

Time course of left ventricular remodelling and mechanics after aortic valve surgery: aortic stenosis vs. aortic regurgitation

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Aims

Pressure overload in aortic stenosis (AS) and both pressure and volume overload in aortic regurgitation (AR) induce concentric and eccentric hypertrophy, respectively. These structural changes influence left ventricular (LV) mechanics, but little is known about the time course of LV remodelling and mechanics after aortic valve surgery (AVR) and its differences in AS vs. AR. The present study aimed to characterize the time course of LV mass index (LVMI) and LV mechanics [by LV global longitudinal strain (LV GLS)] after AVR in AS vs. AR.

Methods and results

Two hundred and eleven (61 ± 14 years, 61% male) patients with severe AS (63%) or AR (37%) undergoing surgical AVR with routine echocardiographic follow-up at 1, 2, and/or 5 years were evaluated. Before AVR, LVMI was larger in AR patients compared with AS. Both groups showed moderately impaired LV GLS, but preserved LV ejection fraction. After surgery, both groups showed LV mass regression, although a more pronounced decline was seen in AR patients. Improvement in LV GLS was observed in both groups, but characterized by an initial decline in AR patients while LV GLS in AS patients remained initially stable.

Conclusion

In severe AS and AR patients undergoing AVR, LV mass regression and changes in LV GLS are similar despite different LV remodelling before AVR. In AR, relief of volume overload led to reduction in LVMI and an initial decline in LV GLS. In contrast, relief of pressure overload in AS was characterized by a stable LV GLS and more sustained LV mass regression.

Keywords

aortic stenosis • aortic regurgitation • LV remodelling • LV mechanics • LV mass regression • LV global longitudinal strain

Introduction

In severe aortic stenosis (AS) and severe chronic aortic regurgitation (AR), aortic valve replacement (AVR) is indicated when patients have symptoms or show signs of left ventricular (LV) dysfunction.^{1,2} The pathophysiology of these two valvular heart diseases and the time course of LV remodelling and development of symptoms are

different. While AS induces a pressure overload on the left ventricle, AR imposes both a pressure and a volume overload. These abnormal haemodynamic conditions induce different remodelling responses of the LV: concentric hypertrophy due to increased muscle fibre diameter and parallel addition of new myofibrils occurs in AS, whereas in AR the growth of cardiomyocytes and the addition of new sarcomeres in series induces eccentric remodelling and LV dilatation.³ In

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both situations, there is increased formation of interstitial fibrosis that may not regress when the volume and/or pressure overload are relieved after AVR.^{3–6} These structural changes influence LV mechanics and, although LV ejection fraction (LVEF) may be preserved for long time, measures of LV deformation such as LV global longitudinal strain (GLS) assessed by speckle tracking imaging have shown that the LV systolic function may be impaired at earlier stages.^{7–10} Impaired LV GLS prior to AVR has been correlated with adverse outcomes after AVR in both AS and AR.^{11,12} However, little is known about the time course of LV remodelling and LV mechanics after AVR and how they differ in patients with AS vs. patients with AR. Therefore, the present study characterizes and compares the time course of LV remodelling and changes in LV mechanics as assessed with 2D speckle tracking LV GLS after AVR in AS vs. AR.

Methods

Study population and data collection

From an ongoing registry of patients with aortic valve disease, patients with severe AS or AR who underwent surgical aortic valve replacement or repair (AVR) at the Leiden University Medical Center were selected based on echocardiographic data available at baseline (prior to surgery) and at least one or more echocardiograms at specific follow-up times: within 1 year after surgery and/or at ~2- and/or 5-year follow-up. Patients with concomitant coronary artery bypass grafting (CABG) and elective aortic surgery were not excluded. If reoperation of the aortic valve and/or aortic root was performed during follow-up, the last transthoracic echocardiogram before reoperation was used for the analysis. Reasons for patient exclusion were non-severe AS or AR, previous mitral valve replacement or AVR, active endocarditis, Type A aortic dissection, or non-feasible LV GLS analysis due to insufficient data.

Demographic and clinical data were collected using electronic records (EPD Vision, version 11.4.29.0, EPD Vision, Leiden, The Netherlands). Echocardiographic data were digitalized and stored in the departmental server (Imagevault, GE Healthcare, Norway). The institutional review board approved this retrospective analysis of clinically acquired data and waived the need for patient written informed consent.

2D transthoracic echocardiography

All patients underwent transthoracic echocardiography prior to surgery. Images were acquired with patients at rest in the left decubitus position using commercially available ultrasound systems (System 5, Vivid 7 and E9, GE Healthcare, Vingmed, Horten, Norway) equipped with 3.5-MHz or M5S transducers. 2D, colour, pulsed-, and continuous-wave Doppler data were obtained in parasternal and apical views. Data were stored digitally and analysed offline retrospectively on a dedicated workstation (EchoPac version BT13; GE Medical Systems). Apical two- and four-chamber views were used for quantification of LV end-diastolic and end-systolic volumes, and LVEF was calculated using the Simpson's biplane method.¹³ LV dimensions and wall thicknesses were measured on M-mode recordings of the parasternal long-axis view. LV mass was calculated according to the formula as recommended by the American Society of Echocardiography and the European Association of Cardiovascular Imaging¹³ and was indexed (LVMI) to body surface area. 2D speckle tracking longitudinal strain analysis was performed on the apical two-, three-, and four-chamber views to calculate LV GLS.¹³ Aortic valve mean and peak gradients were evaluated using continuous-wave Doppler on the three- or five-chamber LV apical views with the simplified Bernoulli equation. Pulsed-wave Doppler recordings of the LV outflow tract were

obtained on the same apical views and the aortic valve area was calculated using the continuity equation.¹⁴ Severe AS was defined based on an aortic valve area <1.0 cm² or indexed aortic valve area <0.6 cm²/m² and/or mean transvalvular pressure gradient ≥40 mmHg and/or peak aortic jet velocity ≥4 m/s.¹⁴ AR grade was assessed using a multi-parametric approach that included the measurement of the vena contracta on the parasternal long-axis view or the apical three- or five-chamber views, the ratio between the regurgitant jet width and the LV outflow tract diameter on colour M-mode recordings on the parasternal long-axis views and the diastolic flow reversal velocity on the suprasternal view of the aortic arch.¹⁵ In addition, the pressure half time was measured from continuous-wave Doppler recordings of the regurgitant jet on the apical three- or five-chamber views.¹⁵

Follow-up

Patients were evaluated after AVR at the outpatient clinic of the Leiden University Medical Center. Transthoracic echocardiography was performed at the discretion of the treating physician. LV dimensions and function, including LV GLS, were measured. Echocardiograms performed within 1 year (0–12 months), at 2 years, and/or at 5 years after AVR were analysed. If more than one echocardiogram was performed within one time period, the latest one was analysed.

Statistical analysis

Categorical variables are presented as numbers and percentages. Continuous variables are presented as mean ± standard deviation or median and interquartile range if not normally distributed. Comparison of continuous variables between the AS and AR groups at baseline (prior to AVR) were performed using the Student's independent t-test or the Mann-Whitney *U* test (for normally or non-normally distributed variables, respectively) and categorical variables using the χ^2 test or the Fisher's exact test, as appropriate. Linear mixed model analyses were used to assess changes in LV GLS, LVMI, LVEF and stroke volume between sequential time points. Correction for age, gender, LV end-diastolic diameter (LVEDD) at baseline, and time between echocardiograms was applied by incorporating these parameters in the models as fixed variables (for gender) or covariates (for age, LVEDD, and time to follow-up). Analyses were performed per separate time interval (between baseline and 1-year follow-up, 1- and 2-year follow-up, and between 2- and 5-year follow-up, respectively) using a stepwise approach: first, interaction between the AS and AR groups and time (*P* for interaction) was assessed and then excluded from the model if not statistically significant and secondly, time as factor (*P* for time) was assessed and then excluded from the model if not statistically significant. All statistical analyses were two-sided and *P*-values <0.05 were considered statistically significant. The SPSS software (version 23.0; IBM, Armonk, NY, USA) was used to perform the analyses.

Results

Clinical characteristics

A total of 211 patients (mean age 61.3 ± 13.5 years, 61% male) with echocardiographic follow-up were evaluated: severe AS was present in 132 (63%) patients and severe AR in 79 (37%) patients. The clinical characteristics of the overall population and both subgroups are shown in *Table 1*. Patients with AS were older and more often had coronary artery disease and prior myocardial infarction as compared to patients with AR. Cardiovascular risk factors were equally distributed with the exception of current smoking, which was more prevalent in AR patients. Patients with AR were more frequently treated with

Table 1 Baseline clinical characteristics of patients with AS and AR undergoing AVR

	Total population (n = 211)	Patients with AS (n = 132)	Patients with AR (n = 79)	P-value
Female gender, n (%)	83 (39)	55 (42)	28 (35)	0.370
Age (years)	61.3 ± 13.5	64.6 ± 11.5	55.9 ± 14.7	<0.001
BSA (m ²)	1.90 ± 0.19	1.90 ± 0.19	1.91 ± 0.18	0.598
Logistic EuroSCORE (%)	4.7 (2.3–8.1)	4.6 (2.7–8.0)	5.3 (2.2–9.7)	0.548
Creatinine level (µmol/mL)	85 (74–99)	87 (74–101)	84 (74–99)	0.576
Cardiovascular risk factors, n (%)				
Hypertension	122 (58)	73 (55)	49 (62)	0.339
Hyperlipidaemia	81 (38)	55 (42)	26 (33)	0.206
Diabetes mellitus	22 (10)	17 (13)	5 (6)	0.132
Current smoking	50 (24)	24 (19)	26 (33)	0.018
Coronary artery disease, n (%)	85 (40)	70 (53)	15 (19)	<0.001
Prior MI, n (%)	25 (11)	20 (15)	5 (6)	0.055
Systolic blood pressure (mmHg)	140.3 ± 22.7	141.8 ± 23.2	138.0 ± 21.7	0.241
Diastolic blood pressure (mmHg)	76.4 ± 11.9	79.1 ± 11.1	71.8 ± 12.0	<0.001
Medications, n (%)				
Beta-blocker	108 (52)	66 (51)	42 (54)	0.708
ACE-inhibitor or AT-receptor antagonist	121 (59)	62 (48)	59 (76)	<0.001
Diuretics	65 (31)	39 (30)	26 (33)	0.641
Spironolactone	9 (4)	2 (2)	7 (9)	0.011
Statins	100 (48)	74 (57)	26 (33)	0.001
Aspirin	81 (39)	58 (45)	23 (30)	0.027
Anticoagulation	40 (19)	19 (15)	21 (27)	0.029
Symptomatic status, n (%)	163 (78)	97 (74)	66 (85)	0.074
NYHA-classification, n (%)				0.909
I–II	159 (76)	100 (76)	59 (76)	
III–IV	50 (24)	31 (24)	19 (24)	

ACE, angiotensin-converting enzyme; AR, aortic regurgitation; AS, aortic stenosis; AT, angiotensin; AVR, aortic valve replacement or repair; BSA, body surface area; MI, myocardial infarction; NYHA, New York Heart Association functional classification.

angiotensin-converting enzyme-inhibitors, spironolactone, and anticoagulants, whereas patients with AS received aspirin and statins more frequently. Both patient groups had comparable logistic EuroSCORE and New York Heart Association heart failure symptoms.

Echocardiographic and procedural characteristics at baseline

Baseline echocardiographic and procedural characteristics of the total population and both AS and AR patient subgroups are displayed in Table 2. The majority of patients had tricuspid anatomy of the aortic valve (77%), whereas bicuspid valve anatomy was present in 23%. AR patients more frequently showed bicuspid aortic valve anatomy (33% vs. 17%, $P=0.010$). Patients with AR had larger LVEDD (59.8 ± 7.4 vs. 50.2 ± 8.3 mm, $P<0.001$) as compared to AS patients. Both groups showed a preserved LVEF. During AVR, both AS and AR patients received biological prostheses more often than mechanical prostheses. Larger valves were implanted in patients with AR. Concomitant CABG was more frequently performed in AS patients (42% vs. 24%, $P=0.009$).

Time course of LV remodelling and LV mechanics

The total median echocardiographic follow-up duration was 57 (51–65) months, with the first post-operative echocardiogram performed at 3 (0–7) months and subsequent controls at 21 (17–25) months and 58 (53–66) months of follow-up after AVR.

Table 3 shows LVMI, LV GLS, LVEF, and stroke volumes at baseline and follow-up. At baseline, due to eccentric remodelling, LVMI was larger in patients with AR (154.9 ± 40.0 vs. 127.1 ± 36.0 g/m², $P<0.001$; respectively) and stroke volume was larger (115.4 ± 45.6 vs. 80.9 ± 22.4 , $P<0.001$; respectively) compared to patients with AS. Both patients with AR and AS showed preserved LVEF (53.5 ± 9.1 vs. $55.9 \pm 11.4\%$, $P=0.117$; respectively) and moderately impaired LV GLS (-15.3 ± 4.1 vs. $-15.1 \pm 4.3\%$, $P=0.685$; respectively). Examples of a patient with AR and a patient with AS before and after AVR are displayed in Figure 1.

Figure 2 presents the changes in LVMI (Figure 2A) and LV GLS (Figure 2B) between sequential time points after AVR for both groups. Despite differences in LV remodelling at baseline, both AS and AR patients showed LV mass regression and improvement in LV GLS after AVR. Both patient groups showed significant LVMI

Table 2 Baseline echocardiographic and procedural characteristics of patients with AS and AR undergoing AVR

	Total population (n = 211)	Patients with AS (n = 132)	Patients with AR (n = 79)	P-value
Baseline echocardiography				
Valve anatomy, n (%)				0.010
Bicuspid	49 (23)	23 (17)	26 (33)	
Tricuspid	162 (77)	109 (83)	53 (67)	
LVEDD (mm)	53.8 ± 9.2	50.2 ± 8.3	59.8 ± 7.4	<0.001
LV end-diastolic volume (mL)	142.9 ± 62.7	115.2 ± 42.0	188.6 ± 64.6	<0.001
LV end-systolic volume (mL)	66.8 ± 39.2	53.5 ± 33.4	88.7 ± 38.3	<0.001
LV ejection fraction (%)	55.0 ± 10.6	55.9 ± 11.4	53.5 ± 9.1	0.117
Aortic valve area (cm ²)	1.42 ± 1.1	0.82 ± 0.2	2.72 ± 1.0	<0.001
AVPG (mmHg)	53.3 ± 32.3	71.5 ± 24.6	21.9 ± 15.0	<0.001
AVMG (mmHg)	32.7 ± 21.5	44.4 ± 17.5	12.5 ± 9.2	<0.001
Procedural variables				
Concomitant CABG, n (%)	74 (35)	55 (42)	19 (24)	0.009
Valve type, n (%)				0.293
Biological	135 (64)	95 (72)	40 (51)	
Mechanical	59 (28)	37 (28)	22 (28)	
Aortic valve repair	18 (9)		18 (23)	
Implanted valve size (mm), n (%)				<0.001
21	24 (12)	19 (14)	5 (6)	
23	53 (25)	47 (36)	6 (8)	
25	62 (29)	42 (32)	19 (24)	
27	34 (16)	19 (14)	15 (19)	
29	22 (10)	5 (4)	17 (22)	
CPB duration (min)	149 (116–189)	140 (113–183)	170 (120–217)	0.053
Aorta clamp time (min)	116 (80–143)	109 (78–139)	122 (83–154)	0.109

AR, aortic regurgitation; AS, aortic stenosis; AVMG, aortic valve mean gradient; AVPG, aortic valve peak gradient; AVR, aortic valve replacement or repair; CABG, coronary artery bypass grafting; CPB, cardiopulmonary bypass; LV, left ventricular; LVEDD, left ventricular end-diastolic diameter.

regression within 1 year after AVR (P for time < 0.001), although this decline was more pronounced in patients with AR (P for interaction = 0.001), leading to comparable LVMI after AVR in both groups (AR: 126.1 ± 30.5 vs. AS: 113.4 ± 31.6 g/m², $P = 0.512$). LVMI continued to regress in both AS and AR patients from 1- to 2-year follow-up (P for time < 0.001) and from 2- to 5-year follow-up (P for time = 0.041) with a slightly steeper decline for the AR patient group during the second year (P for interaction = 0.08). At 2- and 5-year follow-up, no differences in LVMI were observed between the groups ($P = 0.217$ and $P = 0.485$, respectively).

At 1-year follow-up after AVR, the AR group showed a further decline in LV GLS (P for time = 0.810) resulting in a significantly more impaired LV GLS compared to the AS group ($-14.0 \pm 3.9\%$ vs. $-15.0 \pm 4.0\%$, respectively; $P = 0.014$). Thereafter, LV GLS significantly improved over time during the second year (P for time < 0.001) in similar magnitude for both AS and AR patients (P for interaction = 0.152) and remained stable in the period between 2- and 5-year follow-up.

Discussion

The present study demonstrates that in severe AS and severe AR patients undergoing AVR, LV mass regression and changes in LV GLS

are similar despite showing different LV remodelling before AVR. However, in patients with AR, the reduction in LVMI was more pronounced during the first year of follow-up as compared to AS patients. In addition, LV GLS showed an initial impairment in AR patients during the first year after AVR. The reduction (within 1 year) in LV end-diastolic dimensions as a response to the relief of volume overload led to a reduction in LVMI and, according to the Frank–Starling law, an impairment in LV GLS in AR patients. In contrast, relief of pressure overload in patients with AS was characterized by a stable LV GLS and a more sustained regression of LVMI during follow-up.

Pre- and post-operative LV remodelling in AS and AR

Haemodynamic overload conditions imposed onto the left ventricle, characterized by pressure overload in AS and both volume and pressure overload in AR, result in remodelling of the LV to normalize wall stress and to maintain the systolic function. AS is characterized by LV concentric hypertrophy, whereas in AR eccentric hypertrophy with LV dilatation is observed. These structural changes are accompanied by myocardial oxygen demand mismatch and progressive myocardial fibrosis that may lead to LV systolic dysfunction in both AS and AR,

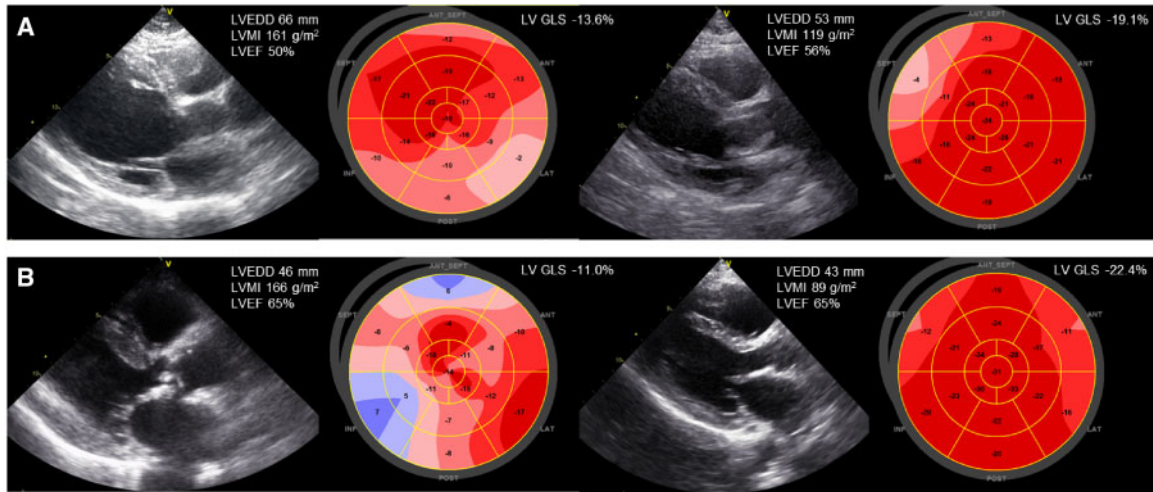


Figure 1 Examples of two patients with preserved LV ejection fraction before (left) and 2 years after AVR (right). (A) Patient with AR with eccentric LV hypertrophy due to increased LVEDD. (B) Patient with AS with concentric LV hypertrophy due to thickening of inter-ventricular septum and posterior wall. Despite differences in LV remodelling, both patients show LV mass regression and improvement of LV GLS after AVR. AVR, aortic valve replacement or repair; GLS, global longitudinal strain; LV, left ventricular; LVEDD, left ventricular end-diastolic diameter; LVEF, left ventricular ejection fraction; LVMI, left ventricular mass index.

Table 3 Course of LVMI, LV GLS, LV ejection fraction, and stroke volume in AS and AR after AVR

	Patients with AS	Patients with AR	P value
At baseline	N = 132	N = 79	
LVMI (g/m ²)	127.1 ± 36.0	154.9 ± 40.0	<0.001 ^a
LV GLS (%)	-15.1 ± 4.3	-15.3 ± 4.1	0.685 ^a
LVEF (%)	55.9 ± 11.4	53.5 ± 9.1	0.117 ^a
Stroke volume (mL)	80.9 ± 22.4	115.4 ± 45.6	<0.001 ^a
At 1-year follow-up	N = 119	N = 72	
LVMI (g/m ²)	113.4 ± 31.6	126.1 ± 30.5	0.512 ^b
LV GLS (%)	-15.0 ± 4.0	-14.0 ± 3.9	0.014 ^b
LVEF (%)	55.1 ± 9.3	50.4 ± 9.7	0.310 ^b
Stroke volume (mL)	82.5 ± 22.4	82.9 ± 26.0	<0.001 ^b
At 2-year follow-up	N = 102	N = 62	
LVMI (g/m ²)	103.6 ± 26.6	110.3 ± 31.2	0.217 ^b
LV GLS (%)	-16.8 ± 4.0	-16.3 ± 4.1	0.133 ^b
LVEF (%)	56.4 ± 8.8	55.1 ± 8.6	0.158 ^b
Stroke volume (mL)	86.2 ± 20.1	89.2 ± 25.0	0.913 ^b
At 5-year follow-up	N = 129	N = 53	
LVMI (g/m ²)	100.7 ± 28.8	104.7 ± 28.9	0.485 ^b
LV GLS (%)	-17.2 ± 4.1	-17.1 ± 3.6	0.301 ^b
LVEF (%)	56.8 ± 8.9	54.8 ± 8.1	0.255 ^b
Stroke volume (mL)	85.9 ± 23.5	85.9 ± 27.3	0.328 ^b

AR, aortic regurgitation; AS, aortic stenosis; AVR, aortic valve replacement or repair; GLS, global longitudinal strain; LV, left ventricular; LVEF, left ventricular ejection fraction; LVMI, left ventricular mass index.

^aCalculated using the Student's independent t-test.

^bCalculated using linear mixed models correcting for gender, age, LV end-diastolic diameter at baseline, and time between echocardiographic follow-up points.

even before symptoms develop.^{3,16} At this time, AVR is strongly recommended.^{1,2} Following surgery, the immediate reduction in afterload has been shown to improve LV systolic function and to result in prompt LV mass reduction in both AS and AR patients.^{16,17} In AS, multiple studies have demonstrated that excessive LV hypertrophy is independently associated with increased mortality after AVR.^{18–20} Similarly, larger LV mass in severe AR patients is associated with mortality and impaired LV systolic function after intervention.^{21,22} LV mass regression after AVR seems critical for clinical improvement and long-term survival.²³ Several studies have described the time course of LV mass regression in severe AS patients within 12 months after intervention.^{24,25} In a more recent longitudinal assessment of the cohort A in the Placement of AoRTic TraNscatheter Valve Trial (PARTNER) I trial, Daubert *et al.*²⁶ showed sustained LV mass regression up to 5 years after surgical AVR. In contrast, the post-operative LV mass regression process in patients with AR is less extensively studied. Studies performing sequential measurements of LV dimensions showed a steep decline of indexed LV diameters in the early post-operative period and at 1- to 2-year follow-up with stabilization of this reduction thereafter.^{22,27}

How the time course of LV mass regression after aortic valve intervention in severe AS compares to patients with severe AR has not been extensively studied. Monrad *et al.*¹⁷ demonstrated that the greatest fall in LVMI occurred 1–2 years after surgery for both AS and AR and that LV mass regression continued until late post-operative follow-up (mean 8.1 ± 2.9 years). More recently, and using the presently recommended formula for the calculation of LVMI¹³, Une *et al.*²⁸ showed a steep decline in LVMI in both AS and AR during the first 24 months after surgery without further significant reduction at longer term follow-up in both patient groups. Of note, patients with AR showed a larger LV mass regression as compared to AS patients.²⁸ However, the reduction in LVMI among AR patients

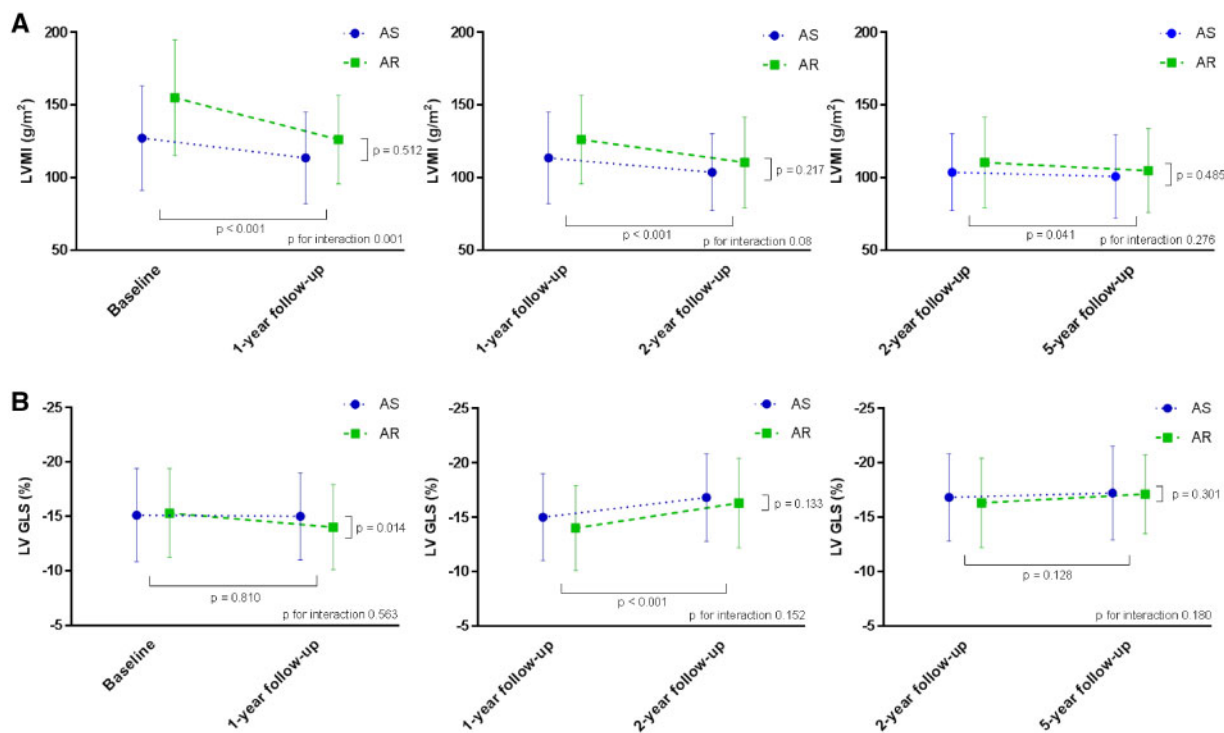


Figure 2 Changes in LV mass index (A) and LV mechanics (B) over time after AVR for patients with AS and AR after correction for age, gender, baseline LV end-diastolic diameter, and time between echocardiographic follow-up points. AR, aortic regurgitation; AS, aortic stenosis; AVR, aortic valve replacement or repair; GLS, global longitudinal strain; LV, left ventricular; LVEF, left ventricular ejection fraction; LVMI, left ventricular mass index.

occurred at a slower pace than in patients with AS. The present study provides further insight in the time course of LV mass regression in AS and AR patients by demonstrating, using sequential echocardiographic measurements, a marked decline of LVMI in both patient groups within 1 and 2 years after surgery which continued up to 5 years after AVR. Interestingly, patients with AR showed a more pronounced and faster regression in LV mass during the early post-operative phase as compared to AS patients.

Pre- and post-operative LV mechanics in AS and AR

Timing for AVR in both AS and AR is largely guided by the presence of symptoms or LV systolic dysfunction, conventionally expressed as a LVEF <50%.^{1,2} However, multiple studies have demonstrated that even in an early stage, when patients are still asymptomatic, subclinical myocardial dysfunction can occur.^{7,11,12} Impaired LV GLS has been shown in patients with severe AS or AR with preserved LVEF and has been associated with poor prognosis, even after AVR.^{10,11,29–31} Studies evaluating the time course of LV GLS after surgery in AS and AR are limited. In severe AR patients treated with AVR, Smedsrud *et al.*³² and Regeer *et al.*³³ demonstrated a significant improvement of LV GLS normalized for LV end-diastolic volume at 229 ± 159 days and 26 (16–64) months, respectively. For severe AS, LV GLS has been described to improve as early as several days after AVR.^{34,35} The present study is first to describe an impaired LV GLS in AR patients in the early period (within 1 year) after AVR compared to AS patients, reflecting the different responses in LV mechanics to a

relief of volume vs. pressure overload. According to the Frank–Starling law, volume overload by AR results in an increase in preload which stimulates force of contraction by stretching of the myocytes to maintain LV systolic function in the progressively dilated LV. After AVR, this preload will dramatically fall and chamber dimensions will decrease, resulting in a decrease of activation of the Frank–Starling mechanism. As a consequence, myocardial contraction force will drop and most likely, longitudinal shortening of myocardial fibres will also decrease, manifesting as a more impaired LV GLS as seen in the present study.

Limitations

Several limitations should be acknowledged. The present study was retrospective in design and performed in a single tertiary centre. Referral for AVR was left at the discretion of the treating cardiologist. Therefore, referral and selection bias could have been introduced. In this study, LVMI was calculated on 2D transthoracic echocardiography using the Devereux formula.¹³ Especially in the presence of eccentric hypertrophy, a prevalent finding in AR, this approach may be inaccurate due to reliance on the LV end-diastolic diameter of this formula. However, our analyses were corrected for LV end-diastolic diameter at baseline, resulting in a fair comparison between AS and AR despite differences in remodelling at baseline. Future studies using 3D imaging methods (e.g. cardiac magnetic resonance imaging) are needed to optimize accurate assessment of LV mass.

Conclusions

After AVR, LV remodelling and improvement of LV mechanics occur both in AS and AR. However, post-operative LV mass regression in AR patients was characterized by a steep decline within 1 year after surgery and associated with a less preserved LV GLS compared to patients with AS, who showed a more gradual and sustained LV mass regression and improvement of LV mechanics. These findings provide further insight in the differences in myocardial response to the relief of pressure overload in AS and a combination of pressure and volume overload in AR, as displayed by the distinct patterns in LV remodelling and mechanics.

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