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STATE-OF-THE-ART REVIEW

The Last Decade in Tricuspid Regurgitation

How Imaging Shaped a Field

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ABSTRACT

The tricuspid valve has become a major focus of novel structural heart interventions, with the Conformité Européenne approval of 5 devices in Europe and the U.S. Food and Drug Administration approval of 2 devices in the United States. Multiple meta-analyses and large population-based registries have shown that although significant tricuspid regurgitation (TR) often accompanies left heart or pulmonary vascular diseases, it is associated with an increased risk of mortality and a reduced quality of life after adjusting for these comorbidities. Echocardiography remains the imaging modality of choice for diagnosing the etiology and assessing the severity of TR. However, advanced imaging techniques have played an essential role in the rapid advancement of the structural field and, in particular, transcatheter interventions for TR. Herein, we review the advances made in this field, focusing on the role that imaging has played in shaping a new field of study. (JACC Cardiovasc Imaging. 2025;18:1013–1041) © 2025 Published by Elsevier on behalf of the American College of Cardiology Foundation.

In the decade since the first-in-human implantation of a transcatheter annuloplasty device for the treatment of severe, symptomatic tricuspid regurgitation (TR),[1] the "forgotten" tricuspid valve (TV) has become a major focus of novel structural heart interventions, with the Conformité Européenne approval of 5 devices in Europe and U.S. Food and Drug Administration approval of 2 devices in the United States. Large population-based databases show a prevalence of moderate and severe TR of ~5% to 6% and ~1.5% to 2%, respectively.[2,3] In addition, emerging evidence now suggests that secondary TR (STR) is independently linked to adverse outcomes.[4] Although surgical treatment has been associated with high in-hospital mortality rates,[5] transcatheter device therapy has proven to be effective in reducing TR and improving functional and quality of life measures.[6,7]

The central issues for the field have been to: 1) understand the anatomy and pathophysiology of a

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ABBREVIATIONS AND ACRONYMS

3DE = 3-dimensional echocardiography

CIED = cardiac implantable electronic device

CMR = cardiac magnetic resonance

CTA = computed tomography angiography

EROA = effective regurgitant orifice area

MPR = multiplanar reconstruction

PASP = pulmonary artery systolic pressure

PISA = proximal isovelocity surface area

RVEF = right ventricular ejection fraction

STR = secondary tricuspid regurgitation

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

TAPSE = tricuspid annular plane systolic excursion

TEE = transesophageal echocardiography

TEER = transcatheter edge-to-edge repair

TTE = transthoracic echocardiography

TTVI = transcatheter tricuspid valve intervention

TTVR = transcatheter tricuspid valve replacement

VCA = vena contracta area

heterogeneous disease; 2) quantify the disease process involving the valve, the right ventricle, and the right atrium; 3) identify patients at risk for adverse outcomes to determine the optimal timing of intervention; and 4) refine patient selection for surgical or transcatheter intervention to optimize outcomes.

The ongoing advancements in our understanding of TR pathophysiology and outcomes have accelerated the development of transcatheter therapies. Enhanced cardiac imaging techniques have provided deeper insights into disease mechanisms, established novel methods for assessing TV as well as right heart structure and function, introduced a new classification system for TR etiology, and developed innovative approaches for quantifying and grading TR severity.[8] The current paper examines the pivotal role of imaging in shaping this evolving field and presents a glimpse into the future of imaging applications in TR (**Central Illustration**).

ANATOMY OF THE TV APPARATUS

ECHOCARDIOGRAPHIC ASSESSMENT OF TV ANATOMY. Echocardiography remains the first-line imaging modality for TR. Because of the anterior and inferior positions of the TV in the mediastinum, either transthoracic echocardiography (TTE) (**Figure 1**) or transesophageal echocardiography (TEE) (**Figure 2**) can be used for imaging the TV with 2-dimensional (2D) or 3-dimensional echocardiography (3DE) techniques. Advanced 3DE has emerged as a groundbreaking innovation, transforming our approach to imaging the TV. The multiple 3DE display modes,[9] including biplane imaging, volume and surface rendering, and multiplanar reconstruction (MPR), have enhanced our understanding and ability to quantify the complex TV[10] and subvalvular apparatus morphology,[11,12] as well as cardiac chambers (**Figures 3 and 4**).[13-16] 3DE MPR enables tomographic reconstruction of multiple long- or short-axis imaging planes from within the 3DE data set and is integral to accurately image anatomy and disease severity irrespective of the 2D acquisition plane (**Figure 5**).[17-19] 3DE MPR of color Doppler data sets can quantify TR severity irrespective of the shape of the regurgitant orifice or the presence of device therapies.[19] 3DE MPR as well as other imaging advances such as echocardiographic-fluoroscopic fusion imaging (**Figure 2**, Video 1) have become important tools for guiding transcatheter TV interventions.[20,21]

MULTIMODALITY IMAGING OF TV ANATOMY. Cardiac computed tomography angiography (CTA) is an integral part of preprocedural evaluation for transcatheter tricuspid valve replacement (TTVR),[21,22] with growing evidence of its utility for transcatheter repair[23,24] providing pre-procedural prediction of the main fluoroscopic angles, caval offset, and catheter simulation for intervention, right ventricular (RV) size and global contractile function,[24,25] and segmentation of the tricuspid annulus and its distance from surrounding structures.[26] It can confirm echocardiographic assessments such as leaflet morphology, coaptation gaps, tenting height, leaflet tenting angles, and anatomical regurgitant orifice area (**Figure 6**, Video 2).[23,27]

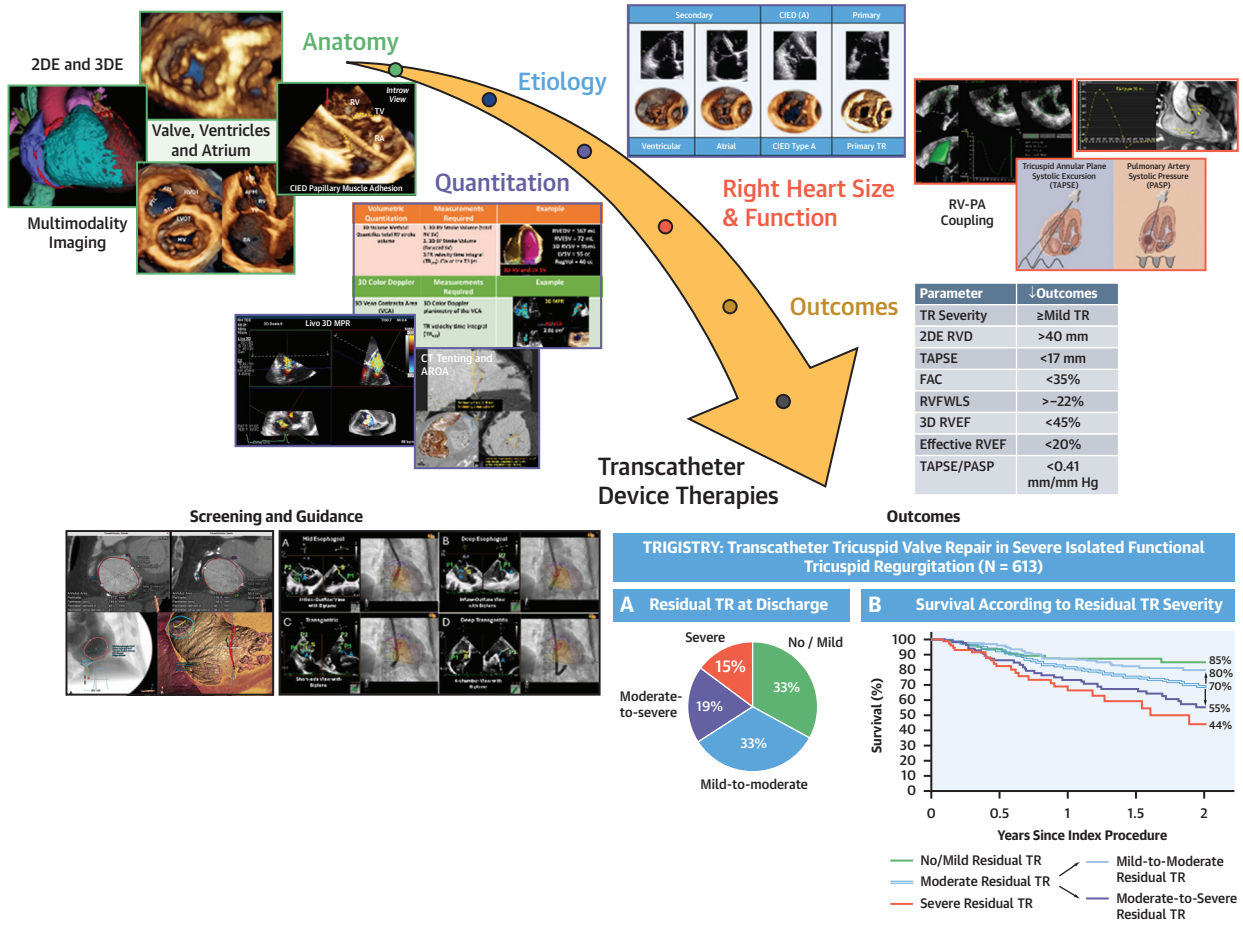
Cardiac magnetic resonance (CMR) can assess right-sided remodeling, structure, and function.[28,29] Identifying myocardial fibrosis (ischemic or nonischemic) or other infiltrative pathologies such as cardiac sarcoidosis or amyloidosis using myocardial delayed enhancement has implications for disease management and prognosis, beyond that of TR itself. Although infrequently required given the advancements in 3DE capabilities, CMR can be used when echocardiographic imaging is limited to evaluate RV remodeling and contractile function, which is a critical prognostic parameter in patients being considered for percutaneous TV intervention (**Figure 7**, Video 3).[30,31]

NOVEL CLASSIFICATION OF TR ETIOLOGY

The novel classification proposed by the PCR Tricuspid Focus group[32] and the TVARC (Tricuspid Valve Academic Research Consortium)[33] now stratifies regurgitant disease into primary TR, atrial-STR, ventricular-STR, and cardiac implantable electronic device (CIED)-related TR (**Figure 8**), relying heavily on imaging to make the diagnosis. Primary TR is due to direct TV leaflet involvement. Conversely, in STR, TV leaflets are structurally normal, and regurgitation results either from the predominant dilation and dysfunction of: 1) the right atrium and tricuspid annulus (ie, atrial-STR); or 2) the right ventricle (ie, ventricular-STR).[34] The distinction is important given the better survival associated with atrial-STR.[35,36]

Although a number of different definitions of atrial-STR have been used in the published reports

CENTRAL ILLUSTRATION **Role of Imaging in the Evolution of Tricuspid Regurgitation Diagnosis and Management**



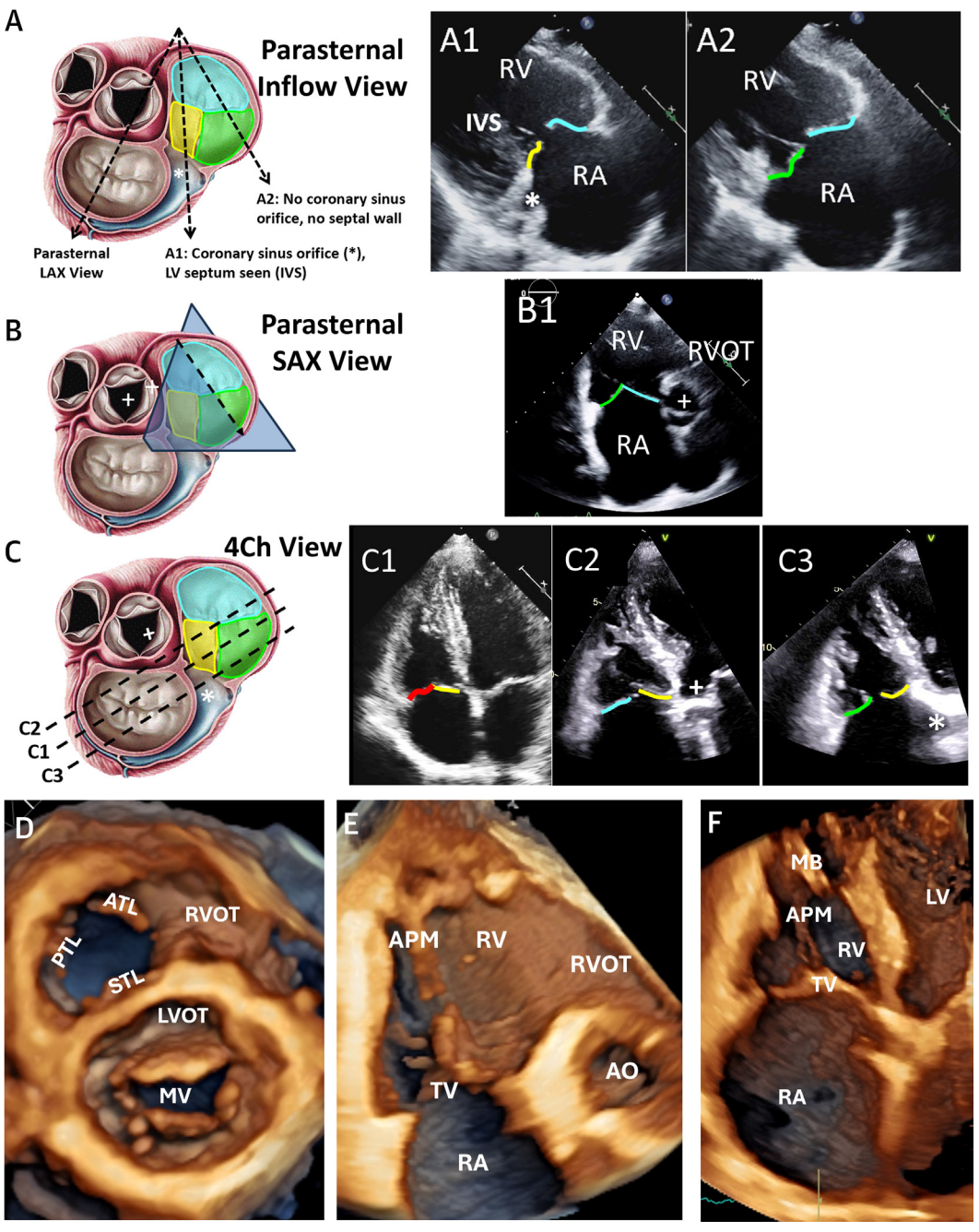
Hahn RT, et al. JACC Cardiovasc Imaging. 2025;18(9):1013-1041.

Advanced imaging techniques have significantly advanced our understanding of both normal and abnormal anatomy of the tricuspid valve and right heart, as well as the classification of the causes of tricuspid regurgitation (TR). Novel quantification methods and grading schemes may enhance the detection and facilitate earlier treatment of the condition. Assessing appropriate measures of right heart function can help predict outcomes and determine the timing of interventions. Furthermore, imaging has been crucial in evaluating the feasibility and efficacy of transcatheter device therapies. 2DE = 2-dimensional echocardiography; 3DE = 3-dimensional echocardiography; FAC = fractional area change; PASP = pulmonary artery systolic pressure; RV = right ventricle; RVD = right ventricular basal diameter; RVEF = right ventricular ejection fraction; RVFWLS = right ventricular free-wall longitudinal strain; RV-PA = right ventricular-pulmonary arterial; TAPSE = tricuspid annular plane systolic excursion; TV = tricuspid valve.

(Table 1),[37] TAVRC[33] and the PCR Tricuspid Focus group[34] have suggested a unifying definition of atrial and ventricular STR (Table 2). The most recent studies have attempted to simplify these criteria, highlighting the importance of the echocardiographic-measured ratio of right atrial (RA) to RV volumes or area, in combination with left ventricular (LV) and/or RV function to help define these subcategories and predict outcomes.[38,39] A CIED lead may directly cause TR (CIED-causative) by interfering with leaflet mobility either by direct mechanical impingement or entanglement of any part of the valve apparatus, by causing structural damage to the TV (leading to significant regurgitation), or by causing pacing-induced RV dysfunction.[40] Three-dimensional (3D) imaging plays a key role in making the diagnosis (Figure 9).

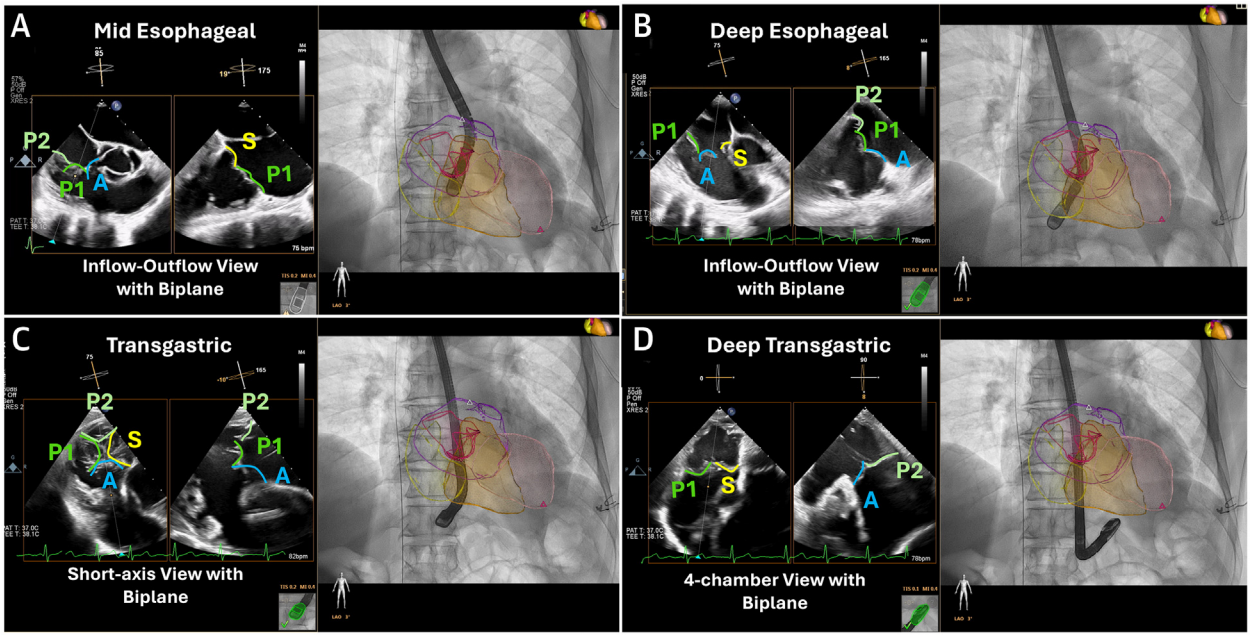
The outcomes of TR vary significantly depending on its etiology.[35] Echocardiography plays a pivotal role in the classification of TR by identifying key features such as leaflet morphology and mobility, annular dilation, and chamber remodeling and function.[8] The use of multimodality imaging and right

FIGURE 1 Transthoracic Imaging Views of the TV



(A to C) Position of the probe on the short-axis cartoon is shown on the left, with associated 2-dimensional imaging on the right. The parasternal inflow view (A) is obtained by angulating the probe from the parasternal long-axis view, toward the right hip; the coronary sinus ostium (asterisk) may be imaged (A1) in addition to the muscular IVS, allowing imaging of the anterior (blue) and septal (yellow) TV leaflets. With the transducer angled more acutely inferiorly and to the right with no IVS seen (A2), the anterior (blue) and posterior (green) leaflets are imaged. The parasternal SAX view (B) will typically image the anterior and posterior leaflets (PTLs). From the apical RV-focused view (C and C1), the septal leaflet and a lateral (red) leaflet are imaged; to image the anterior leaflet, the probe is angled more superiorly and the LVOT (plus sign) may be seen (C2). To image the PTL, the probe is angled inferiorly (C3), and the coronary sinus (asterisk) may be seen. (D to F) 3-dimensional echocardiography rendering of the right heart allows for a more reproducible and accurate identification of the tricuspid valve leaflets and apparatus by reconstructing SAX views (D), inflow-outflow views (E), and apical views (F). 4Ch = 4-chamber; AO = aorta; APM = anterior papillary muscle; ATL = anterior leaflet; IVS = interventricular ventricular septum; LAX = long-axis; LVOT = left ventricular outflow tract; MV = mitral valve; RA = right atrium; RV = right ventricle; RVOT = right ventricular outflow tract; SAX = short-axis; STL = septal leaflet; TV = tricuspid valve.

FIGURE 2 **Multilevel Transesophageal Echocardiographic Imaging of the TV**



There are 4 transesophageal imaging levels of the TV: mid-esophageal (A), deep esophageal (B), transgastric (C), and deep transgastric (D). In this example of a quadricuspid TV with 2 posterior leaflets (P and P2), identification of the leaflets may be more nuanced. Simultaneous multiplane imaging from the mid-esophageal or deep esophageal levels help clarify which leaflet is imaged. The anterior leaflet (A, blue line) is typically seen adjacent to the aorta, the septal leaflet (S, yellow line) attached to the interventricular septum, and the P2 leaflet (light green) is adjacent to the coronary sinus. The P1 leaflet (dark green line) is the most lateral leaflet, directly opposite the septal leaflet. The fusion imaging on the right side of the panels shows the right lateral and retro flexion that may be required for some patients. Modified from Hausleiter et al.[22] Abbreviation as in Figure 1.

heart catheterization has enhanced diagnostic precision and offers detailed information about the consequences of TR on the right heart structures, function, and hemodynamics.[34]

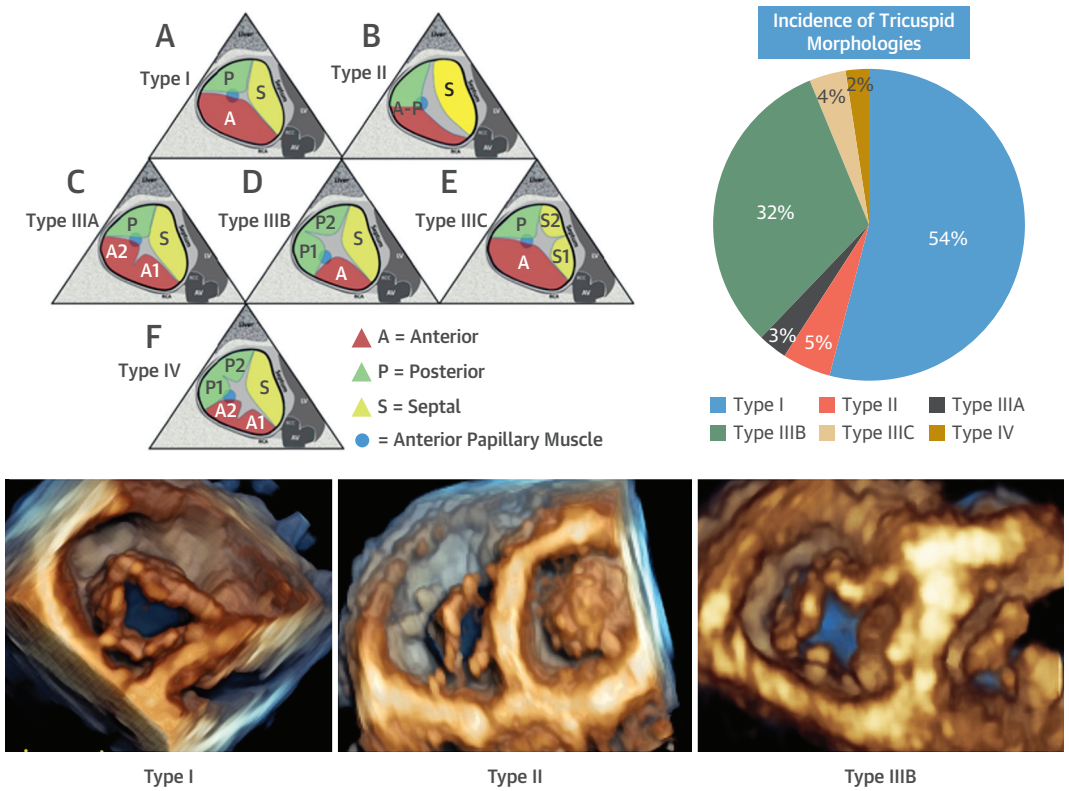
NEW DEFINITIONS OF TR SEVERITY

STANDARD TR GRADING. Traditionally, TR severity has been categorized as mild, moderate, and severe by integrating various structural, qualitative, semiquantitative, and quantitative criteria (Figure 10).[41] Quantification of TR should always be attempted, with the most validated parameter being the effective regurgitant orifice area (EROA) measured by using proximal isovelocity surface area (PISA) (Figure 11). However, the multiple limitations of the PISA methodology should be acknowledged.[42] Correction of the PISA quantitation, by including a parameter for correcting leaflet angle and the low systolic velocity, suggest that there is significant underestimation of TR severity using the uncorrected method[43] and that the corrected method improves the association of quantitative parameters with outcome.[44]

Novel quantitative measures of TR severity are the Doppler volumetric method, the 3DE volumetric method, and direct planimetry of the 3D-vena contracta area (VCA) from 3DE color Doppler data sets (Figure 11).[41,42,45] Although the innovative methods for quantifying TR require further validation, the principle of volumetric quantification measures the total RV stroke volume (SV), which is the combination of both forward and regurgitant volumes, and then subtracts the forward SV. One method uses 3DE full-volume data sets to quantify RV end-diastolic and end-systolic volumes, with the difference then representing the total RV SV. The other method measures the diastolic SV across the TV annulus, ideally by measuring the 3D annular area and multiplying by the annular velocity time integral. Novel 3DE software automates measurements of the TV annulus[15,16,46] but has not been evaluated for TR quantitation.

The accuracy of the 3D-VCA method depends on the acquisition of a high temporal and spatial resolution color Doppler volume data set, frequently requiring a multibeat spliced image, which can be a

FIGURE 3 Complex Tricuspid Valve Morphology



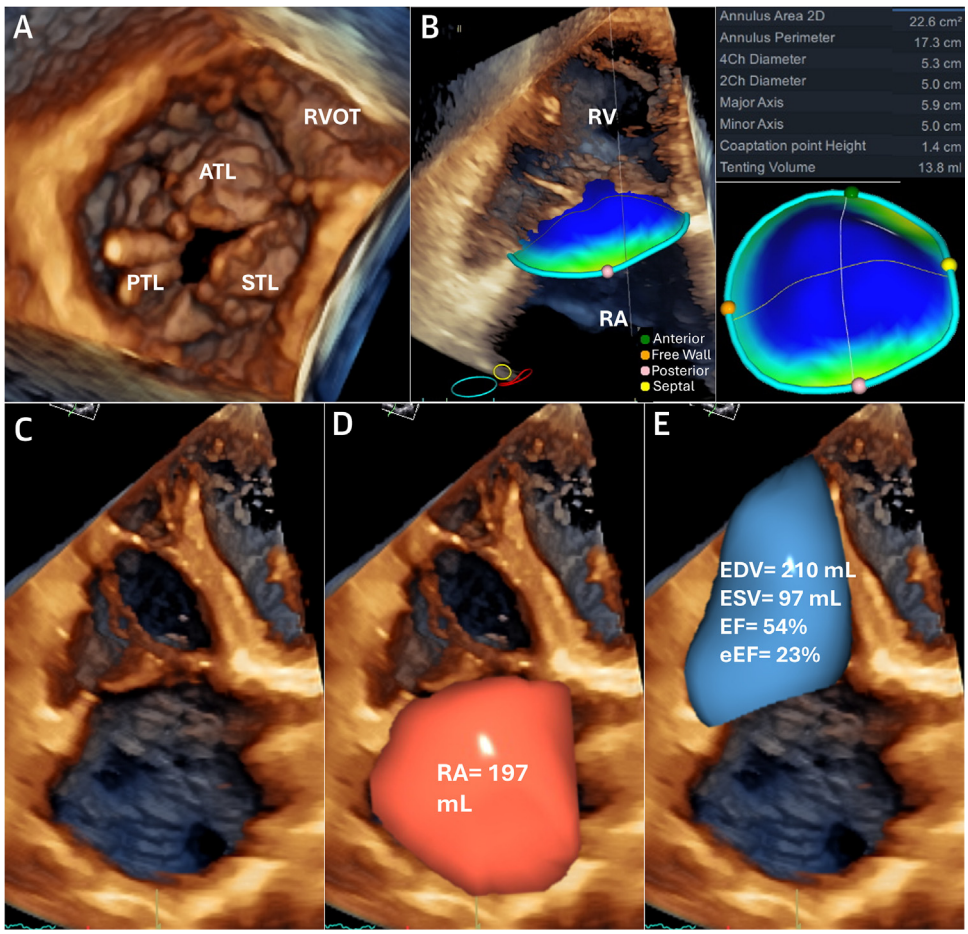
A novel tricuspid valve leaflet nomenclature classification scheme generated by using 2-dimensional (2D) and 3-dimensional (3D) transesophageal echocardiographic imaging identified 4 types of tricuspid valve morphologies with the relative prevalences shown. Transthoracic 3D echocardiography examples are shown in the bottom row. Modified from Hahn et al.[10]

limiting factor in the setting of arrhythmias. Studies suggest that a 3D-VCA cutoff of ~60 mm^2 may best differentiate moderate TR from severe TR.[18,19]

TR severity is strongly influenced by intracardiac pressures and intravascular volume. TR grading integrates not only intrinsic TR severity but also preload and afterload, and can be misleading in the presence of pulmonary hypertension, atrial fibrillation, or increased RA stiffness. Thus, the assessment of TR severity should be performed when patients are euvolemic, receiving stable up-titrated diuretic therapy, or receiving optimal medical therapy for volume overload, left heart disease, pulmonary hypertension, and atrial fibrillation.[22,47,48]

NOVEL ADVANCED GRADING SCHEME. In cases of late diagnosis and presentation of symptomatic TR, the established linear relationship between mortality and TR severity[49,50] supports the use of an extended grading scheme.[51] Increasing evidence highlights its effectiveness in predicting outcomes for patients receiving medical treatment[52-54] as well as for those undergoing transcatheter tricuspid valve interventions (TTVIs).[6,55,56] The grading scheme includes the following word and number categories: mild (1+), moderate (2+), severe (3+), massive (4+), and torrential (5+). Initial early feasibility studies[57-59] and a recent randomized controlled trial of transcatheter repair therapy[6] found that > severe TR was associated with a lower likelihood of achieving residual ≤ moderate TR. The degree of symptom improvement parallels the TR grade reduction when using the 5-grade scale.[6] The European Association of Cardiovascular Imaging guidelines[45] as well as the TVARC[33] support the use of the 5-grade scheme for patients undergoing interventions (**Table 3**). The new 5-grade TR severity scheme suggests different cutoffs reflecting the underestimation of EROA by the standard PISA method compared with 3D-VCA. Studies of volumetric methods for measuring regurgitant volume and calculating EROA have shown good correlation with 3D-VCA, although this quantitative method requires further validation.[18,60]

FIGURE 4 **3D Echocardiographic Imaging of the TV and Right Heart**

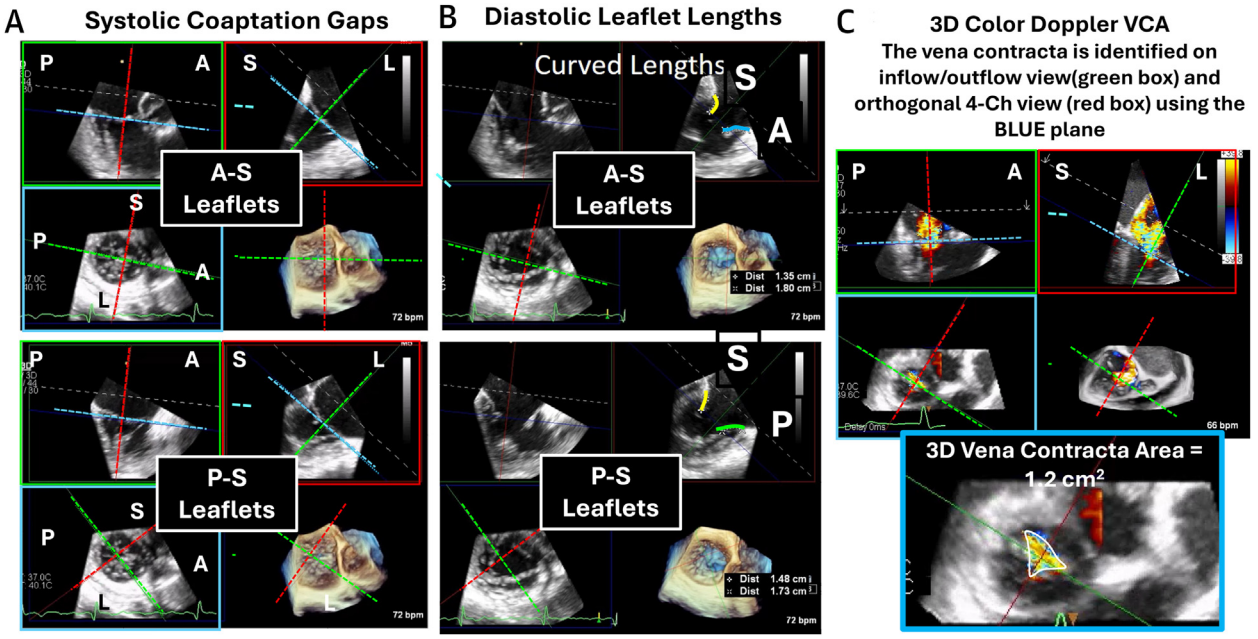


3D echocardiographic rendering with artificial intelligence-enhanced border detection significantly improved the ability to identify and quantify the right heart. Using transthoracic 3D echocardiography, the tricuspid leaflets and attached chordae are shown from the ventricular aspect (A), and the automated quantitation of the tricuspid annulus and leaflet tenting characteristics are also shown (B). Acquisition of 3D echocardiographic right heart data sets (C) allows for quantitation of right atrial (D) and right ventricular (E) volumes. From end-diastolic volumes (EDV) and end-systolic volumes (ESV), the stroke volume and ejection fraction (EF) can be calculated. In this example, the stroke volume = 113 mL; however, the regurgitant volume is 65 mL, and thus the forward stroke volume is 48 mL, and the effective ejection fraction (eEF) (the percentage of the total volume that that is ejected forward) = 23% (reduced). 2Ch = 2-chamber; other abbreviations as in Figures 1 and 3.

ADDITIONAL IMAGING MODALITIES TO GRADE TR. When echocardiography quantification of TR severity is inconclusive, CMR may be used to quantify regurgitant volume and fraction (Figure 7).[41] Regurgitant volume is calculated as the difference between RV SV and pulmonary artery forward flow measured on phase-contrast imaging (assuming no significant pulmonary regurgitation). The regurgitant fraction is derived by dividing the regurgitant volume by the RV SV. Data from a single-center study indicated that specific cutoff values for CMR-derived regurgitant volume and regurgitant fraction, which are associated with poorer outcomes, are ≥45 mL/beat and ≥50%, respectively.[28] On the other hand, Wang et al[61] demonstrated that lower thresholds of TR severity (ie, regurgitant fraction of 30%)—considered by current guidelines as "moderate"—already associated with cardiac and extracardiac TR consequences impacting long-term outcomes. Therefore, research is needed to redefine CMR-based thresholds of the TR severity associated with chamber remodeling and outcomes.

Computed tomography (CT) imaging has been used to assess the dimensions and area of the TV annulus, which reveals a linear relationship to TR severity and could be used as a surrogate measure. Direct

FIGURE 5 3D Echocardiography and MPR



The 3D data set can be sliced in real-time using multiplanar reconstruction (MPR). Standardization of the 2D imaging orientation may be different depending on the imager and interventionalist. In this example, the 3D volume is acquired from a mid-esophageal inflow-outflow view, which forms the primary view in the green box; posterior (P) and anterior (A) anatomical positions are noted at the top of the box. The orthogonal plane shown in the red box corresponds to the septal (S) and lateral (L) anatomical positions. The blue line is aligned with the annulus in both the green and red boxes, creating a short-axis annulus displayed in the blue box. By repositioning the green and red lines (which represent the positions of the green and red boxes, respectively), imaging of individual coaptation zones (A) and leaflet lengths (B) can be performed. (C) By using this method with a color Doppler 3D data set, the vena contracta area (VCA) can be visualized and measured by using a planimeter on the blue plane by aligning the blue lines with the vena contracta in the green and red boxes. Abbreviations as in Figures 3 and 4.

planimetry of the anatomical regurgitant orifice area using dual-source CT imaging is another potential indicator of TR severity; in one small study, this approach showed high intraobserver and interobserver reproducibility and a strong correlation between tricuspid anatomical regurgitant orifice area and 3D-VCA ($r = 0.93$; $P < 0.001$).[27]

REVISITING RIGHT HEART ANATOMY AND PHYSIOLOGY

The chronic volume overload resulting from TR causes gradual remodeling of the right atrium and right ventricle, initiating a vicious cycle in which right heart dilation exacerbates TR, and, in turn, the worsening TR further contributes to right heart dilatation, thereby negatively affecting survival outcomes.[8,62,63] In the early compensated stages of TR, RV dilation occurs with maintenance of systolic function, but the right ventricle works inefficiently, as a fraction of the SV flows back to the right atrium and the preload of the left ventricle is reduced. LV SV may be further impaired due to ventricular interdependence and pericardial constraint. RV dilatation results in a shift of the interventricular septum leftward, which contributes to the impairment of LV diastolic relaxation and filling and a further increase in pulmonary pressures.[64] In later stages, RV function starts to decline due to chronic volume overload and maladaptive RV remodeling.[65] RV remodeling has been shown to be an independent determinant of patients' prognosis[66,67] and is thus an integral assessment for all patients with TR.

RV SIZE AND FUNCTION. Echocardiography is the cornerstone for assessing right heart structural and functional abnormalities in patients with significant TR; however, significant strengths and weaknesses of these parameters should be recognized (Table 4). Despite these limitations, multiple studies have shown that RV function parameters can predict outcomes in patients with TR (Table 5). Assessment of RV free-wall longitudinal strain by 2D speckle-tracking echocardiography identifies higher rates of RV

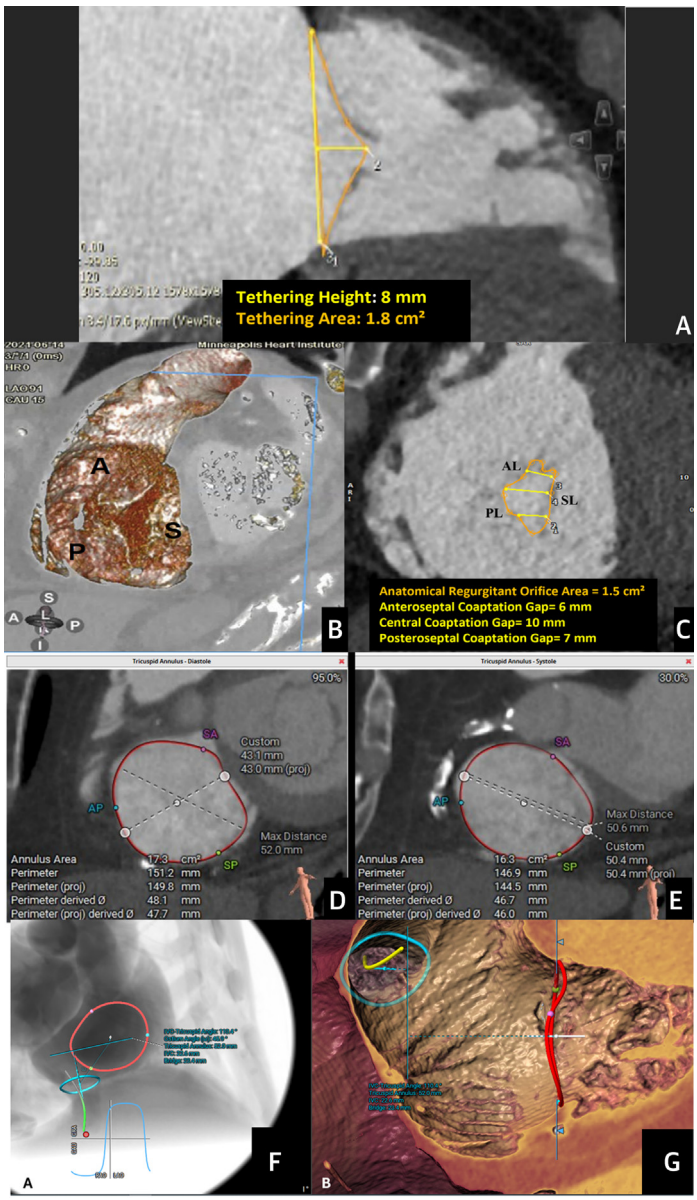
dysfunction and is associated with worse outcome beyond conventional echocardiographic parameters of RV systolic function such as tricuspid annular plane systolic excursion (TAPSE) or fractional area change.[68,69] Thresholds of several imaging parameters used to assess RV function are shown in **Table 6**. RV volumes and RV ejection fraction (RVEF) according to 3DE show excellent correlation with CMR.[70-72] In a recent study of patients with severe STR undergoing tricuspid transcatheter edge-to-edge repair (T-TEER), impaired preprocedural 3DE RVEF (<45%) was associated with an increased mortality, but the postprocedural decrease of 3DE RVEF after TTVR was not.[73]

Similarly, RVEF <45% according to functional CTA[74] has also been shown to be associated with worse outcomes after T-TEER despite successful TR reduction. Components of 3DE RVEF (longitudinal, radial, and anteroposterior) and their contribution to global RVEF[75] may also improve our understanding of RV remodeling in various etiologies of TR.[76] The load dependency of the "shortening" indices of RV function such as RVEF can be partially overcome by accounting for the loss of forward SV due to TR. The effective RVEF is calculated by subtracting the regurgitant volume from the total RV SV (resulting in the forward SV) and dividing the result by the RV end-diastolic volume. Hinojar et al[77] used CMR-measured effective RVEF and reported the strongest association with outcomes incremental to RVEF in patients with severe STR. The same parameter can be obtained by combining 3DE and Doppler echocardiography.[78]

Multiple echocardiographic parameters have been used to assess RV-pulmonary arterial (PA) coupling, which theoretically describes the efficiency with which RV SV is transferred into the pulmonary artery. The most studied has been the ratio between TAPSE and pulmonary artery systolic pressure (PASP), which has been associated with outcomes in patients with severe STR.[79,80] Using 3DE, RVEF/PASP may have greater prognostic value in STR.[14,81] Because of the inherent inaccuracy of the RA pressure estimate by echocardiography,[82] the predictive value of TAPSE/PASP is significantly higher when invasively measured PASPs are used.[12,82,83] RV-PA coupling measured as the ratio of 3DE-measured forward RV SV and RV end-systolic volume does not need the Doppler estimation of PASP and may have a stronger association with outcome than TAPSE/PASP in patients with STR.[14] Another way to adjust for loading conditions is the use of the RV load adaptation index also known as Dandel's index.[84] In patients post-TEER, the RV load adaptation index was a significant predictor of 2-year rehospitalization rates.[85]

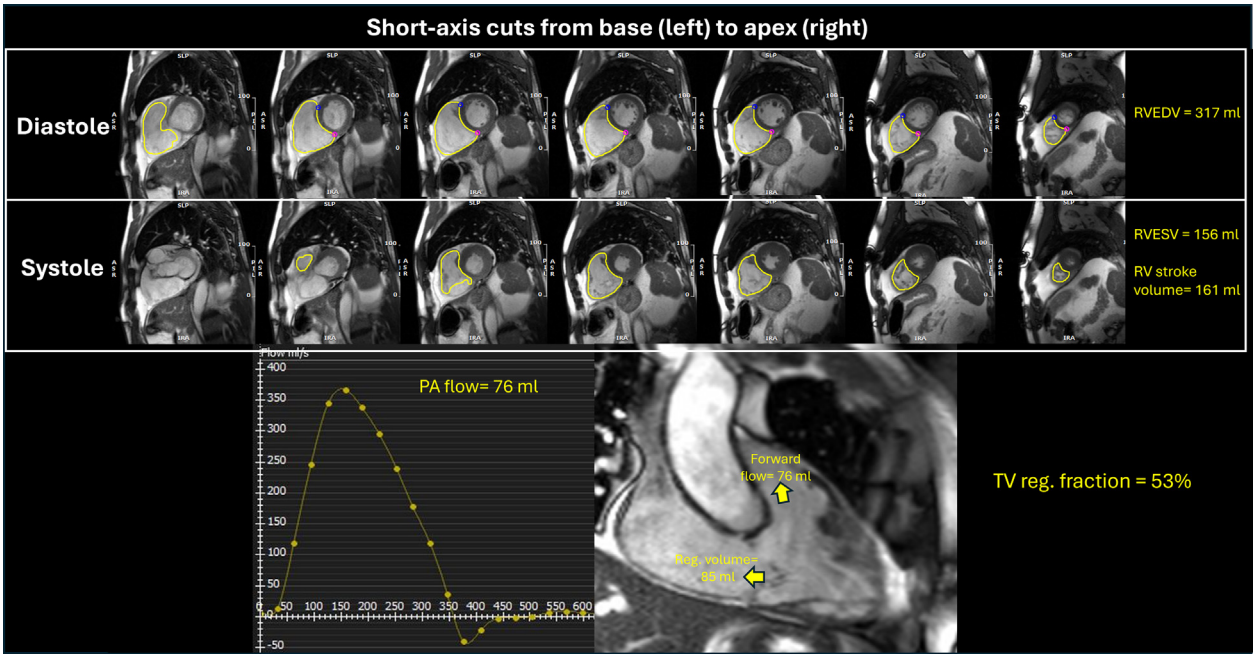
Recently, a novel echocardiographic assessment of RV myocardial work components by noninvasive

FIGURE 6 **Computed Tomography Evaluation of the TV Apparatus Anatomy**



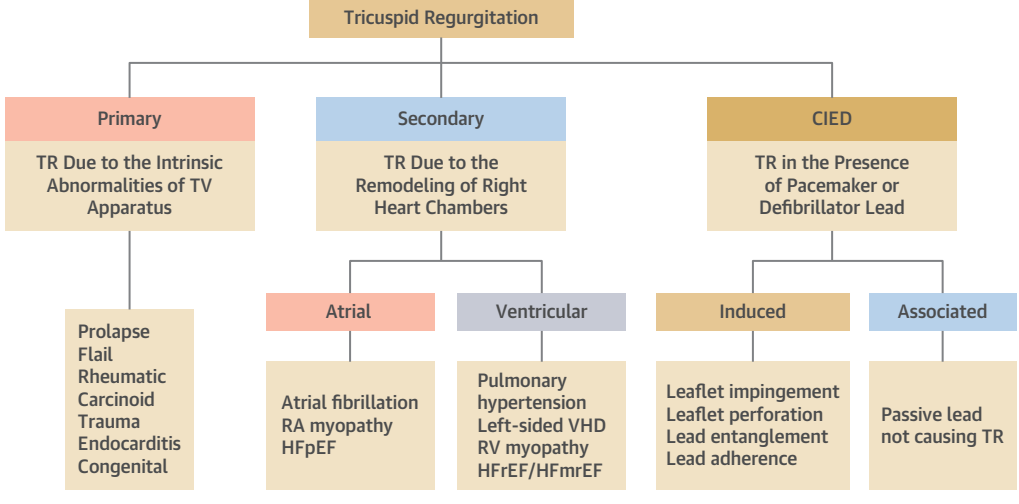
(A) Evaluation of TV tenting height and area. (B) TV leaflets are clearly identifiable along with each commissure. (C) Assessment of the anatomical regurgitant orifice area and the site and size of each coaptation gap. Diastole (D) and systole (E) show TV annular anatomical measurements. (F, G) Finally, pre-procedural planning for percutaneous TV repair includes the evaluation of the main orthogonal fluoroscopic angles, orientation and distance to approach the TV from the inferior vena cava (with 3D-rendered reconstruction). Abbreviations as in **Figures 1, 4, and 5**.

FIGURE 7 Example of How to Calculate TV Regurgitant Volume and Regurgitant Fraction by CMR



By analyzing a stack of short-axis slices that encompass both ventricles from base to apex, the endocardial and epicardial borders are delineated, allowing for the calculation of EDV and ESV. The right ventricular stroke volume (RVSV) is then determined as the difference between these 2 volumes. TV regurgitant volume is then calculated by subtracting pulmonary artery (PA) forward flow from RVSV, and TV regurgitant (reg.) fraction by dividing regurgitant volume by RVSV. CMR = cardiac magnetic resonance; RVEDV = right ventricular end-diastolic volume; RVESV = right ventricular end-systolic volume; other abbreviations as in Figures 1 and 4.

FIGURE 8 New Classification of TR Etiology



The expanded tricuspid regurgitation (TR) classification by etiology currently separates secondary TR into atrial secondary and ventricular secondary disease. When cardiac implantable electronic device (CIED) leads are present, lead-associated TR may be subcategorized into type A, whose CIED is causing the TR, and type B, whose CIED is incidental. Reproduced with permission from Hahn et al.[33] HFmrEF = heart failure with mid-range ejection fraction; HFpEF = heart failure with preserved ejection fraction; TA = tricuspid annulus; RHD = rheumatic heart disease; other abbreviations as in Figures 1, 4, and 7.

TABLE 1 Various Definitions of A-STR and Associated Outcomes

	Definition A-STR	Findings
Topilsky et al[167]	• No significant structural left valve disease • Absence of pulmonary hypertension (PASP <50 mm Hg) • LVEF ≥50% • No CIED	• Prevalence of A-STR (general population with ≥moderate TR) = 8.1% • Yearly mortality rate = 12%
Gavazzoni et al[36]	• Atrial fibrillation • LVEF >60% • PASP <50 mm Hg • No left-sided valve disease • Normal-appearing TV leaflets	• Prevalence of A-STR (among patients with STR) = 26% • A-STR 1-year combined endpoint of all-cause death and HFH = 14% • V-STR had a 2.7-fold higher risk (HR: 2.7 [95% CI: 1.3-5.7]) combined endpoint
Schlotter et al[168]	• TV tenting height ≤10 mm • Midventricular RV diameter ≤38 mm • LVEF ≥50% (Note: parameters determined by cluster analysis)	• Prevalence of A-STR (among patients with significant STR treated with either conservative management or T-TEER) = ∼15% • A-STR had significantly better 10-year survival than nonatrial TR in those conservatively treated (33% vs 57%; $P < 0.001$) • A-STR had a lower 1-year rate of mortality and HFH than nonatrial TR in the T-TEER cohort (12% vs 36%; $P = 0.017$)
Galloo et al[35]	• Absence of left-sided cardiac disease (LVEF ≥50%, < moderate aortic/mitral disease) • Absence of pulmonary hypertension (PASP <50 mm Hg) • No significant RV dysfunction (RV FAC ≥30% and/or TAPSE ≥15 mm) • LVEF ≥50% • Annular dilatation due to RA dilatation (related to atrial fibrillation or HFpEF)	• Prevalence of A-STR (among patients with severe STR) = 23% • Cumulative 10-year survival rates were significantly better for patients with A-STR (78%) compared with V-SFTR (46%; log-rank test; $P < 0.001$) • All V-STR subtypes were independently associated with worse overall survival compared with A-STR (HR: 2.292; $P < 0.001$ for V-STR)
Stolz et al[39]	• Ratio of end-systolic RA:RV area ≥1.5 • Preserved RV function (TAPSE >17 mm)	• Prevalence of A-STR (among patients with STR presenting for T-TEER) = 31% • A-STR at 1-year survival 84% (vs 78% for V-STR; $P < 0.0001$) • A-STR at 2-year survival 79% (vs 64% for V-STR; $P < 0.0001$) • A-STR independent predictor of 2-year survival rate free from HFH (HR: 0.65 [95% CI: 0.45-0.95]; $P = 0.025$)
Clement et al[38]	• Ratio of end-systolic RA:RV end-systolic area ≥1.6 • Ratio of end-systolic RA:RV end-systolic volume ≥1.4 • Additional parameters in multivariable model: 3DE LVEF, 3D RVEF, RA maximum volume, PASP	• Prevalence of A-STR (among a general population of any severity STR, with full 2DE and 3DE data sets) = 41% • No significant difference in outcomes using ES RA:RV volume ratio ≥1.4 in defining A-STR • Multivariable model performed the best in defining A-STR (AUC: 0.97 [95% CI: 0.95-0.98]) • A-STR using ES RA:RV area ratio ≥1.6 (but not ES RA:RV volume ratio) had higher cumulative 2-year survival (67% ± 4% vs 45% ± 5% for V-STR; $P = 0.0024$)

Reproduced with permission from Hahn RT.[38]

2DE = 2-dimensional echocardiography; 3D = 3-dimensional; 3DE = 3-dimensional echocardiography; A-STR = atrial secondary tricuspid regurgitation; AUC = area under the curve; CIED = cardiac implantable electronic device; ES = end-systolic; FAC = fractional area change; HFH = heart failure hospitalization; HFpEF = heart failure with preserve ejection fraction; LVEF = left ventricular ejection fraction; PASP = pulmonary artery systolic pressure; RA = right atrial; RV = right ventricular; RVEF = right ventricular ejection fraction; STR = secondary tricuspid regurgitation; TAPSE = tricuspid annular plane systolic excursion; TR = tricuspid regurgitation; T-TEER = tricuspid transcatheter edge-to-edge repair; TV = tricuspid valve; V-STR = ventricular secondary tricuspid regurgitation.

pressure-strain loops has been developed (**Figure 12**).[86,87] Studies show that, compared with normal control subjects, patients with STR increase constructive work and wasted work, although work efficiency is reduced.[88]

RA SIZE AND FUNCTION. Until recently, the right atrium has been considered to passively dilate due to the chronic volume overload imposed by TR.[89] Growing evidence suggests that the RA enlargement and the subsequent tricuspid annular dilation[90,91] may be the strongest determinant of atrial STR pathophysiology.[92] Therefore, the precise evaluation of RA size is pivotal to defining the STR phenotypes and refining the patient's prognosis.[93] Finally, recent data support using RA longitudinal strain analysis by 2D speckle-tracking echocardiography as an independent predictor of cardiovascular events in patients with STR.[36,94-97]

ESSENTIAL CONTRIBUTION OF IMAGING TO THE DETERMINATION OF OUTCOMES

Nearly all studies of the natural history of TR by severity have relied on qualitative echocardiographic grading (**Figure 10**, **Table 3**),[2,4] although PISA quantitation indicates a continuous increase in mortality associated with TR severity, underscoring the importance of accurately quantifying TR severity.[49,98,99] However, TR is a heterogeneous disease, and risk scores thus play a role in predicting outcomes both for patients undergoing conservative therapy as well as those considered for surgery or transcatheter interventions. Each score is presented in detail,

TABLE 2 Current Parameters That May Be Used to Distinguish Atrial From Ventricular Secondary TR

	A-STR Phenotype	V-STR Phenotype
Leaflet morphology		
Tenting height (4-chamber), mm	≤9	>9
Tenting area (4-chamber), cm^2	<2.1	≥2.1
Tenting volume, mL	<2.5	≥2.5
Right heart chamber size		
RV midventricular diameter, mm	≤38	>38
RV midventricular diameter index, mm/m^2	<21	≥21
RV end-diastolic volume index, mL/m^2	<80	≥80
RV end-systolic volume index, mL/m^2	<21	≥21
2D sphericity index	<55	≥55
End-systolic RA:RV volume or area ratio	≥1.5	<1.5
RV systolic function		
TAPSE, mm	>17	<17
FAC, %	≥35	<35
RVFWS, %	≥20	<20
RV TDI S', cm/s	≥9	<9
3D RVEF, %	≥50	<50
LVEF	≥50	(variable)
Invasive pulmonary vascular hemodynamics		
PCWP, mm Hg	≤15	(variable)
mPAP, mm Hg	<20	usually >20
PVR, WU	<2.0	(variable)

Modified from Hahn et al[33] and Muraru et al.[34]

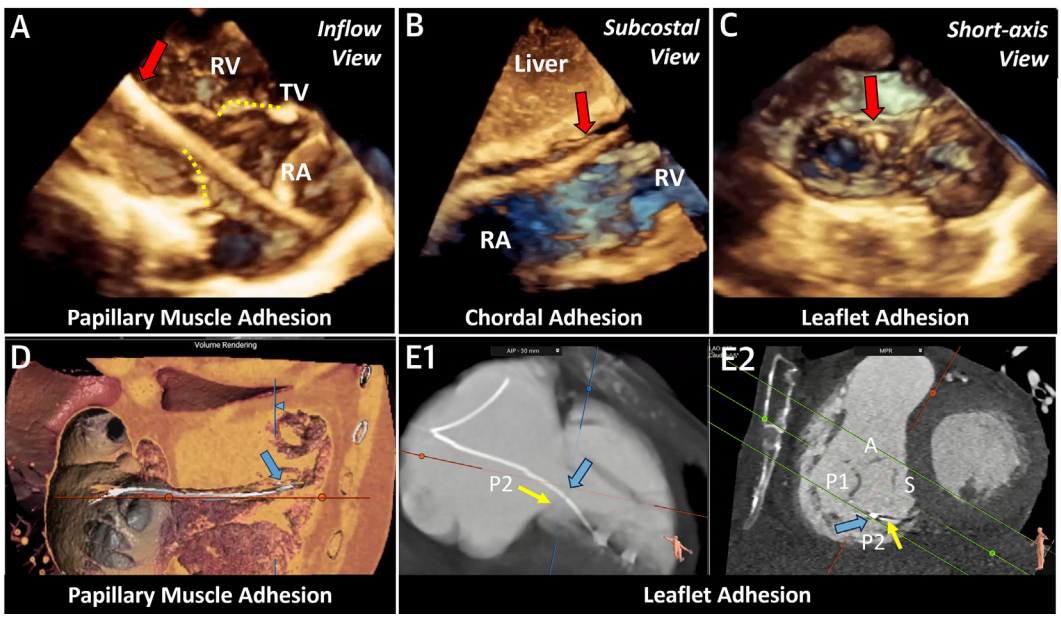
2D = 2-dimensional; mPAP = mean pulmonary arterial pressure; PCWP = pulmonary capillary wedge pressure; PVR = pulmonary vascular resistance; TDI = tissue Doppler imaging; RVFWS = right ventricular free wall strain; other abbreviations as in Table 1.

including potential impact and limitations, in Table 7, with an in-depth discussion in the Supplemental Methods.

Conservatively managed patients have been evaluated by Wang et al[100] and Hochstadt et al,[101] with the scores generated by these studies predictive of short and long-term mortality. Both scores include echocardiographic assessments of LV function and/or RV function. Nonspecific surgical scores had been used to assess risk of isolated TV surgery, including the EuroSCORE (European System for Cardiac Operative Risk Evaluation) I and II[102,103] and the STS (Society of Thoracic Surgeons) score;[104] however, these scores did not include an assessment of right heart function or symptoms associated with right heart failure.

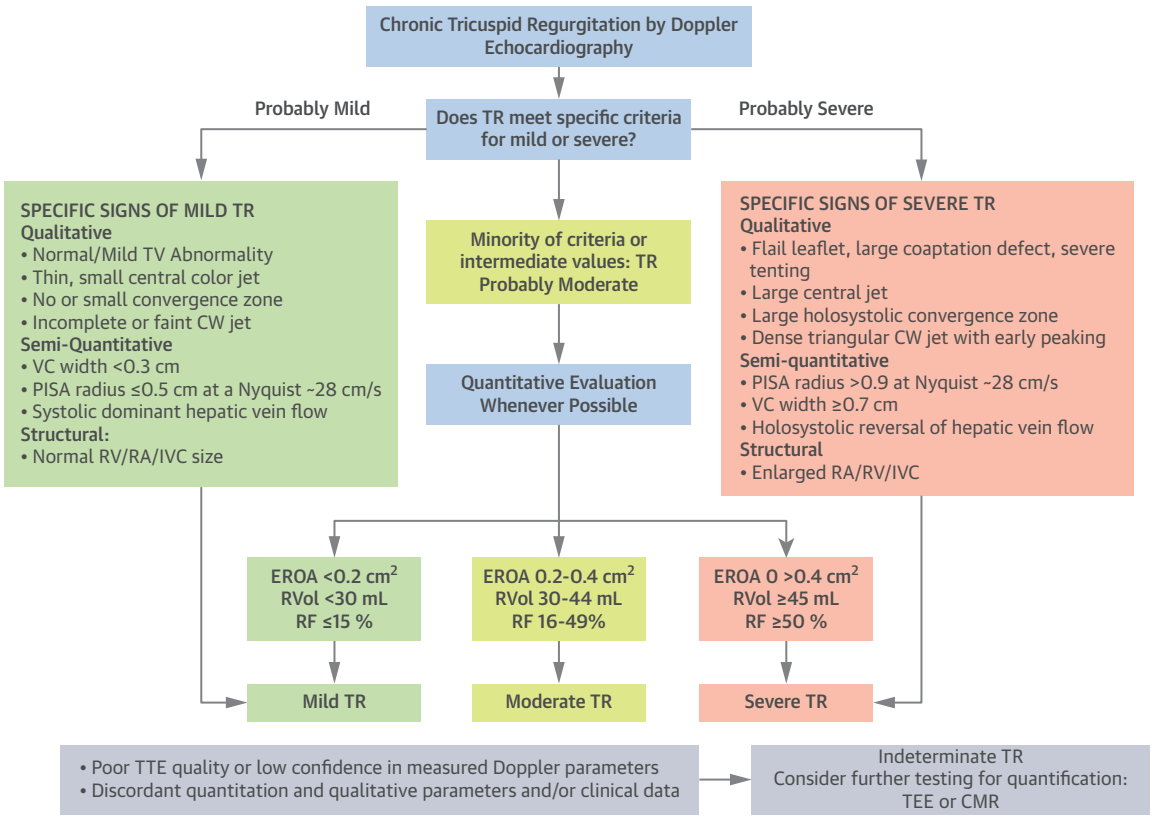
Dedicated surgical risk scores have recently been developed. The new STS-TV-specific risk score leverages a large database of patients to model multiple different operative outcomes. However, it is limited by the data collected in that registry with no echocardiographic/imaging measure of TR severity and no assessment of RV function.[105] The TRI-SCORE[106] was specifically developed to predict postoperative mortality after isolated TV surgery and includes RV function and symptoms as 2 important parameters. This score has been externally validated in multiple countries.[107,108] Divided into 3 risk levels

FIGURE 9 CIED-Related TR



The 3D transthoracic echocardiographic reconstruction of the TV can be used to determine the location of the CIED (red arrow) to show papillary muscle adhesions (A), chordal adhesions (B), and leaflet adhesions/impingement (C). Cardiac computed tomography imaging is also helpful in identifying papillary muscle adhesions (D) and leaflet adhesions/impingement (E). A = anterior leaflet; P = posterior leaflet; S = septal leaflet; other abbreviations as in Figures 1, 3, and 8.

FIGURE 10 Algorithm for the Integration of Multiple Parameters of TR Severity



Current guidelines assume good-quality echocardiographic imaging and complete data acquisition. Specific criteria for severe TR continue to be refined given the ellipsoidal or irregular shape of the regurgitant orifice. The four-chamber vena contracta (VC) width criteria, which assumes a circular orifice, often may underestimate TR severity when using newer methods of TR quantification. If imaging is technically difficult, transesophageal echocardiography (TEE) or cardiac magnetic resonance (CMR) should be considered. TR severity may be indeterminate due to poor image quality, technical issues with data, internal inconsistency among echocardiographic findings, or discordance with clinical findings. Modified from Zoghbi et al[41] and Lancellotti et al.[45] CW = continuous wave; EROA = effective regurgitant orifice area; IVC = inferior vena cava; PISA = proximal isovelocity surface area; TTE = transthoracic echocardiography; RF = regurgitant fraction; RVol = regurgitant volume; other abbreviations as in Figures 1, 5, and 8.

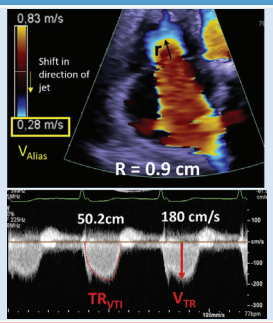
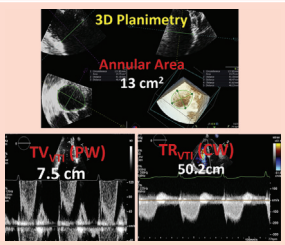
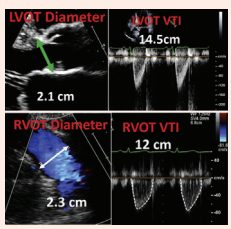
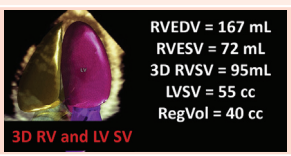
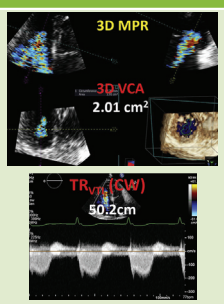
(low, intermediate, and high), it predicts 2-year mortality in surgically, medically, or transcatheter-treated patients.[109-113]

Finally, the TRIVALVE (International Multisite Transcatheter Tricuspid Valve Therapies Registry) score[114] was developed to predict 1-year mortality or hospitalization after percutaneous treatment of TR. It is based on data from an international multicenter registry and includes LV function assessment and signs of right heart failure but no assessment of right heart function.

ANATOMICAL AND PHYSIOLOGICAL CRITERIA FOR DEVICE CHOICE

Device selection for TV interventions still involves several uncertainties and unknowns concerning appropriate indications and patient profile for each treatment modality. Echocardiography and multimodality imaging play a crucial role in determining anatomical suitability and predictions of procedural success (Video 1).[115,116] In current clinical practice, T-TEER is the most commonly used transcatheter intervention given its commercial availability and low risk of complications; however, it achieves mild or less residual TR in approximately 50% of patients.[6] TTVR has only recently become commercially available and can address simple as well as complex anatomies,[22] with >95% of patients achieving mild or less residual TR, but it may be associated with higher bleeding and pacemaker rates.[7] The appropriate timing and use of these devices continue to be studied (Figure 13).[22,117] Prediction of TR reduction with T-TEER has relied heavily on echocardiographic

FIGURE 11 Standard and Novel Methods to Quantify TR

Proximal Isovelocity Surface Area	Measurements Required	Example	Calculation
Proximal Isovelocity Surface Area (PISA)	**1. Aliasing velocity (V_{Alias}):** Color Doppler with baseline shift in the direction of regurgitant jet **2. Radius of PISA (r)** **3. TR peak velocity (V_{TR}):** CW of the TR jet **4. TR velocity time integral (TR_{VTI}):** CW of the TR jet		**PISA EROA:** EROA = $2\pi r^2(V_{Alias}) \div V_{TR}$ **TR Regurgitation Volume =** EROA × TR_{VTI} **Example:** 1. PISA EROA = (6.282 × 0.90 × 28 cm/s) ÷ 180 cm/s = 0.79 cm^2 2. Reg Vol = 0.79 cm^2 × 50.2 cm = 40 mL
Volumetric Quantitation	**Measurements Required**	**Example**	**Calculation**
Doppler Method: Quantifies diastolic stroke volume across the tricuspid valve	**1. 3D Diastolic $TV_{Annular}$ Area** **2. TV velocity time integral (TV_{VTI}):** PW Doppler sample volume at the annulus **3. TR velocity time integral (TR_{VTI}):** CW of the TR jet **4. Forward Stroke Volume by Doppler (see below)**		**TV Diastolic Stroke Volume =** $TV_{Annulus}$ Area × TV_{VTI} **TR Regurgitation Volume =** TV diastolic volume - Forward Stroke Volume **EROA** = RegVol ÷ TR_{VTI} **Example:** 1. TV Diastolic SV = (13 cm^2 × 7.5 cm = 97 mL 2. TR Reg Vol = 97 mL - 50 mL = 47 mL 3. EROA = 47 mL ÷ 50.2 cm = 0.93 cm^2
Forward Stroke Volume used to quantify RegVol by Doppler Method	LVOT Stroke Volume LVOT Diameter LVOT PW RVOT Stroke Volume RVOT Diameter RVOT PW		**Forward Stroke Volume =** $LVOT_{annulus}$Area × $LVOT_{VTI}$ **Example:** LV SV = (0.785 × [2.1 cm]^2) × 14.5 cm = 50 mL **Forward Stroke Volume =** $RVOT_{annulus}$Area × $RVOT_{VTI}$ **Example:** RV SV = (0.785 × [2.3 cm]^2) × 12 cm = 50 mL
3D Volume Method: Quantifies total RV stroke volume	**1. 3D RV Stroke Volume (total RV SV)** **2. 3D LV Stroke Volume (forward SV)** **3.TR velocity time integral (TR_{VTI}):** CW of the TR jet		**TR RegVol =** Total RV SV - Forward SV **EROA** = RegVol ÷ TR_{VTI} **RegFraction** = RegVol ÷ Total RV SV **Example:** **TR RegVol = 95 mL - 50 mL = 40 mL** **EROA = 40 mL** ÷ 50.2 cm = 0.80 cm^2
3D Color Doppler	**Measurements Required**	**Example**	**Calculation**
3D Vena Contracta Area (VCA)	3D Color Doppler planimetry of the VCA TR velocity time integral (TR_{VTI})		EROA ≅ VCA **TR Regurgitation Volume =** VCA × TR_{VTI} **Example:** 3D VCA = 2.01 cm^2 Reg Vol = 2.01 cm^2 × 50.2 cm = 100.9 mL

Continued on the next page

TABLE 3 Echocardiographic Parameters and Relative Cutoffs for TR 5-Tier Grading

	Mild (1+)	Moderate (2+)	Severe (3+)	Massive (4+)	Torrential (5+)
Qualitative					
Tricuspid morphology	Normal or mildly abnormal	Moderately abnormal	Severely abnormal (flail leaflet, large coaptation gap, marked tethering)		
Color-flow jet area	Small, narrow, central	Moderate central	Large central, or eccentric, wall impinging		
Flow convergence zone	Not visible, transient, or small	Intermediate in size and duration	Large throughout systole		
CWD contour	Faint, partial, parabolic	Dense, parabolic	Dense, parabolic, or triangular	Dense, often triangular, may have low peak velocity	Dense, usually triangular, often low peak velocity
Right heart dimensions	Usually normal	Normal or mild dilatation	Usually dilated	Dilated	
Semi-quantitative					
VCW (biplane)[a]	<3 mm	3-6.9 mm	7-13.9 mm	14-20.9 mm	≥21 mm
PISA radius[b]	≤5.4 mm	5.5-8.9 mm		≥9 mm	
Hepatic vein flow[c]	Systolic dominant	Systolic blunting		Systolic flow reversal	
Tricuspid inflow (PWD)	A-wave dominant	Variable		E-wave dominant (≥1 m/s)	
Quantitative					
PISA EROA	<20 mm^2	20-39 mm^2	40-59 mm^2	60-79 mm^2	≥80 mm^2
Regurgitant volume (2D PISA)	<30 mL	30-44 mL	45-59 mL	60-74 mL	≥75 mL
New quantitative methods					
Regurgitant fraction	≤15%	16%-49%		≥50%	
3D-VCA	-	-	75-94.9 mm^2	95-114.9 mm^2	≥115 mm^2
2D Doppler EROA	-	-	75-94.9 mm^2	95-114.9 mm^2	≥ 115 mm^2

[a]At a color Doppler scale between 40 and 60 cm/s. Note that some studies suggest that an average vena contracta width (VCW) >9 mm should define severe TR. [b]Color Doppler Nyquist shift down toward 28 cm/s, until the hemispherical flow convergence zone is clearly visualized. [c]Unless other reason for flow reversal (ie, atrial fibrillation, RA elevated pressures/noncompliance). Adapted from Zoghbi et al,[41] Lancellotti et al,[45] and Hahn and Zamorano.[51] Modified with permission from Hahn et al.[33]

CWD = continuous wave Doppler; EROA = effective regurgitant orifice area; PISA = proximal iso-velocity surface area; PWD = pulsed wave Doppler; VCA = vena contracta area; other abbreviations as in Tables 1 and 2.

assessment of variables such as septolateral coaptation gap, chordal structure density, *en face* TR jet morphology, TR jet location, and image quality.[116] Multiple other predictors of residual TR after TTVI are listed in Supplemental Table 1.

IMPLANTATION AND FOLLOW-UP OF TRANSCATHETER DEVICE THERAPIES

Imaging, particularly echocardiography, plays a crucial role in patient selection, procedural monitoring, and follow-up of patients considered for transcatheter valve therapies.[118] The procedural and technical success of transcatheter device therapies depends on echocardiographic and fluoroscopic imaging. Transcatheter TV device implantation is particularly nuanced given the variety of devices currently approved for commercial use, or undergoing clinical trials (**Figure 14**), and the challenges involved in imaging the far-field TV using TEE. The establishment of recommended standards for the performance of pre-interventional TEE,[119] along with societal training guidelines,[120,121] have solidified interventional echocardiography as a distinct subspecialty. TEE guidance of structural heart disease procedures must be performed by highly skilled

FIGURE 11 Continued

The standard method for assessing the severity of TR is the PISA method. However, newer methods of quantifying TR severity are currently being investigated. For volumetric quantitation, 2 methods are shown: the Doppler method and the 3D volumetric method. The Doppler method quantifies the diastolic RVSV by multiplying the measured tricuspid annular area and annular diastolic velocity time integral (VTI); the forward stroke volume (SV), calculated from either across the RVOT or LVOT, is then subtracted from the diastolic RVSV to obtain the regurgitant volume (RegVol). The 3D volumetric method directly measures the SV of the right and left ventricles with the difference being the RegVol. Finally, 3D color Doppler planimetry of the VCA (3D-VCA) can be performed but typically requires multi-beat acquisitions/high line density for accurate planimetry of the regurgitant orifice. LVSV = left ventricular stroke volume; other abbreviations as in Figures 1, 4, 5, 7, 8, and 10.

TABLE 4 Strengths, Limitations and Clinical Value of Echocardiographic Indexes of RV and RA Geometry and Function in Patients With TR

	Threshold Value for Poor Outcomes	Strengths	Limitations	Use/Value in the Clinical Setting
Right heart size				
2DE basal diameter	>40 mm	• Easy, fast, and widely available • May be performed on 2D images (TTE or TEE)	• Depends on the degree of the probe rotation • Limited reproducibility due to lack of anatomical landmarks	• Poor correlation with RV volumes • It overestimates the size of the right ventricle in patients with A-STR
3DE volumes	(not yet defined)	• Artificial intelligence-assisted quantitation • Independent of geometric assumptions about RV shape • Extensively validated against CMR	• Availability of 3DE systems and expertise in acquiring and post-processing 3D data sets of the right ventricle • Requires dedicated analysis software packages • Dependent on image quality	• Can be used to quantify total RV SV • Can be used to measure RV-PA coupling (ie, Forward SV ÷ RV end-systolic volume)
2DE RA volume	>48 mL/m^2	• Easy and fast • Widely available	• Heavily dependent on view (RV focused apical view recommended)	• Documented prognostic value, particularly if associated with reduced RA longitudinal strain
Right heart function				
TAPSE	<17 mm	• Easy to obtain, fast, and widely available • Highly reproducible	• Unreliable in patients with prior cardiac surgery • Reflects only longitudinal function • Neglects the contribution of IVS and RVOT • Load- and angle-dependent	• It has shown prognostic value, with better survival in patients with preserved TAPSE than in those with significant TAPSE reduction • Its utility in predicting outcomes after tricuspid surgery or percutaneous repair remains controversial
S′ (TDI)	<9.5 cm/s	• Easy to obtain, fast, and widely available • Reproducible • Less load dependent than TAPSE	• Reflects only longitudinal function of basal lateral segment • Neglects contribution of IVS and RVOT • Angle dependent	• Not been tested in patients with significant TR
FAC	<35%	• Simple and widely available • Reflects both longitudinal and radial contraction • Fair correlation with CMR-derived RV EF	• Heavily dependent on image quality and view (RV-focused apical view recommended) • Poor reproducibility • Neglects contribution of the RVOT • Load dependent	• Documented prognostic value in T-TEER
dp/dt	≤400 mm Hg/s	• Easy and widely available • Poor reproducibility	• Load dependent	• Inapplicable in patients with significant TR
RVFWLS	>22%	• Measures longitudinal myocardial deformation by speckle tracking • Less angle and load dependent • Less confounded by RV geometry and passive motion • Reproducible, especially with advanced software	• Dependent on image quality and view (RV-focused apical view recommended) • More reproducible than conventional echocardiographic measurements of RV function • Requires postprocessing using dedicated software packages • Neglects contribution of the RVOT • Vendor dependency, for which caution is required in follow-up studies	• Strong association with clinical outcomes (ie, death or HFHs) in various stages of STR
3DE RVEF	<45%	• Independent of geometric assumptions about RV shape • Includes all the components of RV function • Extensively validated against CMR	• Availability of 3DE systems and expertise in acquiring and post-processing 3D data sets of the right ventricle • Requires dedicated analysis software packages • Dependent on image quality • Cannot measure RV volumes >350-400 mL • Load dependent	• Established prognostic value, superior to other RV parameters obtained with conventional echocardiography • Novel methods for calculating components of RVEF (longitudinal EF, radial EF, and circumferential EF) which may predict outcomes
Effective RVEF	<20%	• All the pros of 3DE RVEF • Takes into account the volume overload by calculating effective RVEF as the RV forward SV ÷ RV end-diastolic volume	• Requires accurate calculation of the regurgitant volume • All limitations of 3DE apply	• Established prognostic value, superior to RVEF and other conventional echocardiography parameters of RV function

Continued on the next page

TABLE 4 Continued

	Threshold Value for Poor Outcomes	Strengths	Limitations	Use/Value in the Clinical Setting
TAPSE/PASP	>0.406 mm/mm Hg	• Easy and fast to obtain, widely available and reproducible • Normalize RV function for afterload	• Pre-load dependent • PASP estimation is inaccurate in patients with severe TR • All the limitations of TAPSE apply	• Established prognostic value, both in patients under medical treatment and those who underwent T-TEER • Use of invasive PASP improves the prognostic value of the parameter
FWLS/PASP	≤0.34%/mm Hg	• All the pros of FWLS apply • Normalize RV function for afterload	• Pre-load dependent • PASP estimation is inaccurate in patients with severe TR • All limitations of 2DE longitudinal strain apply	• Associated with all-cause mortality in patients with severe STR • Independent of TAPSE/PASP
Forward SV/RV end-systolic volume	<0.40	• All the pros of RVEF apply • Does not require the estimation of PASP • Normalize RV function for both volume and pressure overload	• All the limitations of 3DE RVEF apply • Requires accurate calculation of the regurgitant volume	• Documented association with outcomes • Stronger association with outcomes than TAPSE/PASP and FWLS/PASP
2DE RA volume	>48 mL/m^2	• Easy and fast • Widely available	• Heavily dependent on view (RV-focused apical view recommended)	• Documented prognostic value, particularly if associated with reduced RA longitudinal strain
2DE RA longitudinal strain	<13%	• Easy, reproducible	• Heavily dependent on image quality and view (RV-focused apical view recommended) • Requires postprocessing using dedicated software package	• Documented prognostic value in severe STR, independent of RV volume

CMR = cardiac magnetic resonance; EF = ejection fraction; FWLS = free wall longitudinal strain; FWS = free wall strain, IVS = interventricular septum; RV-PA = right ventricular–pulmonary arterial; RVFWLS = right ventricular free wall longitudinal strain; RVOT = right ventricular outflow tract; SV = stroke volume; TEE = transesophageal echocardiography; TTE = transthoracic echocardiography; other abbreviations as in Tables 1 and 2.

echocardiographers who can provide rapid, accurate, and high-quality image acquisition and interpretation in real time, using advanced imaging tools such as MPR,[21] echocardiographic-fluoroscopic fusion,[22] and 3D intracardiac echocardiography.[122] Guidance regarding specific TTVIs are included in the Supplemental Methods and Videos 4 to 13.

FOLLOW-UP OF SURGICAL OR TRANSCATHETER INTERVENTIONS. Follow-up imaging after TV intervention primarily uses echocardiography.[13,33] The postprocedural assessment focuses on evaluating device integrity, position, and stability, as well as assessing residual or recurrent TR, residual valve area and gradient, RV dimensions and function, and RA remodeling. In addition, the impact on left heart size, function, and forward flow may provide additional insight into the efficacy of the device. Both CMR and CTA can be used in cases of challenging echocardiographic imaging or discrepancies between echocardiographic and clinical findings, and particularly for the evaluation of the device performance and reverse RV remodeling.[123]

ASSESSMENT OF RESIDUAL TR. Patients undergoing TV interventions present unique challenges for residual TR assessment: 1) lack of well-validated quantitative parameters of TR severity after device implantation; and 2) specific device-related challenges of color Doppler evaluation (more likely after edge-to-edge repair with multiple, noncoaxial jets with proximal flow restricted by the device). Although PISA is commonly used, it has intrinsic methodologic limitations and can be challenging postintervention due to multiple jets or device shadowing.[18,45,124,125] TR severity might be better appreciated using TEE, although it is rarely performed unless clinically indicated. Three-dimensional methods, such as 3D-VCA, can overcome PISA limitations but are mostly validated on native valves.[18,19] In patients with good image quality, the 3D volumetric method seems promising, does not rely on any geometric assumption, and can be used with all devices. CMR with advanced technologies may be used after device therapy to quantify residual TR severity even in the setting of arrhythmias but requires expertise, dedicated imaging acquisition protocol and experience.[126] Therefore, the approach to the postprocedural evaluation of post-device TR severity is currently to integrate multiple parameters rather than emphasize or depend on a single measurement to mitigate the effects of technical or measurement errors inherent to each method (Table 8).[127-129]

Device stability, mean gradient, transvalvular leak, paravalvular leak, leaflet excursion, thickening, valve area, and presence of masses (eg, vegetation, thrombus) are the essential elements that should be evaluated after TV replacement (surgical or percutaneous).[128] TTVR may be more prone to thrombotic

TABLE 5 RV Function Predictors of Adverse Outcomes in Patients With TR

First Author	Patient Population	RV Dysfunction Cutoff	Outcome
Dietz et al[63]	1,292 patients with significant STR	TAPSE <17 mm	• All-cause mortality • TAPSE predicted (log rank chi-squared test = 17.95; $P < 0.001$)
Schlotter et al[169]	684 patients from the multicenter registry compared with 914 conservatively treated patients from 2 tertiary care centers	1. Preserved (TAPSE >17 mm) 2. Mid-range (TAPSE 13-17 mm) 3. Reduced (TAPSE <13 mm) RV function	• 1-year all-cause mortality • Only in patients with mid-range RV function was TTVI associated with an improved survival (log-rank test; $P = 0.004$). In these patients, procedural success was associated with a reduced HR for all-cause mortality (HR: 0.22 [95% CI: 0.09-0.57]).
Prihadi et al[69]	896 patients with significant functional TR	FAC <35%, TAPSE <17 mm RVFWLS >−23%	• All-cause mortality • FAC log rank chi-squared test = 10.47; $P = 0.001$ • TAPSE log rank chi-squared test = 7.4; $P = 0.006$ • RVFWLS log rank chi-squared test = 28.4; $P < 0.001$
Hinojar et al[68]	151 patients with at least severe STR (severe, massive, or torrential) and the absence of a formal indication for tricuspid valve intervention	RVFWLS >−20%	• Composite of mortality and heart failure • RVFWLS was independently associated (adjusted HR for abnormal RVFWLS: 5.90 [95% CI: 3.17-10.99]; $P < 0.001$)
Ancona et al[170]	79 patients with severe TR who underwent isolated TV surgery	RVFWLS <20%	• RVFWLS best predictor (vs TASPSE, FAC, S') perioperative mortality (AUC: 0.833 [95% CI: 0.72-0.95]; $P = 0.005$; sensitivity, 68%; specificity, 100%) • Adding RVFWLS to TRI-SCORE improved global chi-squared test ($P = 0.008$)
Vogelhuber et al[171]	262 patients with high surgical risk, undergoing T-TEER	TAPSE <17 mm and RVFAC <35%	• RV dysfunction at baseline was associated with increased risk of all-cause mortality (HR: 2.54 [95% CI: 1.49-4.40]; $P < 0.001$) • No association between reduction in RV function and outcomes
3D RVEF			
Orban et al[73]	75 patients with severe TR undergoing T-TEER	3DE RVEF <45%	• 3D RVEF (highest tertile, 44.6%-61.8%; HR: 0.18; $P = 0.007$), whereas both lower tertiles were associated with high 1-year mortality • No association between reduction in RV function and outcomes
Tanaka et al[172]	264 consecutive patients with TR undergoing TTVI (T-TEER and T-annuloplasty)	CT-RVEF <45%	• RVEF associated with higher risk of the composite outcome[a] (HR: 2.99 [95% CI: 1.65-5.41]; $P = 0.001$)
Hinojar et al[77]	75 patients conservatively treated	CMR eRVEF ≤34%	• eRVEF was the strongest predictor of outcomes, incremental to RVEF (ΔC-statistic: 0.139 [95% CI: 0.040-0.237]; $P = 0.005$)
Clement et al[78]	513 patients treated conservatively	3DE eRVEF <20%	• eRVEF <20% was associated with a 3-fold increased risk of experiencing the composite outcome[b] (HR: 3.54 [95% CI: 2.61-4.79]; $P < 0.001$)
RV-PA coupling			
Fortuni et al[80]	1,149 patients with moderate or severe secondary TR	$TAPSE/PASP_{ECHO}$ <0.31 mm/mm Hg	RV-PA uncoupling independently associated with all-cause mortality at 5 years (HR: 1.462 [95% CI: 1.192-1.793]; $P < 0.001$)
Brener et al[79]	444 patients undergoing TTVI	$TAPSE/PASP_{ECHO}$ ≤0.406 mm/mm Hg	TAPSE/PASP ratio >0.406 vs ≤0.406 was associated with a decreased risk of all-cause mortality (HR: 0.57 [95% CI: 0.35-0.93]; $P = 0.023$)
Stolz et al[83]	848 patients who underwent T-TEER	$TAPSE/PASP_{ECHO} = 0.39$ $TAPSE/PASP_{INVASIVE} = 0.30$	Both predict 2-year all-cause mortality however significantly higher c-index when using $TAPSE/PASP_{INVASIVE}$
Sugiura et al[82]	206 patients who underwent T-TEER	$TAPSE/PASP_{INVASIVE}$	• Primary outcome was all-cause mortality or heart failure rehospitalization within 1 year • $TAPSE/PASP_{INVASIVE}$ was inversely associated with the risk of the primary outcome (per 0.1-point increase: adjusted HR: 0.67 [95% CI: 0.56-0.82]; $P < 0.001$)
Gavazzoni et al[14]	180 patients with moderate or severe STR	3DE RV forward SV/RVESV RV FWLS/PASP <−0.42%/mm Hg TAPSE/PASP <0.36 mm/mm Hg	• 2-year composite endpoint of death of any cause and heart failure hospitalization • RV forward SV/ESV <0.40 (HR: 3.36 [95% CI: 1.49-7.56]; $P < 0.01$) • RV FWLS/PASP <−0.42%/mm Hg (HR: 3.1 [95% CI: 1.26-7.84]; $P = 0.01$) • TAPSE/PASP <0.36 mm/mm Hg (HR: 2.69 [95% CI: 1.29-5.58]; $P = 0.01$)
Kassar et al[85]	364 patients with complete data sets in the EuroTR Registry	$RV_{LAI} = (TR_{VTI} \times RV_{Length}) \div RV_{Area}$ RV_{LAI} <20.5%	• Higher RV_{LAI} >20.5 was associated with improved survival following T-TEER

[a]All-cause mortality and HFH, within 1 year after TTVR. [b]All-cause death and HFH at 2 years.

CT = computed tomography; eRVEF = effective right ventricular ejection fraction; ESV = end-systolic volume; EuroTR = European Registry of Transcatheter Repair for Tricuspid Regurgitation; $PASP_{ECHO}$ = PASP by echocardiography; $PASP_{INVASIVE}$ = PASP by invasive catheterization; RV_{Area} = end-diastolic right ventricular area; RVESV = right ventricular end-systolic volume; RV_{LAI} = right ventricular load adaptation index; RV_{Length} = end-diastolic right ventricular length; TR_{VTI} = tricuspid regurgitation velocity time integral; TTVI = transcatheter tricuspid valve intervention; other abbreviations as in Tables 1, 3, and 4.

TABLE 6 Proposed Echocardiographic Cutoffs For RV Function

	Normal RV Function	Mild Dysfunction	Moderate Dysfunction	Severe Dysfunction
TAPSE, mm	≥17	12-16	8-11	<8
RV TDI S', cm/s	≥10	8-10	6-8	<5
RVFWLS (%) in absolute	≥20	17-19	14-16	<14
FAC (%)	≥35	30-34	25-29	<25
RVEF (3D)	>45%	40-45%	35-40%	<35

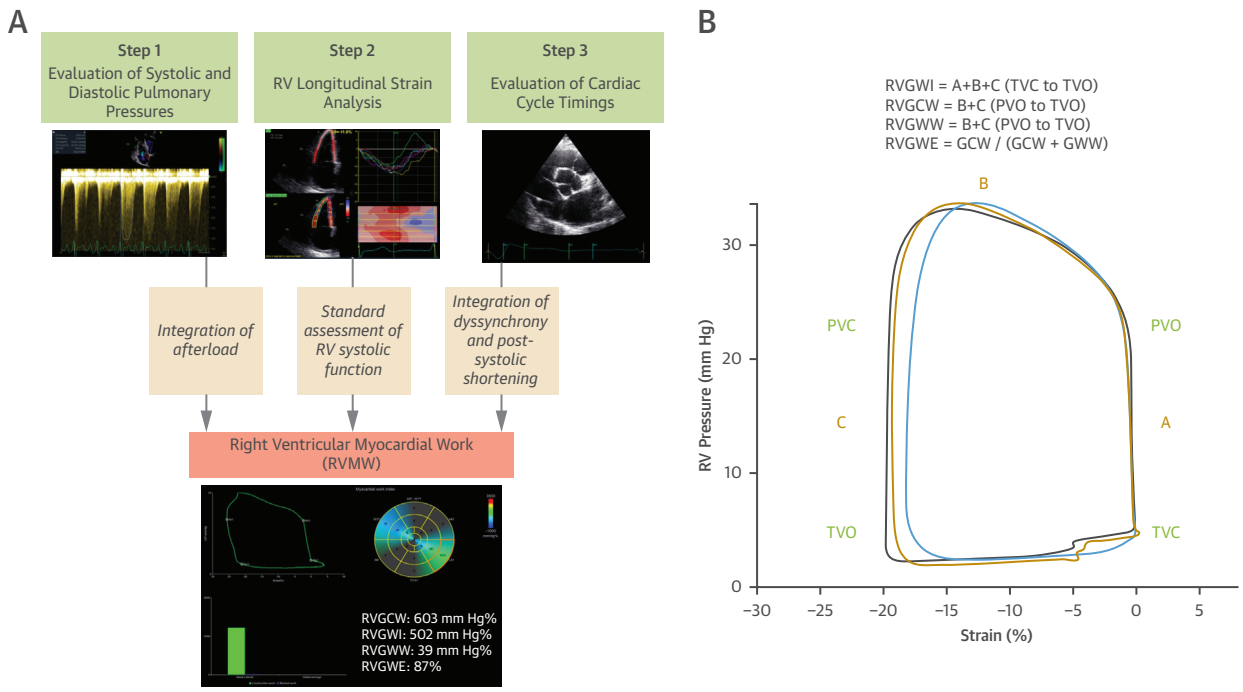
Modified from Hahn et al.[33]
Abbreviations as in Tables 1 and 4.

complications in the setting of low intra-cardiac right heart pressures, RA myopathy and/or dilatation, presence of atrial fibrillation, and lack of antithrombotic therapy. TTE or TEE suspicion or evidence for thrombus (ie, visualized thrombus/leaflet thickening or increased transvalvular gradient) should be confirmed by CTA, which can detect hypo-attenuating leaflet thickening (Figure 15, Video 14). Routine post-TTVR TTE follow-up should be performed every 6 to 12 months,[33] with the frequency dependent on an assessment of the risks for thrombosis.

OUTCOMES AFTER TTVIs

OUTCOMES ACCORDING TO BASELINE TR SEVERITY. In most published studies on TV interventions, ≥ severe TR at baseline is present in 50% to 70% of patients,[6,7,115,130] with higher TR grades associated with lower likelihood of achieving ≤2+ residual TR with T-TEER devices. However, nearly all patients who underwent TTVR with the Evoque device exhibited postprocedural mild or less TR, irrespective of baseline TR severity,[7] and in fact, showed greater improvement in the Kansas City Cardiomyopathy Questionnaire-Overall Summary Score for patients with baseline TR massive or torrential TR, compared with severe TR.[131]

FIGURE 12 Method for the Calculation of RVMW



Indices of right ventricular myocardial work (RVMW) can be calculated from pressure-strain loops. (A) Method for incorporating speckle-tracking echocardiography-derived RV strain, pulmonary pressures, and cardiac cycle timings into a pressure-strain loop. Cardiac cycle timings are determined by pulmonic and TV opening and closure events, identified through either direct visualization of 2D images or by pulsed-wave Doppler interrogation. Integration of event timings allows for the quantitative evaluation of RV dyssynchrony and post-systolic contraction. (B) Calculation of various indices of RVMW. (A) Reproduced with permission from Butcher et al.[87] PVO = pulmonic valve opening; RVGCW = right ventricular global constructive work; RVGWE = right ventricular global work efficiency; RVGWI = right ventricular global work index; RVGWW = right ventricular global wasted work; TVC = tricuspid valve closure; TVO = tricuspid valve opening; other abbreviations as in Figures 1 and 3.

TABLE 7 Presentation of Each Risk Score to Assess Outcomes in Patients With TR

Study Name	Inclusion Period, Center Type	Country/Region	Intervention	Primary Outcome
LES[102] ESII[103] (N = 22,381)	May-July 2010, multicenter	Europe, North America, Asia, Oceania	Major cardiac surgery	In-hospital mortality
STS-TV[105] (N = 13,587)	2017-2023, multicenter	United States and Canada	ITVS (repair and replacement)	30-day mortality
LaPar et al[104] (N = 2,050)	2002-2014, multicenter	United States (VA and MI)	ITVS (repair and replacement)	Postoperative mortality
Cleveland[100] (N = 9,045)	2004-2018, single center	United States	Mostly medically managed	1-year mortality
TRIVALVE[114] (N = 483)	Multicenter	Europe, North America	Transcatheter tricuspid valve repair	1-year mortality or rehospitalization
TRI-SCORE[109] (N = 466)	2007-2017, multicenter	France	ITVS (repair and replacement)	In-hospital mortality

+ = potentially useful for risk assessment; − = of limited use for risk assessment; AUC = area under the receiver operator characteristic curve; ESII = European System for Cardiac Operative Risk Evaluation II; ITVS = isolated tricuspid valve surgery; LES = Logistic European System for Cardiac Operative Risk Evaluation; STS-TV = Society of Thoracic Surgeons Isolated Tricuspid Valve Score; TR = tricuspid regurgitation.

Continued on the next page

OUTCOMES ACCORDING TO RESIDUAL TR SEVERITY. The overall rate of severe residual TR after T-TEER remains high (between 13% and 20%), and the rate of moderate TR reaches 32% to 52%.[6,39,132-134] These results contrast with those seen after surgery[109] or TTVR,[135] in which >95% of patients achieve mild or less TR. A recent large T-TEER registry suggests that procedural results may improve with advances in technology, appropriate patient selection, and increasing operator experience.[130] All studies, whether from registries or randomized controlled trials, consistently described the negative impact of residual TR on patient outcomes.[39,130,134,136,137] Although some studies suggest that ≤ moderate TR has similar outcomes to ≤ mild TR,[39,115] a recent registry of 613 patients who underwent transcatheter TV repair (T-TEER or annuloplasty) suggested that moderate residual TR is a heterogeneous group, and patients with mild and mild to moderate residual TR have better outcomes than those with moderate to severe and severe residual TR (**Central Illustration**).[134] How to quantify mild to moderate and moderate to severe TR deserves further study.[138]

The effect of residual TR may be related to the extent of reversed remodeling of the right heart,[7,139] which is associated with improved outcomes after surgical[140] or transcatheter[141] intervention. In a detailed imaging substudy of the TRILUMINATE (Trial to Evaluate Treatment With Abbott Transcatheter Clip Repair System in Patients With Moderate or Greater Tricuspid Regurgitation) randomized controlled trial using CTA and CMR, RV reverse remodeling and increases in the effective RVEF (percentage of forward SV compared with end-diastolic RV volume) were associated with the degree of TR reduction.[123] In the setting of a fixed pericardial space and ventricular interdependence, reduction in RV volumes has been associated with a septal shift toward the right heart, increases in LV end-diastolic dimensions/volume, and an increase in LV SV and cardiac output.[31,142,143]

IMAGING ENDPOINTS FOR TRIALS AND REGISTRIES

Primary endpoints for pivotal trials must address the hypothesis that treated patients experience a reduction of untoward events or feel better with treatment. Thus, imaging endpoints are usually considered secondary endpoints but may represent primary endpoints in early feasibility studies without power to detect clinically meaningful endpoints. Defining appropriate endpoints for TTVI trials is challenging because of the multifaceted nature of TR and the diversity of patient populations. The following are key considerations for defining imaging endpoints in TTVI trials.[33] Most of these parameters are dynamic and load dependent, and thus assessments should be performed under stable conditions:

- TR reduction directly measures the success of the intervention, which is the primary goal of any TTVI. Ideally, TR severity should be assessed quantitatively; however, an integrative multiparametric approach is still recommended given the limitations mentioned.[45] The grading system should offer enough granularity in both severe and moderate ranges to accurately reflect procedural success, similar to mitral valve repair.[51,144]
- Device performance provides information regarding TTVI safety and efficacy. Critical aspects include full device deployment, anterograde gradient, residual valve area, perivalvular leak, device detachment/migration/fracture, tissue erosion, or leaflet thickening/thrombosis. This

TABLE 7 Continued

C-Statistic	Internal Validation	External Validation	Potential Use in TR Population	Medical Cohort	Surgical Cohort	Transcatheter Cohort
–	No	Poor discrimination: in-hospital mortality after ITVS: AUC = 0.67 for LES and 0.62 for ESII	+/–	No	+	No
0.81 (in-hospital mortality), 0.76 (morbidity and mortality)	No	No	+	No	+	No
0.74 (in-hospital mortality), 0.76 (major morbidity)	No	No	+	No	+	No
0.71	Yes	No	+	+	No	No
0.68	No	No	+/–	No	No	+/–
0.81 (in-hospital mortality)	No	Yes: other surgical cohorts, including Asia (C-statistic between 0.82 and 0.87)	+++	+	+++	+

assessment supports the evaluation of clinical events by adjudication committees.

- RV function and remodeling provide insights into the physiological effects of the intervention. Conventional echocardiography is imprecise, but 3DE or strain imaging are promising modalities, as are the various measures of RV-PA coupling. Incorporating multimodality imaging into clinical trials may also provide invaluable mechanistic information on the physiological reverse remodeling effects of device therapies.[123]
- Hemodynamic consequences ultimately reflect the benefit of TTVI. Both an improvement in forward SV[6,142] as well as a reduction in venous congestion[145] may result in hemodynamic and end-organ benefit. Along with remodeling patterns, echocardiographic parameters of RV and LV forward SV may be secondary endpoints. Although biomarkers of liver and renal function may capture the effects of reduced passive congestion, studies of noninvasive transient elastography ultrasound measurements of liver stiffness have been associated with severity of TR[146] and may predict outcomes in patients with moderate/severe TR.[147] CMR may also detect liver stiffness and passive congestion.[148]

THE ROLE OF IMAGING CORE LABORATORIES

Core imaging laboratories provide a standardized and homogenized evaluation, reducing variability and ensuring consistency across sites. They are less prone to subjective bias favoring the intervention arm and offer the opportunity to implement standardized and comprehensive imaging protocols, especially during randomized trials, ensuring high-quality data oversight and management. Imaging core laboratories also offer the opportunity to explore and validate novel methods (ie, novel TR quantitation methods) or modalities (ie, 3DE, CMR, CT imaging) to assess outcomes. The implementation of artificial intelligence (AI) tools may improve the accuracy and reproducibility of current and future quantitative measures.

Conversely, the involvement of a core laboratory introduces logistical complexity and expense, especially in large, multicenter trials. Centralization can also lead to delays in enrollment (turnaround time). Imaging registries and self-reported imaging parameters may, on the other hand, offer a relatively low-cost, real-life practice database that often includes more diverse patient populations, thereby enhancing the generalizability of the findings. Critically, a quality control process can rarely be implemented in such registries given the variability in imaging protocols, data quality, and operator measurement variability because it is not standardized across sites, which affects data reliability and reproducibility.

ADVANCES IN IMAGING FOR TR

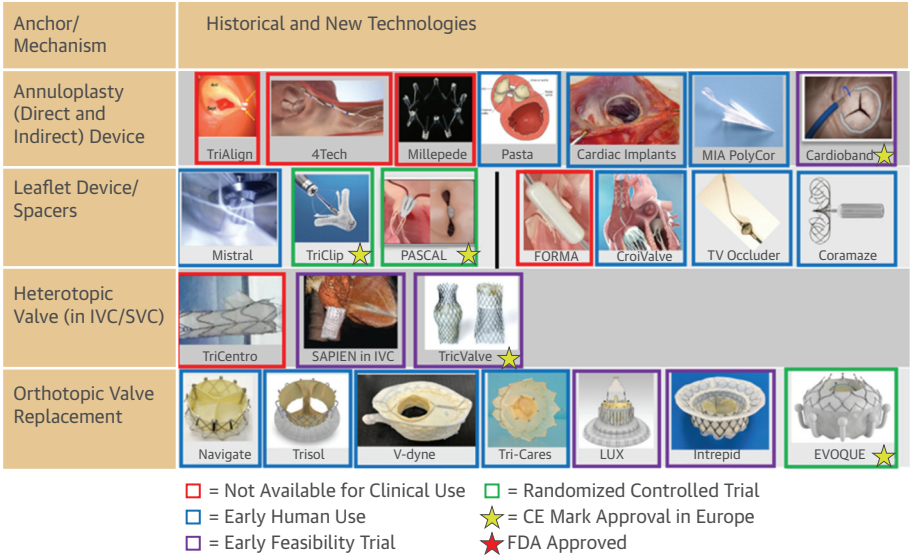
AI AND TV ANALYSIS. There is significant interest regarding the increasing role of AI in aiding the diagnosis and management of cardiovascular diseases. In particular, the ability to simultaneously integrate and process large amounts of data to identify information that might be missed by the human eye is very appealing.[149,150] In the field of TR, machine learning models have emerged providing better prediction of RV-PA coupling by estimating the invasive mean arterial pulmonary pressure from echocardiographic parameters[151] or by automating the segmentation of functional CTA data sets for the assessment of RVEF that is associated with outcomes.[25] These models may provide fast and predictive tools for imaging reconstruction and interpretation for a large number of patients to

FIGURE 13 Anatomical Factors Involved in the Decision to Perform Transcatheter Repair or Replacement of the TV

Favorable Anatomy for T-TEER	Feasible Anatomy for T-TEER	Criteria Favoring Replacement
• Small septolateral coaptation gap ≤7 mm • Anteroseptal jet location • Localized prolapse or flail • Bi- or trileaflet morphology • No CIED lead • Good echocardiographic window for leaflet visualization	• Septolateral coaptation gap >7 but ≤8.5 mm • Posteroseptal jet location • Multiple leaflets (>3) • Lead-associated TR without permanent leaflet interaction • Sufficient echocardiographic window for leaflet visualization	• Large septolateral coaptation gap >8.5 mm • Anteroposterior jet location • Multiple leaflets (>3) and indentations • Leaflet thickening/shortening (rheumatic, carcinoid)/perforation • Pronounced leaflet tethering • CIED-related TR (impingement, adhesion, perforation, subvalvular entanglement) • Insufficient echocardiographic leaflet visualization

Multiple anatomical predictors of device efficacy for TR reduction have been described based on primarily echocardiographic parameters (Supplemental Table 1) and are summarized in the graphic. as the complexity and severity of TR increases, the transcatheter edge-to-edge repair (TEER) device may not achieve adequate TR reduction, and transcatheter tricuspid valve intervention (TTVI) may be more appropriate. Modified from Hausleiter et al.[22] T-TEER = transcatheter tricuspid edge-to-edge repair; other abbreviations as in Figures 1, 8, and 10.

FIGURE 14 Classes of Transcatheter Devices for Treatment of TR



The broad categories of device therapies for treatment of severe, symptomatic TR are: annuloplasty, leaflet device or spacers, heterotopic valve replacement, and orthotopic valve replacement. The annuloplasty devices may be directly implanted in the annulus or affect annular reduction indirectly. The leaflet/spacer devices attempt to reduce the coaptation gap by either bringing the leaflet tips together (leaflet coaptation devices) or filling the regurgitant orifice (spacers). Heterotopic valve replacement devices address the passive congestion resulting from TR, whereas the orthotopic valve replacement devices are seated within the annulus. FDA = U.S. Food and Drug Administration; SVC = superior vena cava; other abbreviations as in Figures 1, 8, and 10.

TABLE 8 Imaging Parameters for Assessing TR Severity following Transcatheter Tricuspid Valve Intervention

	Baseline	Post-TEER	Post-Annular Repair	Post-TTVR (Orthotopic)
Qualitative parameters				
Flow convergence zone	+	+	+	+
CWD jet density/shape	+	+	+	+
Semi-quantitative parameters				
Color flow jet area	++	+	+	+
VC width (average of orthogonal views)	+++	+ (adding multiple jets has not been validated)	+++	+++
PISA radius	+++	± (abnormal shape of proximal flow may result in overestimation)	+++	+++ (for central TR)
Hepatic vein flow pattern	++	± (abnormal RA compliance may affect specificity)	± (abnormal RA compliance may affect specificity)	± (abnormal RA compliance may affect specificity)
Quantitative parameters				
PISA EROA	+++	± (abnormal shape of proximal flow may result in overestimation)	+	+++ (for central TR)
2D Doppler quantitative EROA	++	–	+	–
3DE VCA	+++	+++	+++	+++
Regurgitant volume				
Echocardiography	+++	++ (3DE)	++ (3DE)	++ (3DE)
CMR	+++	++	++	++
Regurgitant fraction				
Echocardiography	+++	++ (3DE)	+++	++ (3DE)
CMR	+++	++	++	++

Given the diastolic flow restriction by the device, diastolic SV will be overestimated. Regurgitant fraction may be performed if a total RV SV is obtained by other methods (ie, 3DE or CMR), and regurgitant volume is quantified by 3D-VCA. Modified from Hahn et al.[33]

Abbreviations as in Tables 1, 2, and 4.

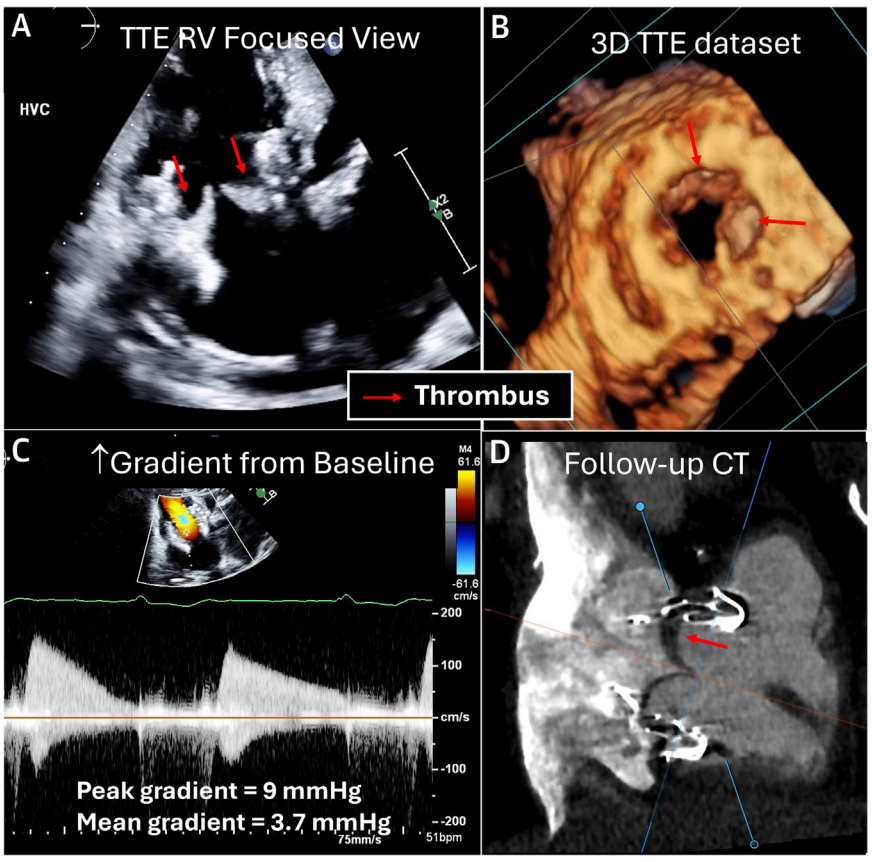
reduce barriers to care and provide timely diagnosis and assessment of eligibility for a specific transcatheter treatment.

The promise of AI must be tempered by the known biases of these methods, particularly for imaging modalities such as echocardiography.[152] For diseases such as TR specifically, in which arrhythmias, respirophasic variability, or image quality may be particularly problematic, the use of AI for disease identification might be limited. Recent errors of exported Digital Imaging and Communications in Medicine structured reporting data have also highlighted the possibility of misdiagnosis of valvular heart disease if careful curation of data is not performed.[153]

Moreover, AI may help to identify phenogroups with different clinical risk profiles that could potentially enrich event rates for clinical trials.[154-156] Future prospective trials and/or registries are required to determine the role these AI models might have in tailoring individual-based decisions regarding the best time and type of TR treatment. Finally, AI-guided procedures hold the promise of reducing operating times and intraprocedural complications while simultaneously increasing the numbers of procedures performed. The role of AI in treating patients with TR has yet to be explored.

COMPUTATIONAL MODELING. Computational modeling integrates multiple data sets to predict the best personalized diagnostic and therapeutic approaches to a specific patient. In the field of tricuspid disease, it may significantly improve our ability to understand TV biomechanics[157,158] and to predict the results of a planned intervention.[159,160] This could be particularly relevant considering that the number of available devices will progressively increase along with the complexity of preprocedural planning. Although the current focus has been on preclinical engineering models,[157,161,162] improvements in multidimensional data annotation and curation could allow computational simulations with creation of digital twins and important practical applications for patient selection and prediction of procedural outcomes.[163] Adding the right ventricle into computational models of TR will allow prediction of the short- and long-term effects of tricuspid intervention on RV function, further improving candidate selection. Some areas of investigation in which computational modeling could be helpful include refinement of patient selection for repair vs replacement, prediction of low cardiac output syndrome post-TV intervention, repair strategy (number and location of clips), post-TTVR pacemaker need and post-CIED lead management, and prediction of hypo-attenuating leaflet thickening.

FIGURE 15 Assessment of TTVR Function



TTE is recommended for follow-up of device position and function. (A,B) In this example, thickening of the base of 2 transcatheter tricuspid valve replacement (TTVR) leaflets is shown, confirmed on 3D reconstruction (B), and suspicious for thrombus (red arrows). The change in mean gradient from 2.0 to 3.7 mm Hg (C) confirms the hemodynamic importance of imaging findings, and a computed tomography (CT) scan shows hypoattenuated leaflet thickening (D) grade 3 involving the anterosuperior leaflet (red arrow) and associated reduced leaflet mobility (Video 14). Abbreviations as in **Figures 4, 10, and 12**.

NOVEL PREDICTORS OF OUTCOMES. Conventional echocardiographic parameters for assessment of RV function are load dependent and may overestimate intrinsic function in patients with significant TR. Imaging surrogates of the RV-PA coupling[164] and effective RVEF by either CMR[77,123] or 3DE[78] may account for the load dependency of other parameters of RV function. Noninvasively measured pressure-volume loops to assess contractility, RV-arterial coupling, and RV myocardial work using an assumed reference RV pressure curve and strain imaging[87,165,166] require further study.

SUMMARY

Significant STR is associated with an increased risk of mortality and a reduced quality of life.

Advanced imaging techniques have played an integral role in the rapid advancement of our understanding of normal and abnormal right heart anatomy and pathophysiology. Imaging has reshaped our classification of disease morphology, redefined our understanding of disease severity, contributed to our assessment of disease risks, defined anatomical and physiological feasibility of interventions, and helped determine the therapeutic goals of therapy.

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HIGHLIGHTS

- TR is independently associated with increased risk of mortality and reduced quality of life.
- Advanced imaging modalities affected the rapid advancements in understanding TR pathophysiology.
- Leveraging novel imaging parameters may improve early detection and patient selection for appropriate management options.

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KEY WORDS echocardiography, multimodality imaging, tricuspid regurgitation

APPENDIX For an expanded Methods section as well as supplemental videos, figures, tables, and references, please see the online version of this paper.

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