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ORIGINAL ARTICLE

Electrocardiographic Left Ventricular Hypertrophy Criteria and Ambulatory Blood Pressure Monitoring Parameters in Adults

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BACKGROUND
To examine the relationship between ambulatory blood pressure monitoring parameters (ABPM) and electrocardiographic criteria for left-ventricular hypertrophy (LVH) in adults.

METHODS
This study analyzed 1,544 subjects from the EVIDENT study (mean age = 55 ± 14 years; 61% women). A standard electrocardiograph (ECG) and 10 criteria were used to detect LVH. Office and ABPM were performed, and we analyzed 24-hour systolic blood pressure (SBP) and diastolic blood pressure (DBP), percentage of time awake with SBP ≥135 mm Hg, percentage of time asleep with SBP ≥120 mm Hg, and central aortic blood pressure.

RESULTS
LVH according to some electrocardiographic criteria was found in 11.30% of the patients (16.60% of men and 7.70% of women). The patients with LVH were older; had higher values for office, 24-hour and, central aortic blood pressure; were more likely to be men; and had a higher prevalence of obesity, diabetes, and antihypertensive or lipid-lowering drug use. In the logistic regression analysis, the association between the parameters of ABPM and LVH, after adjusting for age, sex, body mass index, and heart rate, remained statistically significant.

CONCLUSIONS
Twenty-four hour blood pressure, the percentage of time with elevated awake and asleep SBPs, and the central systolic blood pressure are related to the presence of LVH as determined by ECG in adults. These results indicate the potential importance of the monitoring and control of different 24-hour parameters of blood pressure in addition to the standard clinic blood pressure with respect to the development of LVH.

CLINICAL TRIAL REGISTRATION
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Left-ventricular hypertrophy (LVH) is considered to be a cardiac condition secondary to hemodynamic stress and is also related to genetic, metabolic, and environmental factors.[1] Detecting and monitoring LVH is important because LVH is a strong predictor of cardiovascular disease and death.[2] Furthermore, its regression with antihypertensive therapy reduces the risk of an adverse outcome.[3,4]

Different methods of diagnosis exist, but the most commonly used in clinical practice is the electrocardiograph (ECG). The ECG has been shown to have prognostic value in different studies in the general population,[5] although this value is variable for the different criteria used in each study.[6] Because the ECG criteria identify patients with different profiles,[7] the sensitivity can be improved by using combined criteria and automating the ECG readings.[8–10] To this effect, the North American guidelines recommend the use of multiple ECG criteria to detect LVH in clinical practice.[11]

Moreover, ambulatory blood pressure monitoring (ABPM) is a stronger cardiovascular risk and target organ damage predictor[12,13] than office blood pressure.[14,15]

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Table 1. Left-ventricular hypertrophy criteria (definitions) and prevalence of left-ventricular hypertrophy

Criteria	Equations	LVH diagnostic value	Prevalence LVH (n = 1,544)
Voltage			
Sokolow	S (V1) + max (R(V5, V6)	≥3.8 mV (38 mm)	0.7%
Cornell	R (aVL) + S (V3)	Male: ≥2.8 mV (28 mm);	0.3%
		Female: ≥2.0 mV (20 mm)	
Gubner-Ungerleider	R (I) + S (III)	>2.5 mV (25 mm)	2.2%
Lewis	(R (I) + S (III)) − (R (III)+S(I))	>1.7 mV (17 mm)	8.4%
Voltage-duration QRS product			
Sokolow	Sokolow voltage × QRS duration	Male: >367.4 mV ms (3,674 mm ms)	0.9%
		Female: >322.4 mV ms (3,224 mm ms)	
Cornell	Male: R(aVL) + S(V3) × QRS duration;	>244 mV (2,440 mm)	1.5%
	Female: (R(aVL) + S(V3) + 0,6) × QRS duration		
Gubner-Ungerleider	Voltage × QRS duration	>207 mV ms (2,070 mm ms)	3.7%
Estimation LVMI			
Novacode LVMI	Male: 0.010(R V5) + 0.0203 (Q or S V1) + 0.0287(Q or S III) + 0.1819(T V6) − 0.1482(T aVR) + 1.0485(QRS duration) − 36.429	Male: ≥ 130 g/m ^{2}	0.2%
	Female: 0.0178(R V5) + 0.0528 (Q or S V5) − 0.1128(Q or S I) + 0.1075(T V1) + 0.1701(T aVF) − 0.0939(T V6) + 88.4357	Female: ≥ 115 g/m^2	
Composite criteria			
Minnesota Code	(RV5/V6 or	> 2.6 mV (26 mm)	3.6%
	RI/II/III/aVF or	> 2 mV (20 mm)	
	RaVL)	> 1.2 mV (12 mm)	
Framingham-adjusted Cornell voltage	Male: [RaVL + SV3 + 0.0174 × (age – 49) + 0.191 × (BMI – 26.5)];	≥ 2.8 mV (28 mm)	0.3%
	Female: [RaVL + SV3 + 0.0387 × (age – 50) + 0.212 × (BMI – 24.9)]	≥ 2.0 mV (20 mm)	
Combined criterion	All criteria	At least 1 positive criterion	11.3%

Abbreviations: BMI, body mass index; LVH, left ventricular hypertrophy; LVMI, left ventricular mass index; qRS, QRS complex of the ECG.

The nondipping pattern of blood pressure is associated with increased cardiovascular events in hypertensive patients and in the general population.[16–18] ABPM measurements correlate equally well with LVH indices in individuals with hypertension or type 2 diabetes.[19,20]

We did not find any study that analyzed the relationship between LVH defined by electrocardiographic criteria and different measures of blood pressure assessed by ABPM in an adult population.

The objective of this study was to examine the relationship between ABPM and central aortic parameters and electrocardiographic LVH criteria in adults.

METHODS

The 1,544 patients analyzed in this study were part of the EVIDENT study (ClinicalTrials.gov identifier NCT01083082).[21]

Subjects

Patients ranging 20–80 years of age were selected through random sampling from general practitioner offices in 6 health centers. The exclusion criteria were the following: known coronary or cerebrovascular atherosclerotic disease; heart failure; moderate or severe chronic obstructive pulmonary disease; walk-limiting musculoskeletal disease; advanced respiratory, renal, or hepatic disease; severe mental disease; treated oncological disease diagnosed in the past 5 years; terminally ill patients; and pregnant women.

Of the 1,553 subjects included in the EVIDENT study, 9 were excluded because they did not have an ECG. Therefore, this study analyzed 1,544 subjects in total. The sample size calculation indicated that the number of patients included in the study (n = 1,544) was sufficient to detect a correlation coefficient of 0.10 between ABPM parameters and ECG criteria, accepting an alpha risk of 0.05 and a beta risk of 0.10 in a 2-sided test.

Table 2. Baseline demographic and clinical characteristics in patients with and without left-ventricular hypertrophy

Variables	Total (n = 1,544)	LVH (+) (n = 174)	LVH (−) (n = 1,370)	*P* value
Age, y	55.7 (45.2–65.8)	60.2(51.2–68.4)	55.1 (44.7–65.4)	<0.01
Male	39.7%	58.6%	37.3%	<0.01
Office BP				
SBP, mmHg	125 ± 17	133 ± 18	124 ± 17	<0.01
DBP, mmHg	77 ± 10	82 ± 11	76 ± 10	<0.01
PP, mmHg	48 ± 13	51 ± 13	48 ± 13	<0.01
HR, bpm	72 ± 12	69 ± 12	72 ± 11	<0.01
Hypertensive	40.6%	63.8%	37.7%	<0.01
Antyhypertensive drugs	29.1%	48.3%	26.6%	<0.01
Diuretics	11.8%	17.2%	11.1%	0.02
ACE inhibitors	9.1%	12.1%	8.7%	0.14
ARBs	8.4%	12.1%	8.0%	0.06
Calcium channel blockers	1.4%	4.6%	1.0%	<0.01
Smokers	21.8%	14.4%	22.7%	0.01
Body mass index, kg/m^2	26.6 (24.0–29.6)	27.9 (25.8–31.1)	26.4 (23.8–29.4)	<0.01
Obesity	21.8%	35.1%	20.1%	<0.01
Total cholesterol, mg/dl	213.9 ± 39.0	214.4 ± 39.5	213.8 ± 38.9	0.85
Hypercholesteremie	31.6%	38.5%	30.7%	0.04
Lipid-lowering drugs	19.2%	27.0%	18.2%	<0.01
Fasting glucose, mg/dl	89 (83–98)	93(83–104)	89(83–98)	0.02
Diabetics	11.5%	13.2%	11.3%	0.46
Antidiabetic drugs	8.0%	8.0%	8.0%	0.97

Continuous variables are presented as mean ± SD or median (interquartile range). Data for qualitative variables are expressed as percentages. *P* values are for overall comparison all subgroups by Student *t* test independent groups, Mann–Whitney *U* test, and χ^2 or Fisher test. Hypertensive was defined as systolic blood presusre ≥ 140 and/or diastolic blood pressure ≥ 90 mm Hg or presence of antihypertensive medication. Obesity was defined as a body mass index > 30 kg/m^2. Hypercholesteremie was defined as total cholesterol ≥ 250 mg/dl or on statin treatment.

Abbreviations: ACE, angiotensin-converting enzyme inhibitor; ARB: angiotensin receptor blocker; BP, blood pressure; DBP, diastolic blood pressure; HR, heart rate; LVH, left ventricular hypertrophy; PP, pulse pressure; SBP, systolic blood pressure;

The study was approved by the independent ethics committee of Salamanca University Hospital (Spain), and all of the participants gave written informed consent according to the general recommendations of the Declaration of Helsinki.[22]

Measurement

A detailed description has been published elsewhere for how the clinical data were collected, the anthropometric measurements were made, and the lab tests were obtained.[21]

Cardiac assessment. Standard 12-lead ECGs were digitally acquired using a General Electric MAC 3.500 ECG System (General Electric, Niskayuna, NY) at 10 mm/mV calibration and a speed of 25 mm/sec. All of the ECGs were read automatically for measures of voltage and duration of waves. The definition of the 10 criteria and cutoff points for diagnosing LVH are presented in Table 1. We considered LVH to be present when at least 1 of the 10 criteria used exceeded the cutoff established in Table 1. We defined Lewis-Voltage duration product (VDP) = (R (I)+S (III)) − (R (III)+S(I)) × QRS duration. Where R is R wave of the QRS complex in the ECG, I is Derivation DI, III is Derivation DIII, S is S wave of the ECG, and QRS wave complex of the ECG.

Office or clinical blood pressure. The office blood pressure was calculated as the average of the last 2 of 3 measurements of systolic (SBP) and diastolic blood pressure (DBP), made with a validated sphygmomanometer (OMRON model M10-IT; Omron Health Care, Kyoto, Japan). The measurements were made on the right upper arm of participants in a seated position after at least 5 minutes of rest, with a cuff of appropriate size as determined by a measurement of the upper-arm circumference and following the recommendations of the European Society of Hypertension.[23] The pulse pressure was estimated from the mean values of the 2nd and 3rd blood pressure measurements. Hypertension was defined as the use of antihypertensive medications or when the mean of 3 recordings in the clinic at baseline and at separate times, was ≥140 mm Hg for SBP and/or ≥90 mm Hg for DBP.

Ambulatory blood pressure monitoring. ABPM was performed on a day of standard activity with a radial tonometer. A radial pulse wave acquisition device (B-Pro; HealthSTATS International, Singapore) validated according to the protocol of the European Society of Hypertension, the Association for the Advancement of Medical Instrumentation, and the British Hypertension Society[24,25] was used. Valid registries were required to fulfill a series of pre-established criteria, including ≥80% successful SBP and DBP recordings during the daytime and nighttime periods over a period of 24 hours and with ≥1 blood pressure measurement per hour. The monitor was scheduled for obtaining blood pressure measurements every 15 minutes during the day and night.

Central blood pressure. Central blood pressure was measured with Pulse Wave Application Software (B-Pro; HealthSTATS International) using tonometry to capture the radial pulse and an equation to estimate the central blood pressure.[26,27]

Statistical analysis

The continuous variables were expressed as the mean ± SD for normally distributed data and as the median (interquartile range) for asymmetrically distributed data. Frequency distributions were used for categorical data. Statistical normality was checked using the Kolmogorov–Smirnov test. The χ^2 and Fisher exact tests were used to analyze the association between qualitative variables. The Student *t* test

Table 3. Ambulatory blood pressure monitoring parameters and central blood pressure in patients with and without left-ventricular hypertrophy

	LVH (+) (n = 174)	LVH (−) N= 1370	
Variables	**Mean ± SD**	**Mean ± SD**	***P* value**
24-h SBP, mm Hg	129 ± 18	120 ± 17	<0.01
24-h DBP, mm Hg	82 ± 12	76 ± 11	<0.01
24-h PP, mm Hg	47 ± 12	44 ± 12	<0.01
Awake SBP, mm Hg	134 ± 18	124 ± 18	<0.01
Sleep SBP, mm Hg	119 ± 18	111 ± 17	<0.01
Awake DBP, mm Hg	85 ± 13	111 ± 17	<0.01
Sleep DBP, mm Hg	75 ± 11	79 ± 11	<0.01
Sleep/awake ratio SBP	0.89 ± 0.07	0.90 ± 0.07	0.14
% Awake time SBP >135 mm Hg	45.2 ± 35.7	29.7 ± 32.5	<0.01
% Sleep time SBP >120 mm Hg	40.7 ± 41.4	28.7 ± 37.7	<0.01
Central SBP, mmHg	123 ± 17	116 ± 16	<0.01
Central DBP, mmHg	11.5%	13.2%	11.3%
Central PP, mmHg	8.0%	8.0%	8.0%

Continuous variables are presented as mean ± SD. *P* values are for overall comparison all subgroups by Student *t* test independent groups. Abbreviations: DBP, diastolic blood pressure; LVH, left ventricular hypertrophy; PP, pulse pressure; SBP, systolic blood pressure.

and the Mann–Whitney *U* test were used, as appropriate, to analyze the association between quantitative and qualitative variables from 2 categories. Spearman or Pearson correlation coefficient was used to estimate the relationship between quantitative variables. Five logistic regression models were developed, including as the dependent variable the absence (0) or presence (1) of LVH. The independent variables for each model were 24-hour SBP, 24-hour DBP, central aortic SBP, percentage of time awake with an SBP ≥135 mm Hg, and percentage of time asleep with an SBP ≥120 mm Hg. The enter method was used in all of the models to include the adjusting variables: sex (male = 1; female = 0), age, body mass index, heart rate, and antihypertensive drug use (yes = 1; no = 0). The data were analyzed using Statistical Package for the Social Sciences software version 20.0 (SPSS, Chicago, IL, USA). A value of $P < 0.05$ was considered to be statistically significant.

RESULTS

Table 1 shows the criteria used and the percentage of patients presenting with LVH according to each variable and considered globally. Of the 1,544 patients studied, 174 (11.30%) presented some ECG criteria of LVH. The proportion of patients with LVH was 7.70% (n = 72) for the women and 16.60% (n = 102) for the men.

The descriptive data for the patients with and without LVH on ECG are shown in Table 2. The patients with LVH were comparatively older, and a higher proportion were men. In addition, they had a higher clinic blood pressure, body mass index, and fasting glucose. They also used more antihypertensive and lipid-lowering drugs than the patients without LVH. All of the parameters from ABPM and central aortic SBP were higher for the group of patients with LVH, with the exception of the sleep/awake SBP and DBP ratios (Table 3).

The ECG criteria showed a positive correlation with the 24-hour SBP, 24-hour DBP, central aortic SBP, percentage of time awake with an SBP ≥135 mm Hg, and percentage of time asleep with an SBP ≥120 mm Hg and a negative correlation with the office heart rate and sleep/awake SBP and DBP ratios. The criteria that most closely correlated with the 24-hour DBP ($r = 0.19$), percentage of time awake with an SBP ≥ 135 mm Hg ($r = 0.14$), percentage of time asleep with an SBP ≥120 mm Hg ($r = 0.09$), and sleep/awake ratio for DBP ($r = -0.11$) were the Cornell criteria. The criteria that most closely correlated with the 24-hour SBP were the Voltage duration product (VDP) Gubner–Underleider criteria ($r = 0.14$). The criteria that most closely correlated with the central aortic SBP were the VDP Gubner–Underleider and Lewis criteria ($r = 0.19$). Finally, the criteria that most closely correlated with the sleep/awake SBP ratio were the VDP Cornell criteria ($r = -0.12$) (Table 4).

In the logistic regression analysis, using the presence or absence of LVH according to some of the criteria as the dependent variable and 24-hour SBP, 24-hour DBP, central aortic SBP, percentage of time awake with an SBP ≥135 mm Hg, and percentage of time asleep with an SBP ≥120 mm Hg as the independent variables, the odds ratio in all of the models, after adjusting for age, sex, body mass index, and heart rate, maintained statistical significance (Table 5).

Table 4. Correlations of left-ventricular hypertrophy criteria and ambulatory blood pressure monitoring measurement

	Office HR	24-h SBP	24-h DBP	Sleep/awake SBP ratio	Sleep/ awake DBP ratio	Central aortic SBP	% Awake time SBP ≥135 mm Hg	% Sleep time SBP ≥120 mm Hg
Sokolow	−0.13**	−0.06*	0.08**	−0.04	−0.04	−0.01	0.06*	0.01
Cornell	−0.19**	0.14**	0.19**	−0.11**	−0.11**	0.17**	0.14**	0.09**
Gubner–Ungerleider	−0.13**	0.12**	0.17**	−0.06*	−0.07*	0.18**	0.12**	0.08**
Lewis	−0.12**	0.12**	0.17**	−0.07**	−0.06*	0.19**	0.12**	0.09**
VDP Sokolow	−0.15**	0.08**	0.09**	−0.04	−0.04	0.02	0.08**	0.04
VDP Cornell	−0.17**	0.09*	0.14**	−0.12**	−0.08*	0.12**	0.03	−0.01
VDP Gubner–Ungerleider	−0.14**	0.14**	0.17**	−0.07**	−0.07**	0.19**	0.13**	0.09**
Novacode LVMI	−0.07	0.03	−0.01	−0.06	−0.06	−0.05	0.03	−0.01
Framingham-adjusted Cornell voltage	−0.15**	0.11*	0.17**	−0.10*	−0.06	0.17**	0.04	0.01
VDP Lewis	−0.13**	0.12**	0.17**	−0.07**	−0.07*	0.19**	0.12**	0.09**

P values by Spearman or Pearson correlation.
Abbreviations: DBP, diastolic blood pressure; HR, heart rate; LVH, left ventricular hypertrophy; SBP, systolic blood pressure; VDP, voltage duration product.
*$P < 0.05$.
**$P < 0.01$.

Table 5. Logistic regression with left-ventricular hypertrophy (some criteria) as dependent variable and ambulatory blood pressure monitoring parameters as independent variable

Dependent variable: combined criterion LVH	OR (95% CI)	*P* value
Model 1: Including 24-h SBP	**1.02 (1.01–1.03)**	**<0.01**
Sex (male = 1; female = 0)	1.85 (1.31–2.61)	<0.01
Age	1.01 (1.00–1.03)	0.10
BMI	1.06 (1.02–1.10)	0.01
Antihypertensive drugs (yes = 1; no = 0)	1.60 (1.10–2.31)	0.01
Office HR	0.97 (0.99–0.99)	<0.01
Model 2: Including 24-h DBP	**1.04 (1.02–1.05)**	**<0.01**
Sex (male = 1; female = 0)	1.85 (1.31–2.61)	<0.01
Age	1.05 (1.00–1.03)	0.03
BMI	1.06 (1.02–1.10)	0.01
Antihypertensive drugs (yes = 1; no = 0)	1.68 (1.16–2.43)	0.01
Office HR	0.97 (0.96–0.99)	<0.01
Model 3: Including % awake time SBP ≥135 mm Hg	**1.01 (1.00–1.01)**	**0.01**
Sex (male = 1; female = 0)	1.90 (1.34–2.68)	<0.01
Age	1.01 (1.00–1.04)	0.059
BMI	1.06 (1.02–1.10)	0.01
Antihypertensive drugs (yes = 1; no = 0)	1.58 (1.09–2.30)	0.02
Office HR	0.97 (0.96–0.99)	<0.01
Model 4: Including % sleep time SBP ≥120 mm Hg	**1.01 (1.00–1.01)**	**0.01**
Sex (male = 1; female = 0)	1.97 (1.40–2.78)	<0.01
Age	1.02 (1.01–1.03)	0.04
BMI	1.07 (1.03–1.11)	0.01
Antihypertensive drugs (yes = 1; no = 0)	1.58 (1.09–2.30)	0.02
Office HR	0.97 (0.96–0.99)	<0.01
Model 5: Including central aortic systolic pressure	**1.01 (1.01–1.03)**	**0.02**
Sex (male = 1; female = 0)	1.91(1.36–2.68)	<0.01
Age	1.01 (1.00–1.03)	0.07
BMI	1.06 (1.02–1.10)	0.01
Antihypertensive drugs (yes = 1; no = 0)	1.51 (1.04–2.19)	0.03
Office HR	0.97 (0.96–0.99)	<0.01

Abbreviations: BMI, body mass index; CI, confidence interval; DBP, diastolic blood pressure; HR, heart rate; LVH, left ventricular hypertrophy; OR, odds ratio; SBP, systolic blood pressure.

The bolded values indicates the principal data of each model.

The association found between 24-hour SBP and LVH is independent of the office SBP. However, the association between 24-hour DBP and LVH is dependent on the office DBP.

DISCUSSION

This study analyzed the relationship between electrocardiographic criteria for LVH with different ABPM parameters and central aortic SBP in a random sample of patients from a primary care, multicenter study.

According to the results of this study, most of the electrocardiographic criteria for detecting LVH in an adult population showed a positive correlation with different ABPM measurements and central aortic SBP. Likewise, in the logistic regression model after adjustment, the association remained between the different parameters and the presence of left-ventricular hypertrophy. Finally, the data from this work suggest that the 24-hour SBP and office DBP are the best predictors of the presence of LVH by electrocardiographic criteria. This fact can be explained by the variability in the SBP in clinical practice. These findings suggest the importance of using long-acting antihypertensive drugs to achieve good blood pressure control over a 24-hour period.

Several studies have shown a good correlation between the mean 24-hour SBP and parameters to assess LVH among

hypertensive subjects.[28] Coll-de-Tuero *et al.*[15] analyzed the influence of blood pressure, as assessed by ABPM, on yearly changes in LVH after adjustment in a multivariable analysis. They showed that hypertensive patients with a baseline ABPM >135/85 mm Hg had a worse evolution of LVH (odds ratio = 1.9; 95% confidence interval = 1.5–2.5). In addition, a good correlation was found between electrocardiographic criteria and the mean SBP and DBP as assessed by home blood pressure but only for night measurements.[29] The PAMELA study[30] demonstrated a better correlation of the left ventricular mass index with 24-hour SBP than with 24-hour DBP.

In this study, we show the importance of other parameters obtained by ABPM that were not previously analyzed and found that more than the mean SBP value is associated with the LVH. The percentage of time awake with a SBP ≥135 mm Hg and the percentage of time asleep with a SBP ≥120 mm Hg were the most likely to influence the development and/or evolution of LVH.

The Hermex study[31] analyzed a sample of 2,564 patients in the general population, using a combination of 17 criteria, and found the prevalence of LVH to be 36.20%. This number contrasts with the 11.30% found in our study, most likely because of the high number of criteria used in the Hermex study, which increases the ability to detect LVH in the population analyzed.

This result implies a detection rate more than twice that when using only Cornell-VDP, which is the most widely recommended criteria. This observation reinforces the idea that the different electrocardiographic criteria can be complementary and that their combined use in detecting LVH offers greater sensitivity.[7] Moreover, the American guidelines recommend the use of multiple criteria ECG in clinical practice based on varying ability of each criterion to detect LVH in different patients according to sex, age, race, body type, or even the geometric pattern of the hypertrophy.[11]

The main limitation of this study was that the data originated from a cross-sectional study, which prevented us from establishing a temporal relationship between the different ABPM measurements and LVH. We must also take into account the usual circadian variability pressure because the blood pressure will vary on different days. The other limitation is that LVH was measured using electrocardiographic criteria and not by echocardiography. The latter is considered to be the gold standard for diagnosing LVH.

Twenty-four hour blood pressure, the percentage of time that the awake and asleep SBPs are above the reference values, and the central systolic blood pressure are related to the presence of LVH as determined by ECG in adults. These results indicate the potential importance of the monitoring and control of different 24-hour parameters of blood pressure in addition to the standard clinic blood pressure with respect to the development of LVH. Further prospective studies are needed to clarify the biological mechanisms and the contribution of each of these parameters in the development of the LVH.

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DISCLOSURE

The authors declared no conflict of interest.

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