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Echocardiographic Assessment and Surgical Management of Tricuspid Valve Regurgitation: Review Article

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ABSTRACT
Tricuspid valve regurgitation (TR) remains a significant clinical challenge, predominantly occurring as secondary or functional disease. This paper explores the echocardiographic assessment, pathophysiology, and operative management of tricuspid valve regurgitation, emphasizing the importance of repair techniques over replacement. Echocardiography plays a pivotal role in diagnosing TR and guiding surgical interventions, offering insights into annular dilation and leaflet tethering. Surgical repair, including the De Vega procedure, ring annuloplasty, and autologous pericardial band placement, is preferred for preserving valve function and improving outcomes. Functional TR associated with left-sided heart pathologies often warrants simultaneous repair during related surgeries. Medical management complements surgical approaches, addressing heart failure and pulmonary hypertension in appropriate cases. By reviewing the current techniques and their outcomes, this study underlines the importance of personalized surgical strategies for optimizing patient care and long-term prognosis.
Keywords: Echocardiography, Surgical Management, Tricuspid Valve Regurgitation.

INTRODUCTION
The TV, refers to tricuspid valve, is the greatest and most apically located of the 4 valves of the heart, with a normal orifice region ranging from seven to nine centimeter squared. Due to its reduced pressure and great size variances among the right ventricle (RV) and right atrium (RA), peak transtricuspid diastolic velocities usually remain below one meters per second, with mean gradients of below two millimeters of mercury. Comparable to the mitral valve, the mitral valve may be separated into 4 components: the chordal attachments, the leaflets, the papillary muscles, in addition the annulus (with the connected ventricle and atrium). The leaflets and their association to the papillary muscle and chordae are crucial for tricuspid valve closure throughout systole and might be integrally associated with right ventricular size and function [1].

This study aimed to evaluate the echocardiographic features, pathophysiology, and surgical techniques for tricuspid valve (TV) regurgitation management, emphasizing repair strategies like suture annuloplasty, ring annuloplasty, and pericardial band placement.

Tricuspid valve leaflets
The tricuspid valve typically has 3 leaflets: posterior, anterior, and septal though anatomical variations like bicuspid or more than three leaflets can occur in healthy individuals. Anterior leaflet, greatest and most mobile, has the greatest radial length, whereas the posterior leaflet, frequently scalloped and circumferentially shortest, may merge with the anterior leaflet in about ten percent of cases. The septal leaflet, the shortest radially and minimum mobile, is connected to the tricuspid annulus over the interventricular septum with chordae often inserting directly into the septum ≤10 mm apically from the anterior mitral leaflet's septal insertion.

Commissures vary anatomically, with the septal-posterior commissure near the coronary sinus and the septal-anterior commissure adjacent to noncoronary sinus of Valsalva. Anteroseptal commissure is longest due to larger circumferential size of these leaflets. Normal coaptation happens at or slightly under the annulus, with a five to ten millimeters coaptation length serving as a reserve to accommodate annular dilation before malcoaptation develops [2] (Figures 1 - 3).

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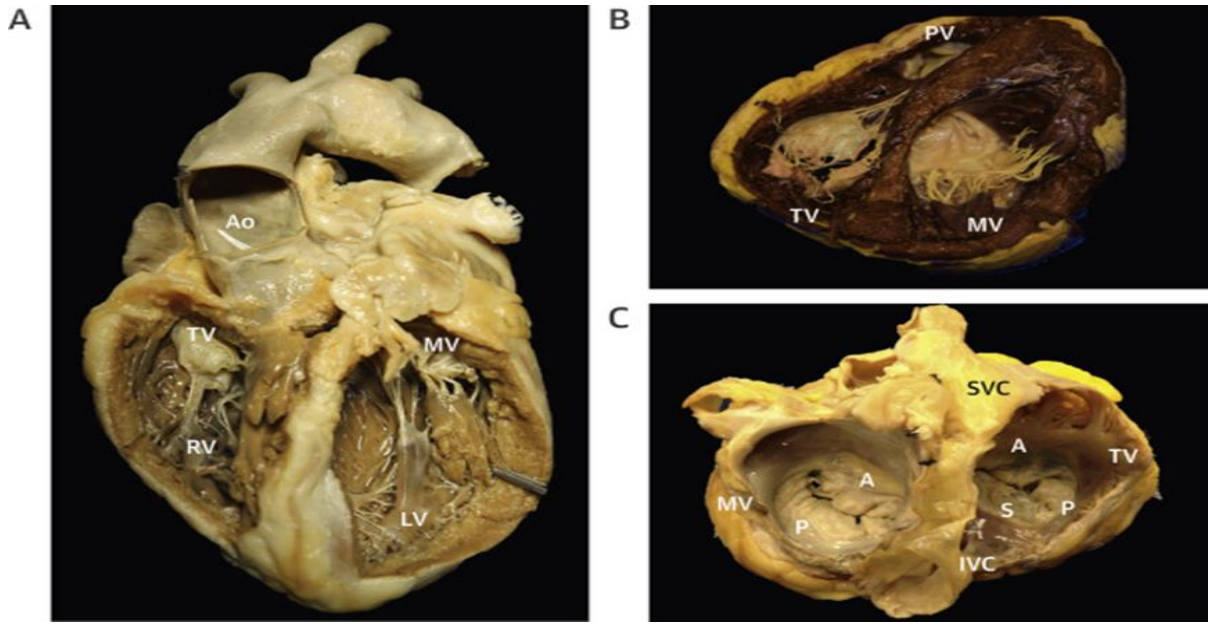

Figure (1): Gross anatomy of tricuspid valve. **(A)** TV is associated with the structures of the left heart. The TV is, in actuality, most anterior and apical of the four valves of the heart, despite usually being depicted in this orientation. **(B)**The tricuspid valve related to both the right ventricular outflow tract and pulmonary valve. Notably, the fibrous continuity among these two valves is absent. **(C)** Relative positioning of tricuspid valve from atrial aspect. A indicates the anterior leaflet; Ao means the aorta; IVC refers to the inferior vena cava; MV indicates the mitral valve; PV indicates the pulmonic valve; P refers to the posterior leaflet; LV indicates left ventricle; S means the septal leaflet; SVC refers to the superior vena cava; and TV indicates the tricuspid valve [1].

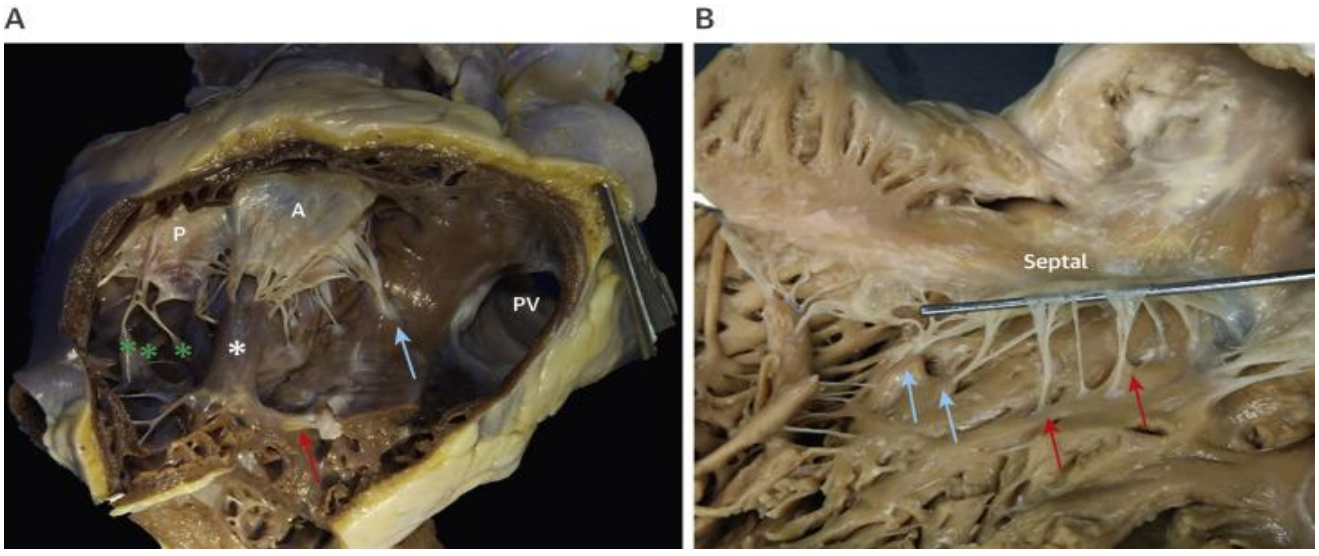

Figure (2): Papillary muscles. **(A)** Usual distribution of papillary muscles for tricuspid valve. Anterior papillary muscle usually is greatest (shown by the white asterisk), giving chordal support for the posterior and anterior leaflets. moderator band shown by **(orange arrows)** might connect to this papillary muscle. Posterior papillary muscle is frequently trifid or bifid shown by **(green asterisks)** and provides chordal support to septal and posterior leaflets. The septal papillary muscle demonstrates variability **(blue arrow)**. **(B)** Chordal attachments of septal leaflet to septal papillary muscle are indicated **(blue arrows)** and directly from septal myocardium **(orange arrows)** [3].

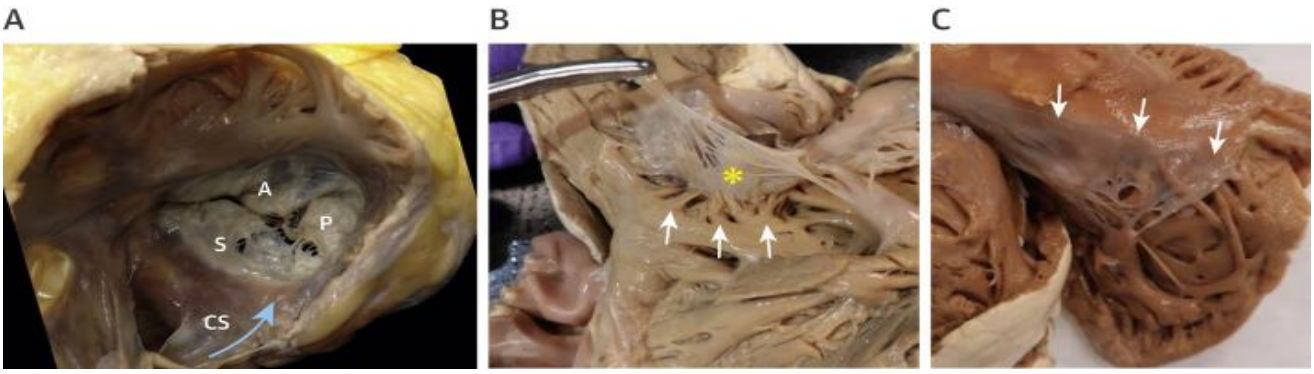

Figure (3): (A) TV Annulus. The TV is observed from atrial side, showing usually D-shaped annulus consist of a flat septal area and curved posterior and anterior area. **(B)** Ventricular surface of anterior leaflet **(asterisk)** has several "crisscrossing" muscle attachments **(arrows)** directly connected to base of the leaflet. **(C)** atrial surface of the anterior leaflet annulus shown by **(white arrows)** is non-fibrous and exhibits a smooth transition from the atrium to the ventricle. CS indicates the coronary sinus. **(curved blue arrow)** [2].

Tricuspid Valve Tensor Apparatus

The tricuspid valve's tensor device comprises the chordae tendineae and papillary muscles. There are typically 2 main papillary muscles (posterior and anterior) and a 3rd variable one. Anterior papillary muscle, the greatest, supports posterior and anterior leaflets, occasionally combined by moderator band. Posterior papillary muscle, often trifid or bifid, supports the septal and posterior leaflets. Septal papillary muscle is variable, sometimes absent in twenty percent of cases, with chordae potentially arising directly from the septum. Accessory chordae might bind to right ventricle free wall or moderator band. Due to fixed length of chordae, dislocation of the right ventricle lateral or septal wall can have an effect on the coaptation of leaflets. Chordae tendineae consist of collagen (80%), elastin, and endothelial cells, with five types identified by morphology and insertion. Their mechanical properties exhibit lower extensibility than mitral valve chordae, explaining marked tethering with RV dilation or papillary muscle displacement [4].

Tricuspid Valve Annulus

The tricuspid annulus is a D-shaped, dynamic structure that alters area throughout cardiac cycle. It has two segments: a larger, curved segment along the right ventricle free wall and a shorter, straighter segment along ventricular septum. Histological studies show minimal fibrous tissue in the right ventricle free wall segment, with annulus composed mostly of endocardium, epicardium, in addition to surrounding adipocytes. Normal annular circumference and area are 12 ± 1 centimeter and 11 ± 2 square centimeters, correspondingly. The annulus dilates laterally and posteriorly in secondary tricuspid regurgitation (TR), whereas dilation of the septal segment is restricted by the fibrous skeleton. The anterior annulus is less distinct in comparison with the posterior, and changes in shape and size affect the annulus' functional properties during disease progression [5].

Adjacent Anatomy

Three key structures are located near the tricuspid valve:

1. The noncoronary sinus of Valsalva is adjacent to the commissure among septal and anterior leaflets, posing risks for aortic perforation during transcatheter interventions.
2. The AV refers to atrioventricular node and bundle of His cross septal leaflet attachment, with potential for heart block if disrupted.
3. Right coronary artery courses along atrioventricular groove, with its proximity to the annulus being an important consideration for device anchoring [6].

Anatomy and Function of the RV

The RV's distinctive ventriculoatrial anatomy facilitates efficient blood flow through the tricuspid annulus. A helical flow in the right atrium contributes to right ventricle filling and energy conservation. Unlike the LV, which refers to left ventricle, the RV blood flow is directed immediately toward the pulmonary valve without requiring rotational motion. RV systolic function is influenced by ventricular interdependence, contractility, afterload, and preload. Right ventricle is more sensitive to acute afterload increases in comparison with left ventricle. Pulmonary vascular compliance supports low-pressure RV outflow. With rising pulmonary artery pressure (PAP), sequential changes occur: early RA dilation at low PAP and RV dilation at high PAP, reflecting disease progression [7].

Normal Hemodynamic

Right cardiac pumps deoxygenated blood to the pulmonary circulation, whereas the left heart pumps oxygenated blood systemically, with both sides pumping

equal volumes in series. The RV, with a thinner, compliant wall, operates under low pressure and relies on preload, afterload, as well as contractility like the LV. The Frank-Starling mechanism enhances contractility with increased preload.

RV systolic pressure (RVSP) is assessed from TR velocity and right atrial (RA) pressure: right ventricle systolic pressure equal four (TR velocity)² + right atrium pressure. Normal PA pressure is <25 mmHg, and RVSP is ≤35–36 mmHg. Significant pulmonary hypertension (PVR >3 Wood units) indicates pathology. Volume or pressure overload can lead to right ventricle hypertrophy and, in severe cases, ventricular shape changes (e.g., D-shaped LV).

P-V loops clarify RV contractility, preload, and afterload, with ventricular elastance serving as a contractility index. Hemodynamics, governed by cardiac output, is critical for blood flow, assessed via heart rate and ejection fraction [8].

Mechanism

Physiologic blood flow is best understood as laminar flow through compliant vessels, with complexities like viscosity, turbulence, and resistance introduced gradually. Laminar flow is characterized by velocity gradients due to wall shear stress, where blood near vessel walls flows slower due to friction. Turbulence, modeled by the Reynolds number Re=ρVD/μRe = \rho VD/\muRe=ρVD/μ, becomes likely at high ReReRe values and is influenced by blood viscosity, velocity, and vessel diameter [9].

Vessel compliance, defined as C=ΔV/ΔPC = \Delta V/\Delta PC=ΔV/ΔP, measures distensibility under pressure. Veins are more compliant than arteries, creating a high-pressure arterial system and low-pressure venous system. Blood flow (QQQ) follows Ohm's law: Q=ΔP/RQ = \Delta P/RQ=ΔP/R, where flow depends on the pressure gradient (ΔP\Delta PΔP) and systemic resistance (RRR). Resistance increases with narrower vessels but decreases when vessels are in parallel, as observed in capillary beds [10].

Resistance in series adds up (Rtot=R1+R2+…R_{\text{tot}} = R_1 + R_2 + \dotsRtot =R1+R2+…), whereas in parallel, total resistance is reduced (1/Rtot=1/R1+1/R2+…1/R_{\text{tot}} = 1/R_1 + 1/R_2 + \dots1/Rtot=1/R1+1/R2+…), aiding blood flow by distributing pressure more effectively. Capillary beds exemplify parallel arrangements, balancing flow despite their small diameter [11].

Pathophysiology of Tricuspid Regurgitation (TR)

Prevalence:

Topilsky *et al.* (2019) [12] reported significant TR prevalence at 0.55% in 21,020 cases, had severe or moderate tricuspid regurgitation affecting one in twenty-five cases above seventy-five years, more commonly in women. Functional TR, frequently 2ry to left valvular illness or pulmonary hypertension, accounted for most cases [12].

Types of TR (Figure 4 and table 1)

Primary TR: Rare; attributed to congenital (e.g., Ebstein's anomaly) or acquired diseases (e.g., myxomatous degeneration, endocarditis, carcinoid syndrome, rheumatic disease) affecting the tricuspid valve [13].

Secondary TR: More common; occurs without intrinsic valve lesions. Classified into:

1. Precapillary pulmonary hypertension-related TR (PH-TR).
2. Left heart-related TR (LH-TR).
3. Isolated TR (ITR).
4. Right ventricular disease-related TR (RVD-TR) [14].

Pathophysiology of FTR:

FTR involves leaflet tethering, papillary muscle displacement, right ventricular dysfunction, and/or dilation of the RA or annulus. Functional tricuspid regurgitation typically leads to an elliptical regurgitant orifice along anteroposterior edge, with leaflet tethering often contributing, even in absence of annular dilation [1].

Tricuspid Annulus and Evaluation

The tricuspid annulus, partly fibrous and sensitive to pre-/afterload, becomes more circular and planar with regurgitation [15]. Assessing TR needs a multi-parametric echocardiographic technique, involving quantitative (e.g., regurgitant volume, EROA), semi-quantitative (e.g., vena cava width, annulus dilation), and qualitative measures (e.g., RV size, valve morphology) [16].

Right Ventricular Function and Advances in Imaging

Right ventricular function (e.g., TAPSE, RV-FAC, RV-GLS) is critical for evaluating TR. Advances like 3D real-time echocardiography have improved differentiation between TR types. Novel transcatheter therapies may need additional imaging (e.g., CT, MRI) for procedural planning and risk assessment [17].

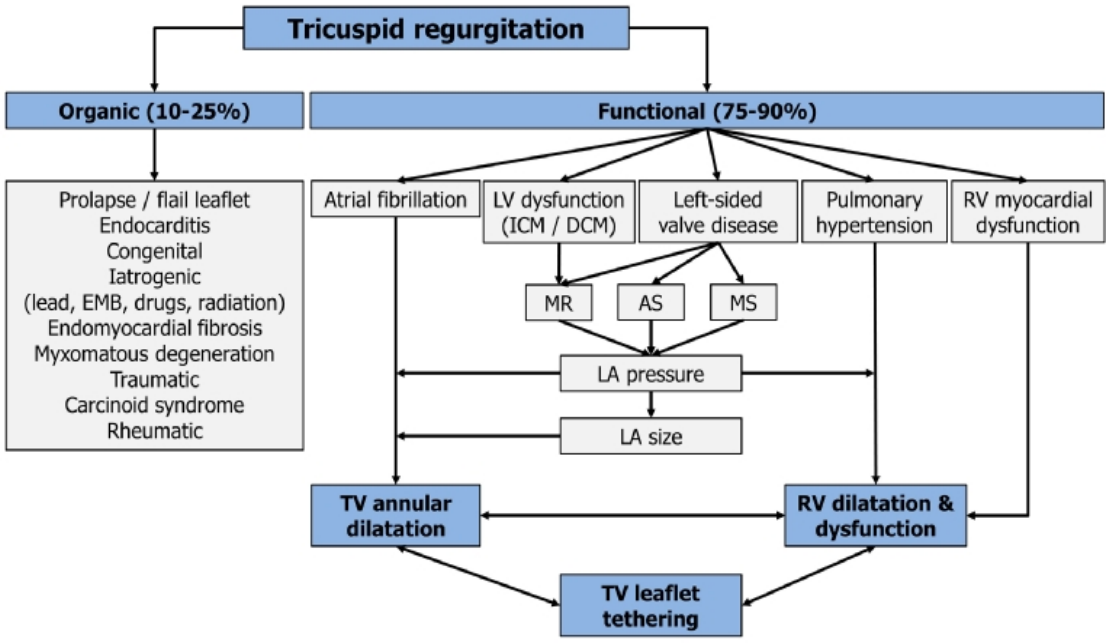

Figure (4): Causes of tricuspid regurgitation.

EMB: endomyocardial biopsy; DCM: dilated cardiomyopathy; ICM: ischaemic cardiomyopathy; AS: aortic stenosis; LV: left ventricular; LA: left atrial; MS: mitral stenosis; TV: tricuspid valve; RV: right ventricular; MR: mitral regurgitation [18].

Table 1. Causes of TR [2]

Primary TR
Congenital
Double orifice tricuspid valve
TV tethering related to perimembranous ventricular septal defect or aneurysm
Ebstein's anomaly
Tricuspid valve hypoplasia, cleft, dysplasia or
Other (giant right atrium)
Acquired
Endocarditis
Myxomatous degeneration (Barlow's illness): tricuspid valve prolapses, flail
Rheumatic illness
Carcinoid syndrome
Trauma (tricuspid valve trauma or chest wall trauma after intracardiac procedures: right ventricle intramyocardial biopsy, and so on)
Device / Pacemaker -related
Secondary Tricuspid Regurgitation
Regarding the underlying illness:
Left-sided heart illness (valve illness and/or LV dysfunction)
Right ventricle dysfunction from any etiology
Pulmonary arterial hypertension from any etiology
Idiopathic (no obvious etiology) commonly related to atrial fibrillation
With regard to the morphologic anomaly:
Dislocation of the papillary muscles
Tethering or tenting of tricuspid valve leaflets
Annular dilation
Right ventricle dilation / dysfunction

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TV = tricuspid valve, RV = right ventricle, RA = right atrium.

Clinical Features and Hemodynamics of Tricuspid Regurgitation

Detection and Clinical Features

Tricuspid regurgitation frequently still unobserved and is normally identified throughout evaluation for co-existing left-sided cardiac illness. Long-standing significant tricuspid regurgitation can lead to right cardiac failure symptoms, including ascites, peripheral edema, weight gain, and liver dysfunction. A faint systolic murmur may occasionally be heard, though it is frequently inaudible because of low right-sided pressures. Severe TR can produce a prominent C-V wave in the jugular pulse, and invasive testing may reveal ventricularization of right atrial pressure [19].

Pathophysiology:

Functional tricuspid regurgitation outcomes from leaflet malcoaptation due to tricuspid annular dilation. The septal leaflet is relatively fixed, limiting compensation for annular dilation as the distance among the right ventricle free wall and septum rises [20].

Hemodynamic Influences on TR

Preload and Afterload: RV preload and afterload significantly impact TR severity. Tricuspid valve regurgitation can worsen with increased afterload (e.g., pulmonary artery constriction) and improve with reduced preload (e.g., nitroprusside infusion) [19].

Pulmonary Artery Pressure (PASP): PASP is strongly correlated with TR severity, but the relationship is variable. Elevated left atrial pressures and left ventricular filling pressures can also indirectly increase TR [21].

Respiratory Cycle and Other Factors: Inspiration increases the regurgitant orifice and volume. Other factors influencing TR include RV size, valvular function, atrial fibrillation, and left heart function [22].

RA Pressure Findings

Severe TR is associated with elevated RA pressures, prominent V waves, and steep Y descents. The ‘‘ventricularization’’ of right atrium pressure is specific but found in a minority of cases [23].

RV Hemodynamic Findings

Significant TR may show elevated right ventricle end-diastolic pressure (RVEDP) and a ‘‘dip and plateau’’ pattern in right ventricle diastolic pressures. TR is also associated with decreased heart output and altered phasic waveforms in central venous pressures. Severe acute TR results in right ventricle and right atrium dilation, raised right ventricle pressures, and loss of function of atrial pump [24].

Pitfalls in Hemodynamic Assessment

Careful interpretation of waveforms and catheter placement is necessary to avoid artifacts that can mimic TR findings [19].

Types of repairs for Tricuspid Valve Disease

Tricuspid valve (TV) disease primarily manifests as regurgitation. Secondary or functional TV disease is generally more amenable to repair, while primary or organic deterioration is less likely to be repaired. Evidence favors repair over replacement, especially when annuloplasty is involved [25]. Functional tricuspid regurgitation often results from pulmonary hypertension caused by left-heart pathology and is associated with annular dilation despite the absence of organic lesions. Guidelines recommend treating severe regurgitation if left-heart operation is planned. For less severe regurgitation, surgery is indicated for a septal-anterior diameter not less than forty millimeters (or not less than twenty-one millimeters per square meter) or signs of progressive right ventricular dysfunction or dilation, even in asymptomatic cases, following prior left-side operation. The cornerstone of tricuspid valve repair is reducing right ventricular afterload and annular diameter [26].

Management of tricuspid valve regurge:

Medical Management

Patients with tricuspid regurgitation (TR) and cardiomyopathy are managed following AHA/ACC and ESC heart failure guidelines. Treatment includes diuretics and ACE inhibitors to address tricuspid valve regurgitation related to chronic cardiac failure and fluid overload. For patients with preserved kidney function, aldosterone antagonists like spironolactone or eplerenone can be added, particularly for heart congestion and 2^{ry} hyperaldosteronism. For 1^{ry} pulmonary hypertension, management options involve endothelin receptor antagonists, calcium channel blockers, phosphodiesterase type five inhibitors, prostacyclin analogues, prostacyclin receptor agonists in addition to guanylate cyclase stimulators. Anticoagulation is recommended for patients with right heart dilatation because of atrial fibrillation or pulmonary embolism, alongside therapies for rhythm control [27].

Surgical Management

Indications for Surgery

Surgical decisions are based on hemodynamic and functional impacts of TR and associated valvular or congenital lesions. Mild to moderate functional tricuspid regurgitation, if uncorrected throughout left-sided valve operation, might develop and lead to worse outcomes. Repair during mitral or aortic valve surgery improves functional outcomes and decreases cardiac-related mortality without significantly rising following operation risks for patients with preserved right ventricle function [28]. Operation is suggested for severe symptomatic tricuspid regurgitation following left-sided valve operation when right ventricle dysfunction or severe lung hypertension is absent. Isolated TV operation for severe

symptomatic tricuspid valve regurgitation is most effective before RV dysfunction develops, although late operation carries higher mortality risks due to comorbidities [18].

Tricuspid Valve Repair Techniques

Suture Annuloplasty

De Vega Procedure

Introduced in 1972, the De Vega annuloplasty decreases the tricuspid annular area using 2 parallel running sutures (2-0 Ticron or 4-0 polypropylene) located counterclockwise from the posteroseptal commissure to the antero-septal commissure. In fragile tissues, pledges may reinforce the sutures to prevent annular cutting [26].

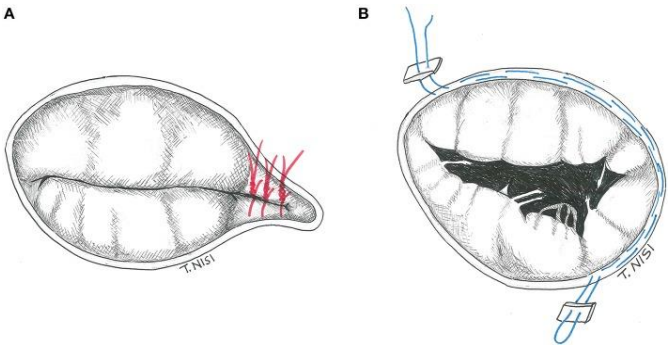

Figure (5): (A) The Kay procedure. (B) The De Vega method [25].

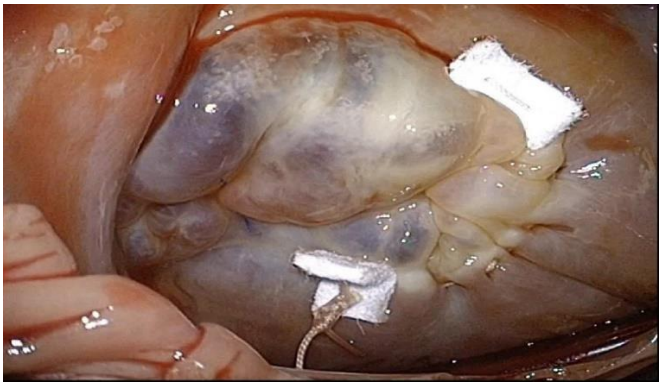

Figure (6): Targeted De Vega repair utilizing a pledgeted suture extending along the annulus from the anteroposterior commissure to the posterior extremity of septal part of the annulus [29].

Ring Annuloplasty

The utilization of a prosthetic ring for the purpose of reinforcing the tricuspid annulus was initially recognized by **Carpentier et al**. [30]. Flexible rings might diminish annular dilatation nevertheless they are unable to restore three-dimensional morphology. Semi-rigid or rigid rings are designed to stabilize the annulus throughout systole and restore the valve's physiologic geometry. Rigid rings are additionally designed to restore the valve's physiologic geometry. When determining the correct dimension of the ring, it is necessary to measure the distance among the antero-septal and posteroseptal commissures, which are located on the anterior leaflet surface. Eight to ten 2-0 Ti-Cron stitches are put in a counterclockwise direction during the implantation process. These stitches begin posteriorly at the middle of the septal leaflet. It is important to avoid causing any damage to the conduction system or the aortic root in close proximity of the septal and anterior leaflets. the final stitch is located over the anteroseptal commissure, and the ring is then parachuted and fixed [25].

Pericardial Band

A pericardial band is prepared from an autologous pericardial patch, immersed in glutaraldehyde solution, and washed with saline. The band length is determined utilizing the Carpentier Edward sizer to match the annulus. Pericardium is rolled with the smooth surface outward and reinforced with 2-0 Ethibond sutures. The band is then utilized to the annulus as an incomplete ring, preventing sutures in the annulus opposite the septal leaflet.

Seven 2-0 Ethibond sutures are typically situated along the posterior and anterior leaflets, with intervals of two to three millimeters in the pericardial band and five to six millimeters in the annulus, starting at the posteroinferior aspect of the septal leaflet and proceeding to anterior septal commissure. Variations in the De Vega repair involve utilizing pledgeted sutures or variant annular sizing approaches, like mitral sizers. A saline test is performed intraoperatively to assess valve competence by injecting saline into right ventricle [31].

CONCLUSION

Effective treatment of tricuspid valve regurgitation needs a multidisciplinary method, integrating advanced echocardiographic techniques with tailored surgical interventions. Repair techniques, including suture annuloplasty, ring annuloplasty, and pericardial band placement, provide superior outcomes compared to valve replacement, especially when combined with appropriate medical management. Early intervention during left-sided heart surgeries and timely repair in symptomatic patients with preserved right ventricular function are crucial for enhancing

survival and reducing complications. Further research into innovative materials and techniques may refine these strategies and improve patient outcomes.

DECLARATIONS

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- **Availability of data and material:** Accessible
- **Conflicts of interest:** No conflicts of interest.
- **Competing interests:** None

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