a survival benefit with either concomitant TV surgery at time of mitral valve surgery, mitral-tricuspid[41] or isolated TV surgery.[42]

TR is often secondary to other cardiac conditions, so combined procedures are frequently performed. Surgery remains the gold standard for concomitant mitral-tricuspid regurgitation unless the patient is at high surgical risk. Isolated TV surgery accounts for only 20% of all TV surgeries due to higher historical operative mortality (>10%), especially in older or high-risk patients.[43,44] However, recent data from the Society of Thoracic Surgeons database suggests that isolated TV surgical mortality is 5.6% overall (mean [SD] age, 52.6 [18.1] years)[45] and up to 7.3% for patients without endocarditis.[46] A new risk score has been developed to better stratify patients undergoing TV surgery[47] but has not been validated for transcatheter therapies, and may be limited by the failure to capture RV function parameters.

Ring annuloplasty is now considered the gold standard for surgical TV repair.[32] Initially, a running suture annuloplasty (DeVega method) was used; however, it has been abandoned because of poor long-term durability.[48] Undersized annuloplasty has had high success rates, especially in secondary TR or in cases without CIED lead interference.[49] Surgical replacement is typically reserved for patients with markedly dilated annuli combined with pronounced leaflet tethering, or nonrepairable valves (congenital heart disease, carcinoid heart disease, endocarditis, or extensively damaged leaflets).[31,32] Both mechanical and bioprosthetic valves are used, with bioprosthetic durability approaching 15 years.[50] While annuloplasty is preferred for leaflet integrity, recurrent TR in patients with significant leaflet tethering and RV dysfunction often leads to valve replacement, especially in those with a tricuspid annular diameter greater than 44 mm.[51]

Transcatheter: Repair vs Replacement

Given isolated TV surgery is rarely performed, transcatheter interventions have become an alternative in high-risk patients. The 2025 European Society of Cardiology guidelines recommend transcatheter TV treatment to improve quality of life (QoL) and RV remodeling in high-risk patients with symptomatic severe TR despite OMT in the absence of severe RV dysfunction or precapillary pulmonary hypertension (class IIa).[11]

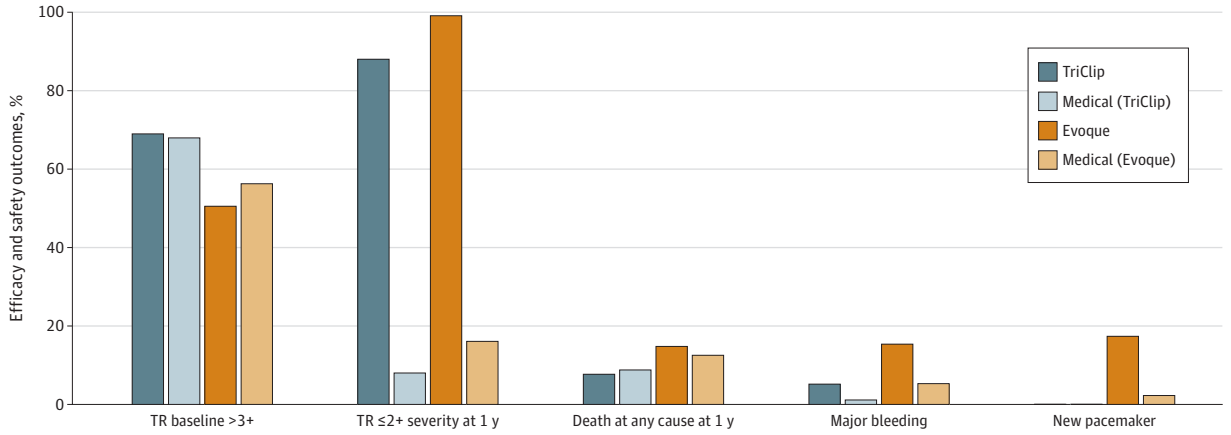
T-TEER and transcatheter TV replacement (TTVR) are 2 commercially available approaches to treat TR.[30,52] The US Food and Drug Administration (FDA) approval letter for the EVOQUE (Edwards Lifescience) TTVR system states that the device is indicated for improving health status in patients with symptomatic severe TR despite OMT, for whom TV replacement is deemed appropriate by a heart team.[53] The FDA approval letter for the TriClip (Abbott) T-TEER system states that the device is indicated for improving QoL and functional status in similar patients in whom T-TEER is clinically appropriate.[54] The US Centers for Medicare and Medicaid Services has approved "coverage with evidence development"[55,56] for both procedures, requiring additional data on device use and comparisons with medically managed patients before definitive coverage decisions.

In addition to the aforementioned devices, a number of novel transcatheter tricuspid technologies are either currently under development and have completed early feasibility studies or have finished enrollment in randomized clinical trials (eTable 1 in the Supplement).[57-60]

Outcomes: T-TEER vs TTVR

T-TEER devices (eg, TriClip G4/G5 and PASCAL Precision [Edwards Lifesciences]) reduce TR by improving leaflet coaptation.[30,61] The

Figure 2. Comparison of Tricuspid Transcatheter Edge to Edge Repair (T-TEER) vs Transcatheter Tricuspid Valve Replacement (TTVR)



The chart on top shows that the cohort of patients treated in the 2 major randomized clinical trials are different as illustrated by a different baseline TR, 1-year death risk, and major bleeding risk in the 2 control groups.[30,36] Therefore, a direct comparison of their efficacy cannot be drawn. TR indicates tricuspid valve regurgitation;

procedure is performed under general anesthesia with TEE and fluoroscopy guidance, often supplemented with 3-dimensional intracardiac echocardiography to confirm leaflet grasping.[62]

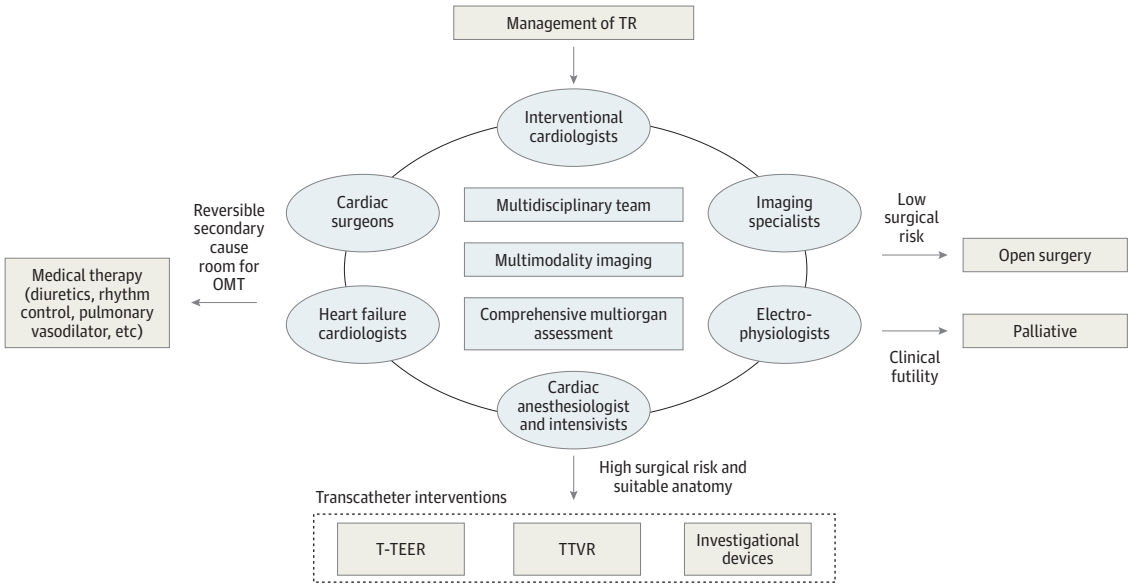
The TRILUMINATE pivotal trial randomized 350 patients with severe TR (mean [SD] age, 78 [7] years; 54.9% women) at intermediate or higher surgical risk to TriClip T-TEER vs OMT. Results demonstrated significant TR reduction, with moderate or lower TR observed in 87% of T-TEER-treated patients vs 4.8% receiving OMT alone at 30 days.[30] Device success was 99% with no procedural deaths and a 0.5% 30-day all-cause mortality.[30] T-TEER superiority at 1 year persisted—a hierarchical composite end point including death, tricuspid surgery, HF hospitalization, and QoL showed a win ratio of 1.48 (95% CI, 1.06-2.13; *P* = .02),[30] driven by improvement in Kansas City Cardiomyopathy Questionnaire (KCCQ) scores. Patients undergoing T-TEER experienced an average KCCQ score improvement of 12.3 points vs just 0.6 in controls (*P* < .001), indicating an improvement in QoL (an improvement of 5 points was deemed clinically meaningful).[58,63] Additionally, nearly 75% of T-TEER patients (n = 110/147) were alive and well at 1 year, defined as a KCCQ score of 60 or greater with no decline greater than 10 points, compared to approximately 46% in the control group (n = 68/148), with a number needed to treat of 3.5 to be alive and well.[64] The 2-year outcomes showed annualized HF hospitalization rates to be significantly lower after T-TEER (0.19 vs 0.26 events per patient-year; *P* = .02). Survival free from death, surgery, or intervention was markedly higher in the T-TEER group compared to control (77.6% vs 29.3%; *P* < .001), driven by increased crossover in the control group.[65] However, the rates of all-cause mortality (17.9% vs 17.1%) were similar.

Outcomes of PASCAL T-TEER device have also been promising.[61] The PASCAL for TR—a European registry (PASTE) involved 1059 patients with symptomatic TR at high risk (mean [SD] age, 79 [9] years; mean [SD] TRI-SCORE risk, 23% [18]).[61] PASCAL T-TEER reduced TR to moderate or lower in 87% (n = 1059).[61] The ongoing Edwards PASCAL Transcatheter Valve Repair System Pivotal Clinical Trial (CLASP II) TR pivotal trial (NCT04097145) randomizes patients to PASCAL T-TEER vs OMT, with 2-year end points including mortality, right ventricular assist device implantation, transplant, TV intervention, HF hospitalization, and KCCQ score improvement. Overall, T-TEER has demonstrated promising midterm benefits in reducing TR severity, improving QoL, and decreasing HF hospitalizations in carefully selected populations.

Orthotopic TTVR involves implanting a bioprosthetic valve within the native TV. The EVOQUE system consists of a self-expanding bovine pericardial valve mounted on a nitinol frame, with a dual-frame design that anchors within the annulus and with leaflet capture. The procedure is performed via a transfemoral venous approach under general anesthesia, guided by TEE and fluoroscopy, occasionally supplemented by 3-dimensional intracardiac echocardiography.[66] The randomized TRISCEND II trial, which enrolled 400 patients with severe TR, demonstrated a win ratio of 2.0 (95% CI, 1.45-2.62; *P* < .001) favoring TTVR over OMT, based on a hierarchical composite primary outcome consisting of all-cause mortality, right ventricular assist device implantation or heart transplantation, TV reintervention, HF hospitalization, an improvement of KCCQ score or 10 or more points, an improvement in New York Heart Association class, and an improvement of 30 minutes or more on the 6-minute walk distance.[36] The trial reported significant improvements in KCCQ scores, New York Heart Association class, and functional capacity at 12 months. However, no difference was observed in 1-year mortality or HF hospitalization. Higher rate of severe bleeding (15.4%) and permanent pacemaker implantation (17.4%) occurred in the TTVR group.[36]

While T-TEER and TTVR show promise, current evidence regarding the reduction of major events remains unclear. Additionally, even for improvements in QoL, the lack of blinding in existing studies introduces uncertainty, highlighting the need to consider potential ascertainment bias and the risk of overattributing benefits. No direct head-to-head trials comparing T-TEER and TTVR exist (**Figure 2**). Current data involves heterogeneous patients and require careful interpretation.[30,52] Key observations include (1) T-TEER safety events are fewer than TTVR, with lower rates of complications such as new pacemakers, bleeding, and acute surgical conversion[30,52]; (2) TR reduction to mild occurs in approximately

Figure 3. A Proposed Management Algorithm for Patients With Severe Tricuspid Valve Regurgitation (TR)



OMT indicates optimal medical therapy; T-TEER, transcatheter edge to edge repair; TTVR, transcatheter tricuspid valve replacement.

95% of TTVR patients regardless of baseline severity, whereas only about 50% of TriClip T-TEER patients achieve mild TR, with efficacy inversely related to initial TR severity[30,52]; (3) a volume-outcome relationship exists for T-TEER procedures, with better results observed in operators with more than 20 procedures,[61] whereas no such relationship has been established for TTVR[36]; and (4) no mortality benefit for transcatheter TV interventions.

Choice Between T-TEER vs TTVR

Both T-TEER and TTVR rely heavily on TEE imaging; careful anatomical assessment is essential to determine which transcatheter therapy will achieve the least TR possible. Analyses of retrospective data show that in T-TEER patients, survival rates are notably lower in those with moderate-severe residual TR compared to those with mild-moderate residual TR (restricted mean survival time difference, 0.27; 95% CI, 0.07-0.47; $P = .006$).[67] According to the FDA based on the TRILUMINATE randomized clinical trial, the goal is to achieve moderate or lower TR; however, emerging trends and the Tricuspid Valve Academic Research Consortium recommend reducing to mild or lower TR.[12] Current imaging standards classify TV anatomy into favorable, feasible, and challenging for T-TEER based on TEE (eTable 2 in the Supplement).[58,68,69] Gercek et al[70] defined the GLIDE score (gap ≥6 mm, jet location, image quality, chordal density, TR morphology) to predict success in T-TEER. Patients with a low GLIDE score (0-1) showed the highest success rate (≥2 grade TR reduction and TR moderate or lower postprocedure). In addition, coaptation gap and TV morphology more than 3 leaflet segments are both predictors of reduced procedural success after T-TEER.[68,69] TTVR offers the potential for TR elimination, but patient selection depends on evaluating comorbidities, RV function, and anatomical suitability via cardiac CT.[4,71] Because TTVR carries higher periprocedural risks, it is important to carefully evaluate the patient's overall tolerance to the procedure. Procedural considerations include venous access, atrial and ventricular size, caval offset, annular shape, leaflet morphology, interactions with CIED leads, and the use of detailed imaging for preprocedural and intraprocedural planning.

Beyond clinical efficacy, several factors influence the choice of transcatheter TV therapy. While T-TEER generally has a better safety profile, its procedural time tends to be longer than TTVR,[30,52] and it can be challenging with CIED leads. Conversely, TTVR traps the lead, risking malfunction[72] or hindrance of extraction, with more than 25% of patients potentially needing alternative pacing[73]; it also requires anticoagulation to prevent thrombosis,[52] and its durability remains uncertain. Eliminating TR may acutely increase afterload and risk RV failure, but such complications are rare, and improvements in forward stroke volume and RV ejection fraction can still occur despite reduced RV function.[74] Advances in TEE and intracardiac echocardiography imaging have improved the reproducibility, safety, and efficiency of both procedures,[66,75] and successful TTVR after failed T-TEER has been reported,[66] with postprocedural CT showing well-expanded prostheses.[76] Both therapies require ongoing OMT to prevent TR recurrence and RV dysfunction. A multidisciplinary assessment that considers clinical, anatomical, and imaging factors is critical to determine the most appropriate treatment approach (**Figure 3**).[26]

Management of CIED Leads and Conduction Disturbances

The optimal approach to CIED-related TR remains uncertain. A TRILUMINATE subgroup analysis[77] found that T-TEER in patients with or without CIEDs had a similar TR reduction (≤moderate: 88% vs

87%). However, these results might only represent highly selected anatomy, as a substantial portion of CIED-related TR is deemed anatomically unfavorable for T-TEER. In fact, only 16% of patients (n = 28/175) had a preexisting CIED lead in the TRILUMINATE trial, compared to nearly 40% (n = 99/259) in the TRISCEND II trial.[30,36] The decision to extract or entrap CIED lead remains a risk-benefit consideration that should involve an electrophysiologist experienced in lead extraction.[73] In patients with CIED-induced TR, lead extraction is unlikely to improve severity. Factors associated with higher complication rates after lead extraction include female sex, lower body mass index, multiple leads extracted, longer implant duration (>10 years), and operator inexperience.

A risk specific to patients undergoing TTVR is new onset conduction disturbances, depending on the implanted device and can be as high as around 20%.[52,57] Preprocedural discussion of alternative pacing strategies following TTVR should involve the multidisciplinary heart team, including an electrophysiologist, and should consider valve-sparing options, such as leadless pacemakers, coronary sinus leads, or epicardial leads.[73] The optimal pacing method depends on individual patient needs, center expertise, and the specific TTVR device used. Lead extraction to facilitate T-TEER and TTVR is infrequent and may damage the TV leaflets, precluding transcatheter procedures. Specifically, lifelong management of CIEDs following T-TEER or TTVR requires careful planning, particularly in younger patients. Advances in transcatheter tricuspid annuloplasty may provide a CIED-friendly option for treating TR. Rapid advances in leadless pacemakers and extravascular implantable defibrillators may provide new options for long-term treatment of patients needing CIEDs after TV interventions.

Future Directions

Further research should focus on several key areas to address significant knowledge gaps and optimize patient outcomes. First, standardized diagnostic criteria and robust risk stratification tools are needed to improve the accuracy of TR assessment and predict patient outcomes. Incorporating advanced imaging modalities and artificial intelligence techniques for imaging analysis to guide treatment strategy is forthcoming.[78] Second, defining the optimal timing for surgical or transcatheter intervention will be critical, and earlier treatment before TR-induced cardiac damage may be beneficial.[79] Third, large-scale, blinded randomized clinical trials with long-term follow-up to assess the hard event reduction and treatment durability would be important. Fourth, in patients deemed unsuitable for transcatheter TV interventions, high-risk surgery may be feasible in operable patients. More contemporary data on isolated TV surgery would benefit the community in identifying suitable patients for surgical intervention.

Conclusions

Management of severe TR is challenging, necessitating a multidisciplinary heart team approach (Figure 3). Importantly, this team should have extensive experience in diagnosis, and management of these patients, with access to surgical and transcatheter options and device trials. While commercial surgical and transcatheter options exist, ongoing research is crucial to refine diagnostic and patient selection criteria, optimize treatment strategies, and improve long-term outcomes.

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REFERENCES

1. Offen S, Playford D, Strange G, Stewart S, Celermajer DS. Adverse prognostic impact of even mild or moderate tricuspid regurgitation: insights from the National Echocardiography Database of Australia. *J Am Soc Echocardiogr*. 2022;35(8):810-817. doi:10.1016/j.echo.2022.04.003

2. Singh JP, Evans JC, Levy D, et al. Prevalence and clinical determinants of mitral, tricuspid, and aortic regurgitation (the Framingham Heart Study). *Am J Cardiol*. 1999;83(6):897-902. doi:10.1016/S0002-9149(98)01064-9

3. Antunes MJ, Rodríguez-Palomares J, Prendergast B, et al; ESC Working Groups of Cardiovascular Surgery and Valvular Heart Disease. Management of tricuspid valve regurgitation: position statement of the European Society of Cardiology Working Groups of Cardiovascular Surgery and Valvular Heart Disease. *Eur J Cardiothorac Surg*. 2017;52(6):1022-1030. doi:10.1093/ejcts/ezx279

4. Hahn RT. Tricuspid Regurgitation. *N Engl J Med*. 2023;388(20):1876-1891. doi:10.1056/NEJMra2216709

5. Hahn RT, Badano L, Praz F, et al. The last decade in tricuspid regurgitation: how imaging shaped a field. *JACC Cardiovasc Imaging*. 2025;18(9):1013-1041. doi:10.1016/j.jcmg.2025.03.003

6. Hell MM, Emrich T, Kreidel F, et al. Computed tomography imaging needs for novel transcatheter tricuspid valve repair and replacement therapies. *Eur Heart J Cardiovasc Imaging*. 2021;22(6):601-610. doi:10.1093/ehjci/jeaa308

7. Zhan Y, Debs D, Khan MA, et al. Natural history of functional tricuspid regurgitation quantified by cardiovascular magnetic resonance. *J Am Coll Cardiol*. 2020;76(11):1291-1301. doi:10.1016/j.jacc.2020.07.036

8. Dahou A, Levin D, Reisman M, Hahn RT. Anatomy and physiology of the tricuspid valve. *JACC Cardiovasc Imaging*. 2019;12(3):458-468. doi:10.1016/j.jcmg.2018.07.032

9. Hahn RT, Waxman AB, Denti P, Delhaas T. Anatomic relationship of the complex tricuspid valve, right ventricle, and pulmonary vasculature: a review. *JAMA Cardiol*. 2019;4(5):478-487. doi:10.1001/jamacardio.2019.0535

10. Hahn RT, Weckbach LT, Noack T, et al. Proposal for a standard echocardiographic tricuspid valve nomenclature. *JACC Cardiovasc Imaging*. 2021;14(7):1299-1305. doi:10.1016/j.jcmg.2021.01.012

11. Praz F, Borger MA, Lanz J, et al; ESC/EACTS Scientific Document Group. 2025 ESC/EACTS guidelines for the management of valvular heart disease. *Eur Heart J*. 2025;2025:ehaf194. doi:10.1093/eurheartj/ehaf194

12. Hahn RT, Lawlor MK, Davidson CJ, et al; TVARC Steering Committee. Tricuspid valve academic research consortium definitions for tricuspid regurgitation and trial endpoints. *J Am Coll Cardiol*. 2023;82(17):1711-1735. doi:10.1016/j.jacc.2023.08.008

13. Hahn RT, Lindenfeld J, Böhm M, et al. Tricuspid regurgitation in patients with heart failure and preserved ejection fraction: JACC state-of-the-art review. *J Am Coll Cardiol*. 2024;84(2):195-212. doi:10.1016/j.jacc.2024.04.047

14. Galloo X, Dietz MF, Fortuni F, et al. Prognostic implications of atrial vs. ventricular functional tricuspid regurgitation. *Eur Heart J Cardiovasc Imaging*. 2023;24(6):733-741. doi:10.1093/ehjci/jead016

15. Prihadi EA, van der Bijl P, Gursoy E, et al. Development of significant tricuspid regurgitation over time and prognostic implications: new insights into natural history. *Eur Heart J*. 2018;39(39):3574-3581. doi:10.1093/eurheartj/ehy352

16. Andreas M, Burri H, Praz F, et al. Tricuspid valve disease and cardiac implantable electronic devices. *Eur Heart J*. 2024;45(5):346-365. doi:10.1093/eurheartj/ehad783

17. Zoghbi WA, Adams D, Bonow RO, et al. Recommendations for noninvasive evaluation of native valvular regurgitation: a report from the American Society of Echocardiography developed in collaboration with the Society for Cardiovascular Magnetic Resonance. *J Am Soc Echocardiogr*. 2017;30(4):303-371. doi:10.1016/j.echo.2017.01.007

18. Lancellotti P, Pibarot P, Chambers J, et al; Scientific Document Committee of the European Association of Cardiovascular Imaging. Multi-modality imaging assessment of native valvular regurgitation: an EACVI and ESC council of valvular heart disease position paper. *Eur Heart J Cardiovasc Imaging*. 2022;23(5):e171-e232. doi:10.1093/ehjci/jeab253

19. Hahn RT, Zamorano JL. The need for a new tricuspid regurgitation grading scheme. *Eur Heart J Cardiovasc Imaging*. 2017;18(12):1342-1343. doi:10.1093/ehjci/jex139

20. Tomaselli M, Penso M, Badano LP, et al. Association with outcomes of correcting the proximal isovelocity surface area method to quantitate secondary tricuspid regurgitation. *J Am Soc Echocardiogr*. 2025;38(3):195-207. doi:10.1016/j.echo.2024.10.015

21. Hahn RT, Saric M, Faletra FF, et al. Recommended standards for the performance of transesophageal echocardiographic screening for structural heart intervention: from the American Society of Echocardiography. *J Am Soc Echocardiogr*. 2022;35(1):1-76. doi:10.1016/j.echo.2021.07.006

22. Tang GHL, Zaid S, Hahn RT, et al. Structural heart imaging using 3-dimensional intracardiac echocardiography: JACC: cardiovascular imaging position statement. *JACC Cardiovasc Imaging*. 2025;18(1):93-115. doi:10.1016/j.jcmg.2024.05.012

23. van Rosendael PJ, Kamperidis V, Kong WK, et al. Computed tomography for planning transcatheter tricuspid valve therapy. *Eur Heart J*. 2017;38(9):665-674. doi:10.1093/eurheartj/ehw499

24. Schulz-Menger J, Bluemke DA, Bremerich J, et al. Standardized image interpretation and post-processing in cardiovascular magnetic resonance—2020 update: Society for Cardiovascular Magnetic Resonance (SCMR): Board of Trustees Task Force on Standardized Post-Processing. *J Cardiovasc Magn Reson*. 2020;22(1):19. doi:10.1186/s12968-020-00610-6

25. Brener MI, Lurz P, Hausleiter J, et al. Right ventricular-pulmonary arterial coupling and afterload reserve in patients undergoing transcatheter tricuspid valve repair. *J Am Coll Cardiol*. 2022;79(5):448-461. doi:10.1016/j.jacc.2021.11.031

26. Hahn RT, Adamo M, Fam NP. Current evidence on tricuspid regurgitation interventions in heart failure. *JACC Heart Fail*. 2025;13(8):102493. doi:10.1016/j.jchf.2025.04.008

27. Miura M, Alessandrini H, Alkhodair A, et al; TriValve Investigators. Impact of massive or torrential tricuspid regurgitation in patients undergoing transcatheter tricuspid valve intervention. *JACC Cardiovasc Interv*. 2020;13(17):1999-2009. doi:10.1016/j.jcin.2020.05.011

28. Lurz P, Rommel KP, Schmitz T, et al. Real-world 1-year results of tricuspid edge-to-edge repair from the BRIGHT study. *J Am Coll Cardiol*. 2024;84(7):607-616. doi:10.1016/j.jacc.2024.05.006

29. Nickenig G, Weber M, Lurz P, et al. Transcatheter edge-to-edge repair for reduction of tricuspid regurgitation: 6-month outcomes of the TRILUMINATE single-arm study. *Lancet*. 2019;394(10213):2002-2011. doi:10.1016/S0140-6736(19)32600-5

30. Sorajja P, Whisenant B, Hamid N, et al; TRILUMINATE Pivotal Investigators. Transcatheter repair for patients with tricuspid regurgitation. *N Engl J Med*. 2023;388(20):1833-1842. doi:10.1056/NEJMoa2300525

31. Otto CM, Nishimura RA, Bonow RO, et al; Writing Committee Members. 2020 ACC/AHA Guideline for the Management of Patients With Valvular Heart Disease: a report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. *J Am Coll Cardiol*. 2021;77(4):e25-e197. doi:10.1016/j.jacc.2020.11.018

32. Vahanian A, Beyersdorf F, Praz F, et al; ESC/EACTS Scientific Document Group. 2021 ESC/EACTS guidelines for the management of valvular heart disease. *Eur Heart J*. 2022;43(7):561-632. doi:10.1093/eurheartj/ehab395

33. Patlolla SH, Schaff HV, Nishimura RA, et al. Incidence and burden of tricuspid regurgitation in patients with atrial fibrillation. *J Am Coll Cardiol*. 2022;80(24):2289-2298. doi:10.1016/j.jacc.2022.09.045

34. Mustapic I, Bakovic D, Susilovic Grabovac Z, Borovac JA. Impact of SGLT2 inhibitor therapy on right ventricular function in patients with heart failure and reduced ejection fraction. *J Clin Med*. 2022;12(1):42. doi:10.3390/jcm12010042

35. Yang Y, Shen C, Lu J, Fu G, Xiong C. Sacubitril/valsartan in the treatment of right ventricular dysfunction in patients with heart failure with reduced ejection fraction: a real-world study. *J Cardiovasc Pharmacol*. 2022;79(2):177-182. doi:10.1097/FJC.0000000000001162

36. Hahn RT, Makkar R, Thourani VH, et al; TRISCEND II Trial Investigators. Transcatheter valve replacement in severe tricuspid regurgitation. *N Engl J Med*. 2025;392(2):115-126. doi:10.1056/NEJMoa2401918

37. Orban M, Rommel KP, Ho EC, et al. Transcatheter edge-to-edge tricuspid repair for severe tricuspid regurgitation reduces hospitalizations for heart failure. *JACC Heart Fail*. 2020;8(4):265-276. doi:10.1016/j.jchf.2019.12.006

38. El-Eshmawi A, Tang GH, Verma S, Yanagawa B, Ruel M, Adams DH. Innovations in tricuspid valve intervention. *Curr Opin Cardiol*. 2017;32(2):166-173. doi:10.1097/HCO.0000000000000377

39. Alnajar A, Chatterjee S, Olive JK, et al. Outcomes of minimally invasive isolated tricuspid valve repair and replacement through right mini-thoracotomy. *JTCVS Open*. 2023;17:98-110. doi:10.1016/j.xjon.2023.12.009

40. Bhasin R, Nisivaco S, Rybar D, Kitahara H, Balkhy HH. Robotic totally endoscopic tricuspid valve surgery: early results and midterm outcomes. *Innovations (Phila)*. 2025;20(2):188-193. doi:10.1177/15569845251326593

41. Gammie JS, Chu MWA, Falk V, et al; CTSN Investigators. Concomitant tricuspid repair in patients with degenerative mitral regurgitation. *N Engl J Med*. 2022;386(4):327-339. doi:10.1056/NEJMoa2115961

42. Axtell AL, Bhambhani V, Moonsamy P, et al. Surgery does not improve survival in patients with isolated severe tricuspid regurgitation. *J Am Coll Cardiol*. 2019;74(6):715-725. doi:10.1016/j.jacc.2019.04.028

43. Zack CJ, Fender EA, Chandrashekar P, et al. National trends and outcomes in isolated tricuspid valve surgery. *J Am Coll Cardiol*. 2017;70(24):2953-2960. doi:10.1016/j.jacc.2017.10.039

44. Scotti A, Sturla M, Granada JF, et al. Outcomes of isolated tricuspid valve replacement: a systematic review and meta-analysis of 5,316 patients from 35 studies. *EuroIntervention*. 2022;18(10):840-851. doi:10.4244/EIJ-D-22-00442

45. Thourani VH, Bonnell L, Wyler von Ballmoos MC, et al. Outcomes of isolated tricuspid valve surgery: a Society of Thoracic Surgeons analysis and risk model. *Ann Thorac Surg*. 2024;118(4):873-881. doi:10.1016/j.athoracsur.2024.04.014

46. Chen Q, Bowdish ME, Malas J, et al. Isolated tricuspid operations: the society of thoracic surgeons adult cardiac surgery database analysis. *Ann Thorac Surg*. 2023;115(5):1162-1170. doi:10.1016/j.athoracsur.2022.12.041

47. Society of Thoracic Surgeons. Risk Calculator—Isolated Tricuspid Valve Surgery STS. Accessed October 30, 2025. https://isolatedtvsurgcalc.research.sts.org/

48. Tang GH, David TE, Singh SK, Maganti MD, Armstrong S, Borger MA. Tricuspid valve repair with an annuloplasty ring results in improved long-term outcomes. *Circulation*. 2006;114(1)(suppl):I577-I581. doi:10.1161/CIRCULATIONAHA.105.001263

49. Carino D, Sala A, Zancanaro E, et al. Surgical treatment of tricuspid valve regurgitation in patients with cardiac implantable electronic devices: long-term results. *Eur J Cardiothorac Surg*. 2022;62(3):ezac239. doi:10.1093/ejcts/ezac239

50. Anselmi A, Ruggieri VG, Harmouche M, et al. Appraisal of long-term outcomes of tricuspid valve replacement in the current perspective. *Ann Thorac Surg*. 2016;101(3):863-871. doi:10.1016/j.athoracsur.2015.09.081

51. Park SJ, Oh JK, Kim SO, et al. Determinants of clinical outcomes of surgery for isolated severe tricuspid regurgitation. *Heart*. 2021;107(5):403-410. doi:10.1136/heartjnl-2020-317715

52. Webb JG, Chuang AM, Meier D, et al. Transcatheter tricuspid valve replacement with the EVOQUE system: 1-year outcomes of a multicenter, first-in-human experience. *JACC Cardiovasc Interv*. 2022;15(5):481-491. doi:10.1016/j.jcin.2022.01.280

53. FDA approval letter Edwards EVOQUE tricuspid valve replacement system. Accessed May 20, 2025. https://www.fda.gov/medical-devices/recently-approved-devices/edwards-evoque-tricuspid-valve-replacement-system-p230013

54. FDA approval letter TriClip G4 system. Accessed May 20, 2025. https://www.accessdata.fda.gov/cdrh_docs/pdf23/P230007A.pdf.

55. US Centers for Medicare & Medicaid Services. Transcatheter edge-to-edge repair for tricuspid valve regurgitation (T-TEER). Published on July 2, 2025. Accessed on July 7, 2025. https://www.cms.gov/medicare-coverage-database/view/ncacal-decision-memo.aspx?proposed=N&ncaid=316

56. US Centers for Medicare & Medicaid Services. Transcatheter tricuspid valve replacement (TTVR). Published on March 19, 2025. Accessed on March 20, 2025. https://www.cms.gov/medicare-coverage-database/view/ncacal-tracking-sheet.aspx?ncaid=314

57. Stolz L, Cheung A, Boone R, et al. Transjugular transcatheter tricuspid valve replacement: early compassionate use outcomes. *JACC Cardiovasc Interv*. 2024;17(16):1936-1945. doi:10.1016/j.jcin.2024.06.014

58. So KC-y, Xu J, Kam KK-h, et al. Current status of tricuspid valve interventions in Asia Pacific region. *JACC Asia*. 2025;5(3 pt 2):405-423. doi:10.1016/j.jacasi.2024.10.008

59. Fam NP, Ong G, Estevez-Loureiro R, et al. Transcatheter tricuspid valve replacement with the cardiovalve system. *JACC Cardiovasc Interv*. 2024;17(4):586-588. doi:10.1016/j.jcin.2023.12.023

60. Sorajja P, Gorgorishvili I, Burns M, et al. First-in-human description of a novel transcatheter tricuspid valve prosthesis to preserve the asymmetric shape of the right ventricle. *EuroIntervention*. 2023;19(8):662-663. doi:10.4244/EIJ-D-23-00079

61. Wild MG, Stolz L, Rosch S, et al; PASTE Investigators. Transcatheter valve repair for tricuspid regurgitation: 1-year results from a large European real-world registry. *J Am Coll Cardiol*. 2025;85(3):220-231. doi:10.1016/j.jacc.2024.10.068

62. Wang L, Tokhi B, Petrossian GA, et al. Streamlined imaging and procedural workflow for tricuspid transcatheter edge-to-edge repair. *JACC Cardiovasc Imaging*. 2025;18(4):500-510. doi:10.1016/j.jcmg.2025.01.010

63. Spertus JA, Jones PG, Sandhu AT, Arnold SV. Interpreting the Kansas City Cardiomyopathy Questionnaire in clinical trials and clinical care: JACC state-of-the-art review. *J Am Coll Cardiol*. 2020;76(20):2379-2390. doi:10.1016/j.jacc.2020.09.542

64. Arnold SV, Goates S, Sorajja P, et al; TRILUMINATE Pivotal Trial Investigators. Health status after transcatheter tricuspid-valve repair in patients with severe tricuspid regurgitation. *J Am Coll Cardiol*. 2024;83(1):1-13. doi:10.1016/j.jacc.2023.10.008

65. Kar S, Makkar RR, Whisenant BK, et al; TRILUMINATE Pivotal Investigators. Two-year outcomes of transcatheter edge-to-edge repair for severe tricuspid regurgitation: the TRILUMINATE Pivotal Randomized Controlled Trial. *Circulation*. 2025;151(23):1630-1638. doi:10.1161/CIRCULATIONAHA.125.074536

66. Safi LM, Lerakis S, Tang GHL, et al. Intraprocedural guidance for transcatheter tricuspid valve replacement in challenging scenarios. *JACC Cardiovasc Imaging*. 2025;18(3):382-388. doi:10.1016/j.jcmg.2025.01.002

67. Dreyfus J, Taramasso M, Kresoja KP, et al; TRIGISTRY Investigators. Prognostic implications of residual tricuspid regurgitation grading after transcatheter tricuspid valve repair. *JACC Cardiovasc Interv*. 2024;17(12):1485-1495. doi:10.1016/j.jcin.2024.04.023

68. Sugiura A, Tanaka T, Kavsur R, et al. Leaflet configuration and residual tricuspid regurgitation after transcatheter edge-to-edge tricuspid repair. *JACC Cardiovasc Interv*. 2021;14(20):2260-2270. doi:10.1016/j.jcin.2021.07.048

69. Mehr M, Taramasso M, Besler C, et al. 1-Year Outcomes after edge-to-edge valve repair for symptomatic tricuspid regurgitation: results from the TriValve registry. *JACC Cardiovasc Interv*. 2019;12(15):1451-1461. doi:10.1016/j.jcin.2019.04.019

70. Gerçek M, Narang A, Körber MI, et al. GLIDE score: scoring system for prediction of procedural success in tricuspid valve transcatheter edge-to-edge repair. *JACC Cardiovasc Imaging*. 2024;17(7):729-742. doi:10.1016/j.jcmg.2024.04.008

71. Tanaka T, Vogelhuber J, Öztürk C, et al. Eligibility for transcatheter tricuspid valve interventions in patients with tricuspid regurgitation. *JACC Cardiovasc Interv*. 2024;17(23):2732-2744. doi:10.1016/j.jcin.2024.09.019

72. So KC, Stolz L, Fam N, et al. Transjugular transcatheter tricuspid valve replacement in patients with cardiac implantable electronic devices. *JACC Asia*. 2025;5(10):1260-1269. doi:10.1016/j.jacasi.2025.07.009

73. Hahn RT, Wilkoff BL, Kodali S, et al; Heart Valve Collaboratory and Heart Rhythm Society. Managing implanted cardiac electronic devices in patients with severe tricuspid regurgitation: JACC state-of-the-art review. *J Am Coll Cardiol*. 2024;83(20):2002-2014. doi:10.1016/j.jacc.2024.02.045

74. Weckbach LT, Stolz L, Chatfield AG, et al. Right ventricular reverse remodeling after transcatheter tricuspid valve replacement in patients with heart failure. *J Am Coll Cardiol*. 2023;81(7):708-710. doi:10.1016/j.jacc.2022.12.005

75. Hahn RT, Makkar R, Makar M, et al. EVOQUE tricuspid valve replacement system: state-of-the-art screening and intraprocedural guidance. *JACC Cardiovasc Interv*. 2024;17(18):2093-2112. doi:10.1016/j.jcin.2024.07.034

76. Penta B, Tang GHL, Onishi T, et al. Computed tomographic analysis of the EVOQUE transcatheter tricuspid valve replacement system: a first-in-human series. *JACC Cardiovasc Interv*. 2025;18(11):1492-1494. doi:10.1016/j.jcin.2025.04.002

77. Naik H, Price MJ, Kapadia S, et al. Tricuspid transcatheter edge-to-edge repair in patients with transvalvular CIED leads: the TRILUMINATE pivotal trial. *JACC Clin Electrophysiol*. 2025;11(5):1012-1020. doi:10.1016/j.jacep.2025.01.001

78. Xie S, Liu H, Su L, et al. A deep learning-based method for assessing tricuspid regurgitation using continuous wave Doppler spectra. *Sci Rep*. 2024;14(1):27483. doi:10.1038/s41598-024-78861-x

79. Dietz MF, Prihadi EA, van der Bijl P, Ajmone Marsan N, Delgado V, Bax JJ. Prognostic implications of staging right heart failure in patients with significant secondary tricuspid regurgitation. *JACC Heart Fail*. 2020;8(8):627-636. doi:10.1016/j.jchf.2020.02.008