Published in final edited form as:
Int J Cardiovasc Imaging. 2010 April ; 26(4): 459–468. doi:10.1007/s10554-010-9584-5.

Body size adjustments for left ventricular mass by cardiovascular magnetic resonance and their impact on left ventricular hypertrophy classification

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Abstract

Methods to index left ventricular (LV) mass, measured by cardiovascular magnetic resonance (CMR), for body size have not been investigated. The purposes of this study were to develop allometric indices for LV mass measured by CMR and compare estimates of the prevalence and predictive value of LV hypertrophy defined by a new allometric height-weight index, LV mass/body surface area (BSA), height indices (a new allometric height index; and previously derived indices from echocardiographic measurements: LV mass/height^2, LV mass/height$^{2.7}$), and non-indexed LV mass. 5,004 participants from the Multi-Ethnic Study of Atherosclerosis (MESA) with CMR measurements of LV mass and no clinical cardiovascular disease at baseline were followed for a median of 4.1 years. The new indices and limits for hypertrophy (95th percentile) were derived from 822 normal-weight, normotensive, non-diabetic MESA participants. 107 events (coronary heart disease or stroke) were observed. The estimated prevalence of hypertrophy at baseline and hazard ratio for event associated with hypertrophy were 8% and 2.4 with the new allometric height-weight index, 11% and 2.2 with LV mass/BSA, 23–24% and 2.0–2.1 with height indices, and 20% and 1.7 with non-indexed LV mass. A statistically significant difference was

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detected between the hazard ratios based on the new height-weight index and non-indexed LV mass. The prevalence of hypertrophy is higher for indices that do not account for weight. The predictive value of hypertrophy is significantly better with the new allometric height-weight index than with non-indexed LV mass and may be better than indices without weight.

Keywords

Cardiovascular risk; Hypertension; Hypertrophy; LV mass index; Magnetic resonance imaging; Obesity

Background

Left ventricular (LV) mass is an important predictor of cardiovascular morbidity and mortality [1–9]. LV mass can be accurately and directly determined using cardiac magnetic resonance (CMR). Abnormally increased LV mass, termed LV hypertrophy, is a strong predictor of cardiovascular disease events for individuals without [1–4] as well as with prior known coronary heart disease [5,6] and congestive heart failure (CHF) [7–9].

Left ventricle mass is known to increase in proportion to overall body size and also differs by gender [10]. Thus, in order to assess an individual's risk for a cardiovascular event based on heart size, an adjustment for the patient's body size must be derived. An LV mass "index" is derived by dividing LV mass by factors that include body height and/or weight. The normal range of LV mass index can then be derived from a *reference sample* of individuals believed to be free of significant risk factors that could otherwise cause LV enlargement. To be clinically useful, an indexed LV mass should be more predictive of a cardiovascular event than non-indexed LV mass.

The optimal method to account for body size remains controversial [11]. LV mass divided by body surface area (BSA) is frequently used clinically to account for body size. However, the most commonly used formula for computing BSA is based on a study of 9 individuals published in 1,916 [12] and its validity is unclear. Other indices to adjust LV mass for body size based on a function of height have been derived by echocardiography studies of LV mass [13–17]. Echocardiographic measurement of LV mass is based on geometric assumptions regarding the shape of the ventricle rather than three-dimensional measurements available with CMR.

An investigation of indices for CMR data has not previously been performed. The purposes of this study were to develop allometric indices for LV mass measured by CMR and compare estimates of the prevalence and predictive value of LV hypertrophy, defined by the new indices and previously derived indices, from a multi-ethnic cohort in the United States.

Methods

Study sample

Data for this investigation were derived from the Multi-Ethnic Study of Atherosclerosis (MESA). The design and objectives of MESA have been described [18]. Briefly, between July 2000 and September 2002, 6,814 men and women age 45–84 years were recruited from six US communities. Participants were recruited from four ethnic groups and were free of clinically recognized cardiovascular disease, including myocardial infarction, angina, coronary revascularization, congestive heart failure, atrial fibrillation, stroke, transient ischemic attack, valvular disease, and peripheral vascular disease.

Two subsets of participants were defined for this investigation: all MESA participants who completed a CMR exam and who had technically adequate data; and a *reference sample*. The reference sample consisted of participants who (a) had a technically adequate CMR examination, (b) were of normal weight, (c) did not have hypertension, and (d) did not have diabetes or impaired fasting glucose. Normal weight was defined as body mass index (BMI) less than 25 kg/m^2, consistent with that defined by the National Heart, Lung and Blood Institute and the World Health Organization [19,20]. Hypertension was defined according to the JNC-VI criteria [21] as a systolic blood pressure value ≥ 140 mmHg, a diastolic blood pressure value ≥ 90 mmHg, or current drug treatment for hypertension. Diabetes was defined as fasting glucose ≥ 126 mg/dL or use of hypoglycemic medication. Impaired fasting glucose was defined as fasting glucose 100–125 mg/dL [22].

Measurements

Participants' weight, height, blood pressure, age, gender, ethnicity, and various cardiovascular risk factors were recorded at the baseline exam. Weight was measured to the nearest pound using a balance scale and height was measured to the nearest 0.1 cm with the participant in light clothing and stocking feet. Blood pressure was measured three times in the seated position with a Dinamap device [23]; the average of the second and third measurements was used in the analysis. Fasting glucose was measured by a thin film adaptation of the glucose oxidase method (Johnson & Johnson Clinical Diagnostics, Inc., Rochester, NY). Body mass index (kg/m^2) was calculated [24] as $weight/height^2$.

Cardiac magnetic resonance imaging

Participants underwent CMR a median of 16 days after enrollment; 95% were completed by 11 weeks after enrollment. CMR examinations were performed using 1.5-Tesla magnets at the 6 MESA field centers: Wake Forest University (Signa CV/i, General Electric Medical Systems, Waukesha, Wisconsin), Columbia University (Signa LX, General Electric), Johns Hopkins University (Signa CV/i, General Electric), University of Minnesota (Vision/Sonata, Siemens, Erlangen, Germany), Northwestern University (Sonata, Siemens), University of California at Los Angeles (Signa LX, General Electric, Vision, Siemens). All imaging was performed with a four-element, phased-array surface coil placed anteriorly and posteriorly, electrocardiogram gating, and brachial artery blood pressure monitoring. The CMR imaging protocol and inter- and intra-observer reproducibility studies have been previously described [10]. In brief, cine images of the left ventricle were obtained in the short axis plane using a fast gradient echo pulse sequence with end-expiratory breath-holding and with temporal resolution less than or equal to 50 ms. Imaging data were analyzed using MASS software (version 4.2, Medis, The Netherlands) at a single reading center by readers trained in the MESA protocol and without knowledge of risk factor information. LV mass was determined by the sum of the myocardial area (the difference between endocardial and epicardial contour) times slice thickness plus image gap in the end diastolic phase multiplied by the specific gravity of myocardium (1.05 g/ml) as described previously [10].

Cardiovascular disease events (combined nonfatal and fatal coronary heart disease and stroke)

The collection, classification, and adjudication of follow-up information from MESA participants have been described [25]. For this report, MESA participants were followed for a median of 4.1 years. Nonfatal coronary heart disease (CHD) events included either definite or probable myocardial infarction (defined primarily on a combination of symptoms, ECG, and cardiac biomarker levels), and resuscitated cardiac arrest. Fatal CHD was defined by a documented myocardial infarction within the previous 28 days, chest pain within the 72 h before death, or a history of CHD, and required the absence of a known non-atherosclerotic or non-cardiac cause of death. Stroke was defined by a rapid onset of a documented focal

neurologic deficit lasting at least 24 h (that was not secondary to brain trauma, tumor, infection, or other non-vascular cause), or a clinically relevant lesion on a brain image.

Indices of LV mass

Previously described indices—LV mass divided by body surface area (LV mass/BSA) is frequently used clinically to index LV mass. BSA (in m^2) was calculated as [12]:

$$\text{BSA}=0.007184 \times (\text{weight in kilograms})^{0.425} \times (\text{height in centimeters})^{0.725} \quad (1)$$

Based on prior population based studies using echocardiography to determine LV mass, two indices based on height alone have been reported: LV mass/height$^{2.7}$ (de Simone et al. [13–15]); and LV mass/height^2 (Lauer et al. [16]).

New indices—Two indices were derived from linear regression models fit to CMR data from the *reference sample* of MESA participants. The indices are allometric (Dewey et al. [11]) as they are proportional to LV mass divided by a body size variable raised to a scalar exponent. Dewey et al. [11] describe potential benefits of the allometric approach.

Percent-predicted LV mass based on height and gender ($ppLVmass_H$): A linear regression model with log-transformed LV mass as the response, and log-transformed height and gender as the predictors was estimated. Gender was included in the model because we expected gender to confound the effect of log-transformed height [16]. The model assumes that the coefficient of log-transformed height, typically called the height exponent [13–16], is the same for males and females. We defined the index, $ppLVmass_H$, as LV mass divided by height raised to the power of the height exponent and by the exponential of the intercept (or the sum of the intercept and coefficient of gender). This leads to a natural interpretation of the index. The index multiplied by 100 is equivalent to the percentage of the value predicted on the basis of height and gender. A $ppLVmass_H$ value of 1 suggests that LV mass is equal to that predicted based on height and gender. A value greater than one suggests LV mass is larger than that predicted, while a value less than one suggests LV mass is smaller than that predicted.

Percent-predicted LV mass based on height, weight, and gender ($ppLVmass_{HW}$): A linear regression model with log-transformed LV mass as the response, and log-transformed height, log-transformed weight, and gender as the predictors was estimated. Weight was included because weight is part of the common formula for computing body surface area, and lean body mass (which is correlated with weight) explains a large proportion of the variability in LV mass. The index, $ppLVmass_{HW}$, was defined as LV mass divided by height raised to the power of the coefficient of log-transformed height, by weight raised to the power of the coefficient of log-transformed weight (or the "weight exponent"), and by the exponential of the intercept (or the sum of the intercept and coefficient of gender).

Definition of LV hypertrophy

Presence of LV hypertrophy was defined by an LV mass index (or non-indexed) value greater than the 95th upper percentile of indexed (or non-indexed) LV mass in the reference sample. The 95th percentile of the indexed LV mass in a healthy sample has commonly been used as an upper limit for "normal" LV mass [26,27]. For each of the previously described indices of LV mass and of non-indexed LV mass, the 95th percentile was estimated (from the empirical cumulative distribution) separately for men and women. For each of $ppLVmass_H$ and $ppLVmass_{HW}$, the 95th percentile was estimated from men and women combined since these indices already account for gender.

Risk of cardiovascular event

The association between time to a cardiovascular event (combined nonfatal and fatal CHD and stroke) and LV hypertrophy was estimated using Cox-proportional hazard models, with adjustment for age and gender. The hazard ratios obtained with the different indices of LV mass and with non-indexed LV mass were compared using a bootstrap approach, similar to that described by Liao et al. [28].

Results

Subject characteristics

Of the 6,814 MESA participants, 5,098 underwent CMR; 5,004 provided technically adequate data. Of these 5,004 participants, 3,458 were overweight or obese, 2,120 had hypertension, and 1,982 had impaired fasting glucose or diabetes. Thirteen participants had unknown diabetes status and were excluded from the reference sample. Thus, the final reference sample consisted of 822 participants. Characteristics of the participants are provided in Table 1.

Indices ($ppLVmass_H$ and $ppLVmass_{HW}$) based on CMR data from the reference sample

The results from fitting the regression models are shown in Table 2. The index, $ppLVmass_H$, was estimated as LV mass divided by 42.5 × (height in meters)$^{1.88}$ for women, and LV mass divided by 51.4 × (height in meters)$^{1.88}$ for men. The index, $ppLVmass_{HW}$, was estimated as LV mass divided by 6.82 × (height in meters)$^{0.561}$ × (weight in kilograms) $^{0.608}$ for women, and LV mass divided by 8.17 × (height in meters)$^{0.561}$ × (weight in kilograms) $^{0.608}$ for men. The height and weight exponents (0.561 and 0.608) in $ppLVmass_{HW}$ differ from the exponents in the common formula for BSA (0.725 and 0.425 in Eq. 1). The constant in $ppLVmass_{HW}$ varies by gender (6.82 for women and 8.17 for men); both constants are larger than the constant for BSA ($0.007184 \times 0.01^{0.725}$ when height is expressed in meters instead of centimeters in Eq. 1). The constant in $ppLVmass_{HW}$ allows $ppLVmass_{HW} \times 100$ to be interpreted as the percentage of the value predicted based on height, weight and gender.

Correlation of the LV mass indices with height and weight

The Pearson correlation coefficients of the LV mass indices with each of height and weight, by gender, in the reference sample are shown in Table 3. An index that adequately accounts for body size should have low correlation with body size in the reference sample. The correlations of $ppLVmass_H$ with height, and of $ppLVmass_{HW}$ with each of height and weight were low (range |r|, 0.01 to 0.04). The correlation of LV mass/height^2 with height was also very low. In contrast, the correlation of LV mass with height and with weight, and of LV mass/height$^{2.7}$ with height were relatively high (range, |r| = 0.19 to 0.51). The correlation between LV mass/Body surface area and each of height and weight were low to moderate (range, |r| = 0.05 to 0.14).

Limits for LV hypertrophy based on CMR data from the reference sample

The estimated 95th percentiles of indexed and non-indexed LV mass are listed in Table 4. The 95th percentile of the $ppLVmass_H$ values was estimated as 1.33. In other words, 95% of the reference sample had LV mass values less than 133% of that predicted on the basis of gender and height. The 95th percentile of the $ppLVmass_{HW}$ values was estimated as 1.31.

Prevalence of LV hypertrophy

The percentage of MESA participants with LV hypertrophy, defined by the different indices and by non-indexed LV mass, is summarized in Table 5. With $ppLVmass_H$ (height

adjustment only), 24% of MESA participants had LV hypertrophy, which was similar to the results using echocardiographic indices (23%). With $ppLVmass_{HW}$ (height and weight adjustment), 8% of MESA participants had LV hypertrophy. Of previously described indices, LV mass/BSA gave results that were most similar to $ppLVmass_{HW}$, with 11% of participants with LV hypertrophy.

All indices demonstrated a higher percentage of MESA participants with LV hypertrophy if hypertension was present. For example, with $ppLVmass_{HW}$, the prevalence of LV hypertrophy was 4 and 13% in normotensive and hypertensive MESA participants. This association was maintained for normal weight participants (5 vs. 15% for normotensive and hypertensive normal weight participants, respectively), as well as overweight participants (3 vs. 13% for normotensive and hypertensive overweight participants, respectively).

In normal weight participants (BMI < 25), 8% had LV hypertrophy by the various indices (6% with non-indexed LV mass). In overweight participants (BMI ≥ 25), the indices that adjust for height and weight ($ppLVmass_{HW}$ and LV mass/BSA) resulted in lower prevalence estimates compared to indices that adjusted for height only ($ppLVmass_{H}$ and the echocardiographic indices). For example, 8–13%of overweight participants had LV hypertrophy when adjusting for height and weight, whereas 30–31% had LV hypertrophy when adjusting for height only. The indices that adjust for height and weight also resulted in lower prevalence estimates in overweight participants after stratification by hypertension status. For example, 13–20% of overweight participants with hypertension had LV hypertrophy when adjusting for height and weight, whereas 41–42% of these participants had LV hypertrophy when adjusting for height only. The difference between the prevalence estimates from indices that adjust for height and weight and from indices that adjust for height only increased with increasing overweight category (results not shown). For example, 19–21%, 44–45%, and 71–73% of participants with 25 ≤ BMI < 30 (N = 2,034), 30 ≤ BMI < 40 (N = 1,331), and BMI ≥ 40 (N = 93), respectively, had LV hypertrophy when adjusting for height only whereas 7–10%, 8–15%, and 8–23% of these same participants had LV hypertrophy when adjusting for height and weight.

Risk of cardiovascular disease event (nonfatal and fatal CHD and stroke)

107 events were observed. The estimated hazard ratios for participants with LV hypertrophy, defined by the different indices and by non-indexed LV mass, relative to participants without LV hypertrophy and of the same age and gender are shown in Table 6. With LV hypertrophy defined by indices that adjust for height and weight ($ppLVmass_{HW}$ and LV mass/BSA), the adjusted hazard ratios were 2.4 and 2.2. The risk of a cardiovascular disease event was 2.4 times greater for a participant with LV hypertrophy defined by $ppLVmass_{HW}$ compared to a participant without LV hypertrophy and of the same age and gender. The adjusted hazard ratios were lower with LV hypertrophy defined by indices that adjust for height only (range of hazard ratios: 2.0–2.1) and by non-indexed LV mass (hazard ratio = 1.7). Higher risk of a cardiovascular event was statistically significantly associated with presence of LV hypertrophy, defined by any of the indices of LV mass or by non-indexed LV mass, as the 95% confidence intervals for each of the hazard ratios in Table 6 excluded one. A statistically significant difference was detected between the hazard ratios based on the new allometric height-weight index and non-indexed LV mass but not among the other hazard ratios (bootstrap confidence intervals for the differences are not shown).

Discussion

Left ventricular hypertrophy is classically considered to be a response to hypertension [29] or valvular dysfunction [30–32] although obesity, diabetes, myocardial infarction and other conditions may also result in increased mass of the left ventricle [33–37] LV mass can be

reduced by appropriate medical therapy; reduction of LV mass as a result of therapeutic intervention reduces cardiovascular events [38–41] indicating LV mass is an important subclinical marker of cardiovascular disease [42]. Because of these therapeutic implications, an appropriate definition of LV hypertrophy becomes quite critical.

LV mass index and obesity

In this study, the prevalence of LV hypertrophy was similar across the various indices in normal weight participants, however in overweight participants, the prevalence was lower with indices that adjust for height and weight compared to indices that adjust for height only. Echocardiographic studies [14,43] have also found that in an obese sample, indices that adjust for height and weight resulted in lower estimates for the prevalence of LV hypertrophy compared to indices that adjust for height only.

Risk of cardiovascular disease event

Despite the differences in prevalence of LV hypertrophy defined by the various indices, in overweight participants and in the MESA cohort, statistically significant differences in cardiovascular disease risk associated with LV hypertrophy defined by the various indices were not detected except when LV hypertrophy was defined by the new allometric height-weight index and by non-indexed LV mass. The hazard ratios tended to be higher with the indices that adjust for height and weight than with indices that adjust for height only. Similar increased risks (hazard ratios) have been reported in the Strong Heart Study [43], a study of subjects with a relatively high prevalence of obesity, and in the MAVI [44] study, a study of hypertensive subjects with a low prevalence of obesity. Liao et al. [28] reported that LV hypertrophy defined by different indices similarly conferred increased risk of mortality in patients with or without coronary artery disease.

Limits for LV hypertrophy from CMR determined LV mass

The 95th percentiles of indexed LV mass in the reference sample (Table 4) were lower than the corresponding percentiles from echocardiographic studies [13–17]. This is not surprising since volumetric CMR derived LV mass is thought to be smaller than M-mode echocardiographically derived LV mass [27]. The percentiles in Table 4 are similar to those reported from the CMR study of 142 healthy subjects by Salton et al. [27] (95th percentile of LV mass equal to 201.4 for men and 134.0 for women; 95th percentile of LV mass/BSA equal to 95.0 for men and 74.7 for women). The mean LV mass for men and for women based on our reference sample (Table 1) were not statistically significantly different (at the 0.05 level) from those in CMR studies of healthy subjects by Marcus et al. [45], and Sandstede et al. [46]. The means were statistically significantly lower but possibly not clinically significantly different from those in CMR studies of healthy subjects by Alfakih et al. [47] (mean ± standard deviation based on fast gradient echo pulse sequence: 166.9 ± 23.4 from 30 men; 110.9 ± 10.3 from 30 women) and Lorenz et al. [48] (mean ± SD: 178 ± 31 from 47 men; 125 ± 26 from 28 women). The limits for LV hypertrophy provided by this study are useful because they were derived from a relatively large, multi-ethnic sample and because previously reported cutoffs from echocardiographic studies appear too high when LV mass is determined by CMR.

Limitations

The study sample was large but included only 5,004 of the total 6,814 MESA participants (and 822 participants in the reference sample). A relatively small number of cardiovascular disease events (107) have been observed in the sample during a median follow-up of 4.1 years and thus the confidence intervals for the hazard ratios are relatively wide.

Conclusion

When LV mass is measured by CMR, the prevalence of hypertrophy is higher for indices without weight. The predictive value of hypertrophy is significantly better with the new allometric height-weight index than with non-indexed LV mass and may be better than indices without weight. Further evaluation of the indices on a continuous scale, and with longer follow-up data from the MESA cohort or CMR data from another large sample would be useful.

Acknowledgments

The authors thank the other investigators, the staff, and the participants of the MESA study for their valuable contributions. A full list of participating MESA investigators and institutions can be found at http://www.mesa-nhlbi.org.

Funding sources This research was supported by contracts N01-HC-95159 and N01-HC-95168 from the National Heart, Lung, and Blood Institute.

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Table 1

Characteristics of the reference sample (Reference sample defined as normotensive participants with BMI less than 25 kg/m^2 with no diabetes or impaired fasting glucose) (N = 822) and the entire MESA cohort with CMR data (N = 5,004)

	Reference sample N = 822				**All participants N = 5,004**			
	Men N = 320		**Women N = 502**		**Men N = 2,382**		**Women N = 2,622[a]**	
Race	%		%		%		%	
White	47		55		39		39	
Chinese	22		22		13		13	
Black	18		12		25		27	
Hispanic	13		11		23		21	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Age (years)	59.5	9.9	58.1	9.8	61.6	10.1	61.5	10.1
Height (m)	1.74	0.08	1.61	0.07	1.73	0.08	1.60	0.07
Weight (kg)	69.1	8.6	57.7	7.0	82.7	14.7	71.9	15.7
BSA (m^2)	1.83	0.15	1.60	0.12	1.97	0.19	1.74	0.19
BMI (kg/m^2)	22.8	1.7	22.2	1.9	27.4	4.1	28.0	5.6
SBP (mmHg)	112.0	12.1	108.5	14.2	125.2	19.3	125.7	23.0
DBP (mmHg)	70.0	7.5	64.4	8.9	74.9	9.4	69.0	10.2
LV mass (g)	148.6	30.7	106.0	19.3	168.9	37.2	123.8	27.4

BSA body surface area, *BMI* body mass index, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *SD* standard deviation

[a] N = 2,622 for measurements from all women participants except for SBP and DBP where N = 2,621

Table 2

Summary of LV mass indices derived from CMR in the reference sample (characteristics of the reference sample are shown in Table 1)

	ppLVmass$_H$[a]	**ppLVmass$_{HW}$**[b]
LV Mass index		
Women	LV mass/(42.5 × Ht$^{1.88}$)	LV mass/(6.82 × Ht$^{0.561}$Wt$^{0.608}$)
Men	LV mass/(51.4 × Ht$^{1.88}$)	LV mass/(8.17 × Ht$^{0.561}$Wt$^{0.608}$)
Intercept		
Women	3.75 (95% CI: 3.62, 3.88)	1.92 (95% CI: 1.50, 2.35)
Men	3.94 (95% CI: 3.79, 4.09)	2.10 (95% CI: 1.67, 2.54)
“Height exponent”	1.88 (95% CI: 1.61, 2.15)	0.561 (95% CI: 0.172, 0.952)
“Weight exponent”	–	0.608 (95% CI: 0.473, 0.742)

LV mass in grams, *Ht* height in meters, *Wt* weight in kilograms, *CI* confidence interval

[a] The index, ppLVmass$_H$, multiplied by 100 is equivalent to the percentage of the value predicted on the basis of height and gender. A ppLVmass$_H$ value of 1 suggests that LV mass is equal to that predicted based on height and gender. A value greater than one suggests LV mass is larger than that predicted, while a value less than one suggests LV mass is smaller than that predicted

[b] The index, ppLVmass$_{HW}$, multiplied by 100 is equivalent to the percentage of the value predicted on the basis of height, weight, and gender

Table 3

Pearson correlation coefficient of LV mass index with height and weight, in the reference sample (Characteristics of the reference sample are shown in Table 1)

Estimate	Height		Weight	
	Women	Men	Women	Men
$ppLVmass_H$	−0.02	0.01	0.17^b	0.19^b
$ppLVmass_{HW}$	−0.01	0.01	−0.04	0.04
LV mass/BSA	0.05	0.08	0.07	0.14^c
LV mass/Ht^2	−0.05	−0.02	0.15^b	0.17^c
LV mass/$Ht^{2.7}$	-0.21^a	-0.19^b	0.03	0.03
LV mass (g)	0.41^a	0.44^a	0.47^b	0.51^a

LV mass in grams, *BSA* body surface area in m^2, *Ht* height in meters

[a] *P*-value < 0.0001,

[b] *P*-value < 0.001,

[c] *P*-value < 0.05

Table 4

95th upper percentiles of LV mass index in the reference sample (Characteristics of the reference sample are shown in Table 1)

	Men and women (*N* = 822)	
$ppLVmass_H$[a]	1.33 (1.29, 1.37)	
$ppLVmass_{HW}$[b]	1.31 (1.28, 1.36)	
	Men (*N* = 320)	Women (*N* = 502)
LV mass/BSA	106.2 (103.8, 110.9)	84.6 (82.2, 88.3)
LV mass/Ht^2	65.7 (62.3, 68.0)	53.0 (51.1, 55.3)
LV mass/$Ht^{2.7}$	45.1 (42.7, 47.0)	38.0 (36.5, 39.5)
LV mass	203.5 (194.4, 216.6)	140.3 (136.0, 146.3)

LV mass in grams, *BSA* body surface area in m^2, *Ht* height in meters

95% confidence interval for the 95th percentile is given in parentheses

[a] The 95th percentile of the $ppLVmass_H$ values equal to 1.33 means that 95% of the reference sample had LV mass values less than 133% of that predicted on the basis of height and gender

[b] The 95th percentile of the $ppLVmass_{HW}$ values equal to 1.31 means that 95% of the reference sample had LV mass values less than 131% of that predicted on the basis of height, weight, and gender

Table 5

Percentage of MESA participants with LV hypertrophy

	Total	**Normal weight**	**Overweight**
		BMI < 25	**BMI ≥ 25**
All participants	***N* = 5,004**	***N* = 1,546**	***N* = 3,458**
$ppLVmass_{H}$	24	8	31
$ppLVmass_{HW}$	8	8	8
LV mass/BSA	11	8	13
LV mass/Ht^2	23	8	30
LV mass/$Ht^{2.7}$	23	8	30
LV mass	20	6	26
Participants without hypertension	*N* = 2,884	*N* = 1,068	*N* = 1,816
$ppLVmass_{H}$	16	5	22
$ppLVmass_{HW}$	4	5	3
LV mass/BSA	6	5	6
LV mass/Ht^2	14	4	20
LV mass/$Ht^{2.7}$	14	5	20
LV mass	13	4	18
Participants with hypertension	*N* = 2,120	*N* = 478	*N* = 1,642
$ppLVmass_{H}$	36	14	42
$ppLVmass_{HW}$	13	15	13
LV mass/BSA	19	16	20
LV mass/Ht^2	35	15	41
LV mass/$Ht^{2.7}$	35	15	41
LV mass	29	11	34

Table 6

Risk of cardiovascular disease event associated with LV hypertrophy

	Adjusted* hazard ratio (95% Confidence interval)
$ppLVmass_{H}$	2.1 (1.4, 3.1)
$ppLVmass_{HW}$	2.4 (1.5, 4.0)
LV mass/BSA	2.2 (1.4, 3.4)
LV mass/Ht^2	2.0 (1.4, 3.0)
LV mass/$Ht^{2.7}$	2.1 (1.4, 3.2)
LV mass	1.7 (1.1, 2.7)

* Adjusted for age and gender