Systolic anterior motion of the mitral valve: A 30-year perspective

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Objective: Systolic anterior motion (SAM) can occur after mitral valve repair (MVr), most frequently in patients with degenerative valve disease. Our initial observations (1981-1990) revealed that most patients with SAM can be successfully treated medically. Here the authors review the last 16 years of their experience with SAM after MVr.

Methods: Between January 1996 and October 2011, 1918 patients with degenerative mitral valve disease underwent MVr at our institution. We performed a retrospective analysis of SAM in this patient population.

Results: The incidence of SAM was 4.6% (89 of 1918) overall, 4.0% (77 of 1906) in patients who did not have SAM preoperatively (de novo). Compared with our previously published report, the incidence of SAM decreased from 6.4% to 4.0% ($P = .03$). Hospital mortality was 2.0% (38 of 1918) overall, 1.3% (14 of 1078) for isolated MVr. One patient with de novo SAM (1 of 77; 1.3%) died after emergency MVr. All patients with de novo SAM were successfully managed conservatively with intravenous fluids, α agonists, and/or β blockers. A higher incidence of SAM was associated with a left ventricular ejection fraction greater than 60% ($P = .01$), posterior leaflet resection ($P = .048$), and hypertrophic obstructive cardiomyopathy ($P < .01$). The incidence of SAM was lower in patients who underwent device mitral annuloplasty with a semirigid posterior band compared with a complete ring ($P = .03$).

Conclusions: In the more recent era, SAM occurs one-third less frequently after repair of degenerative mitral valve disease. Use of an incomplete annuloplasty band rather than a complete ring is associated with a lower incidence of SAM. The mainstay treatment of SAM continues to be medical management. (J Thorac Cardiovasc Surg 2014;148:2787-94)

Systolic anterior motion (SAM) of the mitral valve refers to the paradoxic movement of the anterior leaflet and/or chordae toward the interventricular septum during systole. Initially associated mainly with hypertrophic obstructive cardiomyopathy (HOCM), SAM was first described as a potential complication of mitral valve repair (MVr) in 1977.[1] This led to a significant amount of interest in the phenomenon, including a study of the incidence of SAM in our early MVr series in 1984,[2] and threatened to compromise the concept of MVr. Our observation that SAM occurred most frequently in patients with a large saillike anterior leaflet and excessive height of the posterior leaflet after extensive posterior leaflet resection led us to focus on efforts to lower the height of the repaired posterior leaflet with the attendant posterior relocation of the coaptation line. In addition, numerous other studies have been conducted to help understand the pathophysiology of SAM, and have led to the identification of major preoperative risk factors and adaptation of surgical repair techniques, including the frequently used techniques of sliding plasty or folding plasty repair for patients with the anatomic substrate for SAM requiring posterior leaflet resection. However, despite this knowledge and experience, the current incidence of SAM after MVr in patients with degenerative disease remains in the range of 6.1% to 11.0% in recent large studies.[3-6]

Previously, our group reported a 10-year single-institution experience (1981-1990) with SAM after MVr using mainly complete ring annuloplasty.[7] We now present our last 16 years of data (1996-2011) which incorporates changes in repair techniques, completing a 30-year retrospective on the incidence and management of SAM after MVr.

METHODS

The NYU Langone Medical Center Institutional Review Board granted a waiver of individual informed consent for analysis of de-identified data. Between January 1996 and October 2011, 2687 patients underwent MVr at our institution. Of these, 1918 had degenerative disease and were included in this study. MVr was performed via median sternotomy (n = 473), right anterior thoracotomy (n = 1439), or left thoracotomy (n = 6). All procedures were performed on cardiopulmonary bypass. Mitral valve

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Abbreviations and Acronyms

CABG = coronary artery bypass graft
HOCM = hypertrophic obstructive cardiomyopathy
LVEF = left ventricular ejection fraction
LVOT = left ventricular outflow tract
SAM = systolic anterior motion
TEE = transesophageal echocardiogram

repair techniques were chosen according to the pathology of the individual patient. These techniques were for the most part performed according to Carpentier's principles (Table 2). Exceptions included the introduction of the posterior leaflet folding plasty (n = 705) before the start of this study period as an alternative to the classic sliding plasty technique aimed at reducing posterior leaflet height. Regarding annuloplasty methods, we transitioned from the use of complete rings (n = 409; Carpentier-Edwards classic annuloplasty ring or physio annuloplasty ring; Edwards LifeSciences, Irvine, Calif) to use of semirigid posterior bands (n = 1172; CG Future Band; Medtronic Inc, Minneapolis, Minn) in 2001. We have avoided the use of complete ring annuloplasty since then, except for reduction annuloplasty for functional mitral regurgitation. Patients who underwent MVr with infrequently used annuloplasty devices (Seguin Semi-Rigid Ring, St Jude Medical, St Paul, Minn, n = 46; CG Future Ring, Medtronic Inc, Minneapolis, Minn, n = 78) or without an annuloplasty device specified (n = 214) were not included in the subgroup analysis comparing device types. Annuloplasty device sizes were determined based on the anterior leaflet height and the intertrigonal distance.

Each patient had intraoperative transesophageal echocardiograms (TEE) before and after cardiopulmonary bypass, and a transthoracic echocardiogram before discharge. All intraoperative TEE studies were performed by an echo certified cardiac anesthesiologist and reviewed by a dedicated operating room cardiologist. The diagnosis of SAM was made if present on any of these echocardiograms. SAM was defined as any chordal or mitral leaflet protrusion into the left ventricular outflow tract (LVOT) during systole regardless of the presence of hemodynamic effect. Initial management of postoperative SAM involved a combination of ventricular volume loading, vasoconstriction, elimination of inotropes, and/or β blockade. SAM that was managed medically intraoperatively and resolved was included in the study. Patients who continued to exhibit signs of SAM with moderate mitral regurgitation or LVOT obstruction despite pharmacologic manipulations underwent reoperation for either valve repair or replacement. Neither these practices nor the criteria for the diagnosis of SAM changed during the study period and did not differ from our previous study.

All data were collected prospectively in a database and a retrospective review of this database was performed for this study.[8] Individual echocardiographic images were not reviewed again for this study. Statistical analyses were performed using SPSS 20 (IBM Corp, Armonk, NY). Variables were considered complete if the data were available for more than 97% of the patients included in the study. When data were incomplete for a given variable, separate subgroup analyses were performed using only those patients for whom the data were available. Categorical variables are presented as incidence (percentage), continuous variables as means ± standard deviation. *P* values for univariable analyses were determined by χ^2 or Fisher exact test when appropriate. Multivariable regressions were performed using variables identified by a *P* value of 0.1 or less in the univariable analysis. Regressions were performed on subsets of patients with complete data for the variables of interest. Odds ratios (ORs) of multivariable predictors were determined by stepwise logistic regression.

RESULTS

The mean age of the patients was 62 ± 15 years with a mean New York Heart Association functional class (I-IV) of 2.3 ± 0.6 and a mean left ventricular ejection fraction (LVEF) of 56% ± 17%. Concomitant procedures were performed in 43.8% (840 of 1918), including 182 (9.5%) coronary artery bypass graft procedures and 282 (14.7%) other valve procedures. Baseline characteristics are summarized in Table 1.

In all patients, including those with preoperative SAM, the incidence of postoperative SAM was 4.6% (89 of 1918). Excluding patients with preoperative SAM (n = 12), the incidence of de novo SAM was 4.0% (77 of 1906). No patient with de novo SAM (0 of 77) required reoperation during this time period. One patient who presented with HOCM and SAM underwent reoperative mitral valve replacement 4 days after MVr because of persistent SAM with associated mitral regurgitation and moderate LVOT obstruction (1 of 89; 1.1%). Hospital mortality was 2.0% (38 of 1918) overall and 1.3% (14 of 1078) for isolated MVr. One patient with de novo SAM (1 of 77; 1.3%) died of multisystem organ failure after a prolonged hospital course. This patient had been admitted in cardiogenic shock requiring multiple inotropes and vasopressors before undergoing emergency MVr with posterior leaflet resection and suture reduction annuloplasty.

Univariable analysis (Table 2) showed a higher incidence of SAM in patients with preoperative LVEF greater than 60% ($P = .01$), posterior leaflet resection ($P = .048$), flail posterior leaflet ($P = .04$), severe mitral regurgitation ($P = .03$), and HOCM ($P < .01$). There was a lower incidence of SAM in patients who had a concomitant valve procedure ($P = .02$) and in patients who underwent mitral annuloplasty with a semirigid posterior band ($P = .03$). The incidence of SAM also decreased over time ($P < .01$) during the study period.

Multivariable analyses (Table 3) of preoperative risk factors for SAM identified by univariable analysis revealed that LVEF greater than 60% was associated with an increased risk of post-MVr SAM (OR, 2.7; $P = .04$). Specific preoperative echocardiographic anatomic information was only available in 730 of the patients in the study. Regression analysis of this subgroup demonstrated significant association between HOCM (OR, 14.2; $P = .03$) and flail posterior leaflet (OR, 2.4; $P = .046$) with the development of SAM. In patients who received either a complete ring annuloplasty device or a partial band annuloplasty device as part of their MVr and underwent posterior leaflet resection (n = 1098), multivariable analysis revealed complete ring annuloplasty (OR, 1.9; $P = .02$) to be the only procedural characteristic that was a significant independent risk factor for the development of post-MVr SAM.

TABLE 1. Patient characteristics

Age* (n = 1913)	62 ± 15 y
Male sex	61% (1163 of 1916)
NYHA class (I-IV)* (n = 1907)	2.3 ± 0.6
CHF	34% (654 of 1911)
Diabetes	8.1% (154 of 1911)
Hypertension	55% (939 of 1714)
Angina	22% (417 of 1908)
History of myocardial infarction	6.4% (123 of 1907)
Mitral regurgitation severity (0-4+)* (n = 1473)	2.8 ± 0.6
Mean preoperative LVEF* (n = 981)	56 ± 17%
Mean preoperative LVEDD* (n = 880)	5.4 ± 0.7 cm
HOCM	1.0% (10 of 1000)
Reoperation	8.7% (166 of 1918)
Incision	
Median sternotomy	24% (455 of 1900)
Right thoracotomy	76% (1439 of 1900)
Left thoracotomy	0.3% (6 of 1900)
Isolated MVr	56% (1078 of 1918)
Concomitant CABG	9.5% (182 of 1918)
Concomitant valve procedure	15% (282 of 1918)
Anterior leaflet prolapse	1.4% (26 of 1918)
Posterior leaflet prolapse	5.2% (100 of 1918)
Flail anterior leaflet	3.9% (38 of 983)
Flail posterior leaflet	45% (447 of 984)

NYHA, New York Heart Association; *CHF*, congestive heart failure; *LVEF*, left ventricular ejection fraction; *LVEDD*, left ventricular end-diastolic dimension; *HOCM*, hypertrophic obstructive cardiomyopathy; *MVr*, mitral valve repair; *CABG*, coronary artery bypass graft. *Mean ± standard deviation.

DISCUSSION

In the current report on 1918 patients with degenerative mitral valve disease, the incidence of de novo post-MVr SAM was 4.0%. Preoperative risk factors for SAM included LVEF greater than 60%, HOCM, and flail posterior leaflet pathology. Analysis of intraoperative factors identified the use of semirigid posterior band annuloplasty as a negative predictor of SAM; it was associated with half the risk of SAM compared with complete ring annuloplasty. Although all patients with SAM in this series were managed conservatively, we recognize the occasional need for reoperation in patients in which the post-MVr SAM is severe and persistent.

In our previous 10-year single-institution experience (1981-1990), which looked at postoperative SAM in 439 patients who underwent MVr according to Carpentier principles with rigid annuloplasty rings,[7] the incidence of postoperative SAM over that decade was 6.4%. In comparison, the incidence of SAM in the current report has decreased to 4.0% (a 38% decrease). This seems to be the result of better understanding of the pathophysiology of post-MVr SAM and an improvement in the techniques for its prevention.

The Relation Between Mitral Valve Repair Techniques and the Incidence of SAM

General understanding about the pathophysiology of SAM began with the study by Mihaileanu and colleagues,[9] which highlighted the geometric changes to the base of the left ventricle that occur after rigid ring annuloplasty, specifically the narrowing of the aortomitral angle and its consequences on left ventricular flow patterns. Normally, a flat aortomitral angle maintains the left ventricular inflow and outflow in parallel but opposite directions. Severe narrowing of the aortomitral angle (more acute) produces an overlap of the 2 distinct functional components, creating the potential for SAM. Another important observation was that the anterior leaflet of the mitral valve normally closes most of the mitral orifice in systole. In patients with a narrow aortomitral angle, the systolic pattern is inverted and the posterior mitral leaflet assumes most of mitral orifice closure. This led to the recommendation for less aggressive annuloplasty size reduction with sizing based on the amount of anterior leaflet tissue, and the adoption of the sliding plasty or folding plasty techniques for posterior leaflet resection to decrease the height of the posterior leaflet.[10] These have become cardinal rules when it comes to avoiding postoperative SAM in patients with degenerative mitral valve diseases, and multiple studies have since validated these principles using different approaches.[11-13] In addition, different operative techniques such as sliding plasty, folding plasty, and others have been proposed to reduce the height of the posterior leaflet,[14-18] and many different annuloplasty devices have been developed,[19-24] each with its own potential advantages. Between our previous study and the current study, we adopted the use of posterior leaflet folding and sliding plasty techniques to reduce the height of the posterior leaflet, especially when patients with at-risk pathologies were identified. We believe that this may have partly contributed to the 38% reduction in the incidence of SAM between the 2 studies.

In the present study, we found the use of semirigid posterior band annuloplasty to be significantly associated with a lower risk of postoperative SAM compared with annuloplasty performed with a complete annuloplasty ring. The concept of semirigid posterior band annuloplasty comes from an observation that was made in one of our early studies.[25] Normally, during systole, the base of the anterior leaflet of the mitral valve moves posteriorly, increasing the size of the LVOT. The insertion of a complete rigid ring eliminates this movement, thereby causing a narrower systolic LVOT diameter. The base of the anterior leaflet is attached to the subaortic curtain (also called aortomitral continuity), a fibrous structure running between the 2 trigones and dividing the base of the left ventricle into the aortic and the mitral orifices. Thus, the aortic root and mitral orifice dynamics are closely related. During systole, the subaortic curtain moves posteriorly, away from the ventricular septum toward the posterior wall, increasing the outflow tract dimension to facilitate ventricular emptying. This posterior shift also decreases the anteroposterior

TABLE 2. Incidence of perioperative factors in patients who developed de novo SAM and those who did not

	No SAM	SAM	*P* value
Age >70 y	38% (669 of 1779)	27% (21 of 77)	.07
Sex (male)	61% (1080 of 1778)	55% (42 of 76)	.3
NYHA class III-IV	31% (549 of 1779)	25% (19 of 77)	.2
CHF	34% (604 of 1772)	25% (19 of 77)	.09
Diabetes	8.0% (142 of 1774)	3.9% (3 of 77)	.2
Hypertension	49% (868 of 1775)	43% (33 of 77)	.3
Angina	22% (394 of 1779)	13% (10 of 77)	.05
History of MI	6.6% (117 of 1651)	1.3% (1 of 77)	.06
LVEF ≥60%	78% (1027 of 1312)	92% (57 of 62)	.01
Severe MR	83% (1473 of 1779)	92% (71 of 77)	.03
HOCM	0.8% (7 of 927)	6.7% (2 of 30)	<.01
LVEDD ≤5.5 cm	62% (510 of 823)	63% (17 of 27)	.2
Anterior leaflet prolapse	1.5% (26 of 1779)	0% (0 of 77)	.3
Posterior leaflet prolapse	5.3% (94 of 1779)	3.9% (3 of 77)	.6
Flail anterior leaflet	3.8% (35 of 912)	10% (3 of 30)	.09
Flail posterior leaflet	45% (409 of 914)	63% (19 of 30)	.04
Date of operation*	April 5, 2004 ± 4.0 y	March 20, 2003 ± 3.1 y	<.01
Isolated MVr	44% (776 of 1779)	35% (27 of 77)	.1
Concomitant CABG	10% (174 of 1779)	3.9% (3 of 77)	.09
Concomitant valve procedure	15% (258 of 1779)	5.2% (4 of 77)	.02
Reoperation	8.7% (155 of 1779)	3.9% (3 of 77)	.1
Median sternotomy	24% (418 of 1779)	16% (12 of 77)	.1
Posterior band annuloplasty†	75% (1093 of 1465)	63% (42 of 67)	.03
Annuloplasty device size*	30.5 ± 3.6 mm	31.6 ± 3.7 mm	.05
Any anterior leaflet procedure	27% (486 of 1779)	30% (23 of 77)	.6
Anterior leaflet resection	21% (373 of 1779)	26% (20 of 77)	.3
Posterior leaflet resection	67% (1195 of 1779)	78% (60 of 77)	.048
Posterior leaflet height reduction procedure‡	39% (689 of 1779)	57% (44 of 77)	<.01
Posterior annular plication	4.7% (83 of 1779)	5.2% (4 of 77)	.8
Papillary or chordal procedure	11% (197 of 1779)	5.2% (4 of 77)	.1
Hospital mortality	1.9% (34 of 1779)	1.3% (1 of 77)	.7

SAM, Systolic anterior motion; *NYHA*, New York Heart Association; *CHF*, congestive heart failure; *MI*, myocardial infarction; *LVEF*, left ventricular ejection fraction; *MR*, mitral regurgitation; *HOCM*, hypertrophic obstructive cardiomyopathy; *LVEDD*, left ventricular end-diastolic dimension; *MVr*, mitral valve repair; *CABG*, coronary artery bypass graft. *Mean ± standard deviation. †Compared with complete ring annuloplasty. ‡Posterior leaflet folding plasty or posterior papillary muscle sliding plasty.

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dimension of the mitral orifice, enhancing leaflet coaptation.[26]

Semirigid posterior bands are implanted along the posterior aspect of the mitral annulus up to the trigones to provide posterior remodeling.[24] This restores the native annular dimensions and orifice shape necessary for a durable repair while still allowing for the physiologic motion of the subaortic curtain.[27] With posterior band annuloplasty, the anterior saddle horn of the mitral annulus is not restrained in any fashion, allowing folding away from the left ventricle outflow tract in systole in addition to expansion/contraction of the mitral annulus. In a previous report by Sharony and colleagues,[24] the authors demonstrated that the dynamic anterior annular motion allowed by posterior band annuloplasty was associated with lower mean peak diastolic gradients across the mitral valve compared with rigid ring annuloplasty of the same size. Mesana and colleagues[28] recently reported lower transmitral gradients with improved clinical functional status at late follow-up when a band was used compared with complete ring annuloplasty. We believe that this preservation of the anterior annular folding motion is what may contribute to the statistically significant lower incidence of SAM associated with the partial annuloplasty device reported here, and this should be a consideration when choosing an annuloplasty device for mitral repair.

The Preoperative Risk Factors for SAM

Several studies have identified preoperative risk factors for SAM, including the cause of degenerative disease, posterior leaflet height greater than 15 mm, acute aortomitral angle, sigmoid interventricular septum, short coaptation point-septal distance, and anterior displacement of the anterior papillary muscle.[12,13,26,29-31] Manabe and colleagues[5] detailed the influence of preoperative left ventricular function on the development of SAM. In their study, no patients with an impaired or dilated left ventricle developed SAM. Conversely, the incidence of SAM

TABLE 3. Multivariable analyses* of the preoperative and procedural risk factors for the development of post-MVr SAM

	Odds ratio	*P* value
Preoperative factors (n = 1360)		
Angina		
History of MI		
LVEF >60%	2.7	.04
CHF		
Age ≥70 y		
Severe MR	2.5	.08
Preoperative factors with anatomic echo data (n = 730)		
Angina		
History of MI		
LVEF >60%		
CHF		
Age ≥70 y		
HOCM	14.2	.03
Flail anterior leaflet		
Flail posterior leaflet	2.4	.046
Severe MR		
Procedural factors (n = 1098)		
Posterior band annuloplasty†	0.52	.02
Anterior leaflet resection		
Any anterior leaflet procedure		
Papillary or chordal procedure		
Posterior leaflet height reduction procedure‡	0.62	.10

MI, Myocardial infarction; *LVEF*, left ventricular ejection fraction; *CHF*, congestive heart failure; *MR*, mitral regurgitation; *HOCM*, hypertrophic obstructive cardiomyopathy. *Multivariable analyses performed by stepwise logistic regression. Odds ratios and *P* values are not listed if the variable was eliminated before the final step in the model. †Compared with complete ring annuloplasty. ‡Posterior leaflet folding plasty or posterior papillary muscle sliding plasty.

increased proportionally to the LVEF. In our current report, a preoperative LVEF greater than 60% was also found to be an independent predictor for postoperative SAM. This raises some important issues related to patients with normal ventricles in this era of early surgical indications. Caution should be used within the current surgical trend toward resecting less and using smaller annuloplasty devices in the context of normal left ventricular function and dimensions. The relationship between the amount of leaflet tissue and the dimensions of the mitral orifice after annuloplasty remains a critical determinant in the prevention of post-MVr SAM.[10]

The Management of Postoperative SAM

Although the incidence of post-MVr SAM seems to be decreasing as a result of improved preventative measures, the management of SAM continues to be an area of investigation.[3,4,6,7,11] In our 30-year experience with SAM after MVr, we found that of the 2357 patients who underwent valve repair for degenerative mitral regurgitation, 105 (4.4%) developed de novo SAM, and all were successfully managed nonoperatively with a combination of ventricular volume loading, vasoconstriction, and β blockade. However, there are various surgical techniques recommended for the treatment of post-MVr SAM, including edge-to-edge repair,[32,33] posterior leaflet height reduction techniques,[10,15,34] removal or replacement of the annuloplasty device by a larger device,[3-6,11,12,35] and valve replacement. Although we advocate the role of medical therapy in most cases, these adjunctive surgical techniques may be useful when there is failure of conservative management or when the degree of SAM warrants immediate reintervention. Undoubtedly, some patients with persistent postoperative SAM will still require reoperation for valve replacement. Significantly, with increased emphasis on earlier repair in patients with normal ventricular size and function, surgeons still have to be prepared to manage SAM. Optimization of our repair techniques may help us to further minimize this complication; this will require ongoing investigation.

Recent Related Studies

Other studies have noted similar management strategies for post-MVr SAM in patients with degenerative disease patients. Varghese and colleagues[6] reviewed 785 patients undergoing MVr with an incidence of SAM of 6.6%. Surprisingly, 15% required reoperation during the same hospitalization, whereas 80% had resolution of SAM with appropriate therapy by discharge. Crescenzi and colleagues[4] evaluated 608 patients undergoing MVr with SAM in 9.8%; 8% of these patients required reoperation. They noted that the incidence of SAM was lower when preventative techniques such as sliding plasty (6.1%) and edge-to-edge suturing (6.1%) were used compared with isolated quadrangular resection (16.5%). Manabe and colleagues[12] noted SAM in 6.1% after MVr (n = 441), however nearly half of these patients underwent reoperation after unsuccessful conservative management. Their multivariable analysis found that nondepressed left ventricular function and septal hypertrophy were significantly associated with development of SAM, and noted that patients with a small hyperdynamic heart, such as those patients operated on early in their disease process, are at higher risk for developing SAM. Brown and colleagues[3] reported on 1589 patients with degenerative disease with a post-MVr incidence of SAM of 11% on intraoperative TEE, and similar to our series, the investigators stressed conservative management for most patients as only 4 patients required reoperation. The investigators reported the use of posterior band annuloplasty in 97% of patients, with only 1% receiving complete ring annuloplasty, preventing comparison of the 2 techniques. In contrast to our report and others, they did not attempt to identify risk factors for SAM by statistical methods. The investigators stated that they do not commonly use techniques described in the literature to reduce the risk of SAM, including "sliding plasty, chordal transfer, edge-to-edge repair,

anterior leaflet valvuloplasty, and the Pomeroy procedure,'' given the low requirement for reoperation for SAM. This may have contributed to their higher incidence of SAM.

As with our report, these studies have documented the ability to manage most cases of post-MVr SAM with medical management. Our data also suggest that the incidence of SAM can be reduced by using posterior band annuloplasty instead of complete ring annuloplasty.

Limitations

This was a single-institution, nonrandomized, retrospective study with the inherent limitations. The relatively low incidence of SAM may limit the power to identify additional risk factors for the development of SAM. As there were no patients with de novo SAM who required reintervention, we cannot comment specifically on the surgical management of SAM. We shifted from the use of complete annuloplasty rings to the use of posterior bands in 2001 with limited overlap, creating a potentially confounding factor when comparing the 2 interventions. Although our surgeons evaluated the relationship of the LVOT to the mitral annulus before completing each mitral repair, our echocardiographic database did not quantify the aortomitral angle, a known risk factor for SAM. The actual echocardiographic images were not reviewed specifically for this study, and thus it is possible that transient SAM may have been missed, albeit unlikely as we look specifically for SAM in each case. Although echocardiography has evolved over the study period, it is unlikely that a difference in echo reporting has occurred and contributed to the decrease in the incidence of SAM, as the teams reporting and documenting the echo findings have not changed, and a systematic change from the complete to partial annuloplasty device was shown to be associated with a statistically significant lower incidence of SAM. If the difference in incidence of SAM had occurred secondary to a change in echo technique, we would expect the incidence of SAM to have increased given the likely improved detection of SAM secondary to improvement in echo technology and operator experience. Although SAM secondary to HOCM has an arguably different etiology than post-MVr SAM in patients with degenerative valve disease, we believed that the incidence of de novo SAM in patients with HOCM ($n = 1$) would not significantly affect the outcomes of the study and were included in our report as we believe that mitral surgeons should be cognizant of the increased risk of SAM in this population.

CONCLUSIONS

In the more recent era, de novo SAM occurs one-third less frequently after repair of degenerative mitral valve disease. Mitral annuloplasty with a posterior band is associated with a lower incidence of SAM than with a complete ring. The mainstay treatment of SAM continues to be medical management with volume resuscitation, α stimulation, and β blockade, and the need for operative reintervention is rare.

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Discussion

Dr José Luis Pomar *(Barcelona, Spain)*. Dr Yaffee, as you mentioned, this is a single-center, nonrandomized, retrospective study, and, in addition, the target event has a low incidence. Therefore, as you mentioned very well, some limitations are unavoidable. However, I was expecting to learn more from the cases in which the classic rules to prevent SAM were not only addressed but also properly corrected, like height of the P2, larger selection of the device as shown having an impact on degenerative mitral regurgitation, area of the anterior leaflet and so on. Do you have any data showing the relevance of an adequate adherence to the rules? In your large experience, does the size of the ring or the band have any significant impact on the prevalence of SAM? Did the width of the posterior leaflet resection, the technique, or the use of artificial chords, somehow limiting or preventing the excursion of the P2, have any effect?

Finally, and despite statistics, do you not think that experience in the techniques of repair in a high volume load may be as important as the type of device to avoid this unpleasant complication?

Thanks to your group for bringing this interesting topic and to you for your excellent presentation.

Dr Yaffee. Thank you for your excellent comments and questions. I will try and work through them.

Since our previous study, we have tried to adhere to the classic rules of posterior leaflet height reduction, respect for the anterior leaflet size, use of a larger annuloplasty to prevent overreduction of annular size, and we believe these are some of the main reasons for our decrease in the incidence of SAM since our previous study. We also believe the other main factor is the introduction of posterior band annuloplasty, which partially allows compensation for the risks you mentioned as well as dynamic anterior annular folding.

As far as specific data concerning posterior leaflet height or anterior leaflet area, we routinely use intraoperative transesophageal echo to evaluate these parameters before each repair, however, we do not routinely record these data. So I cannot comment on that specifically for the data in this study.

You mentioned ring size. As touched on, we generally use a larger ring size than we used to because the identification of overreduction of annular size is a risk factor for SAM, and we believe, as I mentioned, this contributed to the reduction in the incidence of SAM from our previous data. In this study, the average size of the band in patients who did not have SAM was 30.5 mm and in patients who did have SAM it was 31.6 mm. This was not statistically significant; however, the slightly larger size of the band in patients with SAM seems a little counterintuitive. We believe this is because we correctly identified the preoperative risk factors for SAM in those patients and compensated by using a slightly larger band size and believe that that is what negated the difference in the risk.

You mentioned size or width of the posterior leaflet resection. Unfortunately, I do not have that data from our database; we do not routinely record that number, so I cannot comment specifically on that.

You mentioned artificial chordae and respect versus resect. We looked at patients who had chordal procedures, either artificial chordae or chordal reimplantation, as well as papillary muscle reimplantation, reduction sliding plasty, and found that there was no difference in the incidence of SAM between patients who underwent those procedures and those who did not.

And then I believe you mentioned surgeon experience and surgical volume. We do believe that they are very important as far as mitral valve repair is concerned. The surgeon must be able to identify the risk factors that you mentioned in order to determine different procedures such as folding plasty or sliding plasty and the need for reduction of the height of the posterior leaflet as well as determine the appropriate annular band size; that is very important in the prevention of SAM.

Dr Pomar. Thank you very much.

Dr Vinay Badhwar *(Pittsburgh, Pa)*. Thank you for your excellent presentation. I wanted to key in on the last comment you just made in your response to Dr Pomar's question on surgeon experience. In your multivariable analysis, I noticed that one thing that was perhaps missing was era of surgery. Before 1999 and Levine's seminal paper on the echocardiographic predictors of SAM, the surgical community had challenges with markers to prevent SAM. After that era, surgeons have become better at preventing and treating SAM, which may be relevant to acknowledge in your manuscript. What are your thoughts on the

impact of era and the surgeon's ability to better navigate SAM predictors after 2000 versus just the band itself?

Dr Yaffee. I think it is a combination of factors. We divided the study into the first half of the study and the second half was I believe in 2004 when we looked, and there was a difference on univariable analysis, as I mentioned, but it fell out of multivariable analysis when we compared it with the other factors discussed. So we did look at that, and we did not see a difference on multivariable analysis. As Dr Pomar mentioned, this was a retrospective nonrandomized study, and we used statistical methods to eliminate those confounding factors as best we could, but we did not find a difference.

Dr A. Pieter Kappetein *(Rotterdam, The Netherlands)*. In addition to this question, was the posterior band used in a different time period compared with the complete ring?

Dr Yaffee. We switched from a complete ring to a posterior band in August 2001. There was not a lot of overlap between those 2 devices.

Dr Kappetein. So that might be a confounding factor?

Dr Yaffee. Right.

Dr Kappetein. If you do not put date of operation in your multivariate analysis, you might be able to identify it as a confounding factor.

Dr Yaffee. We did, and we took into account the date of the operation in the multivariable analysis, and that fell out, but the band did not.

Dr Kappetein. Over time, you gained more experience and therefore that might be the reason why with the band worked better than the complete ring.

Dr Ralph J. Damiano *(St Louis, Mo)*. Congratulations on a great presentation and an excellent series. I was surprised that you included patients with idiopathic hypertrophic subaortic stenosis (IHSS) or hypertrophic obstructive cardiomyopathy, because clearly they have SAM from a different cause. Usually to correct this problem, you need to do a septal myectomy. I wonder if you would comment on that subgroup of patients. Was the IHSS or HOCM defined preoperatively, how did you alter your repair, and how do you usually perform a myectomy? Particularly, I know that you favor the right thoracotomy approach for mitral valve repair? Do you do a transmitral myectomy? What is your approach to these difficult patients?

Dr Yaffee. The subgroup analysis was performed on patients for whom we had that available data. Our database does not always include that specific anatomic information depending on where the patient had their preoperative echo performed. Among the patients for whom we did have that data available, the incidence of IHSS was about 1%. I did not look specifically at the differences, whether these patients had septal myectomies or not. So I do not know if that contributed to the data. Our prevalence of IHSS was relatively low, so we believe it did not affect the overall incidence of SAM, but it was significant on the multivariable analysis, as it obviously would be.