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# Impact of abdominal obesity and ambulatory blood pressure in the diagnosis of left ventricular hypertrophy in never treated hypertensives

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ABSTRACT

*Background and objectives:* The principal objective was to assess the prevalence of left ventricular hypertrophy (LVH) in hypertensive, never treated patients, depending on adjustment for body surface or height. Secondary objectives were to determine geometric alterations of the left ventricle and to analyze the interdependence of hypertension and obesity to induce LVH.
*Patients and methods:* Cross-sectional study that included 750 patients (387 men) aged 47 (13, SD) years who underwent ambulatory blood pressure (ABPM) monitoring and echocardiography.
*Results:* The prevalence of LVH was 40.4% (303 patients), adjusted for body surface area (BSA, $LVH_{BSA}$), and 61.7% (463 patients), adjusted for height$^{2.7}$ ($LVH^{2.7}_{height}$). In a multivariate logistic analysis, systolic $BP_{24h}$, gender and presence of elevated microalbuminuria were associated with both $LVH_{BSA}$ and $LVH^{2.7}_{height}$. Increased waist circumference was the strongest independent predictor of $LVH^{2.7}_{height}$, but was not associated with $LVH_{BSA}$. We found a significant interaction between abdominal obesity and systolic $BP_{24h}$ in $LVH^{2.7}_{height}$. Concentric remodelling seems to be the most prevalent alteration of left ventricular geometry in early stages of hypertension (37.5%).
*Conclusions:* The impact of obesity as predictor of LVH in never treated hypertensives is present only when left ventricular mass (LVM) is indexed to height$^{2.7}$. Obesity interacts with systolic $BP_{24h}$ in an additive but not merely synergistic manner. Systolic $BP_{24h}$ is the strongest determinant of LVH when indexed for BSA.



## Impacto de la obesidad abdominal y la presión arterial ambulatoria en el diagnóstico de hipertrofia ventricular izquierda en hipertensos no tratados

*Palabras clave:*
Hipertrofia ventricular izquierda
Obesidad
Presión arterial ambulatoria
Hipertension
Ecocardiografía
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RESUMEN

*Fundamento y objetivos:* El objetivo principal es determinar la prevalencia de la hipertrofia ventricular izquierda (HVI) en hipertensos no tratados previamente según ajuste por superficie corporal o talla. Los objetivos secundarios son establecer las alteraciones de la geometría ventricular y analizar la interdependencia entre hipertensión y obesidad para inducir HVI.
*Pacientes y método:* Estudio transversal en 750 sujetos (387 varones) con una edad media de 47 años (DE 13) a los que se les practicó monitorización ambulatoria de la presión arterial y ecocardiografía.
*Resultados:* La prevalencia de HVI fue del 40,4% (303 pacientes) ajustando por superficie corporal (HVISC), y del 61,7% (463 pacientes) ajustando por talla 2,7 (HVItalla2,7). En un análisis logístico multivariante, la PA sistólica de 24 h, el sexo y la presencia de microalbuminuria elevada se asociaron

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tanto con HVISC como con HVItalla2,7. La obesidad abdominal fue el mayor predictor independiente de HVItalla2,7, pero no se asoció a HVISC. Encontramos una interacción significativa entre obesidad abdominal y PA sistólica de 24 h en la HVItalla2,7. El remodelado concéntrico parece ser la alteración más frecuente de la geometría ventricular en estadios precoces de hipertensión (37,5%).
*Conclusiones:* El impacto de la obesidad como predictor de HVI en hipertensos previamente no tratados solo tiene lugar cuando la masa ventricular izquierda se ajusta por talla 2,7. La obesidad interacciona con la PA sistólica de 24 h de manera aditiva, pero no meramente sinergística. La PA sistólica de 24 h es el mayor determinante de la HVI cuando se ajusta por superficie corporal.


## Introduction

Left ventricular hypertrophy (LVH) may be detected early in uncontrolled hypertension by echocardiography and is recognized as an independent clinical risk factor for cardiac failure, sudden death, myocardial infarction and stroke.[1] Furthermore, LVH completely fulfils the criteria to be considered an adequate surrogate end-point for morbid events in hypertension.[2]

The definition for echocardiographic LVH, however, is far from homogeneous. A recent review including 39 randomized trials on regression of LVH[3] in hypertensive patients found up to 19 different echocardiographic criteria for defining LVH, which leads to a significant distortion of the association between LVH and cardiovascular disease. This high variability reflects the combination of several partition values for three main sources of the physiologic determination of LV mass (LVM): gender, obesity and lean body mass. The vast majority of studies adjust LVM for body surface area (BSA), and since 2003 the European Society of Hypertension has defined the presence or absence of LVH according to a normalization using BSA.[4] Notwithstanding, in recent years allometric height adjustment of LVM has become increasingly recognized as a useful tool in estimating cardiovascular risk of hypertensive patients more accurately, with separate cut-off values for men and for women.[5]

The principal objective of our study was to assess the prevalence of LVH in hypertensive, never treated patients, depending on adjustment for body surface or height. Secondary objectives were to determine geometric alterations of the left ventricle and to analyze the interdependence of the two principal causing factors, hypertension and obesity, to induce LVH.

## Patients and methods

### *Selection of study participants and design*

This cross-sectional study included subjects, all Caucasians, recruited consecutively from the hypertension outpatient clinic of the Hospital General of Sagunto (Sagunto, Spain) from 1st January 2005 to 31st October 2009. Patients, referred from Primary Care, were selected if they had any of the following Blood Pressure (BP) values: (a) office BP $\geq$140 mmHg for systolic and/or $\geq$90 mmHg for diastolic BP in each of three visits within a month's time, or BP values $\geq$130 mmHg for systolic or $\geq$80 mmHg for diastolic recorded during a 24-h ambulatory BP monitoring (ABPM) during the same period (b) echocardiographic assessment of LVM using an acceptable visualization of interfaces, and (c) no previous antihypertensive therapy. Patients with secondary hypertension, nephropathy, diabetes mellitus and urinary tract infection, or previous vascular, cardiac or cerebral disease were excluded. The study was approved by the Ethical Committee of the Sagunto Hospital, and all participants gave informed written consent.

At the beginning, and when appropriate, a clinical work-up was carried out in order to exclude secondary hypertension. After evaluation, patients were placed in usual care treatment. This included a non-pharmacological treatment consisting of moderate salt restriction and a low-calorie diet, if overweight. If necessary, treatment with antihypertensive drugs was started.

### Procedures

BP was measured using a mercury sphygmomanometer following the recommendations of the British Hypertension Society[6] and with a validated Omron HEM 705-CP monitor after 2009. Systolic BP (SBP) and diastolic BP (DBP) were the average of 3 readings measured at 5-min intervals. Blood samples were obtained in the morning after a minimum of 8 h of fasting. Serum biochemical profiles were measured using a multiple-channel autoanalyzer. The glomerular filtration rate was estimated (EGFR) by the MDRD abbreviated formula.[7] Urinary albumin excretion (UAE) was expressed as the ratio of albumin (mg) to creatinine excretion (g). For each patient, the UAE was considered as the mean value obtained in the two separate samples. The cut-off values for the presence of microalbuminuria[4] were $\geq$22 mg/g in men and $\geq$31 mg/g in women. Obesity was defined according to the waist circumference (102 cm in men, 88 cm in women) on the basis of the Guidelines for the Management of Arterial Hypertension[4] and the Adult Treatment Panel III criteria.[8] The definition of metabolic syndrome (MS) followed also these criteria.

### *Ambulatory blood pressure monitoring (ABPM)*

A portable, non-invasive SpaceLabs 90207 recorder (SpaceLabs, Redmond, WA) was used to perform the 24-h ABPM. The BP readings were performed automatically at 15-min intervals during the day and at 20-min intervals during night-time resting. The time periods were standardized according to the time at which the patients rose and retired. Systolic readings $\geq$260 mmHg or $\leq$70 mmHg, diastolic readings $\geq$150 mmHg or $\leq$40 mmHg, and pulse pressure readings $\geq$150 mmHg or $\leq$20 mmHg were automatically discarded.

### *Echocardiography*

Echocardiography was carried out using commercially available instruments (Hewlett Packard Sonos 1000). The recommendations of the American Society of Echocardiography for image orientation[9] were followed. M-mode echocardiograms with two-dimensional guidance were recorded with the patients in the left lateral decubitus position after a rest of at least 10 min. Standard projections were used (longitudinal, parasternal, 2 and 4 chamber apical, and subcostal), directing the M-mode cursor through the centre of the two-dimensional parasternal short-axis image immediately distal to the tips of the mitral valve leaflets. It was mandatory to align the M-mode cursor perpendicular to the long axis of the ventricle to obtain a clear definition of endocardial and epicardial interfaces. Wall thicknesses to calculate LVM were measured at end diastole on at least three cardiac cycles. Only frames with optimal images and showing simultaneously the interventricular septum, left ventricular internal diameter and posterior wall, were employed. All echocardiographic studies were recorded and read by two expert echocardiographists. The