

Left Ventricular Hypertrophy Evaluation in Obese Hypertensive Patients. Effect of Left Ventricular Mass Index Criteria

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Purpose - To evaluate left ventricular mass (LVM) index in hypertensive and normotensive obese individuals.

Methods - Using M mode echocardiography, 544 essential hypertensive and 106 normotensive patients were evaluated, and LVM was indexed for body surface area (LVM/BSA) and for height² (LVM/h²). The 2 indexes were then compared in both populations, in subgroups stratified according to body mass index (BMI): <27; 27-30; ≥30kg/m².

Results - The BSA index does not allow identification of significant differences between BMI subgroups. Indexing by height² provides significantly increased values for high BMI subgroups in normotensive and hypertensive populations.

Conclusion - Left ventricular hypertrophy (LVH) has been underestimated in the obese with the use of LVM/BSA because this index considers obesity as a physiological variable. Indexing by height² allows differences between BMI subgroups to become apparent and seems to be more appropriate for detecting LVH in obese populations.

Key words: essential hypertension, obesity, left ventricular hypertrophy, left ventricular mass index

Echocardiographic evaluation of hypertensive individuals is based on preestablished guidelines for detecting left ventricular hypertrophy, determined in relation to populations of normotensive individuals. In turn, the definition of normal left ventricular mass implies its correction by influencing physiological factors. Thus, sex, body habitus, and possibly age are of importance in this correction.

The best index of left ventricular mass is that obtained using the physiological scale of weight and height variables, regarding both men and women.

Therefore, the ideal index would be lean body mass¹, but this method is not practical and has not been used. Thus, indexing ventricular mass by body surface area (BSA) is preferred².

However, such an index leads to underestimation of left ventricular hypertrophy in obese individuals (with a greater BSA), because it regards obesity as a continuous physiological variable that would determine increases in left ventricular mass also on a physiological scale³.

To correct this, use of mass by height, whose limits are within the physiological range and thus maintain a normal and not a pathological relation to ventricular mass, has been proposed as an index⁴⁻⁶.

More recent studies⁷⁻¹¹ further suggest that left ventricular mass index should be determined by height or even height raised to a power of 2, 2.7, or 2.13, because no first order relation has been demonstrated between height and left ventricular mass.

In this sense, some selection criteria have been established that have been used for the correction of mass by these proposed indexes (men - 126/143g/m; 49.2g/m^{2.7}; women - 105/102g/m; 46.7g/m^{2.7})^{4,11}. Such criteria, up to the present, have preferentially been used in larger population studies^{11,13-16} with the purpose of detecting the impact of the different indexes used on the prevalence of

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hypertrophy in these populations, known to imply a worse cardiovascular diagnosis¹⁷⁻²².

The purpose of the present study is to compare left ventricular mass index by height squared with the usual index by body surface area in hypertensive and normotensive obese individuals.

Methods

In a cross-sectional study, 544 patients with essential hypertension, 173 men and 371 women, ages ranging from 13 to 84 and 17 to 80 years, respectively, were evaluated as part of a larger study of cardiac morphometric and functional evaluation in this group of individuals.

Individuals with a history of mild to moderate hypertension, with or without use of medicines, were selected based on a survey of their medical records.

Exclusion criteria were: stage 3 hypertension [systolic blood pressure (SBP) ≥ 180 mmHg and/or diastolic blood pressure (DBP) ≥ 100 mmHg on the day of echocardiography]; diabetes (with a preestablished diagnosis and/or fasting glucose ≥ 140 mg/dL); chronic renal failure (defined by serum creatinine ≥ 2.0 mg/dL); coronary disease (diagnosed through angiography, history of myocardial infarct, angina, or a positive ergometric test); clinical signs of congestive heart failure.

Also, 106 normotensive individuals (51 men and 55 women), whose mean blood pressures, measured on 3 consecutive occasions during a 1-week interval, were below 140 mmHg (SBP) and 90 mmHg (DBP), were evaluated.

The following parameters: age, weight, height, body mass index²³ (BMI: weight in kg/height²); body surface area² [BSA: $0.0001 \times 71.84 \times (\text{weight} - \text{kg})^{0.425} \times (\text{height} - \text{m})^{0.725}$]; and time of hypertension were obtained on the day the echocardiogram was performed.

Arterial blood pressure of each hypertensive and normotensive individual was measured before the examination, with 1 measurement in the sitting position and 1 after a 5-minute rest.

Regarding the hypertensives, 84.4% of the men and 86.3% of the women were receiving the usual treatment. In regard to evaluation of pressure levels, 9% had controlled pressure levels, 7.7% had borderline hypertension, 32.3% stage 1, and 50.9% stage 2 hypertension according to the criteria established by the VI Joint National Committee for prevention and treatment of hypertension²⁴.

Both hypertensive (HT) and normotensive (NT) groups were divided into 3 subgroups according to BMI: < 27 kg/m² (normal - 79 NT and 287 HT); 27 to 30 kg/m² (overweight - 15 NT and 136 HT); ≥ 30 kg/m² (obese - 12 NT and 121 HT)²⁵.

For the echocardiographic evaluation, Escote Biomédica, model SIM5000 equipment, with a mechanical 2.5 MHz transducer, allowing bi-dimensional M mode evaluation with pulse and continuous Doppler, was used.

Measures of left ventricle mass (LVM) were calculated by the modified Devereux formula²⁶: $0.8[1.04(\text{DIVS} + \text{DLVD} + \text{DLVPW})^3 - (\text{DLVD})^3] + 0.6$, where DIVS, DLVD, and DLVPW

correspond to measurements of interventricular septum, left ventricle diameter, and left ventricle posterior wall in diastole.

All measurements were performed according to the recommendations of the American Society of Echocardiography²⁷ that considers measurements at the end of diastole, including endocardial thickness measures of the septum and posterior wall. This fact justifies the use of the American Society of Echocardiography (ASE) formula modified by Devereux²⁶. This formula brings the left ventricle mass values obtained by the initially validated ASE formula²⁸ near those obtained by the Penn convention equation²⁹, which, despite being more accurate, uses a less common method for measurement that excludes the endocardial septum and wall thickness from the analysis.

For indexing the ventricular mass and calculation of the prevalence of hypertrophy, body surface area was used, thus obtaining the LVM/BSA parameter, whose usually applied normality criteria regarding hypertrophy have been, 110 g/m² in women and 134 g/m² in men¹³.

As proposed in the literature^{4,5}, we also corrected the mass value by height (LVM/h) and because height^{2,7} or height^{2.13} are not practical, we opted for height squared (LVM/h²).

The prevalence of hypertrophy in the different hypertensive population subgroups was also calculated based on the mass/height² limits established for a normotensive reference population. In this way, using the 95th percentile of the mass/height² ratio in this population, the limits of values of 77.7 g/m² for men and 69.8 g/m² for women were obtained. For comparison, analysis of prevalence, using the limits of mass/body surface area obtained through the 95th percentile of the normotensive population, was performed, leading to 110 g/m² in men and 96 g/m² in women.

Statistical analysis was performed using the Sigma Stat program. For the global analysis of the demographic, pressure, and cardiac-structural parameters, the values of mean \pm standard error were used. The comparative analysis between demographic and pressure variables in the 3 different subgroups of hypertensives and normotensives was performed using a variance test (ANOVA).

For comparison between the cardiac structural parameters of the hypertensive and normotensive subgroups, variance (ANOVA) and covariance tests were necessary, with adjustment for systolic arterial blood pressure in the hypertensive and normotensive groups, respectively.

Spearman's correlation analysis was also used for correlation between anthropometric (weight, height, BSA) and cardiac structural (LVM/h; LVMh²; LVM/BSA) variables.

Values of p below 0.05 were considered significant.

Results

To test the newly proposed indexer (height), the correlations of anthropometric (weight and height) and derived (BSA and BMI) parameters with those of cardiac mass, duly indexed for BSA, height and height² were obtained. As shown in Table I, the LVM/BSA variable has a nonsignificant correlation with all anthropometric variables.

	LVM/BSA	LVM/h	LVM/h ²
Weight	0.07	0.29 *	0.19 *
Height	0.08	0.17 *	0.03
BSA	0.06	0.26 *	0.11 *
BMI	0.03	0.24 *	0.26 *

* p<0.05; LVM/BSA (g/m²) - left ventricular mass corrected by body surface area; LVM/h (g/m) - left ventricular mass corrected by height; LVM/h² (g/m²) - left ventricular mass corrected by height²; BSA (m²) - body surface area; BMI (kg/m²) - body mass index.

Regarding the other structural variables (LVM/h and LVM/h²), the LVM/h variable has a frankly positive relation to the height variable (r=0.17) and a correlation of 0.24 with the body mass index. But when using the correction LVM/h², in addition to a better correlation with the body mass index (r=0.26), a nonsignificant correlation with the height variable (r=0.03) was obtained, indicating that the height variable does not maintain a first order relation to the ventricular mass, as proposed.

Table II shows the demographic and pressure variables of the hypertensive and normotensive groups according to the body mass index distribution.

Figure 1 has the LVM/h² means in 3 hypertensive subgroups. As shown, significantly higher and progressive values exist in the groups with a higher body mass index.

Comparatively, LVM/BSA evaluation did not show significant differences with increased BMI.

The same analysis was performed in the normotensive population, and the results were similar to those of the hypertensive group (Figure 2), although a nonsignificant trend toward an increase in LVM/BSA parallel to the increase in BMI has been observed.

Finally, the prevalence of ventricular hypertrophy was calculated for the 3 hypertensive subgroups, according to the limited criteria for LVM/BSA and LVM/h² established on the basis of the normotensive population. The results (Table III) show nonsignificant differences in the prevalence of hypertrophy between the 3 population subgroups when the correction LVM/h² (110g/m² and 96g/m²) was used, but when the LVM/h² (77.7 g/m² and 69.8 g/m²) criterion was utilized,

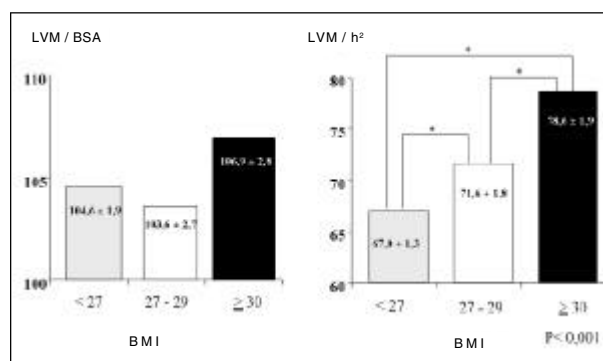


Fig. 1 - Cardiac structure evaluation according to body mass index in hypertensive. * p<0.001; LVM/BSA (g/m²) - left ventricular mass/body surface area; LVM/h² (g/m²) - left ventricular mass/height²; BMI (kg/m²) - body mass index.

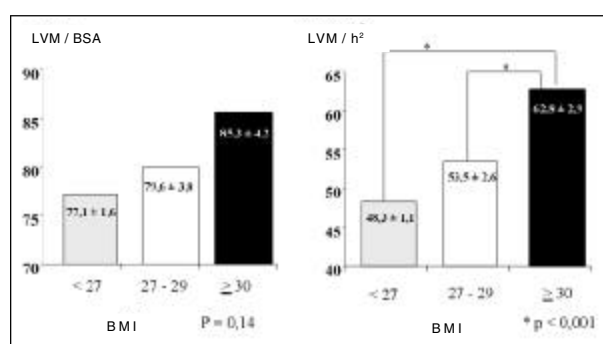


Fig. 2 - Cardiac structure evaluation according to body mass index in normotensive. * p<0.001; LVM/BSA (g/m²) - left ventricular mass/body surface area; LVM/h² (g/m²) - left ventricular mass/height²; BMI (kg/m²) - body mass index.

significant differences between the obese population subgroup and overweight individuals as compared to with those with BMI <27g/m² could be observed.

The utilization of the normally used criteria of LVM/BSA (134g/m² and 110g/m²) for calculating the prevalence of hypertrophy in the hypertensive group, similarly to the 100 and 96g/m² criteria, also did not show significant differences between the 3 subgroups (28.2% vs 27.2% vs 24.8%).

Discussion

Obesity is a risk factor known to be important for left ventricular hypertrophy. The first studies that found an in-

	Normotensive				Hypertensive			
	<27	27-30	>30	P	<27	27-30	>30	P
Kg/m ²	<27	27-30	>30		<27	27-30	>30	
N	79	15	12		287	136	121	
Age	41.2±1.6	44.3±0.7	43.0±3.2	NS	50 ± 0.8	51 ±1.07	50 ±1.15	NS
BMI	23±0.24	27.9±0.18	33.3±0.27	<0.0001	24.3 ± 0.13	28.3 ± 0.07	32.3 ± 0.27	<0.0001
SBP	110 ±1.2	124 ± 3.4	120 ± 2.1	0.02	148± 1.35	144 ± 1.9	148± 2.13	NS
DBP	80± 0.8	81 ± 1.5	80.0 ± 1.2	NS	92± 0.75	93 ±1.1	95.5 ±1.2	NS
SEX	58.3 / 41.7	51.9 / 48.1	46.3 / 53.3	NS	69.3 / 30.7	66.2 / 33.8	67.8 / 32.2	NS
F/M								

BMI (kg/m²)- body mass index; SBP (mmHg)- systolic arterial blood pressure; DBP (mmHg)- diastolic arterial blood pressure

Table III - Prevalence of left ventricular hypertrophy in hypertensive individuals stratified by body mass index according to limits of values established in a normotensive reference population: LVM/BSA (110g/m² men and 96g/m² women) and LVM/h² (77.7g/m² men and 69.8g/m² women).

BMI (kg/m ²)	< 27	27 – 30	≥ 30	p
N	287	136	121	
% LVH	87 (30.3)	59 (43.3) *	58 (47.9) *	< 0.001
LVM/h ²				
% HVE	136 (45.9)	67 (49.2)	53 (43.8)	NS
LVM / BSA				

*p=0.01 vs BMI <27kg/m²; BMI (kg/m²)- body mass index; %LVH-prevalence of left ventricular hypertrophy; LVM/h² (kg/m²)- left ventricular mass indexed squared height; LVM/BSA (kg/m²)- left ventricular mass indexed body surface area.

dependent association between obesity and an increase in left ventricular mass in the 1960s³⁰ were later confirmed by echocardiographic studies^{31,32} and reinforced using larger population studies^{9,33-36}.

Regarding physiology, obese individuals have an increase in intravascular volume and cardiac output to supply the increase in metabolic demands related to increased fatty tissue. In addition, they seem to have an increase in salt intake³⁷ and also a greater sympathetic activity³⁸, which are both mechanisms participating in the genesis of left ventricular hypertrophy^{37,39}.

In addition, obesity is frequently shown to be associated with hypertension⁴⁰, thus being a risk factor besides playing a role in adding to^{32,41} or increasing hypertension with respect to cardiac hypertrophy.

The association of obesity with hypertension can also be seen in studies on body weight reduction that showed falls in blood pressure levels independent of the use of medication⁴². On the other hand, reduction in left ventricular hypertrophy through reduction in body weight independent of falls in blood pressure levels has been shown, also confirming the independent role of obesity to cause hypertrophy⁴³.

Regarding its impact, it has also been demonstrated that obesity is the most potent predictor of the increase in cardiac mass, even surpassing the arterial blood pressure factor^{8,44}.

As has been discussed, the evaluation of the impact of obesity on hypertrophy in certain populations has been subjected to distortion due to indexing with the body surface area, leading to an underestimation of hypertrophy in the obese.

Recent studies, among them the LIFE¹⁵ study and the

VITAE¹⁶ study, conducted in Spain, evaluated the impact of the different selection criteria of the ventricular mass based on different indexes (BSA, height, height²) and identified a higher proportion of obese in the groups where the mass was indexed by height or height raised to a power.

This fact has already been observed in a few studies that evaluated the impact of the different indexes on the prevalence of cardiac structural alterations in their populations^{8,11,14,45}.

In this study, the analysis of ventricular mass correction by body surface area did not show significant differences between the LVM/BSA means of normotensive and hypertensive populations. As already described, the usual correction of mass by body surface area implies the underestimation of hypertrophy in the obese, because it considers obesity as a physiological variable, automatically correcting for weight and height, as can also be seen in Table I.

On adopting the correction by mass/height², in a more practical form than the corrections that use indexed height raised to a power of 2.13 or 2.7, we observe significantly higher mass/height² values in groups with a higher body mass index (Figure 1). This correction, although not being ideal, as well as the correction by lean mass⁴⁶, confers a pathological role to the obesity variable, allowing variations in mass/height² to occur as a function of weight, but exclusively according to changes in body fat (obesity) and not in height. This can be observed in Table I where nonsignificant correlations of height² with mass/height² were obtained.

In view of the fact that mass/height² criteria are not defined for the use in hypertensive populations, we obtained measures for the prevalence of hypertrophy according to the use of our own criterion, based on a local population. Thus, the use of the correction by mass/height² can detect significant differences in the prevalence of overweight and obese populations in relation to individuals with normal body mass index. This fact was not observed using indexes by mass per body surface area, corroborating previous propositions.

Therefore, we may conclude that, for a more reliable evaluation of cardiac hypertrophy in the obese, mainly in populations at higher risk, such as the hypertensive population, limits of values of mass/height² obtained on the basis of normotensive populations, should be used. Thus, we will better detect hypertrophy prevalence in the obese and in this way, better stratify the cardiovascular risk in a situation where two potential risk factors, obesity and hypertension, are already present.

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