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STATE-OF-THE-ART PAPER

Tricuspid Regurgitation in Mitral Valve Disease

Incidence, Prognostic Implications, Mechanism, and Management

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Tricuspid regurgitation (TR) in patients with mitral valve (MV) disease is associated with poor outcome and predicts poor survival, heart failure, and reduced functional capacity. It is common if left untreated after MV replacement mainly in rheumatic patients, but it is also common in patients with ischemic mitral regurgitation. It is less common, however, in those with degenerative mitral regurgitation. It might appear many years after surgery and might not resolve after correcting the MV lesion. Late TR might be caused by prosthetic valve dysfunction, left heart disease, right ventricular (RV) dysfunction and dilation, persistent pulmonary hypertension, chronic atrial fibrillation, or by organic (mainly rheumatic) tricuspid valve disease. Most commonly, late TR is functional and isolated, secondary to tricuspid annular dilation. Outcome of isolated tricuspid valve surgery is poor, because RV dysfunction has already occurred at that point in many patients. MV surgery or balloon valvotomy should be performed before RV dysfunction, severe TR, or advanced heart failure has occurred. Tricuspid annuloplasty with a ring should be performed at the initial MV surgery, and the tricuspid annulus diameter (≥3.5 cm) is the best criterion for performing the annuloplasty. In this article we will review the current data available for understanding the prognostic implications, mechanism, and management of TR in patients with MV disease. (J Am Coll Cardiol 2009;53:401–8) © 2009 by the American College of Cardiology Foundation

A 74-year-old man is admitted to the hospital for the treatment of exacerbation of congestive heart failure from which he has suffered for the last 2 years. He has rheumatic heart disease and had mitral valve replacement (MVR) with a mechanical bileaflet prosthetic valve for predominantly mitral regurgitation (MR) 12 years before his admission. At that time he had good left ventricular (LV) contraction, his pulmonary artery pressure was 32/16 mm Hg, and he had no clinical tricuspid regurgitation (TR). He is in chronic atrial fibrillation (AF), his neck veins are distended with prominent V waves, and he has an enlarged pulsating liver and severe peripheral edema. Echocardiography reveals a normally functioning prosthetic mitral valve (MV), good LV contraction, and no aortic valve disease. The right ventricle (RV) and right atrium are dilated and RV function is reduced. The tricuspid valve (TV) leaflets appear normal, but the tricuspid annulus (TA) is dilated and measures 4.5 cm, and there is severe malcoaptation of the TV leaflets and severe TR (Fig. 1, Online Videos 1 and 2).

The clinical problem. The preceding case illustrates the problem of TR in patients with MV disease. Management of such patients is challenging and difficult at such a stage because of advanced RV dysfunction and failure. The best treatment is in fact prevention at an earlier stage and requires understanding the mechanism, natural history, and consequence of TR in patients with MV disease, which is the focus of this review. Since the problem of late TR was reviewed by Groves et al. in 1992 (1), new data have become available. Unfortunately, randomized prospective trials to guide the management of such patients are lacking, as they are in other areas in valvular heart disease.

Patients who have severe TR at the time of MV surgery should obviously have their TV repaired at the time of the initial MV surgery (2,3). In patients with less than severe TR, however, TR might progress after surgery if the TV is left untreated. Matsuyama et al. (4) reported significant TR (at least grade 3) on echocardiography performed late after MVR in 37% of the patients with grade 2 TR before surgery.

In this review, we will summarize the new available data accumulated regarding TR in patients with MV disease with emphasis on the best criteria for concomitant TV repair in patients undergoing MV surgery.

Incidence of TR in MV disease. Tricuspid regurgitation is frequently present in patients with MV disease, and more than one-third of the patients with mitral stenosis have at least moderate TR (5,6). Clinically severe TR has been reported in 23% to 37% of patients after MVR for rheumatic heart disease (7,8). In 14%, TR occurred in the absence of significant left heart disease, pulmonary hyper-

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Abbreviations and Acronyms

AF = atrial fibrillation
LV = left ventricle/ventricular
MR = mitral regurgitation
MV = mitral valve
MVR = mitral valve replacement
RV = right ventricle/ventricular
TA = tricuspid annulus
TR = tricuspid regurgitation
TV = tricuspid valve

tension, or obvious organic TV disease (7). The incidence of echocardiographically moderate or severe late TR in rheumatic patients is even higher (68%) (8). In most cases TR is diagnosed late after MVR, 10 years on average, but can appear as late as 24 years after the initial surgery (7,8).

Although late TR has most often been reported in patients with rheumatic heart disease, it is not confined to rheumatic patients (9,10). Moderate or severe TR was reported in as many as 74% of patients 3 years after surgical repair of ischemic MR (10). De Bonis et al. (11) reported 14% grade 3 or more TR in patients who had surgery for functional MR secondary to dilated cardiomyopathy (70% ischemic and 30% nonischemic), and those patients had concomitant TV repair. Grade 3 or more TR was still present in 22% of the patients 3.5 years after surgery. Dreyfus et al. (9) reported 34% late TR (grade 3 or 4) in a group of 163 patients with a mixed etiology for MR who were followed for 5 years after MV repair. The most common etiology in this group was degenerative (Barlow's disease in 38%, dystrophic in 27%), followed by ischemic in 13% and rheumatic in only 11%. Tricuspid regurgitation is probably less frequent in patients with MR secondary to MV prolapse. Koukoui et al. (12) reported moderate or severe TR in 15% of patients with MV prolapse and at least moderate MR (n = 477). Tricuspid regurgitation progressed in 14% of the patients during a mean follow-up of 4 years. Late TR is probably less of a problem in MV prolapse, but more data and longer follow-up are needed (13).

Prognostic implications of TR in patients with MV disease. Patients with mitral stenosis and moderate or severe TR before MVR are more likely to have class III or IV heart failure after a mean follow-up of 8 years compared with patients with mild TR (56% vs. 14%) (6). Tricuspid regurgitation after MVR predicts poor outcome. Ruel et al. (14) reported the risk factors for heart failure and death in 708 patients after MVR. Moderate-to-severe TR on echocardiography during follow-up was an independent predictor of New York Heart Association functional class III or IV heart failure, heart failure-related death, and even all-cause mortality during the 5 years of follow-up. Only 77% of the patients in this study, however, had follow-up echocardiography.

In a smaller study (n = 42), Henein et al. (15) reported a 5-year survival of only 50% in rheumatic patients with clinical and echocardiographic severe TR after MV surgery, compared with no deaths in patients with mild TR.

Significant TR requiring TV surgery predicts poor survival in patients undergoing valve surgery (16,17). Tricuspid regurgitation is also a predictor of poor outcome in patients undergoing balloon mitral valvotomy for mitral stenosis. Patients with pre-procedural severe TR have more severe MV disease, higher pulmonary vascular resistance, a smaller increase in MV area after valvotomy, as well as poorer

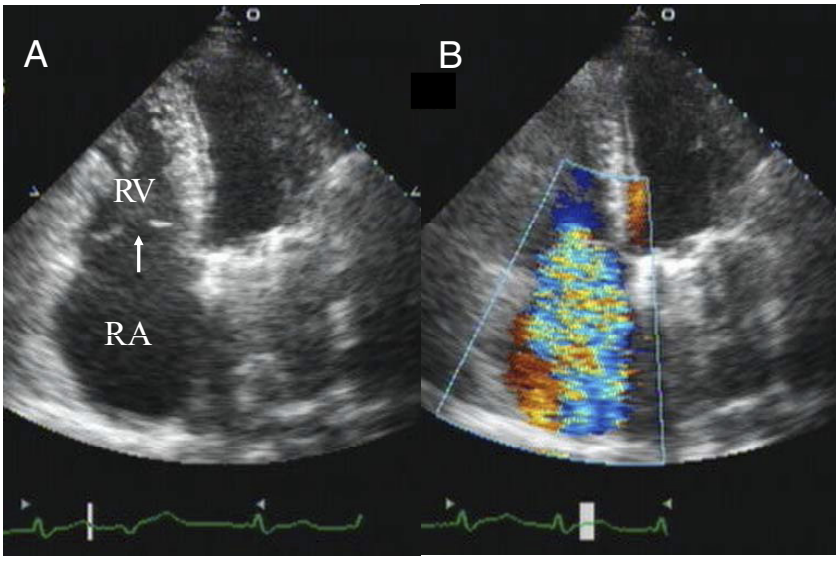

Figure 1 Patient With Late Tricuspid Regurgitation After Mitral Valve Replacement

(A) Apical 4-chamber view showing right ventricle (RV) and tricuspid annulus dilation (tricuspid annulus diameter 4.5 cm) with severe malcoaptation of tricuspid valve leaflets during systole **(arrow)** (Online Video 1). **(B)** Color flow Doppler showing severe tricuspid regurgitation (Online Video 2). RA = right atrium.

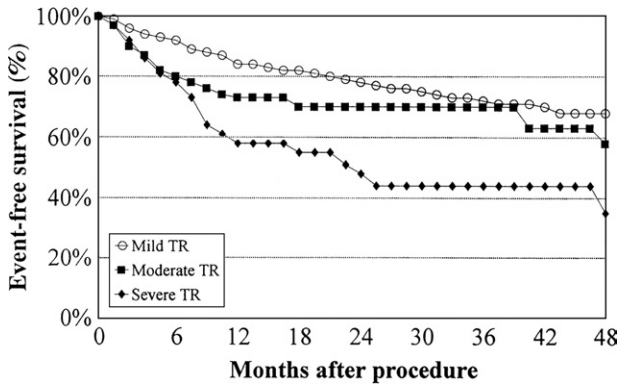

Figure 2 **Event-Free Survival After Balloon Mitral Valvotomy by TR Severity**

Events were defined as death, New York Heart Association functional class III or IV, MV surgery, or repeat mitral valve balloon valvotomy (n = 318). Patients with severe tricuspid regurgitation (TR) had significantly worse event-free survival compared with patients with mild TR. Reprinted, with permission, from Sagie et al. (18).

outcome: lower overall survival, more heart failure, and need for repeat valvotomy or MVR (Fig. 2) (18).

Patients with TR after MVR have reduced exercise capacity compared with patients without TR. Groves et al. (19) have shown that patients with isolated severe TR after MVR have reduced exercise duration, maximal oxygen consumption, and anaerobic threshold compared with patients without TR, despite having good LV and prosthetic valve function.

Patients with severe TR after MVR undergoing isolated TV surgery usually have a poor outcome with high perioperative mortality, poor late survival, and no significant improvement in functional capacity in many of them (20–23). Perioperative mortality might reach 50%, but it was usually reported between 11% and 20% (13,20,23,24). Mangoni et al. (20) reported 20% operative mortality and 1.2 years' median survival in 15 patients, most of them rheumatic, undergoing isolated TV replacement. One-half of the surviving patients were in New York Heart Association functional class III or IV. The results might be better for patients with preserved RV function referred early for surgery (23), but data regarding the outcome of such an approach and the optimal timing for surgery in such patients is lacking.

Pathogenesis of TR in MV disease. The pathogenesis of TR in MV disease is complex and multifactorial (Fig. 3). Most often TR is functional, secondary to RV dilation and dysfunction and tricuspid annular dilation. Mitral valve disease (usually rheumatic or ischemic) leads to mitral stenosis or regurgitation, which in turn leads to increased left atrial pressure and, if severe enough, to secondary pulmonary hypertension. Long-standing pulmonary hypertension might lead to RV dysfunction and remodeling, which leads to TA dilation, papillary muscle displacement, and tethering of the TV leaflets, leading to TR (25–29). Tricuspid regurgitation itself leads to further RV dilation and dysfunction, more TV annular dilation and tethering, and worsening TR. With increasing TR the RV dilates and eventually fails, causing increased RV diastolic pressure and a shift of the interventricular septum toward the LV. Because of ventricular interdependence, this might compress the LV, causing restricted LV filling and increased LV diastolic and pulmonary artery pressure. This phenomenon

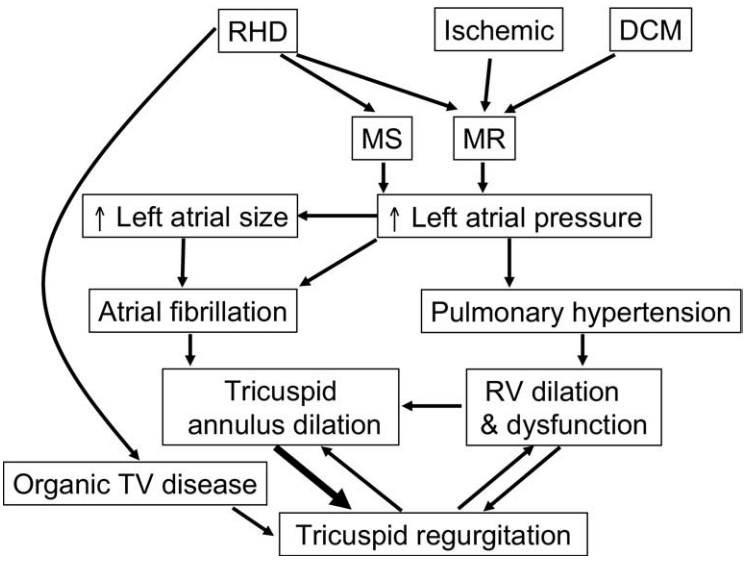

Figure 3 **Pathogenesis of Tricuspid Regurgitation in Mitral Valve Disease**

DCM = dilated cardiomyopathy; MR = mitral regurgitation; MS = mitral stenosis; RHD = rheumatic heart disease; RV = right ventricle; TV = tricuspid valve.

was named "restriction dilation syndrome" by Antunes and Barlow (13).

Increased left atrial size and pressure might also cause AF, which in turn causes right atrial dilation leading to further tricuspid annular dilation. Atrial fibrillation has been recognized as an important risk factor for the development of TR in patients with MV disease as well as for the persistence or occurrence of TR after MV surgery or balloon mitral valvotomy (4,30,31). Furthermore, patients who have a concomitant successful Maze procedure during their MV surgery were reported to have significantly less TR at follow-up (32).

In patients with rheumatic valve disease, organic TV involvement might also cause TR. Tricuspid valve leaflet thickening and restriction had been reported in about one-third of the patients with moderate or severe TR after MVR, but the true incidence of organic TV involvement might be higher (8). With transesophageal echocardiography and 3-dimensional reconstruction, Henein et al. (15) found more subtle organic TV involvement in 12 of 15 patients with isolated severe TR who had MVR for rheumatic heart disease. Scarring, leaflet thickening, and even some chordal shortening are routinely found when resected TVs from patients with functional TR are carefully examined (33). These findings, unfortunately, are nonspecific for rheumatic valvular changes and are frequently found in patients with TR and severe RV hypertension secondary to congenital heart disease (33).

In many patients with severe pulmonary hypertension, pulmonary pressure regresses after successful MVR or balloon valvotomy (34–36). The immediate decrease in pulmonary artery pressure is due to the elimination of the passive component of pulmonary hypertension. It depends on effective lowering of left atrial pressure, which might be hindered by prosthesis-patient mismatch, suboptimal relief of mitral stenosis by balloon valvotomy, or significant MR (35–37). A progressive decline in reactive pulmonary arteriolar vasoconstriction might further decrease pulmonary resistance over a period of 1 week to several months, especially in younger patients with less chronic disease (35,38–40). An irreversible component, caused by pulmonary arteriolar medial hypertrophy, might cause persistent or recurrent pulmonary hypertension (36).

Although pulmonary hypertension is important in the pathogenesis of late TR, it might be normal before MVR (19). Porter et al. (8) reported that pre-operative pulmonary artery pressure did not predict late TR. Kaul et al. (41), in an elegant study, reported the outcome of 86 rheumatic patients with moderate functional TR undergoing MVR. They found that, compared with patients with nonsevere pulmonary hypertension (systolic pulmonary artery pressure 41 ± 6 mm Hg), patients with severe pulmonary hypertension (78 ± 14 mm Hg) had far less late TR at follow-up, better functional capacity, and significantly better survival. The probable reason for this surprising finding was that patients with nonsevere pulmonary hypertension had worse RV function. Right ventricular function is an important determinant of outcome in patients with MV disease and TR (23,42). An alternative explanation is that patients with nonsevere pulmonary hypertension were more likely to have organic TR.

Tricuspid annular dilation is probably the most important factor in the development of late TR, and it is also the target for intervention. The normal TA is saddle-shaped, with the highest points located in an anteroposterior orientation and the lowest points in a mediolateral orientation. With the development of functional TR, the TA becomes dilated and more planar and circular (43,44). Antunes and Barlow (13) suggested that in rheumatic patients direct involvement of the TA by the rheumatic process might weaken the annulus and cause it to dilate. The normal TA diameter, as measured by echocardiography in the 4-chamber view between the base of the septal and the base of the lateral TV leaflets, is 2.8 ± 0.5 cm (28). Sugimoto et al. (27) reported a good correlation between TA diameter and TR regurgitant volume (Fig. 4). In addition, annular dilation and not pulmonary hypertension, RV dilation, or tricuspid tenting is the best determinant of functional TR (28). Reduced tricuspid annular shortening, encountered in patients with severe tricuspid annular dilation and RV dysfunction, also determines TR severity (26,45). Interestingly, in patients with chronic pulmonary thromboembolic hypertension in whom TR resolved after pulmonary thromboendarterectomy and in patients who had successful mitral balloon valvotomy, there was no change in TA diameter after resolution of pulmonary hypertension (31,46). This implies that TA dilation might be irreversible and might explain the mechanism of late TR in MV disease.

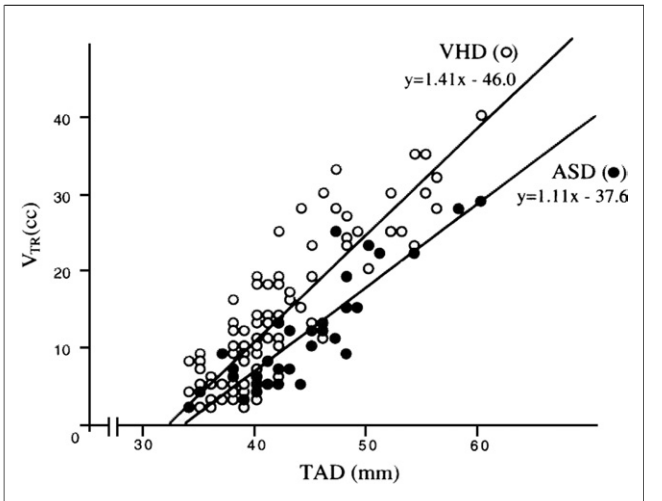

Figure 4 Correlation Between TAD and V_{TR}

There is good correlation in both patients with valvular heart disease (VHD) (r = 0.87) and patients with atrial septal defect (ASD) (r = 0.88). The correlation lines cross the x-axis at a tricuspid annulus diameter (TAD) value of 33 to 34 mm, which is the threshold for tricuspid regurgitation. Reprinted, with permission, from Sugimoto et al. (27). V_{TR} = tricuspid regurgitant volume.

Although late TR after MVR is often isolated and occurs in the absence of significant left heart disease, it is important to look for a dysfunctional prosthetic MV (paravalvular leak or stuck leaflet) by transesophageal echocardiography in patients presenting with severe TR late after MVR (47).

Does TR resolve after correcting the MV lesion? Lessons from patients undergoing balloon mitral valvotomy or MVR. In an early report by Braunwald et al. (33) it was suggested that TR resolves after MVR and there is no need for concomitant TV surgery. Lowering pulmonary artery pressure might eliminate severe functional TR. Severe TR resolved in 70% of the patients with chronic pulmonary thromboembolic hypertension who were examined early after pulmonary thromboendarterectomy (46). Later, it became apparent that TR might not resolve after MV surgery, might become clinically apparent more than 20 years after the surgery, and is fairly common (8,45,48,49). In patients undergoing balloon mitral valvotomy for mitral stenosis, the true natural history of concomitant TR can be determined, because the TV is left untreated. Tricuspid regurgitation did not improve in 49% to 80% of the patients with moderate or severe TR after successful mitral balloon valvulotomy (30,50). Tricuspid regurgitation was more likely to improve in patients with the following characteristics: 1) younger age; 2) functional (as opposed to organic) TR; 3) smaller MV area; 4) severe pulmonary hypertension; 5) larger resolution of pulmonary hypertension after valvotomy; and 6) no AF (30). Song et al. (31) compared mitral valvotomy and surgical treatment (MV surgery and TV repair) in 92 patients with mitral stenosis and severe TR. Although event-free survival was not different in this relatively small retrospective study, patients in the surgical group were older and had more AF and a higher MV score. Event-free survival at 7 years, however, was significantly better in the subgroup of patients with AF who had surgery compared with those with AF who had valvotomy without TV repair, mainly due to heart failure events secondary to TR in the valvotomy group. Furthermore, 98% of the patients in the surgical group were free of grade ≥2 TR, compared with 46% of the patients who had balloon valvotomy.

The resolution of TR reported by Braunwald et al. (33) in their early study might be explained by the fact that their patients were relatively young, with good RV function as evidenced by high systolic pulmonary artery pressure (average 75 mm Hg) and relatively low right atrial pressure (average 11 mm Hg). Furthermore, the follow-up in this study was short (30 months on average), whereas TR might appear many years after MVR.

Management of TR in MV disease. Importance of concomitant tricuspid annuloplasty at the time of initial MV surgery. Because late TR in MV disease is usually due to TA dilation (although rheumatic patients might have organic leaflet disease as well) and carries significant morbidity and mortality, and because the results of repeat surgery for isolated late TR are poor (20,22,24), concomitant TV repair with an annuloplasty ring should be performed at the time of the initial MV surgery. Tricuspid valve annuloplasty adds little time and complexity to MV surgery and results in very few complications (9,51,52). In patients with non-severe organic TV disease, TV repair is probably better than replacement (51). Bioprosthetic valves degenerate with time, and mechanical valves in the tricuspid position tend to thrombose (53). Singh et al. (51) reported a 5-fold risk of early death during the perioperative period with TV replacement compared with TV repair, and in their study TV replacement was an independent predictor of poor survival after surgery. In some patients with severe organic leaflet involvement, TV repair might not be possible and the valve should be replaced to avoid recurrent severe TR or TS, but with less severe leaflet disease suboptimal results with mild residual TR might be well tolerated (in contrast to MV repair, where suboptimal initial results are usually not accepted) (13).

Tricuspid valve repair with an annuloplasty ring resulted in significantly improved long-term survival (15 years), event-free survival, and survival free of recurrent TR compared with De Vega suture annuloplasty in the study reported by Tang et al. (52). Tricuspid valve ring annuloplasty was also an independent predictor of long-term survival in that study. The durability of TV repair was assessed by McCarthy et al. (54). All TV repair techniques had an immediate failure rate (grade 3 TR or more) of approximately 14%. Whereas patients who had a ring annuloplasty with a semi-rigid ring (Carpentier-Edwards) had no progression of TR, more than 30% of the patients who had a De Vega procedure had significant TR after 8 years (54). Some surgeons report good results with a modified De Vega repair with pledgeted sutures in patients with nonsevere organic TV involvement (13).

The best evidence for the utility of TV ring annuloplasty during MV surgery and the importance of TA diameter as a criterion for TV repair comes from the study of Dreyfus et al. (9). They studied 311 patients undergoing MV repair (65% degenerative and only 14% rheumatic MV disease). The TV annulus diameter was measured intraoperatively with a ruler, from the anteroseptal commissure to the anteroposterior commissure. The TV repair with a Carpentier-Edwards ring was performed regardless of the degree of TR, if the TA diameter was ≥7 cm (equivalent to 4 cm by echocardiography [A. Berrebi, personal communication, November 2006]). Tricuspid valve repair was performed in 48% of the patients, despite the fact that 88% had grade ≤1 TR. Although the patients who had TV repair were sicker, they tended to have better survival (90.3% vs. 85.5% at 10 years, $p = NS$) and had significantly less TR (0.7% vs. 34% grade 3 or 4 TR, $p < 0.01$) and better functional capacity at follow-up (0% vs. 14% functional capacity 3 or 4, $p < 0.001$). These results imply that the TA diameter threshold for repair should have been lower, somewhere between 3.0 and 4.0 cm. Perhaps it is best to "freeze" the normal TA at 3.0 cm. Groves et al. (1)

suggested a threshold of 2.1 cm/m^2 (equivalent to 3.6 cm for an average person). They relied on a study by Chopra et al. (55), who found this diameter to best differentiate severe from nonsevere TR. In their study the authors did not attempt, however, to predict late TR. The threshold should probably be lower with increasing degrees of TR and in rheumatic patients. Prospective, randomized trials are needed to better define this cutoff point. On the basis of the available information and personal experience, we believe that prophylactic TV repair should be performed in patients undergoing MVR regardless of TR severity whenever the TV annulus is ≥3.5 cm, especially in rheumatic TR.

We believe that this approach is appropriate for patients with ischemic MR and also for patients with functional MR secondary to dilated cardiomyopathy (ischemic and nonischemic). Radovanovic et al. (56,57) reported good results with systematic MV and TV annuloplasty in relatively young patients (mean age 50 to 55 years) with ischemic and nonischemic dilated cardiomyopathy, but his studies were nonrandomized. Early TV repair before the occurrence of irreversible RV dysfunction is probably also appropriate in patients with isolated TR secondary to TA dilation without left-sided heart disease (58).

Treatment of patients who develop late isolated TR after MVR like the patient we described is difficult (2,3). Aggressive antifailure therapy with loop diuretic drugs and spironolactone is the mainstay of therapy and might retard TR progression. In some patients in whom surgery is no longer an option, chronic dialysis might prove useful, in our experience, in treating volume overload and improving life quality. Early on, when patients have relatively few symptoms, the high-risk surgery might seem unjustified. Later, when symptoms justify surgery, RV dysfunction might already be irreversible. Right ventricular function is still hard to measure by conventional echocardiography. Recently, tissue Doppler imaging was found to be helpful in identifying patients with preserved RV function (tricuspid systolic annular velocity >9.5 cm/s) (23). Newer techniques such as 3-dimensional echocardiography, magnetic resonance imaging, and Doppler indexes such as the RV myocardial performance index (MPI) and isovolumic acceleration index (IVA) might prove useful in the future to better assess RV function and might help to find the ideal timing of surgery to avoid irreversible RV dysfunction (59).

Current guidelines for TV repair and replacement. Both the American College of Cardiology/American Heart Association and the European Society of Cardiology (ESC) guidelines give a class I recommendation for TV repair in patients with severe TR undergoing MV surgery (2,3). The ESC guidelines give a class IIa recommendation for concomitant TV repair in patients with a TA diameter >40 mm or moderate TR, whereas the American College of Cardiology/American Heart Association gives a more vague, class IIb recommendation for patients with less than severe TR. The ESC also gives a class IIa recommendation for TV repair in patients with symptomatic, isolated TR late after left-sided valve surgery, in the absence of left-sided myocardial or RV dysfunction and without severe pulmonary hypertension. On the basis of the current review, we agree more with the European view and support a more aggressive approach toward TV repair. Surgical intervention is not indicated in asymptomatic patients with severe TR developing RV dysfunction, in contrast to asymptomatic patients with severe MR or AR, in whom there is a clear and strong indication for surgical intervention when even mild LV dysfunction is present. Therefore, many patients with severe TR are referred for TV surgery too late, when RV dysfunction has already occurred and in a poor functional class. This might explain the poor surgical results in many of them.

Recommendations. Detailed TV assessment, including measurement of the TA diameter, is mandatory in patients with MV disease. Mitral valve surgery or balloon valvotomy should be performed before RV dysfunction, severe TR, or advanced heart failure has occurred. This is similar to the current approach of performing MV repair before the occurrence of LV dysfunction or closing an atrial septal defect before the occurrence of RV dysfunction.

A TV annuloplasty with a ring is the best procedure to correct or prevent TR in most cases. It improves survival, prevents late TR and heart failure, and should therefore be performed at the time of the initial MV surgery. The TA diameter is the best guide to select patients for TV repair, and the echocardiographic cutoff should be somewhere around 3.5 cm regardless of TR severity and might be lower for rheumatic patients. Patients with significant TR who are older and have longstanding MV disease, AF, nonsevere pulmonary hypertension, and organic TV disease will benefit more from MV surgery and TV repair/replacement than from percutaneous balloon valvotomy.

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Key Words: congestive heart failure ■ mitral valve ■ tricuspid regurgitation ■ tricuspid valve ■ valve surgery.

APPENDIX

For accompanying videos and legends, please see the online version of this article.