International Journal of Multiphysics
Volume 18, No. 3, 2024
ISSN: 1750-9548

An Overview on Tricuspid Valve and Tricuspid Regurgitation

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Abstract:

The tricuspid valve is one of the four cardiac valves, located between the right atrium and right ventricle. It is composed of three leaflets (anterior, posterior, and septal), chordae tendineae, papillary muscles, and the annulus. Its primary role is to maintain unidirectional blood flow during right ventricular contraction by preventing backflow into the right atrium. Tricuspid regurgitation (TR) is the most common abnormality of this valve, characterized by the backward flow of blood into the right atrium during systole. TR can be functional (secondary to annular dilatation or right ventricular remodeling, often due to left-sided heart disease or pulmonary hypertension) or organic (primary, caused by intrinsic valve pathology such as rheumatic disease, endocarditis, trauma, or congenital malformations).

Keywords: Tricuspid valve, Tricuspid regurgitation (TR), Right heart failure, Annular dilatation, Pulmonary hypertension, Functional TR, Organic TR, Valvular heart disease.

Introduction:

Tricuspid regurgitation (TR) is increasingly recognized as a common valvular disorder, with moderate-to-severe forms affecting up to 10–23% of patients with chronic heart failure, highlighting its significant clinical relevance and the importance of early evaluation **(1).**

Once considered the "forgotten valve," TR is now understood to persist even after correction of left-sided valve disease and is strongly associated with right ventricular dysfunction and increased mortality. Recent advances in imaging and interventional techniques have shifted clinical practice toward earlier recognition and treatment of significant TR **(2).**

Anatomy and Function of the Tricuspid Valve

The TV is the largest and most apically positioned of the 4 cardiac valves (Figure 1) with a normal orifice area between 7 and 9 cm^2 . Because of its large size and the low pressure differences between the right atrium (RA) and right ventricle (RV), peak transtricuspid diastolic velocities are typically lower than 1 m/s with mean gradients of <2 mm Hg **(3)**

Similar to the mitral valve, the TV can be divided into 4 components: the leaflets, the papillary muscles, the chordal attachments, and the annulus (with attached atrium and ventricle) **(4)**

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Tricuspid valve leaflets

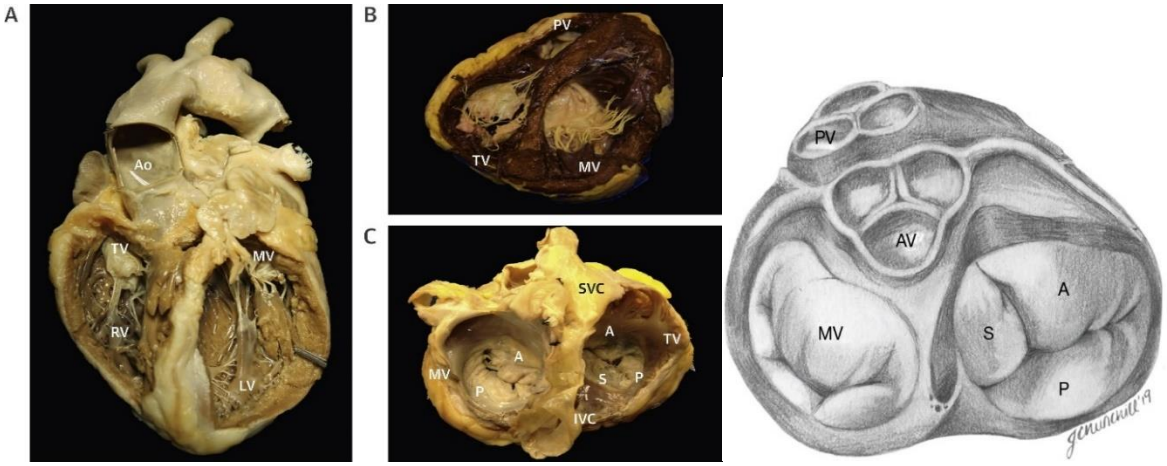


Figure 1 Gross Anatomy of the TV(5)

Although the TV is typically composed of 3 leaflets of unequal size, in many cases, 2 (bicuspid) or more than 3 leaflets may be present as anatomic variants in healthy subjects. **(6).** (figure 2).

A recent study has identified that there may be 4 functional TV leaflets in ≈39% of cases, predominantly comprising 2 posterior leaflets , 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.**(3)**

When described relative to their anatomic position in the body, the 3 leaflets would be the septal, anterior-superior, and inferior leaflets. Typically, however, these leaflets are referred to as the septal, anterior, and posterior leaflets, respectively. **(4)**

The **anterior leaflet** is generally the largest and the longest in the radial direction, with the larger area and the greatest motion. The **posterior leaflet** may have multiple scallops and is the shortest circumferentially.

The **septal** leaflet is the shortest in the radial direction and the least mobile. It is attached to the tricuspid annulus directly above the interventricular septum , it is inserted into the septum ≤10 mm apically to the septal insertion of the anterior mitral leaflet (i.e., apically displaced) **(3) .**

Anatomic landmarks for each leaflet vary significantly, the commissure between the septal and posterior leaflets is usually located near the entrance of the coronary sinus. The noncoronary sinus of Valsalva of the aortic root is adjacent to the commissure between the septal and anterior leaflets. **(7)**

Coaptation of the **TV** normally is located at the level of the **annulus** or just below it, with a coaptation length of 5 to 10 mm. This excess coaptation length functions as the **coaptation reserve**, allowing some dilation of the annulus before mal-coaptation occurs. **(7)**

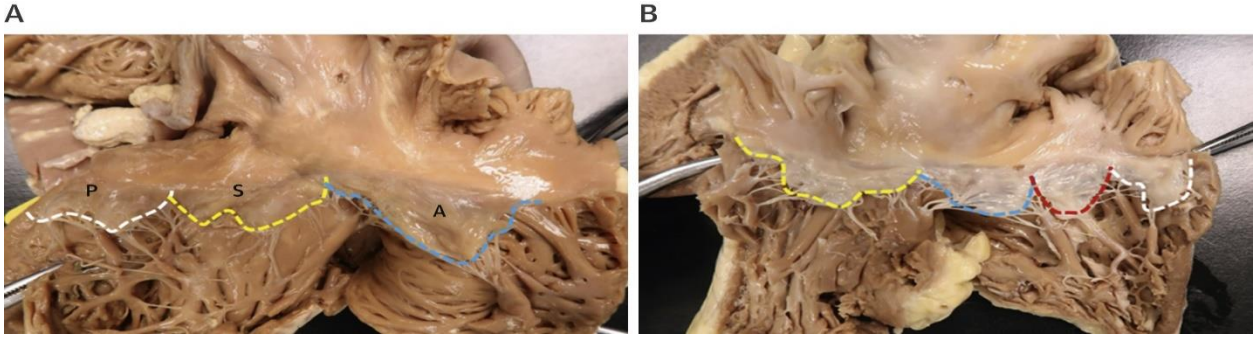


Figure 2 Variable tricuspid valves leaflet number (A) shows the most common configuration 3- leaflet valve

while (B) shows quadricuspid valve (A = anterior leaflet; P = posterior leaflet; S = septal leaflet, orange line in (B) represents the fourth leaflet. (5)

Tricuspid valve tensor apparatus

The papillary muscles and chordae form the "tensor apparatus" of the tricuspid valve. There are 2 distinct papillary muscles (anterior and posterior) and a third variable papillary muscle. The largest muscle is typically the anterior papillary muscle with chordae supporting the anterior and posterior leaflets. The moderator band may join this papillary muscle. The posterior papillary muscle lends chordal support to the posterior and septal leaflets. The septal papillary muscle may be small or multiple or even absent. **(8)**

Chordae of the TV are fibrous cords (80% collagen&20% elastin and endothelial cells) of various lengths that connect the papillary muscles to the tricuspid valve leaflets. The number of chordae varied from 17 to 36 with an average of 25 chordae. **(7)**

From an interventional perspective, the chordae may interact with catheters and devices, causing additional difficulties and challenges during **transcatheter approaches** for the TV. **(9)**

Tricuspid valve annulus

The normal tricuspid annulus is D-shaped and nonplanar with 2 distinct segments: a larger C-shaped segment corresponding to the free wall of the RA and the RV; and a shorter, relatively straight segment that corresponds to the septal leaflet and the ventricular septum. **(10)**

With secondary TR, the tricuspid annulus dilates toward the lateral and posterior free wall and becomes more spherical and planar. Dilation of the septal segment is limited because of its anatomic relation with the fibrous skeleton of the heart . **(11)**

Several important structures lie near the TV. The CS lies near the posteroseptal commissure, the **RCA** courses in the AV groove encircling the annular attachments of the anterior and posterior TV leaflets, the aortic root lies adjacent to the anteroseptal commissure, the AV node and the His bundle lie in the septum adjacent to the septal TV leaflet. **(3)**

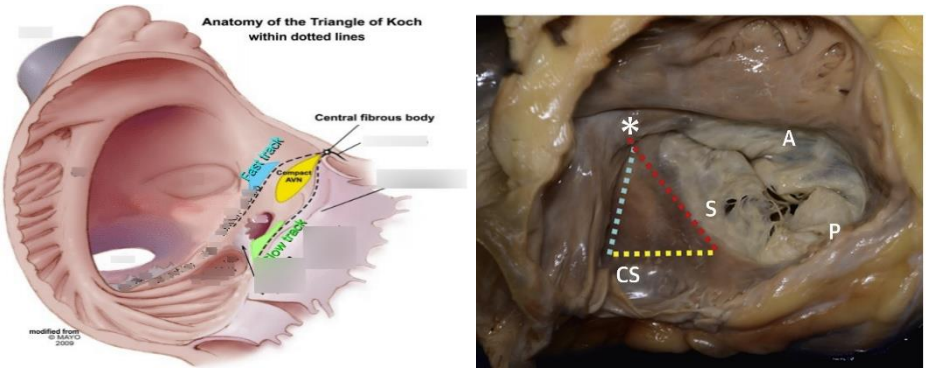


Figure 3. Triangle of Koch and Membranous Septum

The anatomic landmarks of the triangle of Koch. The tendon of Todaro forms one side of the triangle. The hinge point of the septal leaflet forms a second side, and the CS forms the base of the triangle with the apex of the triangle marking the location of the atrioventricular conduction axis near the membranous septum. used as landmark for location of the AV node during procedures such as pacing or ablation.

Tricuspid Regurgitation

Tricuspid valve (TV) disease, mostly seen as tricuspid regurgitation (TR) in adults, is the most common right-sided valvular heart lesion. Physiological trace to mild TR is very common and anatomically normal. For decades, even moderate or greater TR has been considered a benign condition and therefore has been under-treated. Recently, moderate and severe TR have been established a threatening predictor of mortality in patients

with significant left-sided valve disease and LV dysfunction. Clinically **significant** TR, defined as moderate or greater, is more common in women and its prevalence increases by age. **(12)**

TR can be divided into primary and secondary. Primary TR is relatively rare, and it is the consequence of a primitive lesion of the tricuspid valve due to congenital or acquired disease processes that affect the leaflets or chordal structures, or both.

Secondary TR is more common and is secondary to other diseases such as left-side heart diseases, pulmonary hypertension, RV dilation, and dysfunction from any cause, without intrinsic lesion of the TV itself. **(12)**

Causes of Tricuspid Regurgitation

Table 1.classification of causes of TR (5)

Primary TR	Secondary TR
Congenital	**According to the underlying disease:**
Ebstein's anomaly	**Left-sided heart disease**
Tricuspid valve tethering associated with peri-membranous ventricular septal aneurysm or defect	**(valve disease and/or left ventricular dysfunction)**
	Pulmonary arterial hypertension from any cause
TV dysplasia, hypoplasia, or cleft	**RV dysfunction from any cause**
Double orifice tricuspid valve	**Idiopathic often associated with AF**
Other (giant RA)	
Acquired	**According to morphologic abnormality:**
Myxomatous degeneration (Barlow's disease)	**Tethering or tenting of TV leaflets**
Endocarditis	**Displacement of the papillary muscles**
Carcinoid syndrome	**RV dysfunction/dilation**
Rheumatic disease	**Annular dilation**
Trauma	
Pacemaker/device-related	

The most common cause of TR is secondary or "functional" regurgitation. Secondary TR (STR) can be categorized either by the underlying cause or by the morphologic abnormality .

If classified by underlying disease, 4 types are present: **1**) STR because of the left-sided heart disease, **2**) STR because of any cause of pulmonary arterial hypertension, **3**) STR because of any cause of RV dysfunction and **4**) STR with no detectable cause of TR (idiopathic STR).

The morphologic abnormalities associated with STR, which can occur together include:**1**) tethering or tenting of the tricuspid leaflets,**2**) displacement of the papillary muscles, **3**) RV dysfunction, and **4**) dilation of the annulus and/or RA.

The most common causes of Secondary TR are left-sided valve disease (primarily mitral disease), LV and RV cardiomyopathy (ischemic and nonischemic), and RV dilation due to pulmonary disease (cor pulmonale).**(13)**

A unique cause of TR is the result of **pacemaker or defibrillator leads**, which cross from the right atrium into the right ventricle and may directly interfere with leaflet coaptation. In a recent report by Kim et al, the effect of trans-tricuspid permanent pacemaker or implantable cardiac defibrillator leads on 248 subjects with

echocardiograms before and after device placement was studied. The authors found that TR worsened by **1 grade** or more after implant in **24.2%** of subjects and that TR worsening was more common with implantable cardiac defibrillators than permanent pacemakers with baseline mild TR or less. **(14)**

Pacemaker leads can also result in tricuspid stenosis because of leaflet scarring and adhesions.

Pathophysiology of TR

The causes of TR exhibit marked variability among different diseases, particularly in the case of primary TR. Primary TR induces **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. Conditions such as PH and left-sided heart disease can elevate RV afterload, leading to RV dilatation and dysfunction. This remodeling (affecting mainly the anterolateral wall) contributes to tricuspid annular dilatation, further exacerbating TR and 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**. **(15)**

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 the 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**." **(15)**

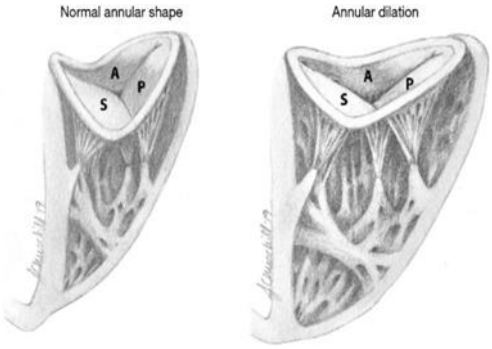


Figure 4 Annular dilatatoin **(16)**

Imaging of the TV

Assessing the severity of tricuspid regurgitation (TR) remains a challenging task, and although **echocardiography** is the test of choice, significant limitations of the current recommendations exist. Recently updated guidelines from the American Society of Echocardiography suggest cardiac magnetic resonance (**CMR**) imaging and computed tomography angiography (**CTA**) may play a significant role.

Transthoracic Echocardiography (TTE)

Transthoracic echocardiography (TTE) and transesophageal echocardiography (TEE) remain the key tools for assessing TR and planning TV interventions.

Current valvular guidelines recommend the use of (**TTE**) to evaluate TR. Assessing right ventricle (RV) size and function, RV systolic pressure, right atrial (RA) size and estimated pressure, and left-sided heart disease are all important. Evaluation of the tricuspid annulus size, leaflet coaptation, and leaflet tethering and motion can help determine the etiology. **(17)**

To fully assess the TV, multiple views with TTE are required, including parasternal RV inflow views, parasternal short-axis view at aortic level, and apical 4-chamber view (RV foused), and subcostal 4-chamber view.

(3)

Based on the ASE Guidelines on right heart imaging, in the **short-axis (SAX) view** at the aortic level, the tricuspid leaflet adjacent to the aortic valve is the **septal** leaflet and the leaflet attached to the basal anterior RV wall is the **anterior** leaflet, Due to the apical position of the septal leaflet, with extreme anterior angulation only the **anterior** leaflet may be seen**. (18)**

In the **parasternal RV inflow** view, the anterior leaflet is always visualized, and based on the angulation of the transducer, the posterior or septal leaflets can be seen.

In **apical 4 chamber** view, the septal leaflet is always seen attached to the septum, but the opposing leaflet could be either the anterior leaflet (when the transducer is angulated anteriorly and the LVOT is in view) or the posterior leaflet (when posterior angulation brings the coronary sinus into view), In the **RV focused** view, the posterior and septal leaflets are almost exclusively seen . **(19)**

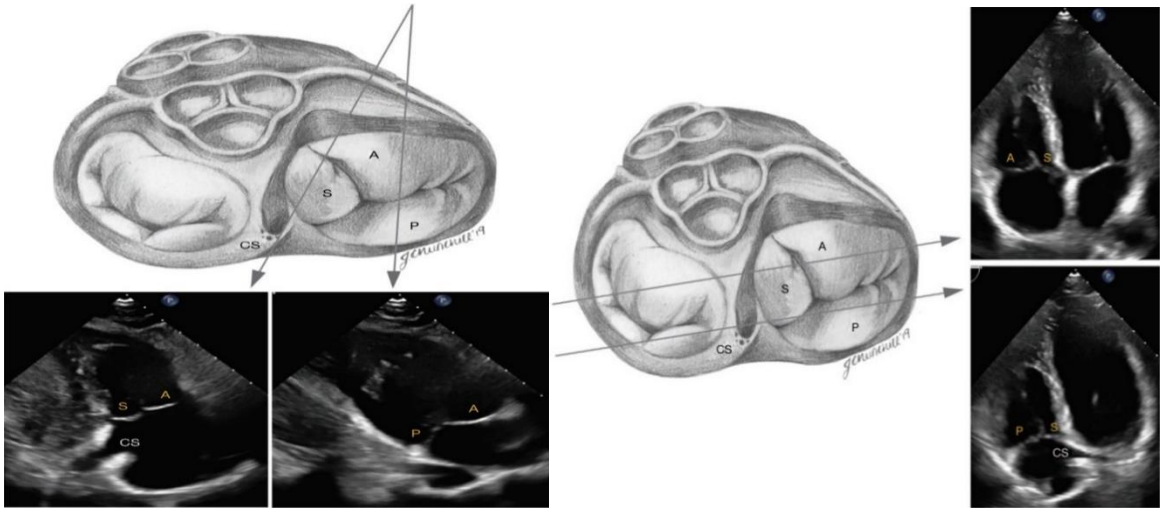


Figure 4 Modified parasternal long axis view (Left) & A4C view (Right) (16)

Recommended Echocardiographic Approaches to Evaluate TR

Most Doppler methods used to characterize left-sided valvular regurgitation (jet size, VC width, proximal convergence analysis, volumetric quantitation, and other characteristics) are directly applicable when assessing the severity of TR keeping in mind that the TR jet is usually a **lower pressure/lower velocity** than is typically found in mitral regurgitation. Except at the extremes of pulmonary hypertension, where pulmonary artery pressure equals systemic pressure. **(21)**

- **Color doppler jet area**

TR severity is traditionally graded by jet area, The relationship between TR jet size and regurgitant severity is governed by the fluid dynamics of turbulent jets, where **severe TR** is defined when the jet area ≥10 cm^2 (with Nyquist limit 50 to 70 cm/sec) **(21)**

Jet area is influenced heavily by blood momentum (flow (Q) = A × V, M = flow(Q) x velocity (V), OR =area(A) x $velocity^2$(V^2), so regurgitant blood traveling through the same effective regurgitant orifice area (EROA) at twice the velocity will have four-times the area. **(3)**

Limitations of Doppler jets are: 1) It is affected by the momentum, which is lower for TR, 2) Reducing the scale reduces the minimal velocity detected, so the jet appear larger 3) Regurgitant jets can be reduced in size due to chamber constraint, (Coanda effect); and 4) multiple views are necessary for adequate TR jet visualization.**(22)**

Despite limitations, jet area was a significant predictor of poor event-free survival after TV repair or bioprosthetic valve replacement. **(23)**

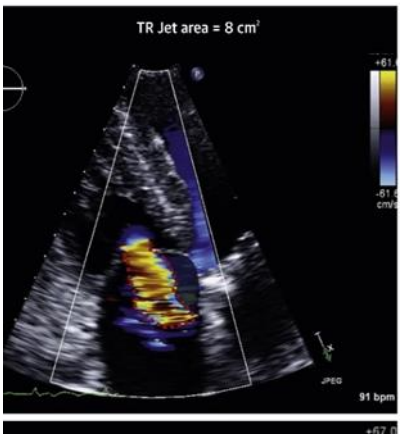


Figure 6: Color Jet Area **(24).**

- **Vena Contracta**

A semiquantitative way to assess TR simply requires measuring the width of the color jet at its narrowest point as it passes through the VC.

The 2017 American Society of Echocardiography valve regurgitation guideline suggests that a VC width <3 mm indicates mild TR, whereas a VC width ≥7 mm indicates severe TR. **(21)**

Limitations: Complex regurgitant jets (often ellipsoid or crescentic) combined with poor lateral resolution make this measure challenging. So, it is recommended that VC is measured as the average of two orthogonal (biplane) views. (Figure7)

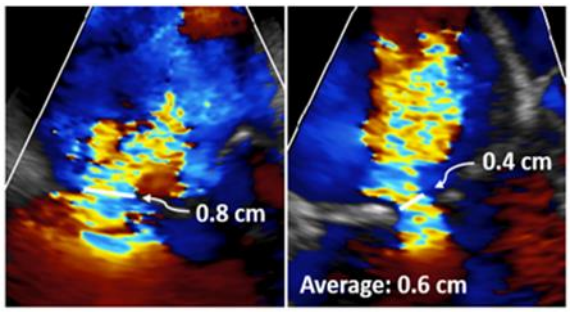


Figure 7: Vena contracta, averaged from biplanar images **(25).**

Continuous wave (CW) velocity profile

The shape and density of the continuous wave (CW) spectral tracing of the TR jet contains useful qualitative information about regurgitant severity.

When the tracing is weak and incomplete, TR is likely trivial or mild, with denser spectra reflecting greater regurgitant volume. Most TR tracings are **parabolic** in shape, reflecting the typical rise and fall of RV pressure.

When TR is severe, RA pressure rises early in systole, resulting in an earlier systolic maximal instantaneous trans-tricuspid gradient and leads to a dense and triangular CW spectral shape. Of note, in the setting of severe, wide open TR, the peak jet velocity is frequently low (<2.5 m/s). **(21)**

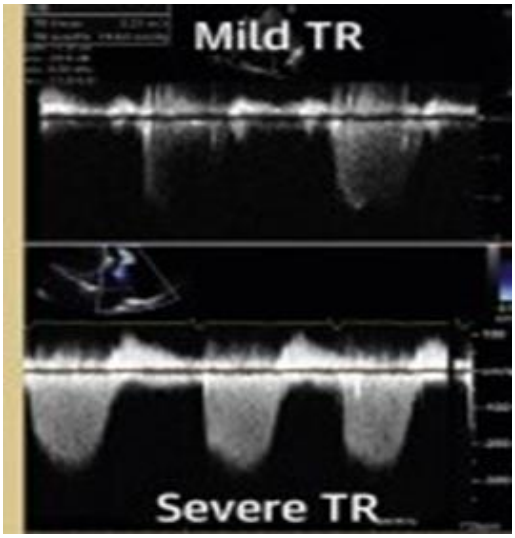


Figure 8: CW doppler of TR **(21).**

Systolic flow reversal in the hepatic vein

Severe TR is often associated with systolic flow reversal in the hepatic veins detected by pulsed wave (PW) spectral Doppler from a subcostal view.

When the RA is small or systemic venous pressure is high ,a smaller amount of TR will raise inferior vena caval pressure enough to reverse flow. When RV function and the annular descent is reduced, a smaller amount of TR may also cause reversal of the systolic flow. One should be aware of potential causes of systolic hepatic vein reversal unrelated to TR, such as ventricular or junctional rhythm with retrograde P-waves. **(26)**

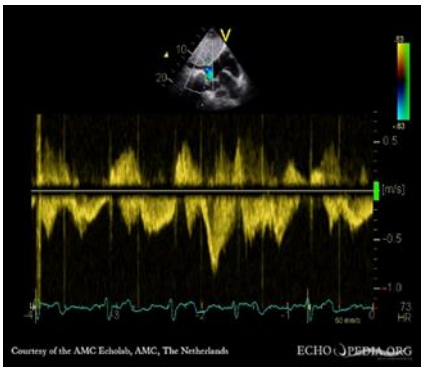


Figure 9 Systolic flow reversal in the hepatic vein

- **Proximal iso-velocity surface area (PISA)**

It is a more **quantitative** technique for assessing TR severity. As regurgitant blood travels toward a narrow orifice, the velocity increases to peak at the narrowest area. This creates hemispheric shells on the upward side of the orifice where blood is traveling at the same velocity.

By shifting the Nyquist limit (recommended to ~30 cm/sec). The radius of this hemispheric shell is then used to calculate flow (Q = area x velocity = hemispheric surface area x $v_a = 2\pi r^2 v_a$).

By the continuity equation, EROA is calculated as flow divided by peak velocity at the exact time when radius was measured, or Q/V_{max} (severe TR ≥ **0.4 cm^2).**

Limitations, PISA may **underestimate or overestimate** TR severity as the orifice is often complex and the shells may be non-hemispheric. **(3)**

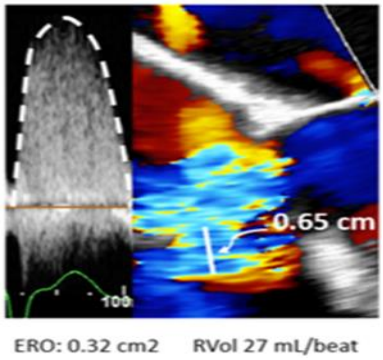


Figure 10 PISA, determined from regurgitant radius **(25).**

- **Volumetric quantification**

In many ways, the optimal approach to any regurgitant lesion is volumetric quantitation, comparing stroke volumes (SV) through the regurgitant valve (i.e., the diastolic stroke volume across the tricuspid valve) with a reference SV from a region of the heart without regurgitation or shunting (i.e., LVOT). (RVol = diastolic flow – stroke volume).

This is useful with dynamic or complex regurgitant jets. Tricuspid annular area is measured using 3D planimetry, also annular diameters measured from biplane imaging or RV inflow and 4-chamber views with an ellipsoid area calculation can be used. Pulsed wave Doppler at the level of the annulus with the measured annular area then gives diastolic flow through the TV, and stroke volume can be estimated by the LVOT (if no shunt) or RVOT or the mitral inflow, or from left ventricular volumetric stroke volume, Severe TR is ≥ 45 mL/beat. **(21)**

Transesophageal Echocardiographic (TEE) Assessment of the Tricuspid Valve

- **Mid-esophageal View**

Using TEE, the first images of the TV are acquired in the mid-esophageal position. The tricuspid annulus and TEE imaging plane are rarely coaxial in this view, so simultaneously imaging all leaflets is difficult. Also, compared to the mitral valve, TV leaflets are thinner and rarely calcified, making them less echogenic. **(17)**

In the four-chamber view, the A and S leaflets are typically in view (Figure 11A). Increasing the transducer angle to 60-70° brings in the RV inflow-outflow view (Figure 11B), and further increasing to 90-130° shows the A and P leaflets (Figure 11C), Withdrawing the probe reveals more anterior portions of the valve (more likely A and S), whereas advancing the probe shows the A and P leaflets, with the coronary sinus (CS) ostium as a useful landmark in the proximal field (Figure 11D). **(17)**

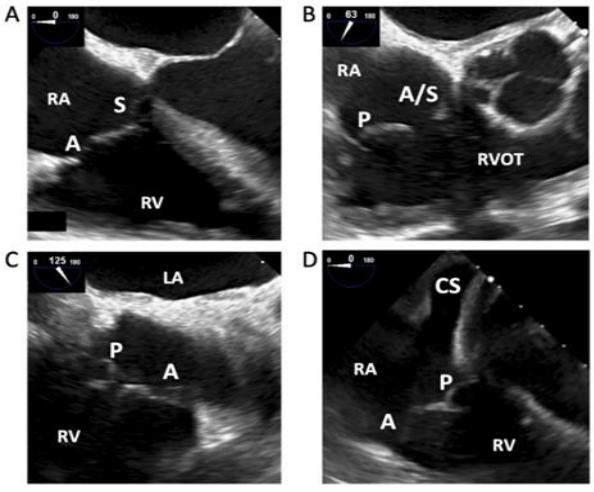


Figure 11: TEE images at the mid-esophageal position **(25).**

- **Trans-gastric view**

The trans-gastric, short-axis view of the TV annular plane is **best to assess the leaflet position** and number. The anteroseptal commissure, septum, and the anterior papillary muscle are identified by advancing and withdrawing the probe to help localize the leaflets, Increasing the transducer to 20-60°, the postero-septal, anteroseptal, and anteroposterior commissures are usually found at 0°, 150°, and 240°, respectively (Figure 12). **(3)**

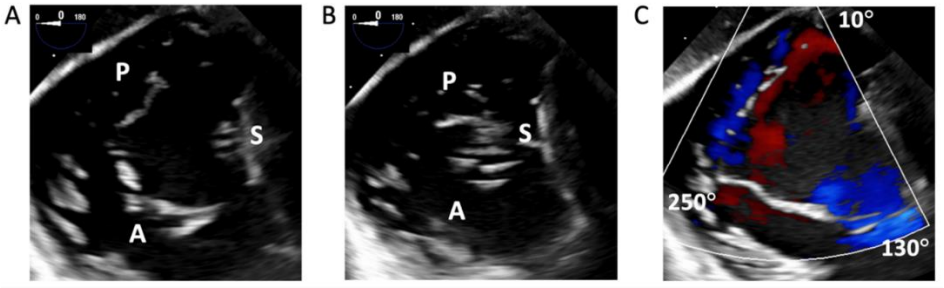


Figure 5 Transgastric View TEE (25).

- **3D Images Acquisition using TEE**

Visualization of the entire TV is possible in 65-70% of routine patients with TEE.3D images are ideally acquired when the leaflets are perpendicular to the beam. As such, the closed valve (during systole) is better imaged in the **deep esophageal view** when leaflets are perpendicular to the insonation beam, whereas the open valve (in diastole) is better imaged in the **transgastric view**. **(17)**

Cardiac Magnetic Resonance (CMR) Quantification of TR

CMR is considered **the gold standard** for right ventricular volumetric quantification due to the complex shape of the right ventricle as well as excellent endocardial definition, independent of body habitus . **(27)**

- **Qualitative assessment by CMR**

One of CMR major advantages is the capability of visualizing the right-sided chamber size and function without ionizing radiation or use of intravenous contrast.

Qualitative assessment of TR by CMR imaging is performed by visualizing the area of local signal drop (spin dephasing) which occurs due to flow turbulence and/or acceleration.

Qualitative grading has been shown to have moderate correlation with quantitative assessment. **(27)**

- **Quantitative assessment by CMR**

CMR-specific cutoff values for TR severity are unknown. The latest guidelines suggest using the same regurgitant severity thresholds that have been used for MR (i.e., a regurgitant fraction of ≤15% for mild TR; 16%-25% for moderate TR; 26%-48% for moderately severe; and >48% for severe TR. **(22)**

Indirect calculation of regurgitant volume

Volumetric analysis of the RV is performed by measurement and interpolation of a short-axis stack of CMR electrocardiographic (ECG)-gated images from the base to the apex of the right ventricle, obtained during breath holding.

The endocardial border of each slice is traced at the end diastole and end systole from which RV volumes are calculated by adding the volumes of the slices using software automation.

After calculation of RV volumetric stroke volume (RVSV), the TR volume (RVol) is then derived as total **RVSV** minus a reference **systemic SV** which can be obtained from: 1) phase contrast (PC) imaging of the pulmonic valve (in the absence of PR) 2) PC imaging of the aortic valve (in the absence of AR) and 3) volumetric

left ventricular stroke volume (in the absence of AR&MR). **(28)**

Direct calculation of regurgitant volume

PC imaging can be used to directly measure regurgitant flow through semilunar valves due to their stable position but challenging for AV valves due to their significant motion and the nonplanar saddle shape of the annuli. **(29)**

- **4D flow imaging by CMR**

A very promising, novel approach is 4D flow velocity-encoded imaging. It has the potential to accurately analyze the complex 3D nature of cardiac flow.

4D flow imaging allows free breathing, time-resolved whole-heart acquisition with velocity encoding in all directions. A standard multiplanar technique can then be used to align the measurement plane perpendicularly to the flow direction offline. **(30)**

- **Limitations of using CMR**

Patients with significant TR develop atrial dilation which causes **atrial arrhythmia** which is an important limitation for CMR quantification of right ventricular volumes and TR feature. **(31)**

The use of compressed sensing, sparsity based methods, and Gadgetron image reconstruction allowed generation of real-time free-breathing cine images with excellent image quality , this sequence has been of great value to patients with AF because it creates clear cine imaging averaging many cardiac cycles, which allows for volumetric analysis and quantification. **(32)**

Computed Tomography (CT)

Cardiac CT plays an important role in the evaluation and management of patients with TV diseases because of high spatial resolution and the ability to perform advanced analysis with specialized software packages. **(33)**

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.

Also, cardiac CT can be used to evaluate the venous system to 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. **(33)**

Intracardiac Echocardiography (ICE)

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 **transcatheter edge-to-edge repair TEER**, tricuspid annulus for percutaneous annuloplasty, or valve structures for transcatheter replacement.

Compared with 2D TEE, 2D ICE has a higher imaging frequency and frame rate acquisition, but this decreases with the addition of color Doppler. **(34)**

symptoms and signs of TR

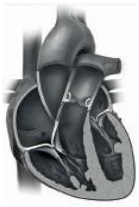
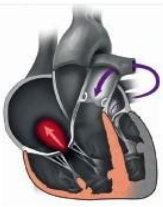
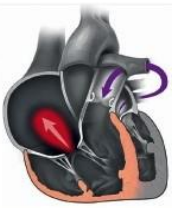
A major challenge in TR management is related to the fact that most patients remain asymptomatic for a protracted span of time, despite having moderate-to-severe TR.

Patients with TR present with the signs and symptoms of right-sided heart failure. The spectrum of presenting symptoms depends on whether the condition is secondary to right ventricular (RV) dilatation or other conditions (eg, left ventricular [LV] dysfunction).

symptoms	Signs
• Dyspnea on exertion, • Orthopnea • Paroxysmal nocturnal dyspnea • Ascites • Peripheral edema • Exercise intolerance may also be observed • Rarely, patients report angina from RV overload and strain, even in the absence of coronary artery disease. **(Sugimoto T et al.,1999)**	• S_3 gallop • RV heaves and S4 gallop that increase with inspiration. • Jugular venous distention with a prominent V wave • Diminished peripheral pulse volume secondary to impaired forward blood flow • Pulmonary rales, if TR is associated with LV dysfunction or MS • Ascites & Peripheral edema • Cachexia and jaundice · Atrial fibrillation

Table 2 Summarizing symptoms and signs of TR (35)

Table 3 A proposed classification to understand better the spectrum of patients presenting with FTR and the proposed treatment strategies. (35)

	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5
					
Symptoms	None	None*	None-vague*	Current or previous episodes of RHF	Overt RHF and/or end-organ damage due to chronic RV volume overload#
TR grade	Less than moderate	>Moderate	Severe	Severe	Torrential
Annular remodelling	Normal	Normal or mildly remodelled	Present	Moderate-severe	Severe
Leaflet coaptation	Normal	Mildly abnormal	Abnormal	Coaptation gap	Large coaptation gap
Tethering	None	None or mildly abnormal (<8 mm)	Abnormal (usually <8 mm)	Significantly abnormal with varying degree of tethering	Significantly abnormal (usually >8 mm)
RV function and remodelling	Normal	Normal function Absent or mild remodelling	Mild RV dysfunction and/or remodelling	>Moderate dysfunction and remodelling	Severe RV dysfunction and remodelling
Medical	No treatment but regular clinical and echo follow-up in patients with high likelihood of developing TR progression such as those in Table 1	None or low-dose diuretics	Diuretics	Moderate to high-dose diuretics and/or requirement for IV diuretics	Multiple admissions for RHF. Frequent need for IV diuretics and/or high-dose combination diuretics
Surgical treatment	No	Consider TV surgery (preferably repair) at time of left-sided surgery	TV surgery (preferably repair) at time of left-sided surgery. Isolated TV surgery (preferably repair) in presence of symptoms or progressive RV remodelling and comorbidities	Isolated TV surgery (repair or replacement) either isolated or at time of left-sided surgery in the absence of severe pulmonary hypertension and severe comorbidities. High risk of perioperative RV dysfunction.	Prohibitive intra- and peri-operative risk
Percutaneous treatment	No	Potential future target for percutaneous options as minimally invasive option could change natural history with minimal risk	Potential candidates for isolated TR surgery who could be enrolled in upcoming IDE RCTs	Current group of patients being treated in EFS if high-risk for surgery. May require combination of annuloplasty and leaflet device or TVR	Prohibitive risk and potentially futile. (Palliative procedures can be considered in highly selected patients)

Treatment of tricuspid regurgitation

Medical treatment options for functional TR

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 primary TR and symptomatic right-sided HF, medical treatment should focus on treating the primary cause of HF and cardiovascular congestion using diuretics **(Class II a)**.

In case of secondary TR, the treatment of TR depends on the treatment of the cause such as underlying cardiomyopathy, PH or resultant structural complications that might further worsen TR such as AF. **(Class II a). (36)**

In case of LV dysfunction, cornerstone management is the use of guideline-directed medical therapy (**GDMT**) which includes renin-angiotensin-aldosterone system blockers/angiotensin neprilysin inhibitors, β-blockers, mineralocorticoid receptor antagonists, and SGLT-2 inhibitors. **(37)**

In case of HF with preserved ejection fraction drugs are limited to diuretics (**Class 1**), SGLT-2 inhibitors (**Class II a**), angiotensin neprilysin inhibitors, MRA, and angiotensin receptor blockers (**Class II a**). **(37)**

If there is suspicion of pulmonary hypertension, further investigations, including right heart catheterization is essential to classify pulmonary hypertension and identify patients who might benefit from specific drug therapy (e.g., calcium channel blockers, endothelin receptor antagonists, PDE-5 inhibitors and guanylate cyclase stimulators, prostacyclin analogues and prostacyclin receptor agonists). **(36)**

Oral anticoagulation could be initiated in patients with right heart chamber dilatation due to (repeated) pulmonary embolism.

In case of Atrial fibrillation or flutter, rhythm management can have an impact on TR, but the effect can vary widely among individuals, Studies have shown that some patients experience a reduction in TR after successful rhythm control, while others may not see significant improvement. **(38)**

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 diuretic therapy after their procedure, but doses may decrease or stabilize over time. **(39)**

Surgical treatment of functional TR

The optimal timing of surgical intervention for TR remains controversial. However, delayed surgery must be avoided due to the risk of irreversible right ventricular damage, organ failure and poor results of later surgical intervention.

The **2017&2021 ESC/EACTS** Guidelines for the Management of valvular heart disease provide a **class I (level of evidence: C)** recommendation for TV surgery in patients with severe functional TR undergoing left-sided valve surgery.

Concomitant treatment of TR during left-sided heart surgery does not increase risk of postoperative morbidity, mortality or permanent pacemaker requirement. According to a recent meta-analysis, concomitant tricuspid valve repair during left-sided valve surgery was associated with a reduction in cardiac-related mortality and improved echocardiographic outcomes for TR after a mean follow-up of six years. **(40)**

A **class IIa** recommendation is given for tricuspid valve surgery in patients with mild or moderate functional TR with a dilated tricuspid annulus (i.e., ≥40 mm or >21 mm/m² by 2D echocardiography) undergoing left-sided valve surgery. **(41)**

Tricuspid valve surgery may be considered in patients undergoing left-sided valve surgery with mild or moderate secondary tricuspid regurgitation even in the absence of annular dilatation when previous recent **right heart failure** has been documented (**class IIb**). **(41)**

In addition, the guidelines provide a **class IIa** recommendation for TV surgery in case of severe TR after previous left-sided surgery if patients are symptomatic, or progressive RV dilatation or dysfunction is present.

Table 3 ESC 2021 guidelines Recommendations on indications for intervention in tricuspid valve disease (42)

Recommendations	Class[a]	Level[b]
Recommendations on tricuspid stenosis		
Surgery is recommended in symptomatic patients with severe tricuspid stenosis.[c]	I	C
Surgery is recommended in patients with severe tricuspid stenosis undergoing left-sided valve intervention.[d]	I	C
Recommendations on primary tricuspid regurgitation		
Surgery is recommended in patients with severe primary tricuspid regurgitation undergoing left-sided valve surgery.	I	C
Surgery is recommended in symptomatic patients with isolated severe primary tricuspid regurgitation without severe RV dysfunction.	I	C
Surgery should be considered in patients with moderate primary tricuspid regurgitation undergoing left-sided valve surgery.	IIa	C
Surgery should be considered in asymptomatic or mildly symptomatic patients with isolated severe primary tricuspid regurgitation and RV dilatation who are appropriate for surgery.	IIa	C
Recommendations on secondary tricuspid regurgitation		
Surgery is recommended in patients with severe secondary tricuspid regurgitation undergoing left-sided valve surgery.[423–427]	I	B
Surgery should be considered in patients with mild or moderate secondary tricuspid regurgitation with a dilated annulus (≥40 mm or >21 mm/m^2 by 2D echocardiography) undergoing left-sided valve surgery.[423,425–427]	IIa	B
Surgery should be considered in patients with severe secondary tricuspid regurgitation (with or without previous left-sided surgery) who are symptomatic or have RV dilatation, in the absence of severe RV or LV dysfunction and severe pulmonary vascular disease/hypertension.[418,433 e]	IIa	B
Transcatheter treatment of symptomatic secondary severe tricuspid regurgitation may be considered in inoperable patients at a Heart Valve Centre with expertise in the treatment of tricuspid valve disease.[f]	IIb	C

Risk stratification using TRI-Score

This score has been proposed for isolated tricuspid surgery based on clinical predictors to predict mortality in order to adequately select patients who will benefit from an intervention or who are eligible for surgery.

Table 4TRI-Score: prediction of in-hospital mortality after isolated TV-surgery. (43)

Risk factors	Points
Age > 70 years	1
NYHA III/IV	1
Right heart failure signs	2
Furosemid > 125 mg/d	2
eGFR < 30 ml/min	2
Elevanted total Bilirubin	2
LVEF < 60%	1
Right ventricular dysfunction	1
Total	12

Score	Mortality in %
0	1
1	2
2	3
3	5
4	8
5	14
6	22
7	34
8	48
≥ 9	65

Surgical repair techniques for functional TR

Surgical tricuspid valve repair with **annuloplasty** (**TV reconstruction**) is the preferred treatment for functional TR in patients with suitable anatomy, preserved right ventricular function and acceptable surgical risk. **(41)**

Tricuspid valve annuloplasty techniques aim to target the pathophysiological hallmark of functional TR, i.e., dilatation of the tricuspid annulus mainly along its posterior portion with an increased size of the tricuspid valve orifice.

Since the 1960s, several surgical annuloplasty methods have been described, including **suture, band and ring techniques**. Amongst them, suture bicuspidisation, known as the **Kay procedure**, intends to reduce TR by obliterating the annular segment corresponding to the posterior leaflet through placement of pledget-supported mattress sutures in the annulus so the tricuspid annular circumference is reduced, and the tricuspid valve is converted into a smaller but competent mitral-like valve. **(44)**

During **De Vega annuloplasty**, 2 C-shaped suture lines are placed along the base of the anterior and posterior tricuspid valve leaflet, starting at the anteroseptal commissure and ending beyond the origin of the coronary sinus. At the anteroseptal and posteroseptal commissures the sutures lines are anchored with pledgets and tied to reduce the tricuspid valve orifice diameter **(44)**

Currently, TV annuloplasty is performed by implantation of an **undersized ring** (rigid, semi-rigid, flexible) or a flexible band. Compared with the use of flexible annuloplasty bands, the implantation of a rigid or semi-rigid ring is associated with a reduced incidence of late, recurrent TR. However, use of a rigid ring may increase the risk of subsequent ring dehiscence **(45)**

Annuloplasty rings or bands do not cover the whole tricuspid annulus but remain open at the septal annulus to avoid injury to the conduction system. In recent years, refined tricuspid annuloplasty rings resembling the **complex 3D** configuration of the native tricuspid annulus have been developed. Retrospective analyses have suggested that ring annuloplasty is **superior** to suture annuloplasty in terms of residual and recurrent TR, redo surgery and survival, however the choice of annuloplasty ring (rigid vs. flexible vs. 3D) is still a matter of debate. **(45)**

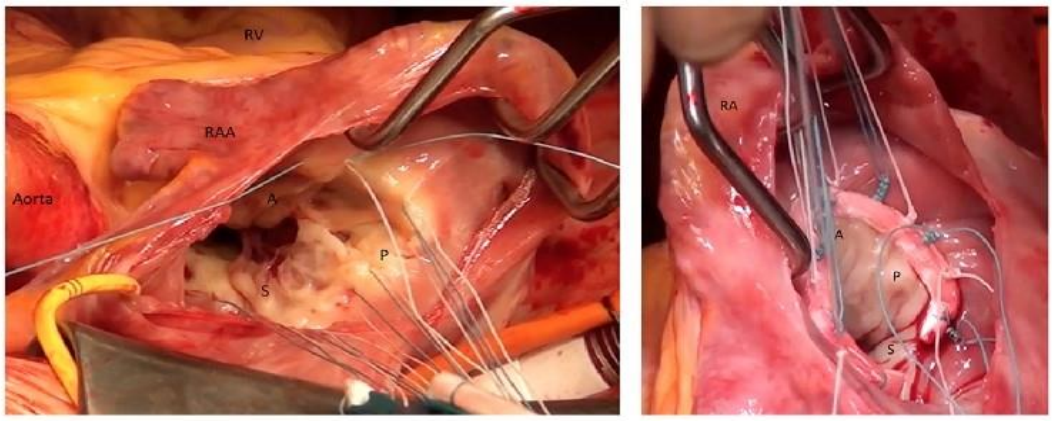


Figure 6 Intraoperative surgical view of the tricuspid valve. Left: annuloplasty ring sutures are placed along the annulus of the tricuspid valve. Right: implantation of an annuloplasty ring into the tricuspid valve. The septal annulus remains uncovered to avoid passing the anchoring sutures close to the conduction system

TV repair is associated with lower perioperative mortality as compared to valve replacement in patients with functional TR and, so, is the preferred surgical option. **(46)**

TV replacement should be considered when valve repair is technically not feasible or predictably not durable (e.g., in patients with profound RV remodeling or dysfunction, or higher pulmonary artery pressures). Studies comparing bioprosthetic and mechanical valves for tricuspid valve replacement indicated similar long-term outcomes. **(44)**

Limitations for surgical TR repair in clinical practice

One of the biggest **limitations** is repeated surgery for symptomatic TR after left-sided valve surgery, which is associated with increased perioperative morbidity and mortality up to 20%, The increased risk of reoperation is attributed to the late referral and poor clinical condition of the patient.

Minimally invasive TV operations through a right thoracotomy may provide an alternative surgical treatment option associated with excellent early outcome even in high-risk patients undergoing elective re-operative TR repair for TR after left-sided heart surgery. **(47)**

Another **limitation** is elderly patients presenting with massive TR and intractable symptoms of RSHF without prior left-sided heart surgery,Up until recently, conventional and/or minimally invasive TV surgery was offered to these patients next to medical treatment. **(48)**

Nowadays, emerging **transcatheter therapies** may provide an alternative treatment option for these patients if surgical risk exceeds the potential benefit.

Emerging transcatheter treatment options for functional TR

In recent years, several devices have entered preclinical and early clinical testing for transcatheter treatment of functional TR.

Current existing transcatheter therapies for functional TR can be divided into three groups according to their mechanism of action and anatomic therapeutic target into **annuloplasty devices**, **coaptation devices** and **heterotopic caval valve implantation**. (Figure 14).

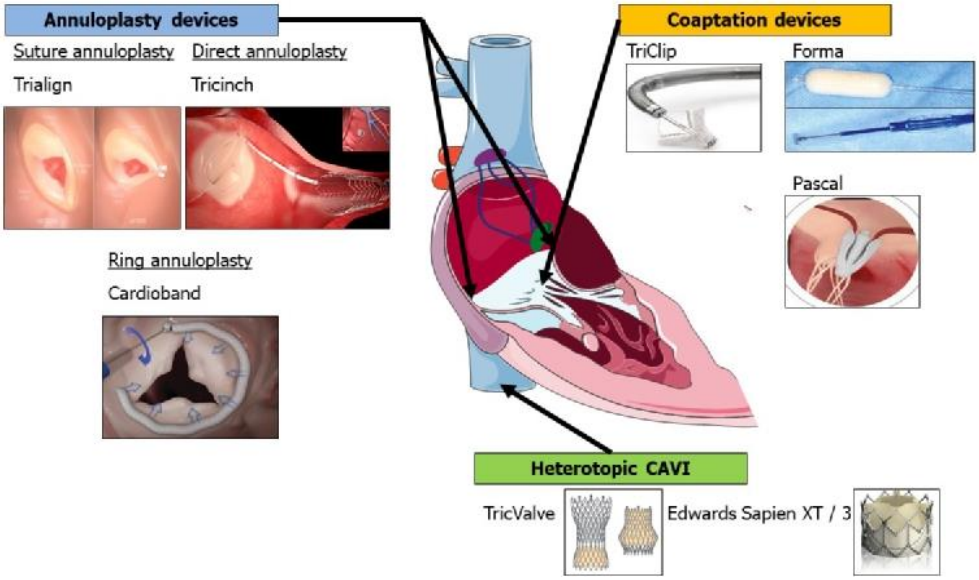


Figure 7 Transcatheter techniques targeting functional TR. (48)

- **Percutaneous tricuspid valve annuloplasty devices**

The first group comprises percutaneous tricuspid valve annuloplasty devices, based either on transcatheter suture or ring implantation techniques.

(1) Trialign™

The Trialign™ device (Mitralign Inc., Tewksbury, MA, USA) is a transjugular suture-based tricuspid valve annuloplasty system which aims to reduce tricuspid annular diameter through tissue plication.

The results of the **SCOUT** trial suggest that TR reduction by the Trialign device translates into improvements in NYHA functional class. **(49)**

(2) TriCinch

The **TriCinch™** system (4Tech Cardio, Galway, Ireland) aims to reduce the septo-lateral tricuspid annular diameter by implanting a nitinol coil anchor in the anteroposterior TA and applying tension on the annulus via a Dacron band fixed to a self-expanding nitinol stent in the inferior vena cava. **(35)**

(3) Cardioband™ Tricuspid Valve Reconstruction System

The **Cardioband** implant (Edwards Lifesciences, Irvine, CA, USA) is composed of an adjustable fabric band with up to 17 anchors for annular fixation and aims to reduce TR via annular reduction and is advanced into the right atrium via transfemoral access.

The **TRI-REPAIR** trial examined safety, efficacy and early functional benefits of TR treatment with the Cardioband tricuspid system. **(35)**

- **Percutaneous tricuspid valve coaptation devices**

The second group currently includes three devices designed to improve leaflet coaptation in functional TR.

(1) Tri-Clip (Used for Tricuspid transcatheter edge-to-edge repair (T-TEER))

According to recent data, the transfer of the **MitraClip®** technique to the tricuspid valve is the most common technique applied for interventional TR treatment. **(50)**

Notably, in patients with both significant mitral and tricuspid regurgitation at high surgical risk, combined transcatheter mitral plus tricuspid valve edge-to-edge repair using the **MitraClip** technique was associated with

superior functional improvement and fewer hospitalisations for heart failure as compared to isolated mitral valve edge-to-edge repair. **(51)**

(2) FORMA

The **FORMA** repair system (Edwards Lifesciences, Irvine, CA, USA) aims to reduce malcoaptation of the tricuspid leaflets by placing a balloon spacer through the central coaptation line, which reduces the regurgitant orifice area. **(52)**

(3) PASCAL

The **PASCAL** system (Edwards Lifesciences, Irvine, CA, USA) has undergone first-in-human, compassionate use experience in 23 patients with severe mitral regurgitation.

Recently, the device was successfully implanted in a patient with severe functional TR for the first time. The novel **PASCAL** system incorporates a spacer to fill the regurgitant jet area, paddles designed to avoid stress concentration on native leaflets, and clasps which allow for independent leaflet capture. Hence, the PASCAL device in a way integrates the mode of action of the **FORMA** and the **TriClip** devices. **(53)**

- **Heterotopic caval valve implantation (CAVI)**

The third group comprises techniques for heterotopic caval valve implantation, where either two dedicated self-expanding bioprosthetic valves (TricValve; P&F Products & Features Vertriebs GmbH, Vienna, Austria,) or balloon-expandable valves used to treat aortic stenosis (29 mm Edwards SAPIEN XT or SAPIEN 3; Edwards Lifesciences, Irvine, CA, USA) are placed in the inferior and superior vena cava.

The use of balloon-expandable valves requires prior implantation of a self-expanding stent in the inferior (and occasionally superior) vena cava to prepare a landing zone, given the large diameter of the vena cava and the hepatic vein confluence. The technique was recently tested in an excess-risk cohort and appeared safe and feasible with a high procedural success rate. Substantial in-hospital and 30 day-mortality rates were attributed to severe and non-device-related comorbidities in these patients. **(54)**

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