Effect of Effective Arterial Elastance on Left Ventricular Systolic Function in Severe Valvular Aortic Stenosis

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ABSTRACT

Background
Left ventricular dysfunction in valvular aortic stenosis (AS) is related to an elevated afterload. This increase may be related to a reduction in aortic valve area (AVA), to changes in ventricular geometry and to the characteristics of the arterial vessels. Effective arterial elastance (E_a) is an index of arterial vascular load which incorporates the characteristic arterial impedance, arterial resistance and arterial compliance.

Objective
To assess the effect of E_a on left ventricular systolic function in severe valvular AS.

Material and Methods
We prospectively studied 54 patients with severe AS; transvalvular gradient, AVA, valvular resistance and energy loss index were assessed. E_a was estimated as the end-systolic pressure/stroke volume ratio. Carotid blood pressure waveform was calibrated against blood pressure measured by conventional cuff sphygmomanometry to calculate end-systolic pressure. We estimated global afterload by the "valvulo-arterial impedance" (Zva) formulated as follows: (systolic arterial pressure + mean net pressure gradient)/stroke-volume index. Zva considers the effect of AS and E_a. Patients were divided in three groups: G1, AS with heart failure (NYHA III-IV) (n = 13), G2, symptomatic AS without heart failure (n = 13), and G3, asymptomatic AS (n =28).

Results
Ejection fraction (EF) (%) was lower in G1 (33±15) compared to G2 (42±16) and to G3 (65±14); $p<0.01$. E_a (mm Hg/ml) and Zva (mm Hg/ml/m^2) were greater in G1 (2.46±0.8 and 6.5±2.2, respectively) than in G2 and G3 (1.83±0.52 and 1.73±0.47; $p<0.01$, and 4.8±1.0 y 4.7±1.5; $p < 0.01$, respectively). When univariate analysis was performed, Zva correlated with E_a ($r=0.88$; $p<0.0001$) and EF ($r=-0.41$; $p<0.01$). In multivariate analysis E_a and Zva were independent predictors for EF.

Conclusions
In severe AS, the increase of the vascular component of the afterload, assessed by the Ea, contributes to a reduction in systolic function.

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Key words > Aortic Valve Stenosis - Left Ventricular Function - Doppler Echocardiography

Abbreviations >

AVA	Aortic valve area	**AVR**	Aortic valve resistance
E_a	Effective arterial elastance	**BSA**	Body surface area
AS	Aortic stenosis	**LVOT**	Left ventricular outflow tract
EF	Ejection fraction	**EDV**	End-diastolic volume
HT	Hypertension	**ESV**	End-systolic volume
AMI	Acute myocardial infarction	**LV**	Left ventricle
ELI	Energy loss index	**SV**	Stroke volume
MAP	Mean arterial pressure	**Zva**	Valvulo-arterial impedance
ESP	End-systolic pressure	**CO**	Cardiac output
PP	Pulse pressure		

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BACKGROUND

The presence of symptoms – angina, syncope or heart failure – along the natural history of valvular aortic stenosis (AS) is associated with high 3-year mortality (75%) in patients not submitted to surgery. (1) However, some patients without previous history of valvular heart disease, dyspnea or angina are hospitalized with congestive heart failure in functional class III-IV (NYHA). This might be due to progressive restriction in physical activity. Afterload is inversely related to myocardial fiber shortening and to left ventricular stroke volume. Thus, reduction in ejection fraction (EF) may be due to increased afterload or reduced inotropism. (2) In the latter, EF is lower than would be expected for a given level of afterload. Ventricular dysfunction in AS is generally due to excessive afterload, except in patients with significant myocardial ischemia, extensive necrosis or associated cardiomyopathy. (3) Increased afterload is a consequence of reduced aortic valve area (AVA), changes in ventricular geometry and characteristics of the arterial vessels. Therefore, in AS the relationship EF/afterload is more complex than would be expected; the sum of external factors that resist ventricular ejection, added to the effect of the obstruction of the left ventricular outflow tract, include physical characteristics of peripheral arteries, such as arterial impedance (determined by the phasic relationship between aortic pressure and aortic volume), peripheral resistance, aortic wall stiffness, (4) inertial properties of blood and properties of the reflection waves produced in arterial bifurcations. (5-7)

Recently, Briand (8) described the concept of valvulo-arterial impedance (Zva) to quantify global afterload in AS (Figure 1A). (9) Zva includes the effect of valvular stenosis by energy loss index (ELI, which takes into account pressure recovery to estimate effective valve area) and arterial compliance. Although Briand found reduced EF in patients with low arterial compliance, there are other parameters that describe more accurately the hydraulic model of arterial vessels. Effective arterial elastance (Ea) described by Sunagawa (10) is a more precise estimation of arterial vascular load, determined by the end-systolic pressure/stroke volume ratio. The goal of our study was to analyze the effect of arterial Ea on left ventricular systolic function in patients with severe valvular aortic stenosis

MATERIAL AND METHODS

The study included 54 patients (28 men, 26 women, mean age 71 ± 10 years) who prospectively underwent an echocardiographic evaluation and were found to have severe aortic valve stenosis defined as the presence of an aortic valve area < 1 cm^2. Patients with coexisting moderate or severe aortic valve regurgitation or mitral valve regurgitation were excluded. Fifteen patients were already hospitalized; 13 of these inpatients had grade III-IV congestive heart failure (11 coronary care unit patients and 2 general ward patients) and 2 patients had been admitted to the coronary care unit with recent onset angina. Eight patients (53%) had no previous history of aortic valve disease. The remaining 41 outpatients had been referred for assessment of an aortic systolic murmur. Symptoms were present in 13 of these patients; 5 patients complaint of angina and 8 had grade I-II dyspnea. Data collected at the time of their echocardiographic evaluation included risks factors for coronary artery disease, presence or absence of symptoms and physical examination. Blood pressure was measured in left lateral decubitus position and carotid pulse waveform was recorded. Inpatients underwent echo-Doppler once they had achieved clinical stability with medical treatment. Hypertension was considered to be present when there was a history of hypertension requiring medical therapy. Patients were considered to have significant coronary artery disease if they had one of the following criteria: 1) history of myocardial infarction, coronary angioplasty, or coronary artery bypass graft surgery; 2) a > 50% stenosis on at least one epicardial artery on coronary angiography; and 3) a regional wall motion abnormality on echocardiogram.

Echocardiogram and Doppler Echocardiography

Echocardiography was performed with a TOSHIBA SS140A scanner with a 2.5 MHz transducer. The patient was positioned in left lateral recumbent and electrocardiographic lead DII was used as reference. Left ventricular outflow tract (LVOT) flow was recorded in the apical five-chamber view, placing the pulsed Doppler sample volume under the aortic valve. Aortic flow was estimated with continuous wave Doppler from the apical, subxiphoid, right parasternal, and suprasternal notch views; the highest velocity detected in any imaging window was used as the peak velocity. Recordings were taken at a sweep speed of 100 mm/s. The study was recorded on a video cassette for subsequent analysis.

The following echocardiographic parameters were assessed:

- Two-dimensional echocardiography: end-diastolic volume (EDV) and end-systolic volume (ESV) were determined by area-length method in the apical 4-chamber and 2-chamber view. The aortic diameter was measured at the level of the aortic ring, Valsalva sinus, sinotubular junction and 1 cm above the sinotubular junction in the ascending aorta.
- Doppler echocardiography: Flow-time integral in the left ventricular outflow tract and aortic flow, and peak and mean transvalvular gradients using the modified Bernoulli equation were estimated.

Calibrated Carotid Pulse Tracing

Carotid pulse tracing was recorded with a TPW – 01 A pulse transducer and blood pressure was measured in the right arm with the use of an arm-cuff sphygmomanometer in left lateral decubitus position. Carotid pulse tracing calibration was carried out according to the method used in our laboratory: (11) systolic and diastolic pressures (phase 5) are derived from the highest and lowest values on the arterial pressure trace. The distance between the maximum and minimum deflections is measured thereafter and is considered differential pressure or pulse pressure. Once pulse pressure is calculated, the distance between end-systolic point and base is measured; this is converted into mm Hg by the simple rule of three and the diastolic arterial pressure is added to it. In this way, end-systolic pressure (ESP) is determined in a non-invasive fashion.

Assessment of Left Ventricular Systolic Function

Ejection fraction was calculated by the conventional method.

Stroke volume (SV) was estimated as the product of flow velocity-time integral through the LVOT, and LVOT cross-section area and cardiac output (CO) as heart rate multiplied by SV.

Assessment of Aortic Stenosis

Effective AVA was calculated using the continuity equation (stroke volume divided by valve flow velocity-time integral).

Peak and mean aortic valve resistance (AVR) were estimated by the formula: AVR (dynes/s/ cm^{-5}) = 1.333. (Peak or mean) gradient (mm Hg)/aortic flow (ml/s). Aortic flow was determined as the ratio between SV (ml) and the left ventricular ejection time (s).

The energy loss index (ELI) was estimated. This index considers pressure recovery at the ascending aorta and is therefore more representative of the real transvalvular gradient than the gradient calculated by the continuity equation as it avoids gradient overestimation. García's formula was used. (12, 13)

$$ELI\ (cm^2/m^2) = [(AVA \cdot A_A) / (A_A \quad AVA)] / BSA$$

where A_A (cm^2) is the cross-sectional area of the aorta at the sino-tubular junction and BSA (cm^2) corresponds to body surface area.

Assessment of Systemic Arterial Hemodynamics

Pulse pressure (PP) was calculated as the difference between systolic and diastolic arterial pressures measured with the use of an arm-cuff sphygmomanometer. The PP/SV ratio was estimated as an index of arterial compliance (14) and arterial elastance (E_a), an adequate method to assess arterial mechanics, was determined as the ratio between ESP and SV (Figure 1 B). Calibrated carotid pulse tracing was used to calculate ESP, as it has been previously described. Systemic vascular resistance was estimated by the formula: (80 x MAP)/CO, where MAP is mean arterial pressure.

Assessment of Global Afterload

In patients with aortic stenosis, left ventricular afterload is determined by the degree of valvular obstruction and the

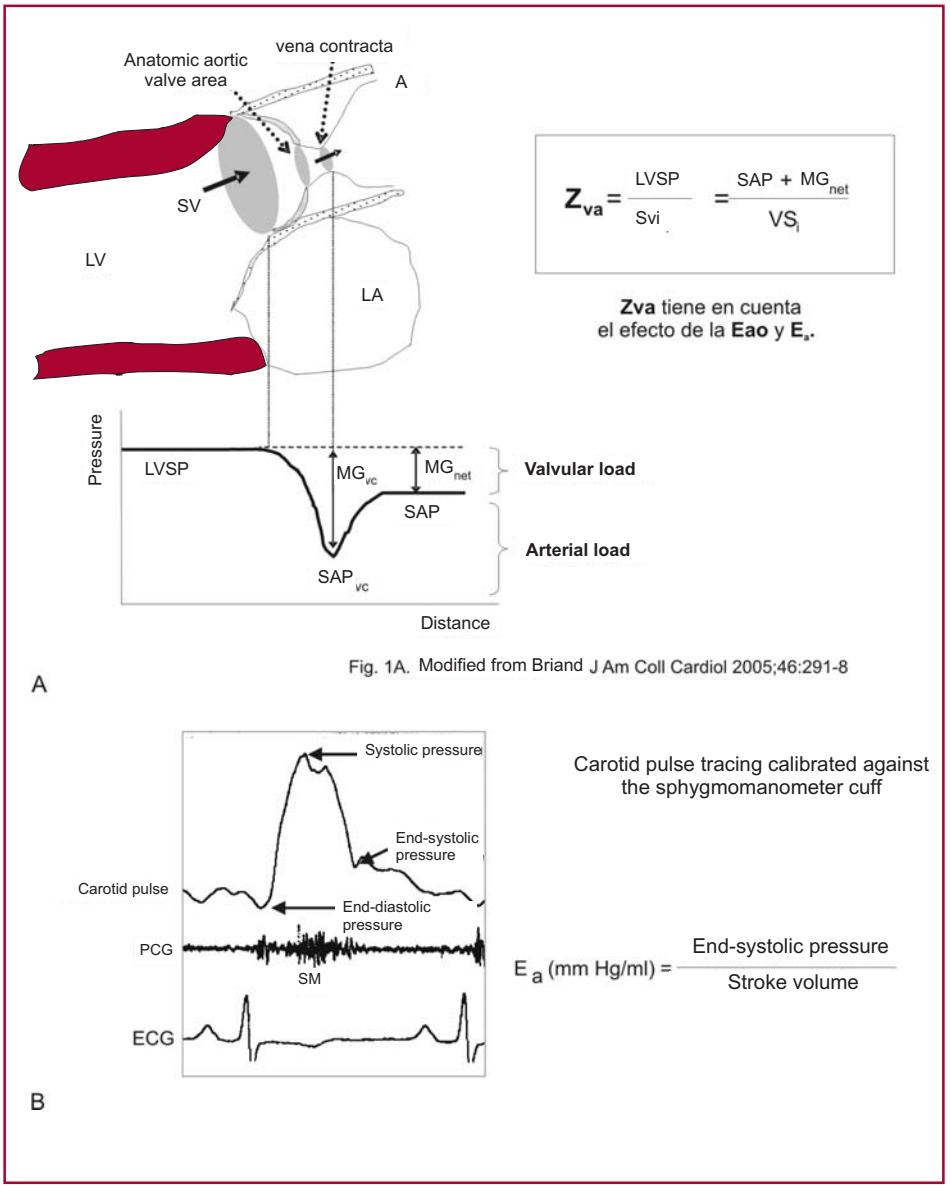

Fig. 1. A. Impedancia valvuloarterial (Zva). Véase explicación en el texto. PSVI: Presión sistólica del ventrículo izquierdo. PAS: Presión arterial sistémica. GMnet: Gradiente medio neto. VS: Volumen sistólico. VSi: Índice de volumen sistólico. GMvc: Gradiente medio a nivel de la vena contracta. VI: Ventrículo izquierdo. AI: Aurícula izquierda. Ao: Aorta. **B.** Registro del pulso carotídeo calibrado en simultáneo con fonocardiograma (FCG) y electrocardiograma (ECG) utilizado para el cálculo de la elastancia arterial efectiva (E_a). SS: Soplo sistólico

load imposed by systemic arterial system. Zva was calculated using the formula (Figure 1 A):

$$Zva\ (mm\ Hg/ml/m^2) = (SAP\ (mm\ Hg) + MGnet\ (mm\ Hg)/SV_i\ (ml/m^2)$$

Where SAP is systemic arterial pressure, MG_{net} is the mean net pressure gradients taking into account pressure recovery according to the equation proposed by Baumgartner (15) and SV_i is SV index.

Patients were divided in three groups: group 1(G1, n = 13) patients with grade III-IV heart failure; group 2 (G2, n = 13) patients with symptomatic aortic stenosis and group 3 (G3, n = 28) asymptomatic patients with aortic stenosis.

Statistical analysis

Continuous variables were expressed as mean ± standard deviation and groups were compared with analysis of variance. Univariate analysis was performed using the coefficient of correlation (r). Forward stepwise multivariate analysis was used to establish independent predictors of EF. A p value < 0.05 was considered statistically significant.

RESULTS

Table 1 shows the demographic and clinical characteristics of the study population. The distribution of age, body surface area and sex was similar among groups; however, the proportion of men was higher in groups 1 and 2. The prevalence of risk factors -hypertension, smoking habits, dyslipemia and obesity- was similar in the three groups. History of coronary heart disease was more frequent in patients in groups 1 and 2. By definition, grade III-IV dyspnea was present in the 13 patients in G1, and 4 of them also had angina. Eight patients in G2 complaint of grade I-II dyspnea, and angina was present in 5 patients.

Systemic Arterial Hemodynamics (Table 2)

There were no significant differences among the three groups in systolic arterial pressure, diastolic arterial pressure, end-systolic pressure and heart rate. Pulse pressure, a clinical parameter that estimates arterial compliance, was also similar in the three groups. Ea was significantly greater in G1 (2.46 ± 0.8 mm Hg/ml) compared to G2 and G3 (1.83 ± 0.52 mm Hg/ml and 1.73 ± 0.47 mm Hg/ml, respectively; $p < 0.01$). PP/SV ratio was greater in G1 (1.1 ± 0.41; $p < 0.03$) and G2 (0.94 ± 0.33; $p < 0.03$) than in G3. This last finding was influenced by the reduction in SV in G1 and G2 (49 ± 18 ml and 55 ± 19 ml $p < 0.03$) compared to G3 (69 ± 18 ml), due to the fact that stroke volume is the denominator in the PP/SV ratio. These findings suggest that in patients in groups 1 and 2 PP does not represent a reduction in arterial compliance as a decreased SV might mask such finding.

Left Ventricular Systolic Function

As it was expected, EF was significantly reduced in G1 (33% ± 15%) compared to G2 and G3 (42% ± 16% and 65% ± 14%; $p < 0.01$). Ejection fraction was lower in G2 than in G3; however, this difference was not significant.

Severity of Aortic Stenosis

Values of aortic valve area, peak and mean valvular resistance and ELI did not differ among groups. Although peak gradient (62 ± 21 mm Hg) and mean gradient (30 ± 13 mm Hg) were significantly lower en G1 compared to G2 (77 ± 36 mm Hg and 37 ± 22 mm Hg) and G3 (77 ± 29 mm Hg and 46 ± 18 mm Hg), this difference was not significant.

Table 1. Demographic and clinical characteristics

	Group 1 (n = 13)	Group 2 (n = 13)	Group 3 (n = 28)
Sex, n			
Male	8	9	14
Female	5	4	14
Age (years)	68 ± 14	68 ± 9	70 ± 11
Body surface area (m^2)	1.87 ± 0.19	1.83 ± 0.19	1.83 ± 0.23
Comorbidities and risk factors (%)			
Coronary artery disease	30	20	0
Previous myocardial infarction	20	20	20
Hipertension	100	100	78
Dyslipemia	43	25	12
Diabetes mellitus	28	0	33
Smoking habits	57	100	22
Obesity	43	0	22
Symptoms			
Grade I-II dyspnea	0	8	0
Grade III-IV dyspnea	13	0	0
Angina	4	5	0

Table 2. Systemic arterial hemodynamics

	Group 1 (n = 13)	Group 2 (n = 13)	Group3 (n = 28)
Systemic arterial pressure			
Systolic arterial pressure (mm Hg)	123 ± 18	124 ± 24	130 ± 20
Diastolica arterial pressure (mm Hg)	75 ± 9	75 ± 14	83 ± 11
Pulse pressure (mm Hg)	48 ± 17	48 ± 13	47 ± 16
End-systolic pressure (mm Hg)	92 ± 14	93 ± 14	97 ± 13
Heart rate (bpm)	76 ± 12	73 ± 11	69 ± 18
E_a (mm Hg/ml)	2.46 ± 0.8 *	1.83 ± 0.52	1.73 ± 0.47
PP/SV	1.1 ± 0.41†	0.94 ± 0.33‡	0.73 ± 0.31
Systemic vascular resistance (dyne/s/cm^5)	2.163 ± 742	1.948 ± 764	1.696 ± 480
SV (ml)	49 ± 18 †	55 ± 19‡	69 ± 18
Cardiac output (L/min)	4 ± 1.2	4.2 ± 1.5	4.4 ± 2
Aortic flow (ml/s)	172 ± 44	176 ± 51	192 ± 51
EF (%)	33 ± 15 *	42 ± 16	65 ± 14
AVA (cm^2)	0.61 ± 0.22	0.58 ± 0.20	0.67 ± 0.18
Peak gradient (mm Hg)	62 ± 21	77 ± 36	77 ± 29
Mean gradient (mm Hg)	30 ± 13	37 ± 22	46 ± 18
Peak valvular resistance (dyne/s/cm^5)	565 ± 282	650 ± 293	557 ± 209
Mean valvular resistance (dyne/s/cm^5)	290 ± 84	391 ± 184	317 ± 140
Energy loss index (cm^2/m^2)	0.39 ± 0.16	0.36 ± 0.14	0.40 ± 0.13
Zva (mm Hg/ml/m^2)	6.5 ± 2.2 *	4.8 ± 1.0	4.7 ± 1.5

* p < 0.01 G1 versus G2 and G3; † p < 0.03 G1 versus G2 and G3; ‡ p < 0.03 G2 versus G3.
Ea: Effective arterial elastance PP: Pulse pressure. SV: Stroke volume. EF: Ejection fraction. AVA: Aortic valve area. Zva: Valvulo-arterial impedance.

Global Afterload

Zva was greater in G1 (6.5 ± 2.2 mm Hg/ml/m^2) than in G2 and G3 (4.8 ± 1 mm Hg/ml/m^2 and 4.7 ± 1.5 mm Hg/ml/m^2, respectively; $p < 0.01$) and had a significant correlation with E_a ($r = 0.88$; $p < 0.0001$, $y = 2.4223x + 0.6784$) and EF ($r = -0.41$; $p < 0.01$ $y = -0.0363x + 6.7738$) (Figure 2). No correlation was observed between Zva and AVA or EF and AVA (figure 2). At multivariate analysis, E_a and Zva were independent predictors of EF (coefficient –4.25 T 2.36; $p < 0.02$).

DISCUSSION

The effect of E_a on global afterload (Zva) and EF in patients with severe aortic stenosis is the main finding of this study, which has potential prognostic and therapeutic implications taking into account that, until recently, surgery was the only therapeutic option. (16) For the purpose of the analysis, patients were divided in three groups, resembling the natural history of severe AS: asymptomatic and compensated patients were included in G3 group; intermediate symptomatic patients were represented by G2, and patients in G1 presented decompensated heart failure. Nevertheless, most patients with heart failure were not aware they had aortic stenosis, probably attributed to progressive reduction in their physical activities and/or absence of early diagnosis. Ejection fraction is inversely related to afterload, but in AS this relationship is more complex due to difficulties in estimating the contribution of ventricular outflow tract obstruction and ventricular/vascular coupling to increased afterload. (9) In patients with AS, increased blood pressure associated with isometric exercise, phenylephrine (17) or angiotensin II (18) causes a reduction in stroke volume and increases left ventricular end-diastolic pressure; in addition, symptoms may be present in patients with hypertension associated with AS with less reduced aortic valve area and lower transvalvular aortic gradients. (19) Severity of AS, compared through transvalvular flow, AVA, peak and mean aortic resistance and ELI, was similar in the three groups, therefore valvular "load" may be considered identical. Peak and mean transvalvular gradients were lower in G1, but this difference was not statistically significant, probably due to reduced SV and transvalvular volume flow rate observed in this group. E_a was significantly greater in patients with heart failure, showing a negative correlation with EF. PP/SV ratio was higher in G1 and G2; nevertheless, this parameter did not show any significant correlation with EF. PP/SV ratio, an index of arterial compliance (20) might have been influenced by the reduction in SV seen in those groups. E_a seems to be more adequate than PP/SV ratio for the assessment of arterial mechanics as it incorporates characteristic impedance (Z_0), resistance (R), arterial compliance (C)

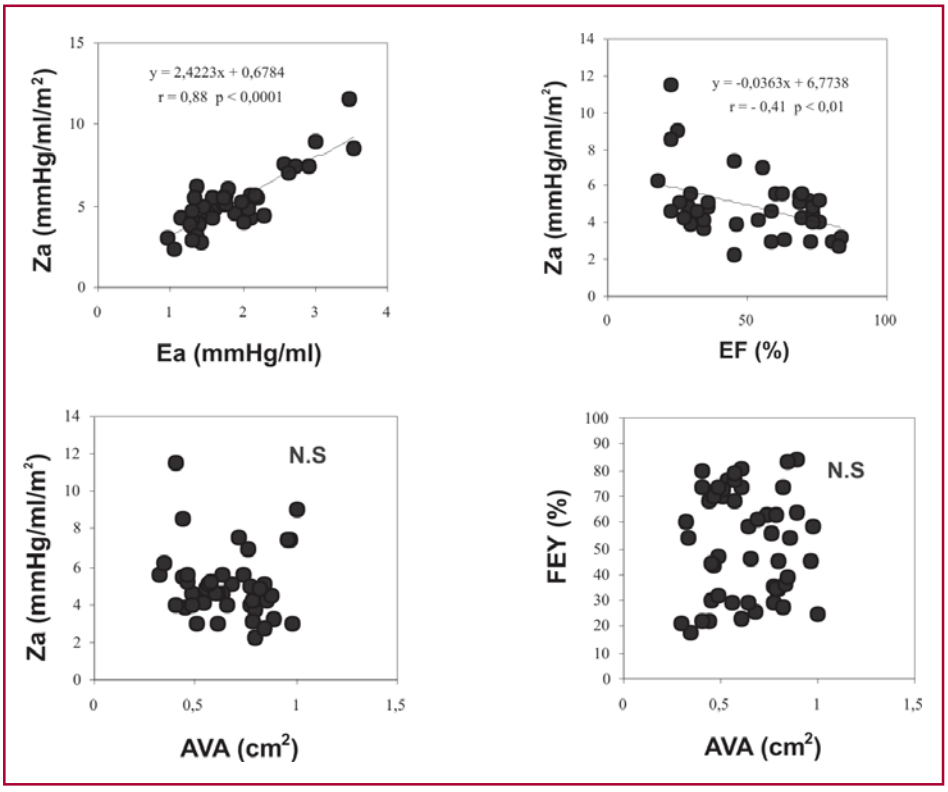

Fig. 2. Correlación de la impedancia valvuloarterial (Zva) con la elastancia arterial efectiva (E_a) y la fracción de eyección (Fey) (superior) y del área valvular aórtica (AVA) con la impedancia valvulo-arterial (Zva) y la fracción de eyección (Fey) (inferior).

and cardiac cycle length (T). (21) Both indices share the same denominator (SV); however end-systolic pressure used by Ea is more accurate to characterize vascular load than pulse pressure (used in PP/SV ratio), (10) as R/T contributes 2.5 times more to Ea than 1/C. (22) Resistance is normalized to cardiac cycle length in R/T ratio; thus this index considers both pulsatile and static (R) blood pressure components. Figure 3 shows that two patients may present the same values of systolic arterial pressure, diastolic arterial pressure and PP but different values of ESP, indicating diverse arterial elastances. (22) According to our findings, vascular "load" was significantly increased in G1 patients, in accordance with increased Zva, a parameter that evaluates global afterload in AS. Zva significantly correlated with Ea, but not with PP/SV. Briand (8) studied patients with moderate and severe AS who were classified in different groups according to arterial stiffness (normal or increased) determined by PP/SV. He reported that PP/SV ratio had a significant impact on EF. Unlike Briand's study, we classified our patients with AS in three groups in an attempt to mimic the natural history of the disease. Even more, the concept of E_a seems more accurate than PP/SV to characterize arterial properties. (24) As Briand, we also found a high incidence of cardiovascular risk factors, such as hypertension, smoking habits, dyslipemia and obesity in all the groups, especially in G2 and G3, which might probably pose a negative influence in the physical properties of the arterial system. Therefore, this finding widens the possibilities of medical treatment of AS, (19, 25, 26) an active process, which requires similar therapy for risk factors as patients with coronary artery disease, especially in early stages of the disease (mild to moderate aortic stenosis). These therapeutic measurements are not opposed to treatment with statins to reduce AS progression, (27) as aortic valve disease and vascular atherosclerosis present similar pathophysiological changes which include lipid infiltration, neoangiogenesis and calcification. (29) However,

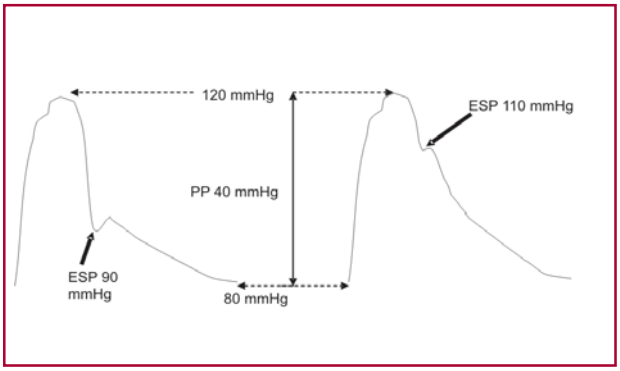

Fig. 3. Véase explicación en el texto.

statins failed to produce any benefit in some studies. (28) Reduction of arterial vascular load is clinically relevant in patients with severe AS, EF < 35% and cardiac index < 2.2 L/min/m^2; treatment with sodium nitroprusside before aortic valve replacement improves the hemodynamic profile of these patients. (30, 31)

Study Limitations

This study failed to establish the precise influence of coronary artery disease on left ventricular function, as coronary arteriography was not performed to most patients due to absence of indication (52% of patients were asymptomatic) or to the fact that data from patients in groups 1 and 2 was not completely available.

CONCLUSIONS

In severe AS, the increase of the vascular component of the afterload, assessed by the Ea, contributes to a reduction in systolic function.

RESUMEN

Efecto de la elastancia arterial efectiva sobre la función sistólica ventricular izquierda en la estenosis aórtica grave

Antecedentes

La disfunción ventricular izquierda en la estenosis aórtica (EAo) está relacionada con el exceso de poscarga. El aumento de la poscarga puede deberse a la reducción del área valvular aórtica (AVA), a cambios en la geometría ventricular y a las características de la vasculatura arterial. La elastancia arterial efectiva (E_a) es un índice de carga vascular que incorpora la impedancia característica, la resistencia y la distensibilidad arterial.

Objetivo

Evaluar el efecto de la E_a sobre la función sistólica del ventrículo izquierdo en la EAo grave.

Material y métodos

Se estudiaron prospectivamente 54 pacientes con EAo grave evaluados mediante los gradientes transvalvulares, el AVA, la resistencia valvular y el índice de pérdida de energía. La E_a se calculó mediante el cociente entre la presión de fin de sístole y el volumen sistólico. La presión de fin de sístole se obtuvo a partir del pulso carotídeo calibrado con la presión arterial medida con esfigmomanómetro. Como medida de la poscarga global, se calculó la impedancia valvuloaórtica (Zva), definida por: (presión sistólica + gradiente medio neto) / índice de volumen sistólico. La Zva tiene en cuenta el efecto de la EAo y la Ea. Los pacientes se dividieron en tres grupos: G1, EAo con insuficiencia cardíaca (NYHA III-IV) (n = 13), G2, EAo sintomática pero sin insuficiencia cardíaca (n = 13) y G3, EAo asintomática (n = 28).

Resultados

La fracción de eyección (Fey) (%) fue menor en el G1 (33 ± 15) en comparación con el G2 (42 ± 16) y el G3 (65 ± 14); p < 0,01. La E_a (mm Hg/ml) fue mayor en el G1 (2,46 ± 0,8) con respecto al G2 y el G3 (1,83 ± 0,52 y 1,73 ± 0,47; p < 0,01), al igual que la Zva (mm Hg/ml/m^2): G1 (6,5 ± 2,2), G2 y G3 (4,8 ± 1,0 y 4,7 ± 1,5; p < 0,01). En el análisis univariado, la Zva se correlacionó con la E_a (r = 0,88; p < 0,0001) y la Fey (r = -0,41; p < 0,01). En el análisis multivariado, la E_a y la Zva fueron predictores independientes de la fracción de eyección.

Conclusión

En la EAo grave, el aumento del componente vascular de la poscarga evaluado a través de la E_a contribuye a la reducción de la función sistólica.

Palabras clave > Estenosis de la válvula aórtica - Función ventricular izquierda - Ecocardiografía de Doppler

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