ORIGINAL ARTICLE

Contribution of Longitudinal and Circumferential Shortening to Stroke Volume and Ejection Fraction in Severe Aortic Stenosis

Contribución del acortamiento longitudinal y circunferencial al volumen sistólico y fracción de eyección en la estenosis aórtica grave

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ABSTRACT

Background: Left ventricular (LV) stroke volume (SV) is ejected by a combination of longitudinal shortening (LS), circumferential shortening (CS) and ventricular twist. Longitudinal shortening is caused by the motion of the mitral annulus towards the apex, causing wall thickening (circumferential and radial shortening), and left ventricular cavity and SV reduction. The role of LS in aortic stenosis (AS) is not defined.
Objective: The aim of this study was to analyze the LS and CS contribution to SV and left ventricular ejection fraction (LVEF) in severe AS.
Methods: The study included 152 patients (70±12 years, 64 women) with severe AS (valve area <1 cm^2) studied by cardiac Doppler echocardiography. The LS contribution to SV was evaluated by considering the base of the heart as a cylinder, with volume=base x height; the base was assumed as a circle with radius equal to (systolic septal thickness+systolic posterior wall thickness+end-systolic diameter)/2; and height, as an average of the mitral lateral, septal, anterior and inferior annulus systolic excursion. The CS contribution to SV was estimated as: SV - LS contribution to SV. Both contributions were expressed in absolute form and as SV percentages (% SV-LS and % SV-CS).
Results: Longitudinal shortening contributed with approximately 2 thirds of SV (68±18 %) and CS with the rest. SV-LS correlated inversely with SV (r= - 0.45 p <0.001) and SV-CS had direct correlation (r=0.45 p <0.001). The % SV-LS contribution was greater in patients with LVEF < 50%. Percent SV-LS correlated inversely with relative wall thickness (RWT) (r=0.32, p < 0.01).
Conclusion: The LS contribution to SV is greater than that of CS, and correlates inversely with SV. Percent SV-LS is higher in patients with LVEF <50%. The aforementioned findings could have implications when considering cut-off points for longitudinal function indices (strain) without considering LVEF and/or ventricular geometry.

Key words: Aortic stenosis – Systolic function – Contractility – Ejection fraction

RESUMEN

Introducción: El volumen sistólico (VS) del ventrículo izquierdo (VI) es eyectado por una combinación de acortamiento longitudinal (AL), circunferencial (AC) y giro ventricular. El AL se produce por el movimiento del anillo mitral hacia el ápex, lo que ocasiona engrosamiento de la pared (AC y radial), reducción de la cavidad ventricular y eyección del VS. No está definido el rol del AL en la estenosis aórtica (EAo).
Objetivo: Analizar la contribución del AL y AC al VS y la fracción de eyección ventricular izquierda (FEVI) en la EAo grave.
Materiales y métodos: Se estudiaron 152 pacientes (70 ± 12 años, 64 mujeres) con EAo grave (área valvular < 1 cm^2) con eco-Doppler cardíaco. La contribución del AL al VS se evalúo considerando a la base del corazón como un cilindro., con volumen = base x altura; la base, un círculo con radio igual a (espesor septal en sístole + espesor pared posterior en sístole + diámetro de fin de sístole) /2; y la altura, el promedio de la excursión sistólica del anillo lateral, septal, anterior e inferior. La contribución del AC al VS se estimó como: VS – contribución del AL al VS. Ambas contribuciones se expresaron en forma absoluta y como porcentajes del VS (% VS-AL y % VS-AC).
Resultados: El AL contribuyó con 2 tercios aproximadamente al VS (68 ± 18 %) y el AC con el resto. El VS-AL correlacionó inversamente con el VS (r = - 0,45, p < 0,001) y el VS-AC tuvo correlación directa (r = 0,45, p < 0,001). La contribución del % VS-AL fue mayor en los pacientes con FEVI < 50%. El % VS-AL correlacionó inversamente con el EPR (r = - 0,32, p < 0,01).
Conclusión: La contribución del AL al VS es mayor que la del AC, y correlaciona inversamente con el VS. Es mayor el % VS-AL en los pacientes con FE < 50%. Los hallazgos mencionados podrían tener implicancias al considerar puntos de corte para los índices de función longitudinal (*strain*) sin considerar la FEVI y/o geometría ventricular.

Palabras clave: Estenosis aórtica - Función sistólica - Contractilidad - Fracción de eyección

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INTRODUCTION

Longitudinal fiber shortening (subepicardial and subendocardial), circumferential fiber shortening (midwall) and ventricular torsion determine left ventricular (LV) wall thickening causing a decrease in the ventricular cavity with the consequent ejection of stroke volume (SV). Longitudinal shortening (LS) is produced by the movement of the mitral annulus towards the apex, which remains immobile, with a mechanism similar to that of a piston (Fig. 1 A). (1) Since the myocardium is considered incompressible, LS determines radial ventricular wall thickening with ventricular cavity reduction. Circumferential shortening (CS) is evidenced by the decrease of the epicardial diameter in the transverse axis, which also contributes to the reduction of the LV cavity during systole. (2) Epicardial shortening can be evaluated as fractional shortening (epiFS) which is lower than midwall FS (mFS) and endocardial FS (eFS) (Fig. 1 B). Longitudinal shortening contribution to SV has been reported to vary between 75% (2) and 17% (3), and its role is not defined in aortic stenosis (AS). Left ventricular LS is directly related to longitudinal strain, so it is essential to determine its contribution when establishing cut-off points in order to assess systolic function through global longitudinal strain or new indices such as myocardial work.

The purpose of this study was to analyze LS and CS contributions to SV and left ventricular ejection fraction (LVEF) in severe AS.

METHODS

A prospective study was conducted on 152 patients (mean age 70±12 years, 88 men and 64 women) with severe AS, defined as aortic valve area index (AVAI) <0.6 cm^2/m^2, and with cardiac echo-Doppler indication. The protocol was approved by the Teaching and Research Committee of the Hospital. Patients who presented significant mitral annulus calcification, or moderate or severe aortic or mitral regurgitation were excluded from the study. History of hypertension was considered as prior antihypertensive treatment and history of ischemic heart disease as the presence of one or more of the following criteria: 1) history of acute myocardial infarction (AMI), angioplasty or revascularization surgery, 2) obstructions greater than 50% in the coronary arteries determined by coronary angiography and 3) akinesia in the echocardiogram. All patients underwent a complete Doppler echocardiogram after interrogation to detect the presence of coronary risk factors and symptoms, a cardiovascular physical examination and blood pressure measurement.

Echocardiogram and cardiac Doppler: The study was performed with an ESAOTE MyLab 40 ultrasound machine and 2.5-3.5 MHz transducer, with the patient lying in left lateral decubitus position and DII lead electrocardiography as reference. M-mode measurements, two-dimensional echocardiogram, and LV eFS, relative wall thickness (RWT), end-diastolic volume (EDV) (Simpson method), end-systolic volume (ESV) and LVEF calculations were performed according to ASE criteria. (4)

Left ventricular mFS was calculated using the Koide formula: (5)

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

where: DD: diastolic diameter; SD: systolic diameter; h: combined posterior wall (PW) and interventricular septum (IVS) diastolic thickness (PWd+IVSd / 2); hfs: combined systolic thickness (PWs+IVSs / 2) and a´: midwall point in systole calculated from the following formula:

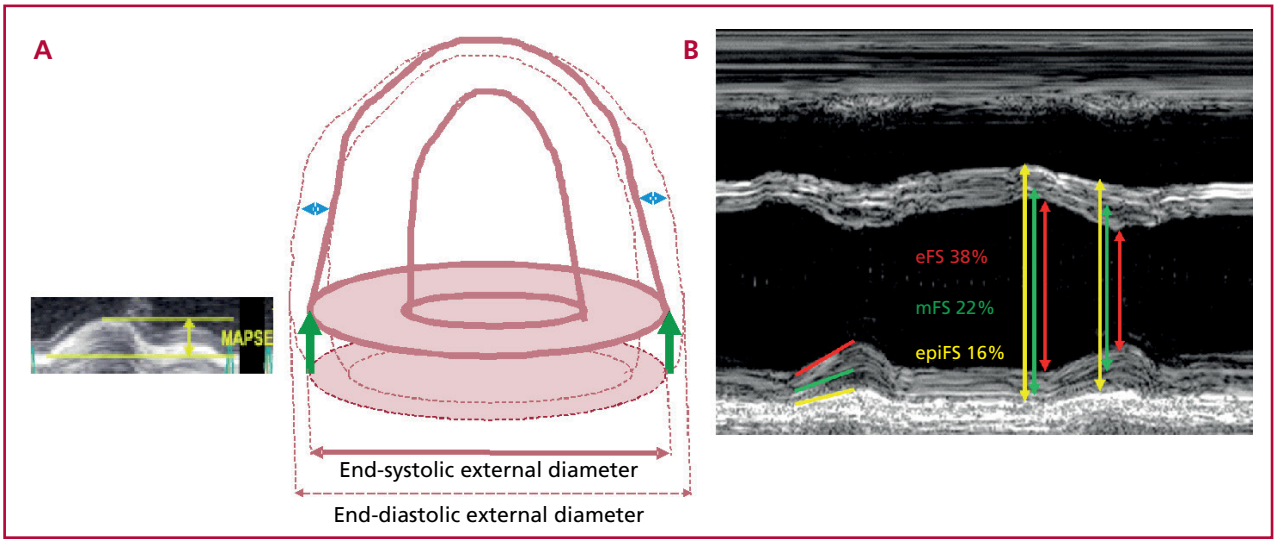

Fig. 1. A. Graphical representation of the left ventricle at end-diastole (dotted lines) and end-systole (full lines). Longitudinal shortening (LS) is represented by the base to apical motion (green arrows) of a theoretical cylinder that would act as a piston, since the apex remains immobile. The lateral mitral annular plane systolic excursion (MAPSE) recording in M-mode, 4-chamber view, is observed to the left. Circumferential shortening, represented with blue arrows, contributes to wall thickening due to myocardial incompressibility; however, it should be mentioned that LS per se already determines part of wall thickening, due to the same cause (myocardial incompressibility) **B.** M mode at mid-ventricular level, representing epicardial (epiFS) in yellow, midwall (mFS) in green and endocardial (eFS) in red fractional shortening. EpiFS is the only one that depends on CS, while mFS and eFS are determined by circumferential (CS) and longitudinal (LS) shortening.

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

Left ventricular mass was calculated according to the Devereux formula (6) and mass index (MI) as mass normalized to body surface area. Epicardial fractional shortening (epiFS) was calculated as (3):

epiFS = [(IVSd + LVDD + PWd) – (IVSs + LVSD + PWs) / (IVSd + LVDD + PWd)] × 100

Continuous Doppler was used to register maximum transvalvular aortic velocity, mean gradient (MG), and the integral of the flow curve from apical, right parasternal, subxiphoid, and suprasternal views. The LV outflow tract flow was obtained from a pulsed Doppler 5-chamber view. Effective AVA was calculated using the continuity equation and AVAI, according to ASE. (7) Stroke volume was estimated as the product of the outflow tract area by the integral of flow at that level, LVEF using the biplane Simpson method, and relative wall thickness (RWT) as 2 × PWd / LVDD. To analyze the LS and CS contribution to SV, an alternative method was used to calculate EDV and ESV. The EDV can be evaluated as the difference between LV total end-diastolic volume (TEDV) that includes the myocardium plus the cavity, minus myocardial volume (MyoV):

$$EDV = TEDV – MyoV$$

Similarly, ESV can be calculated as total end-systolic volume (TESV) minus MyoV:

$$ESV = TESV – MyoV$$

Because the myocardium is considered incompressible, MyoV is similar in diastole and in systole, therefore, SV can be calculated as:

$$SV = EDV – ESV$$

or

$$SV = TEDV – TESV$$

The LS contribution to SV (SV-LS) was evaluated considering the base of the heart as a cylinder (Fig 1A) in which the volume (base × height) was calculated as: circular base (area=ϖ × radius2) with radius equal to: (IVSs+ PWs + LVSD) / 2, and height as the average lateral, septal, anterior, and inferior mitral annulus plane systolic excursion (MAPSE), obtained by M-mode in 4- and 2-chamber views, respectively. (2) The CS contribution to SV (SV-CS) was estimated as: SV–LS contribution to SV. (8) Both contributions were expressed in absolute form and as percentages of SV (% SV-LS and % SV-CS).

Statistical analysis

Statistix 10 software package was used to perform the statistical analysis. Continuous variables were expressed as mean±standard deviation. Analysis of variance was used to compare groups and Pearson's r coefficient to calculate correlation. A p value <0.05 was considered as significant.

RESULTS

Clinical and echocardiographic patient parameters are represented in Tables I and II. Longitudinal shortening contributed with approximately 2 thirds of SV (68±18%) and CS with the remaining third (32±17%). SV-LS was inversely correlated with SV (r= -0.45, p <0.001), which implies that as SV decreases, the LS contribution is greater. SV-CS had a direct correlation with SV (r= 0.45, p <0.001) so that as SV decreases, the CS contribution also decreases (Fig. 2). LVEF had a lower, though statistically significant correlation with % SV-LS and % SV-CS (Fig. 3).

When patients were divided into LVEF ≥50% (n=95) and LVEF <50% (n=57), those with lower

Table 1. Clinical parameters

Age (years)	70 ± 12
Male / female	88 / 64
Body Surface area (m²)	1.82 ± 0.19
Concomitant diseases and risk factors (%)	
Coronary heart disease	30
Previous myocardial infarction	11
Hypertension	58
Diabetes	16
Dyslipidemia	32
Smoking	27
Obesity (Body mass index > 30 Kg/m²)	25
Chronic obstructive pulmonary disease	5
Symptoms (%)	
Angor	9
Syncope	1
Dyspnea I-II	25
Dyspnea III-IV	53
Systolic blood pressure (mmHg)	130 ± 22
Diastolic blood pressure (mmHg)	76 ± 12

Table 2. Echocardiographic parameters

Aortic valve area (cm²)	0.69 ± 0.21
Aortic valve area index (cm²/m²)	0.38 ± 0.12
Peak aortic velocity (m/seg)	4.14 ± 0.85
Peak gradient (mmHg)	72 ± 29
Mean gradient (mmHg)	42 ± 18
Left ventricular ejection fraction (%)	63 ± 8
Systolic volume (ml)	60 ± 19
SV – LS (ml)	41 ± 9
SV – CS (ml)	19 ± 9
% SV – LS	68 ± 18
% SV – CS	32 ± 17
Epicardial fractional shortening (%)	10 ± 5
Midwall fractional shortening (%)	19 ± 5
Endocardial fractional shortening (%)	37 ± 8

SV-LS: Absolute longitudinal shortening contribution to stroke volume
% SV-LS: Percent longitudinal shortening contribution to stroke volume
SV-CS: Absolute circumferential shortening contribution to stroke volume
% SV-CS: Percent circumferential shortening contribution to stroke volume

LVEF had higher EDV and ESV and lower SV when compared with patients with LVEF ≥50% (Table III). When analyzing blood volume ejected during systole due to the displacement of the mitral plane towards the apex, represented by a cylinder, it was possible to observe that although MAPSE was lower in patients with LVEF <50% (9±3 mm vs. 13±3 mm, p <0.0001), the basal area was greater (49±11 cm^2 vs. 40±9 cm^2, p=0.02), which determined that the volume mobilized by LS (SV–LS) was similar in patients with LVEF <50% and LVEF ≥50% (43±15 ml vs. 45±12 ml, p=0.38). As both groups had similar SV–LS but patients with LVEF <50% had lower SV, the % SV-LS contribution was greater in that group than in those with LVEF ≥50% (73±15% vs. 65±16%, p <0.01).

Circumferential function assessed by % SV–CS and epiFS was decreased in patients with LVEF <50% when compared with LVEF ≥50% [(27±18% vs. 35±16%, p <0.01) and (8 ± 4 % vs 11 ± 4 %, p < 0.01), respectively].

Regarding ventricular geometry, concentric hypertrophy predominated in patients with LVEF ≥50% and eccentric hypertrophy in those with LVEF <50% when considering RWT and MI (Table III). In the total group of patients, % SV-LS was inversely correlated with RWT (r= -0.32, p < 0.01), suggesting that eccentric hypertrophy is associated with a greater LS contribution to SV. (Fig.4)

DISCUSSION

The main findings of this study are: 1) LS contributes with two thirds of SV ejected by the LV while CS contributes with the remaining third, 2) SV–LS is inversely correlated with SV, which implies that the LS contribution to SV is greater as SV decreases, and 3) LS (% SV–LS) contribution is greater in patients with LVEF <50%.

Stroke volume is the end product of the heart as a pump, which can be seen as the sum of two parts: the volume generated by the longitudinal function and the volume determined by the circumferential function. Longitudinal function is related to MAPSE and circumferential function to epicardial shortening. In Fig. 5, A represents a theoretical left ventricle without myocardium in which epicardial shortening (circumferential function) determines SV ejection, B shows that LS (longitudinal function) is responsible for SV ejection; C represents a left ventricle with normal wall thickness with LS without CS and D illustrates the effect of hypertrophy, where the increase in wall thickness determines greater endocardial excursion, SV preservation, but higher LVEF due to a decrease in EDV. Thus, there can be wall thickening without CS. (8) The LS and CS contribution to SV is represented in Fig. 1A. According to our findings, LS contributed with approximately two thirds to SV and CS with the remaining third, in agreement with Carlsson (LS 60%) (1) who studied normal individuals, athletes, and patients with dilated cardio-

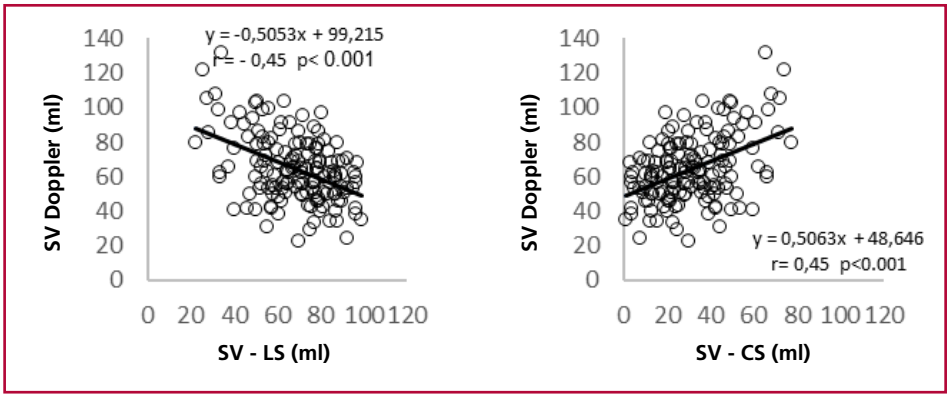

Fig. 2. Correlation and regression between stroke volume (SV), determined by Doppler, and longitudinal shortening (LS) contribution (left panel), and circumferential shortening (CS) contribution (right panel).

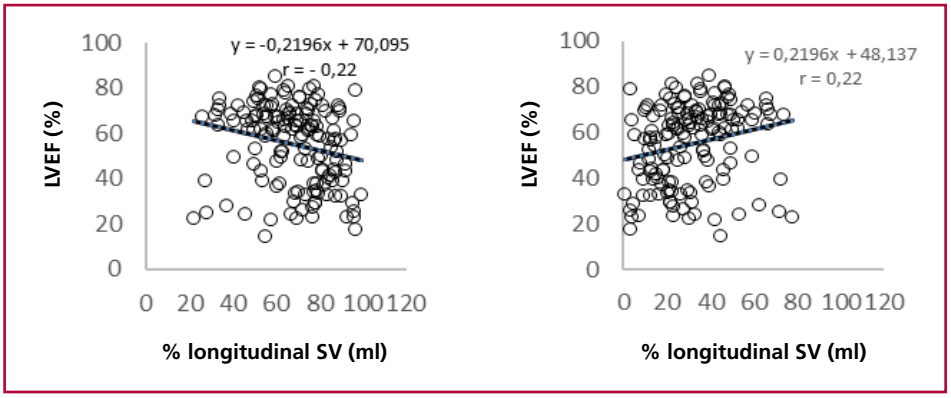

Fig. 3. Correlation and regression between left ventricular ejection fraction (LVEF) and percent stroke volume (SV) as a result of longitudinal shortening (% SV longitudinal) (left panel) and circumferential shortening (%SV circumferential) (right panel).

Table 3. Echocardiographic parameters according to LVEF ≥50% and LVEF <50%

	LVEF ≥50% (n = 95)	LVEF <50% (n = 57)	p
Ejection fraction (%)	67 ± 8	32 ± 11	0.0001
End-diastolic volume (ml)	88 ± 35	156 ± 63	0.0001
End-systolic volume (ml)	31 ± 19	107 ± 52	0.0001
Stroke volume (ml)	66 ± 20	50 ± 21	0.02
Basal radius (cm)	3.36 ± 0.38	3.91 ± 0.47	0.01
Basal area (cm²)	40 ± 9	49 ± 11	0.02
MAPSE (mm)	13 ± 3	9 ± 3	0.0001
SV – LS (ml)	45 ± 12	43± 15	0.38
SV – CS (ml)	20 ± 13	7 ± 5	0.0001
% SV – LS	65 ± 16	73 ± 18	0.01
% SV – CS	35 ± 16	27 ± 18	0.01
epiFS (%)	11 ± 4	8 ± 4	0.001
mFS (%)	19 ± 5	13 ± 5	0.0001
eFS (%)	37 ± 8	25 ± 9	0.0001
Relative wall thickness	0.52 ± 0.18	0.43 ± 0.11	0.0001
Mass index (gr/m²)	145 ± 40	175 ± 48	0.0001

LVEF: Left ventricular ejection fraction
MAPSE: Average lateral, septal, anterior and inferior mitral annular plane systolic excursion.
epiFS: Epicardial fractional shortening, mFS: Midwalll fractional shortening, eFS: Endocardial fractional shortening.
SV-LS: Absolute longitudinal shortening contribution to stroke volume
% SV-LS: Percent longitudinal shortening contribution to stroke volume
SV-CS: Absolute circumferential shortening contribution to stroke volume
% SV-CS: Percent circumferential shortening contribution to stroke volume

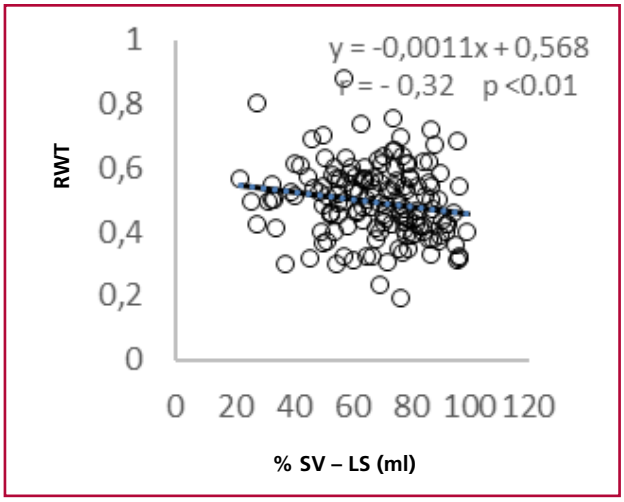

Fig. 4. Correlation and regression between relative wall thickness (RWT) and percent stroke volume (SV) as a result of longitudinal shortening (%SV-LS).

myopathy with magnetic resonance imaging. Stoylen (2) studied normal individuals with echocardiography, finding that 75% of SV corresponded to LS, similarly to results reported by Emilsson (82%). (9) However, Maclver, (3) using a mathematical model, published that LS contributes with 17%, which differs from the previously cited authors. There is no reference about AS. The inverse correlation found between SV-LS and SV indicates that the LS contribution is greater as SV decreases, which also has an impact on the LVEF, since the lower the SV, the lower the LVEF for the same EDV. The LS contribution to SV was different when considering LVEF ≥ 50% and LVEF < 50%. In the first group it was similar to the total number of patients, but in patients with LVEF <50% it was 73%, and this difference was statistically significant. In this group, the paradoxical finding that % SV-LS is increased, but MAPSE -which also evaluates longitudinal function- is decreased, is explained because the base area of the theoretical cylinder that represents the SV is increased, resulting in SV-LS similar to that of the group with LVEF ≥ 50%; however, as the SV is lower, the % SV-LS is higher. The decrease in CS in patients with LVEF <50% is also evidenced by the decrease in epiFS. Stokke (10) reported that longitudinal function (global longitudinal strain) contributes more than circumferential function to LVEF when it is less than 50%, using a mathematical model that was subsequently validated in 100 patients. Ventricular geometry also influences SV composition, since RWT was inversely correlated with %SV-LS, implying that eccentric hypertrophy is associated with higher %SV-LS and concentric hypertrophy with lower %SV-LS.

The aforementioned findings have implications for the interpretation of longitudinal function indices such as global longitudinal strain, used to establish cut-off points of ventricular impairment without considering ventricular geometry (11, 12) or when myocardial work is calculated replacing SV by strain. (13)

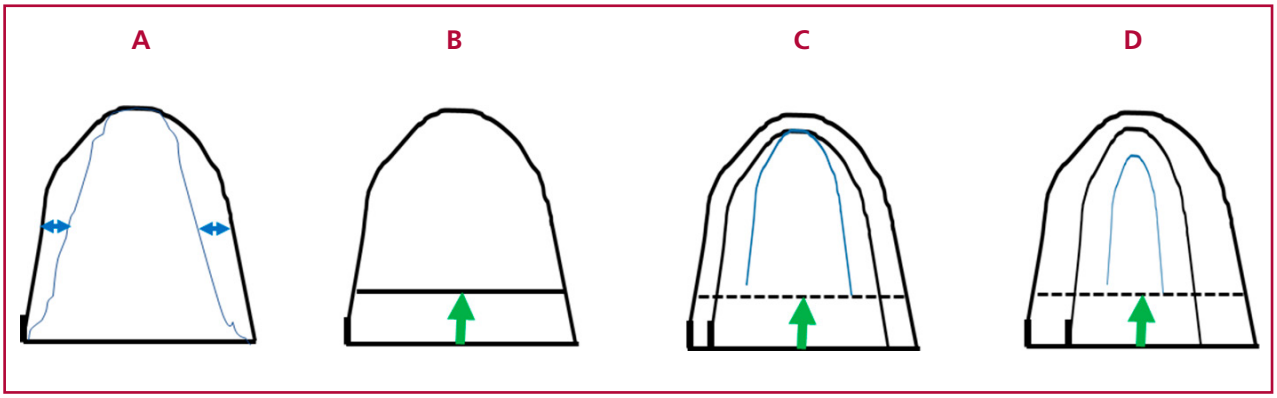

Fig. 5. Graphical representation of the left ventricle (LV) at end-diastole (black) and end-systole (blue) In A, the LV has minimum wall thickness and circumferential shortening (CS) is represented alone, and in B longitudinal shortening (LS) is represented alone. In C, the myocardium has normal thickness and presents only LS, with systolic wall thickening that determines stroke volume (SV) ejection. In D the LV has concentric hypertrophy, so for the same LS it elicits greater endocardial excursion, resulting in higher left ventricular ejection fraction, and evidencing the dependency of this last index on ventricular geometry. In C and D, the SV is the same, as mitral annular plane systolic excursion is similar

Limitations

Cardiac nuclear magnetic resonance is the reference method for calculating ventricular volumes, which can be used to measure mitral annulus displacement and epicardial shortening. (1) This methodology was not available in this study, but echocardiography was the technique used or referred in other studies. (2, 3) Longitudinal strain was measured in a small percentage of patients, so no correlation was made with LS, though it has been previously published. (2)

CONCLUSIONS

The LS contribution to SV is greater (two thirds) than that of CS, and is inversely correlated with SV and RWT. However, this proportion varies when considering LVEF; thus, % SV-LS is greater in patients with LVEF <50% than in those with LVEF ≥50%. These findings could have implications when considering cut-off points for longitudinal function indices (strain) without considering LVEF and/or ventricular geometry.

Conflicts of interest

None declared.

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

REFERENCES

1. Carlsson M, Ugander M, Mosén H, Buhre T, Arheden H. Atrioventricular plane displacement is the major contributor to left ventricular pumping in healthy adults, atheltes, and patients patients with dilated cardiomyopathy. Am J Physiol Heart Circ Physiol 2007;292:H452-9. https://doi.org/10.1152/ajpheart.01148.2006

2. Stoylen A, Dalen H, Molmen HE. Left ventricular longitudinal shortening: relation to stroke volume and ejection fraction in ageing, blood pressure, body size and gender in the HUNT3 study. Open Heart 2020;7:001243. https://doi.org/10.1136/openhrt-2020-001243

3. MacIver DH. The relative impact of circunferential and longitudinal shortening on left ventricular ejection fraction and stroke volume. Exp Clin Cardiol 2012;17:5-11.

4. Lang RM, Badano LP, Mor-Avi V, Afilalo J, Armstrong A, Ernande L, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: un update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. J Am Soc Echocardiogr 2015;28:1-39. https://doi.org/10.1016/j.echo.2014.10.003

5. Koide M, Nagatsu M, Zile M, Hamawaki M, Swindle M, Keech G, et al. Premorbid determinants of left ventricular dysfunction in a novel model of gradually induced pressure overload in the adult canine. Circulation 1997; 95:1601-10. https://doi.org/10.1161/01.CIR.95.6.1601

6. Devereux RB, Alonso DR, Lutas EM, Gottlieb GJ, Campo E, Sachs I, et al. Echocardiographic assessment of left ventricular hypertrophy: comparison to necropsy findings. Am J Cardiol 1986;57:450-8. https://doi.org/10.1016/0002-9149(86)90771-X

7. Baumgartner H, Hung J, Bermejo J, Chambers JB, Edvardsen T, Goldstein S, et al. Recommendations of the echocardiographic assessment of aortic valve stenosis: A focused update from the European Association of Cardiovascular Imaging and the American Society of Echocardiography. J Am Soc Echocardiogr 2017;30:372-92. https://doi.org/10.1016/j.echo.2017.02.009

8. Ugander M, Carlsson M, Arheden H. Short-axis epicardial volume change is a measure of cardiac left ventricular short-axis function, which is independent of myocardial wall thickness. Am J Physiol Heart Circ Physiol 2010;298:H530-5. https://doi.org/10.1152/ajpheart.00153.2009

9. Emilsson K, Brudin L, Wandt B. The mode of left ventricular pumping: is there an outer contour change in addition to the atrioventricular plane displacement? Clinic Physiol 2001;21:437-46. https://doi.org/10.1046/j.1365-2281.2001.00343.x

10. Stokke TM, Hasselberg NE, Smedsrud MK, Sarvari SI, Haugaa KH, Smiseth OM, et al. Geometry as a confounder when assessing ventricular systolic function, Comparison between ejection fraction and strain. J Am Coll Cardiol 2017;70:942-54. https://doi.org/10.1016/j.jacc.2017.06.046

11. Vollema EM, Amanullah MR, Prihadi EA, Ng ACT, Bijl P, Sin YK, et al. Incremental value of left ventricular global longitudinal strain in a newly proposed staging classification based on c https://doi.org/10.1093/ehjci/jeaa220ardiac damage in patients with severe aortic stenosis. EHJ Cardiovasc Imag 2020;21;1248-58.

12. Migliore RA, Adaniya ME, Franco Camacho MI, Barranco MA, Milles Honores J, Cobos SK, et al. Determinantes de la fracción de eyección ventricular izquierda en pacientes con estenosis aórtica grave. Rev Argent Cardiol 2021;89:447-54. http://dx.doi.org/10.7775/rac.es.v89.i5.20439

13. Roemer S, Jaglan A, Santos D, Umland M, Jain R, Tajik AJ, et al. The utility on myocardial work in clinical practice. J Am Soc Echocardiogr 2021;34:807-18. https://doi.org/10.1016/j.echo.2021.04.013