Imaging

Assessment of functional tricuspid regurgitation

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Functional tricuspid regurgitation (FTR) is characterized by structurally normal leaflets and is due to the deformation of the valvulo-ventricular complex. While mild FTR is frequent and usually benign, patients with severe FTR may develop progressive ventricular dysfunction and incur increased mortality. Therefore, FTR should not be ignored, should be appropriately diagnosed and quantified by Doppler echocardiography, and should be evaluated for corrective surgical procedures. At present, referral for surgical correction of FTR is often delayed until patients develop intractable heart failure. However, this strategy frequently translates in poor clinical outcome characterized by notable operative mortality and reduced long-term survival. Appropriate patient selection and proper timing for tricuspid valve (TV) repair or replacement are crucial for optimal outcome, but objective criteria for clinical decision-making remain poorly defined. In the present paper, we review the anatomy of the normal TV, the pathophysiology of FTR, the assessment of its severity and functional significance, and propose an algorithm for selecting patients for surgical treatment.

Keywords Functional tricuspid regurgitation • Tricuspid valve • Echocardiography • Three dimensional • Rightventricle • Pathophysiology

Introduction

The aetiology of tricuspid regurgitation (TR) is generally divided into organic [with intrinsic tricuspid valve (TV) disease] and functional (FTR) in the absence of structural abnormalities of tricuspid leaflets (Table 1). Organic TR results from structural abnormalities of TV apparatus, may be congenital or acquired and accounts for only 8–10% of all severe TRs.1,2 Functional TR is a consequence of deformation of the TV apparatus, such as dilation and geometric deformation of tricuspid annulus (TA), and therefore the term ‘secondary TR’ is also used.

Functional TR is frequently caused by increased right ventricular (RV) afterload and it is associated with advanced stages of left-sided valve, myocardial or pulmonary diseases. In these patients, moderate or severe TR has a significant impact on their functional capacity and long-term survival,1 and surgical repair or replacement of the TV is the only corrective treatment presently available. However, operations for symptomatic FTR have the reputation for being high-risk procedures, particularly in candidates to redo surgery after previous correction of left-sided heart valve disease,1–5 and several investigators support a more aggressive, earlier surgical approach to FTR.6–8 Another issue making the clinical decision-making difficult is the notable rate of recurrence of FTR after surgical repair,9 which emphasizes the importance of a detailed anatomic diagnosis prior to making the decision of a repair vs. replacement approach.

Despite the general agreement regarding the need for appropriate patient selection and optimal timing of surgical treatment of FTR, there is a lack of objective criteria to guide clinicians in proper assessment of patients with FTR.9

In this paper, we review the anatomy of the normal TV, the pathophysiology of FTR, the assessment of its severity and functional significance, and propose an algorithm for selecting patients for surgical treatment.

Functional anatomy of the tricuspid valve complex

The TV is the most caudally located and has the largest orifice among the four cardiac valves.

Classically, the TV complex consists of three leaflets (anterior, posterior, and septal) inserted on the narrow fibrous TA and joined by chordae tendinae to their specific papillary muscles attached to the RV myocardium (Figure 1). Effective valve function depends on the integrity and coordination of these components.
Both the anterior and posterior (also named mural or inferior) tricuspid leaflets arise from the corresponding RV free wall. The three leaflets are unequal in size: the anterior leaflet is the largest, while the posterior leaflet is notable for the presence of multiple scallops. The septal leaflet is the smallest and arises medially directly from the TA above the membranous interventricular septum.

As shown in Figure 1, the anatomy of the mitral and TVs displays many differences beyond the number of leaflets. The papillary muscles are different than those supporting the mitral valve, smaller, often multiple, widely separated, and carrying chordae to a single TV leaflet. Finally, there may be accessory chordal attachments to the RV free wall and to the moderator band. These complex and multiple chordal connections play an important role in the genesis of FTR, as they affect proper leaflet coaptation in the setting of RV enlargement and dysfunction. Indeed, as mitral papillary muscles each serve both leaflets, the separation of leaflets by annular enlargement is limited, while the link of one tricuspid leaflet to only one set of subvalvular support allows wide separation of leaflets with annular and cavity enlargement.

The TA is a complex (less symmetric than the ‘saddle-shaped’ mitral annulus) non-planar, highly dynamic structure, which changes in shape and size during the cardiac cycle due to the contraction of the surrounding myocardium. It is oval in shape, with the postero-lateral portion being ‘lowest’ (towards the RV apex) and the antero-septal portion the highest. Tricuspid annulus diameter, circumference, and area are all larger than that of mitral annulus by about 20%. Although values of major TA diameter of 30–35 mm are described for normal adults, the orifice size is related to body size. Thus, the average normal TA diameter is 21 mm/m². The TA displays a ~19% reduction in annular circumference (~30% reduction in annular area) during atrial systole. It provides a firm and pliable base for the three-valve leaflets and it is in continuity with the aortic and mitral annuli. The septal portion of the TA is relatively fixed due to its position between the fibrous trigones. Therefore, as the RV free wall expands outwards, dilatation of the TA occurs primarily in its free wall aspect.

The diastolic opening of the valve along with corresponding expansion of the annulus provides a tricuspid orifice area of 7–9 cm². The systolic narrowing of the orifice provides an effective valve closure; however, a trivial or mild TR is detectable using Doppler echocardiography in 80–90% of normal subjects.

### Table 1  Aetiology of tricuspid regurgitation

<table>
<thead>
<tr>
<th>Aetiology</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Functional (morphological normal leaflets with annular dilatation)</td>
<td>Left heart diseases (LV dysfunction or valve diseases) resulting in pulmonary hypertension, Primary pulmonary hypertension, Secondary pulmonary hypertension (e.g., chronic lung disease, pulmonary thrombo-embolism, left-to-right shunt), Right ventricular dysfunction from any cause (e.g., myocardial diseases, ischaemic heart disease), Atrial fibrillation, Cardiac tumours (particularly right atrial myxomas), Structural abnormality of the tricuspid valve, Rheumatic Prolapse, Congenital Ebstein anomaly, Tricuspid valve dysplasia, Tricuspid valve hypoplasia, Tricuspid valve cleft, Double orifice tricuspid valve, Unguarded tricuspid valve orifice, Endocarditis, Endomyocardial fibrosis, Carcinoid disease, Traumatic (blunt chest injury, laceration), Iatrogenic Pace-maker/defibrillator lead interference, Right ventricular biopsy, Drugs (e.g. exposure to fenfluramine-phentermine, or methysergide), Radiation</td>
</tr>
</tbody>
</table>

While mild FTR is frequent without detectable abnormality of the valvulo-ventricular complex, the formation of a larger regurgitant orifice requires a combination of TA dilation and valvular deformation. The analysis of respiratory changes of TR by quantitative methods has provided important insights in that regard. All types of TR are characterized by increasing regurgitant orifice during inspiration. This enlarged orifice is opposed by a decline in regurgitant gradient (from RV to right atrium) so that the combination of these opposite changes results in a larger regurgitant volume during inspiration but with an increase of lesser magnitude than that of the regurgitant orifice. The increase in regurgitant orifice area with inspiration is due to a combination of annular enlargement and valvular tenting. These two mechanisms variously combined form the basis of all forms of FTR. Those caused by RV enlargement and dysfunction, that may be primary or secondary to left heart diseases resulting in pulmonary hypertension, are predominantly due to valve tenting, while FTR without pulmonary hypertension is predominantly due to annular enlargement (Figure 2). These factors explain the fact that while pulmonary arterial hypertension from any cause is known to be associated with FTR, not all patients with pulmonary hypertension develop significant TR. Mutlak et al. assessed the determinants of TR severity in 2139 patients with pulmonary hypertension. In this population, elevated pulmonary artery systolic pressure was associated with more severe TR (odds ratio 2.26 per 10 mmHg increase). However, a large number of patients with elevated pulmonary artery systolic pressure showed only mild TR (65.4% of patients with moderate and 45.6% of patients with severe pulmonary hypertension, respectively). Other factors such as atrial fibrillation, pacemaker leads, and RV remodelling were also significant determinants of the severity of TR. Among them, the spherical remodelling of the RV in response to pulmonary hypertension was the most powerful predictor of FTR. Idiopathic dilatation of the TA often

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**Figure 1** Anatomic specimens showing tricuspid and mitral apparatus anatomy. Red arrows indicate the atrio-ventricular to semilunar valve distances. Yellow arrows indicate the specific differences between tricuspid and mitral valves. Tricuspid valve apparatus is characterized by multiple, widely separated papillary muscles which carry chordae to a single tricuspid valve leaflet. Accessory chordal attachments to the right ventricular free wall are also noted (Courtesy of Dr William Edwards, Mayo Clinic).

**Figure 2** Mechanisms of functional or secondary tricuspid regurgitation.
associated with atrial fibrillation can cause FTR, which is sometimes severe. Furthermore, TA dilatation caused by the volume overload of FTR in turn causes further separation of leaflets and progression of FTR. Thus, annular dilatation, RV dilatation or TV tenting, and not pulmonary hypertension in and by itself are the main determinants of FTR.\textsuperscript{15}

These mechanisms of FTR can persist even after successful correction of a potentially causal disease (Figure 3). In patients with chronic pulmonary thrombo-embolic hypertension or mitral stenosis, after successful pulmonary thrombo-endarterectomy or mitral balloon valvuloplasty, there was no change in TA diameter after the resolution of pulmonary hypertension.\textsuperscript{16,17} After repair of ischaemic mitral regurgitation,\textsuperscript{18} TA dilatation also persists and is associated with late recurrent TR. In addition, TA dilatation is usually associated with annular flattening (loss of its saddle shape), which may increase the annulus-to-papillary muscle distance, altering the normal relationship between TV leaflet, papillary muscles, and annulus, and increasing TV leaflet tethering.\textsuperscript{19}

Once TR has become significant, progressive RV remodelling and dysfunction due to chronic volume overload result in papillary muscle displacement and leaflet tethering, which worsen TR and lead to further RV dilatation.\textsuperscript{20} Such valvular deformation involves marked tenting and is frequent in patients with pulmonary hypertension. Post-annuloplasty patients with marked valvular tenting (Figure 3) may incur aggravation of the tethering if the RV does not display reverse remodelling. Hence, coaptation height and tenting area, reflecting TV tethering, are important predictors of the severity of early residual TR after TV repair.\textsuperscript{20} A preoperative coaptation height >1.0 cm has been shown to predict a 55% repair failure immediately after surgery.\textsuperscript{21} An adjunctive procedure (i.e. anterior leaflet elongation with a pericardial patch) to reduce leaflet tethering in patients with a coaptation distance >8 mm or a tenting area >16 mm\textsuperscript{2} has been reported to decrease TR recurrence.\textsuperscript{22,23} With three-dimensional echocardiography (3DE), the extent of TV tethering can be measured in terms of tenting volume and this parameter has been reported to independently determine residual TR after tricuspid annuloplasty. A TV tenting volume >2.3 mL showed a sensitivity of 100% and specificity of 84% to predict severe residual TR after tricuspid annuloplasty.\textsuperscript{24}

Persistently high post-operative RV pressures increase the progression and severity of residual TR\textsuperscript{25,26} possibly through persistent RV enlargement and dysfunction, with valve tethering. Occurrence of atrial fibrillation is also a significant factor in the development of TR in patients with mitral valve disease.\textsuperscript{27} In these patients, the increase in left atrial pressure and size causes atrial fibrosis that in turn causes right atrial dilatation and further increase in TA enlargement.\textsuperscript{16,28}

**Epidemiology**

Using echocardiography, the Framingham Heart Study investigators found a prevalence of moderate or severe TR of 0.8% and an increased prevalence with ageing.\textsuperscript{12} Overall, the prevalence of significant TR was 4.3 times greater in women than in men.

Tricuspid regurgitation is frequently present in patients with mitral valve disease and more than one-third of patients with mitral stenosis have at least moderate TR.\textsuperscript{29,30} Severe TR has been reported in 23–37% of patients after mitral valve replacement for rheumatic valve disease.\textsuperscript{31,32} Functional TR is frequently observed in the advanced stage of left-sided valvular heart disease or myocardial disease,\textsuperscript{33} but 14% of TR may occur in the absence of structural TV alterations, pulmonary hypertension, or left heart dysfunction.\textsuperscript{31}

Finally, the development of haemodynamically significant TR has been reported in 27% of patients who had only mild TR at the time of left-sided valve surgery.\textsuperscript{6} In most cases, TR is diagnosed late after mitral valve replacement, 10 years on average, but can appear as late as 24 years after the initial surgery.\textsuperscript{31,32} Matsunaga and Duran\textsuperscript{18} reported moderate or severe TR in 74% of patients 3 years after surgical repair of ischaemic mitral regurgitation.

**Assessment of patients with functional tricuspid regurgitation**

**Clinical evaluation**

The most prominent physical features of TR are those related to the characteristics of the murmur and to the development of right-sided congestive heart failure. In the early stages of disease, patients remain completely asymptomatic or present with fatigue and shortness of breath before developing overt signs of right-heart failure. With severe right-sided failure, patients with severe TR may present with weight loss and cachexia, cyanosis, and jaundice (reflecting hepatic insufficiency). Ascites and peripheral oedema and even anasarca may be present.

![Figure 3](image-url) Tricuspid leaflet tethering (arrows) after restrictive annuloplasty (yellow dotted line) in the presence of severe dilatation of right-heart chambers. LA, left atrium; RA, right atrium; RV, right ventricle.
Role of imaging techniques

Cardiac catheterization
Before the introduction of 2DE and Doppler echocardiography, cardiac catheterization was the main imaging modality to confirm the presence and severity of TR. The diagnosis of TR posed a greater challenge, as selective angiography into the RV would often distort the TV and catheter position in the RV would be difficult to maintain. At present, diagnostic cardiac catheterization should rarely, if ever, be undertaken for the diagnosis or quantitation of TV disease alone.

Chest X-ray
Chest radiograph is also of limited utility. Cardiomegaly associated with prominent right-heart borders may be noted, but there are no specific findings which suggest a diagnosis of TV disease.

Echocardiography
Two-dimensional echocardiography combined with spectral and colour flow Doppler is now the main modality for detection and quantitation of TR.34 ‘Physiological’ FTR is associated with normal valve leaflet morphology and normal RV and atrial size. The colour jet is localized in a small region adjacent to valve closure (usually <1 cm), is thin, central, and often is limited to early-systole. However, the precise threshold at which FTR transitions from physiological to abnormal remains uncertain.

When TR is detected by colour Doppler and may be abnormal or ‘pathological’ (moderate or more severe), comprehensive description of leaflet morphology and of the pathophysiological mechanisms underlying TR is warranted. In such cases, comprehensive assessment of the morphology of the valvulo-ventricular complex using 3DE (Figure 4) may provide important clues regarding aetiology and mechanisms of valve dysfunction.35–38

Functional TR is characterized by TA dilatation (often >40 mm) and may also involve tethering of the leaflets, with a tenting distance >8 mm. Tricuspid annulus diameter measured with 2DE (both measured in four-chamber view and in parasternal short-axis view) systematically underestimate the actual TA diameter in comparison with 3DE and cardiac magnetic resonance (CMR).35,39 As a consequence, 65% of patients with normal TA diameter at 2DE showed grade 1–2 TR, compared with 30% of patients with normal TA size at 3DE.40 In most severe cases of TA dilation, central leaflet coaptation may be absent, resulting in wide-open regurgitation (Figure 5).

Colour flow Doppler and spectral Doppler are highly sensitive for TR detection and generally provide good semi-quantitative assessment of its severity.34 Tricuspid regurgitation detection by colour flow imaging uses parasternal (tricuspid inflow view and short-axis view at great vessels level), apical, or subcostal (four-chamber view) approaches. Regurgitant jet area correlates roughly with TR severity, <5 cm$^2$ in mild, 6–10 cm$^2$ in moderate, and >10 cm$^2$ in severe cases. In clinical practice, visual estimate rather than actual planimetry is the basis of TR severity assessment, which may participate to the vagueness of the qualitative approach. Detection of eccentric jets adhering, swirling, and reaching the posterior wall of the right atrium suggests organic rather than functional TR and thereby a rather voluminous regurgitation. Conversely, small thin central jets usually indicate mild TR. Colour Doppler evaluation of TR, despite its simplicity, is a source of errors, is limited by technical and haemodynamic factors and therefore it is not recommended to assess TR severity when it

Figure 4 Visualization of tricuspid valve complex by two- (A–C) and three-dimensional (D–F) echocardiography. Apical four-chamber (A), right ventricular inflow (B), and subcostal (C) views. Volume rendering with the cropping plane corresponding to apical four-chamber view (D), allowing a comprehensive visualization of tricuspid leaflets, as well as of subvalvular apparatus, papillary muscles, and moderator band (arrow); en face view of tricuspid valve from the right ventricle (E) from the right atrium (F). ATL, anterior tricuspid leaflet; PTL, posterior tricuspid leaflet; STL, septal tricuspid.
is more than mild. A more quantitative FTR assessment is provided by vena contracta width and proximal isovelocity surface area (PISA) measurements.

Vena contracta represents the cross-sectional area of the blood column as it leaves the regurgitant orifice; it thus reflects the regurgitant orifice area. The vena contracta of the TR flow is typically imaged in the apical four-chamber view using a careful probe angulation to optimize the flow image, an adapted Nyquist limit (colour Doppler scale, 40–70 cm/s) to identify with clarity the neck of the jet, and a narrow sector scan coupled with the zoom mode to maximize temporal resolution and measurement accuracy (Figure 6). Averaging measurements over at least 2–3 beats is recommended. Vena contracta width >6.5 mm is usually associated to severe TR. Intermediate values are not accurate for distinguishing moderate from mild TR. A limitation of measuring vena contracta width is the fact that regurgitant orifice geometry is complex and not necessarily circular (Figure 6). This may explain the limited correlation between vena contracta width by 2D colour Doppler and 3DE planimetry of effective regurgitant orifice area. Particular caution should be used in assessing eccentric jets. With 3DE colour Doppler, planimetered regurgitant orifice area >75 mm² has been associated with severe TR.

Proximal isovelocity surface area radius measurement is by itself a good indicator of severity of regurgitation, but complete application of the method is preferable to obtain physiological information on TR. The apical four-chamber view and the parasternal long- and short-axis views are classically recommended for an optimal visualization of the PISA. The area of interest is optimized by reducing imaging depth and Nyquist limit to ~10–20% of the peak velocity of the FTR jet. The PISA radius is measured at mid-systole using the first aliasing. Qualitatively, a TR PISA radius >9 mm at a Nyquist limit of 28 cm/s has been associated with the presence of significant TR (corresponding to an effective regurgitant orifice area >40 mm² and a regurgitant volume >45 mL, the quantitative thresholds for severe TR), whereas a radius <5 mm suggests mild regurgitation. However, the PISA method also faces several limitations. Eccentric jets may represent a challenge in alignment with the beam of ultrasound. The most challenging issue is the fact that leaflets that are tenting are often not flat and the outer angle formed by these should be accounted for in the calculation of regurgitant flow. It is also crucial to account for the expected respiratory variation of TR and to average measurements performed during inspiration (largest flow convergence, lowest TR velocity) and expiration (smallest flow convergence, highest TR velocity) to provide an average severity over the respiratory cycle to the clinician (Figure 7). Finally, we have to take into account the extreme load dependency of the tricuspid regurgitant volume. Changes in patient’s position and diuretic therapy are among the most frequent causes of inter-technique and day-to-day variability of severity of TR.

Tricuspid regurgitation jet velocity reflects the pressure gradient between the RV and the right atrium throughout systole. The shape of the TR velocity profile using continuous-wave Doppler provides a clue to TR severity. The regurgitation profile is generally parabolic except in severe cases, where high right atrial ‘C–V’ waves result in a rapid equalization with RV pressure, resulting in a rapid deceleration of the Doppler tracing, also described as the ‘V’ wave cut-off sign (Figure 7). Additional indirect clues of regurgitation severity are the density of continuous-wave Doppler profile, the size of RV and atrium, the paradoxical interventricular septal motion and the systolic bulge of interatrial septum towards the left atrium. In severe FTR, hepatic venous systolic flow reversal is a specific but relatively insensitive sign.

In clinical practice, Doppler echocardiographic assessment of TR integrates all signs and measurements obtained by all methods (Table 2). Finally, a comprehensive echocardiographic assessment of patients with FTR should include measurement of RV size and function. Two-dimensional echocardiography quantitation of RV size and function is challenging, due to the anterior position of the RV in the chest, its complex asymmetric geometry, irregularity of the highly trabeculated endocardial border, impossibility to visualize in the same view both inflow and outflow tracts, and lack of realistic geometric models to use for volume quantitation. Three-dimensional echocardiography has been demonstrated to have a good accuracy in measuring RV volumes compared with CMR.

Cardiac magnetic resonance
In the absence of specific contraindications, when echocardiographic imaging is limited by a suboptimal acoustic window, in
patients with equivocal echocardiographic findings and when 3DE is not available for assessing RV volumes and function, CMR is the technique of choice to assess patients with significant TR. Cardiac magnetic resonance is not limited by acoustic window and can image the whole heart in any plane providing excellent myocardial definition. Cardiac magnetic resonance is currently the gold standard for the assessment of RV morphology and function, and accurate assessment of the RV is crucial to understand the underlying mechanisms and address management in patients with TR even if no threshold value of RV volume has been validated by its prognostic value with regard to TR. Anwar et al. have used CMR to assess the configuration of TA.

Indications and timing of tricuspid valve surgery

In the absence of clinical trials, present guidelines are based on expert opinions. Our practice has evolved to include more surgical treatment of FTR even when it is isolated. The principles guiding surgical indications are as follows:

(A) The surgical indication for FTR is considered more actively if:

- (i) another cardiac operation is considered, whether it is for valve surgery, coronary bypass, or MAZE procedure;
- (ii) the FTR is severe, particularly based on quantitative criteria with $\text{ERO} \geq 40 \text{ mm}^2$;
- (iii) the patient is symptomatic from the TR and there are congestive signs directly related to the TR (enlarged pulsatile liver, with pulsatile jugular veins and systolic reversal in the hepatic veins by echo-Doppler). In the absence of congestive signs, marked reduction of functional capacity measured by exercise testing and without other cause than the TR is essential to consider surgery;
- (iv) the comorbid conditions are not overwhelming and life expectancy is of at least several years.

Thus, for example, it is reasonable to consider correction of a moderate TR without congestion if mitral valve regurgitation requires repair or to consider surgery for severe idiopathic FTR in the context of long-standing atrial fibrillation, with marked right-sided congestion. Conversely, it is less enticing to consider surgery for severe FTR with supra-systemic primary pulmonary hypertension. In any case, patients should be referred for surgery before developing secondary liver cirrhosis or severe RV dysfunction since these conditions identify a very high-risk surgical
There is increasing evidence that patients undergoing mitral valve surgery would benefit from tricuspid annuloplasty when the TA is dilated independent on the severity of TR.\(^6,48,49\)

(B) The type of surgery performed is dependent on the morphology of the FTR:

1. If the FTR is purely due to annular enlargement without tenting and the RV function is normal, usually annuloplasty is preferred;
2. If tenting is the main mechanism of FTR and/or RV dysfunction may not be reversible post-operatively, annuloplasty may be

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**Figure 7** Quantification of regurgitation severity by PISA method in a patient with severe functional tricuspid regurgitation. Both colour Doppler images (A and B) and continuous-wave Doppler tracings of regurgitant jet (C) show high respiratory variability stressing the need to average measurements performed during inspiration (largest flow convergence, lowest TR velocity) and expiration (smallest flow convergence, highest TR velocity) to provide an average severity over the respiratory cycle. The severity of the tricuspid regurgitation is also shown by the continuous Doppler signals of relatively low-amplitude, with ’cut-off’ sign (white arrow).

**Table 2** Echocardiographic assessment of tricuspid regurgitation severity (modified from Lancellotti et al. \(^34\))

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
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<tbody>
<tr>
<td>Qualitative</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tricuspid valve morphology</td>
<td>Normal/abnormal</td>
<td>Normal/abnormal</td>
<td>Abnormal/flail/large coaptation defect</td>
</tr>
<tr>
<td>Colour flow TR jet</td>
<td>Small, central</td>
<td>Intermediate</td>
<td>Very large central jet or eccentric wall impinging jet</td>
</tr>
<tr>
<td>CW signal of TR jet</td>
<td>Faint/parabolic</td>
<td>Dense/parabolic</td>
<td>Dense/triangular with early peaking (peak &lt; 2 m/s in massive TR)</td>
</tr>
<tr>
<td>Semi-quantitative</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VC width (mm)</td>
<td>Not defined</td>
<td>&lt; 6.5</td>
<td>&gt; 6.5</td>
</tr>
<tr>
<td>PISA radius (mm)</td>
<td>( \leq 5 )</td>
<td>6–9</td>
<td>&gt; 9</td>
</tr>
<tr>
<td>Hepatic vein flow</td>
<td>Systolic dominance</td>
<td>Systolic blunting</td>
<td>Systolic flow reversal</td>
</tr>
<tr>
<td>Tricuspidinflow</td>
<td>Normal</td>
<td>Normal</td>
<td>E wave dominant (( \geq 1 ) cm/s)</td>
</tr>
<tr>
<td>Quantitative</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>EROA (mm(^2))</td>
<td>Not defined</td>
<td>Not defined</td>
<td>( \geq 40 )</td>
</tr>
<tr>
<td>R Vol (ml)</td>
<td>Not defined</td>
<td>Not defined</td>
<td>( \geq 45 )</td>
</tr>
<tr>
<td>+ RA/RV/IVC dimension</td>
<td></td>
<td></td>
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</table>

CW, continuous-wave Doppler; EROA, effective regurgitant orifice area; PISA, proximal isovelocity surface area; RA, right atrium; RV, right ventricle; R Vol, regurgitant volume; TR, tricuspid regurgitation; VC, vena contracta.
### Table 3  Comparison of American College of Cardiology/European Society of Cardiology clinical guidelines for managing patients with tricuspid regurgitation

<table>
<thead>
<tr>
<th>ACC/AHA guidelines</th>
<th>ESC/EACTS guidelines</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Class IB</strong> TV repair is beneficial for severe TR in patients with MV disease requiring MV surgery</td>
<td>Severe TR in a patient undergoing left-sided valve surgery</td>
</tr>
<tr>
<td><strong>Class IC</strong></td>
<td>Severe organic TR and symptoms despite medical therapy without severe right ventricular dysfunction</td>
</tr>
<tr>
<td><strong>Class IIaC</strong> TV replacement or annuloplasty is reasonable for severe primary TR when symptomatic</td>
<td>Moderate organic TR in a patient undergoing left-sided valve surgery</td>
</tr>
<tr>
<td>TV replacement is reasonable for severe TR secondary to diseased/abnormal TV leaflets not amenable to annuloplasty or repair</td>
<td>Mild-to-moderate secondary TR with dilated annulus (&gt;40 mm or 21 mm/m²) in a patient undergoing left-sided valve surgery</td>
</tr>
<tr>
<td><strong>Class IIbC</strong> Tricuspid annuloplasty may be considered for less than severe TR in patients undergoing MV surgery when there is pulmonary hypertension or tricuspid annular dilatation.</td>
<td>Severe TR and symptoms, after left-sided valve surgery, in the absence of left-sided myocardial, valve, or right ventricular dysfunction and without severe pulmonary hypertension (systolic pulmonary artery pressure &gt;60 mmHg)</td>
</tr>
<tr>
<td><strong>Class III</strong> TV replacement or annuloplasty is not indicated in asymptomatic patients with TR whose pulmonary artery systolic pressure is 60 mmHg in the presence of normal MV</td>
<td>Severe isolated TR with mild or no symptoms and progressive dilatation or deterioration of right ventricular function</td>
</tr>
<tr>
<td>TV replacement or annuloplasty is not indicated in patients with mild organic TR</td>
<td></td>
</tr>
</tbody>
</table>

MV, mitral valve; TR, tricuspid regurgitation; TV, tricuspid valve.

### Figure 8  American College of Cardiology/American Heart Association \(^50\) and the European Society of Cardiology \(^51\) guideline-based algorithm for the management of tricuspid regurgitation in patients who have not previously undergone left-sided valve surgery. RV, right ventricular.
associated with excess recurrence of TR and either valve replacement or elongation of anterior leaflet tissue by pericardial patches may be necessary.

Table 3 summarizes the indications for the management of significant TR from the American College of Cardiology/American Heart Association (ACC/AHA) and the European Society of Cardiology (ESC/EACTS). Patient’s clinical status, concomitant left-sided valve surgery, and the aetiology of TR usually determine the appropriate therapeutic strategy in each individual case (Figures 7 and 8).

**Conclusions**

Assessment of FTR is challenging due to the complex anatomy of both the TV and the RV. However, identification of the mechanism of regurgitation, reliable grading of its severity, accurate quantitative data about extent of TV tethering, TA dilatation, and RV enlargement are crucial for selecting patients who may benefit of early surgical repair and tailor the intervention in order to avoid progression to right-heart failure.

Finally, a better understanding of FTR pathophysiology and anatomy will provide the bases for developing percutaneous procedures to treat TR in high-risk surgical candidates.

**References**


