Relation of Left Atrial Dysfunction to Pulmonary Artery Hypertension in Patients With Aortic Stenosis and Left Ventricular Systolic Dysfunction

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Pulmonary artery hypertension (PAH) increases mortality in patients with severe aortic stenosis. We hypothesized that left atrial (LA) dysfunction would be related to PAH in patients with severe aortic stenosis complicated by left ventricular (LV) systolic dysfunction. The data from 70 patients with severe aortic stenosis and LV systolic dysfunction were analyzed. From the transmitral flow, the peak early (E) and late (A) diastolic velocities were obtained. From the pulmonary vein flow, the peak S-wave, D-wave, and reversed atrial wave velocities were determined. The right ventricular systolic pressure was measured in 50 patients and averaged 38 ± 13 mm Hg. Patients with PAH (n = 19) presented with greater LV diameters, E/A ratio, E-wave velocity, LV mass index, reversed atrial wave velocity, and LA volume ($p < 0.05$) and lower S/D ratio and total and active LA emptying fractions ($p < 0.05$). Simple linear regression analysis revealed that the LA volumes and total and active LA emptying fractions displayed the strongest correlations with the right ventricular systolic pressure. Multiple regression analysis revealed that the minimum LA volume ($r = 0.61$, $p = 0.0001$) independently correlated with the right ventricular systolic pressure, irrespective of the aortic valve (AV) area or gradient. In patients who underwent an echocardiographic examination ≥1 month after AV replacement, LA function had improved significantly. The degree of improvement was related to the degree of recovery of the LV diastolic function and diameter. In conclusion, in patients with severe aortic stenosis and concomitant LV systolic dysfunction, the LA function parameters displayed the strongest correlations with the right ventricular systolic pressure, irrespective of the AV area or gradient and were impaired in patients with PAH. LA function recovered after AV replacement. Additional studies are warranted to determine the prognostic significance of LA function in this setting. **(Am J Cardiol 2010; 106:409–416)**

The left atrial (LA) size is a recognized marker of increased left ventricular (LV) filling pressure and is increased in patients with severe aortic stenosis.[1,2] LA function, expressed as the total LA emptying fraction, is also related to pulmonary artery hypertension (PAH) in patients with heart failure[3] or mitral regurgitation.[4] In patients with heart failure, the maximum LA volume has also been linked to PAH[3,5] and was shown to be an independent predictor of pulmonary artery systolic pressure (PASP).[5] In the presence of mitral regurgitation, the active LA emptying fraction correlated independently with the PASP.[4] Therefore, we hypothesized that the LA volume and function could be potential markers of PAH in patients with aortic stenosis and concomitant heart failure. To test this hypothesis, we analyzed the LA function and its relation to PASP in patients with isolated aortic stenosis who were scheduled to undergo aortic valve (AV) replacement. We also analyzed LA function long term after surgery to assess the reversibility of LA dysfunction. Furthermore, understanding the mechanisms responsible for PAH in patients with aortic stenosis could have physiopathologic and therapeutic relevance.

Methods

The study population was selected from 1,508 consecutive patients who had undergone isolated AV replacement from January 2002 to June 2006 at Cleveland Clinic (Cleveland, Ohio). The selected patients met the following inclusion criteria: (1) severe aortic stenosis, defined as an AV area of ≤ 1.0 cm^2 using the continuity equation; and (2) LV systolic dysfunction, defined as a LV ejection fraction of <50%. Of the 1,508 patients, 132 were selected. Patients with significant aortic regurgitation (greater than grade 2; n = 14), more than moderate mitral regurgitation (n = 6), severe tricuspid regurgitation (n = 1), known coronary artery disease (n = 7), pacing rhythm at the preoperative examination (n = 4), permanent or paroxysmal atrial fibril-

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Table 1
Clinical characteristics and surgical procedure

Variable	Subjects (n = 70)
Clinical characteristic	
Age (years)	69 ± 12
Men/women	50/20
Body surface area (m^2)	2.0 ± 0.3
Coronary angiography	
Normal	35 (50%)
Mild stenosis (<25%)	27 (39%)
Moderate stenosis (26–50%)	8 (11%)
Surgical procedure	
Prosthesis valve	
Bioprosthesis valve	62 (89%)
Carpentier-Edwards	61
3F	1
Mechanical valve	5 (7%)
St. Jude	1
Carbomedics	4
Homograft	3 (4%)
Prosthesis valve size (mm)	23 ± 2
19	8 (11%)
21	13 (19%)
23	19 (27%)
25	22 (31%)
27	7 (10%)
29	1 (1%)

Data are presented as mean ± SD or numbers (%).

Table 2
Two-dimensional echocardiographic characteristics of study patients

Variable	Subjects (n = 70)
Left ventricle	
End-diastolic diameter (cm)	5.6 ± 0.7
End-systolic diameter (cm)	4.3 ± 0.8
End-diastolic volume (ml/m^2)	79 ± 27
End-systolic volume (ml/m^2)	51 ± 24
Ejection fraction (%)	38 ± 10
Mass (g/m^2)	158 ± 41
Left atrium	
Diameter (cm)	4.4 ± 0.7
Area (cm^2)	24 ± 6
Maximum volume (ml/m^2)	40 ± 17
Minimum volume (ml/m^2)	21 ± 12
Precontraction volume (ml/m^2)	30 ± 12
Total emptying fraction (%)	51 ± 15
Active emptying fraction (%)	33 ± 19
Passive emptying fraction (%)	26 ± 12
Aortic valve	
Valve area (cm^2)	0.70 ± 0.19
Peak gradient (mm Hg)	74 ± 25
Mean gradient (mm Hg)	44 ± 16
Transmitral inflow	
E-wave velocity (cm/s)	89 ± 33
A-wave velocity (cm/s)	82 ± 32
E/A ratio	1.3 ± 0.8
E-wave deceleration time (ms)	208 ± 93
Pulmonary vein flow	
Peak reversed A-wave velocity (cm/s)	32 ± 7.0
Peak S-wave/peak D-wave velocities ratio	0.94 ± 0.47

Table 3
Two-dimensional echocardiographic characteristics of study patients

Characteristic	Normal PASP (n = 31)	High PASP (n = 19)
Left ventricle		
End-diastolic diameter (cm)	5.3 ± 0.7	5.9 ± 0.7*
End-systolic diameter (cm)	4.0 ± 0.8	4.6 ± 0.9†
End-diastolic volume (ml/m^2)	74 ± 28	89 ± 30
End-systolic volume (ml/m^2)	47 ± 25	59 ± 27
Ejection fraction (%)	39 ± 10	35 ± 11
Mass (g/m^2)	147 ± 36	182 ± 39†
Left atrium		
Diameter (cm)	4.2 ± 0.6	4.7 ± 0.7†
Area (cm^2)	22 ± 6	27 ± 5*
Maximum volume (ml/m^2)	36 ± 13	51 ± 20†
Minimum volume (ml/m^2)	16 ± 9	29 ± 14*
Precontraction volume (ml/m^2)	26 ± 11	37 ± 14*
Total emptying fraction (%)	57 ± 13	44 ± 11*
Active emptying fraction (%)	39 ± 18	24 ± 15†
Passive emptying fraction (%)	30 ± 11	25 ± 10
Aortic valve		
Valve area (cm^2)	0.71 ± 0.19	0.60 ± 0.19
Peak gradient (mm Hg)	75 ± 24	83 ± 26
Mean gradient (mm Hg)	44 ± 15	51 ± 16
Transmitral inflow		
E-wave velocity (cm/s)	78 ± 27	108 ± 41*
A-wave velocity (cm/s)	88 ± 28	82 ± 47
E/A ratio	1.0 ± 0.5	1.7 ± 1.2*
E-wave deceleration time (ms)	224 ± 98	177 ± 79
Pulmonary vein flow		
Peak reversed A-wave velocity (cm/s)	31 ± 6	37 ± 9*
Peak S-wave/peak D-wave velocities ratio	1.15 ± 0.43	0.66 ± 0.50*

* $p < 0.001$; † $p < 0.05$ versus normal PASP.

lation (n = 20), and inadequate imaging quality (n = 10) were excluded. The data presented were abstracted from our echocardiography and surgical databases, which have been approved by the Institutional Review Board for Clinical Research at Cleveland Clinic. The final population of the study consisted of 70 patients. Of the 70 patients, we identified 28 who had undergone an echocardiographic examination at our institution >1 month after AV replacement. Follow-up echocardiograms were performed according to the recommendation by the patients' physicians.

Studies were performed through several commercially available, ultrasound systems. Cardiac dimensions were measured in accordance with the American Society of Echocardiography recommendations.[6] M-mode echocardiography was used to measure the LA diameter and LV end-diastolic and end-systolic diameters. The LV mass index was calculated according to previously published formulas.[7] The LV and LA volumes were determined using the modified Simpson rule with images obtained from apical 4- and 2-chamber views. Pulsed wave Doppler was obtained in the apical 4-chamber view, positioned at the mitral leaflet tips. From the transmitral recordings, the peak early (E) and late (A) diastolic filling velocities, E/A ratio, and E-wave deceleration time were obtained. The peak and mean transaortic valve gradients were calculated using the simplified Bernoulli equation. The AV area was calculated by continuity equation using the velocity-time integral of the

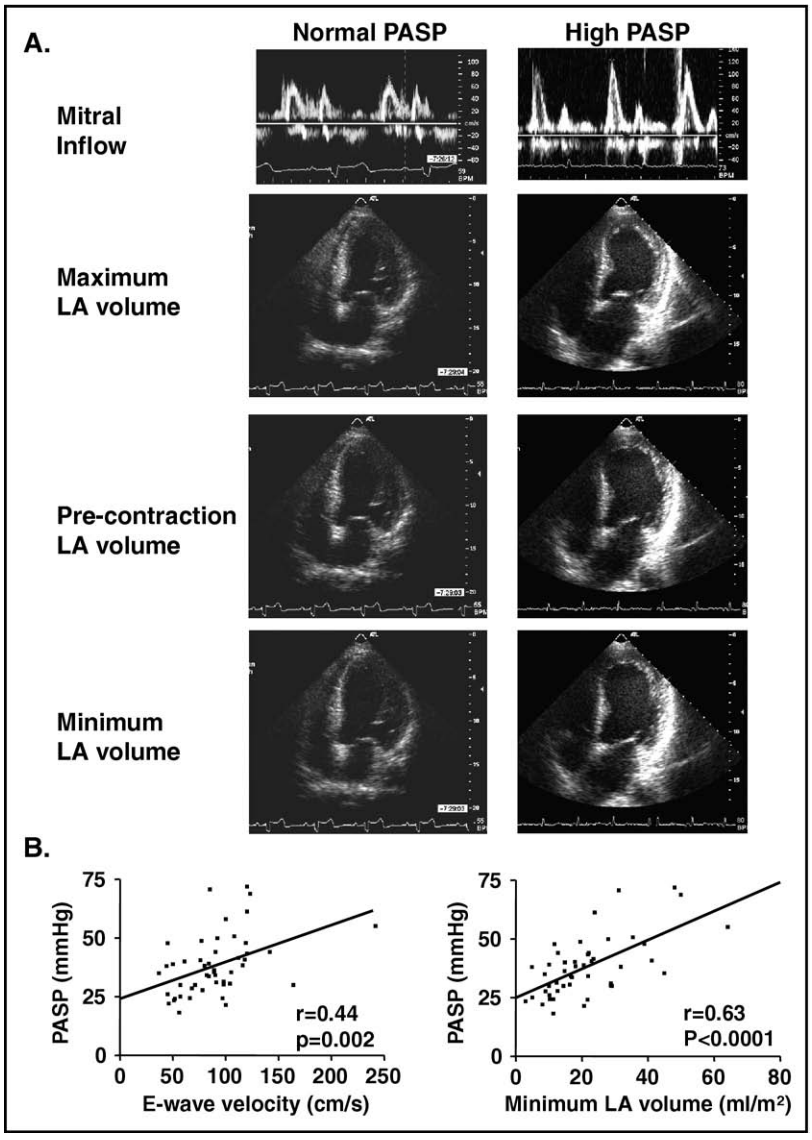

Figure 1. PASP correlated with LA function and LV diastolic function in aortic stenosis. *(A)* Patient with PAH displayed more restrictive pattern of LV diastolic function, with greater E/A ratio, and worse LA function, depicted by a small change in LA area across different phases of cardiac cycle. *(B)* PASP increased significantly with greater E-wave velocity and greater minimum LA volume. A = maximum late wave velocity; E = maximal early wave velocity; LA = left atrial; LV = left ventricular; PAH = pulmonary artery hypertension; PASP = pulmonary artery systolic pressure.

aortic and LV outflow tract flows. Pulmonary venous flow recordings were obtained from the 4-chamber view with the 5-mm sample volume positioned 1 to 2 cm into the right upper pulmonary vein, and the following measurements were taken: peak S-wave inflow velocity during ventricular systole, peak D-wave inflow velocity during the early phase of ventricular diastole and the corresponding S/D ratio, and the peak reversed atrial wave (Ar) velocity during LA contraction. PASP was derived from continuous wave Doppler interrogation of tricuspid regurgitation. The right atrial pressure was estimated from the inferior vena cava size and inspiratory collapse, in accordance with the American Society of Echocardiography recommendations[6] and previously published reports.[8]

The following indexes of LA function were calculated according to a previous study.[9] The total LA stroke volume was calculated as the maximum LA volume minus the minimum LA volume. The active LA stroke volume was calculated as the precontraction LA volume minus the minimum LA volume. The passive LA stroke volume was calculated as the maximum LA volume minus the precontraction LA volume. The total LA emptying fraction was calculated as (total LA stroke volume/maximum LA volume) × 100. The active LA emptying fraction was calculated as (active LA stroke volume/precontraction LA volume) × 100. The passive LA emptying fraction was calculated as (passive LA stroke volume/maximum LA volume) × 100.

The echocardiograms were stored digitally and reviewed off-line with software (Prosolv Cardiovascular Analyzer, Problem Solving Concepts, Indianapolis, Indiana).

The calculations were done using commercially available statistical software (GraphPad Prism, version 3.02, La Jolla, California, and MedCalc, version 9.2.0.2, Mariakerke,

Table 4
Univariate regression analysis of echocardiographic parameters and pulmonary artery systolic pressure (PASP)

Variable	r	p Value
Left ventricle		
End-diastolic diameter	0.37	0.009
End-systolic diameter	0.35	0.01
End-diastolic volume index	0.22	NS
End-systolic volume index	0.25	NS
Ejection fraction	−0.29	0.04
Mass index	0.37	0.01
Left atrium		
Diameter	0.29	0.04
Area	0.48	0.0004
Maximum volume index	0.51	0.0002
Minimum volume index	0.63	<0.0001
Precontraction volume index	0.52	0.0002
Total emptying fraction	−0.59	<0.0001
Active emptying fraction	−0.50	0.0003
Passive emptying fraction	−0.19	NS
Aortic valve		
Valve area	−0.14	NS
Peak gradient	0.07	NS
Mean gradient	0.09	NS
Transmitral inflow		
E-wave velocity	0.44	0.002
A-wave velocity	0.04	NS
E/A ratio	0.34	0.03
E-wave deceleration time	−0.22	NS
Pulmonary vein flow		
Peak reversed A-wave velocity	0.33	0.03
Peak S-wave/peak D-wave velocities ratio	−0.36	0.02

Belgium). Continuous variables are expressed as the mean ± SD and discrete variables as percentages. Comparisons between patients with and without PAH were performed using the unpaired Student t test. Each variable was tested for correlation with PASP using simple linear regression analysis (Pearson's correlation). All variables with a significant univariate association with PASP were entered in a multivariate stepwise regression analysis, with PASP as the dependent variable. The pre- and postoperative LA function parameters were compared using the paired Student *t* test. The null hypothesis was rejected at $p < 0.05$.

Results

The clinical characteristics and surgical procedures of the studied patients are listed in Table 1. All patients underwent isolated AV replacement without serious complications.

The echocardiographic characteristics are listed in Table 2. Of the 70 patients, 24 (34.3%) had mild LV systolic dysfunction, 24 (34.3%) had moderate, and 22 (31.4%) had severe LV systolic dysfunction. Of the 70 patients, 38 (54%) had a mean AV gradient >40 mm Hg, 27 (39%) had a mean AV gradient of 20 to 40 mm Hg, and 5 (7%) had a mean AV gradient of <20 mm Hg. Finally, 32 patients (46%) had bicuspid valves, 37 (53%) had tricuspid valves, and 1 (1%) had a unicuspid valve. The LA volumes were increased. The PASP was measured in 50 patients and averaged 38 ± 13 mm Hg; 19 patients had elevated PASP (>40 mm Hg; 51 ± 11 mm Hg). No significant difference was found in the gender proportion between patients with PAH (13 men and 6 women) or without PAH (20 men and 11 women; p = NS). Also, no significant difference was found in age (71 ± 12 vs 69 ± 13 years, respectively) between patients with and without PAH. Patients with PAH had a larger LA size, LV diameters, LV mass index, E/A ratio, E-wave velocity, and Ar velocity and lower S/D ratio than patients without PAH. The lower LV ejection fraction and AV area in patients with PAH did not reach statistical significance (Table 3).

All LA volumes evaluated were larger in the patients with PAH than in those without PAH. The total and active LA emptying fractions were lower in patients with PAH than in those without PAH, but the passive LA emptying fraction was similar between the 2 groups (Figure 1 and Table 3). Therefore, the LA contractile and reservoir functions were depressed in patients with PAH.

Simple linear regression analysis revealed significant positive correlations between PASP and the LA diameter and area, LV diameters, E-wave velocity, E/A ratio, LV mass index, Ar velocity and negative correlations between PASP and LV ejection fraction and S/D ratio. No significant relation to age was found. The correlations between PASP and the LA volume and function parameters were stronger than with the previously described echocardiographic parameters. PASP correlated positively with the maximum, minimum, and precontraction LA volumes and negatively with the total and active LA emptying fractions (Figure 1 and Table 4).

Multiple stepwise regression analysis, including all variables with a significant univariate association with PASP, revealed that only the minimum LA volume (p = 0.0001) independently correlated with the PASP.

A subgroup of 28 patients underwent follow-up echocardiography at our institution ≥1 month after AV replacement (22 ± 15 months). A significant reduction in the minimum and precontraction LA volumes was seen, with improvement of the total, passive, and active LA emptying fractions after AV replacement (Figure 2). The reduction in the maximum LA volume was not significant. Also, a significant reduction was seen in the LV diameters, volumes, and mass, with improvement in the LV ejection fraction, after AV replacement. The mean and peak AV gradients decreased significantly after AV replacement. The E/A ratio did not change significantly, but an increase occurred in the E-wave deceleration time after AV replacement. The Ar velocity decreased significantly after AV replacement (Table 5).

From those 28 patients, we found significant correlations between an improvement in LA function and a change in LV size or LV diastolic function (Figure 3). The improvement in the total LA emptying fraction correlated with decreases in LV end-diastolic (r = 0.63, p = 0.0007) and end-systolic (r = 0.61, p = 0.001) diameters, E-wave velocity (r = 0.67, p = 0.0003), E/A ratio (r = 0.56, p = 0.007), LV end-diastolic volume (r = 0.40, p = 0.05), and D-wave velocity (r = 0.47, p = 0.04) and with increases in A-wave velocity (r = −0.42, p = 0.05), E-wave deceleration time (r = −0.54, p = 0.005), and S/D ratio (r = −0.52, p = 0.02). The improvement in the active LA emptying fraction correlated with decreases in LV end-diastolic (r =

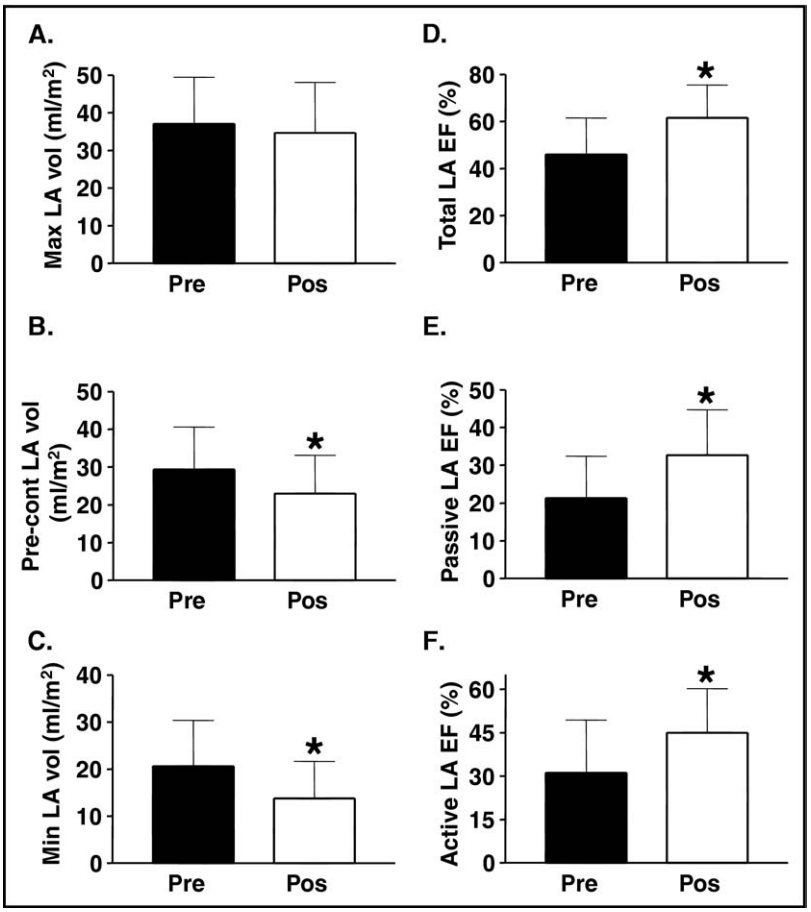

Figure 2. LA function recovery after AV replacement. Subgroup of 28 patients underwent follow-up echocardiography ≥1 month after AV replacement. Although maximum LA volume *(A)* did not change significantly, *(B)* precontraction and *(C)* minimum LA volumes decreased significantly. Thus, the *(D)* total, *(E)* passive, and *(F)* active LA emptying fractions recovered significantly after AV replacement. AV = aortic valve; EF = emptying fraction; LA = left atrium; vol = volume. *p <0.05.

Table 5
Two-dimensional echocardiographic characteristics before and late after surgery

Variable	Preoperative (n = 28)	Postoperative (n = 28)
Left ventricle		
End-diastolic diameter (cm)	5.6 ± 0.5	5.3 ± 0.9*
End-systolic diameter (cm)	4.4 ± 0.7	3.7 ± 0.9†
End-diastolic volume (ml/m²)	79 ± 23	62 ± 23†
End-systolic volume (ml/m²)	52 ± 21	24 ± 15‡
Ejection fraction (%)	37 ± 10	62 ± 12‡
Mass (g/m²)	160 ± 54	120 ± 26‡
Aortic valve		
Peak gradient (mm Hg)	71 ± 28	28 ± 16‡
Mean gradient (mm Hg)	42 ± 18	28 ± 16*
Transmitral inflow		
E-wave velocity (cm/s)	95 ± 22	96 ± 29
A-wave velocity (cm/s)	80 ± 31	90 ± 27
E/A ratio	1.4 ± 0.7	1.1 ± 0.5
E-wave deceleration time (ms)	179 ± 65	220 ± 65*
Pulmonary vein flow		
Peak reversed A-wave velocity (cm/s)	34 ± 7	26 ± 6†
Peak S-wave/peak D-wave velocities ratio	0.77 ± 0.36	0.89 ± 0.51

* p <0.05; † p <0.01; ‡ p <0.0001 versus preoperatively.

0.54, p = 0.01) and end-systolic (r = 0.63, p = 0.001) diameters, E-wave velocity (r = 0.59, p = 0.004), and E/A ratio (r = 0.52, p = 0.02) and with increases in E-wave deceleration time (r = −0.44, p = 0.04) and S/D ratio (r = −0.55, p = 0.02). The improvement in the passive LA emptying fraction correlated with decreases in the LV end-diastolic diameter (r = 0.45, p = 0.04) and volume (r = 0.52, p = 0.01) and with increases in the E-wave deceleration time (r = −0.47, p = 0.03). Also, a trend was seen for a significant correlation between improvement in the passive LA emptying fraction and a decrease in LV mass (r = 0.39, p = 0.08).

Few patients had the PASP measured both before and after AV replacement, precluding an adequate analysis of the factors involved in the changes in PASP after AV replacement. Even so, a significant relation was seen between a change in the PASP and a change in the active LA emptying fraction (r = 0.82, p = 0.007).

Discussion

In the present study, the presence of PAH in patients with aortic stenosis and concomitant LV dysfunction correlated with LV diastolic function, LV mass, and LA function parameters. Furthermore, multiple regression analysis

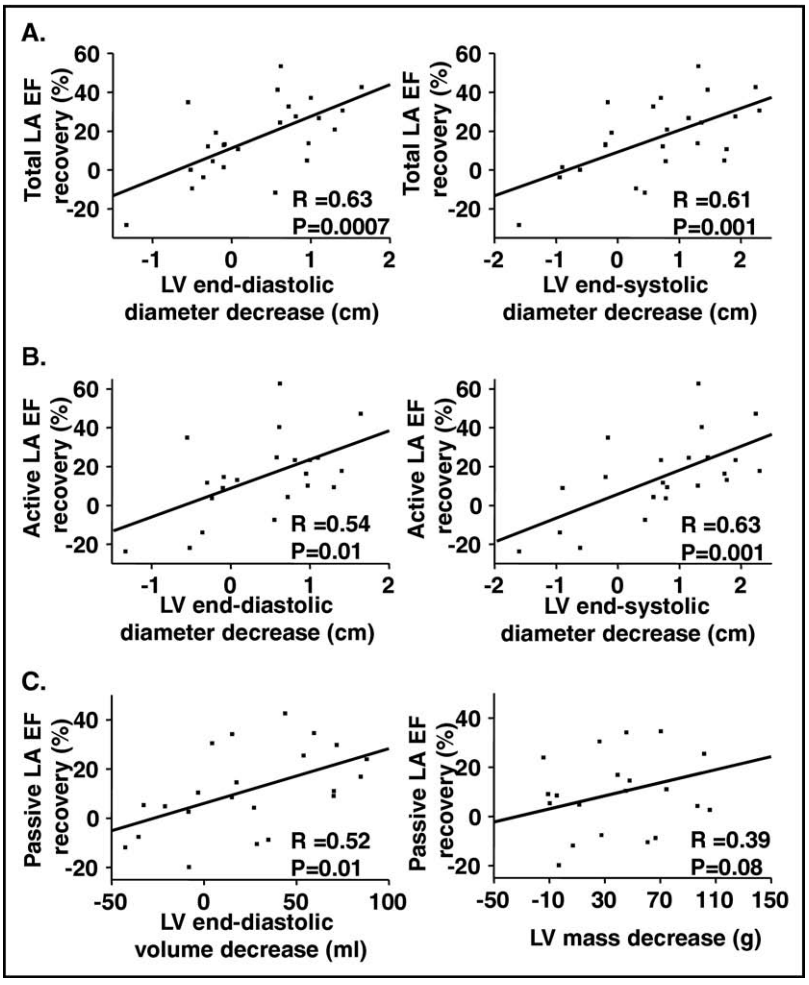

Figure 3. Correlations between LA function recovery and LV geometry improvement after AV replacement Univariate analysis depicted positive correlations between *(A)* recovery of total LA emptying fraction and regression of LV end-diastolic and end-systolic diameters after AV replacement, *(B)* recovery of active LA emptying fraction and regression of LV end-diastolic and end-systolic diameters after AV replacement, and *(C)* recovery of passive LA emptying fraction and regression of LV end-diastolic volume and LV mass after AV replacement. Recovery of LA function calculated as absolute values obtained when LA function parameter after AV replacement was subtracted from values before AV replacement. Improvement in LV geometry calculated as absolute values obtained when LV parameter before AV replacement was subtracted from values obtained after AV replacement. AV = aortic valve; EF = emptying fraction; LA = left atrial; LV = left ventricular.

showed that the minimum LA volume correlated with the PASP, irrespective of the AV area. After AV replacement, LA function improved, and the degree of its improvement was directly related to the degree of improvement in LV size and mass and in LV diastolic function.

PAH is not uncommon among patients with aortic stenosis. Severe PAH was found in 11% to 19% of the patients with severe aortic stenosis,[10–12] and mild to moderate PAH was found in 28% to 50% of the patients.[11,12] Moreover, patients with aortic stenosis and PAH have a dismal prognosis.[13–15] Therefore, PAH is one of the most important clinical questions we encounter with patients with severe aortic stenosis.

In our study, the LV diastolic parameters and LV mass correlated significantly with the PASP. Others studies have reported a greater E/A ratio and shorter E-wave deceleration time in patients with PAH[10] and significant associations between PASP and LV mass, E-wave velocity, and early diastolic mitral annular velocity (E′).[16] In the latter study, the E/E′ ratio was the only echocardiographic parameter independently associated with PASP.[16] In studies that included hemodynamic data, the increase in LV end-diastolic pressure[11,17,18] and pulmonary artery wedge pressure[11] correlated significantly with PASP. Other studies also described a high incidence of elevated LV end-diastolic pressure in patients with severe PAH.[12,19] Therefore, LV hypertrophy and diastolic dysfunction triggered by the pressure overload must contribute to the increase in the LV filling pressures that ultimately lead to reactive PAH. However, the role of the LA function in patients with aortic stenosis with PAH was not clear from these studies.

The relation between the AV area and PASP was also addressed by us and others. Although a lower AV area was described as an independent predictor of PAH,[10] we, and others, found a poor correlation between PASP and the AV area[11,16] or gradients.[16] Moreover, in other studies, the AV area and gradients did not significantly differ between patients with and without PAH.[17,19]

The LA volume is a hallmark of longstanding increased LV end-diastolic pressure and is increased in the presence of severe aortic stenosis.[1,2] The maximum LA volume has been linked to PAH[3,5] and was described as an independent predictor of PASP in patients with heart failure.[5] The LA volume increase in those with aortic stenosis is related not only to the AV area, but also to the LV mass, concomitant mitral regurgitation, a history of hypertension, LV end-diastolic volume, and a restrictive filling pattern.[1,2] However, the correlation coefficients published have varied. Although the correlation between the maximal LA volume and LV mass was described as 0.77,[1] the correlation of the same parameter with AV area was only -0.10^2 and -0.18.[20] In our study, the LA volume was greater in patients with PAH than in those without PAH and had a better correlation with the PASP than the LV diastolic parameters. Considering the direct connection of LA and pulmonary vasculatures, this greater association was expected.

We also found a direct association between PASP, not only with the LA volume, but also with LA function. LA contractile function might contribute to keeping LV filling in patients with aortic stenosis. The passive LA emptying fraction is decreased, and the active LA emptying volume is increased.[1] The A-wave velocity is also greater in those with aortic stenosis than in age- and LV systolic function-matched controls.[21,22] Therefore, most patients with aortic stenosis have an increased atrial contribution to LV filling.[22] In our study, the total and active LA emptying fractions were reduced in patients with PAH. In addition, the minimum LA volume was independently related to the PASP. Therefore, we speculated that LA dysfunction makes LA emptying worse and contributes to further increases in LA pressure and consequent reactive PAH in those with aortic stenosis. The importance of LA function was highlighted, because the peak late diastolic mitral annulus velocity was a predictor of cardiac death and the need for AV replacement in patients with aortic stenosis and a normal LV ejection fraction.[20]

Most patients in our studied population presented with mild or moderate LV systolic dysfunction, but 1/3 presented with severe LV systolic dysfunction. Therefore, most patients still presented with a mean AV gradient of >40 mm Hg, and 46% of patients presented with a mean AV gradient of <40 mm Hg. Thus, although it was often difficult to decide to perform AV replacement in patients with a low AV gradient and severe LV systolic dysfunction, this group was also represented in our population. AV replacement in such risky patients is beneficial, under specific recommendations.[23,24]

AV replacement improves survival in patients with aortic stenosis associated with severe PAH[25] and reduces the PASP in the early postoperative period.[18] In our study, AV replacement was also associated with improvement in all components of LA function, indicating that the concomitant hemodynamic burden of severe aortic stenosis and LV systolic dysfunction contributed to LA dysfunction. We observed a significant decrease in the minimum and precontraction LA volumes, with improvement in the total, passive, and active LA emptying fractions after AV replacement. This suggested a reversibility of LA size and function in patients with aortic stenosis, even in the presence of PAH. However, it is unknown whether LA function recovery after AV replacement or whether preoperative LA dysfunction will have an effect on the long-term prognosis of the patients.

The retrospective nature and the selection criteria used limited the conclusions of our study to patients with severe aortic stenosis and concomitant LV dysfunction in sinus rhythm. The extrapolation of these results to other populations remains to be explored. It is also possible that other parameters could emerge as independently related to PASP if the number of patients included in the study had been greater.

It is possible that the LA dysfunction described in the present study was solely the consequence of an increase in LA afterload due to diastolic LV dysfunction and that the same LV diastolic dysfunction was the primary contributor to PAH. However, the statistically independent correlation of the minimum LA volume to PASP points in the direction of an independent contribution of LA dysfunction to the development of PAH in these patients.

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