Surgical as Opposed to Transcatheter Aortic Valve Replacement Improves Basal Interventricular Septal Hypertrophy

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Background: Basal interventricular septum (IVS) hypertrophy (BSH) with reduced basal IVS contraction and IVS-aorta angle is frequently associated with aortic stenosis (AS). BSH shape suggests compression by the longitudinally elongated ascending aorta, causing basal IVS thickening and contractile dysfunction, further suggesting the possibility of aortic wall shortening to improve the BSH. Surgical aortic valve replacement (SAVR), as opposed to transcatheter AVR (TAVR), includes aortic wall shortening by incision and stitching on the wall and may potentially improve BSH. We hypothesized that BSH configuration and its contraction improves after SAVR in patients with AS.

Methods and Results: In 32 patients with SAVR and 36 with TAVR for AS, regional wall thickness and systolic contraction (longitudinal strain) of 18 left ventricular (LV) segments, and IVS-aorta angle were measured on echocardiography. After SAVR, basal IVS/average LV wall thickness ratio, basal IVS strain, and IVS-aorta angle significantly improved (1.11 ± 0.24 to 1.06 ± 0.17 ; -6.2 ± 5.7 to $-9.1 \pm 5.2\%$; 115 ± 22 to $123 \pm 14^\circ$, $P < 0.001$, respectively). Contractile improvement in basal IVS was correlated with pre-SAVR BSH (basal IVS/average LV wall thickness ratio or IVS-aorta angle: $r = 0.47$ and 0.49 , $P < 0.01$, respectively). In contrast, BSH indices did not improve after TAVR.

Conclusions: In patients with AS, SAVR as opposed to TAVR improves associated BSH and its functional impairment.

Key Words: Aortic valve replacement; Aortic valve stenosis; Basal septal interventricular hypertrophy

Basal interventricular septum (IVS) hypertrophy (BSH) is characterized by reduced angle between the basal IVS and aorta and thickened basal IVS (Figure 1A,B). This is common in elderly patients and is associated with reduced contraction in the region.¹ Aortic stenosis (AS) develops mainly in the aged population,² therefore BSH is also common in patients with AS. BSH may potentially contribute to left ventricular (LV) outflow tract (LVOT) obstruction following surgical or transcatheter aortic valve replacement (SAVR and TAVR, respectively) with unfavorable outcome,^{3–5} and may increase the difficulty in accurate positioning and implantation of the prosthetic valve by confounding coaxial alignment of the

guidewire and/or the valve or by resulting in superior displacement of the prosthetic valve during deployment.⁶ Therefore, BSH is especially important in patients with AS. SAVR or TAVR are the standard interventions for symptomatic AS, but the effects of AVR on the associated BSH have not yet been clarified.

Although the mechanism of BSH is not yet established, its configuration suggests augmented compression of the IVS by the longitudinally elongated ascending aorta (Figure 1C). This concept has been suggested by previous imaging studies.⁷ These further suggest a potential of aortic wall shortening to improve the BSH. SAVR includes incision and suture of the anterior wall of the ascending aorta.

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Figure 1. Hypothesis suggested from configurations of basal interventricular septal hypertrophy (BSH). **(A)** Normal interventricular septum (IVS) and aorta configurations. **(B)** Typical BSH. BSH is characterized by reduced angle α between the basal IVS and ascending aorta and thickened basal IVS (yellow arrows). **(C)** Potential mechanism of BSH. Configurations of BSH suggest compression of the IVS by the longitudinally elongated ascending aorta. **(D)** Potential effects of surgical aortic valve replacement (SAVR) on BSH (hypothesis). SAVR includes incision and suture of the anterior wall of the ascending aorta. By taking the margin to the seam, SAVR shortens the anterior wall of the ascending aorta. In the present study, aortic wall shortening was approximately 1 cm with a 0.5-cm margin for the stitch. This can potentially reduce compression of the IVS by the ascending aorta, leading to attenuated post-SAVR BSH and improved contraction.

By taking the margin to the seam, SAVR shortens the aortic wall. The anterior aortic wall shortening in this study was approximately 1 cm, with a 0.5-cm margin for the stitch (**Figure 1D**). This aortic wall shortening may exert a superiorly directed force on the basal IVS, causing the myocardium to be less compressed and therefore less thick, resulting in attenuated post-SAVR BSH configuration and improved contraction of the basal IVS (**Figure 1D**). These potential effects of SAVR on BSH may not be expected from TAVR, given that TAVR does not involve wall shortening. We therefore hypothesized that SAVR as opposed to TAVR may potentially improve associated BSH configurations and its reduced contraction in patients with AS. The purpose of this study was to test the hypothesis using comprehensive echocardiography with speckle tracking analysis in patients undergoing SAVR or TAVR for AS. This is important because post-procedural LVOT obstruction following SAVR or TAVR and superior displacement of implanted prosthetic valve following TAVR are significant problems,³⁻⁵ and data from this study may offer useful information on SAVR or TAVR and facilitate choice of concomitant procedures for BSH at the time of SAVR for treating AS.

Methods

Subjects

Consecutive patients with SAVR or transfemoral TAVR were retrospectively enrolled at the echocardiographic laboratories of 4 collaborating institutions: University of Occupational and Environmental Health (n=34); Asan

Medical Center (n=4); Sakakibara Heart Institute of Okayama (n=12); and Kokura Memorial Hospital (n=18). All patients had significant AS, defined as aortic valve area $<1.0\text{ cm}^2$ on the continuity equation. Patients with SAVR did not undergo concomitant myectomy or myotomy. Patients with transapical TAVR were not included due to the potential influences on LV contraction. Exclusion criteria were (1) concomitant other structural heart disease; and (2) inadequate echocardiography imaging. From a pool of subjects undergoing clinically indicated echocardiography, 20 healthy subjects were randomly selected for the controls. Consequently, 32 patients who underwent SAVR (16 men; mean age, 72 ± 11 years) and 36 who underwent transfemoral TAVR (14 men; mean age, 85 ± 5 years) were included. Post-SAVR and post-TAVR echocardiography was performed 113 ± 147 and 113 ± 142 days after the procedure, respectively. Images were analyzed by a single physician (H.Y.) with 20 years' experience in echocardiography. This study was approved by the ethics committee of each institution, and informed consent was obtained from all subjects.

General Echocardiography

Comprehensive echocardiography was performed using commercially available equipment (iE-33, Philips Medical Systems, Andover, MA USA; Vivid 7, GE Medical Systems, Milwaukee, WI, USA; Artida, Toshiba Medical Systems, Otawara, Japan). Pre- and post-procedural echocardiography was performed using the same scanner from the same vendor to minimize measurement differences by vendors.⁸ In order to avoid error in measuring stroke volume in the

Table 1. Subject Clinical Characteristics

Variable	AS		Control (n=20)
	SAVR (n=32)	TAVR (n=36)	
Clinical			
Age (years)	72±11*. [†]	85±5*	28±9
Male	16 (50)	14 (39)	11 (55)
BSA (m²)	1.5±0.1*. [†]	1.4±0.2*	1.6±0.2
SBP (mmHg)	137±21*	131±18*	118±10
DBP (mmHg)	74±13	63±10	67±8
Heart rate (beats/min)	69±13	64±9	64±7
Hypertension	25 (78)	28 (78)	0 (0)
Hyperlipidemia	13 (41)	11 (31)	0 (0)
Diabetes mellitus	8 (25)	12 (33)	0 (0)

Data given as n (%) or mean±SD. *P<0.05 vs. control, †vs. TAVR. AS, aortic stenosis; BSA, body surface area; DBP, diastolic blood pressure; SAVR, surgical aortic valve replacement; SBP, systolic blood pressure; TAVR, transcatheter aortic valve replacement.

presence of BSH, LV end-diastolic and end-systolic volumes (LVEDV and LVESV) were measured using the Simpson biplane method to derive ejection fraction and stroke volume.⁹ The aortic valve area was then calculated using the continuity equation with continuous wave Doppler. In the apical long axis view, LVOT flow velocity was obtained on pulse wave Doppler by placing the sample volume at the center. From the velocity image, acceleration time (AT), deceleration time (DT), ejection time (ET), peak velocity, and velocity-time integral were measured. Given that LV outflow ET is influenced by heart rate, AT, DT and ET were corrected for heart rate using a correction formula: corrected AT (ATc)=AT/√(R-R interval); corrected DT (DTc)=DT/√(R-R interval); and corrected ET (ETc)=ET/√(R-R interval). Images with regular R-R intervals in the 2 preceding cardiac cycles were used for measurements in patients with atrial fibrillation.¹⁰

BSH-Specific Echocardiography

In apical 4-chamber view, base to apex IVS, as well as the free lateral wall, were trisected respectively into a total of 6 segments. Similarly, base to apex anterolateral and inferior walls were trisected in apical 2-chamber view and posterior and anteroseptal walls were trisected in apical long axis view. Segmental wall thickness was measured in the midportion of each segment in end-diastole in these 18 segments and averaged. Basal IVS/average LV wall thickness ratio was calculated as an index to express BSH (**Figure 1B**). BSH was defined as basal IVS/average LV wall thickness ratio >1.05 (upper normal range in the control group). LV mass was measured at end-diastole as (LV epicardial volume–LV endocardial volume)×1.05 using the area-length method.¹¹ The right ventricular side line of the basal IVS was determined. The anterior aortic root line, including the anterior annulus of the aortic valve and anterior sinotubular junction, was also determined. The IVS-aorta angle was defined as the angle between the 2 lines in parasternal or apical long axis view (**Figure 1B**).¹² LV longitudinal strain of 18 segments was measured on speckle tracking analysis using EchoPac PC BT12, GE Healthcare, QLAB 9, Philips Medical Systems, or 2D Wall Motion Tracking, Toshiba Medical Systems. Global LV longitudinal strain was calculated as the average strain of the 18 segments. The ratio of basal IVS strain to global LV

longitudinal strain was obtained as an index to express segmental dysfunction in the region of BSH.¹ As opposed to tissue Doppler-derived strain, the speckle tracking method has the merit of angle independency.

Statistical Analysis

Categorical variables are presented as frequencies and continuous variables as mean±SD. Differences between proportions were assessed using Fisher exact test. Unpaired continuous variables were compared using the unpaired t-test or Mann-Whitney U-test according to the data distribution. Pre- and post-procedure results were compared using paired t-test. Quantitative results in 3 groups were analyzed with 1-way ANOVA and post-hoc Tukey test. Spearman correlation coefficient was used to investigate the relationship between echocardiography measurements. P<0.05 was considered significant.

Interobserver variability for the measurements of longitudinal strain was obtained by analysis of measurements in 10 randomly selected patients by 2 independent blinded observers. Intraobserver variability was evaluated by analysis of measurements in the other 10 patients by the same observer at 2 different time points. Results were analyzed using both the least squares fit linear regression analysis and the Bland-Altman method.

Results

Clinical Characteristics

Patient clinical and echocardiographic characteristics are summarized in **Tables 1,2**. As expected, patients with SAVR were younger and physically larger compared with those with TAVR. Indexed LVEDV, LVESV were larger, LV ejection fraction was not augmented and LV stroke volume was similar in patients with SAVR compared with those with TAVR. After AVR, indexed LVEDV and LVESV were reduced in SAVR, but not in TAVR.¹³ Preoperative LVOT peak flow velocity and velocity-time integral tended to be or were significantly increased in both types of AS patients compared with controls, potentially reflecting accelerating flow with protrusion of BSH toward LV outflow. Post-procedural increase in peak velocity, potentially due to attenuation of pre-procedural lengthened ET with AS, tended to be less in patients with SAVR

Table 2. Echocardiographic Data Before and After SAVR/TAVR

	SAVR (n=32)		TAVR (n=36)		Control (n=20)
	Before	After	Before	After	
AS severity					
AVA (cm ²)	0.8±0.2	1.4±0.5 [§]	0.7±0.1	1.8±0.5 [§]	
Mean PG (mmHg)	52±16	20±10 [§]	50±14	10±4 [§]	
LVOT flow					
Peak velocity (cm/s)	100±31	108±28*	100±16	111±18* [§]	88±18
VTI (cm)	22±6*	20±5*	24±4*	23±3*	18±4
Corrected acceleration time (ms)	152±32* [†]	118±25* [§]	136±23*	108±27* [§]	92±17
Corrected deceleration time (ms)	198±40*	190±29*	205±34	210±53	226±13
Corrected ejection time (ms)	346±43	304±41 [§]	342±33	318±34 [§]	323±31
LVEDVI (mL/m ²)	65±28* [†]	55±22 [§]	50±17	49±14	55±22
LVESVI (mL/m ²)	29±24* [†]	22±17 [§]	16±12	14±8*	20±4
LVEF (%)	61±19 [†]	63±17	71±12*	73±10*	64±6
LVSVI (mL/m ²)	36±13	32±10	35±8	35±7	35±8
Indexed LV mass (g/m ²)	97±22* [†]	91±20* [§]	81±18*	75±15* [§]	49±9
Average LV wall thickness (mm)	12.1±1.4*	11.5±1.4* [§]	12.0±2.1*	11.4±2.0* [§]	6.9±0.9
Basal IVS wall thickness (mm)	13.5±3.5*	12.2±2.6* [§]	13.3±2.8*	12.7±2.9* [§]	6.8±1.1
Basal IVS/average LV wall thickness ratio	1.11±0.24*	1.06±0.17 [§]	1.11±0.17*	1.12±0.17*	0.99±0.03
Incidence of BSH	12/32	10/32	17/36	18/36	
IVS-aorta angle (°)	115±22*	123±14* [§]	116±17*	116±17*	147±8
Global LV longitudinal strain (%)	-12.9±3.9	-13.9±3.2	-13.8±3.2	-13.8±2.9	-20.2±1.7
Basal IVS strain (%)	-6.2±5.7	-9.1±5.2 [§]	-6.0±6.2	-5.9±5.8	-17.5±2.9
Basal IVS/global LV strain ratio (%)	42±58*	66±38* [§]	41±47*	41±42*	87±12

Data given as mean±SD or n. *P<0.05 vs. control, [†]vs. TAVR, [§]vs. pre-procedural value. AVA, aortic valve area; BSH, basal interventricular septal hypertrophy; HR, heart rate; IVS, interventricular septum; LV, left ventricle; LVEDVI, indexed left ventricular end-diastolic volume; LVEF, left ventricular ejection fraction; LVESVI, indexed left ventricular end-systolic volume; LVOT, left ventricular outflow tract; LVSVI, indexed left ventricular stroke volume; PG, pressure gradient; VTI, velocity-time integral. Other abbreviations as in Table 1.

Figure 2. Correlation between pre-procedural morphological degree of BSH and basal IVS strain. Greater morphological abnormalities in BSH were associated with greater reduction in basal IVS strain. Abbreviations as in Figure 1.

compared with TAVR. Velocity-time integral tended to reduce after procedure in patients with SAVR while it was constant in those with TAVR. These differences in LVOT velocity, however, were not statistically significant. AS patients who subsequently underwent either SAVR or TAVR had significantly increased basal IVS thickness, increased basal IVS/average LV wall thickness ratio,

reduced IVS-aorta angle, and reduced basal IVS/global LV strain ratio compared with controls (P<0.01, respectively; **Table 2**) but without significant differences between the 2 groups. Both types of AS groups had reduced basal IVS strain compared with controls, but statistical comparison was not carried out due to potential errors in vendor differences. Twelve patients with SAVR and 17 with TAVR

Figure 3. Correlations between improvements in the basal IVS strain after SAVR and pre-SAVR degree or post-SAVR change in BSH morphology. Greater post-SAVR improvement in basal IVS strain was associated with greater pre-SAVR BSH and greater post-SAVR improvement of BSH morphology. Abbreviations as in Figure 1.

met BSH criteria ($P=NS$, **Table 2**). The incidence of BSH tended to be reduced after SAVR but the reduction was not significant. Reduced basal IVS strain was correlated with increased thickness of the region and with decreased IVS-aorta angle ($r=0.66$, $P<0.01$; $r=0.56$, $P<0.01$, respectively; **Figure 2**).

Changes in Basal IVS Thickening and Reduced Contraction

All patients had improvement in aortic valve function and symptoms after SAVR or TAVR (**Table 2**). While LVEDV significantly decreased after SAVR ($P<0.01$), it was not changed after TAVR. Indexed LV mass was significantly reduced after the procedure in both groups ($P<0.01$). Following SAVR, average LV wall thickness decreased, basal IVS thickness showed greater reduction, resulting in reduced basal IVS/average LV wall thickness ratio ($P<0.01$). In contrast, average LV wall thickness and basal IVS thickness had similar reduction after TAVR, resulting in no significant reduction in the ratio. IVS-aorta angle significantly increased after SAVR ($P<0.01$), while it remained unchanged following TAVR. Although global LV longitudinal strain did not change after SAVR, basal IVS strain and basal IVS/global LV strain ratio significantly improved following SAVR ($P<0.01$). In contrast, all global LV longitudinal strain, basal IVS strain, and their ratio did

not change after TAVR. In general, basal IVS thickening and reduced contraction significantly improved after SAVR while these did not improve after TAVR.

Improvement in basal IVS strain after SAVR was correlated with pre-SAVR basal IVS thickening (**Figure 3, Upper left**), its post-SAVR reduction (**Figure 3, Lower left**), pre-SAVR reduced IVS-aorta angle (**Figure 3, Upper right**), and its post-SAVR increase (**Figure 3, Lower right**). Therefore, greater post-SAVR improvements in basal IVS strain were associated with greater pre-SAVR BSH morphology and its greater post-SAVR improvement.

BSH: Changes in Basal IVS Thickening and Reduced Contraction

In general, BSH indices similarly improved only after SAVR in 12 patients with BSH (**Table 3**). Improvements in BSH after SAVR tended to be greater in selected patients with pre-procedural BSH compared with the whole group of patients (reduction in basal IVS thickening: -2.7 ± 1.5 vs. -1.3 ± 1.6 mm; improvement in basal IVS contraction: -5.1 ± 3.8 vs. $-3.0\pm4.0\%$; improvement in IVS-aorta angle: 18 ± 12 vs. 8.1 ± 10.8).

Two Representative Patients

The patient in **Figure 4** had severe AS and associated BSH

Table 3. Echocardiographic Data Before and After SAVR/TAVR in Patients With Pre-Procedural BSH				
	SAVR (n=12)		TAVR (n=17)	
	Before	After	Before	After
AS severity				
AVA (cm ²)	0.8±0.2	1.4±0.4*	0.7±0.2	1.7±0.5*
Mean PG (mmHg)	52±9	20±9*	44±11	10±3*
LVEDVI (mL/m ²)	58±28	51±17	52±20	50±16
LVESVI (mL/m ²)	25±28	19±16	15±15	14±10
LVEF (%)	64±20	65±18	74±12	74±11
LVSVI (mL/m ²)	33±9	31±9	37±8	35±8
Indexed LV mass (g/m ²)	91±12	84±15*	85±22	78±18*
Average LV wall thickness (mm)	12.5±1.6	11.6±1.3*	11.5±1.3	10.9±1.4*
Basal IVS wall thickness (mm)	16.8±3.2	14.1±2.5*	14.3±2.6	13.7±3.0*
Basal IVS/average LV wall thickness ratio	1.35±0.21	1.22±0.18*	1.24±0.15	1.24±0.16
IVS-aorta angle (°)	90±14	108±11*	104±16	103±16
Global LV longitudinal strain (%)	−12.5±4.7	−13.4±3.7	−14.2±3.4	−14.0±2.6
Basal IVS strain (%)	−1.2±5.1	−6.3±4.9*	−3.2±7.1	−3.5±7.2
Basal IVS/global LV strain ratio (%)	−4.4±7.0	46±31*	16±5.1	20±5.1

Data given as mean ± SD. *P<0.05 vs. pre-procedural value. Abbreviations as in Tables 1,2.

as shown by basal IVS thickening (white arrows, left pre-SAVR panel), reduced IVS-aorta angle (α in the left pre-SAVR panel) and reduced strain of the region (blue signals pointed by white arrows, right pre-SAVR panel) and subsequently underwent SAVR. Post-SAVR echocardiography showed clear improvements in basal IVS thickness (yellow arrows, left post-SAVR panel), IVS-aorta angle (α in the left post-SAVR panel) and strain of the region (pink signals pointed by yellow arrows, right post-SAVR panel). Morphological and functional improvements of the BSH are shown in **Movie S1**.

The patient in **Figure 5** had severe AS and associated BSH as shown by basal IVS thickening, reduced IVS-aorta angle, and reduced basal IVS strain (white arrows and

angle α in the upper pre-TAVR panel and blue strain signals in the lower pre-TAVR panel, respectively) and subsequently underwent TAVR. In contrast to the patient with SAVR, post-TAVR echocardiography in this patient did not show clear improvements in these BSH indices. No clear morphological or functional improvements of the BSH are shown in **Movie S2**.

Measurement Variability

Good correlations were observed in inter- and intraobserver variability of the LV global longitudinal strain ($r=0.97$ and 0.93). On Bland-Altman analysis, inter- and intraobserver variability were 4.9% and 6.8%, respectively.

Figure 5. Representative transcatheter aortic valve replacement (TAVR). Before TAVR, this patient had severe aortic stenosis and associated BSH, as shown by the increased wall thickness in the basal IVS, reduced IVS-aorta angle and reduced basal IVS contraction (white arrows and angle α in the upper pre-TAVR panel and blue strain signals in the lower pre-TAVR panel, respectively). In contrast to the patient with SAVR in Figure 4, post-TAVR echocardiography in this patient did not show clear improvements in basal IVS thickness, IVS-aorta angle or basal IVS strain (yellow arrows and angle α in the upper post-TAVR panel and blue strain signals in the lower post-TAVR panel, respectively). Abbreviations as in Figures 1,4.

Discussion

This study has shown that abnormalities in morphology and contraction of basal IVS improve after SAVR but not after TAVR in patients with AS. Although both SAVR and TAVR are highly effective in improving aortic valve function,^{14,15} different effects of SAVR and TAVR on regions other than aortic valve have not been previously evaluated. The present study has demonstrated the beneficial effects of SAVR, as opposed to TAVR, on associated basal IVS abnormality, and may provide a unique perspective on the evaluation of different effects of SAVR and TAVR, which may in turn facilitate the choice of SAVR and TAVR and that of concomitant procedures for BSH at the time of SAVR in patients with AS. Compression of the LV segments may reduce pre-load (end-diastolic segment length) and its contraction according to the Frank-Starling law.¹⁶ We consider that inferiorly and posteriorly directed compression of the basal IVS from a longitudinally elongated ascending aorta may lead to reduced contraction on the segment. Beneficial influence of SAVR on basal IVS strain did not lead to improved global LV functional indices such as global LV longitudinal strain, ejection fraction or stroke volume. The reason was not able to be clarified in the present study, but preoperative compensatory mechanism of other LV segments for basal IVS dysfunction may be attenuated after surgery, resulting in similar function of global LV after surgery.

Previous Investigations and Clinical Implications

High incidence of BSH and reduced contraction in the region of BSH in patients with AS have been reported,¹ consistent with the present findings. The present study further suggested a beneficial influence of conventional

SAVR on something other than the aortic valve itself: the associated BSH.

The main invasive procedures for symptomatic AS are SAVR and TAVR. SAVR is a safe and effective procedure and has a long history and established data, including data on improved long-term durability of prosthetic valve;¹⁷ it requires thoracotomy and the use of extracorporeal cardiopulmonary pump. TAVR is also a safe and effective procedure, and is less invasive, given that it does not involve the use of cardiopulmonary pump, and thoracotomy is also not necessary for transfemoral TAVR,¹⁸ which is the major approach and was the target of this study. TAVR is a relatively recent innovation with excellent outcomes and satisfactory intermediate-term durability of the implanted prosthetic valve.¹⁸ Therefore, both SAVR and TAVR are established treatments for AS. The present study has demonstrated the beneficial influences of SAVR on regions other than the aortic valve itself, that is, BSH in this case. BSH is a potential risk for post-procedural LVOT obstruction following both SAVR and TAVR with unfavorable outcome,³⁻⁵ and a potential risk for superior displacement of implanted prosthetic valve at the time of TAVR.¹⁹ Information derived from this investigation will facilitate decision-making with regard to SAVR and TAVR.

One of the additional merits of SAVR is its availability for concomitant procedures, such as coronary artery bypass grafting or mitral valve surgery. When BSH is associated with AS, myectomy in addition to SAVR is often performed, which reduces basal IVS thickening and confers other advantages.²⁰ In the present study, SAVR with aortic wall shortening mainly reduced basal IVS thickening. Reduction of basal IVS thickening by SAVR in the present study was -1.3 ± 1.6 mm in the whole group of 32 patients, and -2.7 ± 1.5 mm in the 12 patients with significant BSH.

This reduction of basal IVS thickening was similar or even greater to that by concomitant myectomy, with a mean reduction of -0.7 to -2.0 mm.^{20,21} Therefore, in the case of AS with BSH, this study may offer useful information on concomitant procedures.

BSH has been described as a phenotype of hypertrophic cardiomyopathy, but it is still controversial as to whether BSH with its low prevalence of gene abnormalities represents primary cardiomyopathy or not.²² We have conducted the present study based on the proposed mechanism of BSH as a secondary compression of the IVS by a longitudinally elongated ascending aorta. Although the mechanism of BSH is not yet established, morphological and functional improvement of BSH after SAVR and the lack of benefits of TAVR in the present study suggest that the proposed mechanism may be present in patients with AS and associated BSH. To confirm the proposed mechanism of BSH, further studies involving computational finite element modeling and others are required.²³ Confirmation of the proposed mechanism, however, is not necessary for the validity of the present findings. This study has demonstrated the beneficial effects of SAVR on associated BSH configurations and function and the lack of such effects for TAVR, which might be useful for decision-making.

Study Limitations

This study has demonstrated the beneficial effects of SAVR on BSH, but morphological and functional abnormalities in the region of BSH still remained after the procedure. Even after SAVR, basal IVS thickness was greater and basal IVS/global LV strain ratio was reduced compared with other segments ($P < 0.01$, respectively), and IVS-aorta angle was also smaller than that of normal controls ($P < 0.01$). The length of the prosthetic vessel used to replace the aneurysmal aorta is often considerably shorter than that of the resected aneurysm.²⁴ Aortic wall shortening of 1.0 cm only in the anterior wall in the present study may not fully correct the whole aortic wall elongation. Individually tailored and whole, as opposed to only anterior, aortic wall shortening based on the evaluation of whole wall elongation seems preferable. The present results may promote such surgical strategies. Regarding the surgical choice of aortic wall shortening, septal myectomy/myotomy and a combination of these, we consider that the following strategy is an option. Before cardiopulmonary bypass and aortotomy, intraoperative procedure by a surgeon to shift the ascending aorta superiorly and anteriorly may straighten deformed IVS. We have seen intraoperative transesophageal echocardiographic observation of BSH improvement during such a procedure by an expert surgeon. Significant reduction in basal IVS thickening after SAVR in this study suggests that BSH may include pseudohypertrophy, and sufficient aortic wall shortening may totally normalize basal IVS thickness without the need for myectomy/myotomy. Insufficient improvement in BSH may indicate the need for additional myectomy/myotomy. After cardiopulmonary bypass and the recovery of heart beat, intraoperative evaluation of BSH on transesophageal echocardiography is also useful to clarify the need for additional procedures. Effects of SAVR and TAVR on LVOT flow profile could be different; in particular, a skewed and accelerated spatial velocity profile due to protruding BSH may be specifically improved after SAVR. In the present study, post-SAVR LV outflow velocity tended to be reduced compared with post-TAVR velocity but not significantly so. Accurate

evaluation of the effects requires flow velocity mapping in the entire LVOT. Effects of SAVR and TAVR on the associated BSH were evaluated several months after, and relatively early after, the procedure. Longer term effects need to be further evaluated. Additional effects of SAVR on BSH were evaluated but the influences of these effects on patient outcome were not evaluated. Functional improvement in basal IVS was evaluated with longitudinal strain in this study. Ideally speaking, all longitudinal, circumferential and radial strains are important, which requires analysis in the short axis view. Due to the deformity of basal IVS, analysis in the short axis view is difficult. In other studies of BSH, functional evaluation was also performed mainly using longitudinal strain with analysis of apical views.¹ LVOT obstruction is an important complication after SAVR and TAVR, and BSH is a potential risk for this obstruction.³⁻⁵ Attenuated BSH, especially with more aggressive aortic wall shortening, by SAVR may have favorable effects on postoperative LVOT obstruction, but this was not evaluated in the present study.

Conclusions

In patients with AS, SAVR, as opposed to TAVR, improves associated BSH and its functional impairment. This may be useful for decision-making with regard to SAVR and TAVR.

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Disclosures

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Supplementary Files

Supplementary File 1

Movie S1. Pre- and postoperative echocardiography in a patient who underwent surgical aortic valve replacement (SAVR) for aortic stenosis. Preoperatively, this patient had associated basal interventricular septal hypertrophy (BSH) as shown by the increased wall thickness in the basal interventricular septum (IVS), reduced IVS-aorta angle and reduced strain in the basal IVS (blue signals). After SAVR, echocardiography showed clear improvements in both basal IVS thickness, IVS-aorta angle and the basal IVS strain (pink signals). LA, left atrium; LV, left ventricle.

Supplementary File 2

Movie S2. Pre- and postoperative echocardiography in a patient who underwent transcatheter aortic valve replacement (TAVR) for aortic stenosis. Preoperatively, this patient had associated BSH, as shown by the increased wall thickness in the basal IVS, reduced IVS-aorta angle and reduced strain in the basal IVS (blue signals). After TAVR, echocardiography did not show clear improvements in basal IVS thickness, IVS-aorta angle or basal IVS strain. Abbreviations as in Movie S1.

Please find supplementary file(s);
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