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iREVIEW

STATE-OF-THE-ART REVIEW

Tricuspid Regurgitation

From Imaging to Clinical Trials to Resolving the Unmet Need for Treatment

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ABSTRACT

Tricuspid regurgitation (TR) is a highly prevalent and heterogeneous valvular disease, independently associated with excess mortality and high morbidity in all clinical contexts. TR is profoundly undertreated by surgery and is often discovered late in patients presenting with right-sided heart failure. To address the issue of undertreatment and poor clinical outcomes without intervention, numerous structural tricuspid interventional devices have been and are in development, a challenging process due to the unique anatomic and physiological characteristics of the tricuspid valve, and warranting well-designed clinical trials. The path from routine practice TR detection to appropriate TR evaluation, to conduction of clinical trials, to enriched therapeutic possibilities for improving TR access to treatment and outcomes in routine practice is complex. Therefore, this paper summarizes the key points and methods crucial to TR detection, quantitation, categorization, risk-scoring, intervention-monitoring, and outcomes evaluation, particularly of right-sided function, and to clinical trial development and conduct, for both interventional and surgical groups.
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ABBREVIATIONS AND ACRONYMS

3D = 3-dimensional

CMR = cardiac magnetic resonance

CT = computed tomography

EFS = early feasibility study

ICE = intracardiac echocardiography

MRI = magnetic resonance imaging

PA = pulmonary artery

RA = right atrium

RV = right ventricular

RVEF = right ventricular ejection fraction

sPAP = systolic pulmonary arterial pressure

TAPSE = tricuspid-annular-plane-systolic-excursion

TEER = transcatheter edge-to-edge repair

TR = tricuspid regurgitation

TTE = transthoracic echocardiogram

Tricuspid regurgitation (TR) represents a considerable clinical and therapeutic challenge. Moderate or severe TR is highly prevalent with an estimated incidence of 160,000-240,000 new cases per year in the United States alone.[1-3] TR is often underdiagnosed,[2,4] due to nonspecific symptoms, lacking or evanescent murmur, and subtle physical signs.[1]

Another challenge is the complex anatomy of the tricuspid valve and the optimal assessment of the regurgitation severity, which has an impact on prognosis and therapeutic approaches.[3-6] Increasing TR severity has been associated with higher mortality, independent of pulmonary hypertension, atrial fibrillation, and right ventricular (RV) systolic function.[7-13] Severe TR is associated with poor outcomes even when these confounders are absent.[14]

Surgical intervention for severe TR without concomitant coronary artery bypass grafting or left-sided valve surgery has high risks with operative mortality averaging 9%-12%.[15] Operative and long-term risks are higher when patients are referred late[16-18] in the course of the disease, with NYHA functional class III/IV dyspnea, ascites, or refractory heart failure.[19] Also, surgical tricuspid repair/annuloplasty is associated with high TR recurrence, from 25%-40%.[20] Similarly, existing transcatheter repair approaches, although encouraging,[21,22] are complicated by persistent/recurrent TR[23] and by residual ventricular consequences of TR.[24] Transcatheter treatment challenges are several, including fragile leaflets, which are highly variable in size/number, the complex RV anatomy, tenuous tricuspid annulus, proximity to conduction system and right coronary artery, and frequently extremely large coaptation gaps. Furthermore, iatrogenic TR from cardiac implantable electronic devices adds to the complexity of transcatheter treatment options.

These therapeutic challenges contribute to current TR undertreatment. Nationwide and observational surveys suggest a 4%-5% overall treatment rate for moderate/severe TR, befitting limited guideline-based surgical indications.[25,26] The >90% undertreatment of patients with moderate/severe TR has been confirmed in multiple clinical cohorts, even those eminently surgical.[3,9-11] Therefore, TR is rarely treated effectively, and even clinical trials such as the recently reported TRILUMINATE (Clinical Trial to Evaluate Cardiovascular Outcomes In Patients Treated With the Tricuspid Valve Repair System Pivotal) trial[27] suffer considerable challenges in affirming therapeutic benefits. These challenges emphasize the need for transformation of clinical practice to better detect/assess/refer affected patients and for better integration of comprehensive multimodality imaging.[28] Consequently, we address herein crucial needs regarding TR diagnosis, quantification, multimodality imaging, and management, emphasizing the role of Heart Team collaboration and appropriately designed clinical trials to improve outcomes for all patients with TR.

COMPREHENSIVE ASSESSMENT OF PATIENTS WITH TR

Patients are often referred late in the course of disease with advanced symptoms, ascites, and liver and renal dysfunction. It is unclear if treating TR in such advanced stages offers meaningful clinical benefit. Furthermore, studies should evaluate effect of earlier treatment of TR.[16] For diagnosis, and definition of causes/mechanisms/consequences, imaging plays a central role. Our plea is for a changing paradigm, with TR treatment considered early and selected carefully based on multimodality imaging to reduce risks of irreversible RV damage, organ failure, and residual heart failure (**Central Illustration**).

DIAGNOSTIC TR IMAGING: TRICUSPID VALVE STRUCTURE AND REGURGITATION SEVERITY.

Echocardiography. Echocardiographic TR assessment is challenging on account of anatomic, physiologic, and technical-imaging peculiarities affecting reliability/consistency of assessment.

Tricuspid valve morphologic assessment mostly done using transthoracic and transesophageal echocardiography is crucial to differentiate TR causes and mechanisms and select appropriate structural interventions or surgery. Tricuspid valve has highly variable anatomy[28] in terms of numbers of scallops of the individual leaflets, shape of regurgitant orifice,

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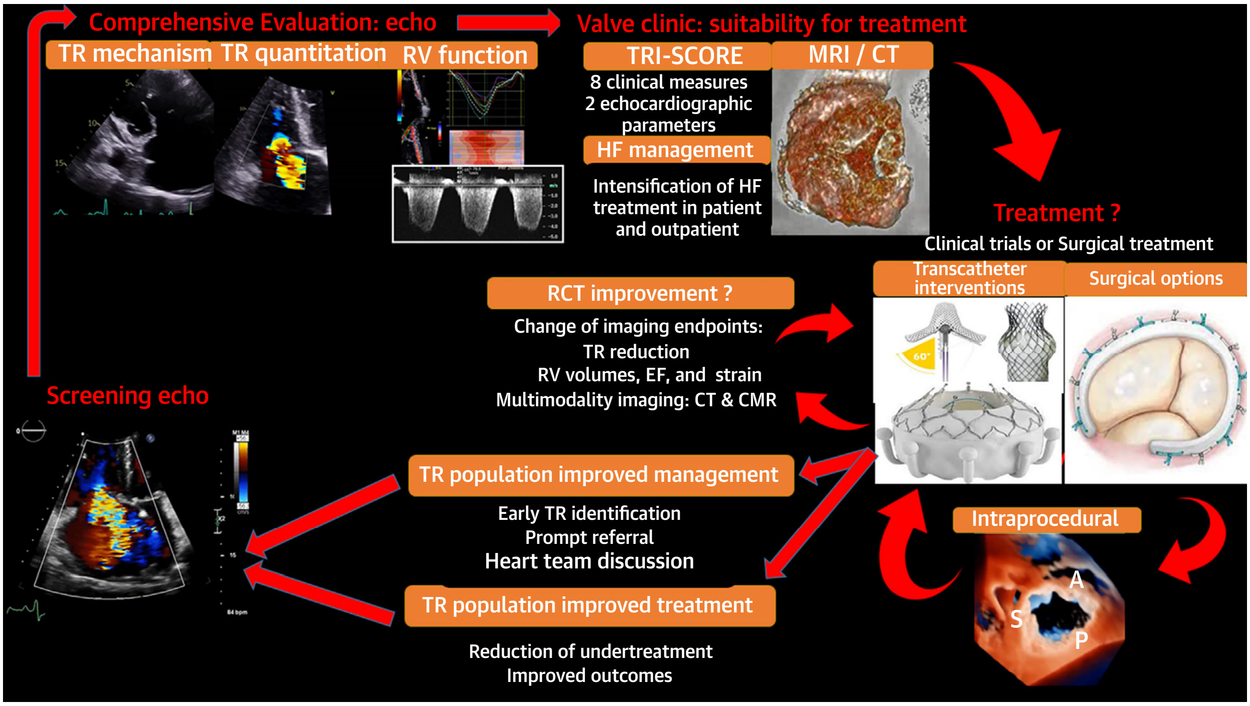
CENTRAL ILLUSTRATION Imaging and Management Pathway for Functional TR



Grapsa J, et al. J Am Coll Cardiol Img. 2024;17(1):79-95.

CMR = cardiac magnetic resonance; CT = computed tomography; EF = ejection fraction; HF = heart failure; MRI = magnetic resonance imaging; RCT = randomized controlled trial; RV = right ventricle; TR = tricuspid regurgitation.

and annular size, which are all important to understand when using comprehensive imaging to decide appropriate intervention.[29,30] Cardiac implantable electronic device leads can also affect the tricuspid valve by impinging on or adhering to leaflets, interfering with subvalvular apparatus, and perforating/lacerating leaflets; also, they may cause leaflet avulsion, particularly during lead extraction.[31,32] Incidence of new or worse TR severity after cardiac implantable electronic devices implantation ranges between 10% and 39%[33] with 5% of patients developing moderate/severe TR 1 year after implantation.[34] It is essential to comprehensively describe TR causes and mechanisms (**Table 1**)[2] and 3-dimensional (3D) echocardiography can be particularly useful.[35] Progressive annular enlargement is the predominant mechanism of TR progression leading to development of coaptation gap

TABLE 1 Classification of TR Causes and Mechanisms

TR Causes	TR Mechanisms: Type I Normal Movement	TR Mechanisms: Type II Excessive Movement	TR Mechanisms: Type III Restricted Movement
Organic	Perforation, AV canal/cleft	Flail leaflet (spontaneous endocarditis), TVP, rupture papillary muscle	Rheumatic, carcinoid Epstein anomaly
Iatrogenic	Perforation (lead/catheter), commissural separation	Ruptured chordae, leaflet tear (biopsy/PM/ICD/ablation)	Ergot derivatives lead impingement
Functional	Atrial FTR (primary, postatrial arrhythmia)		Pulmonary HTN RV dysfunction RV infarction

Reprinted from Topilsky et al.[5]

AV = atrioventricular; FTR = functional tricuspid regurgitation; HTN = hypertension; ICD = implantable cardiac defibrillator; PM = pacemaker; RV = right ventricle; TVP = tricuspid valve prolapse.

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TABLE 2 Limitations of Qualitative TR Assessment With Transthoracic Echocardiography

Imaging Parameter	Limitations
Jet extent into RA	Dependent on jet eccentricity and on fluid dynamics[43]
TR regurgitant volume[44,45]	Varies with respiration,[45] jet may be small in large atrium, displaying less aliasing, yielding TR underestimation[31,32]
Systolic hepatic venous flow-reversal	Dependent on presence of large right atrial V-waves and thus represents a rather insensitive sign

RA = right atrium; TR = tricuspid regurgitation

between the leaflets.[36] Tricuspid valve tenting, due to eccentric RV remodeling,[5] also contributes to TR progression.

Transcatheter edge-to-edge repair (TEER) is most frequently performed now but its procedural success is associated with coaptation-gap ≤8.5 mm, central/anteroseptal jet location, and tethering height ≤1 cm.[29,35,37,38] Therefore, careful anatomic quantitative description and preprocedural imaging are key to TR reduction and improved outcomes.

TR severity assessment using echocardiography. TR severity assessment using echocardiography is crucial for selecting patients for appropriate interventions and evaluating the success and durability of the intervention.[9,10,12,14] Recent TEER trials suggested that TR reduction (vs elimination) may yield clinical benefits,[23,27] and, for TR grading in clinical trials,[28] segmenting further the severe range into massive and torrential grades may be useful.[39] TR improvement by at least one of these extended grades[26] is almost universally observed post-TEER,[27,40,41] whereas transcatheter valve replacement more uniformly reduces TR to mild or less.[42]

Limitations of TR qualitative assessment are summarized in Table 2.[43-48] To address these pitfalls, it is essential that quantitative TR assessment becomes the rule rather than the exception in routine clinical practice, as well as during the selection process for inclusion in clinical trials.

TR quantitation tips and tricks are summarized on Table 3 and Figure 1. TR quantitation displays strong links to outcomes,[46-52] underscoring its relevance.[14,53-55] Severe TR is defined as effective regurgitant orifice area ≥0.40 cm^2 and regurgitant volume ≥45 mL but regurgitant volume is more difficult to integrate in TR grading because it varies with RV afterload, ie, the TR driving force. TR quantitation, when feasible before and after tricuspid interventions, also allows us to precisely define TR improvement postintervention.[56]

Effective regurgitant orifice area has proved to be a direct link to clinical outcomes and these quantitative measurements are indispensable components of trials regarding TR treatment and outcome improvements.[14,53-55]

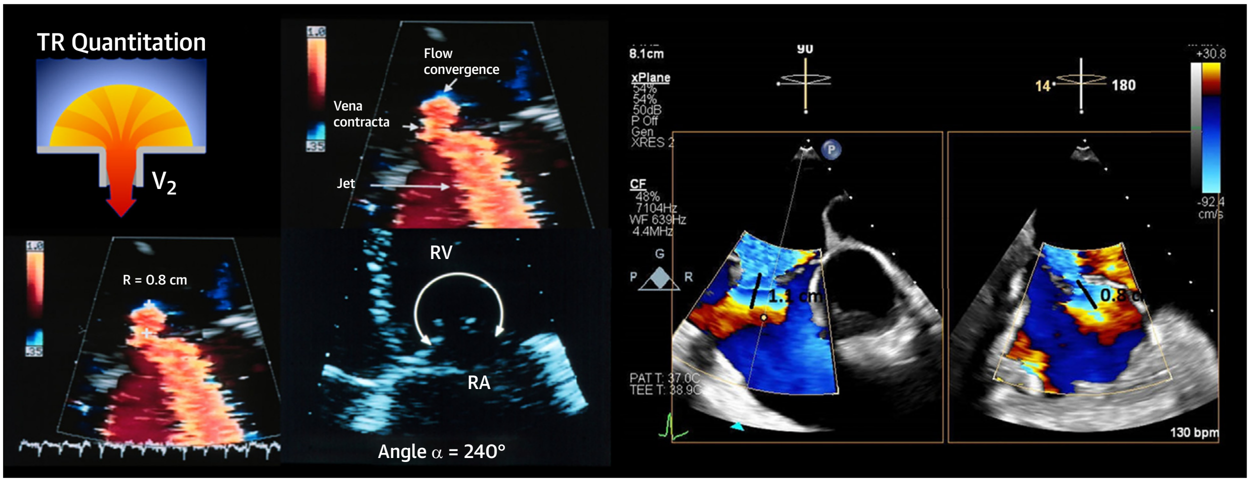
CARDIAC MAGNETIC RESONANCE. Cardiac magnetic resonance (CMR) is important in evaluating right-sided heart function and TR severity, although guidelines suggest using it mostly if echocardiography is inconclusive.[57] CMR imaging can pose challenges in patients with atrial fibrillation, device leads, and chronic kidney disease, however, technology such as compressed sensing with real-time cine in atrial fibrillation allows faster acquisition with superior image quality[58,59] and wideband late-gadolinium enhancement reduces artifacts related to implantable cardiac devices.[60] 3D assessment of tricuspid annulus is feasible using CMR without contrast. Although

TABLE 3 Tips and Tricks for the TR Quantitative Echocardiographic Assessment

	Tips and Tricks
TR vena contracta	Used semiquantitatively, with width >7 mm favoring severe TR and ideally performed in 2 different planes[35,46]
3D TR vena contracta	Less reliable with complex orifice shapes, sensitive to color gain, and may be underestimated with multiple jets[47-49]
EROA/PISA	1. Use an aliasing velocity close to 10% of peak TR velocity[50] 2. Record multiple beats to account for TR respiratory dependency[45] 3. Account for angle of leaflet tenting[51] Correction for both leaflet angle and lower velocity of TR jet is essential to avoid underestimation of TR[52] Important limitations of EROA measurement need to be acknowledged, such as orifice shape influence on EROA reliability, uncertainty about true hemispheric shape of flow convergence, and angle correction for regurgitant flow measurement. These may yield significant underestimation of TR severity.

3D = 3-dimensional; EROA = effective orifice area; PISA = proximal isovolumic surface area; other abbreviations as in Table 2.

FIGURE 1 Quantitative Assessment



Quantitative assessment of TR severity based on TTE views (left) and TEE views (right) and demonstrating the various parts of the regurgitant flow, measurement of the flow convergence, and assessment of the inverted funnel angle. TEE = transesophageal echocardiogram; TR = tricuspid regurgitation; TTE = transthoracic echocardiogram.

tricuspid valve leaflet anatomy using CMR is not typically emphasized, standard cine imaging with contiguous stack perpendicular to the coaptation gap can facilitate visualization of tricuspid morphology and gaps (**Figure 2**).[32,61] 3D assessment using CMR remains under research given large computational requirements. TR severity is measured by subtracting forward pulmonic valve flow by 2-dimensional phase contrast from RV stroke volume, and thresholds such as regurgitant volume ≥45 mL and/or TR fraction ≥50% are associated with high risk for cardiovascular events.[62]

COMPUTED TOMOGRAPHY. Computed tomography (CT) has growing importance in TR evaluation, particularly for transcatheter interventions. Challenges for CT imaging acquisition include atrial arrhythmias, intracardiac devices/leads, and chronic kidney disease. Electrocardiogram gating covering the whole cardiac cycle, without dose modulation, and using the smallest available collimation (0.5-0.625 mm) are key for high-quality functional reconstruction. Technical timing of image-acquisition and contrast-injection is essential to CT quality and yield. CT is complementary to transesophageal echocardiography for visualizing leaflets and TR mechanisms, TR quantification,[63] and anatomical measurements for device selection (**Figure 3**).

CARDIAC CATHETERIZATION. Presence of severe pulmonary hypertension increases operative risk underscoring the importance of comprehensive right-sided heart catheterization. Because severe TR may compromise Doppler-based pulmonary-artery-pressure estimation accuracy, right-sided heart catheterization is often recommended. Although pulmonary hypertension is associated with worse outcomes after TEER, particularly precapillary hypertension,[64] its detection using catheterization vs Doppler may be more specific for these poor outcomes.[65] Patients with pulmonary vascular resistance >3 WUs and/or systolic pulmonary artery pressure ≥70 mm Hg are usually excluded from transcatheter therapies.

DIAGNOSTIC TR IMAGING: CARDIAC REMODELING AND RV FUNCTION. Although interventional trials focused imaging endpoints on TR severity reduction, there is growing interest in reverse RV remodeling.[22] Global RV dysfunction defined as RV ejection fraction (RVEF) ≤45% predicts outcomes after transcatheter therapy,[66] but reduced RV function also affects outcome in patients managed medically alone.[67] Transcatheter TR treatment yields reverse RV and right atrium (RA) remodeling and slight improvement in RV systolic function.[22,41] Several structural trials used tricuspid-annular-plane-systolic-excursion (TAPSE) as an index of RV dysfunction, displaying postprocedural improvement after transcatheter

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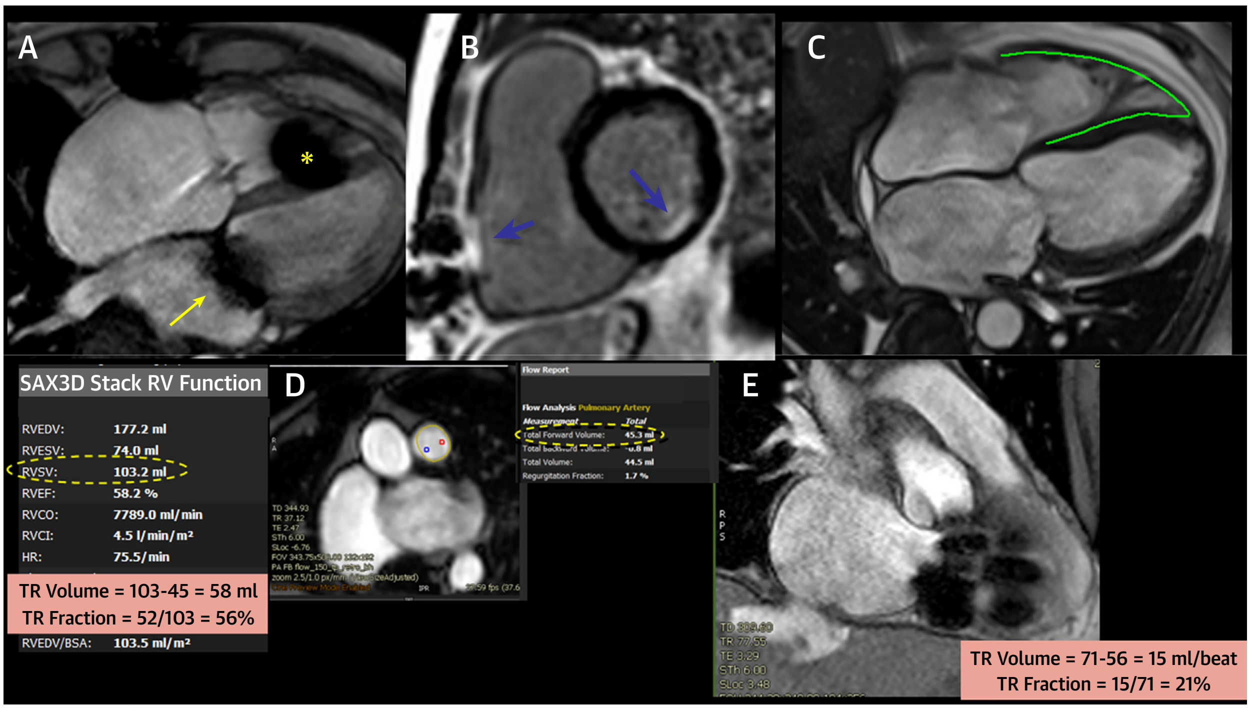
FIGURE 2 CMR in TR



(A) Systolic frame demonstrating dephasing across tricuspid valve consistent with TR in a patient with prior mechanical MV replacement (yellow arrow), and leadless Micra pacemaker (yellow asterisk). (B) Late gadolinium enhancement demonstrates subendocardial infarct (blue arrows) involving the basal inferior and right ventricle. (C) Feature-tracking strain of the right ventricle. (D) Assessment of RV volumes and ejection fraction pre TriClip. TR severity is measured by subtracting from RV stroke volume the forward pulmonic valve flow using 2D phase contrast. (E) After 3 TEER devices, note significant TR reduction with decrease in TR volume from 58-15 mL, and TR fraction from 56%-21%. Importantly, there was an improvement of forward RV stroke volume from 45-56 mL (24%). 2D = 2-dimensional; CMR = cardiac magnetic resonance; MV = mitral valve; RV = right ventricular; TEER = transcatheter edge to edge repair; other abbreviations as in Figure 1.

valve replacement.[68] However, results of various registries are discordant. Few studies suggest that TAPSE is associated with survival after TR procedure and TR elimination,[24,69] but most find no independent role of TAPSE in predicting outcome after TEER.[66,70-72] Limitations of TAPSE are its volume dependency and restriction to RV longitudinal function.

RV-pulmonary artery (PA) coupling refers to the ability of RV to respond to the RV afterload, currently measured as TAPSE/systolic pulmonary arterial pressure (sPAP) ratio.[73,74] RV-PA uncoupling has been shown to be associated with poor outcome in patients treated medically and also those undergoing intervention.[75,76] In a TEER registry, TAPSE/sPAP ≥0.41 (preserved RV-PA coupling) was associated with lower all-cause mortality, irrespective of baseline TAPSE value.[75] Decrease of TAPSE/sPAP is observed after successful TR reduction in patients with preserved coupling, although no change is observed in those with reduced coupling. RV contractile reserve assessment based on stress echocardiography is promising in pulmonary hypertensive and heart failure,[77] and a dedicated transcatheter trial is underway (NCT04141683).

Although intrinsic RV function assessment with severe TR is challenging, RVEF and indexed right ventricular end-systolic volume derived using CMR has been shown to provide prognostically important information in severe TR before surgical intervention.[78-80] CMR pre-TEER showing RVEF <45% is associated with worse mortality/heart failure, whereas reduced longitudinal function compensated by an increase in circumferential function with preserved RVEF had favorable outcomes.[66] In addition to volumetric assessment, CMR feature-tracking RV free-wall longitudinal strain provides incremental risk stratification to clinical and imaging variables in severe functional TR[81] (**Figure 3**). Small series imaged serially using CMR showed sustained post-TEER RV reverse remodeling and cardiac index improvement.[82] Whether RV fibrosis quantitation[79] is impactful on TR outcome remains to be evaluated.

CT also importantly contributes to evaluation of RV size, function, and dynamic changes throughout the cardiac cycle.[83] Although it can be time consuming, semiautomated machine-learning algorithms will facilitate postprocessing for routine clinical assessment. Similar to CMR, functional CT can also assess therapeutic impact of tricuspid interventions. The TRILUMINATE Imaging substudy (NCT03904147) and ongoing TRI-FR substudy (NCT04646811) with systematic multimodality and sequential TR evaluation will inform the design of future imaging in structural interventions.

Thus, comprehensive RV assessment is crucial in clinical trials, in the outcome of TR treated medically vs transcatheter interventions, and in defining RV indices thresholds at which interventions may be beneficial or futile.[24] Thus, prospective registries and trials should include systematic RV volumes and function measurements based on 3D echocardiography,[84] CT, or magnetic resonance imaging (MRI) and by RV strain.[85]

INTRAPROCEDURAL MONITORING/IMAGING

Transesophageal echocardiography remains the imaging of choice for comprehensive tricuspid evaluation[86] and guiding transcatheter interventions.[28] Intraprocedural imaging requirements depend on transcatheter procedure types: leaflet coaptation devices (ie, TEER or spacers), annular repair devices, and orthotopic and heterotopic valve replacement. Superior and inferior vena cava provide easy transcatheter access to the tricuspid valve and should be comprehensively imaged to detect lateralization and convergence of outlets predominant in older subjects.[87]

For TEER, TR etiology, leaflet lengths, tethering characteristics, and coaptation gap at regurgitant orifice allow us to determine location, orientation, and number of devices that may be needed (Supplemental Table 1). Guiding devices safely into the RV, imaging leaflet insertion, and ascertaining grasping are key to procedural success.[88] Transgastric views yield unobstructed leaflet/device imaging, crucial for orientation and positioning. Conversely, mid-esophageal views are essential to assess leaflet insertion, using live 3D reconstruction. Hence, alternating imaging views are often warranted. Transesophageal echocardiographic assessment of residual TR will determine whether additional devices are required.

For annular repair devices, transesophageal imaging detects anchor annulus insertion and leaflets' hinge points avoidance. Acoustic shadowing often mandates transgastric views to ascertain anchors' deployment and sometimes intracardiac echocardiography.[89] Transesophageal intraprocedural assessment of TR severity allows real-time adjustments and optimal TR reduction.

Orthotopic valve replacement encounters similar challenges of acoustic shadowing from large devices in the RA. Depending on anchoring method, leaflet hinge-points/annulus must be clearly imaged, relying heavily on real-time 3D imaging. These devices have shown the most complete TR reduction,[42] which may result in afterload increases and secondary RV function alteration. Thus, careful assessment of baseline and postdevice RV size and function is warranted.

Finally, heterotopic valve replacements are used in patients who are not candidates for other devices.[90] Placement of these devices in vena cava may not rely on transesophageal but rather fluoroscopic landmarks. Nonetheless, transesophageal echocardiography assesses changes in TR and forward stroke volume often with RV/RA-gradient increase associated with successful TR reduction.

In monitoring these implants, real-time multiplanar reconstruction is a novel tool allowing independent imaging views without fixed angulation. 3D multiplanar reconstruction allows visualization of peripheral structures and exact orthogonal distance measurements during the procedure without need for off-line analysis despite low-spatial resolution compared with biplanar modes.

Intracardiac echocardiography (ICE) is promising because transesophageal guidance is often impaired by shadowing through foreign bodies, particularly pacemakers/defibrillators, left-sided mechanical or biological prosthesis, and surgical rings.[31,32] Lipomatous septum or malpositioned horizontal heart may shadow the septal leaflet and impede its visualization and capture. Few patients may have contraindications for transesophageal echocardiography. ICE for transcatheter tricuspid therapies can be complementary or an alternative imaging modality (Supplemental Table 1).[91]

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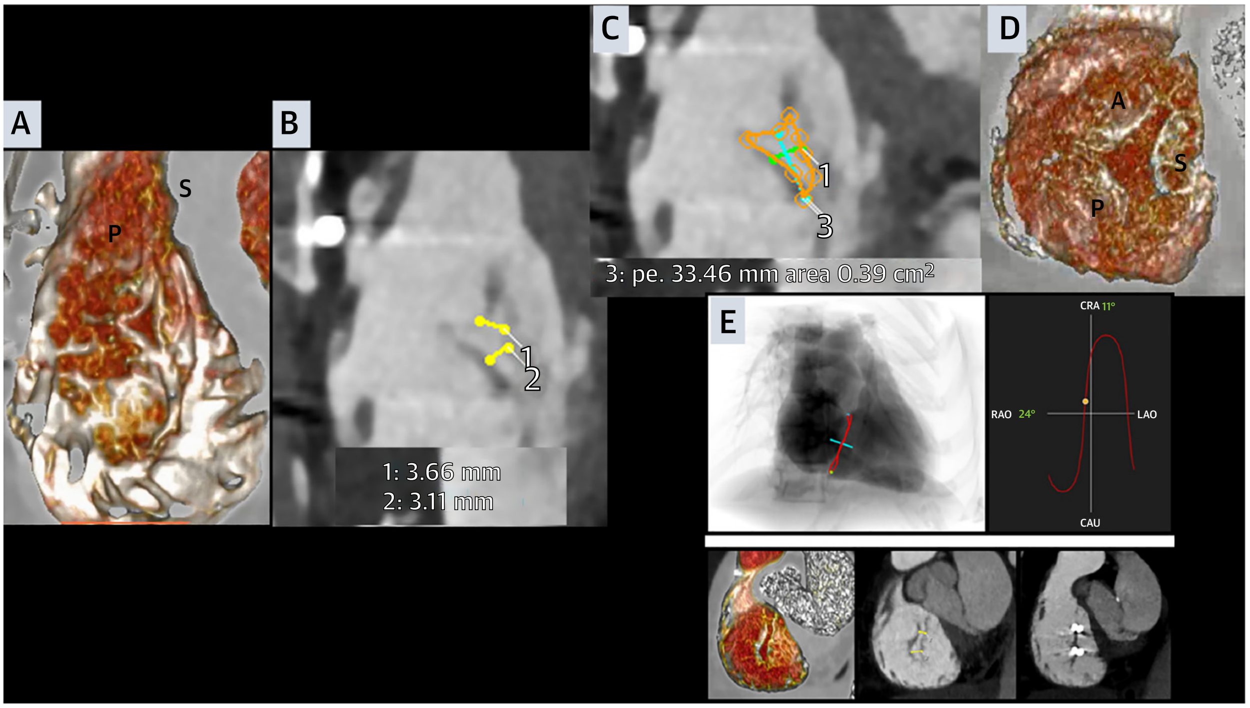
FIGURE 3 **CT Tricuspid Valve Imaging**



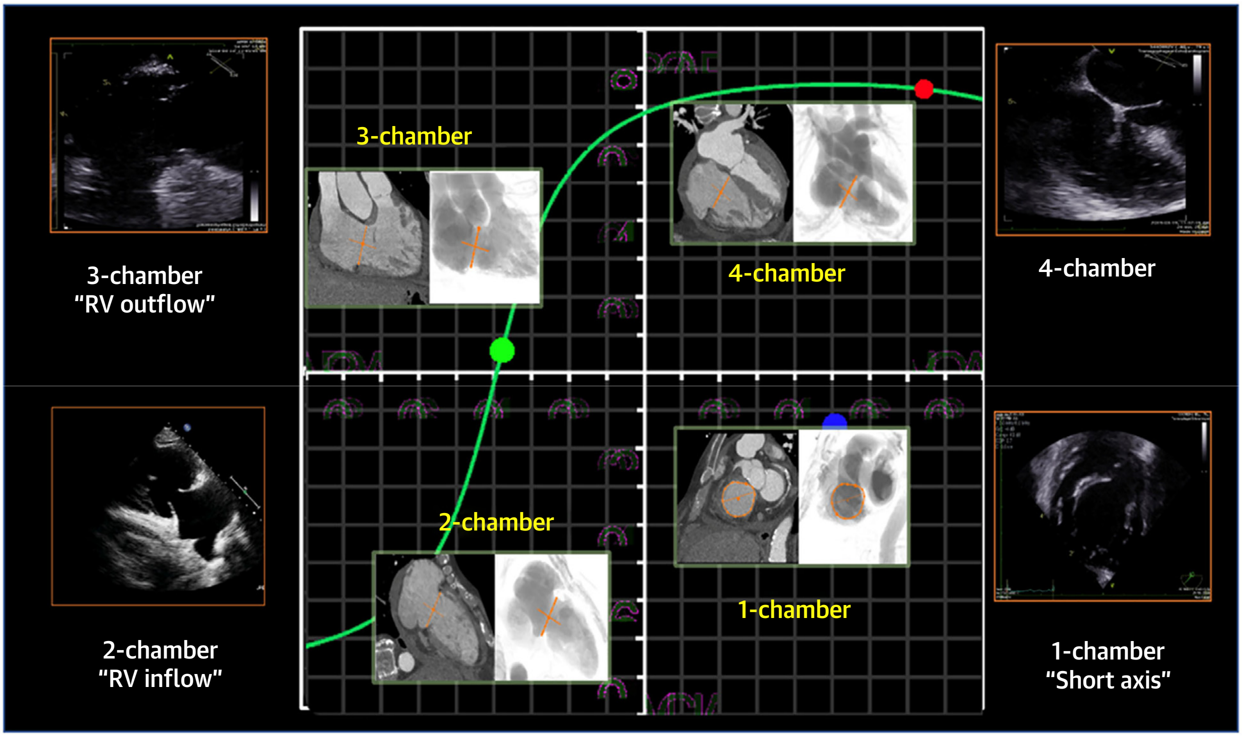
(A) Trileaflet (A = anterior; P = posterior; S = septal) tricuspid valve from RV en-face view in systole with central coaptation gap. (B) Small anteroseptal gap (#1 = 4 mm) and posteroseptal gap (#2 = 3 mm). (C) Tricuspid anatomical regurgitant orifice area = 0.4 cm^2, which is consistent with severe TR. (D) Different patient with trileaflet TV and large systolic gaps. (E) Comprehensive TEER planning using cardiac CT with orthogonal fluoroscopic angle. CT = computed tomography; TV = tricuspid valve; other abbreviations as in **Figures 1 and 2**.

Easy accessibility of right atrium and new 4-dimensional steerable ICE catheters offer potential advantages for guidance. In addition to Doppler, 2-dimensional and M-mode, real-time imaging in several planes (multiplanar reconstruction), as well as instantaneous acquisition and display of 3D volumes is possible. Successful ICE-guided procedures have been reported with outcomes similar to transesophageal,[92,93] which may allow tricuspid procedures under local anesthesia/sedation.

Hybrid or fusion imaging of fluoroscopy and other modalities is increasingly used for guiding percutaneous procedures but might have a role in the future also for planning and selection of the best device. Overlay of transesophageal echocardiography or CT images with fluoroscopy has been used in facilitating transcatheter interventions for congenital and structural heart disease.[94,95] Fusion of CT-echo imaging seems also promising for annulus sizing, procedural landmarks, and access planning. Although further improvement of automated recognition of right-sided heart structures is needed, harmonizing 2 imaging modalities provides better understanding of 3D relationships of anatomy and devices and promises procedural efficiency and safety. CT fusion does not allow synchronous movement of fused images during the heart cycle and limited applicability for valve treatment.

Tricuspid valve S-curve obtained using CT provides fluoroscopic viewing angles of the annulus in-plane (**Figure 4**).[96] Thus, planar/en-face views of tricuspid annulus can eliminate parallax and improve accuracy in device delivery and deployment. Fluoroscopic "chamber views," like those obtained with echocardiography, can be derived from patient-specific CT. Fluoroscopic right-sided 1-chamber (short-axis), 2-chamber, 3-chamber (inflow/outflow), and 4-chamber views can be obtained in left-anterior caudal, right-anterior caudal, right-anterior cranial, and left-anterior cranial positions. Thus, patient-specific views with annulus in-plane or en-face can be obtained.[97] Irrespective of imaging modality (eg, echocardiography, CT, fluoroscopy, MRI), relative positions of structures remain constant in specific

FIGURE 4 Fluoroscopic/Hybrid Imaging of the Tricuspid Valve



The tricuspid annulus S-curve and associated chamber views. Alongside fluoroscopy are the corresponding and identical echocardiographic chamber views.

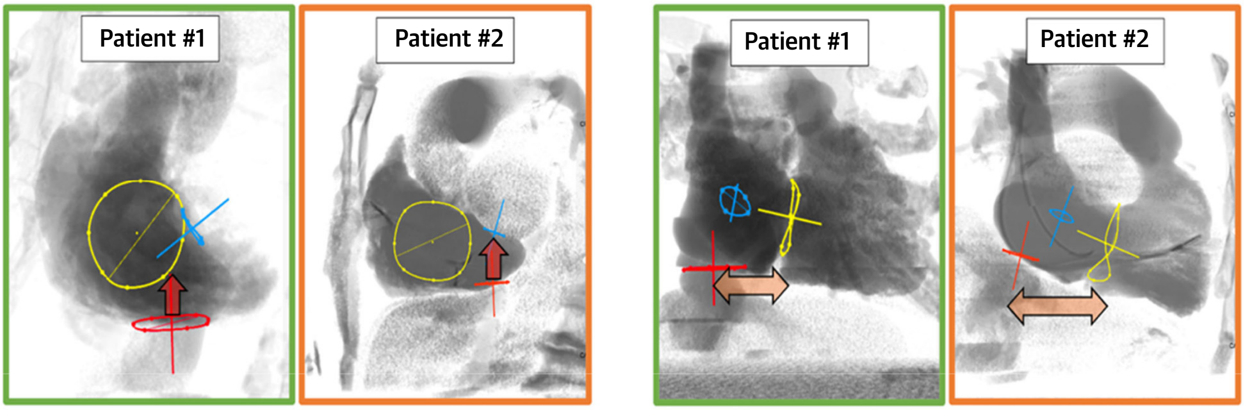
chamber views, providing foundation for "bridging knowledge" across imaging modalities. Attitudinal heart description (anterior vs posterior, superior vs inferior, right vs left) of CT and fluoroscopy can also provide true anatomical position of structures and facilitate accurate device delivery/deployment. For example, these views highlight orientation of the "posterior leaflet" as altitudinally inferior to "anterior/septal leaflets," thus allowing appropriate catheter movements during the procedure (Figure 5).

POSTPROCEDURAL EVALUATION

After surgical/transcatheter tricuspid interventions surveillance occurs at standard times or clinically triggered by new events.[88] Surveillance imaging is typically recommended before discharge, at 1 month, 6 months, and 1 year postintervention and thereafter on a yearly basis.[28,98] TTE is standard for surveillance, complemented by CT/CMR in case of abnormalities or for assessment of reverse-remodeling, generally at 6-month follow-up. Residual TR assessment follows standard guidelines, although implanted devices may challenge the assessment due to multiple/eccentric jets or shadowing.[99] Quantitative methods (proximal isovolumic velocity surface area/3D vena contracta) are recommended but not always achievable for similar reasons. Although triangular systolic continuous-wave Doppler TR signal or markedly abnormal motion/detachment of devices suggest severe residual/recurrent TR, CMR quantification may be particularly useful.[100]

Structural abnormalities such as thrombosis/endocarditis may occur as complications of orthotopic or valve-in-valve valve replacement,[101] and warrant thorough evaluation.[98] Leaflet abnormalities and differentiation of degenerative changes, calcification, and thrombus formation rely on transesophageal echocardiography or gated CT angiography.[102] Prosthesis obstruction is detected using transprosthetic gradient. After TEER, although gradient may detect rare obstructions, structural integrity assessment relies on device position and leaflet attachments.[40] Tricuspid annuloplasty, surgical or transcatheter, aims at restoring normal coaptation length of 5-6 mm,[103] but can fail not only by

FIGURE 5 Hybrid Imaging



(Left) Images for patient 1 and patient 2 demonstrate a short-axis view of the tricuspid valve annulus (yellow closed spline) along with the inferior vena cava (red closed spline) and atrial septum (blue closed spline) both in plane. The red arrow is perpendicular to the inferior vena cava and highlights the trajectory of a delivery catheter. Note that a delivery catheter would be more coaxial with the tricuspid valve annulus in patient 1 than patient 2. The atrial septum and inferior vena cava are shifted more posteriorly in patient 2. (Right) Images for patient 1 and patient 2 demonstrate a 3-chamber view of the right-sided heart with tricuspid valve annulus (yellow closed spline) and inferior vena cava (red closed spline) both in plane. This view also helps understand the trajectory (and flex requirements) of a delivery catheter from the inferior vena cava to tricuspid valve annulus. Furthermore, the orange double arrow highlights the variability in distance from inferior vena cava to tricuspid valve annulus that can be associated with changes in right atrial size.

ring dehiscence but also due to leaflet tenting.[37] Thus, various new devices in clinical trials warrant careful assessment of their structural integrity over time. After TR correction, forward stroke volume and cardiac output tend to increase.[104] Although hemodynamic changes are generally favorable, immediately after TR intervention/surgery with elevated pulmonary resistance, RV afterload may increase and be poorly tolerated.[105] Long-term reverse remodeling occurs with a wide range of responses to TR treatment, quantifiable by timed CT/CMR right-sided heart assessment.[82]

Therefore, although many uncertainties remain regarding success and complication rates after transcatheter/surgical TR correction, protocolized care is warranted to systematically analyze individualized responses and thresholds for poor outcomes, with new therapies.

CURRENT AND DEVELOPING INTERVENTIONS

The tricuspid valve is a difficult target for interventional therapies due to its peculiar anatomy and heterogeneity, explaining the large number of devices and clinical trials. Interventional repair is challenged by anatomical complexity/variability and suboptimal transesophageal imaging. As far as replacement is considered, the tricuspid valve is large, highly variable, and not calcified and the right ventricle is thin, challenging device delivery and anchoring. Proximity of the conduction system also makes therapies prone to complications. To address these challenges, numerous directions are being explored.

Tricuspid annuloplasty is the standard of care for surgical repair.[103,106] A preventive approach in patients undergoing mitral surgery, involving systematic surgical annuloplasty of enlarged annuli in practice,[107] and in clinical trials[108] has reduced progression to severe TR and improved outcome. Transcatheter annuloplasty in the TRI-REPAIR (TrIcuspid Regurgitation RePAIr With CaRdioband Transcatheter System) study reduced septolateral annular diameter, TR grade, and NYHA functional class.[109] However, implantation remains challenging and numbers of treated patients small,[110] but second-generation annuloplasty devices are tested.[111] However, because surgical annuloplasty is associated with 25%-40% TR recurrence risk,[20] with advanced RV remodeling,[37] strict selection processes are required.

Leaflet repair is rarely performed surgically, although the concept of tricuspid edge to edge was introduced by Alfieri et al[112] 20 years ago. Conversely, transcatheter leaflet therapies are the most used and currently dominated by TEER.[23,113] The TRILUMINATE trial, first randomized vs medical therapy, reported that tricuspid TEER is safe, reduces TR severity, and is associated with improvement in quality of life,[41] proportionately to TR reduction.[27] However, mortality, tricuspid-valve surgery and hospitalizations for heart failure were not reduced by interventional vs medical treatment.[27] Residual TR with or without single-leaflet attachment is non-negligible,[40,114] and may have been underestimated using TTE.[27] Whether residual TR is responsible for lacking survival/heart failure improvements or the anatomical selection criteria skewed the randomized population, survival improvement is unrealistic short-term, or heart failure severity is poorly measured by hospitalization in the specific TR context remain to be determined, emphasizing the crucial importance of continued follow-up and of new randomized trials.[27]

Incorporation of a spacer in the TEER device may present a future application.[114] Furthermore, transcatheter leaflet extensions, subvalvular manipulations, as well as new devices are being currently investigated.[115] In all these treatment considerations, pacing leads crossing the tricuspid valve are sources of difficulty, although in the TRI-VALVE registry[38,71] leads did not impact success. Lead extraction may yield significant complications so that transcatheter valve replacement is more often considered with pacemaker-leads-associated TR than complex transcatheter repair.[31,32]

Valve replacement is often preferred in patients with severely dilated right ventricle and distorted subvalvular apparatus undergoing isolated surgery.[116]

Orthotopic transcatheter tricuspid valve replacement is currently tested with various anchoring systems to tricuspid leaflets/annulus,[117] appearing to provide powerful control of residual TR.[42,68] However, many uncertainties remain regarding very large/asymmetric regurgitant orifices, atrio-ventricular blocks, canting, or multiscalloped tricuspid valves, warranting comprehensive imaging/trials. Heterotopic transcatheter tricuspid valve implantation or caval valve implantation is considered when direct TR control is not possible[90,118] to minimize TR consequences on venous circulation. Although early reports mention functional improvement, clinical outcome improvements provided by these devices remain to be defined by rigorous trials.

ENDPOINTS AND CLINICAL TRIALS

RISK SCORING IS WARRANTED. Isolated tricuspid valve surgery is considered high-risk,[119,120] but is of heterogeneous outcome, more related to clinical presentation severity than to the intervention itself.[121] Beside TR severity, advanced sign of heart failure, ascites, dose of diuretics, RV dilatation/dysfunction, systolic pulmonary pressure, RV/PA coupling, kidney insufficiency, liver stasis, and cirrhosis are major prognostic factors.[32,75,121,122] In clinical practice, staging and scoring systems should integrate TR severity, heart failure severity, and comorbidities.[18,123-125] Importantly, such TR staging/scoring will also provide a tool to compare cohorts and their respective outcomes.

There is no guideline-based risk score for isolated tricuspid surgery. Initial attempts were not well calibrated for high-risk patients.[126] Although the MELD (Model for End-Stage Liver Disease) score is linked to mortality after tricuspid surgery,[127] it is hindered by dependence on coagulation alterations impossible to verify in most patients with TR who are on anticoagulation. Therefore, the TRI-SCORE was developed to predict operative mortality risk for tricuspid surgery. It includes clinical (age ≥70 years, NYHA functional class III-IV, right-sided heart failure signs, daily dose of furosemide ≥125 mg), biomarkers (glomerular filtration rate <30 mL/min, elevated total bilirubin), and echocardiographic parameters (left ventricular ejection fraction <60%, moderate/severe RV dysfunction). The TRI-SCORE provides excellent risk assessment independently of TR mechanism and etiology.[128] It delineates patients at low-risk, best suited for surgery and those at high surgical risk and may be considered to define futility in TR treatment vs those to involve in clinical trials.[129]

SURGICAL TRIALS. Growing interest in TR and its transcatheter management has vicariously renewed interest in tricuspid surgery, with an increased number of interventions.[119,121,130] However, short- and long-term outcome have not yet measurably improved.

So far, the only randomized controlled trial of tricuspid valve surgery was conducted in context of concomitant mitral surgery with less than severe TR but with annular dilatation.[108] The trial showed that concomitant "preventive" tricuspid annuloplasty yielded less progressions to severe TR at the cost of more pacemaker implantation. This trial indirectly suggests that tricuspid surgery performed early does not entail outsized operative risk and can result in improved outcomes. It suggests also that more surgical trials are important (Supplemental Table 1 summarizes completed and ongoing trials).

Early feasibility studies (EFS) are pivotal in investigating emerging tricuspid therapies. Although their small cohort size (approximately 10-50 patients) typically limits the ability to establish clinical efficacy, EFS investigations do inform pivotal trial

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TABLE 4 Current Interventional Randomized Trials Enrolling in the United States and Europe

	Anticipated No. of Patients	Treatment Strategy	Sponsor(s)	Primary Endpoints	Anticipated Completion Date (Primary Endpoint)
CLASP II TR	825	T-TEER (PASCAL)	Edwards Lifesciences	Composite of all-cause mortality, RVAD implantation or heart transplantation, tricuspid valve intervention, heart failure hospitalizations, and quality-of-life improvement (measured using KCCQ score) at 24 mo	December 2024
TRILUMINATE Pivotal Trial	700	T-TEER (TriClip)	Abbott Vascular	Composite of number of participants with all-cause mortality or number of participants with tricuspid valve surgery, rate of heart failure hospitalizations, and assessment of quality-of-life improvement using the KCCQ at 12 mo	August 2022
TRISCEND II Pivotal Trial	820	Transcatheter valve replacement (EVOQUE)	Edwards Lifesciences	• TR grade reduction and composite endpoint including: KCCQ improvement, NYHA functional class improvement, and 6-min walk test distance improvement • Rate of MAE at 30 d • Composite endpoint including all-cause mortality, RVAD implantation or heart transplantation, tricuspid valve intervention, heart failure hospitalizations, KCCQ improvement, NYHA functional class improvement, and 6-min walk distance improvement	June 2024
TRI-FR	300	T-TEER (TriClip)	Rennes University Hospital	Milton Packer clinical composite score at 12 mo	August 2025
TRIC-I-HF	360	Transcatheter tricuspid valve repair (annuloplasty and T-TEER)	Ludwig Maximilians-University, Munich	All-cause mortality or heart failure hospitalization at 12 mo	March 2026

KCCQ = Kansas City Cardiomyopathy Questionnaire; MAE = major adverse events; RVAD = right ventricular assist device; T-TEER = tricuspid transcatheter edge-to-edge repair; other abbreviations as in Table 2.

design, and, therefore, can affect commercial applications for tricuspid therapies. EFS usually focus on surrogate endpoints, previously established as indicative of long-term prognosis in larger clinical studies. Guidance from EFS can occur with planning and execution of mechanistic studies, particularly with comprehensive imaging evaluations. Conversely, although biological (B-natriuretic peptide, creatinine/renal function) or functional (symptoms, quality-of-life, walking-distance) endpoints may be useful to measure, these are generally not the primary endpoints of EFS in patients with TR. Patients enrolled in EFS are not candidates for established "commercial" therapy, and thus may have extensive tricuspid, cardiac, or comorbid conditions that should be fully documented to ascertain applicability to the wider cohort of patients with TR. Although pulmonary hypertension is not an endpoint of TR treatment, it is a major determinant of outcome,[9] and enrollment thresholds and impact on physiological benefit of therapy should be carefully noted for future pivotal trials.[131-133]

TEACHING POINTS FROM PIVOTAL TRIALS. Because TR is profoundly undertreated by surgery,[9,10] the comparator to transcatheter therapies in pivotal trials is medical treatment, mostly centered on diuretics.[25,26] In the TRILUMINATE trial,[27] TEER superiority for the combined primary endpoint at 12 months was driven by quality-of-life improvements associated with marked TR reduction. The lack of hard endpoint (survival and heart failure hospitalization) differences between device and control group has several potential explanations. Among those, the frequency of residual TR, the inadequacy of heart failure hospitalization as a measure of success of TR treatment, and the necessity of longer follow-up to detect hard endpoints improvement are widely discussed. In the consideration for dispersion of these therapies and future/ongoing trials, operator experience, intraprocedural imager experience, development of tertiary centers with true Heart Teams, and appropriate multimodality imaging are thought to be fundamental. Table 4 summarizes currently enrolling interventional randomized trials and their respective primary endpoints.

THE WAY FORWARD FOR TR ASSESSMENT, TRIALS, THERAPIES, AND CLINICAL CARE

Due to TR heterogeneity and complexity of tricuspid valve, assessment, care, and treatment of patients with TR represents a considerable challenge. The ultimate goal is to surmount the current vast undertreatment of these patients and dire outcomes they

HIGHLIGHTS

- TR is profoundly undertreated by surgery and is often discovered late in patients presenting with right-sided heart failure. This review sheds light on the multimodality imaging of TR, the existing gaps in the published reports, and how to optimize outcomes of clinical trials.
- TR detection, quantitation, categorization, risk-scoring, intervention-monitoring, and outcomes evaluation, particularly of right-sided function, are crucial for the appropriate management of these patients and for clinical trial development and conduct, for both interventional and surgical groups.
- Protocolized care with routine standardized definition of TR causes/mechanisms and application of standardized quantitative methods to measure TR is a crucial step. Beyond the cursory assessment of cardiac remodeling and RV function, cardiac CT and MRI quantification represent crucial steps that warrant establishing rigorous sequences applicable in routine practice.

endure. It is now well understood that TR is a serious condition, associated in all contexts with poor outcomes if left untreated.

As part of the improvement processes of surgical and transcatheter technologies, it is essential to transform the entire chain of medical care. This encompasses awareness of practitioners and routine cardiologic evaluation of TR, from diagnosis to referral to tertiary heart valve centers. It is also important to enhance development of new devices, clinical trials, EFS and pivotal, to application in the "commercial" TR treatment. For this purpose, protocolized care with routine standardized definition of TR causes/mechanisms and application of standardized quantitative methods to measure TR is a crucial step. Beyond the cursory assessment of cardiac remodeling and RV function, cardiac CT and MRI quantification represent crucial steps that warrant establishing rigorous sequences applicable in routine practice. Based on these quantitative methods, new treatment, surgical or interventional, should be comprehensively evaluated by standardized approaches.

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KEY WORDS clinical trials, endpoints, multimodality imaging, structural interventions, tricuspid valve

APPENDIX For a supplemental table and videos, please see the online version of this paper.