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STATE-OF-THE-ART PAPER

The Growing Clinical Importance of Secondary Tricuspid Regurgitation

Maurizio Taramasso, MD,* Hugo Vanermen, MD,† Francesco Maisano, MD,* Andrea Guidotti,* Giovanni La Canna, MD,* Ottavio Alfieri, MD*

Milan, Italy; and Aalst, Belgium

Functional or secondary tricuspid regurgitation (STR) is the most frequent etiology of tricuspid valve pathology in Western countries. Surgical tricuspid repair has been avoided for years, because of the misconception that tricuspid regurgitation should disappear once the primary left-sided problem is treated; this results in a large number of untreated patients with STR. Over the past few years, many investigators have reported evidence in favor of a more aggressive surgical approach to STR. Consequently, interest has been growing in the physiopathology and treatment of STR. The purpose of this review is to explore the anatomical basis, pathophysiology, therapeutic approach, and future perspectives with regard to the management of STR. (J Am Coll Cardiol 2012;59: 703–10) © 2012 by the American College of Cardiology Foundation

Functional or secondary tricuspid regurgitation (STR) refers to tricuspid regurgitation (TR) occurring secondary to left-sided heart disease (LHD) or pulmonary hypertension in the absence of organic lesions of the tricuspid valve (TV) apparatus and is the most frequent etiology of TV disease in Western countries (1). The "functional" label used regarding TR secondary to pulmonary hypertension or LHD is possibly a misnomer, in that because of the absence of any organic pathology, it could be incorrectly interpreted as a benign condition that would resolve itself after the correction of the primary disorder.

Therefore, STR is not a truly functional entity, because it entails intrinsic anatomical abnormalities of the TV apparatus, such as annular dilation and deformation.

Until recently, surgical avoidance of TV repair was easily accepted in patients with STR, on the basis of the incorrect concept that TR would disappear once the primary LHD had been treated (2). The physiopathology and the treatment of this condition have been neglected for a number of years. This conservative approach still influences surgical practice today, and TV repair remains an all too infrequent procedure at most surgical centers. Over the past few years, many investigators have reported evidence in favor of a more aggressive surgical approach to STR (3–5). Currently, moderate to severe TR affects approximately 1.6 million patients in the United States, of whom only 8,000 undergo tricuspid surgery annually (6); this results in an extremely large number of untreated patients with STR.

Significant residual TR has been reported in 10% to 45% of patients after TV repair with different techniques (7–11). Consequently, interest has been growing in the physiopathology and treatment of STR. The present report is aimed at both medical cardiologists and cardiac surgeons, because their roles in the management of patients with TR are usually different; generally, it is the cardiac surgeon who makes the decision concerning whether or not to perform TV repair during left-heart surgery, while medical cardiologists are reluctant to recommend reoperative tricuspid surgery after surgical correction of the LHD that initially provoked TR, because of the increased morbidity and mortality associated with reoperative open-heart operations (12). This review should foster active preoperative and intraoperative collaboration between cardiologists and surgeons in performing TV repair in individual patients.

Anatomy

The orifice of the TV is semilunar in shape because of the way the right ventricle is wrapped around the left ventricle.

The TV complex (Fig. 1) consists of 3 leaflets (septal, posterior, and anterior), chordae tendineae, and usually 3 papillary muscles (the anterior, the posterior, and a third variable papillary muscle). The septal cusp, shaped like part of a semicircle, has 1 anatomical feature of importance: a number of third-order chordae tendineae that join the body of the cusp are attached directly to the septum.

From *Cardiothoracic Surgery, San Raffaele Scientific Institute, Milan, Italy; and †Cardiothoracic Surgery, Onze Lieve Vrouwziekenhuis, Aalst, Belgium. Dr. Vanermen has relationships with Edwards Lifesciences, Medtronic, Valtech, USB Medical, and 4-Tech. Dr. Maisano has relationships with 4-Tech and Valtech. Dr. La Canna has received speaker's fees from Abbott Vascular. All other authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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Abbreviations And Acronyms

AV = atrioventricular
LHD = left-sided heart disease
LV = left ventricular
MV = mitral valve
RV = right ventricular
STR = secondary tricuspid regurgitation
TA = tricuspid annulus
TR = tricuspid regurgitation
TV = tricuspid valve

The parietal attachment of the TV is normally encircled by the right coronary artery. Other structures of major surgical significance surrounding the TV are the coronary sinus, the atrioventricular (AV) node and the bundle of His.

The AV node lies in the atrial septum bordering the septal leaflet. The exact location of the AV node can be approximated at the apex of the triangle of Koch, which is composed of the septal annulus and the tendon of Todaro as its sides and the coronary sinus orifice as its base. Extending from the AV node is the bundle of His, which penetrates the right trigone under the interventricular component of the membranous septum.

Along the line of attachment, a fibrous "ring" is not as easy to define as it is around the mitral valve (MV), although it remains identifiable.

Compared with the MV, the tricuspid orifice is larger and more triangular. In physiologic conditions, the tricuspid annulus (TA) has an elliptic, nonplanar shape. The superior points of the TA are the anterior and the posterior ones, while the septal and lateral points are inferior, toward the right ventricle (13,14).

The nonplanar and noncircular structure of the TA must be taken into account when considering STR repair; specifically shaped annular prostheses are currently used for surgical correction of STR (15).

Pathophysiology of Secondary Tricuspid Regurgitation

TV closure during systole is a dynamic and complex mechanism that requires the harmonious interaction of the whole valve apparatus. The most common etiology of STR is RV dilation and dysfunction from LHD, as in cases of MV disease (2,16). Dilated cardiomyopathy and hypertensive pulmonary disease are less frequent causes of STR (2).

The pathophysiology of STR may be divided into 3 phases (17). In the first phase, initial dilation of the right ventricle results in TA dilation; TR may or may not be present, depending on the degree of annular dilation (3). In the second phase, with progressive RV and TA dilation, significant STR occurs because of the failure of leaflet coaptation (18). Finally, with progressive RV distortion and eccentricity, tethering of the leaflets occurs, in addition to TA dilation, because of the attachment of the papillary muscles of the leaflets to the free wall of the right ventricle (19).

TA dilation and dysfunction. STR is typically associated with TA dilation (3). The TA is a component of both the TV and the right ventricle. With gross RV enlargement, the shape of the orifice of the TV becomes much more circular and prevents normal leaflet coaptation (20). dilation of the TA occurs mainly in its anterior and posterior portion, corresponding to the free wall of the right ventricle, while the dilation of the septal segment is limited because of its close anatomical relationship with the fibrous skeleton of the heart (3) (Fig. 2).

There are both differences and similarities between functional tricuspid insufficiency and functional mitral insufficiency, but the differences are more significant.

The first is that, on the left side, the single anterior leaflet is the part that moves the most and is the most important part functionally, because it covers a disproportionately large area of the orifice in systole. On the right side, however, the multiple leaflets and scallops of the mural part of the valve move the most and cover a disproportionately large area of the orifice in closing (16).

The second difference is that the mitral papillary muscles arise from the middle of the left ventricle, and their displacement by ventricular dimension changes is a more potent source of functional mitral insufficiency than annular dilation. The tricuspid papillary attachments arise, in the case of the septal leaflet, directly from the upper septum and otherwise from extremely variable papillary muscles attached to the trabecular septum. As such, they are very little affected by dilation of the free RV wall, and lengthening of the mural annulus is by far the dominant mechanism of STR (3,16).

RV remodeling and TV tethering. The pathophysiologic explanation for STR in patients with LHD is rising left atrial pressure transmitted through the lungs as pulmonary hypertension (2). This typically causes RV dilation, which

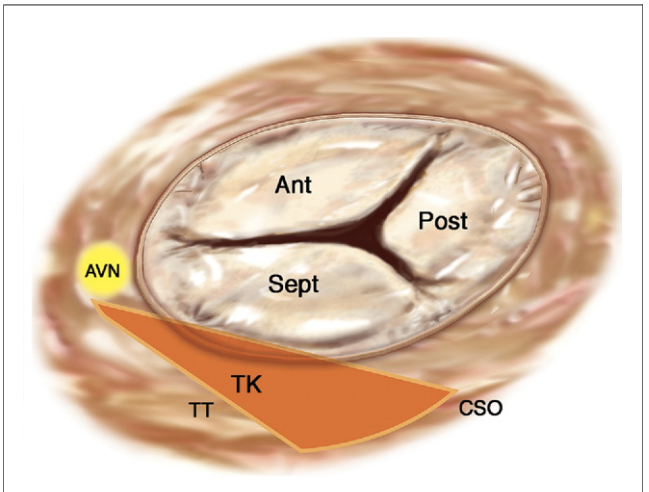

Figure 1 The Tricuspid Valve

Surgical anatomy of the tricuspid valve: the triangle of Koch (TK) is indicated by the orange area. Ant = anterior; AVN = atrioventricular node; CSO = coronary sinus orifice; Post = posterior; Sept = septal; TT = tendon of Todaro). Figure illustration by Craig Skaggs.

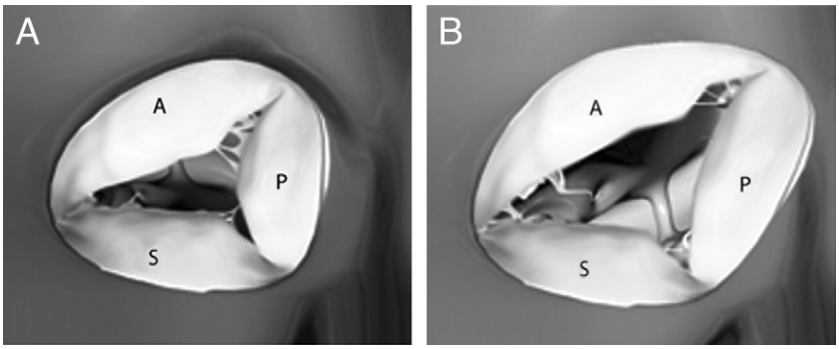

Figure 2 Tricuspid Annular Dilation

(A) Normal tricuspid valve. **(B)** Asymmetric dilation and deformation of the tricuspid annulus leading to tricuspid regurgitation. A = anterior leaflet; P = posterior leaflet; S = septal leaflet.

leads to regurgitation by dilation of the TA and subsequently by tethering of the TV leaflets (17).

The flattening of the TV annulus that occurs with TA dilation in STR can potentially alter the normal papillary muscle-to-leaflet and annulus relationship, because the low points of the annulus may be stretched away from the papillary muscles, thereby increasing tethering (20).

Changes in RV geometry and consequent papillary muscle displacement are also critical factors in the pathophysiology of STR, leading to tenting volume enlargement and inadequate leaflet coaptation (21,22) (Fig. 3).

It is extremely important to bear in mind that once the TV is dilated, its size cannot spontaneously return to normal, and it may continue to dilate further. Moreover, complete reverse remodeling of the right ventricle may not occur (23).

Pulmonary hypertension. Pulmonary hypertension is not a necessary prerequisite to developing STR. Fukuda et al. (24) observed septal leaflet tethering in patients with STR and normal pulmonary artery pressures. When the septum is hypokinetic, dyskinetic, or dilated, there can be a tethering effect on the TV, independently from pulmonary hypertension, because of the right ventricle's septal wall, which is the area of origin of the papillary muscles to the septal leaflet of the TV (21).

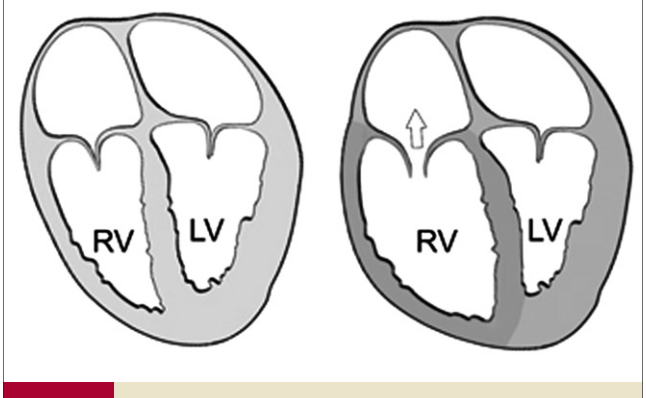

Figure 3 Tricuspid Valve Tethering

Schematic of the pathophysiology of secondary tricuspid regurgitation secondary to right ventricular remodeling and leaflet tethering. LV = left ventricle; RV = right ventricle.

Natural History and Prognostic Implications

Severe TR is an independent predictor of long-term mortality (65% 1-year survival rate in patients with severe TR compared with 90% of patients without TR) (25). Several studies reported a higher long-term mortality rate, lower quality of life, and reduced exercise capacity in patients who developed severe TR after MV surgery (26,27).

The prevalence of STR in patients with MV disease is high: >30% of patients with degenerative mitral regurgitation have TR ≥ 2+ at the time of mitral surgery (28), and up to one-third of patients with significant mitral stenosis have moderate to severe TR (29).

The prevalence of STR is also particularly high in patients with ischemic mitral regurgitation (>30% of patients who underwent revascularization and MV surgery) (23).

Significant TR occurring late after left-heart surgery is observed in up to 40% of patients, with a median survival of 5 years. An increase of >2 grades in TR with respect to preoperative echocardiography is reported in about 50% of patients who undergo isolated MV repair (26,30).

Late TR onset after isolated mitral surgery is associated with decreased exercise tolerance and poor quality of life (31). Many of these patients undergo reoperative TV surgery, with an early mortality rate of 10% to 25% (13).

Tager et al. (32) reported that concomitant TV repair at the time of MV surgery results in TR resolution in about 85% of patients, while several other groups observed a higher incidence of post-operative TR in patients in whom TR was not treated than in patients who underwent TV

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repair. Therefore, it is almost universally accepted that TV annuloplasty during mitral surgery results in a lower incidence of late TR.

However, because of the absence of controlled randomized trials, it is still debated whether a lower incidence of late TR after surgery may improve survival and heart failure symptoms.

Conservative Management

Conservative management of STR includes optimization of RV preload and afterload. RV overload may benefit from progressive use of diuretics and angiotensin-converting enzyme inhibitors. However, an excessive reduction in central venous pressure may result in worsening of TR severity (2).

The use of angiotensin-converting enzyme inhibitors is supported by evidence that chronic RV pressure overload activates the renin-angiotensin-aldosterone system, which may contribute to fluid retention and ventricular remodeling (33); B-type natriuretic peptide is usually increased in cases of RV pressure or volume overload (34).

Although a causal relationship between TR severity and more severe renal failure has not been demonstrated, an association between TR grade and lower estimated glomerular filtration rate, higher blood urea nitrogen, and higher blood urea nitrogen/creatinine ratio has been described (35).

Surgical Management of Secondary Tricuspid Regurgitation

The principles of therapy for STR include elimination of increased afterload to the right ventricle (by correction of LHD and optimization of left ventricular [LV] function) and correction of TA dilation and dysfunction, usually by TV annuloplasty (17).

Current European and U.S. guidelines regarding TV surgery are reported in Table 1 (36,37).

Annular dilation. TA dilation is invariably associated with STR (3). Some studies have suggested that concomitant tricuspid annuloplasty should be performed during left-heart surgery if the TA is dilated, irrespective of the grade of TR (3,38). Moderate annular dilation may not be detected by 2-dimensional echocardiography; 3-dimensional echocardiography probably offers a more accurate assessment (39). Because of the nonplanar saddle shape of the TA, there is no echocardiographic standardized approach to define annular dilation (40,41).

An alternative approach to measuring the TA is direct intraoperative assessment (3).

Preoperative and intraoperative echocardiography should determine which patients undergoing MV surgery should also have tricuspid repair by measuring the TA: tricuspid repair should be performed in cases of TA >40 mm in the 4-chamber view (42).

Moderate TR. Moderate TR is associated with poorer long-term survival (4,25,43). Because of the difficulties in predicting the patients in whom moderate regurgitation will resolve or progress after left-sided heart surgery, the systematic repair of a moderately regurgitant TV during MV surgery should be considered, especially in cases of dilated TA.

The arguments against systematic repair of moderate STR are that it may resolve after correction of LHD, prolonged duration of cardiopulmonary bypass and cross-clamp time, and that annuloplasty may introduce incremental risks such as AV block (44,45).

Pulmonary hypertension. In cases of TV dysfunction, the grade of pulmonary hypertension has been shown to be associated with the severity of TR (20). Pulmonary hypertension may persist after mitral surgery in about 25% of cases; progressive tricuspid dysfunction may also occur in unrepaired valves (46). However, isolated pulmonary hypertension in the presence of a normal TV is not considered an indication for TV repair.

The risk of acutely exposing the right ventricle to increased afterload after TV repair should be taken into account, especially in situations in which severe pulmonary hypertension may persist, because early postoperative RV dysfunction has a prognostic value after tricuspid surgery (47).

Secondary TR and ischemic mitral regurgitation. Concomitant STR is a frequent finding in patients with ischemic cardiomyopathy and functional mitral regurgitation (23). When left untreated, STR progression after surgical revascularization and mitral surgery in ischemic cardiomyopathy negatively affects prognosis (48).

In summary, the aforementioned clinical studies could lead to general recommendations as to when, or when not, to correct STR.

Moderate to severe STR should be corrected at the same time as left-heart surgery (36,37). If severe TV tethering is

Table 1 Guidelines for Tricuspid Valve Repair for Secondary TR

European Society of Cardiology (2007)
Class I: severe TR in a patient undergoing left-sided valve surgery (C)
Class IIa moderate TR with dilated annulus (40 mm) in a patient undergoing left-sided valve surgery (C)
Class IIa: symptomatic severe TR late after left-sided valve surgery in the absence of left-sided myocardial, valve, or right ventricular dysfunction and without severe pulmonary hypertension (C)
American College of Cardiology/American Heart Association (2008)
Class I: severe TR in patients undergoing MV surgery (B)
Class IIb: "less than severe TR" in patients undergoing MV surgery, with pulmonary artery hypertension or tricuspid annular dilation (C)

Levels of evidence are shown in parentheses.
MV = mitral valve; TR = tricuspid regurgitation.

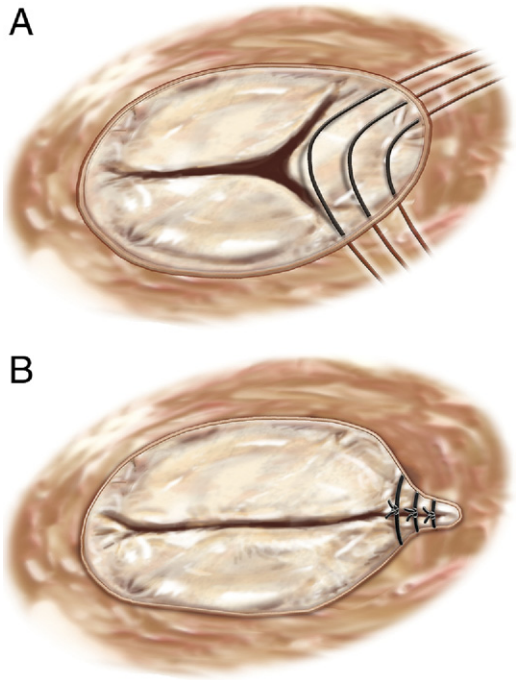

Figure 4 Kay Repair Technique

(A) Tricuspid valve bicuspidization is accomplished by plicating the annulus along the posterior leaflet. **(B)** The sutures are tied, obliterating the posterior leaflet, creating a bicuspid valve. Figure illustration by Craig Skaggs.

present (tethering distance >0.76 cm or tethering area >1.63 cm^2), the use of adjunctive surgical techniques to tricuspid annuloplasty or TV replacement should be considered (7,17). Less than severe STR should be surgically corrected in cases of concomitant LHD requiring surgery in the presence of TA dilation (TA >40 mm on preoperative echocardiography or >70 mm on intraoperative inspection) (3,38).

In cases of less than severe asymptomatic STR, in the absence of LHD requiring surgery, conservative management and clinical follow-up should be carried out (17).

Surgical Treatment of Secondary Tricuspid Regurgitation

TV annuloplasty is the basis of current surgical therapy for STR and aims to correct annular dilation and restore annular geometry, resulting in improved leaflet coaptation.

Two principal surgical methods are used to treat STR: suture annuloplasty techniques and the ring annuloplasty techniques.

With suture annuloplasty methods, annular size is reduced by using a continuous suture to purse string the annulus. Most suture annuloplasty techniques are modified versions of Kay bicuspidization (Figs. 4A and 4B) (49) or De Vega annuloplasty (Fig. 5A), which consists of the plication of both the posterior and anterior annulus (50).

With prosthetic ring annuloplasty (Fig. 5B), the annulus is permanently fixed in a systolic position by suturing in a rigid or semirigid ring (51).

Recent long-term studies have suggested that prosthetic ring annuloplasty repair is more durable than suture repair and confers significant improvements both in overall and event-free survival. In many series, freedom from moderate or severe TR 10 years after surgery is >85% for patients who underwent ring annuloplasty (8,52).

The goal of reconstructive valve surgery is to preserve leaflet mobility and to create a large surface of coaptation, while preventing further annular dilation.

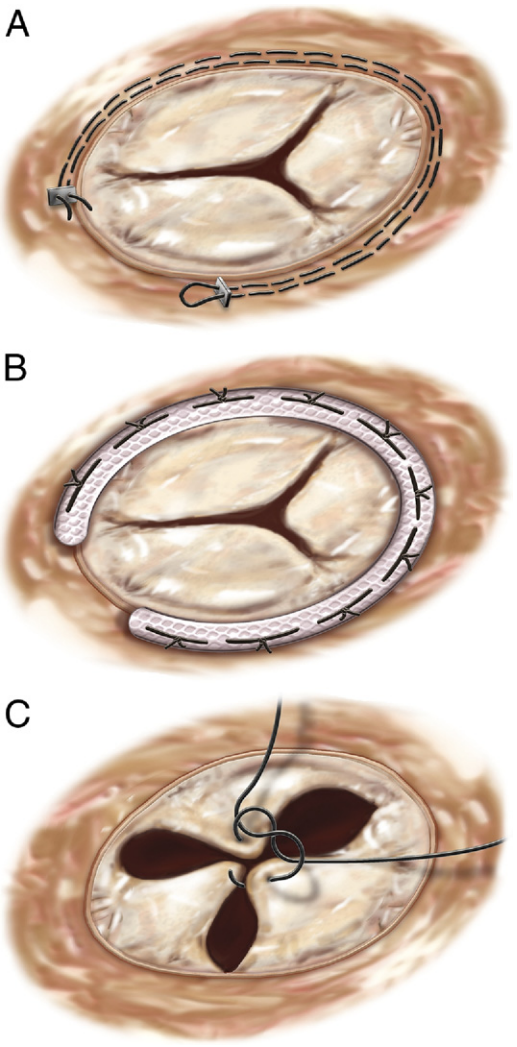

Figure 5 Other Surgical Tricuspid Repair Techniques

(A) De Vega repair: a single suture is placed around the tricuspid annulus, avoiding the area of the atrioventricular node. The suture is tied, completing the annuloplasty. **(B)** Tricuspid annuloplasty with rigid prosthetic ring: a sizer measuring the intertrigonal distance is used to determine the ring size. Multiple interrupted sutures are placed at the atrioannular junction. **(C)** The clover technique: the middle parts of the free edges of the tricuspid leaflets are sutured together, producing a clover-shaped valve. Figure illustration by Craig Skaggs.

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The most widely used prosthetic rings in surgical practice are currently the Carpentier-Edwards annuloplasty ring (Edwards Lifesciences LLC, Irvine, California), the flexible band Cosgrove-Edwards annuloplasty system (Edwards Lifesciences, LLC), and the Edwards MC3 ring system (Edwards Lifesciences, LLC).

The Carpentier-Edwards ring has been the repair device of choice for many years. The oval tricuspid ring conforms to the configuration of the normal tricuspid orifice and has an opening in the anteroseptal commissure that allows surgeons to avoid sutures in the area of the bundle of His. It has a 2-dimensional shape and is semirigid. Despite its good results (97.5% freedom from TV reoperation at 10 years) (8), it has been suggested that the mismatch in configuration between the Carpentier-Edwards ring and the TA has been responsible for incidental repair failure (15).

In 2002, a new generation of prosthetic ring was introduced. The MC3 ring has adopted the concept of remodeling annuloplasty. This ring has a 3-dimensional design and is preconfigured to best accommodate the saddle shape of the annulus.

Observational studies have compared the results with the 2 most used tricuspid rings, but no randomized trials have yet been conducted. Navia et al. (53) reported an incidence of recurrent TR ≥3/4+ 5 years after surgery with the standard Carpentier-Edwards ring and with the MC3 ring of 10% and 14% respectively, but this difference was not statistically significant.

Excellent reductions in TR severity early after surgery with the MC3 ring have been reported by other investigators (15). However, in some series, up to 14% of patients had significant TR 1 year after surgery: preoperative severe TR and increased leaflet tethering were the most significant predictors of residual TR (54).

Adjunctive repair techniques include the edge-to-edge "clover" technique (Fig. 5C), which consists of suturing the free margins of the leaflets in conjunction with ring annuloplasty (55) and anterior tricuspid leaflet augmentation (56). Excellent results with the clover technique were reported in a recent observational study (57); the combination of edge-to-edge repair with tricuspid annuloplasty is associated with a very low rate of recurrent significant TR (8.8% at 2 years after surgery) (53).

Risk factors for repair failure included higher preoperative TR severity, higher pulmonary artery pressures, larger ring size, MV replacement rather than repair, worse LV dysfunction, increased LV remodeling, suture annuloplasty, and the presence of pacemaker leads through the valve area (7,10,24).

Despite the absence of randomized trials, it is currently accepted by the majority of surgeons that ring repairs are more durable than suture repairs and are associated with better long-term and event-free survival (8,10,52,53).

Future Perspectives

Percutaneous procedures may be an attractive alternative to surgery for patients deemed to be high-risk surgical candidates. Whereas over the past few years, the development and clinical use of percutaneous approaches to the aortic valve and MV have been widespread, few data are available about the feasibility and the efficacy of the percutaneous TV approach. Some of the concepts that have been developed for the percutaneous treatment of mitral regurgitation may be adapted to percutaneous repair of the TV (percutaneous annuloplasty, edge-to-edge repair) (58).

Different new devices are currently under preclinical development: the Millipede system (Millipede, LLC, Ann Arbor, Michigan) involves the placement of a tricuspid annular ring with an attachment system via either minimally invasive surgical or percutaneous methods to restore the native tricuspid annular shape and diameter. It is repositionable and retrievable before deployment.

The feasibility of the implantation of a valved stent into the tricuspid position has been described using in vivo animal models (59). Iino et al. (60) reported the feasibility of off-pump transapical tricuspid valved stent implantation in an acute animal model, using a novel valved stent with a self-expandable, superabsorbent polymer to reduce paravalvular leakage. Hon et al. (61) described beating-heart transcatheter TV implantation to treat a degenerated bioprosthesis in tricuspid position through direct transatrial access.

An alternative approach to percutaneous treatment of TV is the implantation of separate valves in the superior vena cava and inferior vena cava to prevent damage to the liver and other organs (62).

The transcatheter implantation of a bioprosthesis in the tricuspid position presents challenging issues such as the large dimension of the TA, the slow flow of the right-heart side, and the trabeculated structure of the right ventricle (58).

With regard to access, the angulation of the annulus in relation to the superior vena cava and inferior vena cava requires careful consideration. Alternatively, transapical RV access may be used, but the RV wall is thinner than the LV wall, and multiple chordae may prevent the advance of the delivery system (58).

A 3-dimensional finite element model of the TV has been described, which may provide a starting point for the development of a predictive tool to evaluate quantitatively TV diseases and surgical or percutaneous repair procedures (63).

Conclusions

The treatment of STR has long been neglected but is now the subject of hot debate in the field of heart valve disease therapies.

Several studies have demonstrated improvements in functional status along with TR grade among patients undergo-

ing concomitant TV repair during mitral surgery, although data on survival benefits are equivocal. However, results are imperfect, possibly because of the complex physiopathology of the disease and the lack of knowledge. Percutaneous TV technologies may be useful for a large number of patients with STR undergoing left-side operations who are at high risk for open-heart surgery.

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Reprint requests and correspondence: Dr. Maurizio Taramasso, Cardiac Surgery Department, San Raffaele Scientific Institute, Via Olgettina 60, 20132 Milan, Italy. E-mail: taramasso.maurizio@hsr.it.

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