Strategies for tricuspid valve repair

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Abstract

Tricuspid valve repair is in many ways more challenging than mitral valve repair, especially since tricuspid valve anatomy is more complex with three leaflets, a saddle-shaped dynamic annulus and a complex subvalvular apparatus. The late referral of patients for tricuspid valve surgery adds to this challenge and contributes to poor prognosis. Nevertheless, studies have shown that the presence of moderate or greater tricuspid valve regurgitation leads to poor survival. Consequently, tricuspid valve surgery is now being performed more often, in order to improve the quality of life and survival. Tricuspid valve disease can be broadly classified into congenital and acquired tricuspid valve pathologies. Various repair techniques besides simple annular reduction maneuvers are used which are primarily aimed at restoring the complex interplay of various anatomical components. This review is a summary of the various operative techniques which provide successful reproducible results and achieve a competent and durable tricuspid valve repair with satisfactory late outcomes.

Keywords Tricuspid valve repair · Tricuspid valve · Cone repair

Introduction

The tricuspid valve (TV) has been labeled as a “neglected valve” for a long time since most patients tolerate right heart failure better and longer than left ventricular dysfunction [1, 2]. However, moderate or greater tricuspid valve regurgitation (TR) has now been identified as an independent risk factor for mortality irrespective of the left heart function [3]. Consequently, TV surgery is now being performed more often with the aim to improve survival and quality of life [4].

The TV has a complex anatomic structure that includes: (a) three leaflets, (b) a dynamic saddle-shaped annulus [5], (c) chordae and papillary muscles, and (d) underlying right ventricular (RV) myocardium. The competency of the TV depends on the interplay of these components [6]. Congenital TV pathologies can broadly be classified into Ebstein and non-Ebstein malformations [7, 8]. While Ebstein malformation (EM) is an RV myopathy with non-delamination of the leaflets, the non-EM congenital causes include leaflet

dysplasia, annular dilation from chronic RV volume overload in patients with left to right shunts, and right atrioventricular valve abnormalities of atrioventricular septal defects. Acquired TV pathology ranges from annular dilation (functional TR secondary to left-sided pathology or pulmonary hypertension) to ruptured chordae to dysplastic leaflets secondary to trauma from endocardial pacing leads [6].

The goal of this review is to provide a summary of the operative techniques used to repair TV, which provide successful, reproducible results, and satisfactory late outcome.

Preoperative evaluation

Imaging and Anatomy

The decision to proceed with TV repair should be made preoperatively and not in the operating room since the degree of TR is typically underestimated under anesthesia by at least one grade. A thorough preoperative evaluation of TV anatomy using transthoracic echocardiography is essential. We use transesophageal echocardiography (TEE) and 3-dimensional echocardiography to provide greater valvar anatomic detail if transthoracic images are insufficient. For patients with congenital causes and longstanding TV pathology, we use magnetic resonance imaging for information about RV size and

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function. Intraoperative TEE is used routinely for confirmation of preoperative findings and for post-repair assessment.

During TV repair, it is important to identify the anatomical landmarks with regards conduction tissue or the right coronary artery and its major branches in order to avoid any injury to them that can lead to adverse outcomes. The conduction tissue is located in the triangle of Koch, and in the absence of complex congenital heart disease, its location is consistent and constant. The right coronary artery runs in the atrioventricular groove, and the takeoff of the posterior descending and posterolateral branches is at the crux of the heart just anterior to the coronary sinus where the right ventricular free wall meets the ventricular septum. Since most repair techniques require suture placement into the annulus, inadvertent injury to either of these two structures can likely lead to poor outcomes.

The subvalvular apparatus of TV has more variable anatomy than the subvalvular apparatus of the mitral valve. Chordal support for the TV usually consists of one or two major papillary muscles arising from the anterior free wall, one in the region of the junction between the inferior RV wall and the ventricular septum. There are usually multiple chordae to the septal leaflet. Importantly, leaflets may get tethered as the normal crescent-shaped RV becomes dilated and rounded, pulling the leading edges of the tricuspid leaflets away from each other. This tethering of leaflets explains, in part, why simple annular reduction maneuvers may not suffice to eliminate central TR when there is severe RV dilation.

Principles of tricuspid valve repair

Operative steps

Surgical approach

We prefer to cannulate centrally via a median sternotomy with standard aortic and bicaval cannulation with right-angle venous cannulae. If a redo-sternotomy with very little sub-sternal space or a robotic-assisted TV repair is being planned, percutaneous cannulation of superior vena cava using Seldinger technique over a line placed in the neck into the internal jugular vein and peripheral groin cannulation of femoral vein and artery are preferred.

Cardioplegia

In general, we inspect the TV under cardioplegic arrest to ensure we understand the anatomical details and mechanism of TR. We incorporate the pulmonary artery in the cross-clamp so filling of the RV both at the beginning of the operation and at the end would facilitate analysis of valve competence and TV pathology. If upon inspection, we decide that repair is going to be restricted to a simple suture or ring

annuloplasty and the tissue quality appears to be good, we remove the cross-clamp and proceed to repair the TV on a beating heart. If it appears that repair will involve additional leaflet or subvalvar work, or if the tissue quality appears to be fragile, we perform the repair on an arrested heart. This prevents any motion trauma that can result in tearing of delicate leaflet or annular tissue while ensuring accurate suture placement and the tying/securing of sutures.

Exposure of tricuspid valve

The right atrium is incised parallel to atrioventricular groove starting from the right atrial appendage and heading halfway between inferior vena cava and the atrioventricular groove. We stay clear of the atrioventricular groove in order to avoid injury to the right coronary artery during atriotomy closure. Tacking sutures of monofilament 4–0 are placed above the anterior annulus of TV at 10:00 and 2:00; a third tacking suture is placed in the crista terminalis to reflect the lower edge of the atriotomy laterally. This opens the right atrium widely and brings the TV into complete view without the need on an additional right atrial retractor and allows for better assistance.

Valve inspection

Valve inspection is essential in order to identify the pathology and target the repair. The annulus is assessed for dilation, followed by leaflet examination—identification of fenestrations, dysplasia, flail segments, leaflet height, etc. The leaflets are retracted up with a blunt hook to visualize the subvalvar apparatus; the chordae and papillary muscles are assessed. After this anatomical assessment, saline is injected into the right ventricle and valve competency is analyzed. The site of the leak is identified, and the corresponding cause delineated. If the valve appears anatomically normal, and the leak is central, the cause is mostly due to a dilated annulus. Failure of coaptation may also be due to the relatively shorter height of the leaflet (anterior most common). The portion of tricuspid valve annulus that dilates the most corresponds to the inferior leaflet followed by anterior leaflet. In contrast to the mitral valve, the septal tricuspid annulus can also dilate (Fig. 1).

Ring Annuloplasty (Fig. 2a)

Since the geometry of the tricuspid annulus is saddle-shaped and changes throughout the cardiac cycle, our preference is a C-shaped flexible ring. The absence of a rigid prosthesis allows a normal interaction between the tricuspid annulus and right ventricle. This may help optimize right ventricular function in the long term. In adults, a 26 mm ring is used routinely, while a 28 mm ring is used in patients weighing more than 110 kg. We prefer to use the monofilament, 2–0 nonpledgeted horizontal mattress sutures since the monofilament

Fig. 1 Tricuspid annular dilatation. Maximum dilatation occurs along the inferior annulus followed by the anterior annulus

suture tends to slide through the delicate annular tissue a lot easier when compared to the more traumatic braided suture.

The first stitch in the annulus is placed at anteroseptal junction. Subsequent stitches are placed in a clockwise fashion along the anterior and inferior annulus. The last stitch is pledgeted and anchored in the os of the coronary sinus. This ensures that the ring stays intact as opposed to the more superficial bites along the septal annulus that are susceptible to

dehiscence. Care should be exercised in not placing this last stitch too deep as it may penetrate or injure the distal right coronary artery, posterior descending, or posterolateral coronary arteries. We avoid extending the suture placement into the septal annulus to minimize any potential injury to the conduction tissue. An additional pledgeted stitch is often placed at the anteroseptal junction.

Pacing or ICD lead repair (Fig. 2b–e)

Injury to TV due to pacemaker or ICD leads is becoming increasingly common [9]. Mechanisms leading to TR from lead injury include restricted mobility of normal-appearing leaflets or subvalvular apparatus, scarring or fusion of leaflets to each other, leaflet adherence to the leads, leaflet perforation, and/or chordal entrapment. Our experience has shown that a valve repair may not always be feasible due to the leaflet destruction from pacing lead injury, necessitating a valve replacement. However, every attempt to repair and salvage the TV should be made since our results have demonstrated that repair offers better long-term survival [9].

Before we attempt to repair the valve, we check the threshold of the ventricular leads. An effort to preserve the endocardial lead is made if the threshold is very good. If the threshold is not ideal or if lead removal is necessary to accomplish a successful repair, we excise the endocardial leads and proceed with TV repair. Once the repair is complete, an epicardial pacing system is placed in to replace the endocardial leads.

Fig. 2 a Ring annuloplasty with pledgeted stitches at anteroseptal and coronary sinus region. b–d Suture repair of leaflet, exteriorization of pacing lead and ring annuloplasty. e Pericardial patch augmentation of leaflet, exteriorization of pacing lead, and ring annuloplasty

If a decision has been made to preserve the endocardial lead, we are careful to use a rubber-protected forceps while manipulating the lead. This prevents any injury to its outer insulation. If the lead is adherent to a leaflet, it is carefully dissected off and the leaflet examined for any damage. If there is a hole or the leaflet tissue appears deficient, a pericardial patch may be used to plug the hole or augment the leaflet. If a part of the leaflet has to be sacrificed due to marked scarring/dysplasia, it can similarly be augmented with a pericardial patch. When the pacing lead is tangled in the subvalvar apparatus, the lead is mobilized as much as possible. The involved leaflet is then incised from the leading edge to annulus. We reposition the lead against the annulus at one of the commissures (usually infero-septal) to prevent any compromise of leaflet excursion. The leaflet is re-approximated from the leading edge up to the annulus leaving it slightly loose at the level of the lead-annulus junction.

If the mechanism of TR was due to restricted mobility of the leaflets without any evidence of leaflet penetration or chordal entrapment, the lead is positioned against the annulus at the most convenient commissure (usually antero-septal or infero-septal commissure). A loose commissural stitch is placed to prevent the lead from wandering into the orifice of the valve and impinging on leaflet excursion. A ring is applied at the completion of the procedure. The lead is usually maintained outside the ring, but should be movable so that future removal by laser extraction, if needed, is not compromised.

Artificial chordae (Figs. 3 and 4)

In the setting of a flail, unsupported leaflet, we use a double-armed, pledgeted Gore-Tex 5–0 as artificial chords. The

location of the artificial-chords depends on which leaflet is unsupported. If the anterior leaflet is flail, we anchor the Gore-Tex chords with pledgeted horizontal mattress sutures into the heavy RV trabeculation towards the apex at its junction with the ventricular septum with each arm of the suture placed through the apico-septal trabeculation without tying the suture at that level. Unlike mitral valve repair, we rarely place the artificial chords into the anterior papillary muscle(s) since measurement is difficult in the setting of a dilated RV that will eventually become smaller with RV remodeling. The anchoring point of the artificial chords towards the apex allows the coaptation between the anterior and septal leaflets, with the leading edge of the anterior leaflet brought closer to septal. This also makes the measurement of the chord length more reproducible since a severely dilated RV would make it difficult to gauge the appropriate chordal length if the artificial chord is attached to the papillary muscle(s).

Each limb of the suture is then passed through the leading edge of the unsupported segment of anterior leaflet twice (this allows the leaflet to retain its position without the leaflet slipping in or out too much prior to tying). Two or three pairs of artificial chords are used. The chord lengths are then adjusted to place the edge of the leaflet in the plane of the annulus. Annular traction sutures are temporarily loosened and the valve is examined with saline instillation into the RV. The position of the leaflet may be further adjusted inward or outward until valve competence is obtained. Sutures are then tied carefully (usually two-handed ties to ensure they are square) maintaining the leaflet at the same level.

When it is the septal leaflet which is unsupported, artificial chords are similarly directed towards the apex but more on the septal side. When the leaflet is thin and fragile, we use

Fig. 3 Artificial chords to anterior leaflet with pledgets anchored in heavy trabeculations towards the right ventricular apex

Fig. 4 Artificial chords to septal leaflet, with pledgets towards the right ventricle apex on septal side

pericardial pledgets when tying knots against the leaflet edge. The length of the chords adjusts itself with no knot on the apico-trabecular side, making sure that the edge of the leaflet lies at the level of the annulus. A ring is placed as described previously.

Leaflet augmentation repair (Fig. 5)

Ideally, we prefer successful leaflet coaptation with reasonable valve competence prior to annular reduction maneuvers. A deficient/dysplastic leaflet can be supplanted with autologous

pericardial patch to allow enough leaflet coaptation. If the height of the anterior leaflet upon valve inspection appears short, we patch augment the anterior leaflet with an ellipse of autologous, untreated pericardium. This increases the height to facilitate leaflet-to-leaflet coaptation. The anterior leaflet is incised off the annulus starting from 9:00 and extending it clockwise to approximately 4:00 towards the mid-point of inferior leaflet. An ellipse of fresh autologous pericardium is then harvested and sutured fixed at four points with 4–0 or 5–0 monofilament: 9:00, 12:00, 3:00, and 6:00. We run the sutures towards each other between the points of fixation with

Fig. 5 **a** Autologous pericardial patch augmentation of the anterior leaflet to increase leaflet height and improve coaptation. **b** Artificial chordae to the anterior leaflet are also shown

occasional locking stitches to prevent a purse-string effect. A ring is then applied.

Similarly, a damaged segment of leaflet from a pacing lead injury can be augmented with a triangular patch of autologous pericardium. The damaged leaflet edges are fashioned into a triangular defect, and a triangular piece of pericardium, of the desired size, is fixed at the three points of the defect with monofilament 5–0 sutures, respectively. The sutures are then run towards each other with occasional locking stitches.

Cone repair (Fig. 6)

EM accounts for 1% of congenital heart disease and results from RV myopathy that leads to failure of TV delamination [10]. The TV morphology in EM is extremely variable. It generally involves a large anterior leaflet with apical displacement of non-delaminated septal and inferior leaflets (septal more than inferior) creating a functional TV annulus further close to the apex of RV, and a proximal atrialized thinned out segment of RV which appears smooth and non-trabeculated. While numerous repair techniques (mostly monocusp) have been described with acceptable early and mid-term results, the need for TV re-repair has persisted with many. Cone repair provides near anatomical repair of the TV and can be performed across all age groups. In our experience, it is safe and has provided satisfactory results with sustained reduction of TR and beneficially impacts RV function with effective RV remodeling in the long term [7]. However, it does have a significant learning curve [7].

The principle of cone repair is complete surgical delamination and recruitment of all undelaminated leaflet tissue. This recruited leaflet tissue is then anchored to the true right atrio-ventricular junction, creating a 360-degree “leaflet cone” of the neo-TV [7, 11]. Running monofilament sutures (locking every other stitch) are carefully used to approximate the delaminated leaflets to avoid a purse-string effect, which can otherwise potentially shorten the height of the reconstructed

leaflet. If it appears that leaflet tissue is insufficient, pericardial patch augmentation is done. If the anterior leaflet is diminutive, it is detached from the annulus from 9:00 to 4:00 and an ellipse patch of bovine or autologous pericardium is placed with tacking sutures at 9:00, 12:00, 3:00, and 6:00 positions and then monofilament sutures are run towards each other from these tacking points (Fig. 6a). If despite surgical delamination, the cone appears deficient in the septal area, a triangular pericardial patch augmentation between the septal and inferior leaflets of the newly fashioned TV cone can be done (Fig. 6b). The leaflet height can also be increased by several small plications along the annular free edge of the leaflet (Fig. 6a). Autologous neochordae may be created surgically when there is a linear attachment of the leading edge of the anterior or septal leaflets to the right ventricular endocardium by performing several fenestrations apically in the linearly attached leaflet at the distal one third of the cone; this creates autologous neochordae. This also allows unrestricted forward blood flow into the ventricle (Fig. 6c). Finally, Gore-Tex 5–0 artificial chords could also be placed as described previously to provide added leaflet support for areas with absent chordal support. The eventual neo-TV cone has two orifices, the proximal orifice which is located at the level of true annulus after reattachment and a distal orifice towards the apex of the cone (Fig. 7).

Plication of the atrialized, inferior RV is performed internally in a horizontal fashion starting from the apex and going towards the true annulus (Fig. 8a–c). Since the true annulus is often dilated, the plication typically crosses the true annulus to partially downsize it. The plication bites are placed in a linear equally spaced fashion that take only part of the endocardial RV wall (they are never full thickness) to avoid distortion or compromise of the right coronary artery and its branches, especially at the crux of the heart just anterior to the os of coronary sinus. If there is a discrepancy in size between the true annulus and the neo-TV, additional annular plication is performed as needed at multiple sites around the anterior and

Fig. 6 **a** Pericardial patch augmentation of anterior leaflet, **b** triangular patch augmentation of cone between the septal and inferior leaflets, **c** creation of autologous chordae by making surgical fenestrations in the linear attachment of the delaminated leaflet

Fig. 7 Cone repair: two orifices, proximal inflow orifice at the level of the annulus, distal outflow orifice

inferior annulus to avoid an exaggerated plication of atrialized RV in one location that could compromise the right coronary artery. On occasion, a dilated RV prevents central coaptation of the neo-TV despite downsizing the annulus as it pulls on the leaflet. If patch augmentation of the leaflet does not overcome this tethering, a modified Sebening stitch which involves approximating the head of the mobilized “base-intact” papillary muscle to the ventricular septum; we generally try and avoid this stitch since dehiscence can occur, particularly in the setting of severe RV dilation.

The neo-TV is attached to the true annulus with continuous monofilament suture with the septal leaflet attached to the ventricular side of the conduction tissue. A flexible annuloplasty band (anteroseptal commissure clockwise to

inferoseptal commissure as described previously) is used to stabilize the repair as the true tricuspid annulus is usually much dilated. The band also reduces the tension on the neo-TV attachment to the annulus. The normal adult TV annular size is 20 to 24 mm. A useful guide to prevent any stenosis is the ability to pass a Hegar dilator ≥ 20 mm in size through the newly created TV orifice. The use of a 26 mm ring is possible in many younger patients without creating stenosis since the annulus is usually very dilated. In children less than 5 years of age or when somatic growth is a concern, only partial band support from anteroinferior to inferoseptal commissure with a pericardial strip is used as this is the area under the greatest tension. The reanchoring of the neo-TV, which consists of surgically delaminated circumferential leaflet tissue, with its hinge point at the atrioventricular groove mimics normal TV anatomy, namely, an “anatomic repair.”

Comment

The TV repair, if feasible, should be the preferred approach to TV disease with several studies showing improved survival benefit with repair irrespective of the etiology, whether it is secondary TR [12], organic [13], congenital [14], or even traumatic TR [9]. In many ways, TV repair is more challenging and less forgiving than mitral valve repair. Leaflet tissue tends to be more fragile, and the severely dilated RV with altered geometry makes successful leaflet coaptation and competency more difficult. In addition, there are multiple aspects of tricuspid surgery that can result in harm, specifically coronary or conduction tissue injury.

While TV repair is generally the goal, there are some situations where replacement may be preferred. This includes patients with pulmonary hypertension, massive annular or severe RV dilation, and those with marked abnormalities with leaflets or subvalvar apparatus where a repair is not feasible or unlikely to provide satisfactory long-term results. Some surgeons suggest a valve replacement may be a better option if

Fig. 8 a–c Plication of atrialized right ventricle

future re-repair is to be avoided [15]. However, given the risk of prosthetic valve complications, and the low morbidity associated with re-repair, we believe that TV repair should be the preferred approach when feasible [16].

The goal of any TV repair is to achieve a competent and durable repair. A pragmatic approach to TV repair adds further credence. When the primary indication for surgery is organic TR, it is more important to accept an excellent result ($\leq$ mild TR). This is in contrast to secondary TR (e.g., in the setting of mitral regurgitation) where the degree of TR is expected to improve after correction of the left-sided pathology, and accepting a less than perfect result ($\leq$ moderate TR) would be the goal. Finally, it is important that the choreography of this (and every) operation should be neatly orchestrated into a series of steps that will ensure that the operation is predictable and reproducible [17].

Compliance with ethical standards Ethical statement, informed consent, human and animal rights statement not applicable being a review article

Conflict of interests The authors declare that they have no conflict of interest.

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