Transcatheter and surgical management of tricuspid valve disease: multidisciplinary lifetime management considerations

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Tricuspid valve disease (TVD) has long been underrecognized compared with left-sided valvular disorders, yet it represents a major determinant of cardiovascular morbidity and mortality. Tricuspid regurgitation (TR), the predominant manifestation of TVD, is being recognized with increasing frequency as populations age and the burden of left-sided heart disease rises. While mild TR is often physiological, moderate and severe TR are associated with adverse outcomes, independent of left ventricular function or pulmonary pressures. Secondary TR, driven by right atrial or right ventricular remodeling, constitutes the majority of cases, whereas primary TR due to intrinsic valvular pathology is less frequent. Cardiac implantable electronic device (CIED)-related TR represents a distinct and increasingly prevalent mechanism that often requires dedicated management considerations. Historically, management of TVD has often been delayed until the onset of advanced right heart failure or end-organ dysfunction, resulting in poor outcomes. Contemporary evidence emphasizes the importance of early recognition, precise etiologic characterization, and timely intervention within a multidisciplinary framework. Surgical repair, particularly annuloplasty, remains the reference standard in suitable candidates, offering durable results when performed before irreversible right ventricular remodeling develops. Transcatheter tricuspid valve interventions have expanded therapeutic options for high-risk or inoperable patients, demonstrating symptomatic and hemodynamic improvement in early studies. Optimal management of TVD follows a lifetime approach, integrating multimodality imaging, risk stratification, and individualized treatment strategies. General physicians and cardiologists play a key role in early detection, while coordinated collaboration among imaging specialists, electrophysiologists, heart failure experts, interventional cardiologists, cardiac surgeons, and anesthesiologists is essential for comprehensive care. Ultimately, a patient-centered lifetime management strategy initiated early and adapted to disease progression offers the best opportunity to preserve right heart function, improve survival, and maintain quality of life in patients with TVD.

Keywords: Tricuspid valve disease (TVD); management; surgery; intervention; prognosis

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Introduction

The tricuspid valve (TV) has long been referred to as the "forgotten valve" (1). Historically, tricuspid valve disease (TVD) received limited clinical and research attention compared with left-sided valve disorders, but it is now recognized as a major determinant of cardiovascular outcomes (2-6). Tricuspid regurgitation (TR) is frequently detected incidentally on routine echocardiography and represents the most common valvular heart disease in older adults, as demonstrated in contemporary population-based prevalence studies (7). While mild TR is often considered a benign physiological finding, moderate or severe TR carries adverse prognostic implications and is associated with increased mortality and heart failure hospitalizations. The progression from mild to clinically significant TR is influenced by age and sex, with a higher prevalence in elderly patients and women (8).

The epidemiology of TVD has shifted considerably over recent decades, largely due to the decline in rheumatic heart disease and the growing recognition of secondary TR. Only a small proportion (<10%) of patients in developed countries present with primary TR, characterized by anatomical abnormalities of the TV apparatus (8). Secondary TR, caused by right ventricular (RV) or right atrial (RA) remodeling, represents the vast majority of cases.

TVD has often been managed late in the disease course, when RV dysfunction and end-organ dysfunction are advanced, limiting the benefits of intervention (9-11). Contemporary evidence now emphasizes early recognition, timely referral, and multidisciplinary evaluation to optimize outcomes. A multidisciplinary Heart Team approach at Heart Valve Centers involving cardiologists, imaging specialists, electrophysiologists, heart failure experts, cardiac surgeons, and anesthesiologists is essential to ensure accurate risk stratification, individualized therapy, and optimal timing of interventions.

Moreover, TVD acts both as a marker and a driver of disease progression, supporting a model of lifelong, multidisciplinary management that integrates evolving technologies, patient comorbidities, and symptom relief (11-13).

This review summarizes contemporary surgical and transcatheter management of TVD, with emphasis on multidisciplinary decision-making, patient selection, and lifetime management strategies aimed at improving long-term outcomes and quality of life.

Classification of tricuspid valve regurgitation

The TV is the largest and most apically positioned valve and is a complex, dynamic structure. Its functional anatomy, similar to that of the mitral valve, can be divided into four components: leaflets, fibrous annulus (with attached RA and RV), chordal attachments, and papillary muscles (14,15). The functional integrity of this apparatus depends on the interplay between leaflet motion, annular geometry, and RV contractility. Disruption of this balance through ventricular, annular, or atrial remodeling leads to secondary TR. The right coronary artery and the atrioventricular node lie in close anatomical proximity to the tricuspid annulus, representing critical structures at risk during both surgical and transcatheter interventions.

TR is classified as primary or secondary depending on whether the underlying pathology originates from intrinsic abnormalities of the leaflets and/or subvalvular apparatus or from geometric and functional alterations of the annulus and surrounding cardiac structures (*Table 1*) (2-6).

Primary TR etiologies include rheumatic disease, infective endocarditis, congenital malformations such as Ebstein's anomaly, carcinoid heart disease, radiation-induced valvulopathy, myxomatous degeneration, or iatrogenic injury (e.g., after endomyocardial biopsy) (8).

Secondary TR represents the vast majority of patients presenting with TR. With an aging population, improved survival of patients with left-sided heart disease, and increasing prevalence of heart failure with preserved ejection fraction (HFpEF), the occurrence of secondary TR is rising (16). The leaflets are structurally normal in secondary TR, and regurgitation arises from annular dilatation and/or leaflet tethering secondary to RA and/or RV remodeling. Two main phenotypes are recognized: (I) atrial secondary TR, typically due to atrial fibrillation or HFpEF, characterized by annular dilatation with preserved RV geometry and function; and (II) ventricular secondary TR, due to annular dilatation and leaflet tethering as a consequence of left-sided valvular or ventricular disease, pulmonary hypertension, or primary RV pathology. As the disease advances, these phenotypes frequently overlap, underscoring the need for early and precise etiologic classification to guide management.

Cardiac implantable electronic device (CIED)-related TR has become increasingly prevalent due to the increasing use of CIEDs and represents a distinct clinical entity.

Table 1 Pathophysiological classification of tricuspid regurgitation

Classification	Leaflet morphology	Pathophysiology	Etiology	Imaging
Primary	Abnormal	Loss of leaflet coaptation due to intrinsic changes, excessive mobility, or perforation	- Myxomatous disease - Endocarditis - Rheumatic heart disease - Trauma - Carcinoid - Iatrogenic (biopsy) - Congenital	- According to etiology - Description of etiology, lesions, and dysfunction
Secondary				
Atrial	Normal	RA enlargement and dysfunction leading to annular dilation and remodeling of the RV	- Atrial fibrillation - (HFpEF)	- Severe RA remodeling - Basal RV diameter may be enlarged despite normal RV volume - Leaflet tethering absent or limited
Ventricular	Considered normal	RV enlargement and/or dysfunction leading to leaflet tethering and annular dilation	- Left ventricular systolic dysfunction - Left-sided valvular heart disease - Pulmonary hypertension - RV cardiomyopathy - RV infarction	Dominant mechanism is leaflet tethering ± annular dilation
CIED-related				
Primary	Abnormal	- Leaflet impingement - Leaflet/chordal entanglement - Leaflet adherence - Leaflet laceration/perforation - Leaflet avulsion (post lead extraction)	- Pacemaker - Implantable cardiac defibrillator - Cardiac resynchronization therapy	3D echocardiography (± color)
Secondary	Normal	RV enlargement and/or dysfunction/dyssynchrony due to pacemaker stimulation leading to significant leaflet tethering and annular dilation	Chronic RV pacing	Dominant mechanism is leaflet tethering ± annular dilation

CIED, cardiac implantable electronic device; HFpEF, heart failure with preserved ejection fraction; RA, right atrium/right atrial; RV, right ventricle/right ventricular.

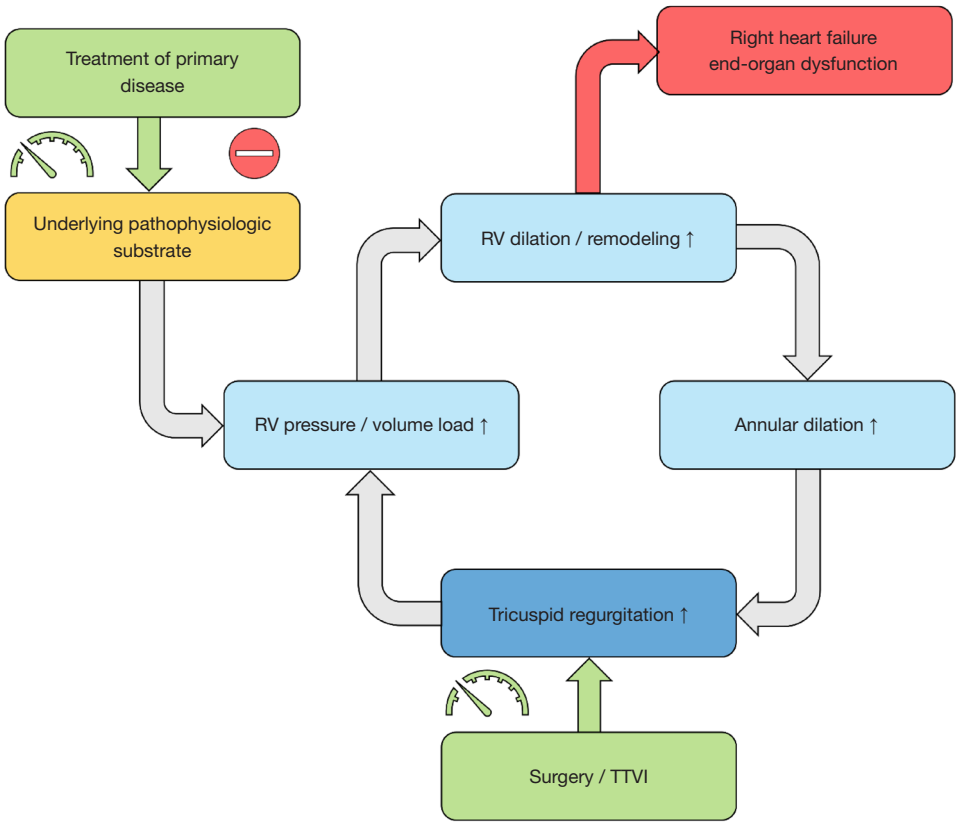

Figure 1 Progression of tricuspid regurgitation and main therapeutic targets. RV, right ventricle/right ventricular; TTVI, transcatheter tricuspid valve intervention.

Determining whether the lead is causative (CIED-related TR) or a bystander (CIED-associated TR) is essential, as mechanisms such as lead impingement, entanglement, or adherence can directly interfere with leaflet motion and may worsen TR.

Natural history and clinical course of TR

Severe TR is increasingly recognized as both a phenotypic manifestation ("marker") and a pathophysiologic contributor ("driver") of adverse cardiovascular outcomes, independently associated with increased mortality and heart failure hospitalizations regardless of ventricular function, pulmonary pressures, or comorbidities (17). The natural course of TR is characterized by progressive deterioration, evolving from a compensated to a decompensated state. In the early stages, mild TR is often well tolerated and may remain asymptomatic for years, particularly when secondary to left-sided heart disease or pulmonary hypertension. During this phase, RV size and function are typically preserved, and functional TR may regress if the underlying cause, such as left-sided valvular pathology or atrial fibrillation, is effectively treated. However, persistent pressure or volume overload promotes RV dilation, annular enlargement, and leaflet tethering, which perpetuate regurgitation and initiate a vicious cycle of progressive TR, worsening RV dysfunction, and venous congestion (*Figure 1*). Breaking this cycle early is key to preventing irreversible right-sided remodeling. In advanced stages, progressive venous congestion causes peripheral edema, ascites, and hepatic dysfunction. Once severe RV failure and end-organ impairment develop, prognosis is poor and therapeutic options are limited, highlighting the need for early recognition and intervention (18).

Diagnostic evaluation of tricuspid valve regurgitation

Echocardiography remains the cornerstone for the evaluation of TVD, providing comprehensive information

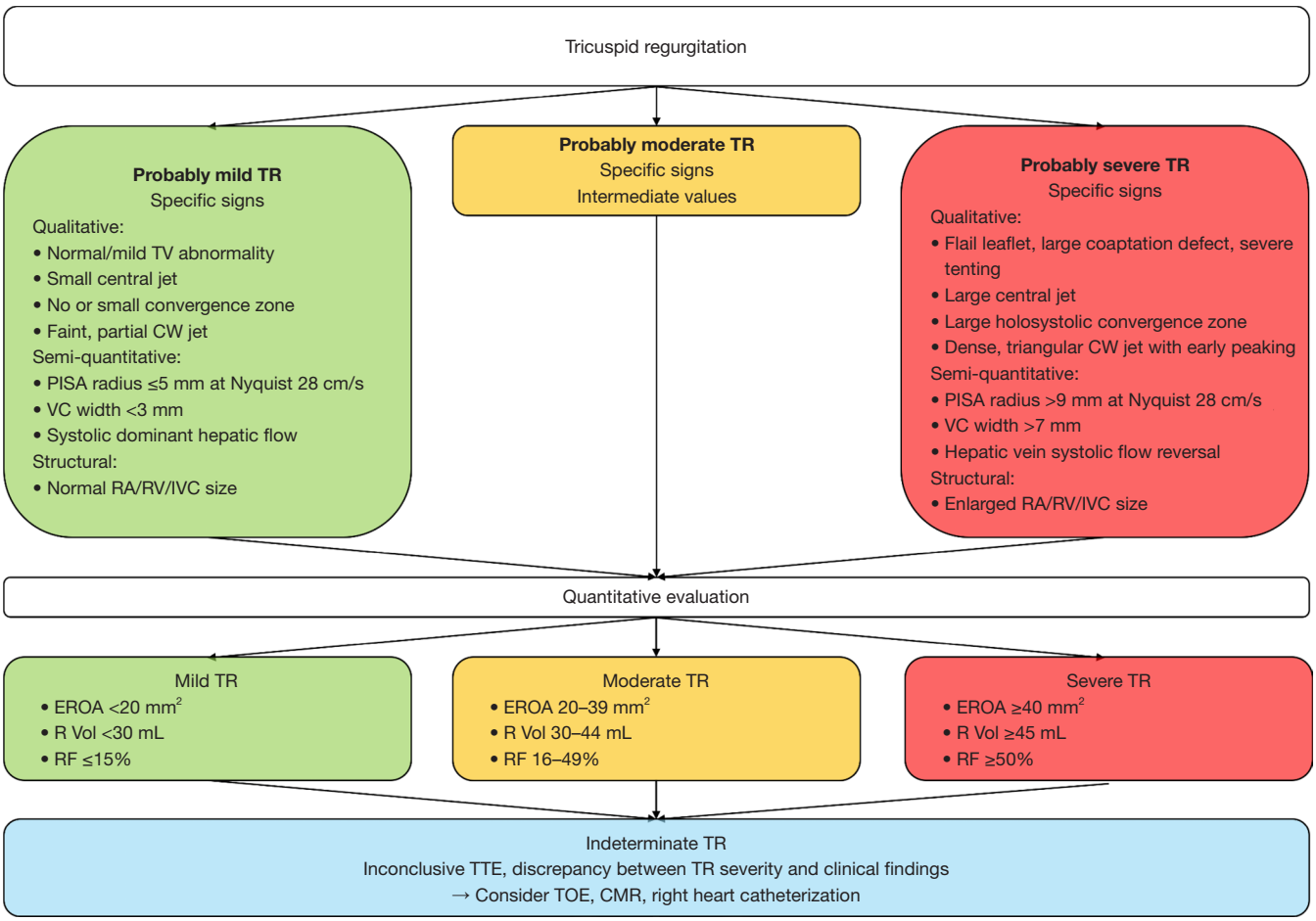

Figure 2 Algorithm for the assessment of tricuspid regurgitation severity. Adapted according to (15). CMR, cardiac magnetic resonance imaging; CW, continuous wave; EROA, effective regurgitant orifice area; IVC, inferior vena cava; PISA, proximal isovelocity surface area; R Vol, regurgitant volume; RA, right atrium/right atrial; RF, regurgitant fraction; RV, right ventricle/right ventricular; TOE, transesophageal echocardiography; TR, tricuspid regurgitation; TTE, transthoracic echocardiography; TV, tricuspid valve; VC, vena contracta.

on the etiology, severity, and hemodynamic consequences (14,15). A grading scheme extending beyond "severe" to include "massive" and "torrential" TR has been introduced to refine post-interventional assessment and improve correlation with clinical outcomes (19). This expanded TR severity classification, as proposed by the Tricuspid Valve Academic Research Consortium (TVARC), is not included in current guideline-based grading and is primarily applied in the context of transcatheter tricuspid valve interventions (TTVI) to better characterize advanced disease and procedural success.

TR should be evaluated in an optimal volume state, with optimized systemic and pulmonary pressures. The integrated multiparametric approach combines qualitative, semi-quantitative, and quantitative indices (*Figure 2*) (14,15). Transthoracic echocardiography generally provides sufficient diagnostic information, including TR severity, mechanism, right and left ventricular size and function, and the presence of cardiac damage and concomitant left-sided valve disease. Transesophageal echocardiography allows the detailed evaluation of leaflet morphology and function (including mobility, tethering, and coaptation gaps) as well as number, size, and location of TR jets, annular morphology and size, and subvalvular anatomy. Such details are essential for procedural planning and device selection.

Cardiac magnetic resonance (CMR) imaging serves as the reference standard for accurate quantification of RV volume and function and is particularly valuable when echocardiographic findings are inconclusive or discordant (20,21). Beyond quantification of TR severity and RV

volumes, CMR enables assessment of TR etiology and identification of structural confounding factors that may influence the prognosis of TV repair, including RV myocardial strain abnormalities and septal or RV fibrosis (22). When available, 4D flow CMR may provide additional insights into regurgitant flow characteristics and RV loading conditions (23).

Right heart catheterization provides direct hemodynamic assessment of RA, RV, and pulmonary pressures. This information is essential to exclude severe pre-capillary pulmonary hypertension, guiding therapeutic decision-making and refining risk assessment, particularly in those patients in whom an isolated TV intervention is being considered.

Electrocardiogram-gated cardiac computed tomography allows exact annular sizing and characterization of the RA, RV, as well as vena cava anatomy, and is crucial for procedural planning.

Beyond anatomical and hemodynamic evaluation, comprehensive risk stratification is essential. The Society of Thoracic Surgeons (STS) isolated TV risk calculator predicts in-hospital mortality after isolated tricuspid surgery (24). The TRI-SCORE can be used in combination with the STS score, and is particularly useful in identifying patients in whom TV intervention is futile (25). Integration of these models with imaging and clinical parameters supports individualized decision-making within the Heart Team framework.

Surgical management

Historically, patients with TVD have been referred too late for surgical intervention, often after the onset of advanced RV dysfunction or secondary end-organ failure (9-11). As a result, earlier reports of isolated TV surgery consisted mostly of patients with high operative risk and correspondingly poor outcomes. However, more contemporary studies have demonstrated that earlier referral, improved perioperative care, and advanced surgical techniques have markedly improved outcomes, with contemporary operative mortality decreasing from >10% to <5% with low rates of major complications such as stroke and renal failure (26,27). In addition, minimally invasive approaches and TV surgery performed with a beating heart strategy have emerged as safe and effective alternatives in high-volume, experienced centers (28,29). Hepatic impairment represents an important contributor to adverse outcomes after tricuspid intervention and the Model for End-Stage Liver Disease (MELD) score appears to provide additional information beyond traditional surgical risk scores (30).

Whenever feasible, valve repair is preferred over valve replacement as outcomes are consistently superior for repair compared with replacement (31,32). Annuloplasty is highly effective in secondary TR caused by annular dilation or when CIED leads do not impede leaflet mobility (33,34). Targeted valve repair may be required in cases of structural pathology such as endocarditis, trauma, or degenerative disease and offers several advantages over valve replacement, including preservation of the native subvalvular apparatus, and maintenance of RV geometry. Valve replacement becomes necessary when repair is not technically feasible, such as in cases of severe leaflet tethering or extreme annular dilatation. Comparative studies show similar mid-term survival and valve durability between mechanical and bioprosthetic prostheses, although mechanical prostheses carry higher thromboembolic and bleeding risk (35). Bioprosthetic valves are therefore preferred, particularly given the low-pressure right-sided circulation and growing feasibility of transcatheter valve-in-valve reintervention.

CIED-related TR requires individualized management. In selected cases, mobilization or extraction of impinging or entangled leads and conversion to epicardial pacing can restore valve function and prevent recurrence. Close collaboration between electrophysiologists and surgeons is essential in these cases, especially with regards to indication and timing of post-CIED removal reimplantation (36).

A recognized drawback of tricuspid annuloplasty is the risk of conduction disturbances, particularly if rigid rings are used (37,38). To prevent damage to a newly repaired or replaced valve during subsequent transvenous lead displacement, there is a low threshold for prophylactic placement of permanent epicardial leads at the time of TV surgery, particularly if heart block is present in the operating room. However, CIED implantation has been associated with higher long-term rates of heart failure hospitalization, infective endocarditis, and mortality, emphasizing the need for careful patient selection (37). In this context, placement of a prophylactic epicardial pacing lead at the time of surgery, which can be tunneled transthoracically and stored in a subcutaneous pocket, may preserve future pacing options while avoiding transvalvular leads that could damage the prosthesis or increase the risk of infection.

Concomitant TV repair during left-sided valve surgery remains a cornerstone of modern management. Isolated correction of left-sided valve disease rarely leads to durable

improvement of coexistent TR, and reoperation for late TR after prior cardiac surgery carries significant risk (39). Approximately 25% of patients with uncorrected moderate TR or significant annular dilatation at the time of left-sided surgery develop progressive regurgitation, resulting in worse long-term outcomes (40,41). Large studies and randomized trials consistently demonstrate that concomitant tricuspid annuloplasty limits TR progression and promotes reverse RV remodeling (42-44). In the most recent randomized trial by Gammie *et al.*, patients undergoing mitral valve surgery with concomitant tricuspid repair had a significantly lower incidence of the composite primary endpoint of TR progression, reoperation, or death at 2 years compared with mitral surgery alone, despite a higher rate of permanent pacemaker implantation. The protective effect of TV repair was particularly strong in patients with moderate TR. Whether reduced TR progression translates into long-term survival benefit remains under investigation (45).

Both European and American guidelines support a proactive surgical approach (2,3). According to the 2025 ESC/EACTS guidelines, tricuspid repair is recommended for severe primary or secondary TR at the time of left-sided valve surgery (Class I), and tricuspid repair should be considered for moderate secondary TR (Class IIa) or mild TR with annular dilatation (Class IIb) (2). The American College of Cardiology (ACC)/American Heart Association (AHA) 2021 guidelines similarly recommend concomitant repair in progressive TR with annular dilatation or right-sided heart failure symptoms (3).

For isolated TR, European guidelines recommend early surgery in symptomatic severe primary TR before severe RV dysfunction develops (Class I) and consideration of surgery in asymptomatic severe TR with progressive RV dilation or dysfunction (Class IIa) (2). The American guidelines are conceptually aligned but apply slightly more conservative thresholds to avoid intervention in patients with advanced RV or end-organ failure (3).

In summary, durable surgical repair, particularly ring annuloplasty, remains the reference standard when performed before irreversible RV remodeling. A multidisciplinary Heart Team at dedicated Heart Valve Centers ensures appropriate timing, optimized perioperative management, and coordination with future transcatheter options.

TTVI

TTVI have transformed the management of high-risk or inoperable patients with severe TR (46,47).

The current device landscape can be broadly divided into four categories: (I) tricuspid transcatheter edge-to-edge repair (T-TEER); (II) transcatheter annuloplasty devices; (III) orthotopic transcatheter tricuspid valve replacement (TTVR) systems; and (IV) heterotopic transcatheter systems for caval valve implantation. Among these, T-TEER is the most established approach, with the TriClip system (Abbott) receiving both Food and Drug Administration (FDA) and CE approval, while the PASCAL system (Edwards Lifesciences, Irvine, CA, USA), which has CE mark approval in Europe and a strong clinical evidence base, represents another widely used T-TEER device. Transcatheter annuloplasty devices, such as the Cardioband system (Edwards Lifesciences), have obtained CE approval only. In the field of valve replacement, orthotopic TTVR has recently advanced, with the EVOQUE system (Edwards Lifesciences) receiving FDA and CE approval. Heterotopic transcatheter systems for caval valve implantation, including the TricValve® transcatheter bicaval valve system (P&F Products and Features GmbH), have received CE mark approval and FDA Breakthrough Device designation, with U.S. use currently restricted to investigational studies. In the context of TTVI, a new TR grading scheme has been developed, which includes two more grades (massive and torrential TR) beyond severe (*Table 2*) (19). This is due to the fact that "severe TR" does not capture the true range of pathology encountered in TTVI candidates, nor the incremental benefits of therapy when TR was reduced but still technically "severe". This refined grading system therefore better stratifies patients with extreme TR and provides a quantitative framework for assessing procedural success and prognosis as outlined in the TVARC document (48). Of note, however, the 2025 ESC/EACTS guidelines on valvular heart disease did not include such grades of extreme TR (2), because the task force wanted clinicians to refer patients before they get to this stage and because "less than severe" should be the minimum goal of any TV intervention.

T-TEER approximates leaflets to improve coaptation and reduce regurgitant orifice area, mimicking the principle of the surgical Alfieri edge-to-edge stitch. Transcatheter annuloplasty replicates the most common surgical repair procedure, aiming to restore annular geometry and reduce dilatation, thereby improving leaflet coaptation while preserving the native valve. Due to the procedural complexity, transcatheter annuloplasty is only performed in very experienced centers and with decreasing frequency

Table 2 Severity of tricuspid regurgitation in the context of transcatheter tricuspid valve interventions

Parameters	Severe	Massive	Torrential
Semi-quantitative parameters			
VC width (mm)[†]	7–<14	14–<21	≥21
3D VC or quantitative Doppler EROA (mm^2)	75–<95	95–<115	≥115
Quantitative parameters			
EROA by PISA (mm^2)[‡]	40–59	60–79	≥80
Regurgitant volume (mL)	40–59	60–74	≥75

[†], at a Nyquist limit of 50–60 cm/s. [‡], baseline Nyquist limit shift of 28 cm/s. EROA, effective regurgitant orifice area; PISA, proximal isovelocity surface area; VC, vena contracta.

over time, despite its compelling rationale. TTVR offers a promising alternative for patients with complex anatomy and advanced disease stages (e.g., large tricuspid annular dimensions or coaptation gaps, excessive leaflet tethering) (47). Heterotopic or caval valve implantation serves primarily a palliative role to reduce venous hypertension when other strategies are unsuitable.

TTVI devices can anchor at various sites (leaflets, annulus, RA, RV, or vena cavae). Comprehensive pre-procedural imaging, combining transthoracic and transesophageal echocardiography with computed tomography is fundamental for selecting appropriate candidates, predicting procedural efficacy, and ensuring technical success. Detailed echocardiographic assessment of leaflet morphology and function (e.g., mobility, tethering, coaptation gaps, and presence of an additional TV leaflet), TR jet grade, size and location, annular morphology and size, and subvalvular anatomy (e.g., location and density of chordae, number/location/height of papillary muscles) is essential (46). Computed tomography provides an anatomical roadmap for TTVI, complementing echocardiography regarding aspects of device delivery (e.g., femoral access, caval veins offset), anchoring, and location of relevant adjacent anatomic structures.

T-TEER includes two widely used systems (TriClip and PASCAL) with the strongest evidence base amongst all forms of TTVI. In the TRILUMINATE Pivotal trial, the TriClip significantly reduced TR severity and improved New York Heart Association (NYHA) class and quality of life compared with optimal medical therapy at 1 year (49). These benefits were sustained through 2 years, at which point a significant reduction in heart failure hospitalizations emerged compared with the control group, while all-cause mortality remained unchanged (50). Similarly, the randomized Tri.Fr trial confirmed symptomatic and quality-of-life improvements with TriClip therapy (51). For the PASCAL device, the CLASP TR trial reported a 30-day mortality of 3% and sustained TR reduction at 1 year, with 86% of patients achieving ≤2+ TR, accompanied by significant improvements in quality of life and functional status (52). Comparable outcomes were observed in the PASTE post-market registry, which included >230 patients, confirming the feasibility, safety, and durability of the PASCAL repair system in a real-world population (53). The TRISCEND II trial demonstrated that orthotopic TTVR with the EVOQUE system was superior to medical therapy alone, driven primarily by improved symptoms and quality of life (54).

Current European Society of Cardiology (ESC)/European Association for Cardio-Thoracic Surgery (EACTS) guidelines recommend considering TTVI for high-risk patients with symptomatic severe TR, provided there is no advanced RV dysfunction or severe pre-capillary pulmonary hypertension (2). Long-term follow-up of previous studies and additional ongoing randomized studies (e.g., CLASP TR, TRICI-HF, TRICAV) will provide further insights into the long-term prognostic impact of these therapies.

Optimal management of TR

TR is a complex, multifactorial condition that serves both as a surrogate of advanced cardiopulmonary disease and as an independent contributor to progressive heart failure and end-organ dysfunction. Its clinical course is frequently complicated by primary underlying or secondary

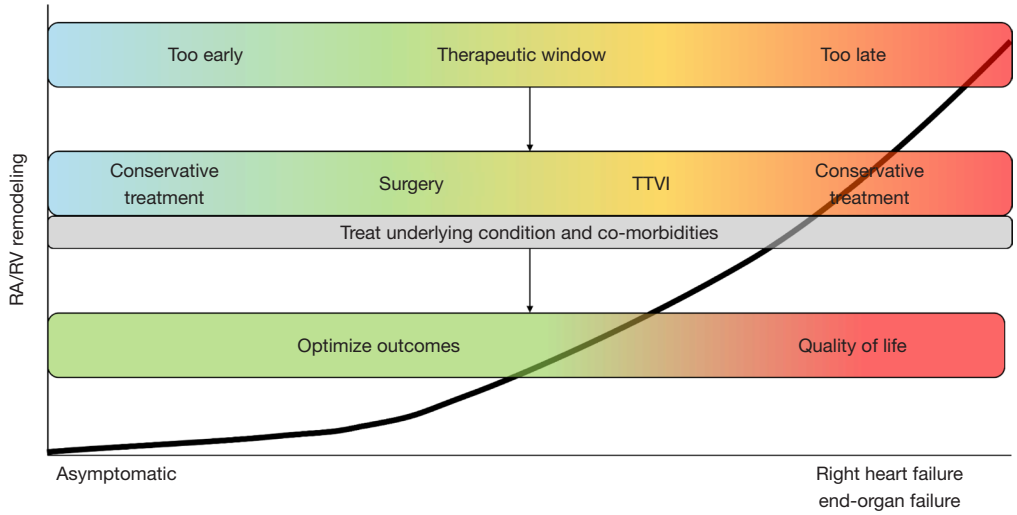

Figure 3 Therapeutic window and treatment strategies in tricuspid regurgitation. RA, right atrium/right atrial; RV, right ventricle/right ventricular; TTVI, transcatheter tricuspid valve intervention.

comorbidities, which affect prognosis and therapy. These complexities highlight the need for integrated, multidisciplinary management at experienced Heart Valve Centers.

Comprehensive evaluation of TR etiology, disease stage, surgical risk, and anatomical suitability for isolated TV procedures within the confines of a multidisciplinary Heart Team is essential, and this has received a class I recommendation in the most recent guidelines (2). Realistic therapeutic goals, whether focused on prognostic benefit, RV remodeling, or symptom relief, should be clearly defined.

The optimal timing of intervention remains a major challenge (*Figure 3*). Contemporary evidence indicates that outcomes are best when therapy is performed before advanced RV dysfunction or irreversible end-organ dysfunction develops, highlighting the concept of a "therapeutic window". Once severe RV or hepatic dysfunction occurs, interventions may still relieve symptoms but seldom improve long-term survival, while the risk for postinterventional right heart failure and suboptimal recovery is increased. Further, quantitative markers of RV remodeling and function, including RV end-diastolic volume index and RV ejection fraction, have been proposed as predictors of outcomes and may help refine the optimal timing of TTVI before irreversible RV dysfunction develops (55).

In primary TR, early diagnosis, close monitoring, and timely surgical intervention are key to preventing progressive right heart failure, systemic deterioration, as well as complicated postoperative courses.

Identifying the etiology of secondary TR, including the distinction between atrial and ventricular forms, has important prognostic implications and therapeutic consequences. Atrial secondary TR, characterized by isolated annular dilation and preserved RV geometry, generally carries a more favorable prognosis and responds well to early repair (56). In the early stages of disease, rhythm control via catheter ablation may improve long-term outcomes (57). It has been demonstrated that in patients with moderate to severe secondary TR and drug-refractory atrial fibrillation, catheter ablation reduced TR severity in over two-thirds of patients (58). A surgical approach is also attractive in lower risk patients, since causal annular dilation and atrial fibrillation can be directly addressed at the time of surgery, as well as concomitant mitral valve repair and left atrial appendage occlusion (59).

Ventricular secondary TR, arising from left-sided heart disease or pulmonary hypertension, is associated with more advanced myocardial remodeling and worse outcomes. These underlying conditions and co-morbidities also independently drive prognosis. Etiology-directed therapy remains the first step, but rarely reverses TR or completely halts its progression. Optimized guideline-directed medical therapy for heart failure is of utmost importance, and TR reduction is also observed after cardiac resynchronization therapy or transcatheter mitral valve repair (60,61). Surgical and transcatheter interventions are additional options to

slow down the cascade of TR and mitigate adverse RV remodeling, thereby improving symptoms and potentially outcomes.

In significant left-sided valvular heart disease, surgical correction with concomitant tricuspid repair remains the cornerstone of management, as it can address both underlying causes and TR itself.

Direct randomized comparisons between surgical and transcatheter approaches are lacking (62). A German multicenter propensity score–matched observational study suggests that minimally invasive TV surgery provides more complete and durable TR reduction compared with T-TEER, albeit with a slightly higher 30-day mortality, while 1-year survival appears comparable between modalities (63). Similarly, among Medicare beneficiaries with TR, the 2-year mortality rate was comparable between T-TEER and surgical repair (64). T-TEER showed advantages in perioperative outcomes, including lower in-hospital mortality and pacemaker implantation rates, whereas tricuspid valve reinterventions were more frequent in the T-TEER group. As in other valve diseases, surgery is associated with a higher upfront procedural risk, whereas transcatheter approaches may show a late catch-up of adverse events. Surgical intervention remains the most definitive treatment option, and TTVI provides a less invasive treatment option for high-risk patients, with growing evidence for symptomatic improvement and quality-of-life benefit. Therefore, TTVI should be viewed as complementary to surgery rather than competitive.

Tricuspid valve stenosis

While TR constitutes the vast majority of TVD, tricuspid stenosis (TS) is discussed briefly for completeness, given its rarity but continued clinical relevance.

TS is most often associated with congenital malformations or metabolic disorders such as Whipple's or Fabry's disease. It may also occur as an acquired condition, either as an isolated manifestation of rheumatic heart disease or in combination with aortic and/or mitral valve involvement. Additional causes include carcinoid heart disease, drug-induced valvulopathy (e.g., fenfluramine or methysergide), and inflow obstruction due to CIED-associated thrombus formation or infective endocarditis with large vegetations.

Echocardiography is the cornerstone of TS diagnosis, providing both anatomical and functional assessment. Leaflet thickening with or without calcification and commissural fusion are characteristic for rheumatic involvement. A mean diastolic transvalvular gradient >5 mmHg at a normal heart rate typically indicates severe TS (65).

Conservative therapy with sodium restriction and diuretics is largely supportive and aims to alleviate symptoms until definitive intervention can be performed. While valve repair is preferred, valve replacement is often necessary (66). Bioprosthetic valves are favored due to the high thrombogenic risk in the low-pressure right-sided circulation. In cases of bioprosthetic degeneration, transcatheter valve-in-valve implantation offers a reasonable alternative to surgical redo (67).

TTVI experience in TS remains limited. Balloon valvuloplasty may be considered in selected patients with isolated TS without significant TR or combined rheumatic mitral and tricuspid disease (68). However, unlike rheumatic mitral stenosis, rheumatic tricuspid pathology frequently involves both stenosis and regurgitation, limiting the applicability and long-term durability of balloon valvuloplasty (2).

Multidisciplinary lifetime management

TVD requires a multidisciplinary and longitudinal management strategy that evolves across the course of the disease. Early detection of TR by general practitioners and cardiologists is critical to initiate timely diagnostic evaluation and optimize comorbid conditions. Early involvement of specialized teams, including imaging experts, heart failure specialists, electrophysiologists, cardiac surgeons, and interventional cardiologists is essential for accurate etiologic classification, timely referral, and coordinated management (2).

A comprehensive diagnostic work-up using multimodality imaging and hemodynamic assessment should define the mechanism and severity of TR, guiding etiology-specific therapy. Conservative management, including optimal heart failure therapy, rhythm control, and treatment of pulmonary hypertension or left-sided valve disease, remains the foundation for early care. When TR progresses despite optimal therapy, timely intervention before irreversible RV remodeling or end-organ dysfunction is vital to improve outcomes. The Heart Team at dedicated Heart Valve Centers serves as the central coordinating structure to determine procedural suitability, anticipate future reinterventions, and ensure continuity of care within a lifetime management model. In the context of

lifetime management, tricuspid valve-in-valve procedures represent an important option for the treatment of failed bioprosthetic valves and should be anticipated at the time of the index intervention. Accordingly, management of CIED leads is a critical consideration when recurrent valve intervention is likely, with strategies including lead extraction, repositioning, or alternative pacing approaches (e.g., epicardial or leadless systems) to preserve future transcatheter access and avoid interference with prosthetic valve function. In advanced stages, when curative options are no longer feasible, management focuses on symptom relief and preservation of quality of life. Ultimately, a patient-centered multidisciplinary strategy initiated early and maintained throughout the disease trajectory offers the best chance to prevent deterioration and sustain quality of life across the continuum of TVD.

Future directions and research priorities

Despite major advances, significant knowledge gaps remain. Standardized TR grading and improved imaging-based quantification are needed to harmonize assessment and follow-up. Determining the optimal timing of intervention remains a key challenge. Integration of computational modeling and multimodal imaging may further refine patient selection and procedural planning. Collaborative registries and large randomized trials are essential to define long-term outcomes, device durability, and comparative effectiveness of surgical and transcatheter approaches.

Conclusions

Once considered the "forgotten valve", TVD is now recognized as a key determinant of cardiovascular outcomes. Early identification, multimodality assessment, and timely intervention guided by dedicated Heart Teams at Heart Valve Centers are essential to improve prognosis. Advances in surgical and transcatheter techniques have transformed management into a lifetime, patient-centered continuum focused on preserving right heart function and quality of life.

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References

1. Hahn RT. Tricuspid Regurgitation. N Engl J Med 2023;388:1876-91.
2. Praz F, Borger MA, Lanz J, et al. 2025 ESC/EACTS Guidelines for the management of valvular heart disease. Eur Heart J 2025;46:4635-736.
3. Otto CM, Nishimura RA, Bonow RO, et al. 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. Circulation 2021;143:e72-e227.
4. Davidson LJ, Tang GHL, Ho EC, et al. The Tricuspid Valve: A Review of Pathology, Imaging, and Current Treatment Options: A Scientific Statement From the American Heart Association. Circulation 2024;149:e1223-38.
5. O'Gara PT, Lindenfeld J, Hahn RT, et al. 10 Issues for the Clinician in Tricuspid Regurgitation Evaluation and Management: 2025 ACC Expert Consensus Decision Pathway: A Report of the American College of Cardiology Solution Set Oversight Committee. J Am Coll Cardiol 2026;87:447-86.
6. Adamo M, Chioncel O, Pagnesi M, et al. Epidemiology, pathophysiology, diagnosis and management of chronic right-sided heart failure and tricuspid regurgitation. A clinical consensus statement of the Heart Failure Association (HFA) and the European Association of Percutaneous Cardiovascular Interventions (EAPCI) of the ESC. Eur J Heart Fail 2024;26:18-33.
7. Cohen D. Defining the prevalence of valvular heart

disease in older Americans: the PREVUE-VALVE study. Presented at: TCT 2025. October 27, 2025. San Francisco, CA.
8. Topilsky Y, Maltais S, Medina Inojosa J, et al. Burden of Tricuspid Regurgitation in Patients Diagnosed in the Community Setting. JACC Cardiovasc Imaging 2019;12:433-42.
9. 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;18:840-51.
10. Dreyfus J, Flagiello M, Bazire B, et al. Isolated tricuspid valve surgery: impact of aetiology and clinical presentation on outcomes. Eur Heart J 2020;41:4304-17.
11. Guenther T, Noebauer C, Mazzitelli D, et al. Tricuspid valve surgery: a thirty-year assessment of early and late outcome. Eur J Cardiothorac Surg 2008;34:402-9; discussion 409.
12. Chorin E, Rozenbaum Z, Topilsky Y, et al. Tricuspid regurgitation and long-term clinical outcomes. Eur Heart J Cardiovasc Imaging 2020;21:157-65.
13. Topilsky Y, Inojosa JM, Benfari G, et al. Clinical presentation and outcome of tricuspid regurgitation in patients with systolic dysfunction. Eur Heart J 2018;39:3584-92.
14. Hahn RT. State-of-the-Art Review of Echocardiographic Imaging in the Evaluation and Treatment of Functional Tricuspid Regurgitation. Circ Cardiovasc Imaging 2016;9:e005332.
15. Lancellotti P, Pibarot P, Chambers J, et al. 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:e171-232.
16. Heitzinger G, Pavo N, Koschatko S, et al. Contemporary insights into the epidemiology, impact and treatment of secondary tricuspid regurgitation across the heart failure spectrum. Eur J Heart Fail 2023;25:857-67.
17. Wang N, Fulcher J, Abeysuriya N, et al. Tricuspid regurgitation is associated with increased mortality independent of pulmonary pressures and right heart failure: a systematic review and meta-analysis. Eur Heart J 2019;40:476-84.
18. Benfari G, Antoine C, Miller WL, et al. Excess Mortality Associated With Functional Tricuspid Regurgitation Complicating Heart Failure With Reduced Ejection Fraction. Circulation 2019;140:196-206.
19. Miura M, Alessandrini H, Alkhodair A, et al. Impact of Massive or Torrential Tricuspid Regurgitation in Patients Undergoing Transcatheter Tricuspid Valve Intervention. JACC Cardiovasc Interv 2020;13:1999-2009.
20. Zhan Y, Senapati A, Vejpongsa P, et al. Comparison of Echocardiographic Assessment of Tricuspid Regurgitation Against Cardiovascular Magnetic Resonance. JACC Cardiovasc Imaging 2020;13:1461-71.
21. Hahn RT, Lerakis S, Delgado V, et al. Multimodality Imaging of Right Heart Function: JACC Scientific Statement. J Am Coll Cardiol 2023;81:1954-73.
22. Ogawa M, Kuwajima K, Yamane T, et al. Prognostic Implication of Right Ventricular Free Wall Longitudinal Strain and Right Atrial Pressure Estimated By Echocardiography in Patients With Severe Functional Tricuspid Regurgitation. J Am Heart Assoc 2024;13:e033196.
23. Sularz A, Negm AS, Chavez Ponce A, et al. Prospective Quantification of Tricuspid Regurgitation With Echocardiography vs 4D Flow Cardiac Magnetic Resonance. JACC Adv 2025;4:101759.
24. O'Brien SM, Feng L, He X, et al. The Society of Thoracic Surgeons 2018 Adult Cardiac Surgery Risk Models: Part 2-Statistical Methods and Results. Ann Thorac Surg 2018;105:1419-28.
25. Dreyfus J, Audureau E, Bohbot Y, et al. TRI-SCORE: a new risk score for in-hospital mortality prediction after isolated tricuspid valve surgery. Eur Heart J 2022;43:654-62.
26. 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;115:1162-70.
27. Pfannmueller B, Budde LM, Etz CD, et al. Postoperative outcome after reoperative isolated tricuspid valve surgery-is there a predictor for survival? Eur J Cardiothorac Surg 2021;60:867-71.
28. Pfannmueller B, Verevkin A, Borger MA, et al. Role of tricuspid valve repair for moderate tricuspid regurgitation during minimally invasive mitral valve surgery. Thorac Cardiovasc Surg 2013;61:386-91.
29. Russo M, Di Mauro M, Saitto G, et al. Beating Versus Arrested Heart Isolated Tricuspid Valve Surgery: Long-term Outcomes. Ann Thorac Surg 2022;113:585-92.
30. Färber G, Marx J, Scherag A, et al. Risk stratification for isolated tricuspid valve surgery assisted using the Model for End-Stage Liver Disease score. J Thorac Cardiovasc Surg 2023;166:1433-1441.e1.
31. Dreyfus J, Juarez-Casso F, Sala A, et al. Benefit of isolated surgical valve repair or replacement for functional tricuspid

regurgitation and long-term outcomes stratified by the TRI-SCORE. Eur Heart J 2024;45:4512-22.
32. Nammalwar S, Tam DY, Alabbadi S, et al. Long-term outcomes of isolated tricuspid surgery in 3706 patients: Implications for the future. J Thorac Cardiovasc Surg 2025;170:1548-1555.e13.
33. Hahn RT, Wilkoff BL, Kodali S, et al. Managing Implanted Cardiac Electronic Devices in Patients With Severe Tricuspid Regurgitation: JACC State-of-the-Art Review. J Am Coll Cardiol 2024;83:2002-14.
34. Andreas M, Burri H, Praz F, et al. Tricuspid valve disease and cardiac implantable electronic devices. Eur Heart J 2024;45:346-65.
35. Negm S, Arafat AA, Elatafy EE, et al. Mechanical Versus Bioprosthetic Valve Replacement in the Tricuspid Valve Position: A Systematic Review and Meta-Analysis. Heart Lung Circ 2021;30:362-71.
36. 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:ezac239.
37. Iribarne A, Alabbadi SH, Moskowitz AJ, et al. Permanent Pacemaker Implantation and Long-Term Outcomes of Patients Undergoing Concomitant Mitral and Tricuspid Valve Surgery. J Am Coll Cardiol 2024;83:1656-68.
38. Pfannmüller B, Doenst T, Eberhardt K, et al. Increased risk of dehiscence after tricuspid valve repair with rigid annuloplasty rings. J Thorac Cardiovasc Surg 2012;143:1050-5.
39. McCarthy PM, Bhudia SK, Rajeswaran J, et al. Tricuspid valve repair: durability and risk factors for failure. J Thorac Cardiovasc Surg 2004;127:674-85.
40. Song H, Kim MJ, Chung CH, et al. Factors associated with development of late significant tricuspid regurgitation after successful left-sided valve surgery. Heart 2009;95:931-6.
41. Kwak JJ, Kim YJ, Kim MK, et al. Development of tricuspid regurgitation late after left-sided valve surgery: a single-center experience with long-term echocardiographic examinations. Am Heart J 2008;155:732-7.
42. Dreyfus GD, Essayagh B, Benfari G, et al. Outcome of consistent guideline-based tricuspid management in patients undergoing degenerative mitral regurgitation correction. JTCVS Open 2021;7:125-38.
43. Pettinari M, De Kerchove L, Lazam S, et al. Mid-term results of a randomized trial of tricuspid annuloplasty for less-than-severe functional tricuspid regurgitation at the time of mitral valve surgery. Eur J Cardiothorac Surg 2019;55:851-8.
44. Dreyfus GD, Corbi PJ, Chan KM, et al. Secondary tricuspid regurgitation or dilatation: which should be the criteria for surgical repair? Ann Thorac Surg 2005;79:127-32.
45. Gammie JS, Chu MWA, Falk V, et al. Concomitant Tricuspid Repair in Patients with Degenerative Mitral Regurgitation. N Engl J Med 2022;386:327-39.
46. Maisano F, Hahn R, Sorajja P, et al. Transcatheter treatment of the tricuspid valve: current status and perspectives. Eur Heart J 2024;45:876-94.
47. Hausleiter J, Stolz L, Lurz P, et al. Transcatheter Tricuspid Valve Replacement. J Am Coll Cardiol 2025;85:265-91.
48. Hahn RT, Lawlor MK, Davidson CJ, et al. Tricuspid Valve Academic Research Consortium Definitions for Tricuspid Regurgitation and Trial Endpoints. J Am Coll Cardiol 2023;82:1711-35.
49. Sorajja P, Whisenant B, Hamid N, et al. Transcatheter Repair for Patients with Tricuspid Regurgitation. N Engl J Med 2023;388:1833-42.
50. Kar S, Makkar RR, Whisenant BK, et al. Two-Year Outcomes of Transcatheter Edge-to-Edge Repair for Severe Tricuspid Regurgitation: The TRILUMINATE Pivotal Randomized Controlled Trial. Circulation 2025;151:1630-8.
51. Donal E, Dreyfus J, Leurent G, et al. Transcatheter Edge-to-Edge Repair for Severe Isolated Tricuspid Regurgitation: The Tri.Fr Randomized Clinical Trial. JAMA 2025;333:124-32.
52. Kodali SK, Hahn RT, Davidson CJ, et al. 1-Year Outcomes of Transcatheter Tricuspid Valve Repair. J Am Coll Cardiol 2023;81:1766-76.
53. Wild MG, Löw K, Rosch S, et al. Multicenter Experience With the Transcatheter Leaflet Repair System for Symptomatic Tricuspid Regurgitation. JACC Cardiovasc Interv 2022;15:1352-63.
54. Arnold SV, Hahn RT, Thourani VH, et al. Quality of Life After Transcatheter Tricuspid Valve Replacement: 1-Year Results From TRISCEND II Pivotal Trial. J Am Coll Cardiol 2025;85:206-16.
55. Kim HK, Kim YJ, Park EA, et al. Assessment of haemodynamic effects of surgical correction for severe functional tricuspid regurgitation: cardiac magnetic resonance imaging study. Eur Heart J 2010;31:1520-8.
56. Galloo X, Dietz MF, Fortuni F, et al. Prognostic implications of atrial vs. ventricular functional tricuspid regurgitation. Eur Heart J Cardiovasc Imaging 2023;24:733-41.

57. Ye Q, Li Y, Zhang W, et al. Catheter Ablation or Surgical Therapy in Severe Atrial Functional Mitral Regurgitation Caused by Long-Standing Persistent Atrial Fibrillation-Propensity Score Analysis. J Am Heart Assoc 2024;13:e035695.
58. Cha MJ, Lee SA, Cho MS, et al. Reduction of moderate to severe tricuspid regurgitation after catheter ablation for atrial fibrillation. Heart 2024;110:523-30.
59. Kagiyama N, Kaneko T, Amano M, et al. Clinical Outcomes of Mitral Valve Surgery in Atrial Functional Mitral Regurgitation in the REVEAL-AFMR Registry. JAMA Netw Open 2024;7:e2428032.
60. Stassen J, Galloo X, Hirasawa K, et al. Tricuspid regurgitation after cardiac resynchronization therapy: evolution and prognostic significance. Europace 2022;24:1291-9.
61. Adamo M, Pagnesi M, Ghizzoni G, et al. Evolution of tricuspid regurgitation after transcatheter edge-to-edge mitral valve repair for secondary mitral regurgitation and its impact on mortality. Eur J Heart Fail 2022;24:2175-84.
62. Saito T, Kuno T, Aikawa T, et al. Long-term outcomes with medical therapy, transcatheter repair, or surgery for isolated tricuspid regurgitation: a systematic review and network meta-analysis. Clin Res Cardiol 2025;114:272-80.
63. Wilde N, Silaschi M, Alirezaei H, et al. Transcatheter edge-to-edge valve repair versus minimally invasive beating-heart surgery of the tricuspid valve: an observational study. EuroIntervention 2023;19:659-61.
64. Shimoda TM, Ueyama HA, Miyamoto Y, et al. Comparison of Transcatheter Versus Surgical Tricuspid Repair Among Patients With Tricuspid Regurgitation: Two-Year Results. Circ Cardiovasc Interv 2025;18:e014825.
65. Baumgartner H, Hung J, Bermejo J, et al. Echocardiographic assessment of valve stenosis: EAE/ASE recommendations for clinical practice. J Am Soc Echocardiogr 2009;22:1-23; quiz 101-2.
66. Cevasco M, Shekar PS. Surgical management of tricuspid stenosis. Ann Cardiothorac Surg 2017;6:275-82.
67. Schamroth Pravda N, Vaknin Assa H, Levi A, et al. Tricuspid Structural Valve Deterioration Treated with a Transcatheter Valve-in-Valve Implantation: A Single-Center Prospective Registry. J Clin Med 2022;11:2667.
68. Yunoki K, Naruko T, Itoh A, et al. Images in cardiovascular medicine. Percutaneous transcatheter balloon valvuloplasty for bioprosthetic tricuspid valve stenosis. Circulation 2006;114:e558-9.

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