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The Tricuspid Valve: A Review of Pathology, Imaging, and Current Treatment Options: A Scientific Statement From the American Heart Association

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ABSTRACT: Tricuspid valve disease is an often underrecognized clinical problem that is associated with significant morbidity and mortality. Unfortunately, patients will often present late in their disease course with severe right-sided heart failure, pulmonary hypertension, and life-limiting symptoms that have few durable treatment options. Traditionally, the only treatment for tricuspid valve disease has been medical therapy or surgery; however, there have been increasing interest and success with the use of transcatheter tricuspid valve therapies over the past several years to treat patients with previously limited therapeutic options. The tricuspid valve is complex anatomically, lying adjacent to important anatomic structures such as the right coronary artery and the atrioventricular node, and is the passageway for permanent pacemaker leads into the right ventricle. In addition, the mechanism of tricuspid pathology varies widely between patients, which can be due to primary, secondary, or a combination of causes, meaning that it is not possible for 1 type of device to be suitable for treatment of all cases of tricuspid valve disease. To best visualize the pathology, several modalities of advanced cardiac imaging are often required, including transthoracic echocardiography, transesophageal echocardiography, cardiac computed tomography, and cardiac magnetic resonance imaging, to best visualize the pathology. This detailed imaging provides important information for choosing the ideal transcatheter treatment options for patients with tricuspid valve disease, taking into account the need for the lifetime management of the patient. This review highlights the important background, anatomic considerations, therapeutic options, and future directions with regard to treatment of tricuspid valve disease.

Key Words: AHA Scientific Statements ■ heart failure ■ heart valve prosthesis implantation ■ hypertension, pulmonary ■ tricuspid valve ■ tricuspid valve insufficiency

The past decade has seen increasing interest in the tricuspid valve (TV), once referred to as the forgotten valve, as a result of a combination of improved imaging and therapeutic possibilities plus a clearer appreciation of the progressive nature of TV disease and its impact on clinical outcomes.[1,2] It is estimated that moderate or severe tricuspid regurgitation (TR) has an age- and sex-adjusted prevalence of 0.55% in the United States, with a higher incidence in women and with increasing age.[3] In patients ≥75 years of age, 4% of the population has at least moderate TR. Unfortunately, despite its high prevalence, TR is often underappreciated both clinically and by imaging, likely because of the complexity of imaging required and lack of commercially available treatment options.[3] In addition, patients with moderate TR or greater have increased mortality, with an observed survival of 10.2% at 15 years, with the highest mortality in patients with functional TR associated

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with left-sided heart disease. However, even in patients with isolated TR, the yearly mortality rate is 12.1%.[3] In addition to TR being associated with increased mortality, patients with severe TR often have significant comorbidities, which include pulmonary hypertension (PH), heart failure (HF), and atrial fibrillation.[1,4] Overall, the TV presents specific anatomic, pathological, and technical challenges that are distinct from those of the other cardiac valves. These unique challenges mandate unique treatment approaches, which are the subject of this American Heart Association tricuspid review article.

COMPLEX ANATOMY OF THE TV

The TV apparatus is a dynamic structure that is generally more complex and anatomically variable than the left-sided valves (Figure 1A).[6,7] The classic TV anatomy comprises 3 leaflets, with the septal leaflet being the smallest and arising medially from the interventricular septum, with multiple septal chordal attachments. The anterior is the largest and most mobile of the leaflets, and the posterior leaflet often has multiple scallops. A recent study has identified that there may be 4 functional TV leaflets in ≈39% of cases, predominantly comprising 2 posterior leaflets (Figure 1B), and a new nomenclature scheme has been used to classify valves: Type I has 3 leaflets; type II has 2 leaflets; type III has 4 leaflets (type IIIA with 2 anterior, type IIIB with 2 posterior, type IIIC with 2 septal); and type IV has >4 leaflets.[5] The TV sits anteriorly in the chest, with an annular plane that is nearly vertical and ≈45° from the sagittal plane.[6] The tricuspid annulus (TA) is nonplanar and elliptically shaped, with the posteroseptal aspect being the most ventricular (lowest) and the anteroseptal portion being the most atrial (highest). The tricuspid leaflets are supported by the fibrous TA, chordae tendinae, papillary muscles, and right atrial (RA) and right ventricular (RV) myocardium. The TV apparatus includes 2 to 3 papillary muscles. The largest is typically the anterior papillary muscle with chordae supporting the anterior and posterior leaflets, whereas the posterior papillary muscle provides chords to the posterior and septal leaflets. A variable but small septal papillary muscle may also exist. Because the septum provides more support for the TA as a result of its relation to the fibrous skeleton of the heart, dilation of the TA tends to occur in the anterolateral direction, corresponding to the RV free wall.

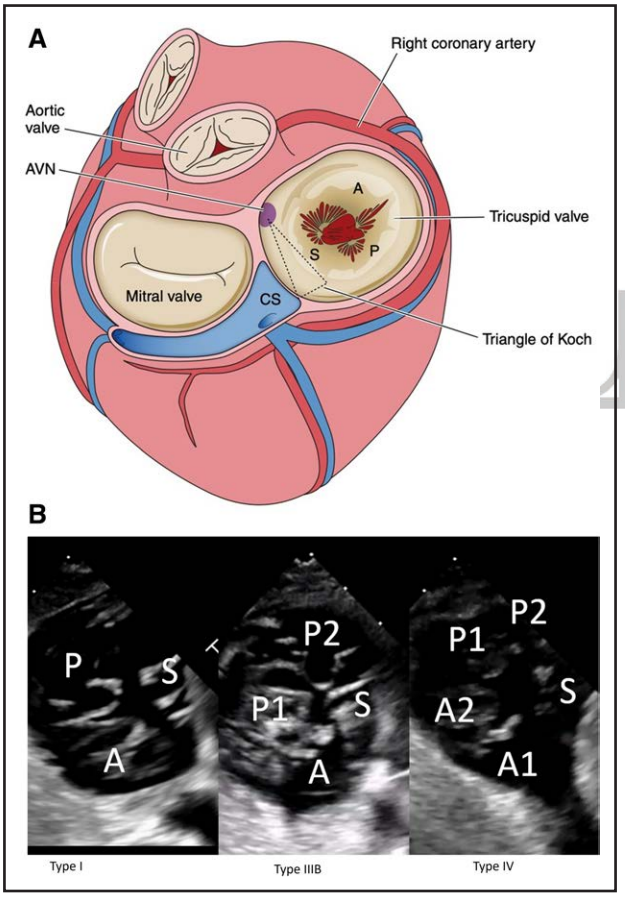

Figure 1. Anatomy of the TV.
A, The tricuspid valve (TV) is a complex anatomic structure that lies in close proximity to the right coronary artery, atrioventricular node (AVN), and coronary sinus (CS). **B**, Although the TV is classically thought to have 3 leaflets (type I), there is considerable variation between individual patients, with some having 2 leaflets (type II), 4 leaflets (type IIIA, 2 anterior [A]; type IIIB, 2 posterior [P]; type IIIC, 2 septal [S]), or > 4 leaflets (type IV).[5]

Several important structures lie in close proximity to the TV. The coronary sinus lies in the RA near the posteroseptal commissure, whereas the right coronary artery courses in the atrioventricular groove encircling the annular attachments of the anterior and posterior TV leaflets, and the aortic root (noncoronary sinus of Valsalva) lies adjacent to the anteroseptal commissure. In addition, the atrioventricular node and the right His bundle lie in the septum adjacent to the septal TV leaflet and the anteroseptal commissure. The inferior vena cava (IVC) also enters the RA in fairly close proximity to the TV but with considerable anatomic variability in location and angle of entry into the RA and therefore variability in the IVC-TA relationship.[7]

PATHOPHYSIOLOGY OF TV DYSFUNCTION

Causes of TR

There are multiple causes of TR that vary in their pathological mechanism and affect decisions about treatment (Table 1). The types of TR can be divided into primary TR and secondary TR, and there is often overlap between the categories. Primary TR is due to a structural abnormality of the valve, which can include rheumatic heart disease, valve degeneration, congenital heart disease, radiation heart disease, trauma, or damage from previous biopsies. Secondary TR,

Table 1. Causes of TR

Primary TR	Secondary (functional) ventricular TR	Secondary (functional) atrial TR
Degenerative	RV dilation due to LV dysfunction	Annular dilation due to atrial fibrillation
Pacemaker impingement/interference (lead entanglement, lead adherence, leaflet perforation, complication of lead extraction)	RV dilation due to left-sided valvular heart disease RV dilation due to PH	Atrial dilation due to HF with preserved ejection fraction
Rheumatic heart disease	RV dilation due to RV infarct	
Damage from endomyocardial biopsy	RV dilation due to chronic RV pacing	
Congenital heart disease		
Endocarditis		
Carcinoid		

HF indicates heart failure; LV, left ventricular; PH, pulmonary hypertension; RV, right ventricular; and TR, tricuspid regurgitation.

or functional TR, is due to pathology that originates from the ventricle or the atrium. Ventricular TR occurs because of disease that causes RV dilation such as PH, coronary artery disease leading to RV infarction, left ventricular (LV) dysfunction, or left-sided valvular heart disease. Atrial TR occurs as a result of atrial dilation, which causes annular dilation, maintains relatively normal leaflet structure, and often leaves the RV spared.[8–10] Atrial TR can still occur in the setting of left-sided heart disease such as in patients with HF with preserved ejection with dilated atria. Notably, atrial TR and ventricular TR may share some features, especially in the later stages of the disease, in which annular dilation leads to progressive dilation of both the atria and ventricles. TR may also result from pacemaker interference with the tricuspid apparatus, and the prevalence of significant TR after cardiac implantable electronic device (CIED) implantation has been reported to be from 7% to 45%.[11–13] CIED-associated TR is often observed in patients who also have atrial or ventricular TR because many patients with CIED have atrial fibrillation or LV dysfunction even before lead implantation.[13–15] Patients with severe TR may at first be asymptomatic but, as the disease progresses, show symptoms of central venous congestion and progressive right HF, leading to poor cardiac output and functional capacity.[16,17] As a result, over time, systemic signs of severe TR may include cardiorenal syndrome and cardiac cirrhosis. For patients with severe TR due to left-sided heart disease, shortness of breath and pulmonary congestion are likely to occur as well.[18] Performing a precise physical examination and obtaining invasive hemodynamics can aid in the assessment of patients with TR. For example, a pulsatile liver, significant pulsatile distention of the jugular vein, and tall RA V waves are indications of severe TR.

RV and LV Pathophysiology

The causes of TR exhibit marked variability among different diseases, particularly in the case of primary TR. Instances of ensuing structural damage encompass leaflet perforation or constriction, commissural fusion, and chordal entanglement or rupture. Primary TR induces a pure volume overload on the right side of the heart, often leading to the concomitant occurrence of annular dilation.

The most prevalent form of secondary TR is associated with PH. Prolonged PH induces maladaptive remodeling in the RV, affecting primarily the anterolateral wall at the midventricular level. This remodeling results in the displacement of papillary muscles, causing the anterior papillary muscle to move into a more caudal position, subsequently leading to tethering of the TV leaflets. Valvular tenting and leaflet tethering are closely related to the elongation and elliptical/spherical deformation of the RV.[19] As TR worsens, the RV dilates further, and RV systolic function progressively deteriorates, leading to an increase in RV diastolic pressure and a shift of the interventricular septum toward the LV. As a result of ventricular interdependence, this shift may compress the LV, raising LV diastolic pressure, exacerbating PH, and contributing to further maladaptive remodeling of the RV. Furthermore, the displacement of papillary muscles and tethering of the TV leaflets may increase with the shift of the interventricular septum, creating a vicious cycle of "TR-generated TR."

The development of precapillary PH is most commonly the result of long-standing left-sided congestion leading to pulmonary vascular remodeling. It is then not surprising that medical therapies treating HF, including right-sided HF, PH, and indirectly TR, are not specific to the RV but target the heart globally.[17,20] The RV plays a crucial role in both the initiation and progression of TR. Beyond anatomic considerations between the RV and TV, there are additional hemodynamic considerations.[21] The function of the RV as a pump for the pulmonary circulation means that it is susceptible to pressure and volume overload. Conditions such as PH and left-sided heart disease can elevate RV afterload, leading to RV dilatation and dysfunction.[22] This ventricular remodeling contributes to tricuspid annular dilatation, further exacerbating TR.[21] RV dilation

and dysfunction cause tricuspid annular dilatation, leading to incomplete leaflet coaptation. The increased annular size prevents the valve leaflets from coming together effectively, causing regurgitant flow during systole. The resulting volume overload further strains the RV, creating a vicious cycle that perpetuates TR. Understanding the intricate interplay between the RV and TR is crucial for clinical assessment and management. Echocardiography and other imaging techniques play a pivotal role in assessing RV size, function, and TV anatomy. Timely identification and monitoring of RV changes are essential for implementing appropriate therapeutic strategies.

Imaging of the TV

Transthoracic Echocardiography

Transthoracic echocardiography (TTE) and transesophageal echocardiography (TEE) remain the key tools for assessing TR and planning TV interventions. This is especially true because it is usually the modality used to first establish the diagnosis of severe TR, which can be easily underestimated because the valve is so large relative to the TR jet origin and this jet location is variable. Therefore, the accurate diagnosis and grading of TR require a comprehensive evaluation with careful image plane manipulation to identify the jet in it is entirety (Figure 2). At these locations, detailed views should be obtained to visualize the local anatomy of the TV; mechanism of TR; grade of TR; right-sided heart anatomy and function; CIED lead location, course, and anatomic interactions; and estimation of pulmonary arterial and central venous pressures.[17,23,24]

To fully assess the TV, multiple views with TTE are required, including parasternal RV inflow views, parasternal short-axis views, and apical 4-chamber views. Standard TTE views may need to be modified to focus specifically on the anterior-septal and posterior-septal leaflet pairs. Images can vary greatly according to probe angulation, requiring panning through the entirety of the valve to fully understand its structure because the severity of TR can be grossly underestimated if the complete large TA is not seen.[6,23] An en face short-axis view showing all of the leaflets simultaneously is possible by slight rightward and apical modification of the parasternal and subcostal mitral short-axis views. Given the potential limitations of 2-dimensional (2D) echocardiography, biplane echocardiography and 3-dimensional (3D) echocardiography with multiplane reconstruction are helpful in providing these data on TV annular anatomy and TR mechanism (Figures 2 and 3).[25] For clinical trial enrollment for transcatheter TV therapies, it is imperative that high-quality TTE imaging is available. One group that can be particularly challenging to image is patients with CIED-induced TR.[14] The use of 3D echocardiography can assist with visualization of the device leads and evaluation of the mechanism of TR.[13,15,26]

Transesophageal Echocardiography

TEE is important to define anatomy for transcatheter TV interventions, especially because it is the primary modality used for image guidance during a procedure. TEE offers the ability to obtain short-axis transgastric views of the TV, which allows visualization of each leaflet at the same time, defines the mechanism of TR, and identifies the specific site of regurgitation. The use of 3D TEE with multiplanar reconstruction (MPR) is fundamental to delineate TV morphology using the en face view and to assess all the TV leaflets and TA size from either the RA or RV perspective.[10] The transgastric view is helpful for evaluating leaflet morphology and visualizing the subvalvular anatomy and can assist with imaging the coaptation zone and with procedural evaluation for transcatheter TV therapies.[27] The mid and deep esophageal views assist with evaluating the leaflets and identifying leaflet capture during transcatheter edge-to-edge repair (TEER) procedures. The 3D and biplane images may further aid in confirming leaflet capture and device orientation and positioning. However, TEE windows can be inadequate because of the distance between the TV and probe,

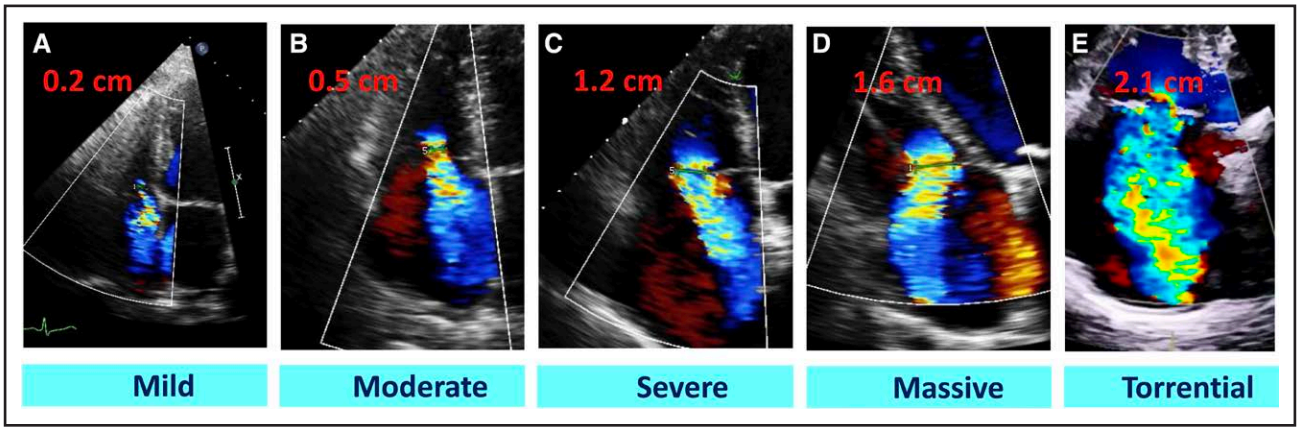

Figure 2. TR severity with TTE with expanded grading scheme.[5]
Example of patients in the (**A**) mild (vena contracta [VC]=0.2 cm), (**B**) moderate (VC=0.5 cm), (**C**) severe (VC=1.2 cm), (**D**) massive (VC=1.6 cm), and (**E**) torrential (VC=2.1 cm) groups with color Doppler in the apical 4-chamber view. TR indicates tricuspid regurgitation; and TTE, transthoracic echocardiography.

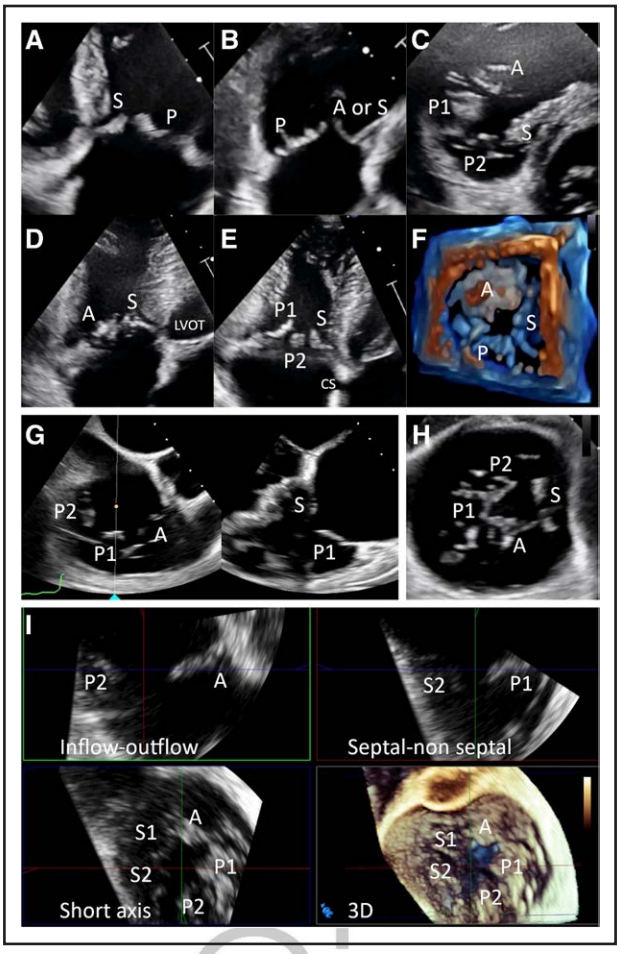

Figure 3. Tricuspid anatomy evaluation with TTE, TEE, and multiplanar reconstruction.
A, Transthoracic echocardiography (TTE), right ventricular inflow with septum (S) in view. **B**, TTE, short-axis aortic valve level. **C**, TTE, short-axis en face view. **D**, TTE, apical 4-chamber view with anterior (A) focus, as demonstrated by the left ventricular outflow tract (LVOT) in view A-S. **E**, TTE, apical 4-chamber view with posterior (P) focus, as demonstrated by coronary sinus in view. P1 with tip of P2 in view (quadricuspid morphology). **F**, Three-dimensional TTE. **G**, Transesophageal echocardiography (TEE) biplane inflow-outflow across P1 showing P1-S in secondary view. **H**, TEE, transgastric en face view of quadricuspid tricuspid valve (TV). **I**, Multiplanar reconstruction of the TV (type IV).

variable TV anatomy, and shadowing from the presence of devices. Because TEE is fundamental for leaflet device procedures, an unsuitable TEE window might be a limitation for some transcatheter therapies, although intracardiac echocardiography (ICE) can be useful when TEE windows are limited.[6]

Grading TR Severity

Grading the severity of TR should be undertaken with multiple qualitative, semiquantitative, and quantitative spectral Doppler, color Doppler, and 3D imaging data (Figure 2). Current guidelines advocate for a multiparametric approach to improve the diagnostic accuracy of severe TR.[23] The American Society of Echocardiography guidelines classify TR as mild, moderate, or severe on the basis of echocardiographic assessment of the vena contracta, effective regurgitant orifice area, regurgitant volume, and other parameters. In 2017, a new grading system was proposed and expanded to a 5-grade scale (mild, moderate, severe, massive, and torrential), which allows a more specific classification of TR that is severe or greater (Table 2).[28] The presence of a dense, early-peaking, triangular envelope by continuous-wave spectral Doppler due to rapid equalization between the RV and RA is indicative of severe TR. The presence of flow reversal in the hepatic vein is indicative of moderate or severe TR.[23] Using adjunctive imaging modalities such as cardiac magnetic resonance imaging or multislice computed tomography (CT) is recommended when echocardiographic quality is poor or the severity parameters are discordant.[23,29] It is notable that TR is dependent on loading conditions and ideally imaging is obtained when patients are euvolemic for the most accurate assessment of severity. In some cases, right-sided heart catheterization may be required to best understand the current hemodynamics.[30]

RV Function and Echocardiography

According to the guidelines, a comprehensive evaluation of the RV should be performed and may require the use of multimodality imaging.[17,23] RV function can be assessed by TA plane systolic excursion, tissue-Doppler systolic velocity S', fractional area change, RV free-wall longitudinal strain measured with speckle tracking, and 3D RV ejection fraction.[31] The degree of RV dysfunction has prognostic implications for patients with TR.[32] The key predictors of adverse post-TV intervention were found to be the RV itself and elevated pulmonary pressures.

Evaluation of only RV systolic function without incorporating the loading conditions may overestimate RV systolic function in patients with severe TR. Evaluation of

Table 2. Grading the Severity of TR by Echocardiography

	VC* width, cm	EROA by PISA, cm²	3D VCA or quantitative EROA, cm²
Mild	<0.3	<0.2	
Moderate	0.3–6.9	0.2–0.39	
Severe	0.7–1.3	0.4–0.59	75–94
Massive	1.4–2	0.6–0.79	95–114
Torrential	≥2.1	≥0.8	≥115

EROA indicates effective regurgitant orifice area; PISA, proximal isovelocity surface area; 3D, 3-dimensional; TR, tricuspid regurgitation; VC, vena contracta; and VCA, vena contracta area.[10,28]

*It is recommended that VC be measured as the average of 2 orthogonal views. For more complex jets, 3D VCA may be useful.[29]

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RV–pulmonary arterial coupling helps to assess whether the RV function has compensated for specific loading conditions, and it has been shown that a high baseline TA plane systolic excursion/systolic pulmonary artery pressure ratio is independently associated with lower all-cause mortality.[33] It must be noted that PA pressures may be underestimated in the setting of greater TR severity, worse RV function, and the presence of a V-wave cutoff sign on spectral Doppler seen with rapid equalization of the pressures.[34]

Computed Tomography

Cardiac CT plays an important role in the evaluation and management of patients with TV disease because of high spatial resolution and the ability to perform advanced analysis with specialized software packages. Contrast-enhanced, cardiac-gated CT imaging sequences allow accurate and repeatable measurements of the TA and its relationship to surrounding cardiac structures such as the right coronary artery.[35,36] Other RV structures, including the complex subvalvular apparatus, moderator band, and trabeculation, are well visualized and are relevant when screening for and planning transcatheter TV replacement due to the expected interaction between the device, annulus, and potentially subvalvular RV anatomy. In many cases, the TV leaflets are also well visualized. If images are taken at multiple points throughout the cardiac cycle, evaluation of leaflet anatomy and mobility can be performed.

In addition, CT can be used to assist in evaluating the severity of TR (Supplemental Figure 1). Quantification of TR is feasible with LV and RV stroke volume differences in the absence of intracardiac shunts or other valvular regurgitation. The anatomic regurgitant orifice area can also be measured on CT, which correlates with the vena contracta seen on 3D TEE but tends to measure higher than the effective regurgitant orifice area.[37]

Last, cardiac CT allows detailed evaluation of the venous system. This helps ensure that the diameter and tortuosity of the femoral veins, iliac veins, and IVC will not prevent successful use of large-bore delivery catheters common to many transcatheter valves. The variable relationship of the IVC entry point in the RA to the TA can also be evaluated to help predict the likelihood of successful transcatheter system manipulation to achieve proper position and trajectory for device delivery.[37]

Cardiac Magnetic Resonance Imaging

Cardiac magnetic resonance imaging is a valuable imaging modality in the evaluation of TV disease and can be particularly helpful when echocardiographic images are suspected to underestimate the degree of TR or RV dysfunction.[38] The spatial resolution of cardiac magnetic resonance imaging allows RV contouring to evaluate RV volumes, systolic function, and quantification of TR when combined with phase-contrast flow evaluation of the pulmonary artery or proximal aorta.[39] A baseline assessment of RV systolic function is particularly relevant for transcatheter TV replacement because the expected elimination of TR and potential direct interaction with the RV may increase the risk of acute afterload mismatch and RV failure, as sometimes seen after TV surgery.[40]

Intracardiac Echocardiography

ICE has been instrumental in facilitating transcatheter TV repair in situations in which TEE imaging is suboptimal in visualizing the critical structures. The ICE catheter is positioned in the RA from the femoral vein to visualize the target leaflets for grasping during TEER, TA for percutaneous annuloplasty, or necessary valve structures for transcatheter replacement. If 3D ICE is to be used, it is imperative to obtain excellent 2D imaging first, before switching to the 3D MPR mode. Compared with 2D TEE, 2D ICE has a higher imaging frequency and frame rate acquisition, but this decreases with the addition of color Doppler.[41] With the addition of 3D MPR, temporal resolution decreases further, with the lowest resolution for 3D ICE. In a study of 15 patients undergoing TEER procedures (10 with 2D ICE and 5 with 3D MPR ICE), 2D ICE was essential in 7 of 10 of the 2D ICE procedures, and 3D ICE was essential in 3 of 5 of the 3D MPR cases.[41,42] Early experience demonstrated feasibility of using ICE as the sole imaging modality or complement to TEE in performing transcatheter TV repair.[42–45] Four-dimensional ICE has been used while performing tricuspid annuloplasty and improves visualization compared with TEE.[46] Some potential disadvantages of ICE use are added cost and overcoming initial operator learning curves.[41]

Treatment Options

Medical Therapy

Medical management is the mainstay for patients with symptomatic TV disease to treat symptoms of fluid overload and slow the progression of RV failure. In patients with primary TR and symptomatic right-sided HF, medical treatment should focus on treating the primary cause of HF and cardiovascular congestion.[47] This may include diuretics (Class 2a). In the setting of secondary TR, the treatment of TR relies on the treatment of the underlying cardiomyopathy and ensuing PH or resultant structural complications that might further worsen TR such as atrial fibrillation (Class 2a).[17] The cornerstone of HF management is the use of guideline-directed medical therapy (GDMT) for LV dysfunction.[20] GDMT for with reduced ejection fraction includes renin-angiotensin-aldosterone system blockers/angiotensin neprilysin inhibitors, β-blockers, mineralocorticoid receptor antagonists, and sodium-glucose cotransporter-2 inhibitors. Class I drugs for HF with reduced ejection fraction have been shown to lower filling pressures and induce structural remodeling, through which they can reduce secondary

TR and MR. Medical therapies in the setting of HF with preserved ejection fraction are limited to diuretics (Class 1), sodium-glucose cotransporter-2 inhibitors (Class 2a), angiotensin neprilysin inhibitors, mineralocorticoid receptor antagonists, and angiotensin receptor blockers (Class 2b).[20] Invasive hemodynamic measurement with a pulmonary artery catheter assists with the management of patients with severe TR and may guide therapies directed at treatment of PH.[48] The use of vasodilators for the treatment of pulmonary arterial hypertension and for rhythm control in patients with atrial arrhythmias is also recommended.[17] Atrial rhythm management, specifically referring to the management of atrial fibrillation or atrial flutter, can have an impact on TR, but it is important to note that the relationship between the two is complex, and the effect of rhythm control on TR depends on various factors. The effect of atrial rhythm management on TR can vary widely among individuals. Factors such as the underlying cause of TR, the degree of atrial dilation, the severity of atrial fibrillation or atrial flutter, and the presence of other heart conditions can influence the outcome.[50] Although there are some data to support the potential benefit of rhythm control for TR in certain patients, the evidence is not consistent. Studies have shown that some patients experience a reduction in TR after successful rhythm control, whereas others may not see significant improvement.[51] Notably, no medical (nonprocedural) therapy can directly reverse either primary or secondary TR but rather can improve TR through change in volume status and atrial/ventricular remodeling.

It is important to recognize that medical therapy after TV intervention is also imperative to achieve excellent outcomes. Many patients will continue to require maintenance diuretic therapy after their procedure, but doses may decrease or stabilize over time and need to be adjusted according to new loading conditions after the intervention.[52,53] Furthermore, patients may require inotropic support immediately after TV surgery to support the RV.[40]

Surgery

Indications for TV surgery have been well defined in current American College of Cardiology/American Heart Association guidelines.[17] In those guidelines, the only Class 1 recommendation for TV surgery is at the time of left-sided valve surgery. Otherwise, guideline recommendations for isolated TV surgery are for patients with signs and symptoms of right-sided HF (Class 2a) or for asymptomatic patients with primary TR and progressive RV dysfunction (Class 2b).[17] As a result, many patients are treated later in the natural history of the disease.[54] There is a paucity of evidence for early intervention for severe TR and the optimal timing of intervention, but the guidelines suggest that treatment of primary TR ideally occurs before signs of end-organ damage and significant RV dysfunction arise.[17]

TV surgery is most commonly performed concomitantly with mitral valve surgery, and isolated TV surgery accounts for only 20% of TV surgeries, with ≈5000 performed in a 10-year period in the United States.[55–57] The 2 major options are replacement and repair (Table 3). TV surgery is performed on cardiopulmonary bypass and can be performed by either sternotomy or minimally invasive approaches. TV replacement is typically reserved for patients in whom the valve is not repairable such as those with congenital heart disease, carcinoid heart disease, endocarditis, or extensively damaged or restricted leaflets. Both mechanical and bioprosthetic valves can be used with variable durability, but durability can approach 15 years for bioprostheses.[58] Studies comparing mechanical valves and bioprosthetic valves have shown that valve failure, mortality, and reoperation rates are similar at 5 years, but mechanical valves may show earlier adverse events.[58,59]

TV repair has evolved over the past few decades, and ring annuloplasty is now the gold standard. Early techniques incorporated a running suture annuloplasty technique rather than implantation (DeVega), but concerns about long-term durability have largely resulted in surgeons abandoning this approach. An undersized annuloplasty device alone can be largely successful in cases in which TR is secondary to a dilated annulus or in cases in which CIED leads do not affect leaflet mobility.[60] Additional repair may be required on 1 or more leaflets of the TV if there is significant leaflet pathology related to either endocarditis or myxomatous degeneration.

Although TV surgery has become reproducible with standardized techniques, overall in-hospital mortality rates remain at 8% to 10%, mostly because patients with severe TR have concomitant comorbidities (RV, renal, and liver dysfunction) that increase perioperative risk. National mortality is 1.7% in patients without these risk factors.[57,61–64] Isolated reports from single centers have been better and may indicate a volume-dependent relationship that is seen in all cardiac surgical procedures.[65] It is notable that, compared with patients who are treated with transcatheter tricuspid therapies, patients treated with TV surgery are significantly younger.[66] Long-term survival and durability data remain rather limited, likely because of limited follow-up in this cohort. A team-based approach to the preoperative and perioperative patient selection and management of patients in conjunction with HF physicians is imperative.

CIED-Associated TR

Current options for the treatment of CIED-induced TR include medical therapy to relieve congestion, lead extraction with replacement or use of different pacing strategies, or surgical TV repair.[67] The use of transcatheter TV repair in patients with CIED-induced TR has been shown to be feasible with similar rates of procedural success, residual TR, symptomatic improvement, and mortality compared with patients with non–CIED-induced TR.[68]

Table 3. Indications, Techniques, Devices, and Potential Complications in TV Surgery

	Repair	Replacement
Indications[17,54]	Primary pathology Myxomatous degeneration Leaflet injury amenable to repair Endocarditis with limited leaflet damage Congenital heart disease amenable to repair (eg, Ebstein anomaly) Secondary pathology Limited annular dilatation, limited tethering and flattening, amenable to optimal and durable repair Leaflet restriction due to CIED lead with adequate leaflet tissue and mobility	Primary pathology Congenital heart disease not amenable to repair Endocarditis with extensive leaflet damage Carcinoid disease Secondary pathology Excessive annular dilatation precluding optimal and durable repair Leaflet restriction/damage due to CIED lead with severe tethering precluding repair
Techniques/devices used	Device-based annuloplasty: Incomplete ring MC3 Tri-Ad Physio Tricuspid Complete ring (largely abandoned) Carpentier Classic Suture-based annuloplasty (largely abandoned): Bicuspidization DeVega annuloplasty Edge to edge	Mechanical valve Bioprosthetic valve Porcine Bovine pericardial
Potential complications	Heart block due to atrioventricular node injury requiring pacemaker Suture-mediated right coronary artery kinking or occlusion Aortic valve injury	

CIED indicates cardiac implantable electronic device; and TV, tricuspid valve.

Transcatheter TV Interventions

A number of transcatheter TV therapies are under development and in clinical trials, including transcatheter TV repair and replacement devices (Figure 4). An area of interest is identifying clinical and anatomic factors that may predict both procedure success and survival in patients undergoing these procedures. In the selection of patients, it is important to recognize the dynamic nature of TR and the changes in RV dimensions, coaptation gaps, and leaflet tethering that can occur, depending on loading conditions for individual patients on the day of imaging. For this reason, if patients are volume overloaded at the time of their initial assessment, imaging may need to be repeated when they are medically optimized. In some cases, patients have been admitted to the hospital before procedures for "prehab" to optimize volume status with intravenous diuresis.[30] It is also imperative to understand whether liver dysfunction exists as a result of either TR or other intrinsic liver disease that may be reversible because this may affect long-term survival and have significant morbidity.[69] In some cases, a hepatology consultation, including duplex of the liver and possible liver biopsy, may be required.

Transcatheter Repair

Transcatheter Edge-to-Edge Repair

TEER is currently the most common transcatheter TV repair procedure being performed, given that 2 devices (TriClip G4, Abbott Structural Heart; PASCAL Precision, Edwards Lifesciences LLC, Irvine, CA) have received the CE mark for clinical use in Europe. Before the development of dedicated tricuspid TEER devices, the MitraClip system (Abbott Structural Heart) had been used in an off-label manner worldwide to perform tricuspid TEER, with promising 1-year outcomes in the international TriValve registry, provided that the coaptation gap is feasible for leaflet grasping.[70] The TriClip G4 system, with 4 different clip sizes to customize repair, is similar to the MitraClip G4 system, but the steerable guide catheter has 2 knobs for steering to improve device trajectory for more efficient leaflet grasping[71] (Supplemental Figure 2).

The TRILUMINATE (TRILUMINATE Study With Abbott Transcatheter Clip Repair System in Patients With Moderate or Greater TR) global early feasibility study (EFS) reported sustained TR reduction at 1 year after TEER with the TriClip system, with TR remaining at moderate or less in 71% of 85 patients and evidence of reversed RA and RV remodeling.[72] The TRILUMINATE

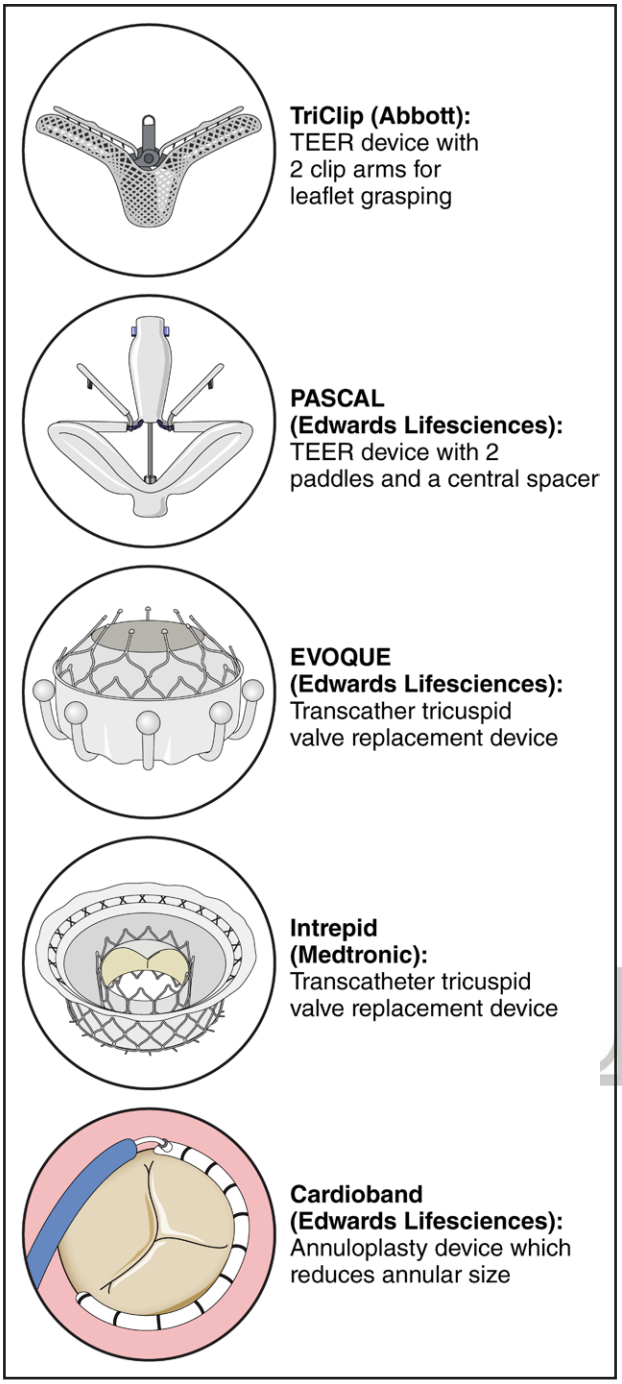

Figure 4. Images of various transcatheter TV devices.
Transcatheter edge to edge, transcatheter annuloplasty, and transcatheter valve replacement devices, among other devices, are currently under evaluation. TEER indicates transcatheter edge-to-edge repair; and TV, tricuspid valve.

Pivotal trial (Trial to Evaluate Cardiovascular Outcomes in Patients Treated With the Tricuspid Valve Repair System Pivotal) is currently the only published randomized clinical trial of transcatheter TV therapies. However, several more trials are underway that will help to further elucidate the effects of transcatheter TV therapies on clinical outcomes.[73] TRILUMINATE randomized patients of intermediate or greater surgical risk to optimal GDMT or TriClip TEER. The primary end point (hierarchical composite of death from any cause or TV surgery, hospitalization for HF, and improvement in quality of life) was in favor of the group who received the device compared with the GDMT group (win ratio, 1.48 [95% CI, 1.06–2.13]; P=0.02); however, the results showed no significant differences in 1-year mortality or HF hospitalizations (annualized rate for HF hospitalizations, 0.21 events per patient year in the device group versus 0.17 events per patient year in the GDMT group). It also demonstrated improved Kansas City Cardiomyopathy Questionnaire-12 scores in the device compared with the GDMT group (12- versus 0.6-point increase, respectively; P<0.001), with greater quality of life improvement when residual TR was moderate or better or if there was >1 grade TR reduction.[73] Eighty-seven percent of patients in the TEER group had no greater than moderate TR at the 30-day follow-up (Supplemental Figure 3). Further subgroup analyses are currently underway to identify those who may benefit the most from tricuspid TEER. The TriClip device recently received FDA approval for the treatment of severe, symptomatic TR to reduce the severity of TR and improve quality of life.

The PASCAL device and delivery system, given its flexibility, can be used for both mitral and tricuspid TEER, thus enabling combined procedures. Unlike the TriClip system, PASCAL has a central spacer in which the arms clasp the leaflets to reduce TR.[74] With the PASCAL system, early experience showed sustained 1-year TR reduction to moderate or less in 86% of 30 patients.[75] The US EFS of the PASCAL system (CLASP TR [Edwards PASCAL Transcatheter Valve Repair System Pivotal Clinical Trial]) with 34 patients showed that 85% had achieved at least 1 grade TR reduction but only 52% had moderate or less TR at 30 days.[76] The CLASP TR pivotal trial randomizing patients between optimal GDMT and TEER with the PASCAL system is ongoing and has a 2-year end point.

Annuloplasty

Transcatheter TV annuloplasty represents a promising, albeit early-stage, minimally invasive technology that emulates its surgical predecessor and could greatly expand treatment options for patients with severe TR. Potential advantages of annuloplasty over other TV transcatheter interventions include preservation of the leaflet anatomy and its role as potentially adjunctive with other interventions (ie, annuloplasty+leaflet intervention).

The Cardioband system (Edwards Lifesciences), analogous to the surgical ring, is the first CE mark–approved (2018) transcatheter therapy for TR. Anchors are implanted in the annulus through the femoral vein, after which the implant is cinched to achieve the desired reductions in annular size and TR. In a multicenter EFS of 37 patients, procedural success was 83%, and at the 1-year follow-up, there was a 2-grade or greater TR

reduction in 73% of patients, with 73% of patients having moderate or less residual TR. In addition, there was a doubling of patients in New York Heart Association class I/II from baseline to 1 year and a clinically significant improvement in Kansas City Cardiomyopathy Questionnaire score.[77] Four-dimensional ICE has been used to enhance imaging in some transcatheter annuloplasty cases, particularly in imaging the lateral annulus, which is visualized better with ICE compared with TEE.[46]

Tricuspid Valve-in-Valve and Valve-in-Ring

In patients with prior surgical TV annuloplasty or replacement, transcatheter treatment to address recurrent TR or prosthetic valve degeneration has become an attractive off-label alternative to repeat surgery, given the multiple comorbidities in this patient population. Although the use of a balloon-expandable transcatheter aortic valve device for TV-in-valve replacement is a straightforward procedure with favorable midterm outcomes, TV-in-ring procedures are technically more challenging with a higher incidence of paravalvular leak and residual TR.[78–82] The presence of RV leads presents additional technical challenges in TV-in-valve or valve-in-ring procedures, with potential risk of lead damage or dislodgement. Devices that are designed for native tricuspid anatomies may be more suitable for transcatheter valve-in-ring replacement than a transcatheter aortic valve device, but more data are needed.

Transcatheter Replacement

Orthotopic transcatheter TV replacement refers to implantation of a bioprosthetic valve within the native TA. Several orthotopic transcatheter TV replacement devices are in development, have completed an EFS, or have completed enrollment for a randomized trial and are now FDA approved.[83]

The EVOQUE system (Edwards Lifesciences) is a self-expanding bovine pericardial valve mounted on a nitinol frame with a fabric skirt and 9 RV anchors designed to stabilize the valve within the annulus and subvalvular apparatus. The multicenter TRISCEND EFS (Edwards EVOQUE Tricuspid Valve Replacement: Investigation of Safety and Clinical Efficacy After Replacement of Tricuspid Valve With Transcatheter Device) reported the outcome of 56 consecutive high-surgical-risk patients with severe symptomatic TR. Technical and procedural success was achieved in 98.2% and 96.4% of patients.[84] Among patients with successful valve deployment, 98.1% had mild or less TR at the 30-day follow-up, and 11% required permanent pacemaker implantation; all-cause 30-day mortality rate was 3.6% (n=2). There were significant improvements between baseline and 30-day New York Heart Association functional class, 6-minute walk distance, and Kansas City Cardiomyopathy Questionnaire scores. Based on the 6-month results of the TRISCEND II randomized trial, the EVOQUE system recently became FDA-approved for the treatment of anatomically suitable patients with symptomatic severe TR, despite receiving optimal medical therapy. It also recently received CE mark approval.

The INTREPID system (Medtronic Plc, Minneapolis, MN) is a bovine pericardial valve mounted within a nitinol dual stent frame and is unique in that it does not require leaflet capture for anchoring. Although originally used in the mitral position, successful tricuspid deployment in 3 patients was reported in 2020, and an EFS is underway in the United States.[85–87] Several other orthotopic transcatheter TV replacement systems are under development and investigation, which include but are not limited to LuX-Valve Plus (Ningbo Jenscare Biotechnology Co, Ningbo, China), Cardiovalve (Cardiovalve Ltd, Or Yehuda, Israel), TriSol (TriSol Medical Ltd, Inc, Yokneam, Israel), and Topaz (TRiCares, Paris, France, and Munich, Germany).[88–91]

Heterotopic TV replacement describes the placement of a prosthetic valve outside the tricuspid annular position to mitigate the symptoms associated with RV failure such as peripheral edema and hepatorenal congestion. The TricValve system (P+F Products+Features, Vienna, Austria) is a transfemoral system consisting of 2 independent self-expanding bovine pericardial tissue valves designed uniquely for deployment in the IVC and superior vena cava. The TRICENTO system (New Valve Technology, Hechingen, Germany) is a custom-made bicaval covered stent that extends from the IVC through the RA into the superior vena cava and directs venous return through a bicuspid pericardial valve oriented toward the native TA. Early experience in 21 high-risk patients with severe TR demonstrated 100% procedural success, no in-hospital mortality, and an improvement in New York Heart Association functional status in a majority of patients at a median follow-up of 61 days.[92]

TRIVALVE REGISTRY

The transcatheter tricuspid landscape has been rapidly evolving, and the TriValve registry is the first and only multicenter global study to report characteristics and outcomes of patients undergoing transcatheter TV interventions.[93,94] Since its initial report in 2017, several substudies have been published, including patients with TEER, individuals with transcatheter intervention versus medical management, patients with RV leads, individuals with combined mitral and tricuspid compared with isolated mitral interventions, patients with massive and torrential TR, patients with RV dysfunction and PH, and patients with prior left-sided valve surgery, as well as studies comparing sex differences.[33,68,70,93–100] These findings have been valuable in guiding clinical trial designs and the development of Tricuspid Valve Academic Research Consortium definitions.

Table 4. Transcatheter TV Therapies*

	TEER	Annuloplasty	TTVR
Benefits	Safe Sustained TR reduction in select anatomies Pacemaker rare Feasible with mitral TEER devices	Mimics surgical technique that has been shown to be effective Preserves other transcatheter options (eg, TEER, TTVR)	Eliminates TR in most cases Procedure reproducible
Limitations	TR rarely eliminated Large gaps unlikely effective Feasibility/difficulty highly imaging dependent Procedural time depends on multiple factors	Feasibility/difficulty highly imaging dependent Long procedural time	Needs CT screening Pacemaker risk Requires anticoagulation Risk of device thrombosis

CT indicates computed tomography; TEER, transcatheter edge-to-edge repair, TR, tricuspid regurgitation; TTVR, transcatheter tricuspid valve replacement; and TV, tricuspid valve.

*Note the importance of accounting for individual patient preferences and shared decision-making when evaluating patients for transcatheter TV therapies.

THERAPY CHOICES AND LIFETIME MANAGEMENT

At present, there are no guidelines to determine whether an individual patient may be better suited for transcatheter TV repair or replacement, and although the understanding of the management of these patients is rapidly growing, the field is still developing (Table 4). However, ongoing clinical trials are promising to determine the best way to approach individual patients. For patients with large annuli due to RV failure, atrial fibrillation, or a combination, tricuspid TEER may be difficult, if not impossible, because of large coaptation gaps. These patients may be better suited for transcatheter TV replacement or transcatheter annuloplasty. In addition, TEER and annuloplasty require precise imaging; therefore, when patients' images are suboptimal, they may not be feasible options. Another consideration is the severity of underlying PH; there are concerns about complete and sudden elimination of TR in patients who have severe PH leading to acute RV failure and hemodynamic instability. Last, the presence of CIED leads may inform choices about transcatheter TV therapy for each individual. It must be understood how the location of the leads may or may not interfere with device placement and what pacemaker options will be available to the patient in the future once a transcatheter TV device is in place.

It is also important to consider the lifetime management of patients with TV disease. When a young patient presents with severe TR, they will need a durable device and ideally the potential option of a second transcatheter device in the future should the first device deteriorate over time. For example, using an annuloplasty device first may leave open options for transcatheter TV replacement in the future, whereas a TEER device may not allow another device to be placed if it becomes necessary. For an elderly, frail patient, alleviating symptoms and improving their present quality of life may be the primary goal; therefore, long-term lifetime management may not be as applicable. As clinical studies continue, a greater understanding of device durability will allow clinicians to better advise patients about the long-term management of TV disease.

UNKNOWNS AND FUTURE DIRECTIONS

TV disease is an underappreciated clinical entity that is strongly associated with morbidity and mortality and historically has limited treatment options. As the world of transcatheter TV therapies has expanded, a greater appreciation of the importance of excellent imaging and better understanding of how to quantify TR have arisen. In the current era, it is clear that the optimal transcatheter tricuspid device is heavily based on individual patients' clinical and anatomic characteristics. It is imperative to consider the lifetime management of patients with TR, taking into account that some patients may require >1 intervention in their lifetime. Currently, there is a lack of information on the durability of the available devices, and as clinical trials progress, a greater understanding will be obtained as to how to select devices that are safe, durable, and most beneficial for individual patients.

ARTICLE INFORMATION

The American Heart Association makes every effort to avoid any actual or potential conflicts of interest that may arise as a result of an outside relationship or a personal, professional, or business interest of a member of the writing panel. Specifically, all members of the writing group are required to complete and submit a Disclosure Questionnaire showing all such relationships that might be perceived as real or potential conflicts of interest.

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This table represents the relationships of writing group members that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all members of the writing group are required to complete and submit. A relationship is considered to be "significant" if (a) the person receives $5000 or more during any 12-month period, or 5% or more of the person's gross income; or (b) the person owns 5% or more of the voting stock or share of the entity, or owns $5000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.

*Modest.

†Significant.

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*Modest.

†Significant.

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