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Left Ventricular Muscle Mass Regression After Aortic Valve Replacement

Implanting a valve that will reduce left ventricular mass is critical in aortic stenosis. Regression of left ventricular hypertrophy in 46 aortic valve replacement (AVR) patients receiving a St. Jude Medical (SJM) valve was assessed by serial electrocardiographic and echocardiographic studies during the preoperative, immediate, and late postoperative periods. The patients were divided into three groups according to valve size; 19 mm group (n=9), 21 mm group (n=20), and 23+mm group (n=17). There was no surgical mortality. The NYHA functional class improved from an average of 2.2±0.8 preoperatively to 1.3±0.5 postoperatively. Left ventricular muscle mass index (LVMI) regression failed to reach statistical significance in the 19 mm group, whereas in the other two groups a steady decrease in the LVMI occurred with follow up. ECG findings were less remarkable showing insignificant differences in voltage among the three groups (p=0.000). In conclusion, the current data suggest that the 19 mm SJM valve may not result in satisfactory left ventricular muscle mass regression despite adequate function, even in small patients. Therefore, additional procedures to accommodate a larger valve may be warranted in the aortic annulus smaller than 21 mm.

Key Words: *Aortic valve; Hemodynamics; Heart hypertrophy; Ventricular function, left; Prosthesis failure*

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INTRODUCTION

Patients with aortic stenosis requiring valve replacement usually have left ventricular hypertrophy. Selecting a prosthetic valve that will reduce the increased left ventricular muscle mass to normal is important. However, aortic valve replacement (AVR) with a small prosthetic valve may preclude left ventricular mass regression and adversely affect long term survival. This may be due to the inherently obstructive nature of prosthetic valves, which become more precipitous with decreasing valve size (1). Studies have shown 21 mm to be the lower limit in size for prosthetic valves in which significant regression of left ventricular hypertrophy failed to occur (2).

The St. Jude Medical (SJM) prostheses has been designed to have minimal resting and post exercise gradients (3, 4). Wortham et al. (5, 6) demonstrated that gradients across the 19 mm SJM valve in the patient with a body surface area (BSA) less than 1.7 m^2 was small and acceptable. Rashtian (7) confirmed this optimistic finding by demonstrating a mean resting gradient of 5 mmHg and a peak gradient of less than 15 mmHg in patients with 19 mm SJM valve implants.

Based on the belief that the purportedly superior hemodynamic qualities of the SJM prostheses would overcome the limitations imposed by the so-called "patient-prosthesis mismatch", small sized standard SJM aortic prostheses (19 and 21 mm) were implanted without annular enlargement at Asan Medical Center since July 1991 to August 1997. The aim of the present study lay in determining possible risk factors that may cause poor regression of left ventricular hypertrophy in patients receiving AVR with SJM valves through a series of ECG and Echo data collected in the preoperative, immediate postoperative, and late (10.7 months after surgery) postoperative periods.

MATERIALS AND METHODS

Between July 1991 and August 1997, 46 patients underwent isolated aortic valve replacement (AVR) with the

SJM valve for predominantly aortic valvular stenosis at Asan Medical Center, Seoul, Korea. Patients less than 16 years of age, and those with other associated cardiac disease or operations were excluded from the present study. Of the 46 patients, nine received 19 mm valves, 20 received 21 mm valves, 14 received 23 mm valves, and three received 25 mm valves. The patients were divided into three groups according to valve implant size; 19 mm, 21 mm, and 23+mm groups. The patients receiving 23 mm and 25 mm valves were placed in the 23+mm group.

Echocardiography

Echocardiograms were performed with a Hewlett-Packard Sonos 2500 apparatus (Hewlett-Packard Co., Palo Alto, CA, U.S.A.) using a 3.5 MHz transducer for imaging and a 2.5 MHz transducer for Doppler echocardiography. All echocardiograms were recorded on Super VHS videotapes for subsequent analysis.

M-mode echocardiograms were used to measure, in mm, the telediastolic interventricular septal thickness (IVST), left ventricular posterior wall thickness (PWT), and telediastolic (LVEDD) and telesystolic (LVESD) left ventricular dimensions. All measurements were repeated for four successive heartbeats and averaged. The measurements were made in accordance with the Penn convention.

Left ventricular mass (LVM) was calculated using the D^3 formula (Devereux and Reichek): LVM (g)=1.04$\times$([LVEDD + IVST + PWT]^3 LVEDD^3)$\times$13.6. LVM index (LVMI, g/m^2) was defined as LVM/BSA, where BSA is body surface area in square meters. Mean and peak pressure drops across the prosthetic valves (MSG; Mean Systolic Gradient, PSG; Peak Systolic Gradient) were estimated using the simplified Bernoulli equation from peak velocity measurements using continuous-wave Doppler echocardiography.

Echocardiography was performed in the preoperative period, immediate postoperative period (just before discharge at 7.1 days postoperatively), and between six and 18 months after operation in the late period.

Electrocardiography

We reviewed the electrocardiograms of all the patients in the immediate preoperative and postoperative periods, and every subsequent clinical follow-up. The ECG taken simultaneously with the echocardiograms were used as the sample for the present study. In three patients, postoperative complete heart block and in another three, bundle branch block was the basis for exclusion from the electrocardiographic study. Measurements of R waves in lead V5 or V6 plus S wave in V1 in mm were used as a criterion for determining LVM. In two cases with atrial fibrillation, the average voltage of three consecutive QRS waves was used as a standard for LVM determination.

Statistical analysis

Data were presented as mean$\pm$1 standard deviation. Using SPSS for Windows, Release 7.5 (7), clinical data and preoperative echocardiographic and electrocardiographic measurements from the three groups were compared by one-way analysis of variance and multiple comparison tests with least significance difference. The paired t-test was used to examine the significance of changes over time in all the patients. Possible significant effects on postoperative left ventricular muscle mass or hypertrophy by age, sex, body surface area, preoperative pressure gradient, postoperative mean and peak gradients in the late period, valve size (19 mm or others), and follow-up duration were each assessed initially by one way analysis of variance (ANOVA). Factors shown to be of significance were entered into a multivariate analysis of variance (General Linear Model), and their independent roles on each echo and ECG parameter were further defined. Post hoc testing of ANOVA was performed using the Dunkan method. A p-value of 0.05 or less was considered statistically significant.

RESULTS

The preoperative characteristics of the three study groups are shown in Table 1. The patients in the larger valve size groups had significantly larger BSAs and were composed predominantly of males, whereas those in the 19 mm valve group were all females. There were no statistically significant differences among the three groups regarding age, NYHA class, degree of stenosis (peak pressure gradient and valve area), or overall LV systolic function. Although the LVEDD was somewhat larger in the 23+ group than the other two groups, the difference in the mean LVMI was statistically insignificant (Table 2). The mean preoperative systolic gradient was largest in the 19 mm (Table 3).

The mean ejection fraction of the study population did not change postoperatively until the end of the study. The LVESD decreased steadily after surgery, but the LVEDD which showed an initial decrease in the immediate postoperative period, failed to decrease thereafter. The left ventricular IVST and PWT, on the other hand, both showed a decrease by the late postoperative period (Table 4). The changes in these parameters were reflected in the LVMI change during follow up. The mean of the

Table 1. Preoperative and operative demographics of the study groups

	19 mm group	21 mm group	23+mm group	*p*
No.	9	20	17	
Age (yr)	58±16	54±13	61±9	NS
Sex (female%)	100	50	0	0.000*
BSA (m^2)	1.47±0.11	1.56±0.14	1.69±0.12	0.000†
NYHA	2.3±0.8	2.0±0.8	2.3±0.9	NS
Pump time (min)	166±62	131±31	121±46	NS
Echo F/U (m)	10.5±4.6	10.6±3.9	10.2±2.8	NS
Total F/U (m)	40.6±27.9	33.4±20.2	27.9±19.4	NS

No., number of patients; BSA, body surface area; NYHA, New York Heart Association functional class; F/U (m), follow-up duration in months

* between any two groups, † between 23+ and 19 or 21 mm groups

Table 2. Preoperative echocardiographic data

	19 mm group	21 mm group	23+mm group	*p*
EF (%)	62±19	57±18	59±13	NS
LVESD (mm)	33±12	33±11	38±8	NS
LVEDD (mm)	46±9	48±7	57±8	0.002*
IVST (mm)	14±3	14±3	15±3	NS
PWT (mm)	14±2	14±3	14±2	NS
LVMI (g/m^2)	218±47	226±54	266±88	NS*

EF, ejection fraction of left ventricle; LVESD, left ventricular end systolic dimension; LVEDD, left ventricular end diastolic dimension; IVST, interventricular septal thickness; PWT, left ventricular posterior wall thickness; LVMI, left ventricular muscle mass index (left ventricular muscle mass/body surface area)

* between 23+ and 19 or 21 mm groups

Table 3. Preoperative echocardiographic data (II)

	19 mm group	21 mm group	23+mm group	*p*
PSG (mmHg)	113±24	102±34	89±22	NS
MSG (mmHg)	84±16	59±21	57±16	0.023*
Valve area (cm^2)	0.60±0.15	0.64±0.25	0.75±0.38	NS

PSG, peak pressure gradient across aortic valve; MSG, mean pressure gradient across aortic valve

* between 19 and 21 or 23+ mm groups

Table 4. Changes in echocardiographic findings and pressure gradients of left ventricle over time

	Preoperative	Immediate post-op.	Late post-op.	*p*
EF (%)	59±15	56±13	62±10	NS
LVESD (mm)	35±10	32±9	29±6	0.031*
LVEDD (mm)	50±9	47±10	47±8	<0.01†
IVST (mm)	14±3	15±3	12±2	0.000‡
PWT (mm)	14±3	14±2	12±2	0.000‡
PSG (mmHg)	98±29	33±11	34±12	0.000§
MSG (mmHg)	62±20	19±7	18±7	0.000§

EF, ejection fraction of left ventricle; LVESD, left ventricular end systolic dimension; LVEDD, left ventricular end diastolic dimension; IVST, interventricular septal thickness; PWT, left ventricular posterior wall thickness; MSG, mean systolic gradient; PSG, peak systolic gradient

* between preoperative and late postoperative values

† between preoperative and immediate or late postoperative values

‡ between late and preoperative or immediate postoperative values

§ between any two groups

Table 5. Mean LVMI at different time points and LVMI variance

	Preoperative	Immediate	Late	Pre-Imm	Imm-Late	Pre-Late	
19 mm	218±47	193±68	175±46	27±47	12±13	51±30	p=0.132*
21 mm	226±54	220±68	157±42	10±61	83±44	84±50	p=0.03*
23+mm	266±88	217±39	167±38	51±68	52±38	96±82	p=0.00*
				p=ns†	p=0.007†	p=ns†	

* significance of LV mass index regression, † ANOVA among the three groups

LVMI as shown in Table 5 has progressively decreased in all three groups. However the LVMI regression in the 19 mm group failed to reach statistical significance (p=0.032). The variance in LVMI change also shows that no significant change in LVMI regression occurred in the 19 mm group between the preoperative and the immediate postoperative periods as well as between the immediate postoperative and late periods (Fig. 1). With the exception of the 23+mm group (p=0.025) significant LVMI regression between the preoperative and the immediate postoperative periods did not occur. Beyond the immediate postoperative period, however, the 19 mm group continued in failing to show significant change in the LVMI variance or LVMI decrease, whereas significant changes in LVMI variance in both the 21 mm and 23+ mm groups occurred in the period between the immediate and late postoperative periods (Fig. 1). The difference in the degree of variance in the LVMI change among the three groups reached statistical significance only in the period between the immediate and late periods, p=0.007 (Table 5, Fig. 1). Between the preoperative and immediate postoperative periods and the preoperative and late postoperative periods, no statistically significant differences were seen in the LVMI variance among the three groups (Fig. 1 and Table 5).

The changes in the mean PSG and MSG measurements showed a progressive decrease with each successive follow up time point (Table 4). In the late period the MSG systolic gradient was highest in the 19 mm group among the three groups (p=0.003) (Table 7).

The change in mean ECG voltage showed a statistically significant progressive decrease throughout the study period in all three groups (Table 6). The variance in the ECG voltage decrease between the preop and immediate post op, the immediate post op and late post op, and preoperative and late postoperative periods were found to be significant in each of the three groups. However, ANOVA of the ECG variance among the three groups during these follow up intervals did not show significant differences (Table 6, Fig. 2). Therefore, the ECG

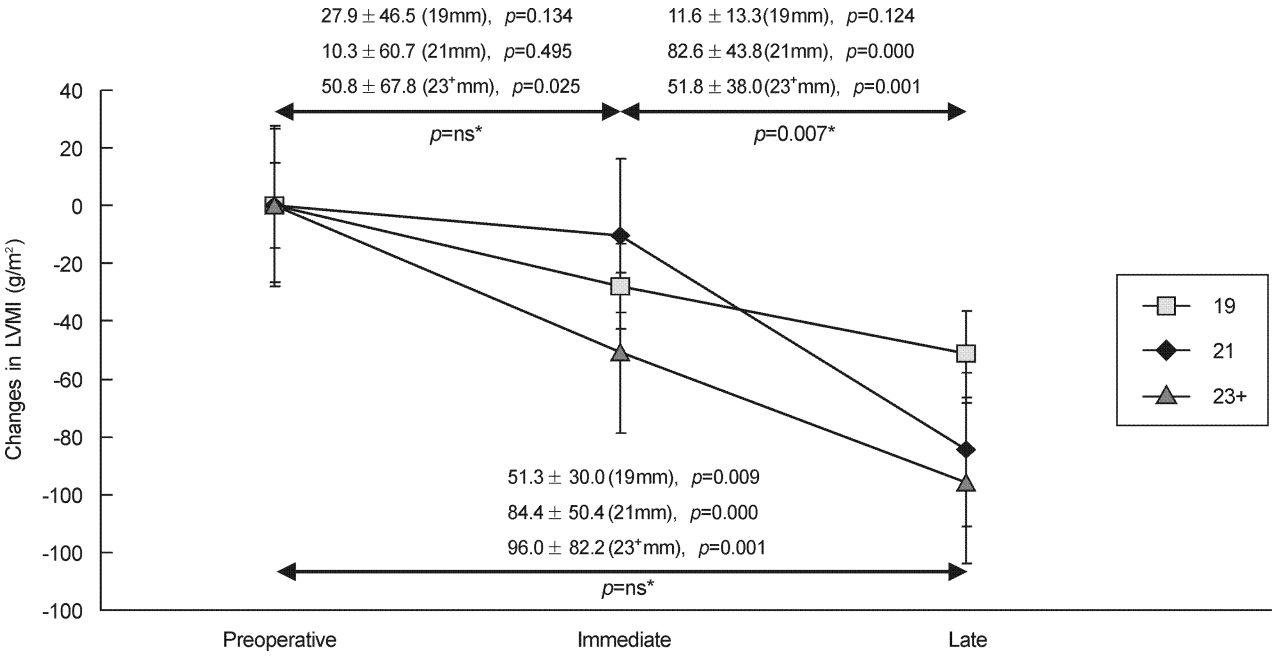

Fig. 1. Changes in LVMI in the three study groups with time. *Comparison among groups by ANOVA. LVMI, left ventricular mass index (g/m^2)

Table 6. Mean ECG voltage change at different time points and ECG variance

Group	Preoperative	Immediate post-op.	Late post-op.	Pre-Imm	Imm-Late	Pre-Late	
19 mm	44.4±7.5	34.2±6.0	30.8±7.4	10.2±6.2	3.4±2.5	13.7±7.3	p=0.000*
21 mm	47.7±9.6	36.8±9.8	27.3±7.5	10.9±6.2	9.5±5.7	13.7±7.2	p=0.000*
23+mm	48.6±11.1	35.2±9.8	28.1±5.8	13.4±11.2	7.1±7.7	20.5±12.9	p=0.000*
				p=ns†	p=ns†	p=ns†	

* significance of ECG voltage regression, † ANOVA among the three groups

Table 7. Pressure gradients in the late period

	19 mm group	21 mm group	23+mm group	p
PSG (mmHg)	45±18	34±7	29±10	0.053*
MSG (mmHg)	26±11	18±4	15±4	0.003*

PSG, peak pressure gradient across aortic valve; MSG, mean pressure gradient across aortic valve
* between 19 and 21 or 23+ mm groups

voltage decreased in all three groups with follow up but in a non specific manner. Multivariate analysis showed valve size and follow up until the late period as significant factors for LVMI regression and ECG voltage decrease (Table 8). The time point in the late follow up that was used in the assessment of the LVMI regression by multivariate analysis was at 12 months.

No statistically significant differences in the NYHA functional class were seen among the three groups (p> 0.05). The preoperative NYHA functional class for the 19 mm, 21 mm, and the 23+mm groups were 2.3+0.7, 2.0+0.8, and 2.3+0.9, respectively, (Table 1) and the postoperative values for each of the three groups were 1.3+0.5, 1.3+0.6, and 1.3+0.8. Analysis of variance showed no statistically significant difference among the groups in the NYHA functionaly class. Accordingly, changes in the pre and postoperative NYHA functional class of the study population were represented by the combined mean of the three study groups (Fig. 3). There was no hospital mortality. The three patients that had complete heart block in the immediate postoperative period required permanent pacemaker implantation. During the entire follow-up period of 100 patient years (mean 32.7 months), there was one stroke related death despite adequate anticoagulation, and another death due to malignancy comprising a 2%/patient-year late mortality

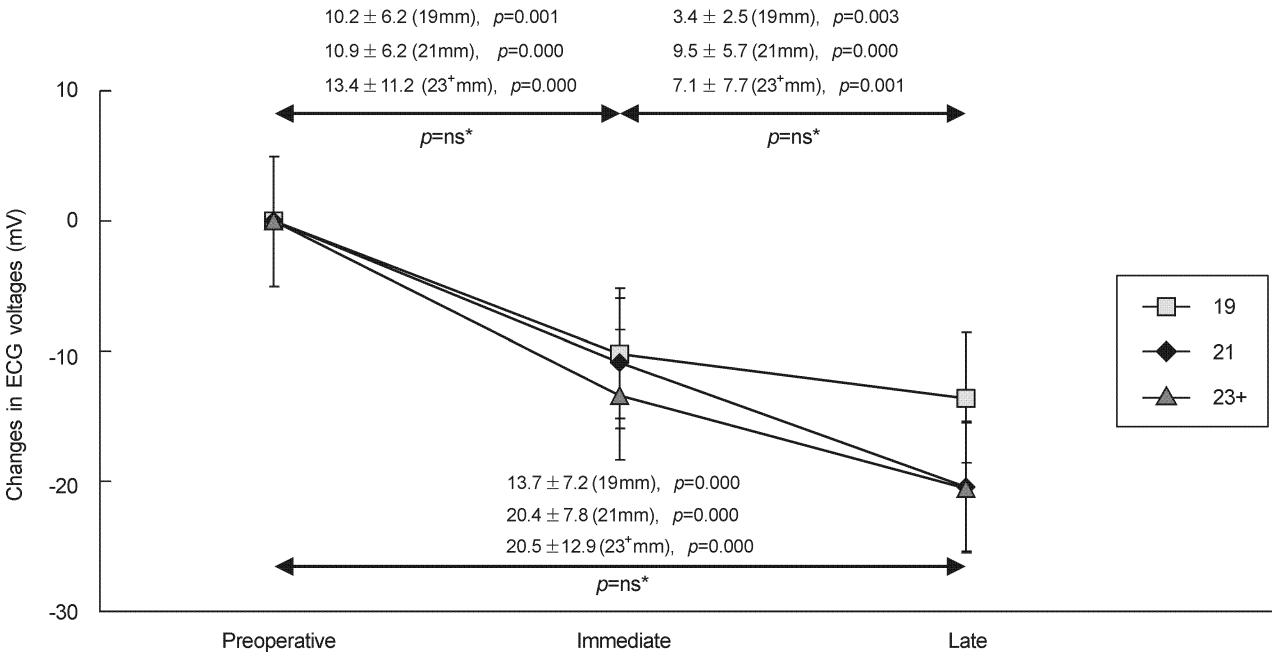

Fig. 2. Changes in ECG voltages in the three study groups with time. *Comparison among groups by ANOVA

Table 8. Multivariate analysis of variance of left ventricular hypertrophy after aortic valve replacement

Item/Intervals	Age	Sex	MSG	V-size	NYHA	F/U*
LVMI						
Preoperative-Immediate	NS	NS	NS	NS	NS	NS
Immediate-Late	NS	NS	NS	0.007	NS	NS
Preoperative-Late	NS	NS	NS	NS	NS	0.047
ECG						
Preoperative-Immediate	NS	NS	NS	NS	NS	NS
Immediate-Late	NS	NS	NS	NS	NS	NS
Preoperative-Late	NS	NS	NS	NS	NS	NS

MSG, mean pressure gradient across aortic valve; V-size, prosthetic valve size; NYHA, new York Heart Association functional class; F/U, the late follow up duration entered in the multivariate analysis was at 12 months; LVMI, left ventricular muscle mass index (left ventricular muscle mass/body surface area); Preoperative-Immediate, difference between the preoperative and the late period; Immediate-Late, difference between the immediate and the late period; Preoperative-Late, difference between the preoperative and the late period; NS, not significant statistically

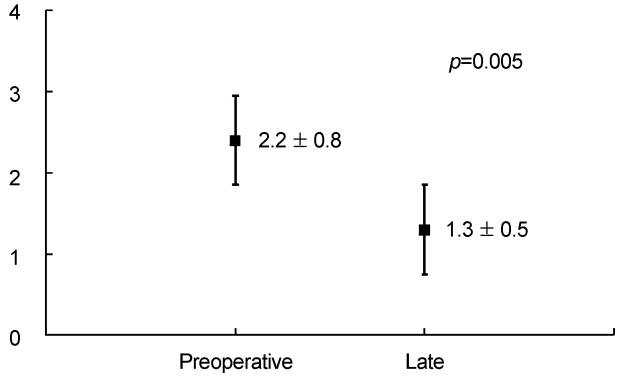

Fig. 3. Changes in NYHA class with time.

rate. There was also a 2%/patient-year of bleeding event (gastrointestinal bleeding). There were no cases of postoperative heart failure or cardiac deterioration.

DISCUSSION

Current aortic prostheses invariably cause some degree of flow obstruction due to inherent design limitations that result in an effective orifice area that is smaller than the native aortic annulus. Notwithstanding, several investigators have observed satisfactory symptomatic improvement after AVR with 19 and 21 mm mechanical valves in patients with small body surface areas (9). Accordingly, the SJM prosthesis has been considered the valve of choice in these situations (10, 11). The current difficulty in valve selection, however, lies in determining the lower size limit below which "patient prosthesis mismatch" and reduced long term survival will likely occur, as no controlled or randomized data are available in the surgical literature to guide the surgeon in making such predictions.

There is a prevailing concern that AVR with a small prosthesis may increase the risk of late complications. It has even been suggested by some that AVR with a small prosthesis is the equivalent of merely replacing one disease with another persistent, but non-progressive type of aortic stenosis. However, Sim et al. (2) demonstrated faster resolution of left ventricular hypertrophy (LVH), a well known risk factor for sudden cardiac death and cardiovascular morbidity (12) after aortic valve replacement. On the other hand, it was also found that left ventricular hypertrophy may not resolve completely if the implanted prosthetic valve was small (13). Concerns have thus been raised regarding the long-term influence of residual LVH after AVR but the relationship between the two has not been fully established (14).

The assessment of left ventricular muscle mass is usually based on echocardiographic and ECG findings. The ECG findings, however, is prone to be nonspecific as it may be affected by factors not directly reflecting changes in the left ventricular muscle mass itself such as pericardial effusion or pericarditis. In the current paper, a larger variance in the ECG voltage decrease was expected in the 21 and 23+mm group compared to the 19 mm group. However, all three groups showed a similar degree of variance in the ECG voltage decrease. Change in the LVMI is a more direct method of assessing left ventricular muscle mass. Of interest, the LVMI was assessed at two follow up time points. Namely, in the immediate postoperative and the late postoperative periods. The 19 mm group failed to show significant regression between the preoperative and immediate postoperative periods as well as the interval beyond the immediate postoperative period until the late period. The 21 and 23+ groups however, both showed significant changes in the LVMI variance between the immediate postoperative period and the late period. Thus variance in the LVMI decrease between the immediate and late postoperative periods among the three groups was statistically significant (p=

0.007) because the decrease in the LVMI in the 19 mm group in this interval failed to reach statistical significance. Therefore, it is suggested in the data that the 19 mm valve compared to the other two sizes, may result in significantly less regression in the left ventricular muscle mass.

The measurement of postoperative gradients has long been used as a tool to assess the adequacy of AVR, and indexing the valve area with BSA was found to improve the correlation between valve area and gradient (15). Gonzalez-Juanatey et al. (16) demonstrated that small valve size was a risk factor for inadequate LVH regression and possibly long-term survival. However, their study was flawed by including three different kinds of prosthetic valves (one mechanical and two tissue valves). Sawant et al. (11), showed satisfactory clinical results with 19 mm SJM prosthesis irrespective of the BSA. They concluded that the 19 mm SJM valve was a viable alternative for valve replacement even in large patients with a small aortic annulus, dismissing patient-prosthesis mismatch as a possible risk factor for poor long-term outcome. Unfortunately, the hemodynamic performance of the implanted 19 mm SJM valves or the regression of LVMI were not studied. In our study, valve size was not found to be an independent risk factor for clinical deterioration, but the patients with a 19 mm SJM prostheses did show a higher pressure gradient across the prosthesis and less regression of left ventricular hypertrophy. Schaff et al. (17) found 20% LVM regression in a 46 month follow up of AVR patients with BSA less than 1.64 m^2 using a 19 mm Bjork-Shiley prostheses. Jin et al. (18) noted 32% to 39% regression of LVM six months after AVR using stentless porcine or homograft valves but only 16% regression with stented valves. Our data also showed regression of just 20% in the 19 mm group, but 32% and 39% LVMI regression at approximately 10.5 months after AVR in the larger 21 and 23+mm groups, respectively. The percent regression in the latter groups were comparable to those of Jin et al.'s stentless and allograft aortic valves (18). From the multivariate analysis, the duration of follow up until the late period was found to be the only independent risk factor for poor LVMI regression on late echocardiography in the 19 mm group. These results support the view that a prosthetic valve with a small valve area may function well in patients with a small BSA, i.e., our 21 mm valve group with a mean BSA of 1.54 m^2. However, the results of our study which is also supported by Jin et al.'s findings on their stented valve group (24 mm with BSA of 1.79 m^2) showed that the 19 mm valve will most likely result in poor LVMI regression.

Calculations of actual valve areas showed a 19 mm SJM valve in a patient with a BSA of 1.7 m^2 to have an effective orifice area index of 0.75 cm^2/m^2 (19). This value is well below the transition point of 0.8 to 0.9 cm^2/m^2, described by Dumesnil et al. (15) as the point in which transvalvular pressure gradients become prohibitively high. Moreover, an aortic valve area index of less than 0.75 cm^2/m^2 is often labeled as severe aortic stenosis. Therefore, it is difficult to understand how patients with such small valves can do well clinically when their aortic valves are actually severely stenotic. Rahimtoola (1) noted that many such patients seemed to do well. Kratz et al. (20) mentioned that there might be at least three possible explanations.

The results of our data suggest that although no clinically detectable short-term negative effects were noted with simple implantation of the largest possible SJM valve, in those with small valve implants, close observation is warranted. A small valve with a residual gradient might adversely affect the incidence of heart failure during long-term follow-up. Indeed, comparison of the preoperative and late postoperative MSG among the three study groups showed the 19 mm valve implants to have the greatest pressure gradient. There was also a clear difference in the degree of LVMI regression with the size of valve implants in the current study, which showed the 19 mm valve to result in significantly less regression in LVMI as compared to the other two larger valve sizes. Thus annular enlargement should be undertaken when the annulus size is diminutive. This is especially important in the patient with associated coronary artery disease (21). Aortic stenosis in an ischemic myocardium may place the patient in danger of oxygen supply/demand mismatch with subsequent ventricular arrhythmia and sudden death (22, 23). Consequently, patients with concomitant coronary bypass were excluded from the present study. This may partially explain the good clinical results that we experienced despite the residual transvalvular pressure gradients across the prosthetic valves. Our results also showed the 19 mm SJM valve to be unsuitable for patients even with a mean BSA as small as 1.48 m^2, since similar degrees of LVMI regression as in the 21 and 23+mm groups could not be attained.

The simplest annular enlarging procedure for the small aortic annulus is a posterior annulus enlarging procedure, i.e, a modified Nick's or Manouguian's procedure (24). The only possible contraindication to these procedures is massive calcification in the non-coronary sinus or the surrounding aorta. However, no such cases were seen in our study. Furthermore, it was always possible to avoid annular enlargement whenever heavy calcification of the aortic wall was present. Sommers and David (24) demonstrated the annulus-enlargement procedures to be safe and acceptable in terms of operative mortality and long-term survival. Carpentier had mentioned that the surgical risk of

adding an annulus enlarging procedure was negligible and recommended its use whenever indicated (25).

As our study was retrospectively conducted on a carefully selected group of patients, the number in each group was small. Likewise, the follow up duration may have been inadequate for any meaningful conclusions to be drawn on the assessment of long-term complications among the study groups. Nonetheless, the present study was designed to see the effects of patient-prosthesis mismatch on the regression of left ventricular hypertrophy, as left ventricular hypertrophy is known to occur mainly during the early postoperative period and continue up to the first six months postoperatively (18). Therefore, the patient population was restricted to those with aortic stenosis only, excluding those with any other type of significant associated cardiovascular diseases. However, due to the limited follow-up duration, a conclusive assessment on the results of LVH regression and clinical correlation was difficult to make. As a further limitation, echocardiographic studies of the SJM valves were performed only in the resting state and exercise hemodynamic studies were not performed. Therefore, exercise hemodynamic studies are currently underway in our institution as part of a long-term study of patients receiving aortic valve replacement for aortic valve stenosis. In conclusion, this intermediate-term study provides evidence to suggest that the 19 mm SJM prosthesis, compared to larger SJM prostheses, may delay or hinder the regression of left ventricular hypertrophy. Accordingly, in the patient with aortic annulus less than 21 mm, we measures to seat larger valves such as annular enlargement is warranted.

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