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Tricuspid Valve Repair

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Abstract

For the previous years, the tricuspid valve (TV), has been studied relatively less than the other heart valves diseases both about pathophysiology, management, surgical intervention, and treatment. However, recent advances in assessment and management of the TV disease have led to redirect the interest in this "forgotten valve." Surgeons often had believed that quick solutions for the left ventricle problems would also improve the secondary tricuspid regurgitation (STR). Every active surgeon has been preferred this quick solution in his whole surgery life many times. Medical treatment options aims to improve the underlying disease and the right ventricle failure. TV surgery have proven to yield good outcomes in surgery indicated patients. For patients who are not available for surgery, trans catheter intervention may be an alternative. Due to limited data, the best surgical techniques are still in question, with no clear answer, particularly for STR. Key factor in determining prognosis, timing for intervention and longer-term outcome is the right ventricular function at the time of prognosis.

Keywords: tricuspid valve, annuloplasty, regurgitation, annular ring, De Vega

1. Introduction

In the past, the TV has received less attention than the other heart valves in terms of pathophysiology and surgical treatment. TV is part of a complex functional structure that involves the right atrium (RA), the right ventricle (RV), and the pulmonary artery (PA) circulation. The prevalence of TV disease is steadily increasing, with the tricuspid regurgitation (TR), the most common form, occurring in an estimated 65–85% of the European population [1]. The most common type of TV disease is functional tricuspid regurgitation (FTR), occurring secondary to the dilation of the tricuspid annulus and/or the tethering of the valve leaflets due to RV dilation and dysfunction. However, with the recent increase in right-sided implantation of transvenous devices (pacemakers, implantable defibrillators), there has been a parallel increase in the risk of organic tricuspid disease. Recent data suggests that TR is not benign, and many patients would benefit from intervention during left-sided valve surgery or in the early period of isolated TV disease (TVD) [2]. The clinical evaluation of TVD is often difficult because of a lack of early clinical characteristics, as the disease might progress when it is diagnosed by a consultant. In order to manage symptoms, prevent complications, and improve quality of life, advanced TVD has to be surgically repaired or replaced [3].

Isolated TR patients are rarely referred for valve surgery and most repairs are performed at the same time with other planned cardiac procedures. With an in-hospital mortality rate up to 37% re-operations for evident TR and heart valve disease or for recurrent TR, generally they are not routinely recommended for most

patients [2]. The current American and European guidelines advocate a more proactive approach for the treatment of TR and/or annular dilatation during left-sided valve surgery. For its better superior long-term outcomes, tricuspid annuloplasty is the preferred technique. This renewed interest in surgical repair has been fueled by the development of a new generation of tricuspid annuloplasty rings and the technological advances in transcatheter treatment, which has expanded to include tricuspid pathologies in otherwise inoperable patients with advanced tricuspid disease and cardiomyopathy. Aggressive approach to surgical treatment is more widely adopted, rather than prophylactic interventions. Still, aggressive tricuspid surgery remains an area of controversy, while surgical repair is considered the gold standard for functional TR [4]. An important note is that presumably because of right ventricle anatomy the pathophysiology of functional TR is understood much less than functional mitral regurgitation (MR). Besides, the left ventricle function has a key role for the function of the right ventricle [5].

2. Anatomy and physiology

The heart has four functional valves and TV is the largest one, with a normal orifice area between 7–9 cm^2 (**Figure 1**) [6], apically located. Due to the low pressure differences between the RA and the RV, the large size of the TV can function at low gradient (<2 mm Hg) and low peak transtricuspid diastolic velocities [6]. The leaflets, the papillary muscles, the chordal attachments, and the annulus (with attached atrium and ventricle) are the components of the TV [7]. The integrity and harmony of these components result successful valve function.

2.1 The leaflets

TV closure during systole needs the normal function of the leaflets and their relationship with chordae and papillary muscle, although they are also closely related to the size and function of RV. RV pressure overload and remodeling was associated with up to 49% increase in TV leaflet size compared to controls in recent screenings, and when this increase in size was insufficient to cover the tricuspid valve closure area, there was a gradual increase in TR severity. observed [8]. The TV typically consists of 3 leaflets of unequal size. Healthy subjects may have anatomical

A　　　　　　　　　　　　　　　　　　　　**B**
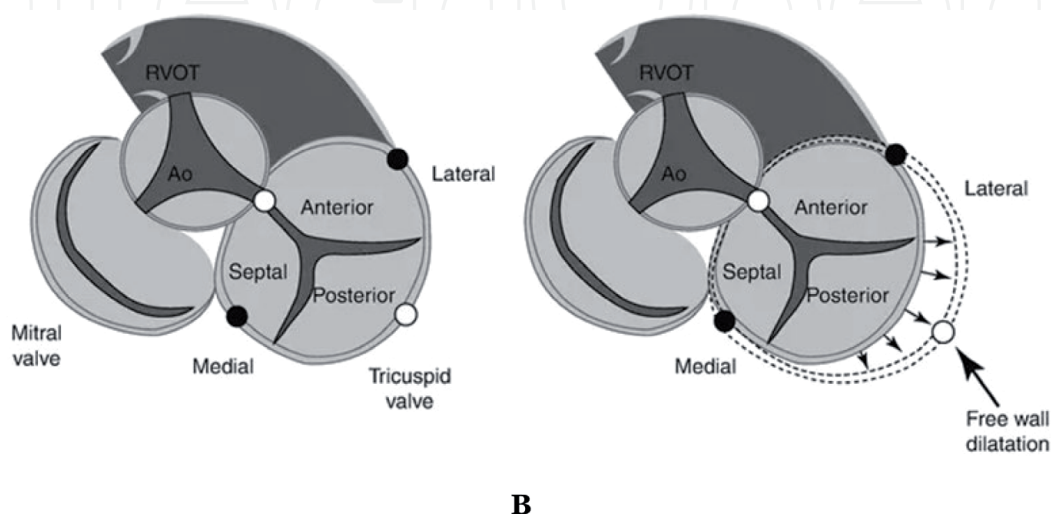
Figure 1.
A) Tricuspid valve and rhe relations with the other valves. B) The weak sies of the tricuspid aparatus with tendency for dilation.

variants consisting of 2 (bicuspid) leaflets or more than 3 leaflets [9]. Definition according to their anatomical positions in the body, these 3 leaflets would be septal, anterior-superior, and inferior: called septal, anterior, and posterior leaflets [10]. The anterior leaflet is the largest, whereas the posterior leaflet is notable for the presence of multiple scallops. The septal leaflet is the smallest and arises medially, directly from the tricuspid annulus above the interventricular septum. It is attached to the tricuspid annulus directly above the interventricular septum [11]. The anatomical markings for each leaflet vary considerably based on the size and shape of the annulus; still, the commissure between the septal and posterior leaflets is often prominent, located near coronary sinus entrance into the right atrium (**Figure 2**).

When we examine the integrity of the four heart valves the noncoronary sinus of valsalva of aortic root is typically adjacent to commissure between the septal and anterior leaflets: the anteroseptal commissure. This is the longest commissure, as the anterior and septal leaflets are often the longest circumferentially. The coaptation of the TV is typically at or just below the level of the annulus with a coaptation length of 5–10 mm [12]. This coaptation length is the potential reserve for keeping the function of the TV when right side of the heart is affected as dilation.

2.2 Chordae and papillary muscles

Tensor apparatus of TV are the papillary muscles and chordae. The posterior and septal leaflets supported by medial papillary muscle group cordae and anterior and posterior leaflets supported by anterior papillary muscle group cordae [6].

The accessory chordae may protrude from the right ventricular free wall or the moderator band. Hence, the septal and anterior leaflets of the TV are attached to the interventricular septum, and the anterior and posterior leaflets are attached to a
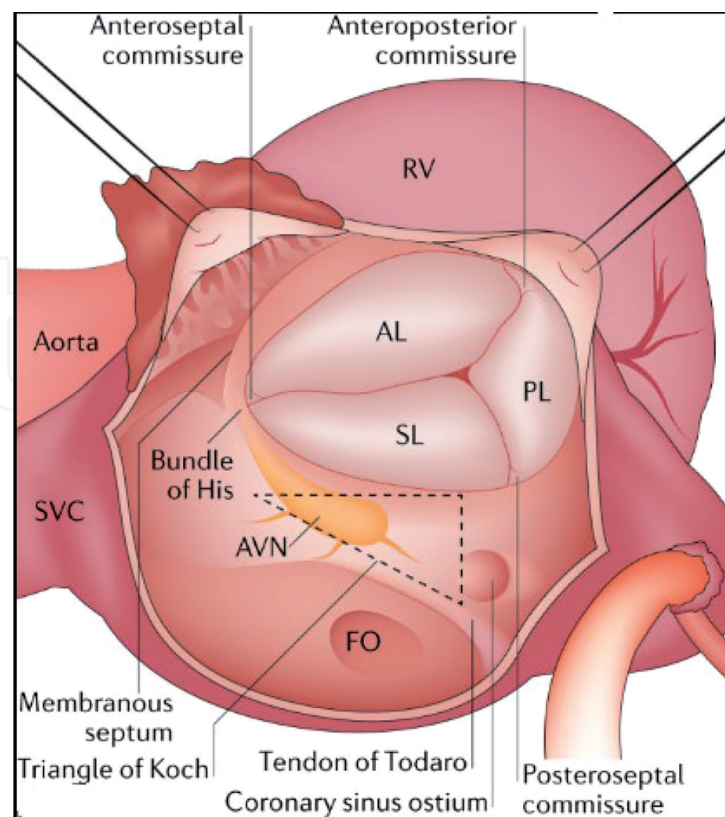
Figure 2.
The schematic seen of the Tricuspid Valve and the relation of heart conduction system (AVN: AN node, SVC: Superior Vena Cava, FO: Fossa Ovalis (Also this is the key anatomic tissue for trans septal mitral valve intervention), RV: Right Ventricle).

large anterior papillary muscle along the anterolateral right ventricular wall. Due to the fixed length of the chordae, the displacement of septal or lateral wall positions of the RV would affect tricuspid leaflet coaptation. Therefore, tricuspid annular sizing algorithms have been based on the dimension of the base of the septal leaflet [13]. The number of chordae varied from 17 to 36 with an average of 25 chordae [7]. Since the septal leaflets that is fixed to the septal wall is quite immobile, there is little space for the free wall of the right ventricle/tricuspid annulus to expand (**Figure 1**) [14].

Transcatheter interventions of the heart, the chordae can interact with catheters and interventional devices, causing additional difficulty during transcatheter approaches for TV. Besides, the mechanical properties and superstructure of the TV chordae tendineae in normal humans are composed of somewhat flat collagen bundles made of collagen fibrin matrices, with less extensibility than normal mitral valve chordae of comparable size [15].

2.3 Tricuspid valve annulus

The TV is situated within an elliptical, nonplanar annulus. The normal tricuspid ring is D-shaped, non-planar with two distinct segments: a larger C-shaped segment corresponding to the free wall of the RA and RV, and a shorter, relatively straighter segment corresponding to the septal leaflet and the ventricular septum [7]. Flexible fibroadipose tissue is the composition the annular ring. During the cardiac cycle composition of the annulus allows geometrical changes, it is rounder during diastole, and during systole it becomes more elliptical by the interventricular septum bulges into the RV [16]. The tricuspid annulus has a complex, three-dimensional structure that differs from the more symmetrical "saddle-shaped" mitral ring. This shape has implications for the design and implementation of new annuloplasty rings, rather than the existing planar annuloplasty rings in the tricuspid position [11]. Fukuda et al. conducted a real-time, three-dimensional transthoracic echocardiographic research and examined 15 healthy subjects and 16 patients with functional TR (12/16 had moderate to severe TR). They mapped the tricuspid annulus throughout the cardiac cycle and reconstructed it using a computer workstation. The healthy subjects had a non-planar, elliptical tricuspid annulus, with the posteroseptal segment being the lowest relative to the right ventricular apex and the anteroseptal segment being the highest. Patients with functional TR often had rather planar annulus compared to the elliptical shape in healthy subjects; the latter mainly expanded in the septal-lateral direction (F resulting in a more circular shape. The authors concluded that novel approaches or rings tailored to the unique shape of the tricuspid ring could improve ventricular function and reduce leaflet stress [17].

The tricuspid annulus is a dynamic structure that can produce significant changes (up to ~30%) in the area it creates during the cardiac cycle. It is greater in end-systolic/early diastole and during atrial systole, as well as under loading conditions [6]. When measured in healthy subjects using 3D echocardiography, the normal tricuspid annulus has a circumference of 12 ± 1 cm and an area of 11 ± 2 cm^2 [17]. During surgery it is more difficult to identify the TV annulus when it compared with the mitral valve annulus. The posteroseptal tricuspid annulus is more ventricular, but anteroseptal portion is more atrial [18].

3. Cause, diagnosis, and natural history

Two forms of TR are primary and secondary. Primary TR is seen less and can be the congenital or acquired disease processes that affect the leaflets or chordal structures, or both. Secondary or functional TR (STR or FTR) is more common and

secondary to other diseases like left-side heart diseases, pulmonary hypertension, RV dilation, and dysfunction from any cause, without intrinsic lesion of the TV itself. Enlargement of the valve annulus and the right ventricle is the main reason of the STR, any cause of left heart dysfunction or disease of myocardial or valvular causes, RV volume and pressure overload, and dilation of cardiac chambers can be the reason. Less common causes of tricuspid valve pathology include rheumatic, congenital, or other causes (endocarditis, leaflet tear/prolapse, chordal rupture, papillary muscle rupture, or myxomatous degeneration of the tricuspid valve) [19].

3.1 Classification of tricuspid regurgitation

See **Table 1**.

3.1.1 Primary tricuspid regurgitation

Primary TR is seen less than the secondary form may be congenital (Ebstein's anomaly) or acquired diseases of the TV (myxomatous degeneration of the tricuspid valve, leading to TV prolapse, endocarditis, carcinoid syndrome, rheumatic disease, radiation, or trauma). The latter is crucial for patient selection and clinical decision-making, so the two must be differentiated. One of the only causes of TR is that the leads of a pacemaker or defibrillator that pass from the RA to the RV can directly affect the leaflet coaptation. This has been reported in case reports and small series, but might be more important and common than currently detected. In a 2008 publication, by Kim et al. researched the effect of a transtricuspid permanent pacemaker or implantable cardiac defibrillator in 248 subjects using echocardiograms before and after device implantation. The authors found grade 1 or greater worsening of TR after implant in 24.2% of the subjects, and TR worsening was more common in implantable cardiac defibrillators than in permanent pacemakers with mild or lower TR at baseline [20]. The current guidelines do not recommended removal in patients with TR and transtricuspid pacing leads due to the potential to damage the valve and result in serious conditions [21].

3.1.2 Secondary (functional) tricuspid regurgitation

The most common cause of TR is secondary or "functional" insufficiency. STR can be categorized based on the underlying cause or the morphological abnormality of the tricuspid apparatus; some morphologies are clearly associated with specific underlying diseases:

1. STR due to left-sided heart disease (valve disease or left ventricular dysfunction),

2. STR due to any cause of pulmonary arterial hypertension (chronic lung disease, pulmonary thromboembolism, left-to-right shunt disease, or systolic

Primary	Secondary
Rheumatic	Pulmonary hypertension with RV remodeling
Infective endocarditis	(primary or secondary to left-sided heart
Iatrogenic (device leads, endomyocardial biopsy)	disease)
Congenital (eg, Ebstein's, levo-transposition of the great arteries)	Dilated cardiomyopathy
	Annular dilation (associated with AF)
Other (eg, trauma, carcinoid, drugs, irradiation)	RV volume overload (shunts/high output)

Table 1.
Tricuspid regurgitation classification table.

pulmonary artery pressure estimated by Doppler >50 mm Hg with no identifiable clinical cause),

3. STR due to any RV dysfunction (myocardial disease or RV ischemia/infarction),

4. STR with no detectable cause of TR (idiopathic STR).

Some morphological abnormalities associated with STR may co-occur, including:

1. tricuspid leaflet attachment or tenting,

2. displacement of papillary muscles,

3. RV dysfunction,

4. enlargement of the annulus and/or RA.

Several studies have concluded that atrial fibrillation (AF), ischemic heart disease associated with mitral regurgitation, rheumatic heart disease, and a large left atrium are associated risk factors of TR [22].

Many factors as preload, afterload, myocardial wall thickness and contractility which can limited by the intact pericardium are determining the right ventricle ejection fraction and the stroke volume. The RV Wall thickness is about 3–4 mm and the mass is approximately six times less than the LV. The RV is adapted to eject blood against a lower pulmonary vascular resistance. RV is low pressure highly compliant cardiac chamber because of low afterload results in reduced wall tension and characterises RV physiology. Before progressive RV dilatation, dysfunction and

Stage	Definition	Valve hemodynamics	Hemodynamic consequences	Clinical symptoms and presentation
B	Progressive TR	Central jet <50% RA Vena contracta width <0.7 cm ERO <0.40 cm^2 Regurgitant volume <45 mL	None	None
C	Asymptomatic severe TR	Central jet ≥50% RA Vena contracta width ≥0.7 cm ERO ≥0.40 cm^2 Regurgitant volume ≥45 mL Dense continuous wave signal with triangular shape Hepatic vein systolic flow reversal	Dilated RV and RA Elevated RA with "c-V" wave	Elevated venous pressure No symptoms
D	Symptomatic severe TR	Central jet ≥50% RA Vena contracta width ≥0.7 cm ERO ≥0.40 cm^2 Regurgitant volume ≥45 mL Dense continuous wave signal with triangular shape Hepatic vein systolic flow reversal	Dilated RV and RA Elevated RA with "c-V" wave	Elevated venous pressure Dyspnea on exertion, fatigue, ascites, edema

c-V wave indicates systolic positive wave; ERO, effective regurgitant orifice; RA, right atrial; RV, right ventricular; and TR, tricuspid regurgitation [23].

Table 2.
Stages of tricuspid regurgitation (AHA Guidelines).

failure progression RV can tolerate this high volume state for prolonged periods. This may occur with or without the development of pulmonary vasculopathy (from chronic high flow). Cresent shape of the RV changes with chronic volume overload to spherical form till limitation of the pericardial capacity. Ventricular interdependence shifts the ventricular septum leftward. LV filling is impaired further compounding a fall in cardiac output (**Table 2**) [8].

4. Diagnosis of tricuspid regurgitation

According to the 2020 AHA guidelines, TTE can differentiate primary TR (abnormal valve leaflets) from STR (normal valve leaflets) for left ventricular-related valve or myocardial disease. PA systolic pressure can be measured by TEE. Characterization of the severity of TR-related regurgitation is based on an integrative assessment of multiple parameters, as recommended by the American Society of Echocardiography and European Association of Echocardiography, but many limitations still remain. In patients with TR undergoing left-heart valve surgery, an annular diastolic diameter >40 mm (or >21 mm/m^2) indicates an increased risk of permanent or progressive TR after isolated mitral valve surgery. PA systolic pressure is estimated from maximal TR velocity. TR velocity for evaluation of RV systolic function is challenged by variation in RV loading condition as well as geometric and image acquisition constraints. Normal RV systolic function is defined by various parameters, including tricuspid annular plane systolic excursion (TAPSE) >16 mm, TV systolic. Other imaging modalities, such as magnetic resonance imaging and CT scanning, can provide more accurate information about the status of the RV [23].

Again according to the AHA guidelines invasive measurement of cardiac index, right –sided diastolic pressures, pulmonary artery pressures, and pulmonary artery pressures and pulmonary vascular resistance, as well as right ventriculography, can be useful when clinical and noninvasive are discordant or inadequate in patients with TR [23].

While TR is one of the main right-sided pathologies that cause right heart failure (RHF), tricuspid stenosis (TS) is a rare etiology for RHF. The increased volume load induces cardiac dilatation, thereby stretching the walls of the cardiac chambers, causing dilation of the tricuspid annulus and enhance the regurgitation of the blood. Therefore, as RHF increases over time, the progressive deterioration of TV functions becomes trapped in a vicious cycle that advances the heart failure. Signs of this pathological mechanism can be seen on echocardiography, and patients with severe TR have right heart enlargement. Other signs observed may include enlargement or pulsation of the inferior vena cava and/or hepatic veins. In addition, the TAPSE can be measured to detect subtle ventricular dysfunction: TAPSE < 8.5 mm is associated with right ventricular ejection fraction less than 25%. If RHF is suspected, investigations including electrocardiogram, natriuretic peptides, and echocardiogram are used to confirm the diagnosis [24].

Echocardiography is routinely used in clinical practice to assess the severity of TR. This includes color doppler flow mapping in at least 2 orthogonal planes, assessment of vena contracta width, flow convergence calculations, and the direction and size of the jet. In addition, the morphology of continuous wave doppler recordings across the valve and pulsed wave doppler of the hepatic veins can be used [25]. Serial evaluations of TR should be interpreted in the clinical context of the patient because, as with functional mitral regurgitation, severity volume status and afterload may be affected by many factors such as The RV shape is complex compared to the left ventricle, appearing as a crescent in cross-section and triangular when viewed from the side [26].

COR	LOE	Recommendations
1	B-NR	1. In patients with severe TR (Stages C and D) undergoing left-sided valve surgery, tricuspid valve surgery is recommended.
2a	B-NR	2. In patients with progressive TR (Stage B) undergoing left-sided valve surgery, tricuspid valve surgery can be beneficial in the context of either 1) tricuspid annular dilation (tricuspid annulus end diastolic diameter >4.0 cm) or 2) prior signs and symptoms of right-sided HF.
2a	B-NR	3. In patients with signs and symptoms of right-sided HF and severe primary TR (Stage D), isolated tricuspid valve surgery can be beneficial to reduce symptoms and recurrent hospitalizations.
2a	B-NR	4. In patients with signs and symptoms of right-sided HF and severe isolated secondary TR attributable to annular dilation (in the absence of pulmonary hypertension or left-sided disease) who are poorly responsive to medical therapy (Stage D), isolated tricuspid valve surgery can be beneficial to reduce symptoms and recurrent hospitalizations.
2b	C-LD	5. In asymptomatic patients with severe primary TR (Stage C) and progressive RV dilation or systolic dysfunction, isolated tricuspid valve surgery may be considered.
2b	B-NR	6. In patients with signs and symptoms of right-sided HF and severe TR (Stage D) who have undergone previous left-sided valve surgery, reoperation with isolated tricuspid valve surgery may be considered in the absence of severe pulmonary hypertension or severe RV systolic dysfunction.

Table 3.
2020 ACC/AHA guideline for the management of patients with valvular heart.

Disease recommendations for intervention timing for TR intervention (Level A: High quality evidence from more than 1 rondomised clinical trial (RCT) or meta-analyses of high –quality RCTs or one or more RCTs corroborated by high-quality registry studies. Level B-R: Moderate-quality evidence from 1 or more RCT sor meta-analyses of moderate-quality RCTs. Level B-NR: moderate –quality evidence from 1 or more well-designed, well-executed nonrandomized studies observational studies, or registry studies or meta-analysis of such studies. Level C-LD: Randomized or nonrandomized observational or registry studies with limitations of design or execution or meta-analyses of such studies or physiological or mechanistic studies in human subjects. Level C-EO: Consensus of expert opinion based on clinical experience.) (**Table 3**) [23].

By using experimental models it has been estimated that left ventricle contractions contribute 20% to 40% of RV systolic pressure and volume output [27]. And also both two ventricles share same biochemical environment, by this way any systemic and local neurohormonal parameters result in improvements in biventricular function. Importantly, left-sided heart failure with chamber enlargement and mitral regurgitation can cause right-sided pressure overload, RV dilation, tricuspid annulus enlargement, and resultant TR. This mechanical step initially led to the concept that surgical or medical treatment of the left-sided abnormality would result in secondary recovery or improvement in TR. Although there is improvement in TR, this is not always the case [11]. Dreyfus et al have shown that a paradigm advocating treatment of only the proposed "primary" lesion, such as mitral valve disease, will not directly correct tricuspid annular dilation or improve right ventricular function, which are key determinants of functional TR. In their study, the TV annulus was evaluated visually in 311 patients who underwent mitral valve repair between 1989 and 2001. Tricuspid annuloplasty was performed selectively only in patients with twice the tricuspid annular diameter (as measured from anteroseptal commissure to

anteroposterior). New York Heart Association class were significantly improved in those who underwent TV annuloplasty. In-hospital mortality and actuarial survival rate were similarly improved in patients undergoing TV annuloplasty, supporting the notion that TV annuloplasty improves patient outcomes during mitral valve repair [18].

In 2020 report by Tirone [28] for degenerative MR they found no correlation between tricuspid annulus of ≥ 40 mm on the development of postoperative TR following mitral valve repair. They concluded that a preoperative echocardiographic diameter of tricuspid annulus ≥ 40 mm is not associated with the development postoperative TR after mitral valve repair for degenerative MR. Furthermore, in their report of extended outcomes of mitral valve repair for degenerative MR, they found that patients were only 2.5% likely to develop severe TR at 20 years after surgery, but the probability increased to 20.8% when moderate-intensity TR was added. It was also very disturbing to find an accumulated new incidence of atrial fibrillation of 32.4% over 20 years. The development of these two adverse events was not associated with repeat MR and may have been interdependent. The reporter recommends that tricuspid annuloplasty should be performed during mitral valve surgery when there is moderate or severe tricuspid regurgitation and in patients with atrial fibrillation or a dilated right ventricular cavity (systolic diameter ≥30 mm), even in the absence of significant tricuspid regurgitation [18, 29].

5. Current surgical approaches to tricuspid regurgitation

In the past decades, intervention was believed to be unnecessary, as repairing secondary TR would resolve the problems in the left ventricle, which was thought to help TR regress as well. The first experience regarding the prognosis of FTR was reported by Braunwald in 1967. Braunwald claimed that untreated FTR naturally resolves after treating mitral valve disease [30]. Later, Carpentier recommended surgical intervention for FTR in patients with mitral valve disease [31]. However, today's understanding suggests aggressive intervention to resolve this disease. Repairing left-sided valve disease has resulted in increased survival rates, making the long-term sequelae of TR more pronounced. During left heart surgery, severe secondary TR should also be corrected. In cases of moderate or even lower right ventricular dysfunction or enlarged tricuspid valve annulus TR should be addressed as well [32]. The indications for tricuspid valve procedure during left heart valve surgery are relatively simple, though there is limited data on how to approach a patient with functional TR with no indications for left heart surgery. The preferred approach for these patients has not been well defined [33]. The main surgical technique to repair functional TR with a dilated ring and normal leaflet and chordal structures is by rigid or flexible annular open or closed rings, which are also used in mitral valve surgery, aiming to reduce annulus size and provide leaflet coaptation [11].

Contrary to findings regarding isolated TV surgery, the increased risk of mortality associated with concomitant tricuspid annuloplasty during mitral valve surgery seems almost negligible in current practice. TV repair takes an additional 15 to 20 minutes, but it can be performed without prolonging the CPB time by removing the cross-clamp in the working and perfused heart. The incidence of heart block requiring pacemaker implantation is potentially greater with concurrent tricuspid surgery, although this has been refused by most comparative series, seeming largely dependent on the preferred annuloplasty technique. Similarly, the current literature

has yet to confirm the theoretical incremental risk of postoperative bleeding with the addition of a right atriotomy suture line [34].

The anatomical characteristics of the TV determine the repair technique:

1. Annulus intervention (annuloplasty),

2. Leaflets (e.g., triangular resection),

3. Chordae (e.g., transfers or new chordae),

4. Papillary muscles (e.g., sliding technique).

5.1 Annuloplasty

Despite the multiple repair strategies for TV, the ring annuloplasty technique is the preferred treatment for preventing the long-term recurrence of TR associated with suture annuloplasty [35, 36]. Tricuspid annuloplasty devices, whether rigid or incomplete semirigid rings for creating annular remodeling, or flexible bands that provide annular reduction, are often focused on restructuring annular movement. To date, there has been no clear evidence of superiority for any annuloplasty device for preventing the recurrence of TR [37]. In functional or secondary TR, the non-planar native tricuspid annulus is larger, flatter, and more circular. Hence, the ideal annuloplasty device should consider the geometric changes and restore the normal three-dimensional elliptical shape of the tricuspid annulus to reduce leaflet stress and tethering It should also focus on remodeling along the right ventricular free wall, but have an open design to protect the transmission system. Ideally, such a device would be "resilient" in areas of three-dimensional motion or areas prone to ring separation, particularly along the membranous septum [38].

5.1.1 Kay annuloplasty

The first type of annuloplasty is suture annuloplasty, which can be done in one of two ways [39]. First Kay annuloplasty was introduced in 1962: involves creating a functional bicuspid valve by placing a suture through the posterior leaflet's commissure [40]. This surgical technique places a pledget-supported bed suture from the anterior-posterior commissure through the posterior annulus to the posteroseptal commissure. Deloche et al. have demonstrated posterior annular dilatation in functional TR, suggesting that this method would work in selected cases [41]. In 1965 Herald Kay reported his Kay bicuspidization procedure, where the posterior leaflet is plicated along the annulus, has shown good mid-term and long-term results [40]. Ghanta et al. have modified this technique (**Figure 3**) [42].
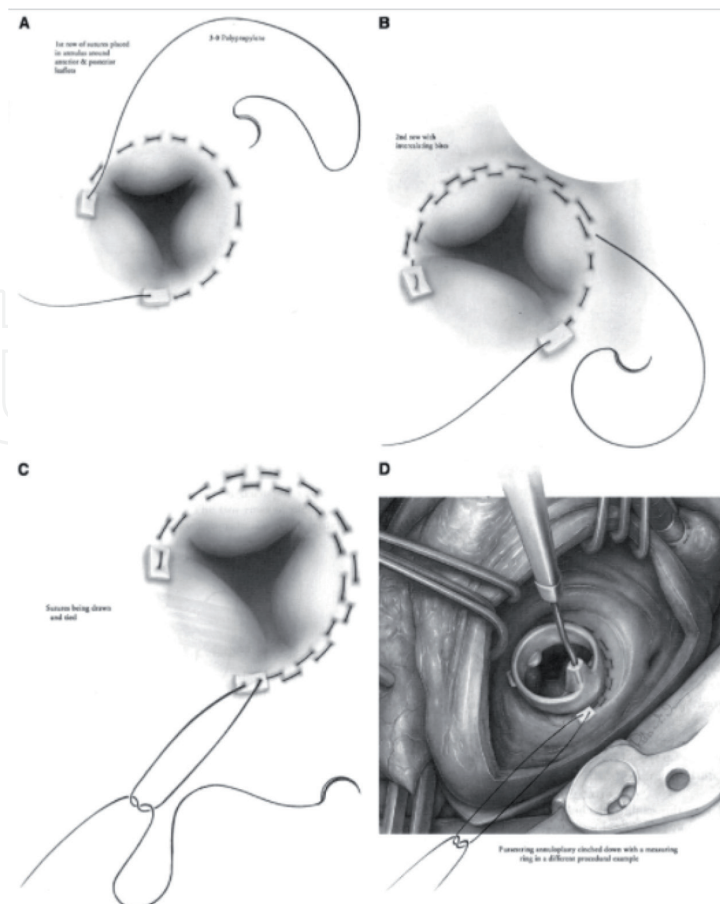
5.1.2 DeVega annuloplasty

DeVega annuloplasty was introduced in 1972 as an alternative [43]. The classical De Vega annuloplasty consists of a pair of continuous sutures, running along the anterior and posterior annulus, often corresponding to the free walls of the right ventricle. The septal part of the annulus is typically not involved in the expansion and is reserved to protect the transmission system. In the classical De Vega technique, a 2/0 or 3/0 polypropylene suture starts from the posterior end of the septal part of the annulus and continues counterclockwise in the posterior and anterior parts. The suture needle is inserted 1 to 2 mm deep, into 5 to 6 mm long knots. When the suture reaches the fibrous trigone, the anteroseptal commissure
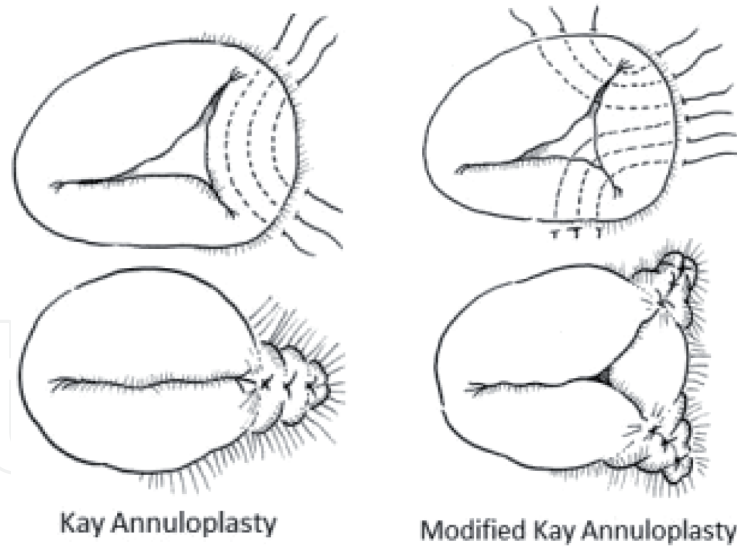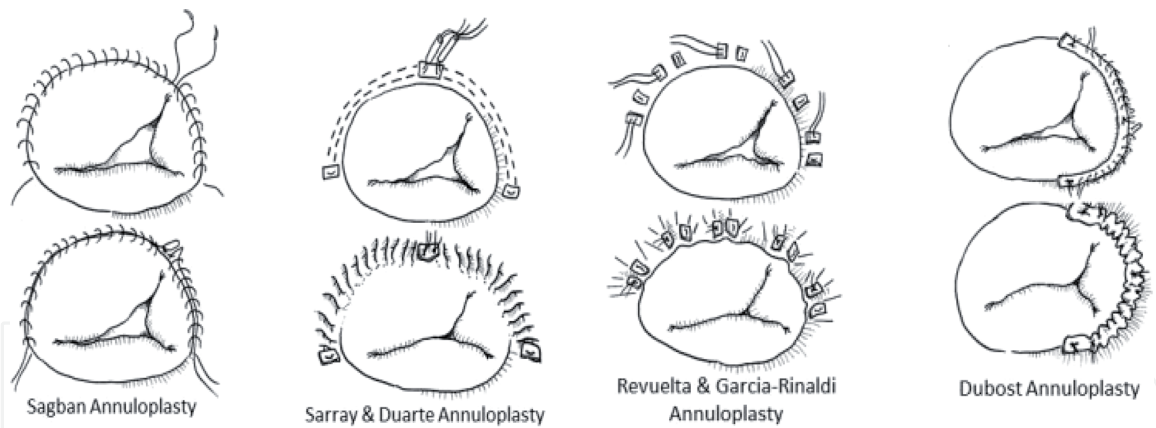
Figure 3.
The original and modified technique of Kay annulopasty.

is inverted on a teflon felt pledget; in the second suture, each knot of the ring interposes with that of the first. The suture ends where it starts, and a teflon felt is attached to the pledget. The degree of narrowing of the annulus can be measured from 25 to 29 mm using a mechanical valve sizer or Hegar dilator, depending on the patient's body surface area. Note that mild stenosis is better tolerated than regurgitation. When performed routinely, the valve can be tested by injecting cold saline into the right ventricle using a bulb syringe (**Figure 4**) [44].

Figure 4.
De Vega Annuloplasty.

Figure 5.
The several modified techniques of De Vega annuloplasty.

As a result of gliding effect recurrent TR secondary to Bowstring phenomenon is seen after classical De Vega annuloplasty often in the setting of moderate to severe regurgitation. There are several modifications of classical De Vega annuloplasty for example, Revuelta and Garcia-Rinaldi, Dubost, Sagban, and Sarray and Duarte (**Figure 5**) [45].

5.1.3 Carpentier ring annuloplasty

The next technique is ring annuloplasty, introduced by Carpentier in 1971. This is where a rigid or semirigid ring is introduced to control the dilatation of the annulus [39].

A rare complication that deserves focus is injury to the right coronary artery (RCA). Plications caused by aggressive reduction of the tricuspid annulus or deep penetration of purulent string sutures may impair RCA flow [37]. Note that severe arrhythmias, persistent ST elevation on electrocardiogram, or RV dysfunction following cardiopulmonary bypass would affect the RCA. Prompt grafting of the distal RCA or removal of the annuloplasty band is an effective strategy to deal with this rare complication, but early recognition is key for good outcomes [37].

A 2014 meta-analysis compared these techniques and found bicuspidization or the Kay method to be associated with a higher risk of recurrent TR. Still, ring annuloplasty was found to give better results in reducing the risk of recurrence. There was no significant difference in late survival rates, although ring annuloplasty provided significant reduction in early mortality [46]. A more recent 2020 meta-analysis concluded that mortality and TR incidence rates were comparable among ring and suture. Other flexible rings were found to have a higher TR ratio than rigid rings. However, this systematic review excluded articles with primary TR. There is still a limited number of suitable research and a lack of large randomized studies (**Figure 6**) [47].

5.2 Leaflets maneuvers

As described by Castedo et al., other approaches include edge-to-edge repairs, like the Alfieri repair, and DeVega-style purse-string suturing techniques to reduce the anterior and posterior parts of the annulus [48, 49].

There have been some auxiliary techniques to increase ring annuloplasty methods, particularly in leaflet tethering formed by right ventricular dilatation and annulus enlargement. Enlarging the anterior leaflet with a pericardial patch increases leaflet coaptation and provides leaflet mobility (**Figure 7**) [50].
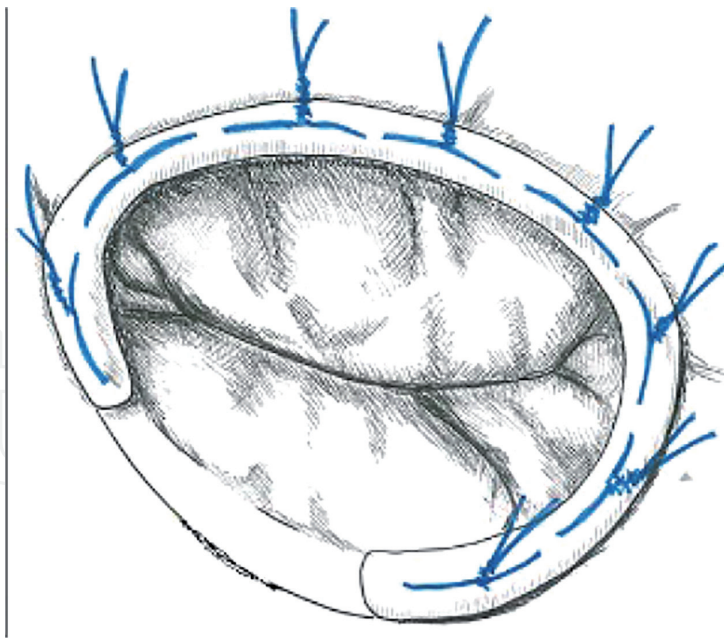
Figure 6.
TV ring annuloplasty.

The Leaflet Augmentation
Technique
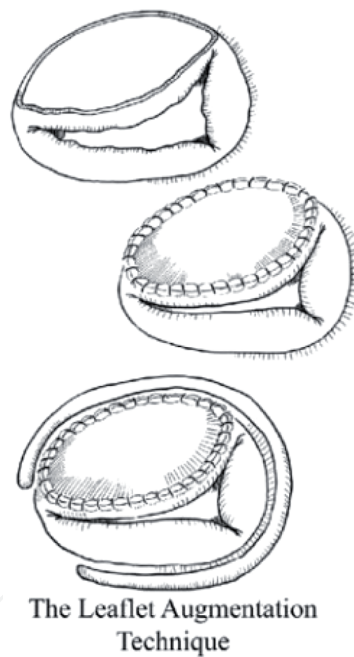
Figure 7.
The anterior leaflet of TV is enlarged by using a pericardial patch.

Double-orifice valve repair is achieved by approximating the free edges of the septal and newly formed anteroposterior leaflets. This is very effective in dealing with complex acquired TR (**Figure 8**) [51].

The 'clover' technique consists of stitching together the middle point of the free edges of the tricuspid leaflets, producing a clover-shaped valve (**Figure 9**) [52].

Combination of some factors like the technique of the operation, decision of annuloplasty type, unrecognized severe leaflet tethering area mismatch between the leaflets and the annuloplasty ring. In cases of severe tethering enlargement of posterior and anterior leaflet can be used. However there is no evidence that this is superior to tricuspid valve replacement [50].
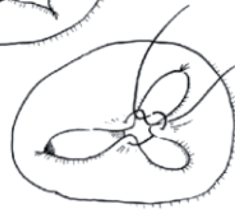
Recurrent TR is probably due to a combination of factors: operative technique, type of annuloplasty used, unrecognized severe tethering of the leaflet, and
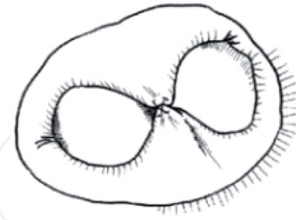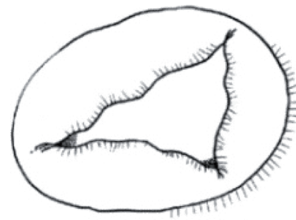
Figure 8.
Double orifice technique like in Alfieri technique.

Figure 9.
The stages of clover technique.

mismatch between the area of the leaflets and the area of the annuloplasty ring. Some surgeons advocate patch enlarging the anterior and posterior leaflets in cases of severe tethering but there is no evidence that this is superior to tricuspid valve replacement [50].

6. Discussion

In the absence of concurrent tricuspid valve repair, the incidence of TR after mitral valve surgery is somewhat dependent on the MR mechanism. A US study on 5223 patients reported worse survival rates at a 4-year follow-up of TR, independent of age, right and left ventricular systolic function, or right ventricular diameter. TR has been associated with less survival times in ischemic or non-ischemic cardiomyopathy with or without heart failure symptoms [32, 53]. Matsuyama et al. followed up 174 patients for 8 years and found that only 16% who underwent non-ischemic degenerative mitral valve surgery without tricuspid valve surgery developed 3 to 4 TRs [54]. TR seems much more common in patients who had

mitral valve repair due to functional ischemic mitral regurgitation. Matsunaga et al. investigated 70 patients who underwent mitral valve repair due to functional ischemic mitral regurgitation and found that 30% (21/70) had at least moderate TR before surgery. Postoperatively, moderate TR increased by 25% in 1 year, 53% in 1 to 3 years, and 74% at the 3-year follow-up [55].

Even after successful mitral valve repair, significant residual tricuspid valve regurgitation contributes to poor postoperative hemodynamic outcomes. King et al. found high early and late mortality rates in patients who required tricuspid valve surgery after mitral valve operation. The authors encouraged liberal use of tricuspid annuloplasty in the first mitral valve surgery. Surgical series have demonstrated significant improvement in recurrent TR, survival, and event-free survival rates with successful tricuspid valve repair (primarily when combined with other valve surgeries) [56].

Rigid annuloplasty rings appear to have lower TR recurrence rates than DeVega and flexible band annuloplasty [57]. Algarni et al. compared rigid rings with flexible bands in 713 TV annuloplasty patients in 2020. The authors found that the type of TV annuloplasty did not affect survival or tricuspid valve reoperation. However, there was a trend of higher cumulative incidence of recurrent moderate TR with flexible bands compared to rigid rings [58].

Dreyfus et al. suggested that annular dilatation measuring 70 mm or more during mitral valve repair surgery is an indication for annuloplasty, even in the absence of TR. The authors also demonstrated that TR was increased by at least 2 degrees in 45% of patients undergoing isolated mitral valve repair, supporting the perspective that tricuspid dilatation is a progressive process that often warrants preventive surgical treatment [18]. Singh et al. found no difference in survival or need for TV reoperation over 10 years of follow-up with TV repair compared to tricuspid valve replacement in patients with organic TR. Tricuspid valve repair is associated with better perioperative, midterm, and event-free survival rates than TV replacement in patients with organic tricuspid disease. TV replacement demonstrated higher mortality. The authors suggested that the higher perioperative mortality with replacement may be due to a hard object (the tricuspid valve) in a deformable low-pressure cavity (the right ventricle), resulting in right ventricular dysfunction and a low-output state perioperatively. The authors concluded that there was no difference in terms of functional class among the groups, although the patients had fewer recurrent TRs with repair versus replacement (62% versus 95% had mild or less TR at the final echocardiographic follow-up) [2].

Due to the significant risks of isolated tricuspid valve surgery and the poor prognosis of TR, less invasive approaches like robotic or endoscopic methods or innovative approaches like transcatheter have been needed. Currently, their use is not as common as mitral procedures, as the anatomical features of the tricuspid valve and associated apparatuses make TR treatment with minimally invasive surgery and transcatheter treatment more difficult [59, 60]. Even though severe TR is largely associated with mortality, patients with normal ventricular function can live for years, even decades, without the tricuspid valve. Arbulu et al. performed tricuspid vulvectomy without changing the tricuspid valve in patients with infective endocarditis secondary to IV drug addiction. During long-term follow-up, most patients (37 of 55; 67%) did not require TV replacement, and only a small number of patients without TV developed severe and permanent right ventricular dysfunction. Therefore, if there is only one underlying cardiovascular disease responsible for TR, this may be more important in patients with secondary TR [61].

Gursoy et al. reported that female sex, low preoperative functional capacity, low body surface area, enlarged left atrial size, enlarged right ventricular size, and increased square root of left ventricular mass index were effective on functional TR progression. After these parameters were studied in a multivariate logistic

regression analysis, only female sex and left atrial size were found to be independent risk factors [62].

The recurrence of significant TR after tricuspid annuloplasty is around 8 to 15% at 1 month after surgery, depending on various factors (e.g., preoperative TR severity, pulmonary hypertension, RV dilation, pacemaker, LV dysfunction, increased LV remodeling, severe tethering of tricuspid leaflets, or the DeVega technique instead of ring annuloplasty). Most of randomized and observational researches have shown that particularly in patients with severe tricuspid annular dilatation or pulmonary hypertension, repair the valve with ring annuloplasty techniques are more durable than suture annuloplasty ones. Long-term survival after tricuspid valve surgery for severe TR is influenced by a variety of preoperative factors like advanced heart failure symptoms, comorbidity, and end-organ dysfunction, rather than the type of surgery or the cause of TR. Ring annuloplasty may be associated with better results compared to the DeVega technique. The results of annuloplasty alone have not always been consistent. This may be associated with the degree of narrowing of the tricuspid opening, among other factors; thus, it has been recommended to reduce the size of the tricuspid annulus to prevent recurrent TR, considering the patient's body size [63].

The ESC 2017 guidelines recommend ring annuloplasty as the preferred modality for STR. Besides, in cases of severe tethering or severe enlargement of the annulus, replacement should be considered. Still, a very recent 2020 meta-analysis found no comparable differences among these techniques. There is still a lack of adequate research on valve interventions in TR, so the most effective intervention has yet to be clarified [64].

7. Conclusion

Intervention for the tricuspid valve disease has entered a new era with evolving guidelines and the development of new surgical annuloplasty devices and techniques, as well as conceptual transcatheter options. Still, the implementation of such novel techniques requires a significant infrastructure and increased costs. The current guidelines now emphasize surgical repair of functional tricuspid regurgitation during left-sided valve intervention, even during the repair of severely enlarged annulus in the absence of significant tricuspid regurgitation. The newly developed annular rings have been redesigned to protect the transmission system from adverse effects and to better mimic or preserve the normal tricuspid geometry. Finally, there is now an increasing early experience with new transcatheter approaches for managing very high-risk patients with advanced tricuspid valve disease. Regarding repair, the findings tend to increasingly favor rings, and among these, rigid rings that preserve the geometry.

According to our opinion, as mentioned above, it is very clear that if the patient has a severe TR, it is not true to left it as it is. If the patient has operation indication due to mitral valve disease, transseptal approach from right atrium with bicaval cannulation will be helpful for both valve intervention. By this way the surgeon consider to minimize the aortic cross clamp time. However which technique will be chosen is up to the some factors like experience of the surgeon, limitation of the sources, the degree of the regurgitation and the size of the TV annulus and right ventricle. If you don't have any annular ring for plasty, De Vega, Kay annuloplasty techniques or the modifications can be preferable. Also using teflon felt or pericardial patch like a ring is the other choices. In De Vega technique we use the same sizer with the replaced prostetic mitral valve for the reducing tricuspid annulus. If the patient has minimum or moderate TR also this kind of simple or particle techniques

also can be preferable. In severe TR, ring annuloplasty has better middle and long term results then the others. Annuloplasty ring sizers can be use or one or double size larger then the prosthetic mitral valve can be used for the TV annular ring. In my opinion after the TV repair testing by saline injection must be done but the result is not certain for the future progress of the valve. Again to our consideration and observation TV intervention with the mitral valve intervention does not effect the early operation mortality and morbidity.

Conflict of interest

The authors declare no conflict of interest.

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