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ORIGINAL ARTICLE

Predicting systolic anterior motion after mitral valve reconstruction: using intraoperative transoesophageal echocardiography to identify those at greatest risk†

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Abstract

OBJECTIVES: We set out to determine if intraoperative pre-bypass transoesophageal echocardiography could assist in predicting which patients are at greatest risk for systolic anterior motion (SAM) after mitral valve repair (MVR).

METHODS: Three hundred and seventy-five consecutive patients who underwent reconstructive MVR surgery for degenerative disease were included. Data were collected using intraoperative echocardiographic images taken prior to the initiation of cardiopulmonary bypass. Based on the physiology of SAM, we postulated that 11 parameters could be potential risk factors for SAM: left ventricular ejection fraction (LVEF), left ventricular end-systolic dimension, left ventricular end-diastolic dimension (LVEDD), basal septal diameter (basal-interventricular septal diameter in diastole (IVDd)), mid-ventricular septal diameter (mid-IVDd), coaptation–septal distance (c-sept), anterior leaflet height, posterior leaflet height, aorto-mitral angle, mitral annular diameter and left atrial diameter. These parameters were measured and recorded by a blinded single operator. Independent predictors of SAM were identified using multiple logistic regression analysis.

RESULTS: Of the 375 patients, 345 (92%) did not develop SAM (No-SAM group), while 30 (8%) developed intraoperative or postoperative SAM (SAM group). The mean age was 56.8 ± 12.8 and 56.7 ± 13.8 in the No-SAM and SAM groups, respectively. The incidence of fibroelastic deficiency, forme fruste and Barlow's disease was similar in both groups. All patients received a complete annuloplasty ring as part of the repair. There was no statistical difference in the mean ring size used in each group. EF was similar in the No-SAM (56.2% ± 8.1) and SAM (57.0% ± 9.2) $P = 0.63$) groups. Independent predictors of developing SAM after valve repair were: EDD <45 mm [odds ratio (OR) 3.90; $P = 0.028$], aorto-mitral angle <120° (OR 2.74; $P = 0.041$), coaptation-septum distance <25 mm (OR 5.09; $P = 0.003$), posterior leaflet height >15 mm (OR 3.80; $P = 0.012$) and basal septal diameter ≥15 mm (OR 3.63; $P = 0.039$).

CONCLUSIONS: The risk for SAM can be predicted using intraoperative transoesophageal echocardiography. The combination of a smaller left ventricle, tall posterior leaflet, narrow aorto-mitral angle and enlarged basal septum significantly increases the risk for SAM. Knowing these parameters prior to valve repair can assist the surgeon in adjusting their repair technique to minimize the risk.

Keywords: Mitral valve repair • Systolic anterior motion

INTRODUCTION

Systolic anterior motion (SAM) is reported to occur in 4–10% of cases after mitral valve repair (MVR) [1–3]. A number of surgical techniques have been suggested to decrease the incidence of SAM after MVR [3, 4]. Knowing which patients are at an increased risk for developing SAM at the time of repair may provide the surgeon with guidance as to the specific repair strategy they should employ. However, those factors that predispose patients to SAM have only been studied in limited detail. Small series and case reports have suggested some predictive factors, such as anterior/posterior leaflet ratios and coaptation point to septal distance as predictive of SAM [5, 6]. However, because of limited sample size, mixed aetiologies and non-consecutive patient inclusion, published studies generally do not provide a robust and objective analysis of predictors for SAM. Hence, the aim of this study was to assess the role of the mitral valve (MV) apparatus, ventricular dimensions and ventricular function in predicting SAM in patients undergoing MVR for degenerative disease.

MATERIALS AND METHODS

Patients and surgery

Between July 2008 and June 2011, 375 consecutive patients with degenerative MV regurgitation underwent MVR. All patients were included in the analysis. Patients who underwent concomitant

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aortic valve replacement or had a preoperative diagnosis of SAM/hypertrophic cardiomyopathy were excluded. We also excluded patients undergoing MV re-repair. A variety of repair techniques, as described elsewhere [7, 8], were employed using a lesion-specific strategy, and all patients received a complete annuloplasty ring.

Patient demographics, aetiologies of the valve dysfunction and surgical techniques used to perform valve repair are listed in Table 1.

Echocardiographic parameters (Table 2) were acquired from intraoperative transoesophageal echocardiographic (TEE) examination performed prior to the initiation of cardiopulmonary bypass.

SAM was defined as any portion of the anterior leaflet migrating into the left ventricular outflow tract (LVOT) after discontinuation of cardiopulmonary bypass. When this occurred, LVOT gradients and the degree of residual mitral regurgitation (MR) were assessed by echocardiography. Management of SAM involved an algorithm that has been previously published [3]. Based on the physiology of SAM, we investigated 11 echocardiographic parameters that could be potential risk factors for SAM: left ventricular ejection fraction (LVEF), left ventricular end-systolic diameter (LVESD), left ventricular end-diastolic diameter (LVEDD), basal septal diameter (basal-IVDd), mid-ventricular septal diameter (mid-IVDd), coaptation-septum distance (C-Sd), anterior leaflet height, posterior leaflet height, aorto-mitral angle, mitral annular diameter and left atrial diameter.

For the purposes of clinical application, we assigned categorical cut-off values for 8 of the 11 parameters and analysed them for significance: LVEF >65%, LVESD <35 mm, LVEDD <45 mm, basal-IVDd >15 mm, C-Sd <25 mm, anterior leaflet height >25 mm, posterior leaflet height >15 mm and aorto-mitral angle <120°.

Echocardiographic data acquisition

Intraoperative echocardiographic parameters were measured retrospectively by a single cardiac anaesthesiologist, board certified by the National Board of Echocardiography (NBE), based on the American Society of Echocardiography guidelines [9]. An LVEF was obtained using the biplane method of discs (modified Simpson's rule). LV internal diameters (LVEDD and LVESD) were measured from the mid-oesophageal two-chamber or transgastric mid-papillary short-axis views. The mid-LV septal wall diameter was measured from the transgastric mid-papillary short-axis view. C-Sd, aorto-mitral angle, anterior and posterior leaflet length, basal septal wall diameter and mitral annular diameter were all obtained from the mid-oesophageal long-axis view. Aorto-mitral angle was defined as the angle created between the mitral annulus plane and the aortic annulus plane. Leaflet lengths were measured, by choosing a frame that provided the best view of the anterior or posterior leaftet in its entirety, reaching from the MV annulus to the leaflet tip. The septal wall diameter was measured at the level of the basal and mid-LV. The left atrial diameter was measured at its antero-posterior linear dimension from the mid-oesophageal four-chamber view.

Statistical analysis

Continuous variables were expressed as the mean value with standard deviation and compared with Student's *t*-test or the Mann–Whitney *U*-test. Categorical variables were expressed as frequency and percentages and analysed by the χ^2 test. For continuous variables, testing for normality was performed by the combination of Kolmogorov–Simirnov test and visual assessment of histograms. Logistic regression analysis was used to identify the univariate and multivariate predictors of SAM. For multivariate analysis, we used a prespecified model with 11 echocardiographic parameters in a continuous format. The model fit and predictive power were validated with the Hosmer and Lemeshow goodness-of-fit test ($P = 0.83$) and *C*-statistics (0.90), respectively. Correlation analysis between each variable was performed (Pearson or Spearman as appropriate) to ensure no violation of the assumption of multicollinearity (with a cut-off correlation coefficient <0.7). For the purpose of clinical application, parameters which were found to be predictors in either univariate or multivariate analysis were also tested in categorical format with a specific cut-off given. This second-step analysis was performed for 8 of the 11 parameters by substituting the categorical variable for the continuous variable. The cut-off for each categorical variable was chosen based on a combination of receiver operating characteristic (ROC) analysis (Fig. 1), normal echocardiographic measurements, surgeon's experience and a literature review. Finally, each surgical technique was entered into the final model to test its influence on predicting SAM. Results are demonstrated as odds ratio (OR) and 95% confidence intervals (CIs). A *P*-value <0.05 was considered statistically significant. The statistical analysis was performed using SPSS for Windows, version 20.0 (SPSS, Inc., Chicago, IL, USA).

RESULTS

The overall incidence of SAM was 8% (30 of 375). Patient demographics are listed in Table 1. There were no significant differences in the baseline demographics or aetiologies of the valve dysfunction. There was a significant portion of patients with Barlow's disease in each group comprising 48% of patients. The mean EF was >55% in both groups.

The aetiologies of the valve dysfunction and surgical techniques used to perform valve repair are listed in Table 2. All patients had a complete ring used at the time of repair with the mean ring size being 32 ± 4 mm, with no significant differences between groups. For patients who developed SAM, a prespecified algorithm was utilized, resulting in resolution of significant SAM (mild or greater MR, LVOT gradient >50 mmHg) in all cases [3]. Baseline echocardiographic measurements are listed in Table 2.

Univariate predictors of SAM (Table 3) included the LVEDD ($P = 0.001$), LVESD ($P = 0.02$), basal-IVDd ($P = 0.002$) mid-IVDd ($P = 0.005$), C-Sd ($P < 0.001$) and aorto-mitral angle ($P < 0.001$). The use of the posterior leaflet sliding plasty technique was similar in both the NoSAM and SAM groups: 41% (143 of 345) vs 43% (13 of 30); ($P = 0.84$), respectively.

In multivariate analysis of continuous variables, the independent predictors of SAM were the LVEDD (OR 0.58, 95% CI 0.40–0.86, $P = 0.006$, per 5 mm increment), C-Sd (OR 0.49, 95% CI 0.32–0.76, $P = 0.001$, per 5 mm increment), posterior leaflet height (OR 1.14, 95% CI 1.02–1.23, $P = 0.023$) and aorto-mitral angle (OR 0.72, 95% CI 0.59–0.88, $P = 0.002$, per 5° increment).

The analysis of variables using prespecified cut-off values was then performed (Table 4). In this analysis, the independent predictors of SAM were LVEDD <45 mm (OR 3.9, 95% CI 1.16–13.2, $P = 0.028$), C-Sd <25 mm (OR 5.09, 95% CI 1.72–15.1, $P = 0.003$), basal-IVDd >15 mm (OR 3.63, 95% CI 1.07–12.3, $P = 0.039$), aorto-mitral angle <120° (OR 2.74, 95% CI 1.04–7.20, $P = 0.041$) and pre-repair posterior leaflet height >15 mm (OR 3.8, 95% CI 1.34–10.7, $P = 0.012$). Neither the aetiology of MR nor the location of leaflet

Table 1: Preoperative patient characteristics and operative data

Variable	All patients (*N* = 375)	Non-SAM (*N* = 345)	SAM (*N* = 30)	*P*-value
Demographics and comorbidities				
Age (year)	57 ± 12.9	57 ± 12.8	56.7 ± 13.8	0.96
Female gender, *n* (%)	136 (36)	124 (36)	12 (40)	0.69
Body surface area (m^2)	1.9 ± 0.23	1.9 ± 0.23	1.9 ± 0.25	0.73
Hypertension, *n* (%)	184 (49)	166 (48)	18 (67)	0.12
Hyperlipidaemia, *n* (%)	98 (26)	86 (25)	12 (39)	0.19
Diabetes mellitus, *n* (%)	19 (5)	17 (5)	2 (6)	0.87
Aetiology of mitral valve disease				
Barlow's disease, *n* (%)	179 (48)	162 (47)	17 (57)	0.31
Fibroelastic deficiency, *n* (%)	195 (52)	182 (53)	13 (43)	0.32
Marfan's syndrome, *n* (%)	1 (0.3)	1 (0.3)	0 (0)	0.76
Operative findings				
Anterior leaflet prolapse, *n* (%)	26 (7)	25 (7)	1 (3)	0.42
Posterior leaflet prolapse, *n* (%)	272 (73)	246 (71)	26 (87)	0.071
Bileaflet prolapse, *n* (%)	73 (20)	3 (10)	3 (20)	0.17
Surgical techniques				
Posterior leaflet resection, *n* (%)	301 (80)	273 (79)	28 (93)	0.061
w/ Sliding plasty, *n* (%)	256 (42)	143 (41)	13 (43)	0.84
Neochordal placement, *n* (%)	224 (60)	208 (60)	16 (53)	0.46
Anterior commissuroplasty, *n* (%)	25 (7)	24 (7)	1 (3)	0.45
Posterior commissuroplasty, *n* (%)	73 (20)	70 (20)	3 (10)	0.17
Mitral ring size (mm)	32 ± 4	32 ± 4	32 ± 4	0.76
Concomitant surgery				
Coronary artery bypass grafting, *n* (%)	20 (5)	18 (5)	2 (7)	0.74
Tricuspid annuloplasty, *n* (%)	248 (66)	226 (66)	22 (73)	0.39
Maze procedure, *n* (%)	38 (10)	36 (10)	2 (7)	0.51

Table 2: Intraoperative pre-bypass echocardiographic parameters

Variable	All patients (*N* = 375)	Non-SAM (*N* = 345)	SAM (*N* = 30)	*P*-value
Ejection fraction (%)	56 ± 8	56 ± 8	57 ± 9	0.63
Left ventricular end-systolic diameter (mm)	33 ± 9	33 ± 8	29 ± 10	0.018
Left ventricular end-diastolic diameter (mm)	55 ± 10	55 ± 10	48 ± 10	0.00070
Left atrial size (mm)	55 ± 10	55 ± 10	54 ± 10	0.82
Mitral annular diameter (long axis; mm)	39 ± 5	39 ± 5	38 ± 4	0.16
Septal thickness (basal; mm)	11 ± 3	11 ± 3	13 ± 3	0.0015
Septal thickness (mid-left ventricle; mm)	10 ± 3	10 ± 2	11 ± 4	0.06
Anterior leaflet length (mm)	30 ± 6	30 ± 6	32 ± 6	0.054
Posterior leaflet length (mm)	15 ± 5	15 ± 5	17 ± 3	0.031
Coaptation-septum distance (mm)	27 ± 7	27 ± 6	20 ± 6	<0.001
Aorto-mitral angle (°)	127 ± 13	128 ± 12	116 ± 12	<0.001

prolapse was found to correlate with the incidence of SAM. No one surgical technique employed to repair the valve was found to increase the risk of SAM or be protective against SAM. The ring size was similar between both groups and was not predictive of SAM. Anterior leaflet height and LVESD were not independent predictors for SAM. The incidence of SAM varied with the height of the posterior leaflet. Prerepair posterior leaflet heights between 15–20 mm were at greatest risk for SAM as illustrated in Fig. 1.

DISCUSSION

SAM has long been recognized as a potential cause of residual MR or outflow tract obstruction following MVR. This study looked at 375 consecutive patients undergoing MV surgery at a high-volume centre in order to further clarify potential risk factors for SAM. We found that the independent predictors of SAM were a smaller left ventricle, enlarged interventricular septum, short co-aptation to septum distance, narrow aorto-mitral angle and a tall posterior leaflet. These predictors suggest what intuitively makes sense—a small left ventricle with a narrow LVOT and a prerepair anteriorly displaced closure line increases the risk of SAM after valve repair.

The coaptation-septal distance was shown to be significant in univariate analysis by Cosgrove and colleagues [5] in a series of 14 patients and Maslow *et al.* [6] in a series of 33 patients after MVR. In this larger series, we found that a preoperative C-Sd <25 mm increased the risk of SAM five-fold. In addition, a tall posterior leaflet increased the risk almost four-fold. These two parameters

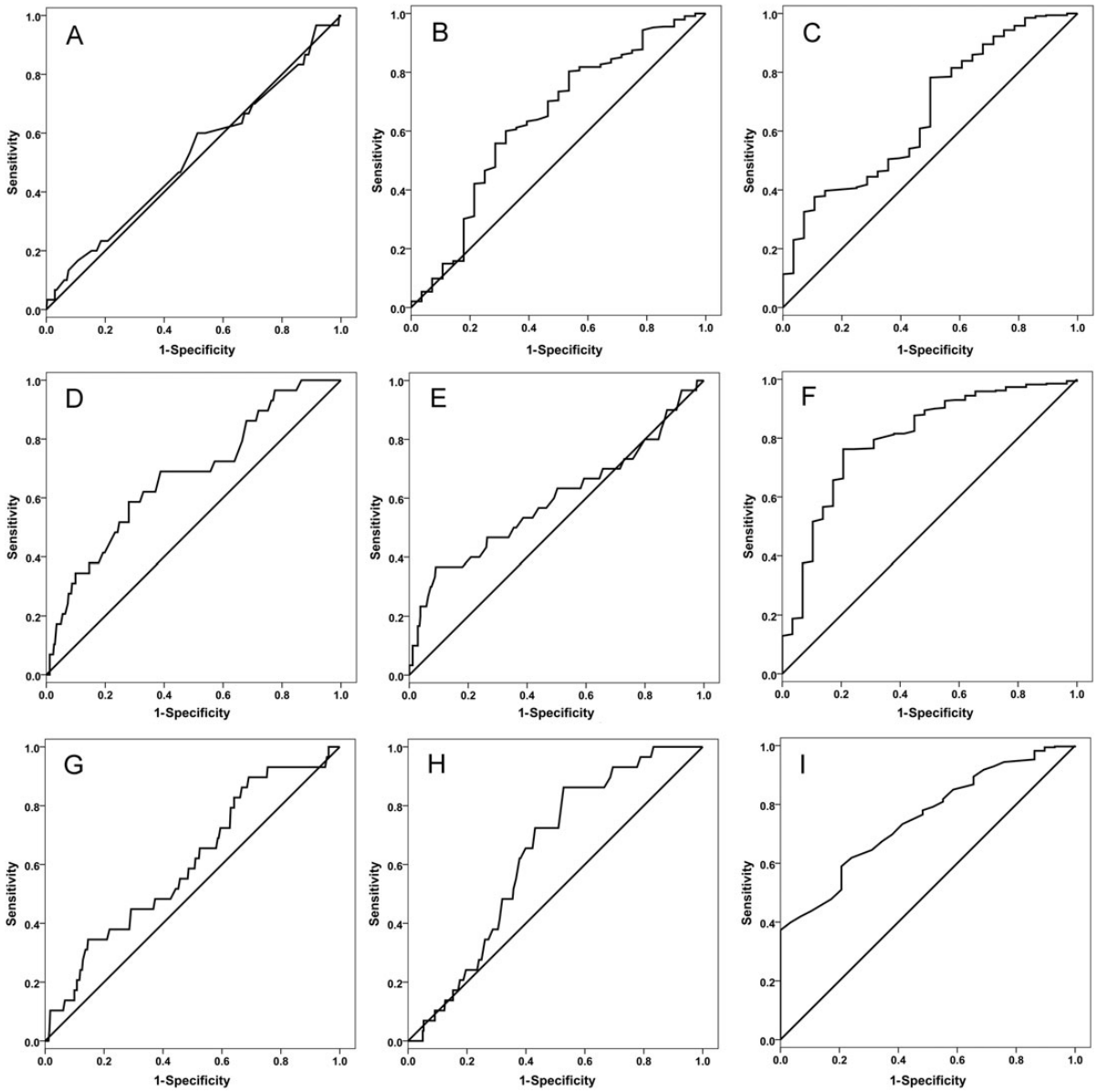

Figure 1: Receiver operating characteristic curves for each variable. (**A**) Ejection fraction; (**B**) end-systolic diameter; (**C**) end-diastolic diameter; (**D**) septal thickness (basal); (**E**) septal thickness (mid-ventricular); (**F**) coaptation-septum distance; (**G**) anterior leaflet length; (**H**) posterior leaflet length and (**I**) Aorto-mitral angle.

suggest that the MV leaflet coaptation point was anteriorly displaced prior to repair, and after repair one is more likely to be left with an anteriorly displaced closure line if this is not appreciated and surgically corrected. For tall posterior leaflets (>3 cm), our usual technique involves resection and sliding leaflet plasty. If after sliding the closure line remains anteriorly displaced, we most often use a relatively shortened neochord to displace the tallest portion of the posterior leaflet into the left ventricle, effectively shortening the posterior leaflet height and moving the coaptation line posteriorly. In our previously published series, 0.76% (6 of 785) of patients undergoing MVR required a return to cardiopulmonary bypass and re-repair of the valve for unresolving intraoperative SAM [3]. In none of these cases did the ring have to be upsized. Other studies have suggested that a smaller ring size increases the incidence of SAM; however, we were not able to support this conclusion in our own dataset, likely due to careful selection of the ring size at the outset.

In this study, we observed that the independent relationship the septum plays in predicting SAM where a basal septal thickness >15 mm independently increased the risk of SAM. As suggested by Sherrid *et al.* [10], a thickened basal septum (>15 mm) may change flow dynamics within the narrowed LVOT, leading to increased velocities through the LVOT that result in a pushing effect on the anterior leaflet into the outflow tract leading to SAM. Said *et al.* [11] suggested in a series of 6 cases that the ratio of the basal septal thickness to the mid-ventricular septal thickness ≥1.3 increased the risk of SAM, and that septal myectomy should be considered. Although this strategy may be effective, we do not advocate managing SAM by resecting the thickened septum if SAM was not present preoperatively, neither have we performed a septal myectomy for unresolving post-repair SAM. We believe that SAM in these cases resulted secondary to the repair and is exacerbated by the thickened septum. In cases where medical therapy does not suffice to resolve significant SAM, re-repair of

Table 3: Independent predictors of SAM (continuous variables)

Variable	Univariate		Multivariate	
	OR (95% CI)	*P*-value	OR (95% CI)	*P*-value
Ejection fraction[a] (%)	1.06 (0.85–1.34)	0.606		0.739
End-systolic diameter[b] (mm)	**0.74 (0.58–0.95)**	**0.019**		0.510
End-diastolic diameter[b] (mm)	**0.69 (0.56–0.86)**	**0.001**	**0.58 (0.40–0.86)**	**0.006**
Septal thickness (basal) (mm)	**1.16 (1.05–1.29)**	**0.003**		0.129
Septal thickness (mid-ventricular) (mm)	**1.20 (1.06–1.36)**	**0.005**		0.364
Coaptation-septum distance[b] (mm)	**0.39 (0.27–0.56)**	**<0.001**	**0.49 (0.32–0.76)**	**0.001**
Anterior leaflet length (mm)	1.06 (0.99–1.13)	0.055		0.204
Posterior leaflet length (mm)	1.06 (0.99–1.14)	0.107	**1.14 (1.02–1.23)**	**0.023**
Aorto-mitral angle[c] (degrees)	**0.69 (0.58 – 0.81)**	**<0.001**	**0.72 (0.59–0.88)**	**0.002**
Left atrial diameter[b] (mm)	0.98 (0.81–1.18)	0.817		0.523
Mitral annulus diameter (mm)	0.96 (0.89–1.03)	0.305		0.403

Hosmer–Lemeshow = 0.831 and *C*-statistics = 0.896
[a]The OR for ejection fraction is shown for each 5% increment.
[b]The ORs for end-systolic diameter, end-diastolic diameter, coaptation-septum distance and left atrial diameter are shown for each 5 mm increment.
[c]The OR for aorto-mitral angle is shown for each 5° increment. Boldface indicates statistically significant values ($P < 0.05$).

Table 4: Independent predictors of SAM (categorical variables)

Variable	Univariate		Multivariate	
	OR (95% CI)	*P*-value	OR (95% CI)	*P*-value
Ejection fraction >65%	1.38 (0.54–1.38)	0.51		0.79
End-systolic diameter <35 mm	2.51 (0.99–6.34)	0.053		0.680
End-diastolic diameter <45 mm	**3.21** (1.37–7.57)	**0.007**	**3.90 (1.16–13.2)**	**0.028**
Septal diameter (basal) ≥15 mm	**4.78 (2.06–11.1)**	**<0.001**	**3.63 (1.07–12.3)**	**0.039**
Coaptation-septum distance <25 mm	**8.40** (3.27–22.5)	**<0.001**	**5.09 (1.72–15.1)**	**0.003**
Anterior leaflet length ≥25 mm	4.10 (0.96–17.6)	0.058	5.70 (0.98–33.2)	0.052
Posterior leaflet length ≥15 mm	**2.61 (1.18–5.79)**	**0.018**	**3.80 (1.34–10.7)**	**0.012**
Aorto-mitral angle <120°[a]	**3.49 (1.62–7.56)**	**0.001**	**2.74 (1.04–7.20)**	**0.041**

[a]The cut-off of the aorto-mitral angle was set at 120° with an ROC analysis with a sensitivity of 73% and a specificity of 59%.

the MV should be considered. This can often be done with a brief period of cardioplegic arrest and effectively displacing the posterior leaflet height using a shortened neochord.

Carpentier and colleagues [12] first postulated the role that the aorto-mitral angle may play a role in causing SAM. In their paper, they discussed a post-repair angle of <120° as being a likely risk factor for SAM. In our dataset, we were also able to find an association between the presence of a prerepair narrow aorto-mitral angle and SAM.

Manabe *et al.* [13] recently observed that a hyperkinetic left ventricle was a risk factor for SAM. In our study, we were surprised to find that the EF did not predict SAM either as a continuous or categorical variable. Most of the patients in this series had a near normal EF, and one may hypothesize whether this would have been predictive for SAM if there was a larger spectrum of heterogeneity in the patient's EF.

When examining the posterior leaflet height, we found that patients whose prerepair posterior leaflet height was between 15 and 20 mm were at greatest risk for SAM (Fig. 1). On either side of this range, the incidence of SAM diminished such that patients with heights >25 mm or <10 mm did not develop SAM. This was an interesting finding and likely a result of the repair technique employed. At the lower end of the spectrum (posterior leaflet height <15 mm), the likelihood of SAM developing was small unless other factors predominated (tall anterior leaflet, thickened septum and small ventricle), and so the repair technique likely did not have an effect on the risk of SAM. At the higher end of the spectrum (posterior leaflet height >25 mm), it was likely that an intervention was undertaken to significantly shorten the posterior leaflet (resection and sliding plasty), hence reducing the likelihood of SAM. It was in the middle range of posterior leaflet heights (15–25 mm) where the incidence was greatest. This may have been due to our repair technique, where we may not have shortened the leaflet much in favour of greater coaptation depth. One must therefore be most cautious in this range of posterior leaflet heights.

The limitations of this study stem from it being a single institution experience where valves are repaired in a fashion that may not be common to other centres. Although we found that repair techniques did not significantly influence the incidence of SAM and the incidence was predominantly predicted by the patients' inherent and surgically uncorrectable anatomical characteristics, this may only be the case in our institution and with our philosophy towards valve repair; specifically, because we regard excess

leaflet tissue as a lesion contributing to valve dysfunction, we have an aggressive approach to resecting excess posterior leaflet and this may result in a different outcome with SAM as opposed to surgeons who use a non-resective approach. The strengths of this study lie in it being a large series of consecutive patients with degenerative MV disease with a large set of clinical and echocardiographic variables that were analysed providing for a robust and thorough analysis of predictors for SAM.

In summary, this study illustrates the important contribution that a small left ventricle, tall posterior leaflet, anterior displaced coaptation line and enlarged interventricular septum play in predicting the risk for SAM. The presence of these parameters on echocardiography would suggest an increased risk for SAM and appropriate measures, such as an upsized ring, ensuring a post-repair posterior leaflet height <15 mm and avoiding inotropes, when weaning from bypass should assist the surgeon in minimizing this complication.

Conflict of interest: David Adams is a consultant and inventor for Edwards LifeSciences. The other authors have no conflicts to declare.

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APPENDIX. CONFERENCE DISCUSSION

Dr R. De Simone *(Heidelberg, Germany)*: The authors provide a precise analysis of the data on systolic anterior motion, an annoying but serious event after otherwise successful repairs. Your study addressed the need to check not only for valve competence, which interests us more, but also to look actively for the motion of the leaflet. You chose the parameters that intuitively make sense, according to the well-known pathomechanism of SAM: smaller left ventricle, larger posterior leaflet, and so on.

Since this study includes a large series of patients with SAM, I would be very happy to see a table in your manuscript showing what happened to each patient according to the surgical technique used; this would give us the unique chance to learn more from such a large experience. I have three questions. First, how many patients underwent re-repair (and how was this achieved), and in how many patients was the SAM resolved just on the basis of optimizing the haemodynamic condition by fluid filling? Secondly, don't you think that it's rather difficult to measure the angle between the mitral and the aortic plane with a 2-dimensional technique? And, finally, how can the knowledge of a higher risk help us to minimize the risk of SAM? Don't you think that care in avoiding SAM is part of our surgical strategy for mitral valve repair?

Dr Varghese: Concerning your first question with respect to our surgical techniques and predicting SAM, and how we managed the patients that did develop SAM, we published that with more detail in our previous series in JTCVS. The majority of our patients that did develop SAM (and again, this included all systolic anterior motion, so 86% of patients that developed SAM in our series that we published earlier this year) resolved with medical interventions intraoperatively. The management of each of the six patients (0.6%) who developed intraoperative SAM requiring return to bypass was shown in a previous slide: they were all able to be fixed with one dose of cardioplegia. What we effectively do is shorten the height of the posterior leaflet with shortened Gore-Tex neochordae; we did not change the ring size, and SAM resolved in those six cases. However, the details of those are available in the previous paper I referred to; in the present paper we primarily focused on the predictors.

Concerning your second question with respect to the aorto-mitral angle, indeed it is very difficult to measure and it's very time-consuming. I don't know about the practicality of actually doing it on every patient. However, because it had been previously published back in 1988 by Carpentier and colleagues where they measured the aorto-mitral angle after the repair, after the ring was put in place, we wondered if it was an important predictor preoperatively, pre-repair. It has never been shown as an independent predictor and that's why we added it to the model because we were already collecting the other data. But I do agree with you that, practically speaking, the aorto-mitral angle is not as important as measuring maybe something like the septal diameter or the leaflet heights of the valve or the ventricular dimensions.

And your final question: what should we do once we do these measurements and a patient has a risk for SAM? I think one thing we always consider before we begin the repair is, what is the risk of SAM in this patient? And there is a certain level of care that is taken to provide the appropriate posterior leaflet height, as well as balancing that with ensuring you have a good coaptation depth of your repair predicting long-term durability. So in these patients where we are concerned, we don't oversize rings, we true size rings. But if we're in between a 30 and a 32, we might lean towards putting a 32 in if the valve was competent prior to tying the ring down. We also do an intraoperative ink test after saline injection to assess how much leaflet is below the coaptation line and that also guides us in choosing between ring sizes. And if we have excess tissue in a Barlow's valve, as I said, we will do a resection technique shortening the posterior leaflet effectively.

Dr M. Borger *(Leipzig, Germany)*: Sometimes in HOCM patients with severe SAM, we see that they have displacement of one or both papillary muscles; for example, the anterolateral papillary muscle can be more anteriorly displaced onto the ventricular septum than you would normally find. Did you see that in any of your 30 patients?

Dr Varghese: In this series, we excluded patients who presented with primary HOCM, and so we didn't observe the displaced papillary muscles as you mentioned. However, when we operate on patients who have primary SAM secondary to HOCM, indeed I do agree with you that there are a lot of thickened papillary muscles that are displaced towards the left ventricular outflow tract, and in those patients our approach is always to first open up the aorta, resect the septum, resect those thickened papillary muscles, take all the chordae that are coming across the outflow tract off the base, and then come off bypass and reinspect the valve. And in the majority of cases the SAM is resolved and we don't have to provide an intervention on the mitral valve.

ADULT CARDIAC

Dr Borger: So you've not seen that in a non-HOCM patient?

Dr Varghese: It's a little bit difficult to look at those chordae from the mitral valve side. And so if a patient doesn't present with primary SAM, then we don't go looking for those.

Dr F. Casselman *(Aalst, Belgium)*: Have you lowered the threshold for myectomy, more specifically, as a predictor for SAM?

Dr Varghese: For patients with degenerative disease?

Dr Casselman: Yes.

Dr Varghese: No, in patients with degenerative disease we don't advocate septal myectomy. If there was no SAM to begin with, coming into the operating room, we believe the SAM is secondary to the mitral valve repair.

Dr Casselman: So just the thickness of the septum doesn't lead you towards a myectomy?

Dr Varghese: No.

Dr T. Mesana *(Ottawa, ON, Canada)*: I have two questions, one following the other. Did you change your practice over the years towards less sliding plasty and more Gore-Tex use? And now that you have this study, did you switch back to resecting more posterior leaflet? Because I think it's still a significant higher number of SAM that you have here.

Dr Varghese: We have performed less resection in patients who present with smaller valves with less tissue, so fibroelastic deficiency or a forme fruste of Barlow's. But in patients that present with true Barlow's disease, we're still performing a sliding plasty with significant leaflet resection, aiming for a posterior leaflet height of around 15 mm. But we do use Gore-Tex neochordae more often, probably, in the last three years.

Dr Mesana: The sliding plasty has been described by Carpentier in great detail and has been the main solution to avoid and prevent SAM. So did you have SAM when you were using sliding plasty? And if yes, why do you think it still happened?

Dr Varghese: Well, in the study here when we looked at the repair technique and its ability to predict the incidence of SAM, we weren't able to show that any technique decreased the incidence of SAM. But we did show that posterior leaflet height was predictive. And it's probably because we just didn't have enough patients in each group to show that sliding leaflet plasty decreased the incidence of SAM. But the posterior leaflet height is a surrogate marker of that and so we do know that shortening that posterior leaflet height does decrease the incidence of SAM and that's exactly what the sliding plasty does. But if you don't do a sliding plasty, a lot of groups who don't do leaflet resection effectively shorten that posterior leaflet height by using shortened Gore-Tex neochordae.