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ORIGINAL RESEARCH

Impact of Left Ventricular Diastolic Pressure Changes on Clinical Outcomes After Transcatheter Aortic Valve Replacement

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BACKGROUND: Changes in left ventricular (LV) diastolic pressure after transcatheter aortic valve replacement (TAVR) or their relationship with subsequent outcomes remain poorly clarified. Accordingly, we aimed to assess the changes in invasively measured LV diastolic pressure and their relationship with long-term outcomes in patients undergoing TAVR.

METHODS: In total, 509 patients with severe aortic stenosis who underwent TAVR at 3 tertiary centers were retrospectively included and divided into 2 groups according to changes in LV pre-A pressure after TAVR: Group 1, with no change or decrease in pre-A pressure, and Group 2, presenting an increase in pre-A pressure after TAVR. The primary outcome was a composite of all-cause death and rehospitalization for heart failure.

RESULTS: Group 1 included 39% (n=198) patients, and Group 2 had 61% (n=311) patients. More patients in Group 2 had diabetes, chronic kidney disease, and a larger aortic valve area than in Group 1. During the follow-up period (median, 28 months), 122 primary outcomes were recorded. In Kaplan-Meier analysis, the cumulative incidence of the primary outcome and all-cause death was significantly lower in Group 1 than in Group 2. In multivariable Cox hazard models, Group 1 was independently associated with a favorable primary outcome (hazard ratio, 0.52 [95% CI, 0.34–0.80]; *P*=0.003).

CONCLUSIONS: Increase in LV pre-A pressure after TAVR is common, and no change or decrease in LV pre-A pressure after TAVR is independently associated with favorable outcomes. Changes in LV pre-A pressure can help identify patient subsets who will maximally benefit from TAVR.

Key Words: aortic stenosis ■ diastolic function ■ transcatheter aortic valve replacement

Aortic valve stenosis (AS) triggers chronic left ventricular (LV) pressure overload, which leads to myocardial hypertrophy, fibrosis, and subendocardial ischemia,[1,2] subsequently resulting in diastolic dysfunction and increased LV filling pressure. These processes are common and critical in patients with severe AS,[3] because they are associated with the development of dyspnea and affect long-term outcomes.[3,4] Transcatheter aortic valve replacement (TAVR), an established treatment, can effectively eliminate pressure gradients across the stenotic aortic valves and improve LV hypertrophy, diastolic dysfunction, and increased filling pressure. TAVR improves LV function and induces substantial improvements in LV diastolic function and the New York Heart

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CLINICAL PERSPECTIVE

What Is New?

- An increase in left ventricular pre-A pressure immediately after transcatheter aortic valve replacement has been frequently observed.
- A reduction in left ventricular pre-A pressure following transcatheter aortic valve replacement is independently associated with improved long-term clinical outcomes.

What Are the Clinical Implications?

- Emphasis should be placed on left ventricular myocardial evaluation to identify individuals who could benefit most from transcatheter aortic valve replacement.
- An increase in left ventricular pre-A pressure after transcatheter aortic valve replacement may serve as an early indicator for the need for more intensive postprocedural monitoring, helping to identify patients at higher risk of adverse events.

Nonstandard Abbreviations and Acronyms

AS	aortic valve stenosis
STS	Society of Thoracic Surgeons
TAVR	transcatheter aortic valve replacement

Association functional class during follow-up.[5] Although the importance of preprocedural diastolic dysfunction in patients who underwent TAVR has been emphasized,[4] changes in LV diastolic dysfunction after TAVR and its relationship with subsequent outcomes remain poorly clarified. Data on invasive intraprocedural hemodynamic assessments and their effects on the outcomes of patients undergoing TAVR are limited. Therefore, we aimed to analyze changes in invasively measured LV diastolic pressure and the association between changes in LV diastolic pressure and clinical outcomes in patients undergoing TAVR.

METHODS

All data used during this study will be available from the corresponding author under reasonable request.

Study Population

This multicenter observational retrospective study was conducted at 3 tertiary medical centers in Korea. Patients who underwent TAVR between 2010 and 2020 were enrolled. Patients with unsuccessful TAVR (n=45), insufficient pre- and postprocedural LV or aortic pressure (n=484), insufficient clinical data (n=41), and additional emergent procedures during or immediately after TAVR (n=27) were excluded. In total, 509 patients were included in the final analysis (Figure 1). Patients were divided into 2 groups according to changes in LV pre-A pressure after TAVR: Group 1, no change or decrease in LV pre-A pressure after TAVR; and Group 2, increase in LV pre-A pressure after TAVR. Figure 2 shows the representative LV and aortic pressure curves pre- and post-TAVR in the 2 groups. This study was approved by the institutional review board of Yonsei University Health System (approval number: 4-2019-0758) and complied with the Declaration of Helsinki. Because this was a registry-based retrospective study, and the data were analyzed anonymously, informed consent from the study participants was not required.

TAVR Procedure and Assessment of LV Diastolic Pressure

Participants were implanted with commercially available self-expanding (CoreValve and Evolut R system; Medtronic, Minneapolis, MN), balloon-expandable (SAPIEN XT and SAPIEN 3; Edwards Lifesciences, Irvine, CA), or mechanically expandable (Lotus Valve; Boston Scientific, Marlborough, MA) valves. A transfemoral approach was used in all cases. Intraprocedural rapid ventricular pacing was used to ensure a transient cardiac standstill during valve positioning and deployment. Pre- or postdilatation with rapid ventricular pacing was performed in selected patients according to the valve anatomy and function on intraprocedural echocardiography. Two pigtail catheters with multiple side holes were placed in the left ventricle and ascending aorta to simultaneously record pressures. LV and aortic pressures were obtained for at least 5 seconds to ensure steady-state conditions.[6,7] LV and aortic pressures, including LV minimum, LV peak, LV pre-A, LV end-diastolic pressure, aortic systolic pressure, and aortic diastolic pressure, were measured at end-expiration. In patients with atrial fibrillation, pre-A pressure was estimated at the point of the plateau before the LV end-diastolic pressure. LV pre-A pressure, the LV diastolic pressure immediately before atrial contraction, was used as a representative value for LV diastolic pressure in the study. This was because LV pre-A pressure is the most correlated with mean left atrial pressure among LV diastolic pressures. Changes in LV pre-A pressure were calculated by subtracting the preprocedure LV pre-A pressure from the postprocedure LV pre-A pressure.

Echocardiography and Follow-Up and Outcomes

All study participants underwent transthoracic echocardiography within 3 months before TAVR. Echocardiographic measurements were obtained using

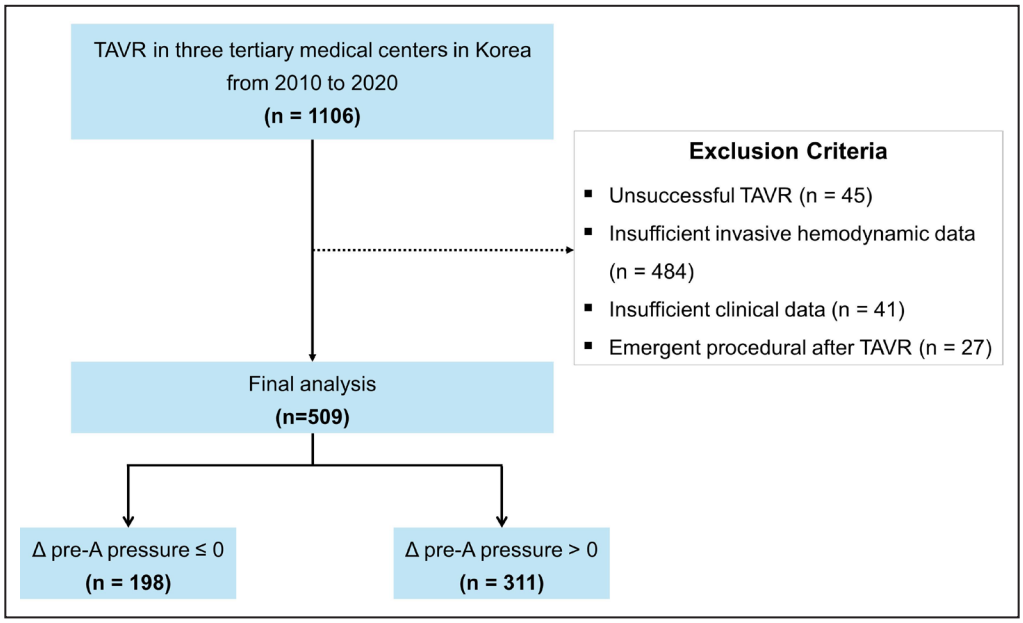

Figure 1. Flow diagram for study patient selection.
TAVR indicates transcatheter aortic valve replacement.

standard methods.[8,9] Speckle tracking echocardiography was performed to measure LV global longitudinal strain.[10] Speckle tracking echocardiography analysis was performed offline using customized software (TomTec-ARENA, version TTA 2.31; Munich, Germany).

All patients were scheduled to visit the outpatient clinic regularly after TAVR. Primary outcomes were defined as a composite of all-cause death and hospitalization for heart failure during the follow-up period. Hospitalization for heart failure was defined as an unexpected presentation to an acute care facility requiring overnight hospitalization for 2 of the following 3 reasons: (1) signs or symptoms consistent with heart failure, (2) radiological or echocardiographic evidence of worsening heart failure, and (3) requiring intravenous diuretics or inotropes or mechanical fluid removal. All clinical characteristics and parameters were obtained by reviewing electronic medical records. Two researchers independently analyzed clinical events, and the occurrence of primary outcomes was determined by agreement between the researchers (J.S. and A.-R.K.).

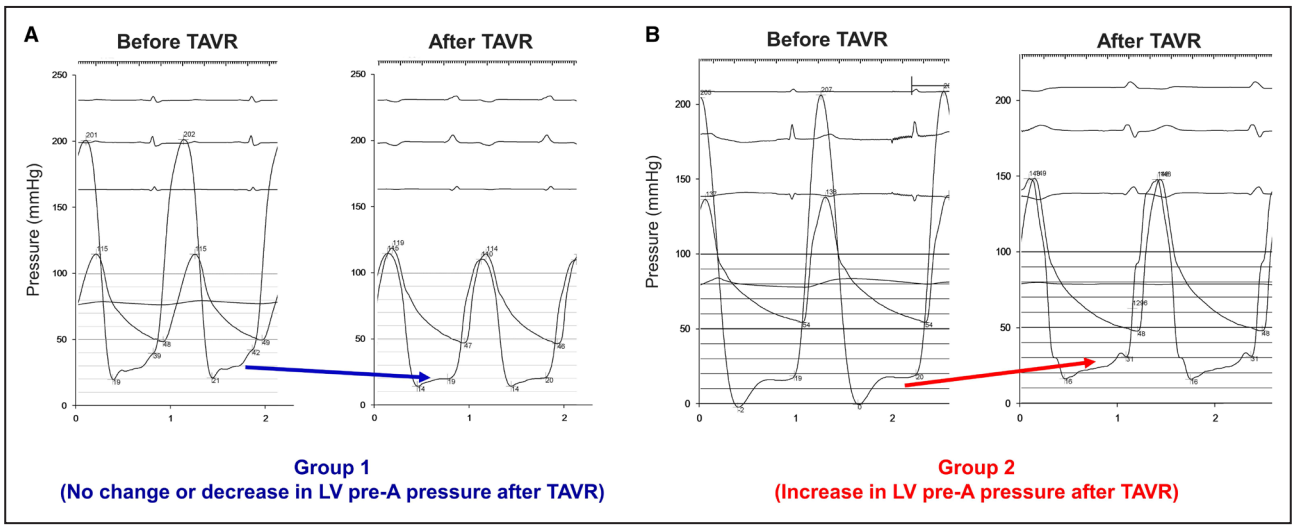

Figure 2. Representative case.
Representative LV and aortic pressure curves before and after TAVR. **A**, Decreased average LV diastolic pressure after TAVR. **B**, Elevated average LV diastolic pressure after TAVR. LV indicates left ventricular; and TAVR, transcatheter aortic valve replacement.

Statistical Analysis

Continuous data are expressed as mean±SD, and categorical data are expressed as number and percentage for each group. Continuous variables were compared between the groups using the Student *t* test; categorical variables were compared using the χ^2 test or Fisher exact test. The Cox proportional hazards assumption was assessed using Schoenfeld residuals. Kaplan-Meier analysis and Cox proportional hazards models were used to assess the associations between changes in LV pre-A pressure and clinical outcomes. Variables with a *P* value <0.1 and clinically important variables were included as potential predictors of primary outcome in a multivariable Cox model. Pearson correlation coefficient was used to assess the association between LV pre-A pressure and echocardiographic parameters, including LV ejection fraction (LVEF) and LV mass index. All tests were 2-sided, and statistical significance was defined as $P<0.05$. Data analyses were performed using R statistical software (version 3.6.0; R Foundation for Statistical Computing, Vienna, Austria).

RESULTS

Baseline Characteristics and Changes in LV Diastolic Pressures

The mean patient age was 81.2±5.4 years, and 243 (48%) were men. AF was documented in 86 (17%) patients, and 257 (51%) were diagnosed with coronary artery disease, including 32 (6.3%) with myocardial infarction. The mean Society of Thoracic Surgeons (STS) score was 4.9±4.3; 284 (56.2%) patients had low risk (STS score <4), 154 (30.5%) had intermediate risk (STS score 4–8), and 67 (13.3%) had high risk (STS score ≥8). Among the 509 patients, 198 (39%) had unchanged or reduced LV pre-A pressure (Group 1), and 311 (61%) had elevated LV pre-A pressure (Group 2) after TAVR. Table 1 compares baseline characteristics and procedural data between the 2 groups. Diabetes was diagnosed more frequently in Group 2 than in Group 1 (36% versus 51%, $P=0.001$). Preprocedural LV minimum pressure, LV pre-A pressure, LV end-diastolic pressure, and LV peak systolic pressure were significantly higher in Group 1 than in Group 2. However, Group 2 had significantly higher postprocedural LV diastolic pressures than Group 1. Although preprocedural aortic systolic pressure was comparable between the 2 groups, postprocedural aortic systolic/diastolic pressure and pulse pressure were significantly lower in Group 1 than that in Group 2. Group 2 had a significantly higher mean change in pulse pressure (postprocedural pulse pressure–preprocedural pulse pressure) than Group 1 (5.0±22.2 versus 15.3±21.0 mm Hg, $P<0.001$). There were no significant differences in the use of pre- and postballoon dilatation or the requirement for ventricular pacing after valve implantation between the groups. Table 2 presents baseline echocardiographic parameters. AS was more severe (higher mean and peak pressure gradient, smaller aortic valve area, and higher LV mass index) in Group 1 than in Group 2. LVEF and LV global longitudinal strain were more impaired in Group 1 than in Group 2.

Clinical Outcomes

During the follow-up period (median, 28 months; interquartile range, 12.7–43.5 months), there were 122 primary outcomes. All-cause death occurred in 78 patients, and hospitalization for heart failure occurred in 44 patients. Kaplan-Meier curves showed a significant difference in both the primary outcome and all-cause death between the groups when divided by the change in LV pre-A pressure after TAVR (Figure 3). Table 3 shows the results of univariable and multivariable Cox proportional hazard analyses for the primary outcomes. Men, higher STS score, self-expandable system, impaired preprocedural LV global longitudinal strain, higher preprocedural LV mass index and mitral E velocity, larger aortic valve area, and increase in LV pre-A pressure (per 1 mm Hg) after TAVR (hazard ratio [HR], 1.05 [95% CI, 1.02–1.08]; $P=0.002$) were associated with primary outcomes. Group 1 (change in LV pre-A pressure after TAVR ≤0 mm Hg) was independently associated with the primary outcome (HR, 0.52 [95% CI, 0.34–0.80]; $P=0.003$) and all-cause death (HR, 0.54 [95% CI, 0.33–0.90]; $P=0.019$) in the multivariable proportional Cox regression analysis. The association between Group 1 and the risk of primary outcomes was consistent in most subgroups (Figure 4).

Factors Linked to Changes in LV Pre-A Pressure

After TAVR, LV minimal pressure, LV pre-A pressure, and LV end-diastolic pressure increased by an average of 3.6±6.6, 2.6±6.3, and 2.3±8.9 mm Hg, respectively. Changes in LV mass index, LVEF, Septal E/e' ratio, and aortic pulse pressure significantly correlated with changes in LV pre-A pressure after TAVR (Figure 5). Of the 509 patients, 448 underwent follow-up echocardiography 12 months post-TAVR. A 1-year follow-up comparison of echocardiographic parameters revealed that patients from Group 1 had significantly greater improvements in LVEF, greater regression in left ventricular mass index, and more improved Septal E/e' ratio compared with patients from Group 2 (Table 4).

DISCUSSION

Herein, our key findings were as follows: (1) Increased LV pre-A pressure immediately after TAVR was

Table 1. Baseline Characteristics According to Changes in LV Pre-A Pressure

Variables	Δ Pre-A pressure ≤0 (n=198)	Δ Pre-A pressure >0 (n=311)	*P* value
Demographic data			
Age, y	80.8±5.4	81.3±5.3	0.301
Male sex, n	97 (49.0%)	146 (46.9%)	0.719
Hypertension, n	165 (83.3%)	255 (82.0%)	0.788
Diabetes, n	72 (36.4%)	160 (51.4%)	0.001
Dyslipidemia, n	99 (50.0%)	166 (53.4%)	0.514
Chronic kidney disease, n	78 (39.4%)	153 (49.2%)	0.038
COPD, n	18 (9.1%)	21 (6.8%)	0.426
Atrial fibrillation, n	30 (15.2%)	56 (18.0%)	0.474
Coronary artery disease, n	101 (51.0%)	156 (50.2%)	0.924
STS score	5.1±4.7	4.9±4.0	0.630
Low	103 (52.6%)	181 (58.6%)	0.187
Intermediate	69 (35.2%)	85 (27.5%)	
High	24 (12.2%)	43 (13.9%)	
Preprocedural pressure			
LV minimal pressure, mm Hg	7.3±6.9	5.7±6.1	0.007
LV pre-A pressure, mm Hg	18.6±6.3	14.2±5.3	<0.001
LV end-diastolic pressure, mm Hg	26.2±8.9	21.0±7.6	<0.001
LV peak pressure, mm Hg	184.1±35.7	176.9±33.0	0.021
Aortic systolic pressure, mm Hg	132.7±27.3	129.0±24.6	0.120
Aortic diastolic pressure, mm Hg	55.3±12.3	54.3±11.1	0.312
Aortic pulse pressure, mm Hg	77.3±22.4	74.8±20.1	0.180
Peak to peak pressure gradient, mm Hg	51.4±29.3	47.9±27	0.166
Heart rate, bpm	64.9±13.8	64.1±11.5	0.484
Postprocedural pressure			
LV minimal pressure, mm Hg	7.6±6.3	11.3±7.2	<0.001
LV pre-A pressure, mm Hg	15.3±5.8	20.6±6.4	<0.001
LV end-diastolic pressure, mm Hg	21.6±7.4	27.6±8.0	<0.001
LV peak pressure, mm Hg	140.6±29.3	151.2±29.3	<0.001
Aortic systolic pressure, mm Hg	137.0±28.4	148.8±28.5	<0.001
Aortic diastolic pressure, mm Hg	54.7±10.9	58.7±12.8	<0.001
Aortic pulse pressure, mm Hg	82.3±23.5	90.1±23.3	<0.001
Heart rate, bpm	70.1±12.3	69.0±11.1	0.316
Procedural data			
Self-expandable system, n	76 (38.4%)	96 (30.9%)	0.028
Balloon-expandable system, n	118 (59.6%)	214 (68.8%)	0.028
Mechanically expandable system, n	5 (8.3%)	1 (1.1%)	0.035
Balloon predilatation, n	114 (57.6%)	182 (58.5%)	0.906
Balloon postdilatation, n	79 (39.9%)	118 (37.9%)	0.727
Postprocedural back-up pacing, n	20 (10.1%)	32 (10.3%)	0.516
Paravalvular leakage, n			0.568
No	105 (53%)	178 (57.2%)	
Mild	81 (40.9%)	111 (35.7%)	
Moderate	11 (5.6%)	18 (5.8%)	
Severe	1 (0.5%)	4 (1.3%)	
Immediate postprocedure CK-MB, ng/mL	10.8±20.7	9.4±7.8	0.389

Δ Pre-A pressure = post-TAVR LV pre-A pressure−Pre-TAVR LV pre-A pressure. CK-MB indicates creatine kinase-myocardial band; COPD, chronic obstructive pulmonary disease; LV, left ventricular; STS, Society of Thoracic Surgeons; and TAVR, transcatheter aortic valve replacement.

Table 2. Baseline Echocardiographic Characteristics According to Changes in LV Pre-A Pressure

Echocardiographic data	Δ Pre-A pressure ≤0 (n=198)	Δ Pre-A pressure >0 (n=311)	*P* value
LV end-diastolic diameter, mm	49.3±6.7	47.9±6.6	0.017
LV end-systolic diameter, mm	33.6±8.2	31.5±7.7	0.005
LV mass index, g/m^2	140.6±38.7	132.0±36.6	0.011
LV ejection fraction, %	58.5±13.2	61.2±11.9	0.016
LV GLS, %	−13.8±4.4	−15.3±5.0	0.001
LA volume index, mL/m^2	53.8±20.9	52.4±20.3	0.448
E velocity, cm/s	85.2±29.3	81.0±29.8	0.122
e′ velocity, cm/s	4.2±1.4	4.3±1.5	0.469
Septal E/e' ratio	22.1±9.5	19.9±7.9	0.008
Peak TR velocity, cm/s	2.7±0.5	2.7±0.5	0.217
AV peak systolic PG, mm Hg	91.9±30.0	87.7±29.4	0.126
AV mean systolic PG, mm Hg	55.6±19.3	52.6±18.3	0.082
Aortic valve area, cm^2	0.64±0.16	0.68±0.17	0.025
Indexed aortic valve area, cm^2/m^2	0.40±0.10	0.42±0.11	0.023
Moderate or severe MR	16 (8.1%)	24 (7.7%)	0.984
Moderate or severe TR	7 (3.5%)	20 (6.4%)	0.223

AV indicates aortic valve; E velocity: Early diastolic transmitral flow velocity; e' velocity: Septal early diastolic mitral annular velocity; GLS, global longitudinal strain; LA, left atrial; LV, left ventricular; MR, mitral regurgitation; PG, pressure gradient; and TR, tricuspid regurgitation.

common (61%). (2) Patients with decreased LV pre-A pressure after TAVR had higher preprocedural LV pre-A pressure and more severe AS. (3) Decreased LV pre-A pressure after TAVR was independently associated with favorable long-term outcomes. (4) Changes in LV pre-A pressure during TAVR significantly correlated with regression of LV mass index and improvement in LVEF and septal E/e′ ratio.

LV Diastolic Dysfunction and Outcomes After TAVR

Data on the association between LV diastolic dysfunction and prognosis after TAVR are insufficient and inconsistent. Reportedly, baseline LV diastolic dysfunction is substantially associated with the prognosis after TAVR.[4,11,12] In a retrospective study of 90 patients undergoing TAVR, baseline LV diastolic dysfunction was the most important echocardiographic factor associated with all-cause mortality at 1 year, whereas post-TAVR LV diastolic dysfunction or changes in diastolic dysfunction grade were not associated with clinical outcomes.[11] In a prospective cohort of 166 patients with AS, progressive stages of LV diastolic dysfunction were associated with increased mortality, irrespective of AS severity.[12] Conversely, no association between baseline LV diastolic dysfunction and survival was noted in a study involving 358 patients despite documented improvement in LV diastolic dysfunction during follow-up.[5] In our study, changes in LV pre-A pressure after TAVR, rather than preprocedural LV pre-A pressure, were independently associated with clinical outcomes, further supporting the importance of postprocedural LV diastolic function over baseline diastolic function after TAVR. Although LV diastolic dysfunction is expected to improve after TAVR, the degree of LV diastolic dysfunction remained unchanged after TAVR in more than half of the patients and even worsened in a notable subset of patients.[4,5,13] These findings are consistent with those of our study, revealing that

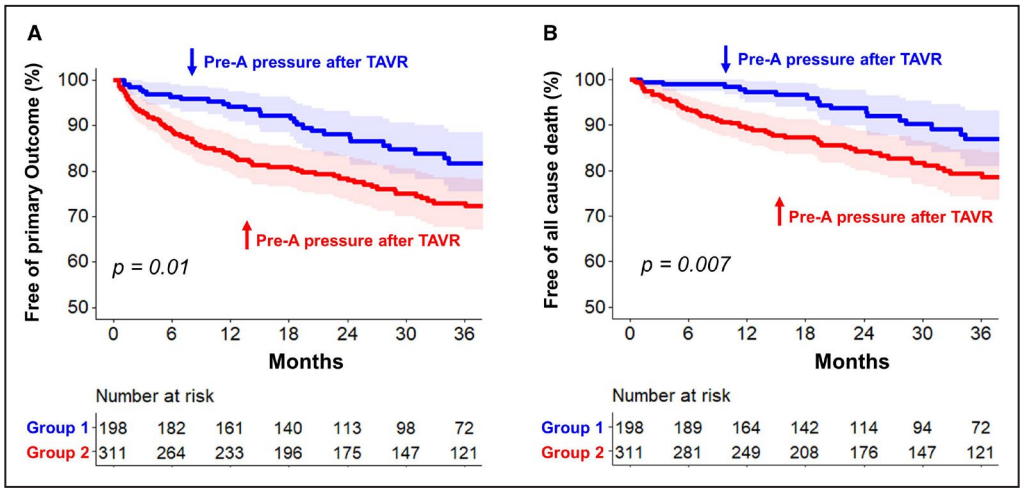

Figure 3. Kaplan-Meier survival curves.
Curve for primary outcome-free survival (**A**) and curve for all-cause death-free survival (**B**) according to changes in left ventricular diastolic pressure after TAVR. TAVR indicates transcatheter aortic valve replacement.

Table 3. Univariable and Multivariable Proportional Cox Regression Analyses of the Primary Outcome

Variables	Univariable		Multivariable	
	Hazard ratio (95% CI)	*P* value	Hazard ratio (95% CI)	*P* value
Age	1.03 (0.99–1.07)	0.103		
Male sex	1.60 (1.11–2.30)	0.011	1.61 (0.99–2.61)	0.055
Coronary artery disease	1.23 (0.86–1.76)	0.251		
Diabetes	1.35 (0.94–1.92)	0.102		
Chronic kidney disease	1.42 (0.99–2.03)	0.056		
COPD	1.16 (0.61–2.22)	0.649		
Atrial fibrillation	1.09 (0.67–1.76)	0.736		
STS score	1.07 (1.04–1.10)	<0.001	1.06 (1.02–1.11)	0.008
Valve size	1.11 (1.03–1.20)	0.005	1.02 (0.90–1.14)	0.803
Balloon expandable system	0.56 (0.39–0.80)	0.002	0.57 (0.34–0.95)	0.030
Balloon predilatation	1.02 (0.71–1.47)	0.901		
Balloon postdilatation	1.05 (0.73–1.51)	0.780		
Pre-LV peak pressure	0.99 (0.99–1.00)	0.394		
Pre-LV minimal pressure	0.98 (0.96–1.01)	0.231		
Pre-LV pre-A pressure	0.97 (0.94–1.00)	0.050		
Pre-LVEDP	0.98 (0.96–1.01)	0.150		
Pre-aortic minimal pressure	0.98 (0.96–0.99)	0.004	0.99 (0.97–1.01)	0.232
LV end-diastolic diameter	1.02 (0.99–1.05)	0.071		
LV end-systolic diameter	1.03 (1.01–1.05)	0.001	0.96 (0.91–1.01)	0.126
LV ejection fraction	0.98 (0.97–0.99)	0.001	0.99 (0.96–1.01)	0.246
LV GLS	1.05 (1.02–1.09)	0.001	1.05 (1.01–1.10)	0.018
LV mass index	1.01 (1.00–1.01)	<0.001	1.01 (1.00–1.01)	0.014
LA volume index	1.01 (0.99–1.01)	0.201		
E velocity	1.00 (1.00–1.01)	0.007	1.01 (1.00–1.01)	0.029
e′ velocity	0.95 (0.83–1.09)	0.470		
Septal E/e′ ratio	1.02 (0.99–1.04)	0.053		
AV Vmax	0.62 (0.48–0.80)	<0.001		
AV mean pressure gradient	0.98 (0.97–0.99)	<0.001		
Aortic valve area	4.87 (1.62–14.60)	0.005	6.03 (1.59–22.87)	0.008
Group 1	0.60 (0.41–0.89)	0.011	0.52 (0.34–0.80)	0.003
Δ Pre-A pressure	1.05 (1.02–1.08)	0.001		

AV indicates aortic valve; COPD, chronic obstructive pulmonary disease; GLS, global longitudinal strain; LA, left atrial; LV, left ventricular; and STS, Society of Thoracic Surgeons.

approximately 60% of patients showed increased LV pre-A pressure after TAVR.

Explanation for Varying Change in LV Pre-A Pressure

The exact mechanism underlying the increased LV pre-A pressure after successful TAVR remains unclear and may involve paravalvular leakage[14–16]; however, we did not detect a significant difference in the presence or severity of paravalvular leakage between the 2 groups. Despite the limited number of patients in previous studies, LV end-diastolic pressure, minimum rate of pressure change (dp/dt), and τ were found to be consistently exacerbated immediately following TAVR.[17,18] This phenomenon is likely attributable to a temporarily obstructed LV outflow tract during the TAVR procedure, compounded by rapid pacing.[19] The possibility of myocardial ischemia, which may have induced an increase in LV pre-A pressure, should be considered. This is supported by elevated creatine kinase-myocardial band (CK-MB) levels in most patients after TAVR in our study.

Given that AS is a disease of the LV myocardium rather than only of the aortic valve,[20,21] LV hypertrophy and myocardial fibrosis are key pathophysiological mechanisms of AS. Therefore, myocardial scars are commonly detected using cardiac magnetic resonance imaging with late gadolinium enhancement in patients with AS.[22,23] Higher preoperative native T1 values that

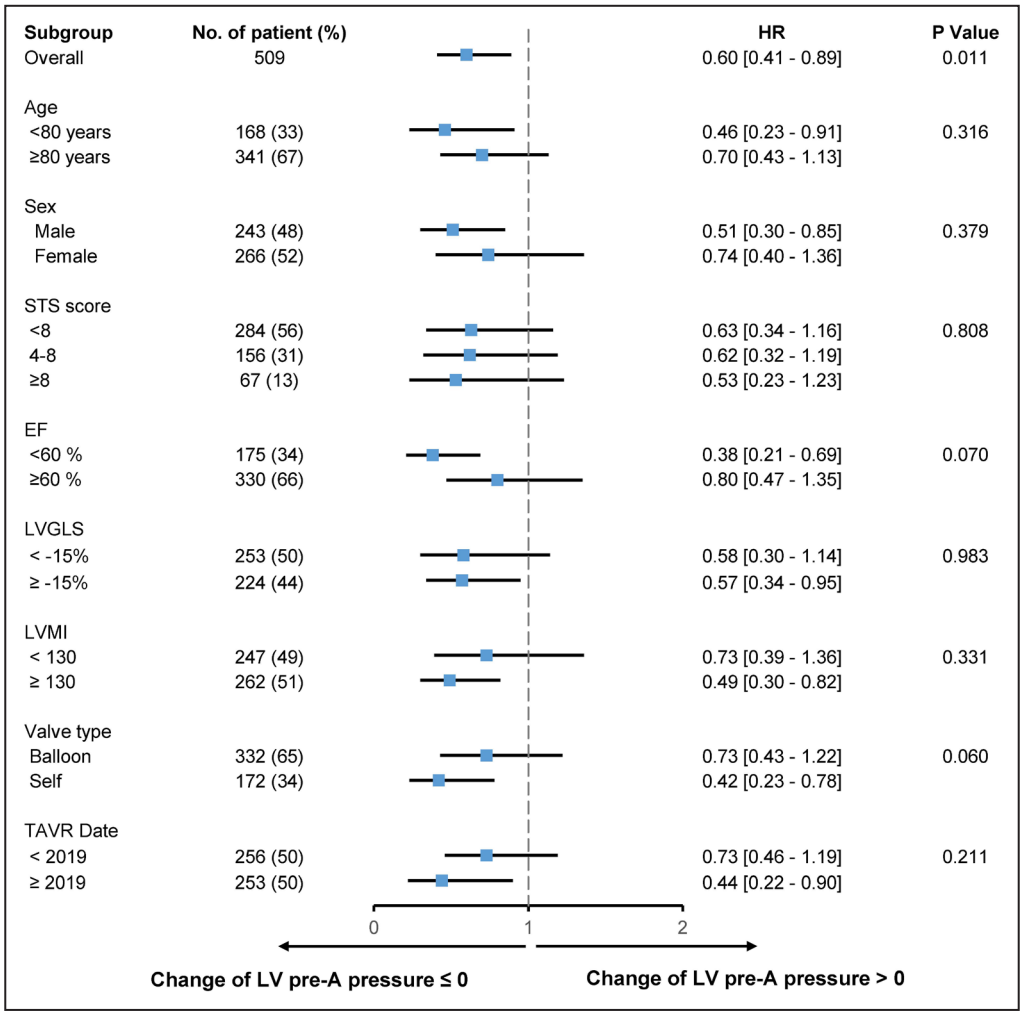

Figure 4. Subgroup analysis.
Risk of primary outcomes associated with an increase or decrease in the LV pre-A pressure in the subgroups. AV Vmax: aortic valve maximum velocity; E velocity: Early diastolic transmitral flow velocity; e' velocity: e' velocity: Septal early diastolic mitral annular velocity; EF indicates ejection fraction; HR, hazard ratio; LVEDP: left ventricular end-diastolic pressure; LV, left ventricular; LVGLS, left ventricular global longitudinal strain; LVMI, left ventricular mass index; STS, Society of Thoracic Surgeons; and TAVR, transcatheter aortic valve replacement.

correlate with histologically proven myocardial fibrosis in AS were recently detected in approximately 70% of patients with severe AS.[24] Transient myocardial damage during TAVR can result in varying responses of LV diastolic pressure changes after TAVR, depending on the extent of myocardial damage from AS. Conversely, in a subset of patients, the burden of LV afterload due to AS is substantial but has little intrinsic LV myocardial problem. Upon resolution of AS through TAVR, there is an immediate and significant decrease in LV diastolic pressure driven by a pronounced unloading effect on the left ventricle. Although transient ischemic damage due to TAVR probably occurred in this group, the remarkable reduction in LV afterload due to TAVR, along with the presence of minimal or reversible LV damage, may have led to a subsequent decrease in LV diastolic pressure after TAVR. Additionally, it can be assumed that resolving AS with TAVR in these patients would yield a relatively favorable long-term outcome, as shown in our results.

Clinical Implications

The relationship between postprocedural increase in LV pre-A pressure and clinical outcomes is important. LV functional evaluation should be further emphasized to identify individuals who would benefit maximally from TAVR. This could be attributed to the fact that AS is not just a valvular disease, and non-AS-induced symptoms may fail to improve after TAVR. Furthermore, if these patients have irreversible changes, vulnerable myocardium, or systemic vascular changes that contribute to

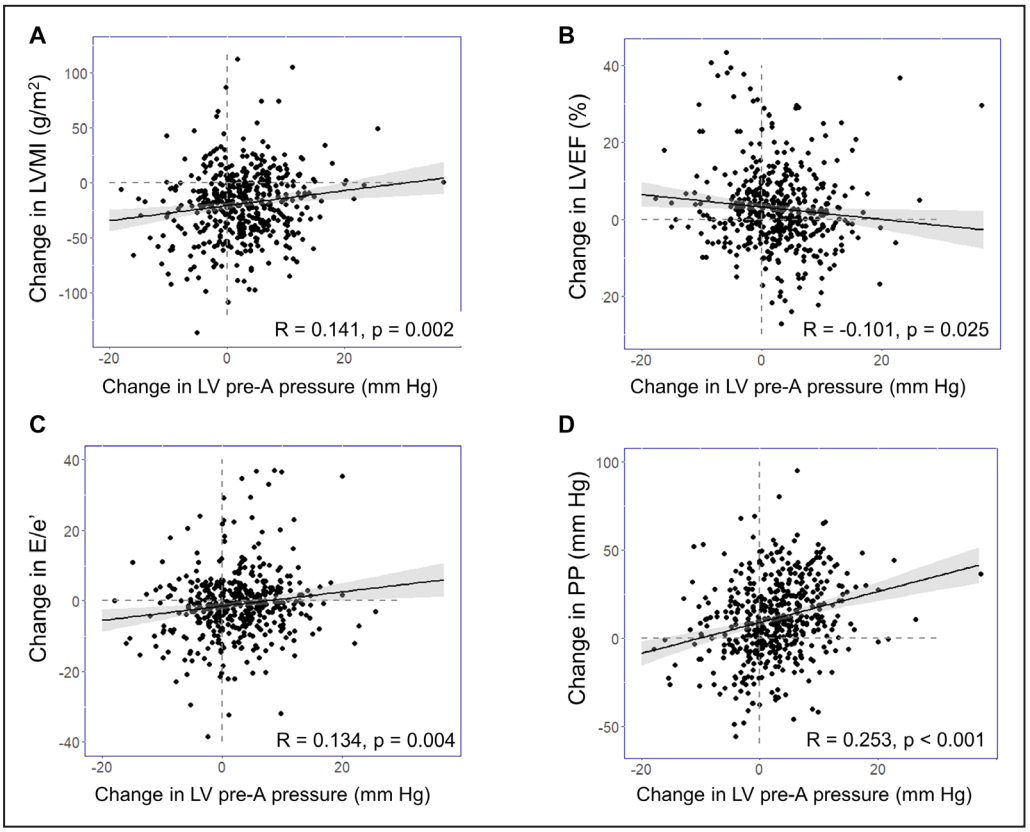

Figure 5. Correlation plot.
Correlation between changes in LV pre-A pressure and (**A**) change in LVMI, (**B**) change in LVEF, (**C**) change in Septal E/e' ratio, and (**D**) change in PP. LV indicates left ventricular; LVEF, left ventricular ejection fraction; LVMI, left ventricular mass index; and PP, pulse pressure.

persistent symptoms, their risk will remain high despite successful TAVR. Reduced LV pre-A pressure after TAVR may help identify a cohort of patients with AS who derive better long-term benefits from TAVR. Valve intervention should be determined by factors including not only valve measurements but also myocardial properties such as myocardial fibrosis.

Although observations of changes in LV pre-A pressure were obtained during the TAVR procedure, our results suggest the necessity for more meticulous patient selection for TAVR. Our study underscores the importance of patient selection for TAVR to get the maximum benefit from the procedure and also provided the important reality of mixed physiology of the current AS population. This study further emphasizes the need for preprocedural assessment of nonvalvular components in patients who are considering TAVR.

Study Limitations

This study had some limitations. First, this was a retrospective study, and only LV and aortic pressure curves were analyzed. Second, because LV pre-A pressure changes cannot be measured before the procedure, it could be considered less valuable as a prognostic predictor than preprocedure parameters. Nevertheless, our study suggests that LV myocardial evaluation should be emphasized to identify individuals who would benefit maximally from TAVR. Moreover, the effectiveness of TAVR may be assessed more accurately under stressful conditions than during the resting state. A diastolic stress test before the procedure may provide additional information on the prognosis after successful TAVR.[25,26] Third, we did not include cardiac magnetic resonance imaging data. Further studies combining cardiac magnetic resonance imaging with late gadolinium enhancement, extracellular myocardial volume, native T1 mapping, and invasive hemodynamics may provide further insights.[23,27,28]

CONCLUSIONS

A significant subset of patients with AS experienced elevated LV pre-A pressure after TAVR. Reduced LV pre-A pressure after TAVR was independently associated with favorable long-term outcomes. Changes in LV pre-A pressure can help identifying a subset of patients

Table 4. Comparison of Echocardiographic Parameters 1 Year After Transcatheter Aortic Valve Replacement

Echocardiographic data	Δ Pre-A pressure ≤0 (n=180)	Δ Pre-A pressure >0 (n=268)	*P* value
LV end-diastolic diameter, mm	47.1±5.9	46.5±5.6	0.300
LV end-systolic diameter, mm	30.3±6.7	29.5±6.0	0.219
LV ejection fraction, %	63.2±10.5	63.2±8.5	0.966
LV mass index, g/m^2	118.8±37.0	111.6±27.6	0.029
LA volume index, mL/m^2	48.7±19.8	47.7±18.5	0.585
E velocity, cm/s	83.5±27.3	82.9±27.1	0.831
e′ velocity, cm/s	4.6±1.4	4.8±1.5	0.112
Septal E/e' ratio	19.5±8.6	18.6±8.1	0.278
Peak TR velocity, cm/s	2.5±0.4	2.5±0.4	0.649
AV peak systolic PG, mm Hg	24.0±9.9	24.5±10.1	0.619
AV mean systolic PG, mm Hg	12.5±5.3	12.9±6.2	0.553
Aortic valve area, cm^2	1.5±0.4	1.5±0.5	0.551
Paravalvular leakage, n			0.866
No	120 (66.7%)	171 (63.8%)	
Mild	52 (28.9%)	82 (30.6%)	
Moderate	7 (3.9%)	14 (5.2%)	
Severe	1 (0.6%)	1 (0.4%)	
Change in LV ejection fraction,* %	5.2±10.9	1.5±9.7	<0.001
Change in LV mass index,* g/m^2	−25.3±29.5	−19.4±29.3	0.037
Change in septal E/e′ ratio*	−2.3±9.5	−0.2±9.3	0.021

AV indicates aortic valve; LA indicates left atrial; LV, left ventricular; PG, pressure gradient; and TR, tricuspid regurgitation.

*1-year follow-up values minus preprocedure values.

who derive maximal benefits from TAVR. Our data suggest that survival after TAVR is not linked to the severity of preprocedural AS but to parameters of myocardial dysfunction in conjunction with other comorbidities.

ARTICLE INFORMATION

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