Imaging Evaluation of Tricuspid Valve: Analysis of Morphology and Function With CT and MRI

OBJECTIVE. In this article we show the morphologic detail of the tricuspid valve (TV) apparatus and discuss the spectrum of diagnostic information that CT and MRI can provide regarding pathologic processes. We also compare the strengths and limitations of these modalities with currently established echocardiographic diagnostic parameters.

CONCLUSION. The TV plays an important role in a number of pathologic states, and its abnormality can directly or indirectly influence morbidity and mortality in different cardiac disorders. However, the importance of the role of the TV has been overlooked primarily because TV malfunction may remain less symptomatic for a long time. Along with rapid development in imaging technology, improvement in postoperative management, and better understanding of the pathophysiologic mechanisms of TV dysfunction, more attention is being given to careful imaging analysis of this “forgotten valve.”

The tricuspid valve (TV) plays an important role in a number of pathologic states, and its abnormality may directly or indirectly influence morbidity and mortality in many clinical scenarios involving right or left heart diseases [1, 2]. In this regard, the unique morphology of the TV apparatus plays a crucial role, and understanding anatomic changes of the TV has helped to explain the pathophysiologic mechanisms of different clinical presentations [1]. The primary imaging of choice for the TV is echocardiography. Because tricuspid dysfunction is often associated with other cardiac abnormalities, especially left heart diseases that are a common indication for cardiac MRI or CT, understanding the spectrum of imaging findings in TV abnormalities can improve CT and MRI interpretation and may be crucial for clinical management of patients. In this article, we review the roles of CT and MRI in the assessment of anatomy and morphologic changes of the tricuspid apparatus and describe the important functional parameters of the TV under normal circumstances and in patients with functional and structural TV abnormalities.

Morphology

Normal Tricuspid Apparatus

The septal leaflet of the TV, aortic leaflet of the mitral valve, and atrioventricular fibrous continuity between these valves develop from the mesenchyme of the inferior and superior atrioventricular endocardial cushions [3]. Later, the tricuspid septal leaflet delaminates from the muscular ventricular septum. The mural leaflets, including the anterior and posterior (inferior) leaflets of the TV and the posterior leaflet of the mitral valve, are mainly derived from mesenchymal cushions that arise laterally in the atrioventricular canal after cushion fusion [3–5]. A complete failure of development of the atrioventricular cushions results in atrioventricular canal defect with variable degrees of insufficiency, whereas incomplete resorption of the myocardial tissue on the TV septal leaflet can result in Ebstein anomaly or accessory conduction pathways [5].

The TV apparatus consists of the leaflets, fibrous annulus, and supporting tension apparatus, the latter consisting of the chordae tendinae (tendinous chords) and papillary muscles (PMs) (Fig. 1). The leaflets include anterior (superior), septal, and posterior (inferior) [6, 7]. The PMs include anterior, posterior, and sometimes medial (septal) leaflets. The posterior leaflet appears to be of lesser functional significance because it can be removed without impairment of valve function. The septal leaflet is smaller than the anterior leaflet and arises medially directly from the tricuspid annulus above.
the interventricular septum. The commis-
sure between the septal and anterior leaflets
is located over the membranous septum and
divides it into the atrioventricular and inter-
ventricular components [7, 8] (Fig. 2). The
anterior PM provides chordae to the anterior
and posterior leaflets, and the posterior PM
provides chordae to the posterior and septal
leaflets. The septal PM of the conus or right
ventricular (RV) septal wall provides chord-
ae to the anterior and septal leaflets. It rep-
resents an important surgical landmark for
location of the right bundle branch to avoid
injury during surgical correction of certain
types of ventricular septal defects (VSDs).

Tricuspid Annulus
The normal tricuspid annulus has an ellip-
soidal shape and appears nonplanar, with
the posterosetal portion “lowest” (toward the
apex) [1] (Fig. 3). As a result of this inferior
migration of the septal leaflet, the distance
between the mitral and tricuspid leaflets is
maximum where posterior cardiac crux is lo-
cated [7] (Fig. 2). In this region, also known
as the muscular interventricular septum, the
cavity of the right atrium is separated from
that of the left ventricle by invagination of the
atrioventricular junction myocardial walls,
which are filled with epicardial fibrofatty tis-
sue where the atrioventricular nodal artery
passes. Therefore, this is not a true muscu-
lar atrioventricular septum [8]. An important
consideration regarding the structure of the
TV annulus that makes it different from the
mitral annulus is the lack of extensive fibrous
elements in the peripheral (mural) part of
the valve to support its leaflets [8].

Imaging of the Tricuspid Valve
Apparatus Morphology
Compared with the mitral valve, which can be
optimally evaluated in most routine coro-
ny CT angiography (CTA) studies, vi-
sualization of the right atrioventricular jun-
tion in detail can be challenging. Clear de-
piction of this region requires homogeneous
enhancement of the structures around the TV
annulus. Most ECG gated or triggered car-
diac CTA techniques modified for the right
heart examination (i.e., 50% contrast agent
and saline chucks in routine coronary CTA)
can provide good-quality motion-free imag-
est of the RV outlet and trabeculated portions.
However, the quality may not be high enough
to show the details of the RV inlet [9]. This
is mainly because of inhomogeneous en-
This text contains information about the morphology and imaging of the tricuspid valve. The tricuspid annulus has an ellipsoidal shape and is nonplanar. The location of the right bundle branch can be challenging during surgery. The quality of imaging for the tricuspid valve can be improved with certain techniques. MRI and CT imaging can be useful for assessing the TV annulus size and shape changes. Congenital defects in the tricuspid valve can be detected using these imaging modalities.
stein anomaly is due to failure of delamination of the valve tissue from the underlying myocardium, resulting in a more apical attachment of the posterior and septal leaflets. In Ebstein anomaly, precise delineation of the degree of apical displacement of the posterior and septal leaflets and volumetric and functional assessment of the ventricles are feasible with MRI. CT is an alternative imaging modality, particularly in patients unable to undergo cardiac MRI (Fig. 5).

The displacement of the TV septal leaflet in Ebstein anomaly results in an "atrialized RV" and a "functional RV" [19, 20] (Fig. 5). In contrast, the anterior leaflet of the TV is rarely displaced but is often redundant or "saillike."

Double-orifice TV presents with TR in 62% of cases and combined stenosis and regurgitation in 25% [21]. Cleft on the anterior or TV leaflet usually presents with TR [22]. Dysplastic or cleft TV may be seen in association with perimembranous VSD.

The imaging characteristics of tricuspid atresia include the classic fatty wedge seen in the right atrioventricular groove on axial or long-axis four-chamber views along with a dilated systemic venous system and right atrial and severe RV hypoplasia [23].

In the congenital form of atrioventricular membranous defect (Gerbode defect), an elongated saillike anterior TV leaflet in a perimembranous VSD may partially close the defect or in some cases direct the shunt from the left ventricle into the right atrium. The acquired form of the disease (e.g., bacterial endocarditis) may not show this phenomenon [24, 25]. In aneurysm of the interventricular membranous septum, it is believed that the aneurysm results from adhesion of the septal or anterior leaflets to the rim of a perimembranous VSD, causing complete or incomplete closure of the defect [25]. The characteristic windsock appearance on CT or MRI results from aneurysmal distention during ventricular systole.

**Tricuspid Valve Masses**

Tumors arising from the valve are usually papillary fibroelastoma, commonly involving the aortic valve and rarely the TV [26–28]. The differential diagnoses of TV masses include valve myxoma, thrombus, vegetation, metastasis, and other rare primary benign or malignant tumors [26–30]. On MRI, papillary fibroelastoma usually presents as a hyperintense mobile pedunculated mass on T2-weighted imaging with some enhancement that can be indistinguishable from myxoma or hemangioma. In contrast, thrombus is usually hypointense on T2-imaging and remains low in signal intensity with no enhancement on delayed contrast-enhanced imaging, although the signal intensity may vary depending on the stage of evolution. Hemangioma and angiosarcoma are among the vascular masses with variable enhancing patterns.

**Function**

**Tricuspid Dysfunction**

Appendix 1 shows the causes of TV malfunction classified into two major groups of primary (intrinsic, organic, or structural) and secondary (functional or anatomically normal) diseases. In the secondary group (more common), tricuspid dysfunction usually presents as mild to moderate TR in a patient with left heart disease or pulmonary hypertension and may resolve after treatment of the primary disease. Isolated primary disease of the TV is less common and usually secondary to rheumatic heart disease and CHD. The valve morphology can help differentiate the two groups. Rheumatic disease has been the leading cause for TV dysfunction [31]. This trend has changed over past decades, and the relative frequency of CHD has increased [32]. In all groups, the most common clinical presentation is TR. TV stenosis, especially in its isolated form, is a rare phenomenon and appears to be congenital (bicuspid, dysplastic) in most cases or, less likely, due to endocarditis, rheumatic disease, or carcinoid [21, 31, 33, 34].

**Tricuspid Regurgitation**

The causes of TR are listed in Appendix 2. Trivial to mild TR is common (60–70%) and appears to be more common in patients more than 50 years old [35]. Although little is known about the significance of mild TR, moderate to severe TR has been associated with increased mortality [35, 36].

**Functional tricuspid regurgitation**—The most common cause of TR is dilatation of the TV annulus caused by pulmonary hypertension or secondary to left heart failure [36]. As mentioned earlier, functional TR is associated with flattening of the tricuspid annulus, dilatation, and decreased annular longitudinal excursion during the cardiac cycle (especially in severe TR) [37, 38]. RV dilatation leads to a distortion of the normal geometric relationships of the tricuspid leaflets, chords, and PMs. With annulus flattening, the lowest point of the annulus is displaced away from the PMs, resulting in tethering of the leaflets and regurgitation due to incomplete coaptation of the leaflets. The critical diameter for annular dilatation to cause functional TR is approximately 27 mm/m² of body surface area [37]. In an in vivo study of porcine TVs by Spinelli et al. [39], all valves lost competence at 40% dilatation.

Tethering of the leaflets as a result of annular remodeling and flattening has been reemphasized by Fukuda et al. [38], who showed that after ring annuloplasty, in spite of reduction of the annulus diameters to normal limits, TR can persist and its persistence is primarily related to leaflet tethering. The distance and area of tethering (tenting) can be measured by echocardiography, MRI, or CT (Figs. 6 A and B). In addition to tricuspid dilatation, three important factors determine whether TR occurs: preload, afterload, and right ventricular function. These factors should be taken into consideration in the imaging and clinical study of TR [40].

**Pulmonary hypertension and tricuspid regurgitation**—Pulmonary hypertension increases RV systolic pressure and may lead to RV dilatation and dysfunction, TR annulus dilatation, PM displacement, tethering of the leaflets, and finally TR. Chronic TR, in turn, contributes to progression of the RV volume overload and right atrial enlargement, which exacerbates RV and tricuspid annular dilatation and can reduce RV systolic function and cardiac output [41] (Figs. 6 D–E).

Among the different causes of pulmonary hypertension and secondary TR, mitral valve disease is of special significance. Moderate TR may be present in up to 30% of patients with mitral valve disease [42]. Over time, significant TR recurs after mitral valve surgery in many patients; this entity has been termed "late TR." Late TR is associated with a very poor prognosis [43]. Once the tricuspid annulus is dilated, its size does not return to normal and may even dilate further, requiring a second operation years after the initial mitral valve repair [40]. That is why almost half of patients who undergo mitral valve repair also undergo tricuspid annuloplasty and a maze procedure if atrial fibrillation exists [40, 44].

**Structural causes of tricuspid regurgitation**—Rheumatic heart disease remains a major origin of heart valve disease in the developing world [45]. The mitral valve is most often involved [46]. TV involvement is seen in 6.0–8.5% of cases, and TR develops in two thirds of those patients. In one third of patients, TR
is functional [47, 48]. Imaging criteria include thickened leaflets with restriction in motion, diastolic doming (hemispheric shape of the leaflets due to adhesion), and encroachment of the leaflet tips on the ventricular inlet [48].

In carcinoid syndrome, the TV is the most frequently involved valve (97%), followed by the pulmonary valve (49%). Endocardial plaques of fibrous tissue on both sides of the TV leaflets and subvalvular apparatus result in moderate to severe regurgitation in 90% and TV stenosis in 10% of cases [49, 50]. Endocardial fibrous plaques in the RV and its valves can be visualized with cardiac MRI on delayed contrast-enhanced imaging [50, 51].

Prolapse is diagnosed if one or more leaflets extend beyond the tricuspid annulus plane in systole (Fig. 7). Myxomatous degeneration and Marfan syndrome affecting the mitral and tricuspid valves can lead to chordal elongation and prolapsing leaflets [52]. TV prolapse is seen in 20% of cases of myxomatous mitral valve disease and primarily involves the anterior and septal leaflets. Determination of tricuspid prolapse is difficult because the tricuspid ring is not easy to define.

Infective endocarditis of TV is seen in 5–10% of infective endocarditis and can involve a native TV or valve prosthesis [53]. TV endocarditis is mainly a disease of IV drug abusers. CT has limited value for detection of small vegetations (< 4 mm) and small valve perforations [54, 55]. Associated findings include thickening, shortening, perforations, or complete destruction of the leaflets and intramural or perivalvular abscesses. TR can rarely develop after inferior wall myocardial infarction and appear to be primarily related to the dysfunction of the PM complex [56]. Patients with a permanent pacemaker or automatic implantable cardioverter-defibrillator leads have an increased prevalence of significant TR [57]. After lead implantation, 18% of patients with baseline mild TR develop moderate to severe TR. TR can be caused by direct lead interference with valve closure, laceration or perforation, infection, and fibrous adhesions.

**Imaging of Tricuspid Function**

Table 1 shows the imaging parameters for assessment of TR using different modalities. MRI evaluation of TV function is performed in a manner analogous to that in the mitral and aortic valves, but in some cases it can be difficult because of more extensive excursion of the valve annulus during the cardiac cycle unless it is corrected for motion [58]. In general, gradient-recalled echo (GRE) pulse sequences are more sensitive to dephasing effects than balanced steady-state free precession (SSFP). Therefore, the jet of regurgitation, especially when it is mild, can be better shown by GRE imaging. Velocity mapping and flow quantification by phase contrast MRI enable direct measurement of the regurgitant flow [59]. On in-plane balanced SSFP or phase contrast cine imaging, the regurgitant flow is typically shown as a triangular jet into the right atrium. Obtaining a short-axis image orthogonal to the jet flow can optimally provide information about the velocity and direction of flow (Fig. 8). Short-axis slices will be obtained at three levels: above, at, and below the valve. In most cases, a velocity encoding (VENC) value of < 200 cm/s is enough to prevent aliasing.

In severe stenosis, higher VENC values are required. Quantification of regurgitation is possible by measuring peak systolic velocity (PSV) across the valve. Quantification of regurgitation is performed by measuring retrograde flow across the valve and dividing this measure by the anterograde flow, thus obtaining the regurgitant fraction. PSV is used to quantify the pressure gradient and thus to estimate RV systolic pressure. The pressure difference (gradient) between the right atrium and the RV is calculated by the Bernoulli equation: $\Delta p = 4. (PSV)^2$. The addition of right atrial pressure (7–10 mm Hg) to the $\Delta p$ gives an estimate of the RV systolic pressure that is equal to the systolic pulmonary artery pressure (if there is no pulmonary valve stenosis) in mm Hg [60].

The regurgitant orifice area can be directly evaluated on short-axis through-plane views. The short plane of the TV is different from the mitral valve and can be best scanned using two orthogonal long-axis RV views. This method is difficult in Ebstein anomaly cases because of the difficulty of identifying the valve level on short-axis images [61]. The 3D three-directional acquisition method with retrospective valve tracking is currently the best technique to overcome the limitations of the 2D technique and to correct for valve motion in the apical-basal direction. Using this technique, velocity encoding in three orthogonal directions is applied and a free-breathing 3D acquisition is performed in approximately 5 minutes with an additional 5 minutes for data processing [62].

An alternative method for calculating tricuspid regurgitant volume is to subtract RV stroke volume calculated by balanced SSFP cine images from forward stroke vol-

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**TABLE 1: Noninvasive Imaging Parameters for Assessment of Tricuspid Regurgitation**

<table>
<thead>
<tr>
<th>Mode</th>
<th>Parameters</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
<th>Imaging</th>
</tr>
</thead>
<tbody>
<tr>
<td>Qualitative</td>
<td>Tricuspid morphology</td>
<td>Normal nonplanar</td>
<td>Nonplanar, tethered</td>
<td>Planar with large coaptation defect</td>
<td>CT, MRI, 3D echocardiography</td>
</tr>
<tr>
<td></td>
<td>TR jet</td>
<td>Small central</td>
<td>Intermediate</td>
<td>Large central or eccentric jet</td>
<td>MRI, echocardiography</td>
</tr>
<tr>
<td></td>
<td>Hepatic vein inflow</td>
<td>Systolic dominance</td>
<td>Systolic blunting</td>
<td>Systolic reversal</td>
<td>MRI, echocardiography, CT</td>
</tr>
<tr>
<td></td>
<td>Tricuspid inflow</td>
<td>Normal pattern</td>
<td>Normal pattern</td>
<td>Early systole wave dominant &gt; 1 m/s</td>
<td>Echocardiography, MRI</td>
</tr>
<tr>
<td>Quantitative</td>
<td>Regurgitant area (cm²)</td>
<td>&lt; 5</td>
<td>6–10</td>
<td>&gt; 10</td>
<td>MRI, echocardiography</td>
</tr>
<tr>
<td></td>
<td>TR length (cm)</td>
<td>2</td>
<td>3–5</td>
<td>&gt; 5</td>
<td>MRI, echocardiography</td>
</tr>
<tr>
<td></td>
<td>TR (%)³</td>
<td>20</td>
<td>20–34</td>
<td>&gt; 35</td>
<td>MRI, echo</td>
</tr>
<tr>
<td></td>
<td>Vena contracta width (mm)</td>
<td>Not defined</td>
<td>&lt; 6.5</td>
<td>&gt; 7</td>
<td>MRI, echocardiography</td>
</tr>
<tr>
<td></td>
<td>PISA radius (mm)</td>
<td>&lt; 5</td>
<td>6–9</td>
<td>&gt; 9</td>
<td>Echocardiography</td>
</tr>
<tr>
<td></td>
<td>Regurgitant orifice area (mm²)</td>
<td>Not defined</td>
<td>Not defined</td>
<td>&gt; 40</td>
<td>CT, MRI, 3D echocardiography</td>
</tr>
<tr>
<td></td>
<td>Regurgitant volume (mL)</td>
<td>Not defined</td>
<td>Not defined</td>
<td>&gt; 45</td>
<td>Echocardiography, MRI</td>
</tr>
<tr>
<td></td>
<td>RA, RV, IVC dimensions</td>
<td>Nonspecific</td>
<td>Nonspecific</td>
<td>IVC &gt; 2 cm</td>
<td>CT, MRI</td>
</tr>
</tbody>
</table>

Note—PISA = proximal isovelocity surface area, RA = right atrium, RV = right ventricle, IVC = inferior vena cava [1, 2, 59,70].

³Ratio of maximal regurgitant area to RA area.
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and the right atrial area (Fig. 9). However, this method is semiquantitative and rather subjective. Direct measurement of the regurgitant area on cine MRI has also been suggested as a reliable method of regurgitation severity analysis on balanced SSFP images (severe > 0.92 cm²) [68]. Values are smaller using phase imaging compared with balanced SSFP.

A more quantitative assessment of TR can be obtained by measuring the width of the vena contracta, which is measured as the narrowest neck of regurgitant flow near the regurgitant orifice and before expansion of the turbulent jet [69]. The vena contracta is typically measured in the four-chamber view, and planimetry of the maximal area of the regurgitant jet or the regurgitant orifice is performed on short-axis images (Fig. 9). Width of the vena contracta greater than 7.0 mm is an additional indicator of severe regurgitation [69, 70]. Intermediate values are not accurate for distinguishing moderate from mild TR. The method is not accurate in assessing eccentric jets.

Tricuspid Valve Surgery

Two common types of tricuspid valve repair include suture annuloplasty using a “purse-string” suture and ring annuloplasty of Carpentier (26- to 36-mm open) [71] (Fig. 10). Annuloplasty with a rigid or flexible ring has better durability compared with purse-string suture and is considered the standard of care. The ring is C-shaped, and its gap is placed at the site of the septal leafllet of the TV to prevent complications to the conduction system (Fig. 10). Posterior annular bicuspidization is another form of annuloplasty that is not commonly used. Any patient with moderate TR or a tricuspid annular diameter greater than 40 mm in any imaging view is considered for TV annuloplasty during any left-sided valve surgery [72]. TV annulus is surgically reduced to 3.0 cm or by at least 20–30% from the values. If the functional TR is purely due to annular enlargement without tenting and the RV function is normal, usually annuloplasty is preferred [38]. Tenting is described as displacement of the TV leafllet tips toward the RV in systole because of cord tethering (Fig. 6). Tenting distances greater than 0.76 cm and areas greater than 1.63 cm² are important predictors of persistence or recurrence of functional TR after annuloplasty, and either valve replacement or elongation of anterior leafllet tissue by pericardial patches may be necessary [38, 73]. Tenting is also seen in pulmonary hypertension or increased afterload (i.e., systemic ventricle) (Fig. 6).

Conclusion

The TV plays an important role in a number of pathologic conditions and its abnormality can influence the morbidity and mortality in a number of acquired and congenital cardiac abnormalities. In this article, we have reviewed the morphologic details of the TV apparatus and discussed the spectrum of diagnostic information that CT and MRI can provide regarding its pathologic processes and compared the strengths and limitations of these modalities with currently established echocardiographic diagnostic parameters.

References


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APPENDIX 1: Causes of Tricuspid Valve Dysfunction

A. Primary
- Rheumatic valve disease
- Endocarditis: infective, hypereosinophilic syndrome
- Carcinoid heart disease
- Toxic: fenfluramine, methysergide
- Tumors: myxoma, metastasis
- Iatrogenic: pacemaker lead trauma
- Trauma: blunt or penetrating injuries
- Degenerative (valve prolapse)
- Congenital: atresia, hypoplasia, Ebstein

B. Secondary
- Right ventricular dilatation
- Pulmonary hypertension
- Global right ventricular dysfunction: cardiomyopathy, myocarditis,
- Segmental right ventricular dysfunction: ischemia/infarction, endomyocardial fibrosis, arrhythmogenic right ventricular dysplasia

APPENDIX 2: Causes of Tricuspid Regurgitation (TR)

A. Functional (secondary) (75%)
1. Right ventricular dysfunction
   - Left heart disease
   - Right ventricular dysplasia
   - Ischemic right ventricle
   - Systemic right ventricle
2. Increased afterloada
   - Primary pulmonary hypertension
   - Secondary pulmonary hypertension
   - Left ventricular dysfunction
   - Mitral valve disease
   - Chronic obstructive pulmonary disease
   - Thromboembolism
   - Left to right shunt
   - Systemic RV
3. Others
   - Physiologic
   - Atrial fibrillation
   - Right atrial tumors

B. Structural (25%)
1. Acquired
   - Rheumatic heart
   - Prolapse
   - Endocarditis
   - Infective, Löffler syndrome
   - Endomyocardial fibrosis
   - Carcinoid
   - Tumors
   - Traumatic
   - Papillary muscle dysfunction
   - Iatrogenic
   - Pacemaker
   - Right ventricular biopsy
   - Drugs, radiation
2. Congenital
   - Ebstein anomaly
   - Dysplasia
   - Leaflet cleft
   - Hypoplasia
   - Double orifice
   - Unguarded orifice
   - Bicuspid

aLeft heart dysfunction is the most common cause of pulmonary hypertension, associated right heart failure, and TR.
bLead implantation.

(Figures start on next page)
Fig. 1—Tricuspid valve (TV) apparatus. A and B, Photographs obtained from cadavers show that TV has largest orifice among four cardiac valves. Anterior (mural) leaflet is largest (A). Septal leaflet base inserts obliquely across membranous interventricular septum where anteroseptal commissure is located (asterisks, A). Inferior (posterior) leaflet has more scallops and is relatively difficult to see with echocardiography compared with CT and MRI. TV papillary muscles (B) are smaller than mitral apparatus, often multiple, and widely separated. AA = ascending aorta, MV = mitral valve, RCA = right coronary artery, LCx = left circumflex artery, PV = pulmonary valve, MPA = main pulmonary artery, AVNa = atrioventricular node artery.

Fig. 2—Cadaveric images of 45-year-old man who had nonspecific chest pain. A–C, Horizontal long axis (A) and CT angiography (B) images obtained through atrioventricular junction septum at three levels, with corresponding levels of slices shown on right heart view of ventricular septum (C). Septal leaflet of tricuspid valve (STV) divides membranous septum (mbS) into two parts: interventricular and atrioventricular. Inferior to membranous septum is anatomic location of "muscular" atrioventricular septum (AVS). Section below that, above coronary sinus (CS) ostium, is where atrioventricular node (AVN) resides. Note offsetting of attachments of mitral and tricuspid valves (TVs) at level of muscular AVS. RBB = right bundle branch, LBB = left bundle branch, MV = mitral valve, PM = papillary muscle, IVS = interventricular septum, IAS = interarterial septum, His = bundle of His, CFB = central fibrous body.
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Fig. 3—32-year-old woman with transposition of great arteries after Mustard procedure for tricuspid valve (TV) annulus. A and B, Color-coded volume rendered four-chamber (A) and superior (B) CT images show normal tricuspid annulus in this patient with systemic right ventricle (RV) demarcated by green ring. Normal nonplanar shape due to oblique attachment of septal (sep) leaflet to septum is well preserved. TV annulus is generally weaker compared with mitral annulus and therefore dilates more easily with pressure or volume overload. 1 and 2 = septal attachment, 3 = lateral (Lat) wall, Post = posterior/inferior attachment, Ant = anterior/superior attachment.

Fig. 4—Shape of tricuspid annulus in healthy 34-year-old man and in 32-year-old woman with functional tricuspid regurgitation (TR) who had right heart enlargement. In normal condition (left), annulus appears oval shaped and is usually 30% longer in medial to inferolateral (double-headed red arrows). In functional TR (right), dilatation primarily involves right ventricle free wall (green double-headed arrows) and annulus becomes rounded. Septal portion of tricuspid annulus (double-headed yellow arrows) remains relatively fixed. Normal tricuspid valve annulus is dynamic, and up to 30% reduction in annular area in atrial systole can occur.

Fig. 5—Color-coded CT angiography in 45-year-old woman with Ebstein anomaly. A, Four-chamber volume rendered and corresponding 2D (inset) views show concept of atrialized right ventricle (aRV) and functional RV (fRV). Mitral valve (Mv) and tricuspid valve (Tv) are shown in blue. Expected tricuspid annular plane is shown by dashed white line. RA = right atrium. B, On inferior surface rendered view, green bands demarcate peripheral attachments of Mv and displaced Tv. Septal and inferior leaflets of Tv are displaced apically, resulting in aRV and fRV. Expected tricuspid annular plane is shown by dashed white line. Dysplastic leaflets and dilated atrioventricular ring both contribute significantly to malfunctioning of Tv and resultant right heart enlargement as was seen in this patient. Of note, anatomically normal Tv typically shows < 8 mm/m² apical displacement relative to hinge point of mitral valve. LV = left ventricle.
Fig. 6—Tricuspid leaflet tenting. A and B, Distance of tricuspid valve (TV) tethering is measured from annulus plane to coaptation point (dashed red line) and tethering area by tracing leaflets from annulus plane on four-chamber (dashed yellow line) (A) or two-chamber (B) images. In this 40-year-old man with hypertrophied systemic right ventricle (RV), tricuspid annulus diameter was normal (30 mm in systole) but with abnormal tenting (10 mm). Tenting distance > 0.76 cm and area > 1.63 cm² are important parameters for predicting residual tricuspid regurgitation (TR) after tricuspid annuloplasty. This phenomenon is also seen in pulmonary hypertension or systemic right ventricle.

C–E, Ancillary findings in severe tricuspid regurgitation due to pulmonary hypertension in 39-year-old woman with history of atrial septal defect. Four-chamber (C) and short-axis (D) CT images show specific signs of TR, lack of coaptation of leaflets (green arrow, C), and annulus diameter of > 40 mm. Nonspecific signs include straightening of septum (orange arrows, D), atrial septal leftward bulging (G), and markedly dilated right atrium (RA). Enlarged pulmonary artery (PA) and mild pulmonary regurgitation (PR) (red arrow, E) are also related to pulmonary hypertension.
Fig. 7—Myxomatous disease of tricuspid valve. A and B, Four-chamber MR image (A) shows mild disease in 32-year-old man, and right ventricle (RV) two-chamber CT image (B) in 21-year-old woman shows moderate disease. Prolapse is diagnosed if one or more leaflets extend beyond tricuspid annulus in systole and usually involves anterior (A) and septal (S) leaflets. Visualization of tricuspid prolapse may be difficult because tricuspid ring is not easy to define. Confirmation in two perpendicular images improves diagnosis. I = inferior leaflet.

Fig. 8—48-year-old man with moderate functional tricuspid regurgitation (TR) and diastolic dysfunction. Pulmonary valve function was normal. Both atria are dilated. Note enlargement of atrial contraction waveform, indicating impaired ventricular relaxation and mild dyssynchrony between right and left ventricles reflected by early onset and peak of tricuspid valve (Tv) diastolic forward flow compared with mitral valve (Mv) possibly due to TR.

Fig. 9—Vena contracta (VC) shown by cine MRI in 48-year-old man with moderate to severe tricuspid regurgitation (TR). A and B, Thickened noncoapting leaflets are shown on short-axis image (A). VC is smallest regurgitant flow diameter (area) before expansion of jet, best measured on four-chamber view (B). It is great quantitative assessment of functional TR and corresponds hydrodynamically with regurgitant orifice area (blue dotted line). Severe TR is defined as TR jet area > 10 cm², ratio of jet-right atrium (RA) area > 35%, regurgitant orifice area > 40 mm², and VC > 7 mm (arrows). RV = right ventricle.
Fig. 10—Surgery in functional tricuspid regurgitation (TR).

A, Short-axis CT image obtained at level of atrioventricular node (arrow, AVN) (red) shows relationship of annuloplasty ring (blue) to AVN.

B, Drawings show annuloplasty types. Standard of care (due to durability compared with purse-string suture) is rigid or flexible ring, which reduces annular size and constricts it to predetermined size. It is preferred if tricuspid regurgitation (TR) is due to pure annular enlargement without tenting and normal right ventricle (RV) function. Posterior annular bicuspidization is pledget-supported mattress suture from anteroposterior commissure to posteroseptal commissure along posterior annulus. S = septal tricuspid leaflet.

C, Short-axis CT image shows tricuspid valve annuloplasty and mitral valve replacement. Open ring spares AVN, thus reducing risk of heart block. Note clear membranous septum (arrow) region, approximate site of AVN.