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Mechanisms of Recurrent Aortic Regurgitation After Aortic Valve Repair

Predictive Value of Intraoperative Transesophageal Echocardiography

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OBJECTIVES The aim of the present study was to examine the intraoperative echocardiographic features associated with recurrent severe aortic regurgitation (AR) after an aortic valve repair surgery.

BACKGROUND Surgical valve repair for AR has significant advantages over valve replacement, but little is known about the predictors and mechanisms of its failure.

METHODS We blindly reviewed all clinical, pre-operative, intraoperative, and follow-up transesophageal echocardiographic data of 186 consecutive patients who underwent valve repair for AR during a 10-year period and in whom intraoperative and follow-up echo data were available. After a median follow-up duration of 18 months, 41 patients had recurrent 3+ AR, 23 patients presented with residual 1+ to 2+ AR, and 122 had no or trivial AR. In patients with recurrent 3+ AR, the cause of recurrent AR was the rupture of a pericardial patch in 3 patients, a residual cusp prolapse in 26 patients, a restrictive cusp motion in 9 patients, an aortic dissection in 2 patients, and an infective endocarditis in 1 patient.

RESULTS Pre-operatively, all 3 groups were similar for aortic root dimensions and prevalence of bicuspid valve (overall 37%). Patients with recurrent AR were more likely to display Marfan syndrome or type 3 dysfunction pre-operatively. At the opposite end, patients with continent AR repair at follow-up were more likely to have type 2 dysfunction pre-operatively. After cardiopulmonary bypass, a shorter coaptation length, the degree of cusp billowing, a lower level of coaptation (relative to the annulus), a larger diameter of the aortic annulus and the sino-tubular junction, the presence of a residual AR, and the width of its vena contracta were associated with the presence of AR at follow-up. Multivariate Cox analysis identified a shorter coaptation length (odds ratio [OR]: 0.8, $p = 0.05$), a coaptation occurring below the level of the aortic annulus (OR: 7.9, $p < 0.01$), a larger aortic annulus (OR: 1.2, $p = 0.01$), and residual aortic regurgitation (OR: 5.3, $p = 0.01$) as risk factors of repair failure.

CONCLUSIONS Our results demonstrate that intraoperative transesophageal echocardiography can be used to identify patients undergoing AR repair who are at increased risk for late repair failure. (J Am Coll Cardiol Img 2009;2:931–9) © 2009 by the American College of Cardiology Foundation

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Aortic valve repair is increasingly favored over replacement with a prosthetic valve for a variety of lesions causing significant aortic regurgitation (AR) (1–6). Potential advantages of aortic valve repair over replacement include a lower incidence of subsequent thromboembolic events, the avoidance of long-term anticoagulation, and reduced risks of endocarditis (7). Despite these potential benefits, the durability of aortic repair procedures has been a matter of concern, with their success rate being varied and greatly dependent upon the etiology and mechanisms of aortic valve dysfunction and the experience of the surgeon (8).

Because it provides excellent real-time visualization of the anatomy of heart valves, intraoperative transesophageal echocardiography (TEE) has become the method of choice in operative decision making, guidance of reconstructive techniques, and the assessment of the adequacy of mitral valve repair (9). We have recently demonstrated that intraoperative TEE was equally useful in delineating the mechanisms of AR and in predicting aortic valve repairability (10). Currently, no data are available on the ability of intraoperative TEE to identify patients who will develop recurrent severe AR after an initially successful aortic reconstruction procedure. Accordingly, the aim of the present study was to examine the causes of recurrent severe AR after an aortic valve repair and to evaluate the ability of intraoperative TEE to identify patients at increased risk for recurrent severe AR.

ABBREVIATIONS AND ACRONYMS

AR = aortic regurgitation

LV = left ventricular

TEE = transesophageal echocardiography

METHODS

Study population. Between December 1995 and January 2007, 244 consecutive patients (79% men, mean age 54 ± 14 years, range 22 to 84 years) underwent aortic valve repair for isolated severe AR at the Cliniques Universitaires St-Luc in Brussels. The mechanism of AR was type 1 dysfunction in 93 patients (38%), type 2 dysfunction in 94 (39%), and type 3 dysfunction in 57 (23%) (see definitions in the data analysis section). Patients were considered for inclusion in the present study if interpretable intraoperative and follow-up echocardiographic images were available either on our echocardiographic image server or on videotapes.

Transthoracic and TEE. Pre-operative, intraoperative, and follow-up echocardiographic examinations were performed with commercially available ultrasound systems. The digitally stored echocardiographic studies were reviewed retrospectively, with the readers being blinded to the initial pathology, the course of the initial operation, and the patient's clinical status. This review was directed to 1) determine the mechanism of recurrent AR based on the follow-up echocardiographic appearance of the repaired aortic valve and on the origin and direction of the regurgitant jet and 2) measure relevant intraoperative aortic root and valve dimensions (Fig. 1). Demographic, etiologic, and echocardiographic variables were compared among 3 groups of patients depending on residual AR severity at follow-up: no/trivial AR, moderate (1+ to 2+) AR, and severe (≥3+) AR.

Data analysis. Aortic cusp and root lesions were categorized as previously described (10,11). In brief, 3 main mechanisms of AR were identified: dilation of the aortic root (type 1); excess cusp motion, with good cusp tissue quality (type 2); and poor cusp tissue quality, including cusp retraction, extensive cusp calcifications, and/or endocarditis (type 3). Type 1 dysfunction was identified when the dimensions of any components of the aortic root exceeded the upper limits of published normal values (12) and no other cause of AR was identified. Type 2 dysfunction was considered in the presence of an eccentric AR jet and either a cusp prolapse or a cusp fenestration. Cusp prolapse was considered whenever the free edge of 1 or more of the aortic cusps overrode the plane of the aortic annulus. Type 3 dysfunction was considered whenever the quality or quantity of the cusp tissue was judged to be poor. Thickened and rigid valves with reduced motion, valves whose leaflet tissue had been destroyed by infective endocarditis, and severely calcified valves were included in this category.

In addition to the careful description of the mechanisms of AR, several echocardiographic measurements were obtained before and after the repair (Fig. 1). All measurements were made according to the leading edge technique, on end-diastolic long-axis still frames of the aortic valve and root (13). Whenever present, post-repair residual AR jets were described as eccentric (when the main axis of the jet formed an angle >45° with the left ventricular outflow tract) or central (when the main axis of the jet paralleled that of the left ventricular outflow tract and was directed toward the apex). The severity of AR was semiquantitatively evaluated by measurement of the width of the vena contracta on the basis of the recommendations of the American Society of Echocardiography (14).

Surgical strategies. The surgical correction was tailored to the mechanisms of valve dysfunction (11). In brief, type 1 dysfunction was corrected by the root reimplantation or remodeling techniques. Type 2 dysfunction was corrected by triangular resection, plication, or resuspension in association with a root procedure whenever needed. Type 3 dysfunctions were treated by shaving, decalcification, resection, and patch-extension techniques.

Statistical analysis. All analyses were conducted using SPSS software version 15.0 (SPSS Inc., Chicago, Illinois). Continuous variables were expressed as mean ± 1 SD and categorical variables as counts and percentages. Differences between groups were analyzed with a 1-way analysis of variance or chi-square statistics where appropriate. A probability value of <0.05 was considered indicative of a statistically significant difference.

All echocardiographic variables were submitted to a Cox proportional hazards survival model. The Cox model was built to identify independent predictors of recurrent ≥3+ AR. Relative hazard ratios for each specific covariate of the final model were computed as the exponential of the regression coefficient. The most powerful predictors of recurrent ≥3+ AR at follow-up were then used to construct a stepwise clinical algorithm that could be used intraoperatively to predict the risk of subsequent recurrent AR. For this purpose, continuous parameters were binarized by receiver-operator characteristic curves analysis. Optimal cut-off values for these parameters were computed as the maximum of the Youden index (15). Finally, survival curves for each predictive factor combination were computed by the Kaplan-Meier method and compared with the log-rank chi-square test.

RESULTS

Population characteristics. Among the 244 patients in whom aortic valve repair was performed during the study period, 186 met the inclusion criteria. Figure 2 shows the patient and selection flow chart. After a median follow-up of 18 months, 122 had no or trivial AR (102 men, mean age 53 ± 13 years, range 22 to 84 years), 23 patients presented with 1+ to 2+ AR (16 men, mean age 63 ± 12 years, range 42 to 82 years), and 41 patients had ≥3+ AR (35 men, mean age 50 ± 16 years, range 22 to 84 years). Table 1 shows the baseline characteristics of these 3 groups. Patients with residual or recurrent AR were more likely to display Marfan syndrome or type 3 dysfunction pre-operatively. At the opposite, patients with continent AR repair at follow-up were more likely to have type 2 dysfunction pre-operatively.

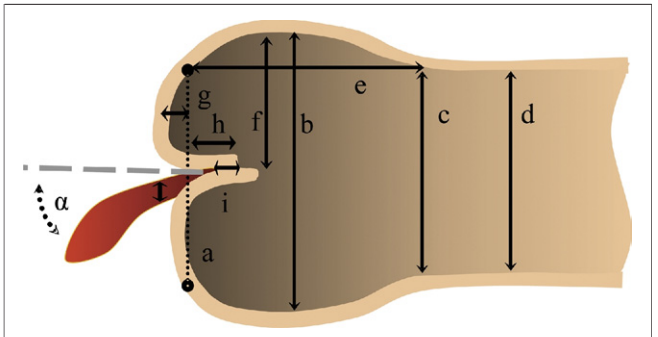

Figure 1. Schematic Representation of the TEE Measurements

Measurements were performed pre-operatively and immediately after bypass. **(a)** Aortic annulus; **(b)** sinuses of Valsalva; **(c)** sino-tubular junction; **(d)** ascending aorta; **(e)** height of the sinus of Valsalva; **(f)** distance from coaptation tips to aortic wall (the symmetry of coaptation within the sinuses of Valsalva was estimated by the absolute difference of the distance separating the tip of the coaptation from the anterior and the posterior border of the sinus of Valsalva); **(g)** distance from the aortic annulus to the belly of the lowest cusp (degree of cusp billowing if present); **(h)** distance from the tip of the cusp coaptation to the aortic annulus (relative level of cusp coaptation); and **(i)** the coaptation length. α = angle between regurgitant AR jet and left ventricular outflow tract. AR = aortic regurgitation; TEE = transesophageal echocardiography.

Mechanisms of late failure of aortic valve repair. Among the 41 patients who had ≥3+ AR at follow-up, 23 needed a reoperation. Indications for redo surgery were development of AR-related symptoms in 21 patients and acute aortic dissection in 2 patients. The surgical findings at the time of the index operation as well as the surgical techniques used to repair the valve in the 41 patients with ≥3+ AR at follow-up are shown in Table 2.

Figure 3 shows representative immediate intraoperative post-repair results in 3 patients with ≥3+ AR at follow-up. Overall, the causes of ≥3+ AR at follow-up included residual or newly appearing cuspal prolapses in 26 patients, restrictive cusp motion in 9 patients, dehiscence of valvular sutures in 3 patients, aortic dissection in 2 patients, and recurrent endocarditis in 1 patient. Among the 26 patients with cuspal prolapses at follow-up, 20 (77%) had an eccentric AR jet before leaving the operating room at the time of the index surgery. Similarly, among the 9 patients with restrictive cusp motion at follow-up, 4 (44%) initially had type 3 rheumatic dysfunction, and 3 others presented with an infective endocarditis pre-operatively. In the 23 patients that needed a reoperation, direct surgical inspection of the aortic valve and root confirmed

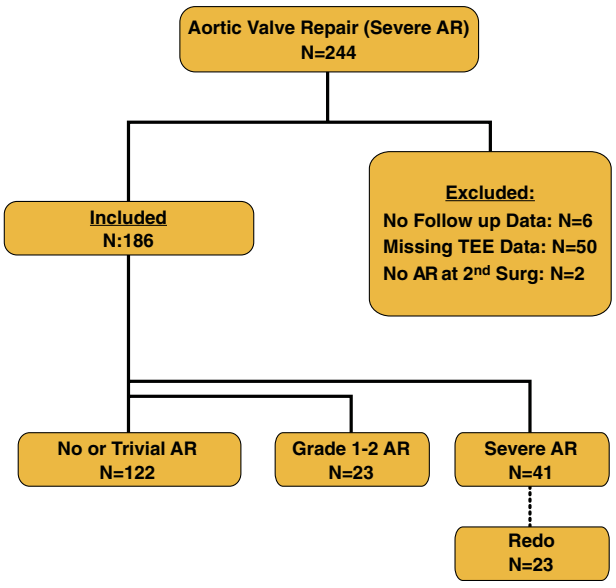

Figure 2. Flow Chart of the Study Protocol

During the study period, aortic valve repair was performed in 244 patients; 56 patients lacking TEE data or follow-up were excluded from the present study. In addition, 2 patients were excluded because their aortic valve was reoperated on in the absence of severe recurrent AR (one had an aorto-right ventricular communication, and the other had a pure calcified aortic stenosis). Ultimately, 122 patients with no or trivial AR, 23 patients with 1+ to 2+ AR, and 41 patients with ≥3+ AR were included. Abbreviations as in Figure 1.

the mechanisms identified by intraoperative TEE. In these patients, reoperation consisted of a prosthetic replacement in 11 patients, a second repair of the native valve in 5 patients, and a Ross procedure in 7 patients. Three of the 5 patients in whom the aortic valve could be successfully re-repaired had a recurrent cusp prolapse, 1 presented with a rupture of a pericardial patch, and 1 presented with a type 3 dysfunction.

Among the 23 patients with 1+ to 2+ AR at follow-up, the mechanism of AR was an isolated cusp prolapse or fenestration in 6 patients, a restrictive cusp motion in 9 patients, and dehiscence of valvular sutures in 2 patients. In the absence of follow-up TEE or reoperation, the cause of recurrent AR remained uncertain in 6 patients.

Table 1. Pre-Operative and Intraoperative Characteristics of the Study Population

	No/Trivial AR (n = 112)	1+ to 2+ AR (n = 23)	≥3+ AR (n = 41)	p Value (F or Chi-Square)
Age (yrs)	53 ± 13	50 ± 16	63 ± 12	0.01
Sex (female)	18%	30%	15%	0.27
Marfan syndrome	2%	9%	15%	0.005
Type 1 AR dysfunction	39%	22%	28%	0.11
Type 2 AR dysfunction	48%	35%	26%	0.035
Type 3 AR dysfunction	13%	43%	46%	<0.001
ECC time (min)	111 ± 37	98 ± 38	119 ± 41	0.20
Clamp time (min)	91 ± 32	78 ± 32	92 ± 35	0.11

AR = aortic regurgitation; ECC = extracorporeal circulation time.

Echocardiographic measurements. As shown in Table 3, pre-operative measurements of the aortic root were similar among patients with and without AR at follow-up. After cardiopulmonary bypass, the dimensions of the aortic annulus, the sinuses of Valsalva, and the sino-tubular junction were all significantly larger in patients with ≥3+ AR at follow-up than in the other 2 groups. The length of coaptation and the level of coaptation relative to the annulus decreased gradually throughout the 3 groups (from patients without AR to those with ≥3+ AR). Patients exhibiting any degree of AR at follow-up were also more likely to exhibit residual AR immediately upon weaning from bypass than patients with no or trivial AR at follow-up. As a consequence, the width of their AR jet was larger than that of patients without residual AR. The AR jet was also more frequently eccentric in patients with ≥3+ AR than in the other groups.

Univariate and multivariate predictors of late repair failure. With univariate analysis, pre-operative type 3 dysfunction, Marfan syndrome, the coaptation length, the degree of cusp billowing, the level of coaptation (relative to the annulus), the diameter of the aortic annulus and the sino-tubular junction, the presence of a residual AR, and the width of its vena contracta were associated with the presence of AR at follow-up. All those parameters (except Marfan syndrome, which was uncommon in our population) were submitted to a Cox proportional hazards survival model. With multivariate analysis, the presence of residual AR, a shorter coaptation length, a low level of coaptation (below the level of the aortic annulus), and a larger aortic annulus were found to independently predict ≥3+ AR at follow-up (Table 4). Using receiver-operator characteristic analysis, a coaptation length <4 mm was found to predict recurrent AR with a sensitivity of 85% and a specificity of 89%.

Intraoperative TEE strategy to recognize patients at risk for repair failure. The 3 most powerful predictors of the presence of ≥3+ AR at follow-up were used to construct a stepwise clinical algorithm based on the immediate post-operative TEE data, which could be used intraoperatively to predict the risk of subsequent recurrent AR (Fig. 4). The first step in this algorithm was to examine the level of coaptation relative to the aortic annulus. Whenever the

level of coaptation was below the aortic annulus, the risk of ≥3+ AR at follow-up was high (71%). In patients whose level of coaptation was above the aortic annulus, the second step consisted in evaluating the presence or absence of residual AR. In the absence of residual AR, the risk of ≥3+ AR at follow-up was low (2%). In patients whose level of coaptation was above the aortic annulus and who exhibited any degree residual AR intraoperatively, the third and last step consisted in measuring the length of coaptation. When the coaptation length was ≥4 mm, the risk of having ≥3+ AR at follow-up was low (5%). By contrast, when the coaptation length was <4 mm, the risk of ≥3+ AR increased to 47%. Figure 5 shows the 4-year freedom from ≥3+ AR in these 4 groups of patients.

Table 2. Clinical and Surgical Characteristics of the Patients With ≥3+ Recurrent AR

	Type 1 (n = 11)	Type 2 (n = 9)	Type 3 (n = 21)
Etiology/mechanisms of index AR			
Marfan syndrome	3/11	1/9	—
Aortic dissection	4/11	—	—
Isolated right coronary cusp prolapse	—	7/9	—
Right and noncoronary cusp prolapse	—	1/9	—
Isolated left coronary cusp prolapse	—	1/9	—
Extensive cusp calcifications	—	—	8/21
Infective endocarditis	—	—	7/21
Rheumatic valve disease	—	—	6/21
Root procedures at index surgery			
Root reimplantation	8/11	4/9	4/21
Root remodeling	2/11	2/9	2/21
Supracoronary replacement	1/11	1/9	5/21
Cuspal procedures at index surgery			
Plication	1/11	4/9	4/21
Resection	—	1/9	11/21
Shaving/decalcification	1/11	4/9	17/21
Free edge reinforcement	1/11	7/9	6/21
Patch extension	—	—	8/21
Subcommisural annuloplasty	1/11	5/9	11/21
Mechanisms of recurrent AR			
Type 1 dysfunction	2/11	—	—
Type 2 dysfunction	9/11	8/9	9/21
Type 3 dysfunction	—	—	10/21
Suture dehiscence	—	1/9	2/21
Procedures at reoperation (if applicable)			
Aortic valve replacement	5/11	1/9	5/21
Re-repair	2/11	—	3/21
Ross procedure	1/11	2/9	4/21

Abbreviations as in Table 1.

DISCUSSION

Although the benefits of aortic valve repair over valve replacement have been documented, the potential for technical problems with this surgery is substantial. Accordingly, the long-term durability of these procedures, as reported in the literature, has varied (7,8). Unfortunately, few data are available on the mechanisms of failure of aortic repair procedures. Besides the experience of the surgeon itself, other factors, such as the etiology and mechanisms of aortic valve dysfunction, likely play an important role. In the present study, we therefore sought to examine the causes of recurrent severe AR after an aortic valve repair and to evaluate the ability of intraoperative TEE to identify patients at increased risk for late AR repair failure.

We found that among patients undergoing AR repair, those with Marfan syndrome or type 3 dysfunction are more likely to exhibit ≥3+ recurrent AR. In patients with ≥3+ recurrent AR, the primary cause of failure was the persistence or the induction of some degree of cuspal prolapse at the time of the initial operation. Also, immediate post-repair intraoperative TEE allows the identification of certain functional and morphological features that are associated with late repair failure, including the persistence of any residual AR (particularly if eccentric), a short coaptation length, a coaptation level occurring below the level of the aortic annulus, and an enlarged aortic annulus.

Residual cuspal prolapse as a cause of recurrent AR after aortic valve repair. Cusp prolapse is a frequent cause of AR (4,6,10,16). It can either exist as an isolated pathology of the aortic valve or can coexist with root dilation. During valve sparing surgery, cusp prolapse also can be induced by reducing the root dimensions, particularly at the level of the sino-tubular junction (17,18). Although initially valves presenting with residual or surgically induced cusp prolapses can function with acceptable degrees of AR, severe AR frequently ensues within the first years after repair. In a recent series of 274 patients with aortic root dilation who were undergoing root remodeling at the University Hospital of Saarland, Aicher et al. (19) observed that the likelihood of developing recurrent grade 2 AR or greater during follow-up progressively decreased from 1995 to 2006 as the proportion of patients undergoing combined root remodeling and prolapse correction increased. Although in this series, only 9 patients needed a reoperation for recurrent severe AR, the reason for reoperation was mostly related to cusp prolapse.

The results of the present study confirm and extend these previous observations. In our series, we also

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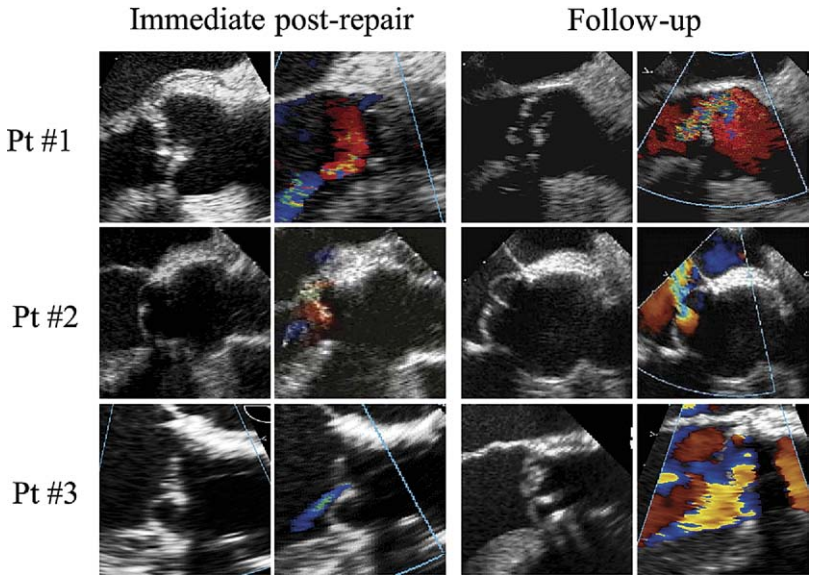

Figure 3. Case Failure Examples

Representative examples of immediate post-repair intraoperative and late post-repair TEE in 3 patients with ≥3+ recurrent AR. The post-repair intraoperative TEE illustrates poor, low coaptation level, and eccentric residual AR jets in all 3 patients. In all 3 cases, the follow-up echocardiography as well as the surgical inspection identified cusp prolapse as the cause of AR recurrence. Abbreviations as in Figure 1.

observed that the number one cause of recurrent severe AR after an initially successful AR repair was related to residual or newly appearing cusp prolapses. Our data show that, in most cases, these prolapses were already present at the end of the initial operation and were readily identifiable by the presence of a residual AR jet, a short length of coaptation, and a "low" level of coaptation relative to the aortic annulus. Interestingly, our results are also in agreement with those recently reported by Pethig et al. (17). In 75

Table 3. Pre-Operative and Intraoperative TEE Measurements of the Study Population

	No/Trivial AR (n = 122)	1+ to 2+ AR (n = 23)	≥3+ AR (n = 41)	p Value (F or Chi-Square)
Pre-operative (mm)				
Aortic annulus	25.4 ± 4.1	23.7 ± 3.5	25.8 ± 5.9	0.27
Sinus of Valsalva	39.4 ± 7.6	39.0 ± 8.6	41.0 ± 13.4	0.61
Sino-tubular junction	34.8 ± 8.9	34.7 ± 8.6	34.1 ± 8.9	0.93
Ascending aorta	41.6 ± 11.4	39.5 ± 8.2	37.2 ± 12.6	0.14
Height of the sinus	25.3 ± 7.5	25.4 ± 5.8	27.3 ± 11.5	0.64
Symmetry of coaptation	1.9 ± 2.2	2.3 ± 1.9	2.2 ± 2.3	0.23
Post-operative				
Aortic annulus (mm)	21.4 ± 3.8	21.0 ± 3.5	25.7 ± 4.4	<0.001
Sinus of Valsalva (mm)	29.1 ± 5.3	29.6 ± 5.0	31.4 ± 5.4	0.04
Sino-tubular junction (mm)	25.6 ± 4.1	23.9 ± 3.7	27.2 ± 3.8	<0.01
Ascending aorta (mm)	27.4 ± 5.1	27.7 ± 5.2	28.4 ± 4.6	0.47
Coaptation length (mm)	6.6 ± 2.8	3.2 ± 1.4	2.2 ± 1.6	<0.001
Coaptation length <4 mm (%)	11	52	85	<0.001
Cusp to annulus distance (mm)	−1.2 ± 2.8	−1.5 ± 3.2	−3.9 ± 4.8	<0.001
Distance from tips to annulus (mm)	6.9 ± 4.3	3.0 ± 3.1	0.6 ± 4.2	<0.001
Tips below the aortic annulus (%)	4	13	49	<0.001
Vena contracta width (mm)	0.6 ± 1.1	2.4 ± 1.7	2.6 ± 1.4	<0.001
Eccentric jet (%)	9	30	73	<0.001

TEE = transesophageal echocardiography; other abbreviations as in Table 1.

Table 4. Intraoperative TEE Predictors of ≥3+ Recurrent AR With the Cox Multivariate Analysis

Multivariate Analysis	HR	95% Confidence Interval	Cox p Value
Coaptation length	0.82	0.63–1.00	0.05
Tips below the level of the aortic annulus	7.9	6.52–9.28	<0.01
Diameter of aortic annulus	1.18	1.03–2.45	0.01
Residual AR	5.3	1.47–6.57	0.01

HR = hazard ratio; other abbreviations as in Tables 1 and 3.

consecutive patients undergoing a David-Feindel root reconstruction, these authors found that the level of coaptation relative to the insertion of the graft was the only predictor of recurrent severe AR.

The mere presence of residual AR in the operative theater has also been identified as a risk factor for post-operative repair failure (20). Our results also confirm and extend these observations by demonstrating that the eccentric character of the jet or its association with a short coaptation length (<4 mm) increases the likelihood of severe recurrent AR during follow-up. Early detection of these unfavorable characteristics by immediate post-repair intraoperative TEE should probably prompt an attempt to immediate re-repair of the valve to avoid post-operative AR recurrence.

Type 3 dysfunction as a risk factor for recurrent AR after aortic valve repair. Besides cuspal prolapses, repair of a type 3 dysfunction was the second most common cause of late repair failure, confirming earlier results from our group (10). Because of the frequent need to excise large areas of diseased tissues (in cases of infective endocarditis or extensive calcifications) in type 3 dysfunction, the surgeon is often left with insufficient good-quality tissue to restore a normal valve function. The resulting defect must then be closed either by direct suturing or interposition of a biological patch. Both of these approaches have potential drawbacks that can secondarily cause recurrent AR. Attempts to direct suturing when lacking tissue can indeed result in excessive shortening of the leaflet and restriction of its movement, causing either aortic stenosis or recurrent AR. However, excess tension on the suture lines, particularly at the level of the biological patches, can lead to disruption and dehiscence.

It is also interesting to note that in approximately one-half of patients with initial type 3 dysfunction who had recurrent AR during follow-up, the mechanisms of recurrent AR were related to recurrent cusp restriction or dehiscence of the biological patches. Our results are in line with those previously reported in similar patients. In 46 patients with rheumatic aortic valve disease treated by pericardial cusp extension, Bozbuga et al. (21) reported a 10-year incidence of recurrent AR of 24%. Similarly, in 61 patients with rheumatic aortic valves in whom an AR repair was attempted, Talwar et al. (22) reported a 10-year freedom from recurrent AR or aortic stenosis of only 52%. This finding highlights the difficulty of maintaining durable results in patients with type 3 dysfunction, particularly when the underlying disease process is rheumatic in origin.

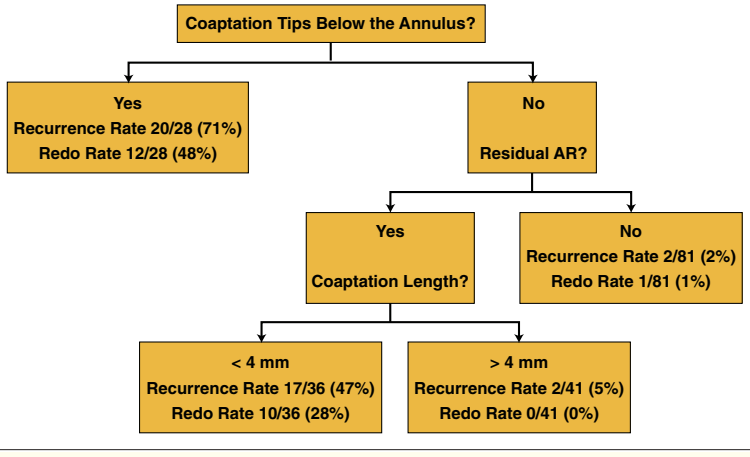

Figure 4. Risk of Repair Failure According to TEE

This chart represents the hierarchical risk of aortic valve repair failure according to 3 powerful predictors identified with the multivariate analysis. When the level of coaptation of the aortic valve relative to the aortic annulus is successfully evaluated, the presence or absence of residual AR and the length of coaptation enables one to identify patients at risk for repair failure. Abbreviations as in Figure 1.

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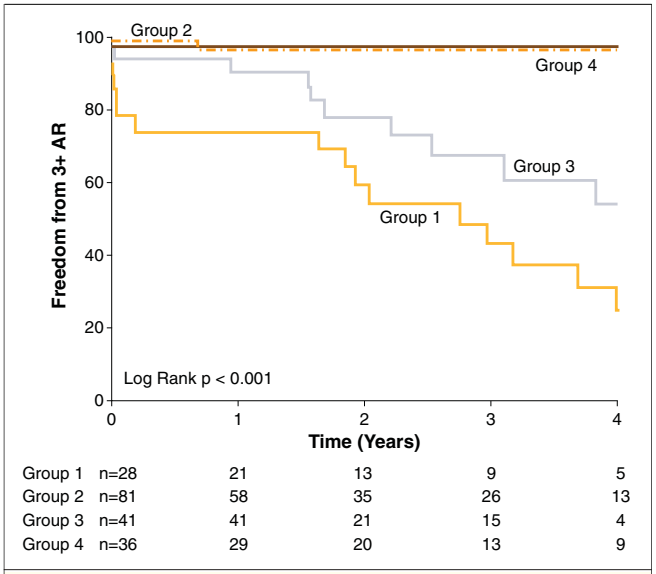

Figure 5. 4-Year Survival Free From Redo According to TEE

Kaplan-Meier estimates of 4-year survival free from recurrence of ≥3+ AR in patients whose coaptation was below the level of the aortic annulus (group 1), in those whose coaptation was above the aortic annulus and either had no residual AR (group 2) or displayed residual AR with (group 3) or without (group 4) a coaptation length <4 mm. Abbreviations as in Figure 1.

Study implications. Our study shows that it is possible to predict intraoperatively which repaired valves could become incompetent over time, which should help the surgeons in deciding whether or not to return on cardiopulmonary bypass for further valve surgery (i.e., second pump runs). Our observations may also influence future strategies in aortic valve repair. We have indeed identified subsets of patients with type 3 dysfunction in whom further refinements of the operative techniques are needed or in whom valve replacement may be a better alternative because of the poor quality of aortic cusp tissue.

Study limitations. This study has several limitations that should be acknowledged. First, intraoperative and follow-up echocardiographic data were available for only 186 (76%) of 244 patients who underwent surgery during the study period. Second, we did not evaluate whether the intraoperative recognition of poor surgical results impacted on the decision of the surgeon whether or not to attempt to re-repair the valve. Moreover, the impact of these second run procedures on the ultimate post-operative results was not evaluated. Third, from a strict statistical point of view, the number of observed end points was too low to permit the multivariate analysis without overfitting. Yet, because our aim was not to build a prognostic index but to select relevant clinical predictors of outcome that can be used in daily clinical practice to evaluate the risk of recurrences, we do not feel uncomfortable with this issue.

CONCLUSIONS

Our results demonstrate that intraoperative TEE can be used to identify patients undergoing AR repair who are at increased risk for developing recurrent severe AR. Intraoperative parameters associated with recurrent AR during follow-up include the persistence of a residual AR jet, a coaptation length <4 mm, a coaptation level occurring below the level of the aortic annulus and an enlarged aortic annulus.

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Key Words: aortic valve repair ■ transesophageal echocardiography ■ aortic regurgitation ■ aortic valve surgery ■ aortic prolapse.

▶APPENDIX

For supplementary methods, please see the online version of this article.