Tricuspid Valve Repair for Treatment and Prevention of Secondary Tricuspid Regurgitation in Patients Undergoing Mitral Valve Surgery

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Secondary or functional tricuspid regurgitation occurring late after mitral valve surgery is associated with high morbidity and mortality. In this article, we review the pathophysiology of secondary tricuspid regurgitation and the evidence supporting the use of tricuspid valve annuloplasty for preventing and treating secondary tricuspid regurgitation. Liberal application of tricuspid valve annuloplasty is recommended to prevent progression of secondary regurgitation, as contrary to widely held opinion, fixing the left-sided valve dysfunction often does not resolve secondary tricuspid valve dysfunction. Based on existing literature, assessing the tricuspid valve annular dimensions can be recommended as part of all mitral valve operations, and annuloplasty strongly considered in patients with tricuspid annular dilatation or moderate to severe tricuspid regurgitation.

Introduction

Secondary or functional tricuspid regurgitation refers to tricuspid regurgitation, typically seen in association with left-sided valve dysfunction and occurring in the absence of macroscopically visible pathology of the tricuspid valve leaflets or chordae. The term "functional" has been used to describe this form of tricuspid regurgitation for several decades. As early as 1950, clinicians described methods of differentiating functional tricuspid regurgitation from "organic" tricuspid regurgitation [1]. This distinction became increasingly relevant in the 1960s with the advent of valve replacement, as surgery's role in managing valvular heart disease expanded rapidly. The thinking at the time was that if regurgitation was "functional," then it should not require surgical treatment as, by definition, it should improve when the left-sided valve is replaced. Reduction in severity of tricuspid regurgitation was sometimes observed after mitral valve replacement, prompting Braunwald et al. [2] in 1967 to recommend conservative management of functional tricuspid regurgitation [2]. Functional tricuspid regurgitation in patients undergoing mitral valve surgery was historically managed conservatively. However, in 1974, Carpentier et al. [3] reported excellent results with tricuspid valve repair and argued for systematic repair of functional tricuspid regurgitation during mitral valve surgery. It was later observed in the 1980s that patients who had undergone successful mitral surgery sometimes returned years later with severe symptomatic tricuspid regurgitation, and when these patients were reoperated on, mortality was very high [4]. This raised the question as to whether the tricuspid regurgitation should have been fixed during the first procedure, supporting Carpentier et al.'s [3] assertion more than a decade earlier that "surgical abstention" may be a somewhat dangerous policy [3].

Despite these observations, only a minority of cardiologists and surgeons embraced using tricuspid valve repair for functional tricuspid regurgitation, and surgical abstention continues in many centers to the present day. This strategy has been increasingly scrutinized since Dreyfus et al. [5••] reported that patients having tricuspid valve repair at the time of mitral valve surgery did better in the long term compared with patients who did not. An increasing wealth of observational data now supports surgical treatment of functional tricuspid regurgitation. In this article, we review the basis for recommending tricuspid valve repair for functional tricuspid regurgitation.

Pathophysiology of Secondary Tricuspid Regurgitation

The term functional tricuspid regurgitation is somewhat of a misnomer. The distinction from organic tricuspid regurgitation infers a normal valve free from disease with

regurgitation occurring due to volume or pressure overload on the valve rather than an intrinsic valve problem. In reality, however, the mechanism of this regurgitation is more complex, and it is unlikely the valve is truly "normal." Using echocardiographic analysis of 109 patients, Sagie et al. [6] demonstrated as early as 1994 that pulmonary hypertension and right ventricular dilatation were not prerequisites for developing functional tricuspid regurgitation. The most consistent feature they found was tricuspid annular dilatation. Recent echocardiographic studies have validated this finding by demonstrating abnormal geometry and function of the tricuspid valve in patients with functional regurgitation [7•,8•]. Because of these valve annular abnormalities, the tricuspid valve cannot be said to be truly disease free. We prefer to use the term "secondary" rather than "functional" to denote a (presumed) pathophysiologic etiology of left-sided valve disease, intrinsic pulmonary disease, or pulmonary valve disease, in contradistinction to primary tricuspid regurgitation, in which the principal etiology is a pathologic process within the tricuspid valve, such as rheumatic or infective infiltration of the valve.

Pulmonary hypertension

The pathophysiologic explanation for secondary tricuspid regurgitation in patients with left-sided valvular disease is a rising left atrial pressure, transmitted through the lungs as pulmonary arterial hypertension, resulting in pressure overload on the right ventricle. This pressure overload can directly result in tricuspid regurgitation or more typically causes right ventricular dilatation, which leads to regurgitation by simple dilatation of the tricuspid valve annulus (Carpentier type I regurgitation [9]) or by tethering of the tricuspid valve leaflets (Carpentier type IIIb regurgitation [9]). The degree of pulmonary hypertension and septal leaflet tethering has been shown by three-dimensional echocardiography to be moderately predictive of secondary tricuspid regurgitation severity [10]. At least in theory, reduction in degree of pulmonary hypertension (eg, by mitral valve repair) could result in less tricuspid regurgitation, but this would first require reverse remodeling of the previously dilated right ventricle, which may not be instantaneous. Therefore, correction of the left-sided valvular dysfunction alone cannot be relied upon to eliminate tricuspid regurgitation in the immediate postoperative period. An assessment of the success of isolated mitral valve surgery in reversing secondary tricuspid regurgitation should not be made until maximal reverse remodeling of the right ventricle has taken place. In practice, complete reverse right ventricular remodeling may not occur, and normalization of pulmonary artery pressures alone will not eliminate tricuspid regurgitation in many patients. This is well demonstrated in the pulmonary thromboendarterectomy literature, in which moderate to severe secondary tricuspid regurgitation is reported postoperatively in 30% to 45% of patients despite successful reduction in pulmonary arterial pressure [11,12]. It is arguable that, as is the case with the mitral valve, "functional" tricuspid regurgitation resulting from ventricular remodeling should be treated as a ventriculoannular disease requiring an annular, and possibly ventricular, solution.

Papillary muscle displacement

Although it was recognized in the 1990s that pulmonary hypertension was not a prerequisite to developing secondary tricuspid regurgitation [6], alternative pathophysiologic mechanisms have only recently been proposed. Modern echocardiographic imaging, particularly real-time three-dimensional echocardiography, has helped clarify the pathophysiology of secondary tricuspid regurgitation. Fukuda et al. [7•], among others, have observed septal leaflet tethering in patients with secondary tricuspid regurgitation who have normal pulmonary artery pressures. The presumed mechanism for secondary tricuspid regurgitation with normal pulmonary artery pressure is left ventricular dysfunction or dilatation. Because the two ventricles are interdependent at the septum, left ventricular septal dysfunction also causes dysfunction of the right ventricle's septal wall, the area of origin of the papillary muscles to the septal leaflet of the tricuspid valve. Through this mechanism, there can be a tethering effect on the tricuspid valve, causing Carpentier IIIb regurgitation, when the interventricular septum is hypokinetic, dyskinetic, or dilated. Such regurgitation can occur irrespective of the size and function of the right ventricle. This may explain why left ventricular dysfunction is an independent risk factor for secondary tricuspid regurgitation (independent of the effect of pulmonary hypertension) [7•]. Kim et al. [13•] support this theory in an echocardiographic study of 75 patients with right ventricular dilatation. They found that the eccentricity of the right ventricle, tricuspid valve tethering area, and end-diastolic tricuspid annular diameter were predictive of the severity of secondary tricuspid regurgitation. The right ventricular dimensions, right ventricular function, and pulmonary artery pressures were not significant predictive factors, suggesting that change in geometry of the right ventricle, and the consequent papillary muscle displacement, is the critical factor in the pathophysiology of tricuspid regurgitation. Whereas such eccentric displacement of the ventricle is more likely to occur in the globally dilated ventricle, it can also occur in normal-sized ventricles [7•].

Tricuspid annular dysfunction

Tricuspid annular dilatation has long been recognized as a constant feature of secondary tricuspid regurgitation [3,6,14]. Recently, other abnormalities in the annulus have been discovered. Using three-dimensional echocardiography, Fukuda et al. [15], Sukmawan et al. [16•], and Ton-Nu et al. [8•] independently demonstrated in recent studies that, unlike the saddle-shaped annulus in normal

subjects, valves with secondary tricuspid regurgitation are dilated, flattened, and circular. Fukuda et al. [15] additionally demonstrated an asymmetric reduction in tricuspid annular contraction in patients with secondary tricuspid regurgitation. The role of tricuspid annular dysfunction is important because if it is a critical pathophysiologic factor in secondary tricuspid regurgitation development, then the presence of annular dilatation predisposes a patient to future regurgitation. In the future, three-dimensional echocardiography may identify patients at risk for secondary tricuspid regurgitation.

Implications for surgical therapy

Based on current understanding of pathophysiology, principles of surgical therapy for secondary tricuspid regurgitation include the following:

1. Elimination of increased afterload to the right ventricle by correction of left-sided valve dysfunctions and optimization of left ventricular function.
2. Maximization of right ventricular remodeling by reducing pulmonary hypertension. Correction of left-sided lesions often suffices, but in cases in which pulmonary hypertension persists, oral pulmonary vasodilators such as sildenafil and bosentan may be helpful in promoting reverse remodeling of the right ventricle [17].
3. Correct tricuspid annular dilatation and dysfunction. This usually consists of a tricuspid valve annuloplasty to restore annular size and geometry.

Rationale for Surgical Correction of Secondary Tricuspid Valve Disease at the Time of Mitral Valve Surgery

Nonregression or progression of tricuspid regurgitation after mitral valve surgery

Tricuspid regurgitation is frequently present months or years after isolated mitral valve surgery. Although repair of left-sided valve dysfunction may reduce the severity of tricuspid regurgitation, a substantial proportion of patients will go on to develop moderate or severe regurgitation. In one series, 43% of patients had severe tricuspid regurgitation at a mean follow-up of 11 years after isolated mitral valve replacement [18].

The percutaneous mitral valve balloon dilatation experience is an ideal model to examine the natural course of tricuspid regurgitation after correction of left-sided valve dysfunction, as in these patients, there is usually prompt reversal of pulmonary hypertension with relief of mitral stenosis and there are no issues of surgical compromise to ventricular function. Sagie et al. [19] were among the first to study the natural course of tricuspid regurgitation after isolated balloon dilatation. Having previously observed a high mortality rate in patients with severe tricuspid regurgitation who underwent valvuloplasty (4-year survival 69% [19]), they examined the outcome in a subset of 20 patients with severe tricuspid regurgitation who had hemodynamically effective relief of mitral stenosis; they found that tricuspid regurgitation degree decreased in only four patients [20]. This was recently corroborated by a study from Song et al. [21•], who compared isolated percutaneous balloon dilatation to mitral valve surgery combined with tricuspid valve repair in 92 patients who had mitral stenosis and severe tricuspid regurgitation. At 1-year follow-up, moderate or severe tricuspid regurgitation persisted in 54% of patients who underwent the percutaneous mitral procedure, compared with only 2% of those who received a tricuspid valve repair with a mitral valve repair or replacement. The nonresolution of "functional" tricuspid regurgitation after correction of the left-sided valve dysfunction has also been noted in the surgical literature, in which 35% to 70% of patients with significant regurgitation at the time of mitral surgery are reported as having persisting moderate or severe regurgitation at mid-term follow-up [18,22,23•].

Although in some patients, significant tricuspid regurgitation will resolve with correction of the left-sided lesion, there is no means of accurately predicting this at the time of mitral surgery; therefore, an additional strategy is required if reducing the prevalence of late tricuspid regurgitation is desired.

Progression of tricuspid valve dysfunction in patients with a dilated annulus

Groves et al. [24] demonstrated an association between tricuspid annular dilatation at the time of initial mitral valve replacement and subsequent development of severe tricuspid regurgitation in the long term. Colombo et al. [25] subsequently systematically repaired all tricuspid valves with an annular diameter greater than 21 mm on echocardiography in patients undergoing mitral valve surgery, although they noted that 30% of patients with annular dilatation did not have severe regurgitation on preoperative echocardiography. This approach of tricuspid repair was popularized by Dreyfus et al. [5••], who also based their decision to operate on tricuspid annular dilatation, but using direct surgical measurement rather than echocardiography. However, the threshold of Dreyfus et al. [5••] for repairing the tricuspid annulus (> 70 mm in a flaccid heart) was likely too stringent, as a third of patients who fit their criteria of a "normal"-sized annulus developed moderate or severe tricuspid regurgitation in the follow-up period. We believe a dilated annulus infers functional and structural abnormality of the tricuspid valve (annulus), and possibly of the right ventricle, a view supported by recent echocardiographic data [8•,15,16•]. At least in some patients, annular dilatation likely represents the early stages of secondary tricuspid regurgitation. In those instances, a ring annuloplasty by reversing annular dilatation could theoretically halt progression to severe secondary tricuspid regurgitation.

Mortality and morbidity associated with tricuspid regurgitation occurring after mitral valve surgery

A substantial rise in mortality and morbidity is associated with severe tricuspid regurgitation occurring after mitral valve surgery. The early mortality, long-term survival, freedom from heart failure, and functional outcome are all significantly worse for patients with severe tricuspid regurgitation compared to those without severe regurgitation [19,24,26–28]. Moderate tricuspid regurgitation, regardless of etiology, is also associated with inferior survival in the nonsurgical setting, but to a lesser degree than for severe regurgitation (79% 1-year survival for moderate, compared to 64% for severe and 90% to 92% for patients with mild or none, respectively [26]). About half the patients who develop severe tricuspid regurgitation will be very symptomatic in New York Heart Association class III or IV [29]. Therefore, the burden of secondary tricuspid regurgitation occurring after mitral surgery is substantial.

Although surgery can be offered to these patients, it may not improve survival and quality of life at this late stage. The mortality of reoperative tricuspid valve surgery is about 10% to 25% [4,30–32], or substantially higher [33,34]. The outlook remains guarded even for survivors of reoperative surgery because of a high rate of persistent or recurrent heart failure and continued elevated risk of death despite resolution of tricuspid regurgitation; the 5-year event-free survival in one series was reported to be 42% [32], and in another, the 3-year patient survival was 19% [33]. This high rate of mortality and failure of resolution of heart failure may partly explain why patients with severe tricuspid regurgitation after mitral valve surgery are often not offered surgical therapy. Because of the poor prognosis for tricuspid regurgitation occurring after mitral valve surgery and limited effectiveness of surgical therapy at this late stage, strategies to prevent or delay late tricuspid regurgitation onset offer the best hope of reducing the net burden of mortality and morbidity associated with the condition.

Indications for Concomitant Tricuspid Valve Repair for Secondary Tricuspid Regurgitation in Patients Having Mitral Valve Surgery

Severe tricuspid regurgitation

Most authorities would now recommend repairing the tricuspid valve in any patient with severe tricuspid regurgitation undergoing any heart operation. The American College of Cardiology and American Heart Association guidelines recommend repair of all valves with severe tricuspid regurgitation in patients undergoing mitral valve surgery [35•].

Moderate tricuspid regurgitation

Patients with moderate degrees of tricuspid regurgitation require careful consideration, particularly because of the possibility that the severity may have been underestimated. The severity of secondary tricuspid regurgitation is highly dependent on conditions of preload, afterload, and right ventricular function. Vasodilators, diuretics, inotropes, fasting, and general anesthesia may result in downgrading of severe tricuspid regurgitation [36]. Any patient who has any prior documentation of severe regurgitation should probably undergo tricuspid valve repair, as severe regurgitation at any point infers an abnormally functioning valve. Coexisting pulmonary hypertension is a relative indication for tricuspid valve repair [35•], and if present in the setting of moderate regurgitation, it should alert the clinician to the possibility that regurgitation severity is being underestimated. Echocardiographic assessment of tricuspid regurgitation severity should not be done intraoperatively, as patients with moderate or severe regurgitation may have only minimal regurgitation when optimized under conditions of general anesthesia.

Because of the secondary tricuspid regurgitation's dynamic nature, patients with mild or moderate degrees of tricuspid regurgitation may still have severe valvular dysfunction. Regardless of severity, any degree of tricuspid regurgitation other than trivial or mild is undesirable, as even moderate tricuspid regurgitation is associated with reduced long-term survival [26]. Evidence from surgical series suggests that the severity of mild to moderate tricuspid regurgitation occurring after mitral valve surgery increases over time and is associated with poorer long-term survival and higher reoperation rates [5••,23•,29,37]. For these reasons, we also recommend tricuspid valve repair for patients with documented moderate tricuspid regurgitation.

Tricuspid annular dilatation

In the early 1970s, Carpentier et al. [3] were the first to recommend tricuspid annular dilatation as an indication for tricuspid valve repair; their method of assessing dilatation was surgical exploration of the valve and the ability of the annulus to admit three fingerbreadths of the surgeon's hand, in which case it would be repaired. Despite subsequent emergence of evidence that tricuspid annular dilatation predisposed to long-term development of tricuspid regurgitation [24], Carpentier's approach of using annular dilatation as an indication for surgery was largely ignored by surgeons for the next three decades. However, following recent reports from two surgical centers that use annular dilatation as the indication for concurrent tricuspid valve repair [5••,25], surgeons have become more receptive to the notion that a dilated tricuspid annulus is diseased and should be fixed to prevent late development of tricuspid regurgitation [38]. Our group, among others, has recently adopted tricuspid annular dilatation as the principal determining criterion for adjunctive tricuspid valve repair [39•]. Because patients with annular dilatation will not necessarily have significant regurgitation on echocardiography (Fig. 1), the regurgitation severity alone is probably not sufficient to screen for secondary tricuspid valve disease [5••].

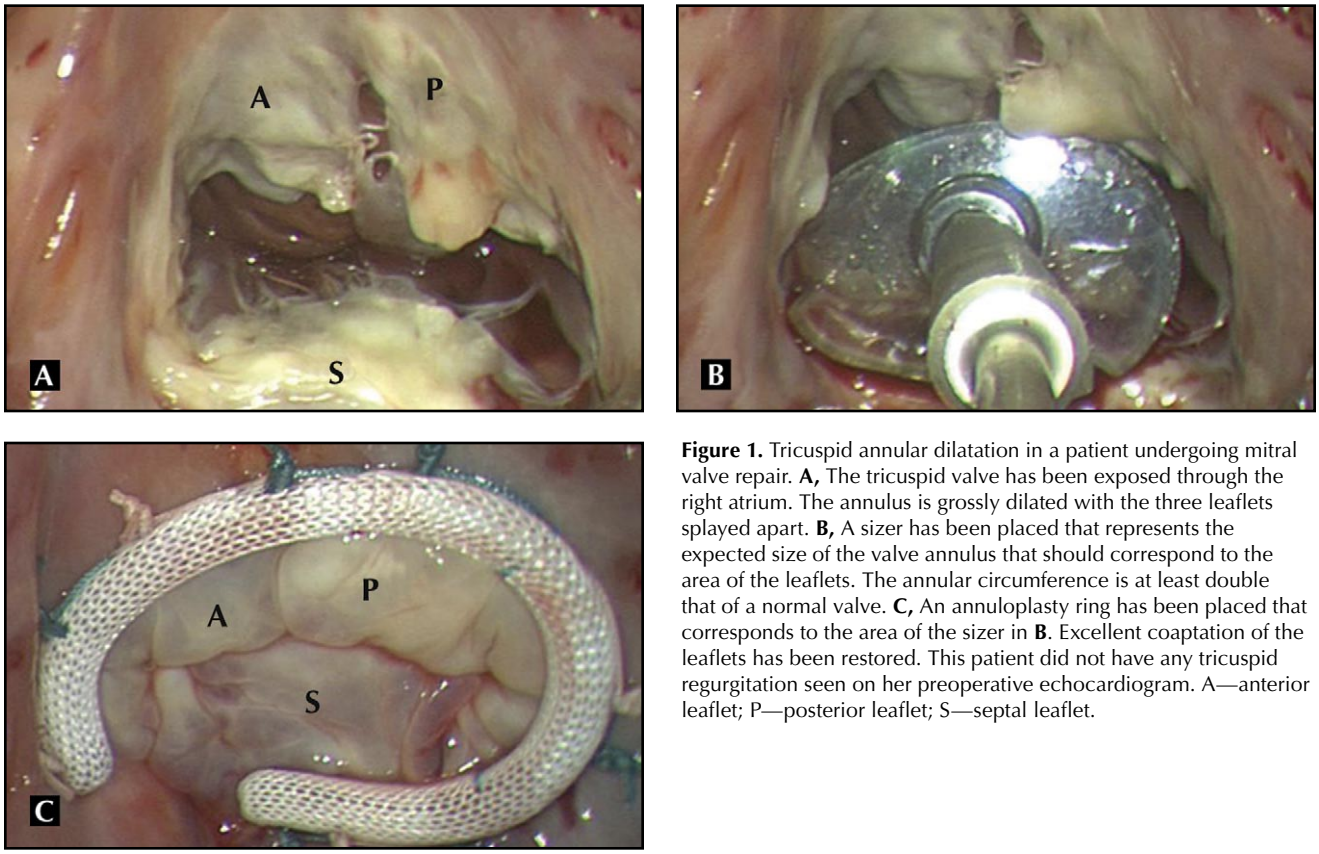

Figure 1. Tricuspid annular dilatation in a patient undergoing mitral valve repair. **A,** The tricuspid valve has been exposed through the right atrium. The annulus is grossly dilated with the three leaflets splayed apart. **B,** A sizer has been placed that represents the expected size of the valve annulus that should correspond to the area of the leaflets. The annular circumference is at least double that of a normal valve. **C,** An annuloplasty ring has been placed that corresponds to the area of the sizer in **B**. Excellent coaptation of the leaflets has been restored. This patient did not have any tricuspid regurgitation seen on her preoperative echocardiogram. A—anterior leaflet; P—posterior leaflet; S—septal leaflet.

Several methods of determining annular dilatation have been described based on echocardiography [25], direct surgical measurement of maximal diastolic intercommissural dimensions in a flaccid heart [5••], or direct assessment of the ratio of anterior and posterior leaflet area to the annulus area [39•]. Assessing the annular diameter should be part of intraoperative transesophageal echocardiographic assessment. An abnormal intercommissural annular diameter recorded on any view of more than 35 mm likely represents a dilated annulus; however, a normal annulus on two-dimensional echocardiography does not necessarily infer the lack of annular dilatation [40]. Three-dimensional echocardiography is probably the most accurate method of assessing tricuspid annulus dilatation [8•,10,15,16•,40] and may supersede two-dimensional echocardiography and direct surgical evaluation as the clinical tool of choice for assessing tricuspid annular dilatation. Because of the difficulties in assessing the tricuspid valve annulus on two-dimensional echocardiography, our surgical approach includes routine inspection of the tricuspid valve in all mitral valve repairs [39•]; currently, 70% of our mitral valve repair patients receive concurrent tricuspid valve repair for annular dilatation or valve regurgitation.

Systematic tricuspid valve repair

Dreyfus et al.'s [5••] observation of lower long-term incidence of significant tricuspid regurgitation and better symptom status in patients having concomitant tricuspid valve repair at the time of mitral valve repair, and also the late occurrence of significant regurgitation in some patients who had a nondilated annulus at initial surgery, raises the question as to whether all patients should have a tricuspid repair at the time of mitral surgery. We believe that because of the low risk of tricuspid repair, the high prevalence of late secondary tricuspid regurgitation, the inability to reliably predict which patients will develop secondary regurgitation, and the high mortality and morbidity associated with severe tricuspid regurgitation, it may ultimately prove beneficial to perform prophylactic tricuspid repair in most patients having mitral valve surgery regardless of the presence of annular dilatation or regurgitation. However, a clinical trial will be necessary to determine the efficacy and safety before this approach can be recommended.

Surgical Techniques for Tricuspid Valve Repair

Two principal surgical methods are used to treat or prevent secondary tricuspid regurgitation: the ring annuloplasty method introduced by Carpentier et al. [41] (Fig. 1C) and the suture annuloplasty method described by De Vega et al. [42]. With the ring annuloplasty technique, the annulus is permanently fixed in a systolic position by suturing in a rigid or semirigid ring (the procedure may

also be done with a flexible band, which may allow annular movement), whereas in the approach by De Vega et al., the annulus size is reduced by using a continuous suture to "purse string" the annulus, relying on continued integrity of the suture and annular contraction and fibrosis to maintain the new annular dimensions. Although either method may be applied for secondary tricuspid regurgitation, recent long-term studies suggest that ring annuloplasty repairs are more durable than suture annuloplasty repairs [33,43]. Data from the surgical literature suggest that more than 85% of patients having a ring annuloplasty will be free from moderate or severe tricuspid regurgitation 10 years after the surgery [5••,33,43]. Therefore, tricuspid valve repair should be effective in reducing the prevalence of secondary tricuspid regurgitation late after mitral valve surgery. Future studies are required to confirm that the reduction in prevalence of secondary tricuspid regurgitation will transform into patient benefit.

Incremental Risks Associated with Tricuspid Valve Repair

Operative mortality

Tricuspid valve surgery has historically been associated with high operative mortality. In a review of all valve procedures reported to the Society of Thoracic Surgeons between 1994 and 2003, the mortality of tricuspid valve procedures, whether in isolation or in combination with a mitral valve operation, was 11% compared with 8% for isolated mitral valve operations [44]. However, these data cannot be reliably extrapolated to contemporary cohorts undergoing tricuspid repair for incidental tricuspid regurgitation or dilatation, as, historically, most surgeons operated on the tricuspid valve at an advanced stage when patients showed evidence of the clinical consequences of severe regurgitation. In the present era, the risk of concurrent tricuspid valve repair at the time of mitral valve surgery is probably negligible, as patients usually do not have associated end-organ dysfunction, which increases perioperative risk as was historically seen in patients having tricuspid valve surgery. Two groups who liberally applied tricuspid valve repair for annular dilatation at the time of mitral valve repair reported an operative mortality of 0.7% and 2% [5••,25]. Tricuspid valve repair takes 10 to 20 minutes, so it does not add significantly to the duration of cardiopulmonary bypass; it can be undertaken with the heart beating and need not prolong the clamp time in a sick ventricle. If there is incremental mortality risk of adding a tricuspid valve repair to concomitant cardiac surgery, it is probably negligible in most patients [39•] and would likely be offset by the survival benefit of preventing late tricuspid regurgitation.

Other risks

Other possible risks of adding a "prophylactic" tricuspid valve repair procedure include the risk of postoperative bleeding, pacemaker requirement, and endocarditis. None of these was significantly increased in a prospective study comparing mitral valve repair alone with concurrent tricuspid valve repair [5••]. Tricuspid valve repair may be associated with a higher rate of pacemaker requirement [5••,39•]; however, without a controlled study, it is difficult to ascribe this to the tricuspid valve repair with certainty, as many patients have also had multivalvular surgery and atrial fibrillation ablation, which also predispose to heart block. There is probably a small incremental risk in morbidity associated with adding a tricuspid valve repair, but the literature and also our own personal experience as an active mitral repair group suggest that this risk is small and likely to be outweighed by the benefit of avoiding the high morbidity associated with persisting tricuspid regurgitation.

Conclusions

Secondary tricuspid regurgitation commonly occurs in combination with left-sided valvular heart disease and will often not improve despite correction of the left-sided valve dysfunction. Because tricuspid regurgitation occurring after prior mitral surgery carries a poor prognosis, surgical repair of the tricuspid valve at the time of mitral valve surgery, preferably using a ring annuloplasty technique, is recommended for treating and preventing secondary tricuspid regurgitation. Assessing for tricuspid valve annular dilatation can be recommended in all mitral valve procedures, as identifying and treating annular dilatation may be associated with superior long-term outcomes. Future randomized trials will determine whether there is a role for prophylactic tricuspid valve repair in all patients undergoing mitral valve surgery.

Disclosures

David H. Adams is an inventor with royalties from Edwards Lifesciences. No other potential conflict of interest relevant to this article was reported.

References and Recommended Reading

Papers of particular interest, published recently, have been highlighted as:

- • Of importance
- •• Of major importance

1. Messer AL, Hurst JW, Rappaport MB, Sprague HB: **A study of the venous pulse in tricuspid valve disease.** *Circulation* 1950, **1:**388–393.
2. Braunwald NS, Ross J, Jr, Morrow AG: **Conservative management of tricuspid regurgitation in patients undergoing mitral valve replacement.** *Circulation* 1967, **35(4 Suppl):** I63–I69.
3. Carpentier A, Deloche A, Hanania G, et al.: **Surgical management of acquired tricuspid valve disease.** *J Thorac Cardiovasc Surg* 1974, **67:**53–65.
4. King RM, Schaff HV, Danielson GK, et al.: **Surgery for tricuspid regurgitation late after mitral valve replacement.** *Circulation* 1984, **70(3 Pt 2):**I193–I197.

5.•• Dreyfus GD, Corbi PJ, Chan KM, Bahrami T: **Secondary tricuspid regurgitation or dilatation: which should be the criteria for surgical repair?** *Ann Thorac Surg* 2005, **79:**127–132.
Prospective study comparing long-term outcomes in patients undergoing mitral valve repair with or without concomitant tricuspid annuloplasty.
6. Sagie A, Schwammenthal E, Padial LR, et al.: **Determinants of functional tricuspid regurgitation in incomplete tricuspid valve closure: Doppler color flow study of 109 patients.** *J Am Coll Cardiol* 1994, **24:**446–453.
7.• Fukuda S, Gillinov AM, Song JM, et al.: **Echocardiographic insights into atrial and ventricular mechanisms of functional tricuspid regurgitation.** *Am Heart J* 2006, **152:**1208–1214.
Two-dimensional echocardiography demonstrates importance of leaflet tethering as a mechanism of functional tricuspid regurgitation.
8.• Ton-Nu TT, Levine RA, Handschumacher MD, et al.: **Geometric determinants of functional tricuspid regurgitation: insights from 3-dimensional echocardiography.** *Circulation* 2006, **114:**143–149.
Three-dimensional echocardiography provides insight on mechanisms of tricuspid regurgitation.
9. Carpentier A: **Cardiac valve surgery—the "French correction".** *J Thorac Cardiovasc Surg* 1983, **86:**323–337.
10. Park YH, Song JM, Lee EY, et al.: **Geometric and hemodynamic determinants of functional tricuspid regurgitation: a real-time three-dimensional echocardiography study.** *Int J Cardiol* 2007, [Epub ahead of print.]
11. Sadeghi HM, Kimura BJ, Raisinghani A, et al.: **Does lowering pulmonary arterial pressure eliminate severe functional tricuspid regurgitation? Insights from pulmonary thromboendarterectomy.** *J Am Coll Cardiol* 2004, **44:**126–132.
12. Menzel T, Kramm T, Wagner S, et al.: **Improvement of tricuspid regurgitation after pulmonary thromboendarterectomy.** *Ann Thorac Surg* 2002, **73:**756–761.
13.• Kim HK, Kim YJ, Park JS, et al.: **Determinants of the severity of functional tricuspid regurgitation.** *Am J Cardiol* 2006, **98:**236–242.
Analysis of echocardiographic predictors of tricuspid regurgitation. Pulmonary hypertension did not independently correlate with regurgitation.
14. Tei C, Pilgrim JP, Shah PM, et al.: **The tricuspid valve annulus: study of size and motion in normal subjects and in patients with tricuspid regurgitation.** *Circulation* 1982, **66:**665–671.
15. Fukuda S, Saracino G, Matsumura Y, et al.: **Three-dimensional geometry of the tricuspid annulus in healthy subjects and in patients with functional tricuspid regurgitation: a real-time, 3-dimensional echocardiographic study.** *Circulation* 2006, **114(1 Suppl 1):**I492–I498.
16.• Sukmawan R, Watanabe N, Ogasawara Y, et al.: **Geometric changes of tricuspid valve tenting in tricuspid regurgitation secondary to pulmonary hypertension quantified by novel system with transthoracic real-time 3-dimensional echocardiography.** *J Am Soc Echocardiogr* 2007, **20:**470–476.
Study using three-dimensional echocardiography to identify and quantify changes such as tricuspid tenting volume and annular dilatation in functional tricuspid regurgitation.
17. Mogollon MV, Lage E, Cabezon S, et al.: **Combination therapy with sildenafil and bosentan reverts severe pulmonary hypertension and allows heart transplantation: case report.** *Transplant Proc* 2006, **38:**2522–2523.
18. Porter A, Shapira Y, Wurzel M, et al.: **Tricuspid regurgitation late after mitral valve replacement: clinical and echocardiographic evaluation.** *J Heart Valve Dis* 1999, **8:**57–62.
19. Sagie A, Schwammenthal E, Newell JB, et al.: **Significant tricuspid regurgitation is a marker for adverse outcome in patients undergoing percutaneous balloon mitral valvuloplasty.** *J Am Coll Cardiol* 1994, **24:**696–702.
20. Sagie A, Schwammenthal E, Palacios IF, et al.: **Significant tricuspid regurgitation does not resolve after percutaneous balloon mitral valvotomy.** *J Thorac Cardiovasc Surg* 1994, **108:**727–735.
21.• Song H, Kang DH, Kim JH, et al.: **Percutaneous mitral valvuloplasty versus surgical treatment in mitral stenosis with severe tricuspid regurgitation.** *Circulation* 2007, **116(11 Suppl):**I246–I250.
Observational study that reports better outcomes in mitral stenosis treated with mitral and tricuspid valve repair versus isolated percutaneous mitral balloon valvuloplasty.
22. Matsuyama K, Matsumoto M, Sugita T, et al.: **Predictors of residual tricuspid regurgitation after mitral valve surgery.** *Ann Thorac Surg* 2003, **75:**1826–1828.
23.• Matsunaga A, Duran CM: **Progression of tricuspid regurgitation after repaired functional ischemic mitral regurgitation.** *Circulation* 2005, **112(9 Suppl):**I453–I457.
Observational study documenting high incidence of postoperative tricuspid regurgitation in patients undergoing predominantly isolated mitral valve repair for ischemic mitral regurgitation.
24. Groves PH, Ikram S, Ingold U, Hall RJ: **Tricuspid regurgitation following mitral valve replacement: an echocardiographic study.** *J Heart Valve Dis* 1993, **2:**273–278.
25. Colombo T, Russo C, Ciliberto GR, et al.: **Tricuspid regurgitation secondary to mitral valve disease: tricuspid annulus function as guide to tricuspid valve repair.** *Cardiovasc Surg* 2001, **9:**369–377.
26. Nath J, Foster E, Heidenreich PA: **Impact of tricuspid regurgitation on long-term survival.** *J Am Coll Cardiol* 2004, **43:**405–409.
27. Izumi C, Iga K, Konishi T: **Progression of isolated tricuspid regurgitation late after mitral valve surgery for rheumatic mitral valve disease.** *J Heart Valve Dis* 2002, **11:**353–356.
28. Groves PH, Lewis NP, Ikram S, et al.: **Reduced exercise capacity in patients with tricuspid regurgitation after successful mitral valve replacement for rheumatic mitral valve disease.** *Br Heart J* 1991, **66:**295–301.
29. Boyaci A, Gokce V, Topaloglu S, et al.: **Outcome of significant functional tricuspid regurgitation late after mitral valve replacement for predominant rheumatic mitral stenosis.** *Angiology* 2007, **58:**336–342.
30. Kwon DA, Park JS, Chang HJ, et al.: **Prediction of outcome in patients undergoing surgery for severe tricuspid regurgitation following mitral valve surgery and role of tricuspid annular systolic velocity.** *Am J Cardiol* 2006, **98:**659–661.
31. Keogh BE, Kinsman R: *Fifth National Adult Cardiac Surgical Database Report 2003*. Henley-on-Thames, UK: Dendrite Clinical Systems; 2004.
32. Staab ME, Nishimura RA, Dearani JA: **Isolated tricuspid valve surgery for severe tricuspid regurgitation following prior left heart valve surgery: analysis of outcome in 34 patients.** *J Heart Valve Dis* 1999, **8:**567–574.
33. McCarthy PM, Bhudia SK, Rajeswaran J, et al.: **Tricuspid valve repair: durability and risk factors for failure.** *J Thorac Cardiovasc Surg* 2004, **127:**674–685.
34. Hornick P, Harris PA, Taylor KM: **Tricuspid valve replacement subsequent to previous open heart surgery.** *J Heart Valve Dis* 1996, **5:**20–25.
35.• American College of Cardiology/American Heart Association Task Force on Practice Guidelines; Society of Cardiovascular Anesthesiologists; Society for Cardiovascular Angiography and Interventions; Society of Thoracic Surgeons, Bonow RO, Carabello BA, Kanu C, et al.: **ACC/AHA 2006 guidelines for the management of patients with valvular heart disease: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (writing committee to revise the 1998 Guidelines for the Management of Patients With Valvular Heart Disease): developed in collaboration with the Society of Cardiovascular Anesthesiologists: endorsed by the Society for Cardiovascular Angiography and Interventions and the Society of Thoracic Surgeons.** *Circulation* 2006, **114:**E84–E231. [Published erratum appears in *Circulation* 2007, 115:E409.]
Regularly updated evidence-based practice guidelines.

36. Rivera JM, Vandervoort PM, Vazquez de Prada JA, et al.: **Which physical factors determine tricuspid regurgitation jet area in the clinical setting?** *Am J Cardiol* 1993, **72:**1305–1309.
37. Kuwaki K, Morishita K, Tsukamoto M, Abe T: **Tricuspid valve surgery for functional tricuspid valve regurgitation associated with left-sided valvular disease.** *Eur J Cardiothorac Surg* 2001, **20:**577–582.
38. McCarthy PM: **Adjunctive procedures in degenerative mitral valve repair: tricuspid valve and atrial fibrillation surgery.** *Semin Thorac Cardiovasc Surg* 2007, **19:**121–126.
39.• Filsoufi F, Salzberg SP, Coutu M, Adams DH: **A three-dimensional ring annuloplasty for the treatment of tricuspid regurgitation.** *Ann Thorac Surg* 2006, **81:**2273–2277.
Observational study reporting low mortality and effective resolution of tricuspid regurgitation in patients undergoing tricuspid annuloplasty.
40. Anwar AM, Geleijnse ML, Soliman OI, et al.: **Assessment of normal tricuspid valve anatomy in adults by real-time three-dimensional echocardiography.** *Int J Cardiovasc Imaging* 2007, **23:**717–724.
41. Carpentier A, Deloche A, Dauptain J, et al.: **A new reconstructive operation for correction of mitral and tricuspid insufficiency.** *J Thorac Cardiovasc Surg* 1971, **61:**1–13.
42. De Vega NG, De Rabago G, Castillon L, et al.: **A new tricuspid repair. Short-term clinical results in 23 cases.** *J Cardiovasc Surg (Torino)* 1973, **Spec No:**384–386.
43. Tang GH, David TE, Singh SK, et al.: **Tricuspid valve repair with an annuloplasty ring results in improved long-term outcomes.** *Circulation* 2006, **114(1 Suppl):**I577–I581.
44. Rankin JS, Hammill BG, Ferguson TB, Jr, et al.: **Determinants of operative mortality in valvular heart surgery.** *J Thorac Cardiovasc Surg* 2006, **131:**547–557.