ORIGINAL ARTICLE

Determinants of the Left Ventricular Ejection Fraction in Patients with Severe Aortic Stenosis

Determinantes de la fracción de eyección ventricular izquierda en pacientes con estenosis aórtica grave

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ABSTRACT

Background: In severe aortic stenosis (AS), reduced left ventricular ejection fraction (LVEF) (<50%) may be attributed to decreased contractility, afterload mismatch (AM) or a combination of both mechanisms. However, when LVEF is ≥50% some patients may have decreased contractility.
Objective: The aim of this study was to assess contractility level (CL), AM and ventricular geometry as determinants of LVEF in patients with severe AS.
Methods: Doppler echocardiography was used to study 184 patients with severe AS and 71 normal individuals (N). Contractility was assessed as the difference between measured and predicted midwall fractional shortening (mFS) minus 2 standard errors for an established meridional end-systolic stress (ESS). Patients with AS were divided into 4 groups: LVEF <50% (n=78), LVEF 50-59% (n=27), LVEF 60-69% (n=45), and LVEF ≥70% (n=34).
Results: Decreased CL was observed in approximately half of the patients with LVEF <60% and in a lesser degree in patients with LVEF ≥60%. Afterload mismatch was found in patients with LVEF <50% with decreased (34%) as well as preserved (45%) CL.
Conclusions: Half of the patients with LVEF <60% presented decreased CL and to a lesser extent this occurred in the rest of the patients, even with LVEF ≥70%. The overestimation of systolic function using LVEF seems to be related to the degree of concentric hypertrophy.

Keywords: Aortic stenosis - Systolic function - Contractility - Ejection fraction

RESUMEN

Introducción: En la estenosis aórtica (EAo) grave la fracción de eyección ventricular izquierda (FEVI) reducida (< 50 %) puede deberse a una disminución de la contractilidad, exceso de poscarga (EP) o a una combinación de ambos mecanismos. Sin embargo, cuando la FEVI es ≥ 50 % algunos pacientes (pacientes) pueden tener la contractilidad disminuida.
Objetivo: Evaluar el nivel de contractilidad (NC), EP y geometría ventricular como determinantes de la FEVI en pacientes con EAo grave.
Material y métodos: Se estudiaron 184 pac con EAo grave y 71 individuos normales (N) mediante eco-Doppler cardíaco. El NC se determinó mediante la diferencia entre la fracción de acortamiento mesoparietal (FAm) medida (EAo) y la FAm predicha (N) menos 2 errores estándar para un valor determinado de estrés meridional de fin de sístole (EFS). Los pac con EAo fueron divididos en 4 grupos: FEVI < 50 % (n=78), FEVI 50 - 59 % (n=27), FEVI 60 - 69 % (n=45) y FEVI ≥ 70 % (n=34).
Resultados: El NC estuvo disminuido aproximadamente en la mitad de los pac. con FEVI < 60 % y en menor grado en los pacientes con FEVI ≥ 60 %. El EP se observó en los pacientes con FEVI < 50 % con NC disminuido (34 %) como así también con NC conservado (45 %)
Conclusiones: La mitad de los pacientes con FEVI < 60 % presentó NC disminuido y en menor proporción ello ocurrió en el resto de los pacientes, incluso hasta con FEVI ≥ 70 %. La sobrestimación de la función sistólica utilizando la FEVI parece estar relacionada con el grado de hipertrofia concéntrica

Palabras claves: Estenosis aórtica – Función sistólica - Contractilidad – Fracción de eyección

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INTRODUCTION

In aortic stenosis (AS), a reduction in left ventricular ejection fraction (LVEF) below 50% is a class I indication for aortic valve replacement in the absence of symptoms according to guidelines of valve disease management, (1-3) due to poor short-term prognosis. However, patients with LVEF between 50% and 60%, same as those with LVEF <50%, have been reported to have worse prognosis when compared with those with LVEF >60%. (4-6) In AS, LVEF may decrease due to reduced contractility, afterload mismatch (AM) or a combination of both mechanisms. Since LVEF expresses endocardial wall motion from end-diastole to end-systole, it is influenced by ventricular geometry, as in the presence of concentric hypertrophy, this motion, as well as wall thickness, increases and can produce a normal LVEF value even when contractility is reduced. (7) To correct the apparent dissociation between chamber function (LVEF) and muscle function, it is more appropriate to use the midwall shortening fraction (mSF) adjusted to afterload [end-systolic stress, (ESS)] in the presence of concentric hypertrophy to estimate the contractility level (CL). (8-11) The aim of this study was to evaluate the CL, AM and ventricular geometry as determinants of LVEF in patients with severe AS.

METHODS

A total of 184 patients (112 men and 72 women) with mean age of 69±11 years and severe AS defined as aortic valve area (AVA) index (AVAI) <0.6 cm^2/m^2, in whom Doppler echocardiography had been requested, were prospectively included in the study. The protocol was approved by the Hospital's Teaching and Research Committee. Patients with mitral annulus calcification and moderate or severe aortic or mitral regurgitation were excluded. A history of hypertension was considered if the patient had previously been prescribed with antihypertensive treatment, and history of ischemic heart disease if the patient presented one or more of the following criteria: 1) history of AMI, percutaneous coronary intervention or coronary artery bypass graft surgery, 2) obstructions >50 % in the coronary arteries assessed by coronary angiography and 3) akinesia in the echocardiogram. As a control group, 71 patients with mean age of 69±13 years and no cardiovascular history or risk factors, or systemic diseases, were analyzed. Prior to a full Doppler echocardiogram, all patients were interrogated to detect the presence of coronary risk factors and symptoms, and a cardiovascular physical examination, blood pressure measurement and carotid pulse recording was performed.

Echocardiogram and cardiac Doppler: The study was carried out with an ESAOTE MyLab 40 ultrasound machine and 2.5-3.5 MHz transducer, with the patient in left lateral decubitus position, using the electrocardiographic lead II tracing as reference. M-mode and two-dimensional echocardiographic measurements, calculation of left ventricular (LV) endocardial fractional shortening (eFS), relative wall thickness (RWT), end-diastolic volume (EDV) (Simpson method), end-systolic volume (ESV) and LVEF were performed according to ASE criteria. (12). Left ventricular midwall fractional shortening (mFS) was estimated using the Koide formula: (13)

$$mFS = [(LVDD+h)-(LVSD+2a')/(LVDD+h)] \times 100$$

where DD: diastolic diameter; SD: systolic diameter; h: composite of diastolic posterior wall (PW) and interventricular septum (IS) thickness (dPW+dIS/2); hfs: composite systolic wall thickness (sPW+sIS/2) and a´: systolic midwall point estimated from the following formula:

$$a' = 1/2 \left[\sqrt{\frac{hfs\ (2\ LVDD+h)\ (LVSD+hfs)}{LVDD+h} + LVDD^2 - LVSD} \right]$$

Left ventricular mass was estimated according to the Devereux formula (14) and mass index (MI) as LV mass normalized to body surface area. According to MI and RWT, ventricular geometry was classified as normal, concentric remodeling, concentric hypertrophy, and eccentric hypertrophy. (15) Maximum aortic transvalvular velocity, mean gradient (MG) and the flow curve integer were recorded with continuous Doppler echocardiography from apical, right parasternal, subxiphoid and suprasternal views. A pulsed Doppler echocardiography 5-chamber view was used to obtain LV outflow tract velocity. Effective AVA, estimated using the continuity equation, AVAI, energy loss index (ELI), stroke volume index and aortic flow were calculated according to ASE criteria. (16) Pulsed tissue Doppler echocardiography of the lateral and septal mitral annulus was recorded and peak e' wave velocity (septal and lateral average) was measured. Transmitral flow was recorded to estimate peak E wave velocity, peak A wave velocity, E/A and E/e' ratios. Pulmonary artery systolic pressure and left atrial (LA) volume index were estimated according to ASE. (16) An average of 3 consecutive measurements was considered for each parameter evaluated. After performing the Doppler echocardiogram, the carotid pulse was recorded with a TPW-01 A pulse transducer connected to a TOSHIBA SSH140A ultrasound machine, and blood pressure was measured with a sphygmomanometer in the right arm with the patient in left lateral decubitus position. The carotid pulse recording (17) to obtain end-systolic pressure (ESP) was calibrated according to the method used in our laboratory. End-systolic meridional wall stress (ESS) was estimated as an afterload index, using the Grossman formula. (18) Contractility was evaluated using the mFS-ESS relationship in the control group with the following regression equation: mFS=28.15-0.12 x ESS, with a standard error of estimate (SE) of 3.75% (r=0.41 p <0.001). For a certain level of ESS (afterload) a value lower than the predicted mFS by the regression equation minus 2 SE was considered as decreased contractility in patients with AS. The CL was defined as the measured mFS minus the predicted mFS for a given ESS value, so a positive value indicates normal contractility and a negative value decreased contractility. (19) Afterload mismatch was considered when ESS was >77 g/cm^2 determined as the average ESS of the control group (53±12 g/cm^2) + 2 standard deviations. The difference between measured LVEF and CL (Δ LVEF-CL) was calculated to evaluate the possible effect of ventricular geometry on the overestimation of systolic function using LVEF.

To assess global LV afterload, valvulo-arterial impedance (Zva) was estimated as:

Zva (mm Hg/mL/m^2) = (SBP (mmHg)+ MATG (mm Hg))/SVI (mL/m^2);

where SBP is systolic blood pressure, MATG mean aortic transvalvular gradient corrected for pressure recovery according to the Baumgartner formula (20) and SVI systolic volume index.

Diastolic function was assessed using the E/A ratio, E/e'

ratio, LA volume index, and pulmonary artery systolic pressure. Patients with AS were divided into 4 groups: LVEF <50% (n=78), LVEF 50-59% (n=27), LVEF 60-69% (n=45) and LVEF ≥70% (n=34).

Statistical analysis

SPSS 25 software was used to perform the statistical analysis. Continuous variables were expressed as mean±standard deviation and categorical variables as percentage. The chi square test was used to compare categorical variables, the analysis of variance for continuous variables and Pearson's correlation r coefficient as a correlation test. A p value <0.05 was considered statistically significant.

Ethical considerations

The Institutional Ethics Committee approved the study, waiving an informed consent due to the observational nature of the study.

RESULTS

There were no significant differences in patient age or body surface area between the 4 groups. Male gender predominated in patients with LVEF <50% and LVEF 50-59%; its prevalence was similar to female gender in the LVEF 60-69% group, while the number of women was higher in the LVEF ≥70% (p <0.01) group (Table 1). The presence of ischemic heart disease, cardiovascular risk factors and chronic obstructive pulmonary disease was more frequent in patients with LVEF <50% and similar among the remaining 3 groups. Regarding symptoms, dyspnea class III-IV predominated in the LVEF <50% group (p <0.01), and angina and dyspnea class I-II in the rest of the groups. There were no significant differences between groups when considering systolic, diastolic and end-systolic pressure. Aortic stenosis severity was similar in the 4 groups according to AVA, AVAI, peak aortic velocity, MG and ELI. Systolic volume index and aortic flow were lower and Zva higher in patients with LVEF <50% compared with those with LVEF ≥60% (p <0.01). The presence of ischemic heart disease, cardiovascular risk factors and chronic obstructive pulmonary disease was more frequent in patients with LVEF <50% and similar among the remaining 3 groups. Regarding symptoms, dyspnea class III-IV predominated in the LVEF <50% group (p <0.01), and angina and dyspnea class I-II in the rest of the groups. There were no significant differences between the groups when considering systolic, diastolic and end-systole blood pressure. The severity of AS was similar in the 4 groups according to AVA, AVAI, peak aortic velocity, GM and ELI. SVI and aortic flow were lower and Zva higher in patients with LVEF <50% compared with those with LVEF ≥60% (p <0.01) (Table 2).

Contractility assessment: Among the 184 patients, 64 (35%) had decreased contractility (CL <0) and 120 (65%) preserved contractility (CL ≥0). When considering the groups according to LVEF, 49% (51/105) of patients with LVEF <60% had decreased contractility, significantly different with respect to patients with

Table 1. Patient characteristics

	Left ventricular ejection fraction (%)				
	< 50 (n = 78)	50 – 59 (n = 27)	60 – 69 (n = 45)	≥ 70 (n = 34)	p
Age (years)	70 ± 12	65 ± 12	71 ± 11	71 ± 11	ns
Male / female gender	56 / 22	19 / 8	25 / 20	12 / 22	< 0.001
Body surface área (m^2)	1.85 ± 0.21	1.90 ± 0.19	1.82 ± 0.19	1.76 ± 0.16	ns
Co-existing diseases and RF (%)					
Coronary heart disease	31	22	15	8	< 0.01
Previous myocardial infarction	10	2	0	0	
Hypertension	52	48	49	42	
Diabetes	15	17	12	10	
Dyslipidemia	22	23	22	23	
Smoking	25	28	26	25	
Obesity (BMI > 30 Kg/m^2)	27	33	20	24	
Chronic obstructive pulmonary disease	5	2	0	0	
Symptoms (%)					
Angina	9	33	16	9	
Syncope	0	11	11	0	
Dyspnea I-II	23	22	20	26	< 0.01
Dyspnea III-IV	54	11	20	6	
Systolic blood pressure (mm Hg)	122 ± 23	131 ± 25	135 ± 20	134 ± 21	ns
Diastolic blood pressure (mm Hg)	72 ± 13	78 ± 13	77 ± 11	76 ± 12	ns
End-systolic pressure (mm Hg)	91 ± 16	97 ± 16	97 ± 18	98 ± 19	ns

RF: Risk factors BMI: Body mass index

Table 2. Echocardiographic parameters

	Left ventricular ejection fraction (%)				
	< 50 (n = 78)	50 – 59 (n = 27)	60 – 69 (n = 45)	≥ 70 (n = 34)	p
LV ejection fraction (%)	33 ± 10	56 ± 3	65 ± 3	75 ± 4	< 0.001
Aortic valve area (cm^2)	0.65 ± 0.21	0.66 ± 0.23	0.71 ± 0.20	0.71 ± 0.20	ns
Aortic valve area index (cm^2/ m^2)	0.35 ± 0.12	0.35 ± 0.12	0.39 ± 0.12	0.40 ± 0.12	ns
Peak aortic velocity (m/s)	4 ± 1	4.1 ± 0.8	4.3 ± 0.8	4 ± 0.9	ns
Mean gradient (mmHg)	40 ± 21	39 ± 15	44 ± 18	40 ± 19	ns
Stroke volume index (mL/m^2)	30 ± 12	32 ± 7	38 ± 10 *	38 ± 11*	
Aortic flow (ml/s)	174 ± 55	183 ± 38	205 ± 53 *	191 ± 46 *	
Energy loss index (cm^2/m^2)	0.40 ± 0.15	0.39 ± 0.15	0.45 ± 0.15	0.46 ± 0.15	ns
Valvulo-arterial impedance ($mmHg/mL/m^2$)	6 ± 2	5.3 ± 1.5	4.7 ± 1.2 *	4.7 ± 1.4 *	
LV diastolic diameter (cm)	5.9 ± 0.8	5.1 ± 0.6	4.9 ± 0.6	4.7 ± 0.6	< 0.01
LV systolic diameter (cm)	4.5 ± 0.9	3.4 ± 0.6	3.0 ± 0.6	2.8 ± 0.5	< 0.01
Endocardial shortening fraction (%)	23 ± 9	33 ± 8	38 ± 7	39 ± 8	< 0.001
Midwall fractional shortening (%)	12 ± 6	16 ± 5	20 ± 4	19 ± 6	< 0.001
End-systole stress (gr/cm^2)	69 ± 28	41 ± 15	38 ± 22	33 ± 11	< 0.001
Contractility level (n (%))					
≥ 0	39 (50)	15 (56)	39 (87)	25 (74)	
< 0	39 (50)	12 (44)	6 (13) *	9 (26) *	
Δ LVEF – CL	33 ± 11	55 ± 5	61 ± 4	73 ± 6	< 0.001
Relative wall thickness	0.42 ± 0.11	0.51 ± 0.09	0.52 ± 0.10	0.54 ± 0.11	< 0.01
Mass index (gr/m^2)	178 ± 50	153 ± 43	149 ± 39	136 ± 32	<0.01.
Ventricular geometry (n (%))					
Normal	1 (1)	0	3 (7)	1 (2)	
Concentric remodeling	2 (2)	5 (19)	5 (11)	6 (18)	
Concentric hypertrophy	29 (37)	19 (70)	32 (71)	23 (68)	< 0.01
Eccentric hypertrophy	46 (59)	3 (11)	5 (11)	4 (12)	< 0.01
E/A ratio	1.8 ± 1	0.9 ± 0.3	1.0 ± 0.7	0.76 ± 0.25	< 0.01
E/e'	19 ± 8	13 ± 5	16 ± 7	14 ± 8	ns
LA volume index (ml/m^2)	60 ± 16	48 ± 13	54 ± 19	45 ± 12	< 0.01
Pulmonary artery systolic pressure (mmHg)	54 ± 18	36 ± 15	35 ± 12	32 ± 7	< 0.01

* p < 0.01 vs LVEF <50 % and LVEF 50–59 %
LV: Left ventricular LA: Left atrial Δ LVEF - CL: Difference between left ventricular ejection fraction and contractility level.

LVEF ≥ 60% [50% (LVEF <50 %) and 44% (LVEF 50-59%) vs. 13% (LVEF 60-69%) and 26% (LVEF ≥ 70%) respectively, p <0.01] (Table 2). Midwall FS was inversely correlated with ESS in patients with preserved and decreased contractility, with the two regression lines parallel to each other, but differing in the Y-axis intercept (mFS 25% vs. 16%, p <0.001). (Figure 1A). When patients in each group were divided into CL <0 and CL ≥0, it was observed that despite not having significant differences in LVEF and ESS within each group, mFS was significantly decreased in all patients with CL < 0, which implies that for similar afterload values (ESS), LVEF could not discriminate between reduced or preserved contractility (Table 3). The Δ LVEF-CL index, which assesses the discrepancy between LVEF and CL, showed a progressive increase in groups from LVEF <50% to LVEF ≥ 70% (Table 2).

Afterload (ESS): Different from results observed in the mFS-ESS relationship, in all the 184 patients, LVEF was inversely correlated with ESS only in patients with decreased contractility and not in patients with preserved contractility (Figure 1B). In the LVEF <50% group, AM (ESS >77 gr/cm2) was present in 13 (34%) of the 38 patients with CL <0 and in 18 (45%) of the 40 patients with CL ≥0 (Table 3). This implies that the cause of LVEF <50% was AM in 23% (18/78) of cases, decreased contractility in 32% (25/78) and AM and decreased contractility in 17% (13/78), leaving 28% in which LVEF decrease was related to an increase in ESS (mean 69±28 gr/cm2), but less than the limit considered as AM. In the rest of the groups, AM was only recorded in 2 patients with LVEF 60-69%.

Ventricular geometry: Eccentric hypertrophy predominated in patients with LVEF <50%, while concentric hypertrophy was more frequent in the rest of

the patients. The Δ LVEF-CL index had a logarithmic correlation with RWT (Figure 2), showing an increase in Δ LVEF-CL up to RWT 0.65, and then decreasing.

Diastolic dysfunction was present in all groups, but was more evident in patients with LVEF <50% (Table 2) characterized by the increase of LA volume index (p <0.01), pulmonary artery systolic pressure (p <0.01) and higher E/e' ratio, although the difference was not significant with respect to the other groups. The aforementioned findings indicate an increase in LV

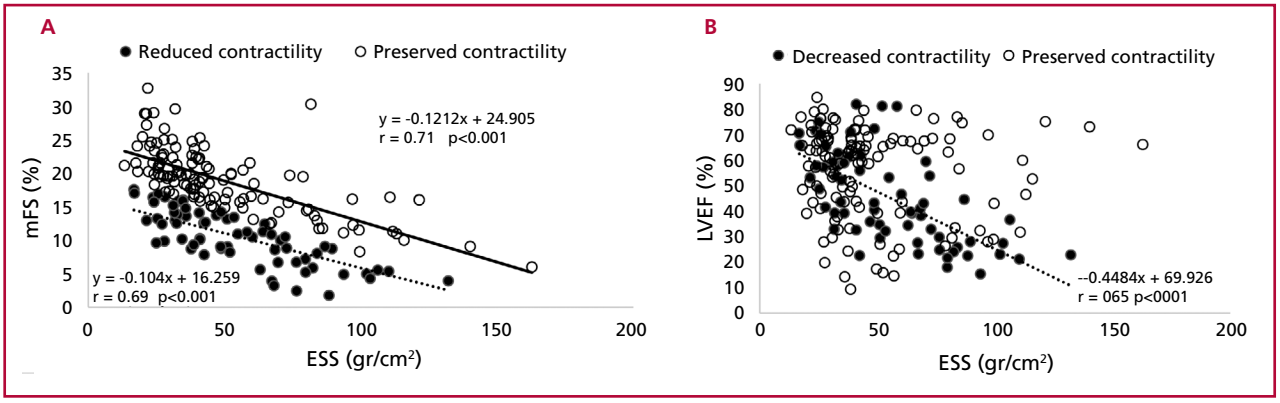


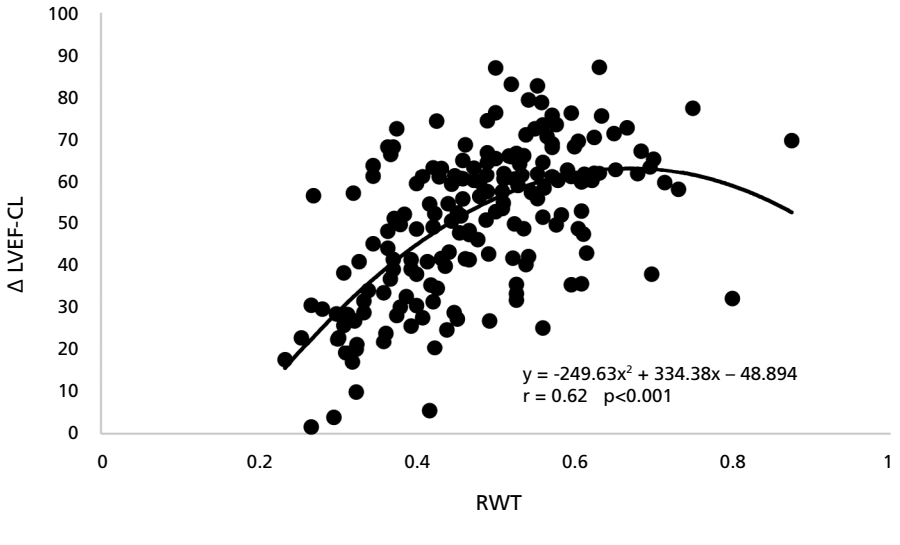
Fig. 1. A. Correlation between midwall fractional shortening (mFS) and end-systolic stress (ESS) in patients with preserved and decreased contractility. **B.** Correlation between left ventricular ejection fraction (LVEF) and end-systolic stress (ESS) in patients with preserved and decreased contractility

Table 3.

	Left ventricular ejection fraction (%)							
	< 50 (n=78)		50 - 59 (n=27)		60 – 69 (n=45)		≥ 70 (n=34)	
	CL<0 n=38 (49%)	CL ≥0 n=40 (51%)	CL <0 n=12 (55%)	CL ≥0 n=15 (45%)	CL <0 n=6 (14%)	CL ≥0 n=37 (86%)	CL <0 n=9 (27%)	CL ≥0 n=25 (73%)
LVEF (%)	32 ± 9	34 ± 11	55 ± 0.8	53 ± 1.3	62 ± 0.7	63 ± 1.2	72 ± 1.7	74 ± 3
mFS (%)	8 ± 3	16 ± 5 *	12 ± 2	19 ± 5 *	13 ± 3	20 ± 5 "	14 ± 4	21 ± 4 Δ
ESS(gr/cm²)	67 ± 25	70 ± 31	43 ± 20	37 ± 8	37 ± 4	46 ± 3,4	30 ± 12	31 ± 9
AM	13 (34 %)	18 (45 %)	-	-	2 (4 %)	-		
RWT	0,.42 ± 0.11	0.41 ± 0.11	0.55 ± 0.11	0.48 ± 0.04	0.61 ± 0.07	0.49 ± 0.1^	0.59 ± 0.13	0.51 ± 0.11

CL<0 vs NC≥0: *p< 0.0001; "p< 0.0002 ^p< 0.01; Δp< 0.00001
CL Contractility level. LVEF: Left ventricular ejection fraction. mFS: midwall fractional shortening. ESS: End-systolic stress, AM: Afterload mismatch, RWT: Relative wall thickness.

Fig. 2. Correlation between the difference between left ventricular ejection fraction and contractility level (Δ LVEF - CL) and relative wall thickness (RWT).



diastolic pressures, suggesting the use of the preload reserve as a compensatory mechanism against AM.

DISCUSSION

The main findings of the present study are: 1) Forty-nine per cent of patients with LVEF <60% presented decreased contractility, 2) LVEF could not discriminate between preserved and decreased contractility, 3) the discrepancy between LVEF and CL increased with the degree of concentric hypertrophy, 4) AM (with or without decreased contractility) was responsible for LVEF <50% in 40% of patients.

Left ventricular ejection fraction is the most frequently used parameter to determine prognosis in most heart diseases. Its value is assessed by the level of contractility and loading conditions, especially afterload. Quantification is carried out (regardless of the method) by measuring volume (from the endocardial contour) at end-diastole and end-systole, normalized to end-diastolic volume. In the presence of concentric hypertrophy, increased wall thickness causes a reduction in ventricular diameter, magnifying the endocardial motion in such a way that the LVEF can be within normal limits in the presence of decreased contractility. (21) This is because LVEF expresses chamber function influenced by ventricular geometry, and it is more appropriate to use mFS, which assesses the shift of the midwall point towards the epicardium during systole, reflecting muscle function. (9, 10, 22) Contractility is defined as the inherent capacity of the myocardium to contract independently of preload and afterload, (23) so it is necessary to use a shortening parameter (LVEF or mFS) normalized to afterload, represented by ESS, in order to evaluate it. Carabello (24) used the LVEF-ESS relationship in 14 patients with AS, heart failure and reduced LVEF (28%±3%) and observed that in most patients LVEF improved after aortic valve replacement due to AM. Ito (25) studied 445 patients evaluating contractility with the mFS-ESS relationship and found that it was decreased in 58% of patients with LVEF <60% and in 24% with LVEF ≥60%, in agreement with our findings. We added the group with LVEF ≥70% in which contractility was decreased in 26% of cases, evidencing that a normal LVEF does not allow us to establish that the inotropic state is also normal. The discrepancy between LVEF and CL was evaluated using the Δ LVEF - CL index which had a logarithmic relationship with RWT, proportionally increasing up to RWT=0.65 and then decreasing, suggesting that this compensatory mechanism (concentric hypertrophy with increased RWT) ends when this value is reached and LVEF cannot increase further. In AS, the increase in LV systolic pressure secondary to valve obstruction stimulates hypertrophy by incorporating sarcomeres added in parallel with increased wall thickness and, according to Laplace's law, normalization of systolic wall stress with concentric hypertrophy. (26) This pattern was more frequent in patients with LVEF >50%, but not in those with LVEF <50% in whom eccentric hypertrophy was observed. In a retrospective study, Ito (27) observed in 928 patients with severe AS and reduced LVEF, who had had a previous echocardiogram when AS was moderate, that they already had previous reduced LVEF with eccentric hypertrophy, while those with LVEF ≥60% did not modify the type of remodeling over time. This could be due to the fact that the patients would have a previous myocardial disorder, secondary to comorbidities (hypertension, ischemic heart disease, diabetes, etc.) which would condition remodeling before the AS is severe, differing from the classic concept that pressure overload only induces concentric hypertrophy. There are other techniques, such as global strain, to assess ventricular function. (28, 29) However, this technique combines the complex relationship between contractility, (11) loading conditions (30,31) and ventricular geometry, (32) so it cannot be used to estimate contractility if it is not analyzed in relation to afterload. Stokke et al (21) studied the relationship between LVEF, strain, and geometry and concluded that LVEF can be preserved despite the reduction in longitudinal and circumferential strain due to the effect of concentric hypertrophy and the consequent decrease in end-diastolic volume. Circumferential strain contributes more than longitudinal strain in maintaining preserved LVEF according to these authors, which supports the use of mFS, as circumferential strain assesses the short-axis midwall circumference shortening and mFS the diameter of this circumference. (33) The decrease of longitudinal strain would depend on subendocardial fiber dysfunction, while the decrease of circumferential strain and mFS reflects a transmural involvement.

In accordance with our and other authors' findings, the limit of reduced LVEF in AS should be reconsidered probably to 60%, taking into account that, in any case, 1 in 4 patients with values higher than 60% could have reduced contractility More studies are required to determine the prognostic value of decreased contractility with preserved LVEF.

CONCLUSION

Left ventricular ejection fraction was inversely correlated with afterload (ESS) only in patients with decreased contractility. Half of the patients with LVEF <60% presented decreased contractility, and the proportion was lower in the rest of the patients even with LVEF ≥70%. Afterload mismatch was observed in patients with LVEF <50% regardless of the level of contractility. The overestimation of systolic function using LVEF seems to be related to the degree of concentric hypertrophy. The determinants of LVEF can be quantitatively evaluated using the mFS-ESS relationship and RWT.

Conflicts of interest

None declared.

(See authors' conflict of interests forms on the web/Additional material.)

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