



Specific Anatomic Considerations for Tricuspid Interventions

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Abstract

Purpose of Review This review discusses the normal anatomy and pathology of the tricuspid valve (TV) and right side of the heart. Emphasis is on those anatomic and pathologic features relevant to interventions intended to restore normal function to the TV in disease states.

Recent Findings TV pathology is less common than aortic and mitral valve pathology, and treatment and outcomes for interventions face considerable hurdles. New innovations and early data showing safety and efficacy in transcatheter interventions have transformed TV interventions into the next frontier in cardiac valve disease treatment. Certain features of the TV and right heart have presented themselves as potential targets, as well as impediments, for TV intervention.

Summary The causes of TV pathology and the anatomy of the TV and right heart bring unique challenges to intervention. Approaches to intervention will continue to progress and change the way we view and treat TV pathology.

Keywords Tricuspid regurgitation · Tricuspid interventions · Tricuspid valve anatomy · Tricuspid valve surgery · Tricuspid stenosis · Transcatheter intervention

Introduction

Interventions for tricuspid valve (TV) pathology have historically trailed behind those for the aortic and mitral valves. While the first aortic valve intervention took place in 1912, the first tricuspid valve intervention occurred a full four decades later in 1954 [1•]. Several factors contributed to the status of the TV as the “forgotten” valve. When tricuspid regurgitation (TR) was first described in 1837, it was concluded that “the tricuspid valve was designed to be (come) incompetent.” [2, 3] Additionally, clinically significant TV pathology is uncommon, with an estimated 1.6 million people in the USA suffering from severe tricuspid regurgitation (TR), and

even fewer suffering from tricuspid stenosis (TS) [4, 5]. In 2002, the first transcatheter aortic valve replacement took place. Right around this time, it became increasingly recognized that TR was an independent risk factor for morbidity and mortality [6–9]. This led to a renewed interest in interventions for tricuspid valve pathology, and the first TV transcatheter interventions in humans were performed in 2010 and 2011 [1•, 10–12]. Numerous emerging transcatheter techniques and devices are now under investigation with promising early results.

While percutaneous transcatheter interventions are an exciting new chapter of TV intervention, a discussion regarding the current surgical interventions is a necessary companion. It is important to note that clinical features, patient preference, and clinician experience play a vastly important role as well, and that in this new frontier, there is still significant variability in terminology and clinical criteria when navigating TV interventions [13, 14].

The anatomy of the right heart and TV is unique and poses several obstacles to interventions not encountered in the aortic and mitral valve. Herein, we will focus on anatomic considerations that factor into indications and contraindications for both surgical and transcatheter interventions, as well as the anatomic features that act as impediments and potential complications for said interventions.

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Cardiac Embryology

To understand TV pathology, its subsequent interventions, and what makes it more complicated than other valve interventions, a basic understanding of cardiac embryologic development is useful. Any perturbation during development, and any acquired deficiency in any component of the TV, can cause significant dysfunction.

The vascular system of the human embryo begins to develop during the third week of gestation. At days 16 to 18, progenitor heart cells migrate from the epiblast into the splanchnic layer and form the primitive heart field, a horseshoe shaped collection of cells which will develop into the atria, the left ventricle, and a portion of the right ventricle (RV). The remainder of the RV and the outflow tracts develop from the secondary heart field, which arise later, at days 20 to 21. The cells of the primary and secondary heart fields then form the cardiac tube, which differentiates into the sinus venosus, primitive atrium, primitive ventricle, bulbus cordis, and truncus arteriosus. Through a complex process of folding and fusing and progression and regression of various structures, the cardiac tube assumes its anatomically normal configuration by about the eighth week of gestation. The embryological structures and their subsequent anatomic structures are provided in Table 1.

The development of the sinus venosus deserves recognition, both for its complex development and subsequent regression of

several structures, and because it becomes a significant portion of the right atrium. The paired vitelline/omphalomesenteric veins, umbilical veins, and common cardinal veins drain into the left and right sinus horns, which converge onto the sinus venosus, draining into the atrium at the sinuatrial junction. The sinuatrial junction is flanked by the right and left venous valves, which fuse at the septum spurium. Eventually, the left sinus horn involutes, and the right sinus horn enlarges and becomes incorporated into the right atrium (RA) (giving rise to the smooth walled sinus venarum portion). When this incorporation occurs, the left venous valve and septum spurium normally fuse with the atrial septum, the superior portion of the right venous valve regresses, and the inferior portion of the right venous valve will develop into the valve of the inferior vena cava (IVC) and the valve of the coronary sinus. The left atrium, in contrast, develops primarily from the primitive atrium and its outpouching which develops into the pulmonary veins, without the complicated process of vessel and valve development and regression seen on the right side [15].

Cardiac Anatomy

Understanding the limitations and impediments to TV interventions requires knowledge of the valve itself, the subvalvular apparatus, and particular features of the right heart.

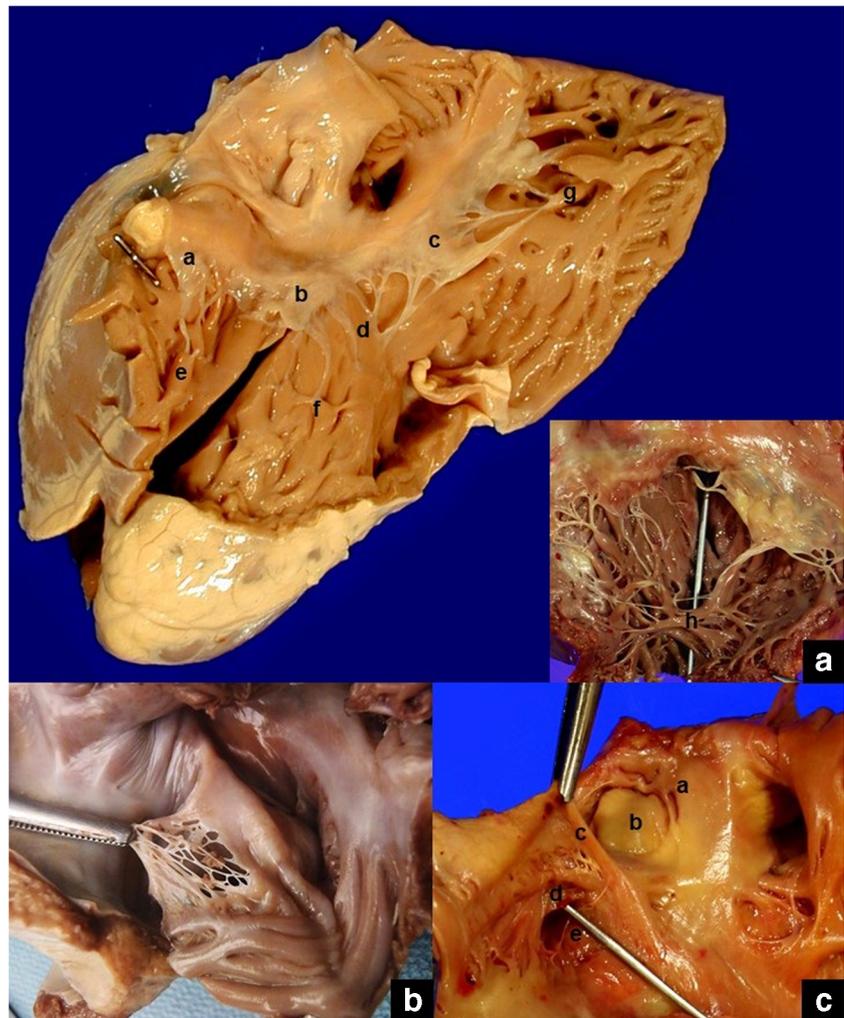
Table 1 Embryologic structures and their subsequent normal anatomic counterparts

| Embryologic structure | Corresponding anatomic structure |
|---------------------------------------|---|
| Sinus venosus | Superior vena cava Inferior vena cava Sinus venarum portion of RA Eustachian valve Thebesian valve Chiari network (anatomic variant) |
| Primitive atrium | Trabeculated portion of RA/RA appendage Portion of interatrial septum Left atrium/Trabeculated LA appendage Pulmonary veins/Smooth portion of LA |
| Atrioventricular endocardial cushions | Portion of interatrial septum Membranous ventricular septum Atrioventricular canals and valves |
| Primitive ventricle | Right ventricle Muscular ventricular septum Left ventricle |
| Bulbus cordis | |
| Proximal portion | Trabeculations of right ventricle |
| Conus cordis | Outflow tract of both ventricles |
| Conotruncal endocardial cushion | Aorticopulmonary septum (creating the aortic and pulmonary channels) |
| Truncus arteriosus | Semilunar aortic and pulmonary valves Roots and proximal portions of great arteries |

Systemic Venous Return and Right Atrium

The right atrium (RA) receives blood flow primarily from the superior vena cava (SVC), IVC, and the coronary sinus. The Thebesian veins also empty into the right atrium and ventricle [16]. The SVC and IVC both have large diameters, with considerable variability between individuals. Furthermore, the convergence of the hepatic veins with the IVC occurs very close to the RA. The RA is divided into a smooth posterior and trabeculated anterior surface with pectinate muscles, separated by a raised C-shaped rim of muscle, the crista terminalis. Three valves are apparent in the RA: the Eustachian (valve of the IVC), Thebesian (valve of the coronary sinus), and valve of the fossa ovalis (with its associated protuberant limbus). One consequence of abnormal embryologic development, while uncommon, is failure of the valves of the sinus venosus to regress. In these circumstances, a meshwork of fibrous connections can remain in the RA, forming Chiari networks, which have been described in up to 6% of people in autopsy series (Fig. 1) [17–19].

Fig. 1 Anatomic features of the right heart. (a) Tricuspid valve and right ventricle: (a) posterior leaflet, (b) septal leaflet, (c) anterior leaflet, (d) chordae with septal attachments, (e) posterior papillary muscle, (f) septal papillary muscle, (g) anterior papillary muscle. Inset: Apical portion of right ventricle demonstrating coarse trabeculations, with probe passing under (h) septomarginal trabeculation/moderator band and exiting through right ventricular outflow tract. (b) Chiari network. (c) Right atrium: (a) limbus, (b) fossa ovalis, (c) valve of inferior vena cava, (d) valve of coronary sinus, (e) probe passing through coronary sinus



Tricuspid Valve

Annulus: The TV is situated within an elliptical, nonplanar annulus. The antero-septal and postero-septal commissures are more apically located, while the region that supports the anterior leaflet is atrially located. The annular ring is largely composed of flexible fibroadipose tissue. This feature allows the geometry of the annulus to change during the cardiac cycle, such that during diastole it is rounder, and during systole as the interventricular septum bulges into the RV, it becomes more elliptical. The TV has a larger orifice than the mitral valve, ranging from 10 to 11.8 cm in circumference (direct open measurements of deceased patients, non-physiologic state). Limited information about the valve annulus size is available through echocardiographic assessment in living humans, though recent data are consistent and suggests that at end-diastole, the tricuspid annulus average diameter is the same size or larger than the mitral valve annulus. The mitral valve in contrast has a saddle shape and is situated in a planar, rounder annulus, with a dense collagenous skeleton. [18, 20, 21, 22•].

Leaflets: As implied by the name, the TV is classically (and over-simplistically) described as having three thin leaflets. In truth, there is substantial variability in the configuration of the leaflets TV. As many as two to six leaflets have been described, and the anatomy is so variable to have been described as “unique to each individual as one’s fingerprint.” [23, 24] As a result, the three classical leaflets suffer from overlapping, redundant, and confusing nomenclature. The classic three leaflets include anterior (anterosuperior/infundibular/mural), posterior (inferior/marginal/mural), and septal (medial). The anterior leaflet is the largest and most consistently present. The posterior leaflet is intermediate in size and is the least consistent. The septal leaflet is the smallest, with intermediate consistency (Fig. 1) [18, 20, 25].

Subvalvular Apparatus

Chordae tendinae: As chordae typically connect leaflets to papillary muscles, the arrangement of these attachments is as varied as that seen with the papillary muscles. However, one of the consistent and defining features of the morphologically RV is the direct attachment of chordae of the septal leaflet to the interventricular septum (Fig. 1).

Papillary muscles: Papillary muscles stabilize the position of the leaflets through their chordal attachments. Arranged perpendicularly to the atrioventricular ring, the papillary muscles contract during systole and draw the annulus and leaflets toward the apex. Though just as variable as the leaflets, the RV is classically described as containing three papillary muscles: anterior, posterior (inferior), and septal (muscle of Lancisi/muscle of Luschka/conal/medial) [18, 26, 27]. The anterior is the most consistently present and often has a single belly. The posterior is the least consistent and often has multiple bellies. The septal shows intermediate consistency (Fig. 1) [28].

Adjacent Structures

There are numerous structures close to the TV, which may complicate interventions. The aortic sinus of valsalva is in close proximity to the TV annulus. Just superficial and anterior to the TV lies the right coronary artery and its nodal branch. The conduction system also travels adjacent to the valve. The atrioventricular (AV) node occupies the space at the base of the atrial septum and is delimited in part by the annulus of the TV (Fig. 2). The bundle of his crosses the annular attachment of the septal leaflet and then travels within the membranous ventricular septum [18].

Right Ventricle

The RV is the most anterior chamber of the heart, situated posterior to the sternum. It has complex geometry; triangular and crescentic in shape, it appears to wrap around the more circular left ventricle. The inlet and outlet portions of the RV are isolated,

separated by the crista supraventricularis. The endocardial surface contains coarse trabeculations. The lumen of the RV has several protruding muscular structures, including the septomarginal trabeculation/moderator band, and the aforementioned papillary muscles (Fig. 1). The RV also has a thin free wall (0.3 to 0.5 cm in thickness, at autopsy). In contrast, the left ventricle has much finer trabeculations on the endocardial surface, only two distinct papillary muscles, and a much thicker wall (up to 1.5 cm in thickness, at autopsy). [18, 20, 29]

Tricuspid Valve Pathology: Anatomic Considerations and Interventions

The TV presents special problems for repair. Perhaps because the blood flow in the right heart is under low pressure, thrombosis of prosthetic devices is a much greater problem than with replacement of left-sided valves. Also, the TV orifice is very oblique, while prosthetic and bioprosthetic devices designed for left-sided valve replacement have a round fixation ring.

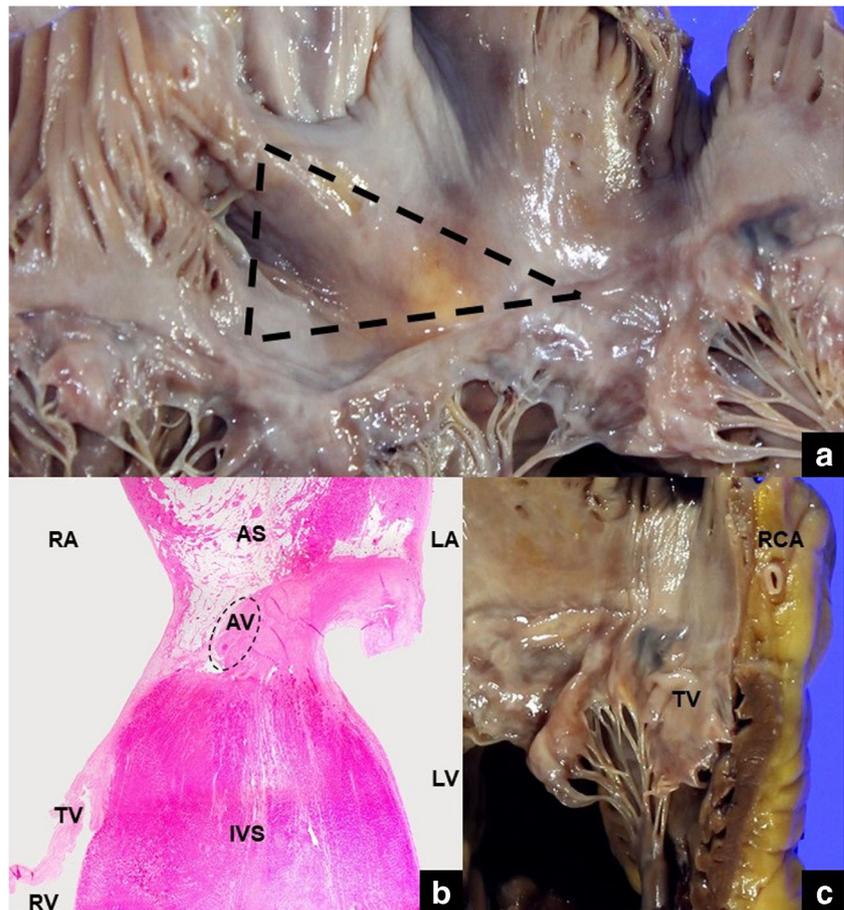
Tricuspid Stenosis

Tricuspid stenosis (TS) is an uncommon valve disease, and most commonly presents in the context of rheumatic heart disease (RHD). Because of its strong association with RHD, it is found less commonly in industrialized countries. Furthermore, in virtually every case of TS due to RHD, there is concomitant mitral valve involvement. Pure isolated TS is very uncommon and can be caused by a variety of factors (many of which, coincidentally, can also cause TR). Approximately 90% of TS is post-inflammatory/RHD, while 10% are non-rheumatic (listed below) [5, 30–35].

- Congenital defects
- Carcinoid syndrome
- Cardiac device lead complications
- Tumors
- Infective endocarditis
- Stenosis of prior prosthesis

The interventions currently available include surgical intervention (both repair and replacement) or transcatheter balloon commissurotomy. There are limited observational data regarding TS interventions, and most recommendations are based on expert opinion and small case series/studies. TV surgery is recommended in patients with severe TS at time of operation for left-sided heart disease (class I recommendation) or for isolated symptomatic severe TS (class I recommendation). Balloon commissurotomy might be considered for isolated, symptomatic, severe TS (class IIb recommendation). While numerous clinical features are used to grade TS severity, the

Fig. 2 Structures adjacent to tricuspid valve. **a** Koch's triangle is delineated by the tendon of Todaro, the coronary sinus, and the septal tricuspid valve leaflet. The apex is the approximate location of the atrioventricular node, which resides in close proximity to the tricuspid valve annulus. **b** Histologic cross section showing the location of the atrioventricular (AV) node in relation to the tricuspid valve (TV). Right atrium (RA), atrial septum (AS), left atrium (LA), right ventricle (RV), interventricular septum (IVS), and left ventricle (LV) are labeled for orientation. **c** The right coronary artery (RCA) is situated in close proximity to the tricuspid valve (TV)



strictly anatomic features incorporated into the grading scheme include thickening, distortion, or calcification of the TV leaflets and TV area $< 1.0 \text{ cm}^2$ [36]. The underlying cause of TS must also be addressed, and in the cases of TS caused by cardiac device leads, consideration should be given to removal of the leads and replacement with epicardial leads. Furthermore, in TS caused by infective endocarditis, tumor, or thrombus, balloon valvuloplasty would be contraindicated [36, 37]. Table 2 lists anatomic features and their indications/contraindications for different interventions.

Tricuspid Regurgitation

TR is much more common than TS [4]. Between 75 and 90% of TR is functional/inorganic. In these cases, the valve and chords are anatomically normal, but leaflet coaptation is hindered by dilation of the TV annulus. Causes of secondary TR are listed below [5, 30, 38–45].

- Left sided heart failure
- Mitral stenosis or regurgitation
- Left to right shunt
- Eisenmenger syndrome
- Pulmonary parenchymal/vascular disease

- Pulmonic valve/artery stenosis
- Hyperthyroidism
- Cardiomyopathy
- Ischemic heart disease
- Atrial fibrillation

Only 10–25% of TR are due to primary causes, listed below. [5, 30, 38–45].

- Ebstein's anomaly
- Dysplastic TV
- Giant RA
- TV tethering associated with ventricular septal defect and ventricular septal aneurysm
- Post-infectious/RHD
- Connective tissue disorders
- Carcinoid syndrome
- Endomyocardial biopsy complication
- Endomyocardial fibrosis
- Myxomatous degeneration
- Cardiac device lead complication
- Infective endocarditis
- Nonbacterial thrombotic endocarditis
- Tumors

Table 2 Anatomic indications/contraindications for specific interventions

| Anatomic consideration | Indications or contraindications for interventions |
|--|---|
| Tricuspid stenosis | |
| Concomitant left sided valve disease | Anatomic feature recognized in AHA guidelines Concurrent surgical intervention for TS can be considered |
| Valve area < 1.0 cm ² | Anatomic feature recognized in AHA guidelines for grading of severe TS, which may make a patient eligible for surgical intervention |
| Thickened/distorted/calcified leaflets | Anatomic feature recognized in AHA guidelines for grading of severe TS, which may make a patient eligible for surgical intervention |
| Isolated TS | Possible candidate for balloon commissurotomy |
| Tumor, thrombus, vegetations | Risk of embolus precludes use of balloon commissurotomy |
| Transvalvular pacemaker leads | Consideration of replacement with epicardial leads |
| Tricuspid regurgitation | |
| Concomitant left sided valve disease | Anatomic feature recognized in AHA guidelines Concurrent surgical intervention for TR can be considered |
| Tricuspid valve annulus diameter > 40 mm or 21 mm/m ² | Anatomic feature recognized in AHA guidelines for grading of severe TS, which may make a patient eligible for surgical intervention |
| Flail/grossly distorted leaflets | Anatomic feature recognized in AHA guidelines for grading of severe TS, which may make a patient eligible for surgical intervention |
| Marked tethering | Anatomic feature recognized in AHA guidelines for grading of severe TS, which may make a patient eligible for surgical intervention Associated with failed annuloplasty repair; adjunctive surgical techniques or valve replacement should be considered |
| Isolated TR | Less invasive surgical approach may be attempted |
| Transvalvular pacemaker leads | Consideration of replacement with epicardial leads |
| Loss of nonplanar shape of TV measured by 3d TEE | May be a feature of more severe TR, warranting intervention |

- Ischemic heart disease
- Drug/radiation complication

The pathogenesis of functional TR involves volume or pressure overload due to left-sided heart disease, which leads to RV dilation and subsequent dilation of the tricuspid annulus. Due to its connection to the fibrous skeleton, the medial/septal aspect of the annulus stays relatively fixed, and dilation occurs along the anterior and posterior aspects as the free wall of the RV distends. Furthermore, RV remodeling can cause alterations in papillary muscle configuration. This ultimately distorts the normal geometry of the valve and subvalvular apparatus, hindering the proper coaptation of the leaflets despite intrinsically normal parts [46].

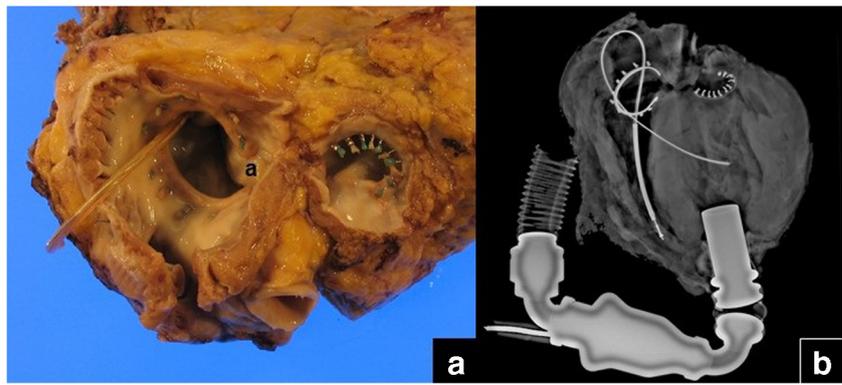
It was historically believed that treatment of the left-sided heart disease would also fix TR, so it was largely ignored. Only recently was it shown that not only did interventions for left sided valve disease not necessarily resolve TR, but that TR was itself an independent risk factor for morbidity and mortality [6–9]. Surgical intervention for TR has historically been associated with the highest mortality of all valve procedures. Because of this, there has been reluctance to perform tricuspid surgery, and isolated TV surgery remains rare [13,

47, 48]. Consequently, the number of patients undergoing percutaneous interventions is increasing under compassionate clinical use. Because of the relative novelty of TR interventions, there are limited data on the risks and long-term outcomes of each type, and no data comparing one intervention to another [39].

Surgical and minimally invasive interventions are mentioned below, though the details will not be discussed as they can be seen in the various comprehensive review articles referenced [1••, 19, 49••]. An example of a surgical TV annuloplasty device is shown in Fig. 3. In short:

- Surgical interventions include suture annuloplasty (Kay bicuspidization or De Vega repair), or ring annuloplasty (flexible annuloplasty band or rigid/semirigid rings).
- Minimally invasive interventions include ones that improve leaflet coaptation: TriClip/MitraClip (the transcatheter intervention with the most experience to date, with more than 600 patients having undergone this treatment), [13] FORMA device (available in 12 mm and 15 mm diameter) [50], TV Occluder Device, and Edwards Pascal transcatheter mitral valve repair system.

Fig. 3 Select example of surgical TV intervention. (a) Explanted heart with ring annuloplasty devices situated in tricuspid and mitral valve annulus. Note the discontinuous portion of the ring (a) along the septal edge, to avoid interference with the nearby conduction system. (b) Corresponding radiograph of explanted heart shown in (a), showing ring annuloplasty devices and portion of left ventricular assist device



- Other interventions target the annular dimensions: Trialign/Mitralign, TriCinch, Cardioband, Millipede annular ring, Cardiac Implants annuloplasty device, pledget-assisted suture tricuspid annuloplasty, minimally invasive annuloplasty, and transatrial intrapericardial tricuspid annuloplasty device (TRAIPTA—currently no experience in human subjects) [51].
- Heterotopic caval implantation targets the symptoms rather than the pathogenesis; these include TricValve (available in 28 to 43 mm diameter) and Edwards Sapien valves (available in 29 mm diameter) [39].
- Excitingly, the first successful in-human, transcatheter orthotopic TV replacements, using the NaviGate device were recently completed [49•, 52].

There are only a few anatomic considerations currently included in the AHA recommendations and grading of TR severity: flail/grossly distorted leaflets, TV annulus diameter > 40 mm or > 21 mm/m², marked tethering, and concomitant left-sided valve disease [36]. One recent study showed loss of nonplanar shape of the TV as measured by 3D transesophageal echocardiography might be another potential anatomic feature used to guide intervention [53]. Some have recently proposed incorporating the degree and location of leaflet coaptation, as well as tethering > 8 mm below the annular plane as additional anatomic parameters to stratify patients [38, 54]. Additionally, severe tethering is an independent risk factor for recurrent TR after surgical annuloplasty and thus may merit adjunctive surgical techniques or valve replacement [19, 55, 56]. In isolated TR, a more minimally invasive approach can be performed [57]. If TR is caused by cardiac device leads, consideration can be given to remove the leads and implant epicardial leads, though some data suggest that lead removal alone may not improve TR, and other interventions may still be necessary [58, 59]. Table 2 lists anatomic features and their indications/contraindications for different interventions.

Anatomic Features and Potential Impediments and Complications

With an understanding of the development of the heart, its normal and abnormal anatomy, and the current interventions available, we can begin to piece together the multiple impediments and complications that make TV intervention such a challenging frontier. A comprehensive list is provided in Table 3. The protuberant crista terminalis, limbus of the fossa ovalis, Eustachian valve, and Thebesian valve act as physical obstructions and possible sites of entanglement for transcatheter devices. Similarly, Chiari networks in the RA, though uncommon, have also been reported to entrap devices [60]. Furthermore, the retrosternal location makes intraprocedural guidance by TEE more difficult than in the mitral valve (though recently a successful TriClip/MitraClip procedure was performed using intracardiac echocardiography alone). [61] The vena cavae are often dilated to a larger diameter than the sizes currently available for heterotopic caval implantation devices. In addition, the dilated tricuspid annulus is often much larger than a pathologic aortic or mitral valve. These large diameters can hinder the ability for an implant to fix appropriately and be a cause of failure due to paravalvular regurgitation. The tricuspid annulus itself has less collagen, is more flexible, and is rarely calcified like the mitral and aortic valves—features that make fixation of an implant difficult. Secondary to the pattern of dilation along the anterior and posterior aspects of the annulus, clipping the anterior and posterior leaflets together may not fix TR, as the main area of insufficiency remains unfixed [62]. Transcatheter interventions, due to the inability to directly visualize the anatomy, can impinge on several important nearby structures, causing arrhythmias/conduction disturbances, myocardial infarcts, and left ventricular outflow tract complications (interestingly, due to its separation by the crista supraventricularis, complications involving the RV outflow tract are negligible). [63]

Table 3 Anatomic impediments/ complications for transcatheter intervention

| Specific anatomic considerations | Potential impediments and complications |
|---|--|
| Systemic venous return | |
| Large and variable dimension of normal IVC and SVC | Poor landing zone for fixation of devices |
| Proximity of hepatic veins | Risk of failure and paravalvular leak |
| Lack of calcification | |
| Right atrium | |
| Valves (Eustachian, Thebesian) | Physical obstruction and risk of entanglement for transcatheter device |
| Possible Chiari network | |
| Limbus of fossa ovalis | |
| Crista terminalis | |
| Tricuspid valve | |
| Larger annulus size | Most transcatheter devices developed for the smaller aortic or mitral valves |
| | Risk of failure and paravalvular leak |
| Flexibility of tricuspid annular tissue | Poor landing zone for fixation of devices |
| Noncalcified annulus in secondary TR | Risk of failure and paravalvular leak |
| Proximity to conduction system | Poorly visualized during transcatheter therapy, injury/impingement may cause arrhythmia/conduction disturbances, ischemia/infarct, or left ventricular outflow tract complications |
| Proximity to right coronary artery and nodal branch | |
| Proximity to coronary sinus | |
| Proximity to aortic sinus of Valsalva | |
| Angulation in relation to SVC and IVC | Complicated geometry is more difficult to maneuver around |
| Nonplanar valve | Devices developed for other valves are round and planar |
| Elliptical annulus shape | |
| Presence of cardiac leads | Possibility of device entanglement |
| Variable anatomy | Difficult to plan access |
| Annular dilation along anterior/posterior aspects | Clipping anterior and posterior leaflets together may not be as effective as clipping anterior and septal leaflets together |
| Right ventricle | |
| Triangular/crescentic shape | Decreased room to maneuver devices |
| Septomarginal trabeculation/moderator band | Possibility of device entanglement |
| More papillary muscles | |
| More chordae tendinae | |
| Coarse trabeculations | Precludes transapical approach |
| Thin RV free wall | |
| Retrosternal location | Increased distance from the esophagus makes intraprocedural imaging with TEE more difficult |

The triangular and crescentic shape of the RV, with its numerous chordae tendinae, papillary muscles, muscular bands, and coarse trabeculations also create a smaller chamber for the devices to operate in, with increased likelihood of entanglement. And finally, the thin wall of the RV and its coarse trabeculations preclude an apical approach to intervention [1•, 19, 39].

Conclusion

The TV, once neglected, has in recent years become a topic of renewed interest and innovation. Associated with significant morbidity and mortality and historically poor outcomes with surgical intervention, transcatheter interventions are rapidly emerging. Early data have been promising, demonstrating

both short term safety and efficacy in case reports, case series, and early clinical trials. The causes of tricuspid pathology and the anatomy of the tricuspid TV and right heart bring unique challenges to surgical and transcatheter intervention. The complex and crowded anatomy of the right heart may require novel approaches to access the TV. Simultaneously, the many structures that may affect TR, including annulus, leaflets, chordae, and papillary muscles, present themselves as discrete targets for intervention. The large size and shape of the annulus, the pattern of dilation along the free wall, increased leaflets, chordae and papillary muscles, and the complex geometry of the right heart with its numerous protruding obstacles must all be considered. This quickly advancing field has progressed from first attempts at transcatheter repair to first successful in-human transcatheter orthotopic TV replacement in a mere decade and will surely continue to progress and change the way we view and treat TV pathology.

Compliance with Ethical Standards

Conflict of Interest Ryan P. Lau, Gregory A. Fishbein, and Michael C. Fishbein declare that they have no conflict of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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- Of importance
- Of major importance

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