Contemporary Reviews in Cardiovascular Medicine

The Tricuspid Valve

Current Perspective and Evolving Management of Tricuspid Regurgitation

Jason H. Rogers, MD; Steven F. Bolling, MD

Abstract—Cardiovascular specialists have entered an era of renewed interest and enthusiasm surrounding the diagnosis and treatment of valvular heart disease, driven in part by emerging percutaneous therapies for the treatment of aortic, pulmonic, and mitral valve disease. Despite this wave of investigation, little or no attention has been given to the treatment of tricuspid valve disease. Tricuspid regurgitation (TR) occurs mainly from tricuspid annular dilation, which can result from left-sided heart failure from myocardial or valvular causes, right ventricular volume and pressure overload, or dilation of cardiac chambers. If untreated at the time of surgical mitral valve repair, significant residual TR negatively impacts perioperative outcomes, functional class, and survival. TR does not reliably resolve after successful mitral valve surgery. If present at the time of mitral valve surgery, TR can usually be effectively addressed with ring annuloplasty. Because reoperations for recurrent TR carry high mortality rates, few patients are offered reoperation for redo tricuspid repair or replacement. As transcatheter therapies for mitral regurgitation arise, parallel percutaneous approaches for TR may be necessary. In this article, we review the anatomy, pathophysiology, and value of mechanical correction of TR, including potential transcatheter therapies for TR. (***Circulation*. 2009;119:2718-2725.**)

Key Words: valve, tricuspid ■ valves ■ catheters ■ surgery

In recent years, multiple percutaneous approaches for treatment of aortic, mitral, and pulmonic valve disease have been introduced. Whereas older methods relied primarily on balloon valvuloplasty or commissurotomy, newer approaches include the development of percutaneously implanted aortic and pulmonic valves and numerous techniques for repairing either functional or degenerative mitral regurgitation. In contrast, there has been far less discussion on existing surgical and potential percutaneous methods for tricuspid valve repair or replacement. Despite the fact that tricuspid regurgitation (TR) can result in significant symptoms, it remains undertreated. Patients are rarely referred for isolated surgical tricuspid valve repair, and most repairs are done in the context of other planned cardiac surgery. Because significant tricuspid regurgitation appears to be a marker for late-stage myocardial and valvular heart disease, reoperations for recurrent TR are especially high-risk surgical procedures (up to 37% in-hospital mortality) and are therefore not routinely offered to many patients.[1,2] In this article, we examine the clinical relevance of surgical-mechanical TR correction and the potential importance of percutaneous treatments for TR.

The Tricuspid Valve Complex

The tricuspid valve complex consists of three leaflets (anterior, posterior, and septal), the chordae tendinae, two discrete papillary muscles, the fibrous tricuspid annulus, and the right atrial and right ventricular myocardium (Figure 1). Successful valve function depends on the integrity and coordination of these components. The anterior leaflet is the largest, whereas the posterior leaflet is notable for the presence of multiple scallops. The septal leaflet is the smallest and arises medially directly from the tricuspid annulus above the interventricular septum. The anterior papillary muscle provides chordae to the anterior and posterior leaflets, and the medial papillary muscle provides chordae to the posterior and septal leaflets. The septal wall gives chordae to the anterior and septal leaflets (note that there is no formal septal papillary muscle as with the anterior and posterior papillary muscles). In addition, there may be accessory chordal attachments to the right ventricular free wall and to the moderator band. These multiple chordal attachments are important mediators of TR, as they impair proper leaflet coaptation in the setting of right ventricular dysfunction and dilation.[3]

Because the small septal wall leaflet is fairly fixed, there is little room for movement if the free wall of right ventricular/tricuspid annulus should dilate.[4] Dilation of the tricuspid annulus therefore occurs primarily in its anterior/posterior (mural) aspect, which can result in significant functional TR as a result of leaflet malcoaptation.[5] The septal aspect of the tricuspid annulus is considered to be analogous to the intertrigonal portion of the mitral annulus in that it is relatively spared from annular dilation. Because of this property, tricuspid annular sizing algorithms have been based on the dimension of the base of the septal leaflet.[6] Other important factors influencing the

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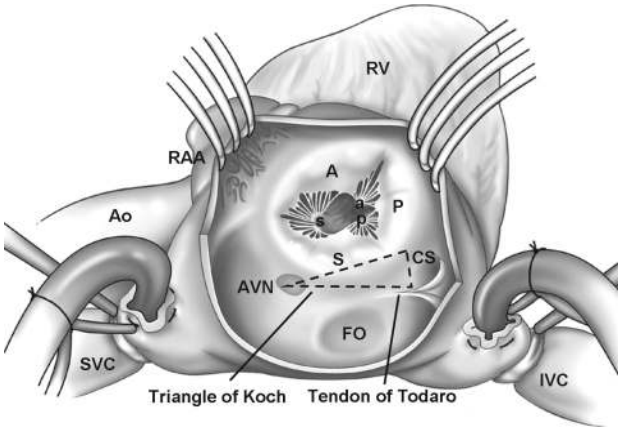

Figure 1. Surgical perspective of the tricuspid valve complex. The tricuspid valve consists of three leaflets: anterior (A), posterior (P), and septal (S). There are 2 main papillary muscles, anterior (a) and posterior (p). The septal papillary muscle (s) is rudimentary, and chordae tendinae arise directly from the ventricular septum. Relevant adjacent structures include the atrioventricular node (AVN), coronary sinus ostium (CS), and the tendon of Todaro, which form the triangle of Koch. Ao indicates aorta; FO, foramen ovale; IVC, inferior vena cava; SVC, superior vena cava; RAA, right atrial appendage; and RV, right ventricle.

degree of TR include right ventricular preload, afterload, and right ventricular systolic function. The influence of intravascular volume status and underlying right ventricular function on tricuspid valve function stems from the fact that the tricuspid annulus is very dynamic and can change markedly with loading conditions. Even during the cardiac cycle, there is a ≈19% reduction in annular circumference (≈30% reduction in annular area) with atrial systole.[7,8]

The tricuspid annulus has a complex 3-dimensional structure, which differs from the more symmetric "saddle-shaped" mitral annulus. This distinct shape has implications for the design and application of currently available annuloplasty rings in the tricuspid position (most currently available rings are essentially planar). In an effort to better understand the shape and movement of the healthy and diseased tricuspid annulus, Fukuda et al[7] performed a real-time 3-dimensional transthoracic echocardiographic study. They examined 15 healthy subjects and 16 patients with functional TR (12/16 had moderate to severe TR). The tricuspid annulus was mapped throughout the cardiac cycle and reconstructed on a computer workstation. Healthy subjects had a nonplanar, elliptical-shaped tricuspid annulus, with the posteroseptal portion being "lowest" (toward the right ventricular apex) and the anteroseptal portion the "highest" (Figure 2). Patients with functional TR generally had a more planar annulus, which was dilated primarily in the septal-lateral direction, resulting in a more circular shape as compared with the elliptical shape in healthy subjects. The authors concluded that novel approaches or rings tailored to the unique tricuspid annular shape might improve ventricular function and reduce leaflet stress.

Cause, Diagnosis, and Natural History

Tricuspid valve regurgitation occurs mainly from annular dilation and right ventricular enlargement, which is often secondary to left heart failure from myocardial or valvular causes, right ventricular volume and pressure overload, and dilation of cardiac chambers.[9] Less common causes of tricuspid valve pathology include rheumatic, congenital, or other (endocarditis, leaflet tear/prolapse, chordal rupture, papillary muscle rupture, or myxomatous degeneration of the tricuspid valve, Table 1).[10] With isolated TR, patients may experience fatigue and decreased exercise tolerance as a result of decreased cardiac output. They may also experience the classic symptoms of "right-sided heart failure" from elevated right atrial pressures, such as ascites, congestive hepatopathy, peripheral edema, decreased appetite, and abdominal fullness. The assessment of intravascular volume status in a patient with severe TR can be difficult because of the pulsatile jugular venous pressure on physical examination. Atrial fibrillation is common as a result of right atrial enlargement.

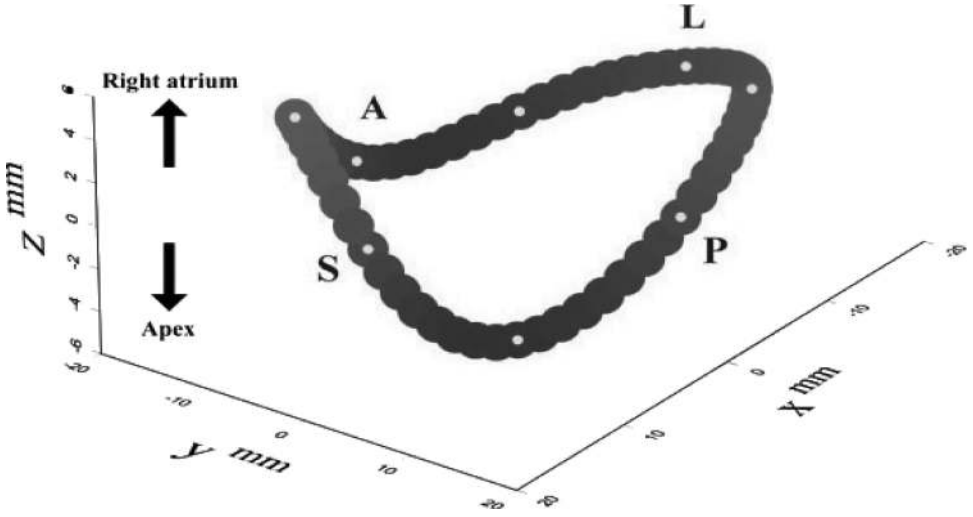

Figure 2. Three-dimensional shape of the tricuspid annulus. Reconstructed representation of the tricuspid annulus based on a 3-dimensional transthoracic echocardiographic study in healthy subjects. Note that the annulus is not planar and that an optimally shaped annuloplasty ring may need to mimic this configuration. A indicates anterior; L, lateral; P, posterior; S, septal. Reprinted from Fukuda et al[7] with permission of the publisher.

Table 1. Causes of Tricuspid Regurgitation

Primary causes (25%)
Rheumatic
Myxomatous
Ebstein anomaly
Endomyocardial fibrosis
Endocarditis
Carcinoid disease
Traumatic (blunt chest injury, laceration)
Iatrogenic (pacemaker/defibrillator lead, RV biopsy)
Secondary causes (75%)
Left heart disease (LV dysfunction or valve disease) resulting in pulmonary hypertension
Any cause of pulmonary hypertension (chronic lung disease, pulmonary thromboembolism, left to right shunt)
Any cause of RV dysfunction (myocardial disease, RV ischemia/infarction)

RV indicates right ventricular; LV, left ventricular.

A unique cause of TR is the result of pacemaker or defibrillator leads, which cross from the right atrium into the right ventricle and may directly interfere with leaflet coaptation. This entity has been reported in case reports and small series but is likely more significant and prevalent than currently perceived. In a recent report by Kim et al,[11] the effect of trans-tricuspid permanent pacemaker or implantable cardiac defibrillator leads on 248 subjects with echocardiograms before and after device placement was studied. The authors found that TR worsened by 1 grade or more after implant in 24.2% of subjects and that TR worsening was more common with implantable cardiac defibrillators than permanent pacemakers with baseline mild TR or less. After lead implantation, 17.8% of patients with baseline mild TR developed moderate to severe TR. Pacemaker leads can also result in tricuspid stenosis as a result of leaflet scarring and adhesions.[12] For patients with existing TR and trans-tricuspid pacing leads, extraction is not recommended on the basis of the current guidelines, because the risks of lead extraction are significant and because there is potential for injury to the tricuspid valve if the lead is adherent to the valve apparatus.[13] It has also been shown that 5 years after successful tricuspid valve repair, 42% of patients with a pacemaker had severe TR, almost double the incidence of those without pacemaker implantation.[14] This suggests removing a trans-tricuspid lead and replacing it with an epicardial lead at the time of tricuspid valve surgery may reduce late repair failure.[1]

Echocardiography is routinely used to assess the severity of TR in clinical practice. This is performed in an integrative manner using color Doppler flow mapping in at least 2 orthogonal planes, assessment of vena contracta width, flow convergence calculations, and the direction and size of the jet. In addition, the morphology of continuous wave Doppler recordings across the valve and pulsed wave Doppler of the hepatic veins can be used.[15] Serial assessments of TR must be interpreted in the patient's clinical context, because, as with functional mitral regurgitation, severity can be affected by multiple factors, such as volume status and afterload. Examples of echocardiographic patterns of TR from various pathologies are shown in Figure 3. Right ventricular shape is complex as compared with the left ventricle, appearing crescent shaped in cross-section and triangular when viewed en face.[16] Right ventricular function can be assessed quantitatively in the 4-chamber view by measuring the end-diastolic area and the end-systolic area to calculate the fractional area change of the right ventricle.[17] Other indices of right ventricular function assessable by echocardiography include the use of velocity vector imaging to measure strain and strain rate and the magnitude of tricuspid annular excursion.[18–20] Although right ventricular chamber dimensions may be obtained during echocardiography, magnetic resonance imaging is emerging as an improved technique for assessing right ventricular diastolic and systolic volumes.[21]

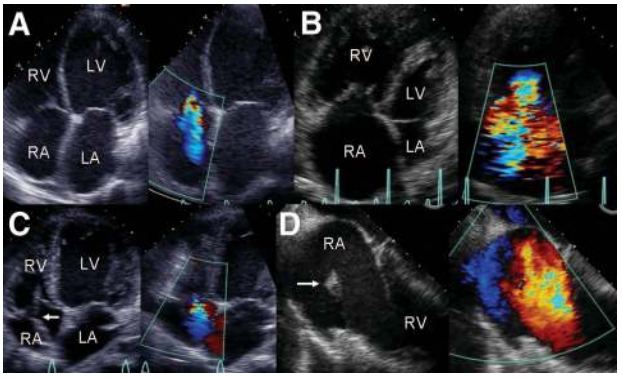

Figure 3. Echocardiographic appearance of tricuspid regurgitation (TR) from various pathologies. (A) Left ventricular (LV) dysfunction with mitral regurgitation and left atrial (LA) enlargement, with secondary right heart enlargement and 2+ TR. (B) Primary pulmonary hypertension with massive RV and RA enlargement and 4+ TR. The LV and LA are underfilled and compressed by the right heart. (C) Nonischemic LV cardiomyopathy with as yet preserved right heart size. An RV pacemaker lead present (arrow) with 2+ TR. (D) Cardiac contusion from motor vehicle accident with anterior papillary muscle rupture. The papillary muscle can be seen prolapsing into the RA with systole (arrow), resulting in torrential TR.

Left and right ventricular interdependence plays an important role in right ventricular function. In addition to a shared interventricular septum, there is continuity between the muscle fibers of the left and right ventricles, resulting in a mechanical union whereby left ventricular contraction augments right ventricular free wall contraction.[22] Experimental models have shown that 20% to 40% of RV systolic pressure and volume outflow results from left ventricular contraction.[23] In addition, the left and right ventricle may share a common biochemical milieu, whereby improvements in systemic and local neurohormonal parameters may result in improvements in biventricular function. Importantly, left-sided heart failure with chamber enlargement and mitral regurgitation can result in right-sided pressure overload, right ventricular chamber enlargement, tricuspid annular dilation, and resultant TR. This mechanistic cascade originally led to the concept that either surgical or medical treatment of the left-sided abnormality will result in secondary improvement or amelioration of TR. Although improvement in TR does occur, this is not invariably the case. Dreyfus et al[24] have demonstrated that a paradigm that advocates treatment of the proposed "primary" lesion only (ie, mitral valve disease) will

not directly correct tricuspid annular dilation or improve right ventricular function, the major determinants of functional TR. In their study, the tricuspid valve annulus was visually assessed in 311 patients undergoing mitral valve repair between 1989 and 2001. Tricuspid annuloplasty was performed selectively only on those patients whose tricuspid annular diameter (as measured from the anteroseptal commissure to the anteroposterior commissure) was greater than twice the normal size ($\geq$70 mm, n=148). In follow-up, TR grade (0.4$\pm$0.6 versus 2.1$\pm$1.0, mitral valve repair plus tricuspid valve repair versus mitral valve repair alone, $P<0.001$) and New York Heart Association class were significantly improved in those who underwent TV annuloplasty. In-hospital mortality and actuarial survival rate were likewise improved in patients undergoing TV annuloplasty, supporting the notion that TV annuloplasty at the time of mitral valve repair results in improved patient outcomes.

Without treatment, TR may become worse over time, leading to severe symptoms, biventricular heart failure, and death.[14] It has been shown in a large retrospective echocardiographic analysis of 5223 Veterans Administration patients by Nath et al,[25] that independent of echo-derived pulmonary artery systolic pressure, left ventricular ejection fraction, inferior vena cava size, and right ventricular size and function, survival is worse for patients with moderate and severe TR than for those with no TR (TR graded using Framingham Heart Study criteria). In this series, the prevalence of TR was as follows: no TR, 11.5%; mild TR, 68.8%; moderate TR, 11.8%; and severe TR, 3.8%. A limitation of this study is that the presence of mitral regurgitation was not included as a covariate in the survival analysis, although severe mitral regurgitation would presumably be reflected to some degree by the pulmonary artery systolic pressure.

Pulmonary artery hypertension from any cause is known to be associated with the development of secondary tricuspid regurgitation. However, not all patients with pulmonary hypertension develop significant tricuspid regurgitation, and the mechanisms of secondary TR in this population are multifactorial. In a recent study by Mutlak et al,[26] 2139 subjects with either mild (<50), moderate (50 to 69), or severe ($\geq$70) elevations in pulmonary artery systolic pressure (PASP, defined as the sum of the peak TR systolic pressure gradient and estimated right atrial pressure in mm Hg) were studied to define the determinants of TR severity. In this analysis, increasing PASP was independently associated with greater degrees of TR (odds ratio, 2.26 per 10 mm Hg increase). However, many patients with high PASP had only mild TR (mild TR in 65.4% of patients with PASP 50 to 69 mm Hg and in 45.6% of patients with PASP $\geq$70 mm Hg). Other factors, such as atrial fibrillation, pacemaker leads, and right heart enlargement, were also importantly associated with TR severity. The authors concluded that the cause of TR in patients with pulmonary hypertension is only partially related to an increase in trans-tricuspid pressure gradient, with remodeling of the right heart in response to elevated PASP as the major mechanism responsible for TR in these patients. An important question that arises is whether surgical correction of TR in patients with elevated PASP is indicated. The guidelines reflect the paucity of data on this subject, with

Table 2. 2006 ACC/AHA Guidelines Pertaining to the Surgical Management of Tricuspid Valve Disease/Regurgitation

Class I
Tricuspid valve repair is beneficial for severe TR in patients with MV disease requiring MV surgery. *(Level of Evidence: B)*
Class IIa
1. Tricuspid valve replacement or annuloplasty is reasonable for severe primary TR when symptomatic. *(Level of Evidence: C)*
2. Tricuspid valve replacement is reasonable for severe TR secondary to disease/abnormal tricuspid valve leaflets not amenable to annuloplasty or repair. *(Level of Evidence: C)*
Class IIb
Tricuspid annuloplasty may be considered for less than severe TR in patients undergoing MV surgery when there is pulmonary hypertension or tricuspid annular dilatation. *(Level of Evidence: C)*
Class III
1. Tricuspid valve replacement or annuloplasty is not indicated in asymptomatic patients with TR whose pulmonary artery systolic pressure is less than 60 mm Hg in the presence of a normal MV. *(Level of Evidence: C)*
2. Tricuspid valve replacement or annuloplasty is not indicated in patients with mild primary TR. *(Level of Evidence: C)*

ACC indicates American College of Cardiology; AHA, American Heart Association; TR, tricuspid regurgitation; and MV, mitral valve.

Class IIb and III recommendations (level of evidence C) for tricuspid annuloplasty in subsets of patients with pulmonary hypertension (Table 2).[27] Although it seems plausible that correction of TR would alleviate unfavorable volume overload of the right ventricle, it remains to be proven that TR correction in the setting of pulmonary hypertension alters the natural course of right ventricular dilation and development of cor pulmonale. Percutaneous or minimally invasive approaches to TR correction could facilitate a clinical trial to investigate this hypothesis further.

In severe TR, elevated right atrial pressure is transmitted to the hepatic veins, which can result in congestive hepatopathy. Over time, this can lead to hepatocyte dysfunction, atrophy, and eventually cardiac cirrhosis (fibrosis). The development of liver dysfunction in patients with heart failure and TR can also occur from an ischemic hepatopathy secondary to decreased cardiac output. Cardiac cirrhosis is typically seen in patients with constrictive pericarditis or any cause of right-sided heart failure and TR.[28] In general, most patients with cardiac cirrhosis present late in the disease state, at which time correction of TR may not be helpful. In select patients, it is conceivable that earlier correction of TR could lead to resolution of liver function abnormalities, but this remains to be shown.

Current Surgical Approaches to TR

The main surgical approaches to rectify functional TR (occurring in the presence of a dilated annulus with normal leaflets and chordal structures) involve rigid or flexible annular bands (open or closed), which are used to reduce annular size and achieve leaflet coaptation, as with mitral valve disease. Another less commonly used technique involves posterior annular bicuspidalization. This surgical technique places a pledget-supported mattress suture from the

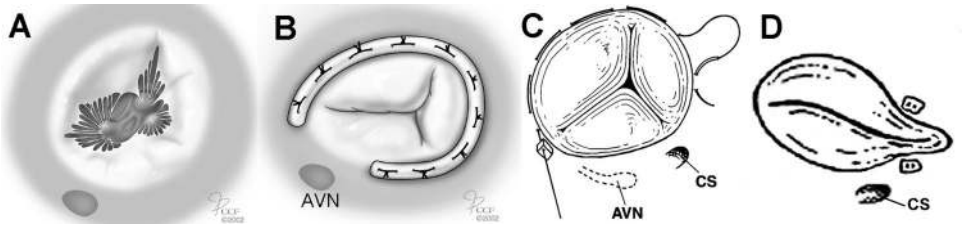

Figure 4. Predominant surgical repair techniques for functional tricuspid regurgitation (TR). The main surgical approaches for correcting functional TR in the presence of a dilated annulus are shown. (A) Dilated tricuspid annulus with abnormal circular shape, failure of leaflet coaptation, and resultant TR. Note that in functional TR, dilation occurs primarily along the mural portion of the tricuspid annulus, above the right ventricular free wall. (B) Rigid or flexible annular bands are used to restore a more normal annular size and shape (ovoid), thereby reducing or eliminating TR. The open ring shown spares the atrioventricular node (AVN), thus reducing the incidence of heart block. (C) DeVega–style suture annuloplasty in which a purse-string suture technique is used to partially plicate the annulus and reduce annular circumference and diameter. (D) Suture bicuspidalization is performed by placement of a mattress suture from the anteroposterior to the posteroseptal commissures along the posterior annulus. CS indicates coronary sinus.

anteroposterior commissure to the posteroseptal commissure along the posterior annulus. This is based on prior studies by Deloche et al[28a] that show posterior annulus dilation occurs in functional TR and that a focal posterior tricuspid annuloplasty can be effective in selected cases.[5] Other approaches include edge-to-edge (Alfieri-type) repairs as described by Castedo et al[29,30] and partial purse-string suture techniques to reduce the anterior and posterior portions of the annulus (DeVega-style techniques, Figure 4). DeVega and flexible band annuloplasties appear to have a lower freedom from recurrent TR than rigid annuloplasty rings.[10,14,31,32]

There are numerous reports describing the presence of TR in patients undergoing surgery for mitral regurgitation. In the absence of simultaneous tricuspid valve repair, the prevalence of TR in the postoperative period after mitral valve surgery depends to some degree on the mechanism of MR. Matsuyama et al[33] reported in a study of 174 patients that only 16% of patients who underwent nonischemic (ie, degenerative) mitral valve surgery without tricuspid valve surgery developed 3 to 4+ TR at 8-year follow-up. Conversely, TR appears to be far more prevalent in patients undergoing mitral valve repair for functional ischemic mitral regurgitation. In the series by Matsunaga et al,[34] of 70 patients undergoing mitral valve repair for functional ischemic mitral regurgitation, 30% of patients (21/70) had at least moderate TR before surgery. In the postoperative period, the prevalence of at least moderate TR increased over time, from 25% at $<$1 year, 53% at 1 to 3 years, and 74% at $>$3 years of follow-up.

Significant residual tricuspid valve insufficiency may also contribute to a poor postoperative hemodynamic result, even after successful mitral valve repair. In one early surgical series by King et al,[35] patients requiring subsequent tricuspid valve surgery after mitral valve surgery had high early and late mortality. The authors encouraged a policy of liberal use of tricuspid annuloplasty at initial mitral valve surgery. Surgical series have shown that successful tricuspid valve repair (primarily when combined with other valve surgeries) resulted in a significant improvement in recurrent TR, survival, and event-free survival. Accordingly, 50% to 67% of patients undergoing surgery for mitral valve disease have been reported to undergo concomitant surgical tricuspid valve repair or replacement (although this may approach 80% in some dedicated centers).[1,10] Investigators have attempted to identify specific patient subsets that should have tricuspid valve repair/replacement at the time of mitral valve repair/replacement. It has been proposed by Dreyfus et al[24] that at the time of mitral valve repair, the presence of tricuspid annular dilation ($\geq$70 mm measured intraoperatively), even in the absence of significant TR, should be an indication for tricuspid valve annuloplasty. This study also showed that TR increased by at least 2 grades in 45% of the patients who received isolated mitral valve repair, supporting the notion that tricuspid dilation is an ongoing, progressive process that often warrants preemptive surgical treatment.

In the series by Singh et al,[1] tricuspid valve repair appears to result in improved mid-term survival (up to 10 years after surgery, primarily as a result of higher perioperative mortality with replacement) as compared with tricuspid valve replacement, although there was no difference in valve-related mortality or need for tricuspid valve reoperation. The authors hypothesized that the higher perioperative mortality with replacement may have been due to a rigid object (tricuspid valve) in a deformable low-pressure cavity (right ventricle), with resultant right ventricular dysfunction and perioperative low output state. Although patients in this series had less recurrent TR with replacement versus repair (95% versus 62% had mild or less TR at most recent echocardiographic follow-up), there was no difference in functional class in either group.

Current Practice Patterns and Guidelines for the Surgical Management of TR

The American College of Cardiology/American Heart Association 2006 Practice Guidelines for the surgical management of patients with TR are shown in Table 2.[27] An individual patient's clinical status and the cause of their tricuspid valve abnormality usually determine the appropriate therapeutic strategy. It is stated in the guidelines that the timing of surgical intervention for TR remains controversial, as do the surgical techniques. Given the adverse consequences of allowing TR to progress to severe (such as worsening symptoms of right heart failure), it would seem logical that earlier intervention for TR, especially in the presence of ongoing right atrial and right ventricular enlargement, would be beneficial. There are currently no data that specifically address this important question. A trial comparing benign neglect of TR versus earlier intervention would seem reasonable, especially if percutaneous or minimally invasive ap-

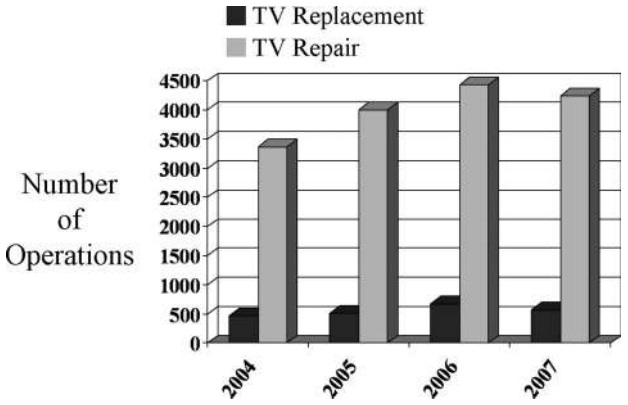

Figure 5. Current tricuspid valve repair and replacement volumes. Shown are the current Society of Thoracic Surgeons National Cardiac Database estimates for total tricuspid valve operations. Totals presented are for tricuspid valve replacements (dark bars) or repairs (light bars). Each bar represents the total tricuspid replacements or repairs in that year, as the sum of isolated tricuspid valve repair/replacement (TVR), TVR with mitral valve repair/replacement (MVR), TVR with coronary artery bypass grafting, and TVR with MVR/coronary artery bypass grafting. Volumes are clearly a small fraction of the total mitral valve operations yearly, which exceed 40 000.

proaches to TR correction were used. At present, surgery on the tricuspid valve for significant TR should occur at the time of mitral valve surgery, as TR does not simply "go away" after mitral valve surgery. TR associated with dilatation of the tricuspid annulus should also be repaired, because tricuspid dilation is an ongoing process that may progress to severe TR if left untreated.[14,24] Rigid annuloplasty should be the preferred surgical approach for significant TR if the leaflets are spared from the disease process.[36–41] Despite guidelines and recent data that support a proactive approach to surgical repair of TR at the time of mitral valve surgery, tricuspid valve repair currently appears underutilized (Figure 5). The current surgical volume of tricuspid valve repair and replacement as quantified in the Society of Thoracic Surgeons National Cardiac Database represents only approximately one-tenth of the >40 000 mitral valve operations performed yearly in the United States.[42] Although repair is preferred, valve replacement is often necessary when the valve leaflets themselves are diseased, abnormal, or destroyed.[43] Thrombosis with mechanical tricuspid valves is rare (<1% per year), and overall survival has been shown to be equivalent between bioprosthetic and mechanical valves in a recent large meta-analysis.[44] Thrombolysis is considered first-line therapy for tricuspid valve thrombosis, as opposed to left-sided valve thrombosis, for which the risks of systemic and cerebral embolism are increased. In patients undergoing sternotomy with underlying conduction disease, placement of an epicardial pacing electrode can avoid the subsequent need to pass a transvenous lead across the native valve. This technique is particularly important in the presence of a bioprosthetic tricuspid valve, in which case placement of a trans-tricuspid pacing lead is generally contraindicated. The major limitation of an epicardial lead is an increased pacing threshold, which significantly shortens generator life. In general, endocardial leads perform better over time than epicardial leads, and if an atrial lead is needed, an endocardial lead is generally preferred.

Relevance in the Percutaneous Era

To date, there have been few reports describing percutaneous approaches to tricuspid valve disease. In 2005, Boudjemline et al[45] described a novel percutaneous tricuspid valve consisting of a bovine jugular venous valve mounted to a self-expanding nitinol frame consisting of 2 disks. The device was deployed through an 18F sheath in the right internal jugular vein, with right ventricular and right atrial disks deployed sequentially to sandwich the native tricuspid valve (Figure 6). The device was successfully implanted in seven normal sheep, but no further work has been done with this device. As percutaneous approaches to valvular heart disease emerge, lessons gained from prior surgical experiences are relevant. Concomitant surgical repair of TR at the time of mitral valve surgery should be considered standard of care, as this approach has been shown to result in improved perioperative outcomes, functional class, and survival. Surgical correction of isolated TR can significantly improve right ventricular volumes and ejection fraction.[46] It is currently estimated that as many as 500 000 people in North America have clinically significant congestive heart failure–associated or functional mitral regurgitation. This constitutes the clinical need on which current percutaneous approaches to functional mitral valve repair are based. There would appear to be an associated need in many these patients for some manner of percutaneous tricuspid annuloplasty/repair or replacement.

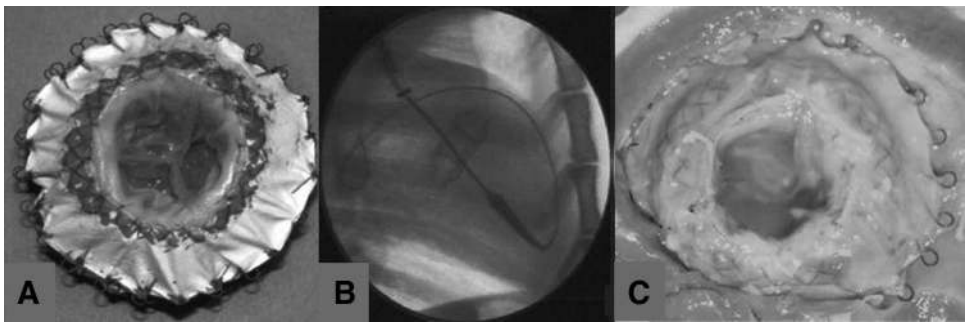

Figure 6. Percutaneous tricuspid valve replacement. (A) Novel nitinol stent-based percutaneous tricuspid valve. An 18-mm bovine jugular venous valve is mounted in the central part of the stent, with a polytetrafluoroethylene membrane sutured to the ventricular disk to assist in sealing. B, Percutaneous tricuspid valve delivered in an ovine model via an 18F sheath in the internal jugular vein under fluoroscopic and echocardiographic guidance. C, Gross appearance of valve explanted at 1 month after implantation showing neoendocardial coverage of the stent (atrial view). Adapted from Boudjemline et al,[45] copyright © 2005, with permission from Elsevier.

Unique challenges for emerging percutaneous approaches to the tricuspid valve include the lack of convenient adjacent structures for device placement (such as the coronary sinus and its relationship to the mitral valve) and the relatively low-flow state in the right heart, which may promote thrombus formation. The coronary sinus ostium, atrioventricular node, and inferior vena cava are adjacent structures that must not be covered by any potential therapeutic devices. The presence of preexisting trans-tricuspid pacemaker or defibrillator leads will undoubtedly require unique percutaneous solutions.

Conclusion

Significant functional TR cannot be ignored when performing corrective surgical procedures for mitral regurgitation. Because TR does not disappear after successful mitral valve surgery, and reoperations for recurrent TR carry high mortality rates, few patients are offered reoperation. Minimally invasive or percutaneous approaches to TR could offer the ability to more readily treat TR concomitantly or after mitral valve intervention. In addition, the less invasive approaches may allow earlier mechanical treatment of TR than is currently offered. Challenges to emerging minimally invasive or percutaneous approaches are numerous, but should be surmountable with evolving surgical, imaging, and interventional techniques.

Disclosures

Dr Rogers is a consultant for Ample Medical, Medtronic, and Sorin-Carbomedics. Dr Bolling is a consultant for St. Jude Medical, Sorin-Carbomedics, Medtronic, and Edwards Lifesciences.

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