Which Arterial and Cardiac Parameters Best Predict Left Ventricular Mass?

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Background—Many cardiovascular and noncardiovascular parameters are thought to be determinants of left ventricular mass (LVM). Complicated interactions necessitate the simultaneous measurement and consideration of each to determine their individual and collective impact on LVM. We undertook such a comprehensive study.

Methods and Results—The influence of anthropometry, cardiac size and contractility, arterial structure and function, as well as indices of lifestyle, physical activity, and dietary salt intake on LVM (by two-dimensionally guided M-mode echocardiography) was analyzed in 1315 Chinese subjects who were either normotensive or had untreated hypertension. Effects of many cardiac and arterial factors were assessed. In univariate analysis, almost all measured noncardiovascular, cardiac, and arterial variables were significantly correlated with LVM. In multivariate linear regression analyses, when age, sex, body habitus, fasting serum C-peptide level, dietary salt, physical activity, and lifestyle were accounted for, the optimum multivariate linear regression main effects model had an adjusted model $r^2$ of 0.740, with 98% of the model variance accounted for by the 5 independent determinants of LVM: stroke volume (49.6%), systolic blood pressure (30.7%), contractility (14.7%), body mass index (1.8%), and aortic root diameter (1.6%). Other proposed arterial indices were significant independent determinants of LVM only when blood pressure was removed from the model and, even then, these indices not only resulted in less powerful prediction but also accounted for only a very small percentage of the total variance of LVM.

Conclusions—In a large population, we (1) confirmed that age, body habitus, and some indexes of arterial structure and function are independent determinants of LVM; (2) found aortic diameter to be an independent structural determinant of LVM; (3) demonstrated that the effects of the derived measures of arterial function were small and provided no better predictive power than blood pressure alone; and (4) showed that when the best measures of cardiac and vascular load were included, the single most potent predictor was an index of left ventricular size. (Circulation. 1998;98:422-428.)

Key Words: hypertrophy ■ cardiac load ■ arterial system ■ vascular load ■ blood pressure

Left ventricular mass is determined by integrated signaling of multiple stimuli, among which are the mechanical loads imposed on the heart. These loads are composed of both cardiac and arterial components. The independent roles of cardiac internal workload and contractility have been well documented.$^{1,2}$ Because the heart and arterial system are physically coupled, some of the workload of the heart is also determined by the arterial system. Elevated BP, being the easiest index of arterial load to measure, is a well-recognized cause of increased LVM. BP is determined by cardiac factors such as heart rate, contractility, and stroke volume on the one hand and arterial size, wall properties, peripheral wave reflections, and peripheral resistance on the other. Technological advances allowing noninvasive measurement of several arterial factors$^{3-9}$ have spawned many studies associating LVM with one or more factors.$^{2,10-14}$ Because of complicated interrelations among the determinants of BP, however, without a comprehensive study, it is difficult to discern the independent predictors of LVM.

This study comprehensively examines the independent and interactive impact of cardiac factors as well as arterial structure and function on LVM while accounting for well-known noncardiovascular modulators of LVM such as age, sex, body size and habitus, salt intake, physical activity, and lifestyle$^{2,7,15-27}$ in a large number of racially homogeneous individuals. We examined noninvasive indexes of arterial structure and function such as aortic and carotid artery diameters, carotid artery wall thickness, blood pressure, arterial pulse wave velocity, carotid artery elastic modulus, arterial compliance, peripheral resistance and carotid aug-
ment index as well as cardiac functional indexes of size, contractility, heart rate, stroke volume, and stroke work to determine which of these factors were independent determinants of LVM.

Methods

Study Population
The study population consisted of 1315 subjects (698 men and 617 women) in Taiwan nearly equally distributed in the 3rd to 7th age decades and older. None of the subjects were receiving antihypertensive therapy, none had a history of angina pectoris, peripheral vascular disease, or diabetes, and none had significant heart disease disclosed by echocardiography. Each participant underwent a 2-hour cardiovascular study including a complete medical history, anthropometric measurements, physical examination, echocardiography, and Doppler flow examination.

LV Parameters
LV dimensions were obtained in all subjects by the same experienced sonographer using a Hewlett-Packard Sonos 500 U (Hewlett-Packard) with a 2.5 MHz transducer in accordance with the published recommendations. LV wall thickness was the average of interventricular septal and posterior wall thickness. LVM was calculated from the 2-D guided M-mode echocardiogram as well as with a 2-D echocardiographic formula obtained by 2D echocardiographic measurements rather than either geometric assumptions. For example, in calculating ESSV, the LV volume used was that obtained noninvasively with a tonometer. Peripheral vascular resistance was calculated as the ratio of MBP to cardiac output.

Noncardiovascular Modulators of LVM
In addition to age and sex, the effects of most other reported modulators of LVM were accounted for. Lifestyle (urban or rural domicile) and physical activity level (high for farmers or laborers and low for retirees or white collar workers), were treated as categorical variables. BMI and BSA were calculated from measurements of weight and height (BMI [kg/m²] = weight[kg]/height[m²]; BSA [m²] = 0.0001 × 71.84 × weight[kg]/height[m²]), and WHR was measured.

Statistical Analysis
Results are expressed as mean ± SD. Carotid E was logarithmically transformed because of a skewed distribution. Unpaired Student t tests were used to compare parameter means between men and women. To delineate the independent determinants of LVM, multiple regression models were created. Univariate regression was first performed between LVM and each categorical and continuous parameter. Only those variables having a significant correlation with LVM were included in the subsequent multiple regression models. We entered pairwise (omitting linear combinations) SBP and DBP, SBP and PP, and MBP and PP and found little difference among the pairs so we used SBP and PP. In the final reduced model, variables with nonsignificant regression coefficients (P > 0.1) in the initial models were first removed. Then variables that exhibited a high degree of collinearity were added or deleted iteratively. This was to achieve a model with consistency, conciseness, the highest adjusted total variance and the least multicollinearity (assessed with the variance inflation factor). Thus models in which the sign of a variable was opposite that in the univariate analysis were rejected. In some cases we could retain only one of several closely related variables. For example, Disten and RDist could not both be retained because a model with both rendered one of them insignificant. Likewise, including LVEDV rendered SV insignificant. Whenever possible, multicollinearity effects were further minimized by using variables not derived from another variable in the model. For example, in calculating ESSV, the LV volume used was that obtained by 2D echocardiographic measurements rather than either of the volumes derived from the M-mode parameters.

Once the final main effects model was obtained, the relative importance of each independent variable was determined by performing forward stepwise multiple regression analysis by calculating the ratios of individual partial r² to the full model r². Expansion of the optimal main effects model to include each possible pairwise interaction was also evaluated. Finally, because many of the derived vascular functional parameters as well as contractility contained one of the blood pressure parameters and/or LV chamber volume,

Vascular Structural and Functional Parameters
Sitting brachial artery SBP and DBP were measured with conventional sphygmomanometry. Aortic root diameter was determined by M-mode echocardiography. We also measured on-line from frozen, digitized images (with a 7-MHz vascular probe incorporated in the echocardiographic unit) the right common carotid artery systolic and diastolic diameters (Ds and Dd) and the intimal-medial thickness of the posterior wall.

Wall properties of the aorta and carotid arteries were indexed by the trunk PWV, E, and carotid distensibility. PWV was calculated from sequential nondirectional Doppler (Parks model 802) flow velocity and a simultaneous ECG at the right carotid artery and femoral artery. The elastic modulus was calculated as E=4πb²Dp/[(b²−a²)DΔ], where a and b are the internal and external radii, and ΔP and ΔD are the pressure and diameter changes over the cardiac cycle. Carotid artery distensibility (Disten) and relative distensibility (RDist) were also calculated as ΔD/ΔP and ΔD/ΔD/ΔP, respectively. Additionally, AC, (the ratio of SV to PP), arterial elastance (Ea, ratio of an index of ESP to SV) were measured with ESP defined as (2SBP+DBP)/3, AGI was calculated from the right common carotid arterial pressure wave contour obtained noninvasively with a tonometer. Peripheral vascular resistance was calculated as the ratio of MBP to cardiac output.

Selected Abbreviations and Acronyms

AC = aortic compliance
AGI = carotid augmentation index
BMI = body mass index
BP = blood pressure
BSA = body surface area
DBP = diastolic BP
E = carotid elastic modulus
Ea = arterial elastance
ESP = end-systolic pressure
ESSV = end-systolic meridional stress to volume ratio
LV = left ventricular
LVEDV = LV end-diastolic volume
LVM = LV mass
MBP = mean blood pressure
PP = pulse pressure
PWV = pulse wave velocity
SBP = systolic BP
SV = stroke volume
SW = stroke work
TSVR = total systemic vascular resistance
WHR = waist to hip ratio
2-D = two-dimensional

\[
\text{AC} = \frac{(\text{end-systolic pressure} \times \text{LV end-systolic dimension})}{(\text{LV wall thickness} \times \text{LV end-systolic volume})}
\]
TABLE 1. Characteristics of the Study Population

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Men (n=698)</th>
<th>Women (n=617)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Anthropometric parameters</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>24.3±3.2</td>
<td>25.2±4.0</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>BSA, m²</td>
<td>1.72±0.15</td>
<td>1.56±0.13</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Height, cm</td>
<td>165±7</td>
<td>153±6</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>6.1±10.6</td>
<td>59.2±9.9</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>WHR</td>
<td>0.89±0.06</td>
<td>0.84±0.06</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td><strong>Cardiac parameters</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ESSV, mm Hg/mL</td>
<td>8.3±2.6</td>
<td>10.0±3.2</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Heart rate, bpm</td>
<td>73±10</td>
<td>74±10</td>
<td>NS</td>
</tr>
<tr>
<td>LVEDV, mL</td>
<td>120±26</td>
<td>107±25</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LV load, mm Hg/cm²</td>
<td>27130±6969</td>
<td>24187±6958</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LVM, g</td>
<td>173±42</td>
<td>153±45</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>SV, mL</td>
<td>54±15</td>
<td>48±14</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>SW, mL · mm Hg</td>
<td>5517±1840</td>
<td>4914±1894</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td><strong>Vascular parameters</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AC, mL/mm Hg</td>
<td>1.87±0.64</td>
<td>1.58±0.57</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>AGI, %</td>
<td>7.5±14.4</td>
<td>19.7±13.9</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Aortic diameter, cm</td>
<td>3.2±0.4</td>
<td>2.9±0.3</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Carotid diameter, mm</td>
<td>6.59±0.78</td>
<td>6.32±0.84</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Carotid IMT, mm</td>
<td>1.05±0.27</td>
<td>1.01±0.25</td>
<td>0.004</td>
</tr>
<tr>
<td>Distensibility, mm/mm Hg</td>
<td>0.007±0.004</td>
<td>0.007±0.004</td>
<td>NS</td>
</tr>
<tr>
<td>Ea, mm Hg/mL</td>
<td>2.40±1.0</td>
<td>2.82±1.2</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>lnE, 10⁶ dyne/cm²</td>
<td>8.3±0.7</td>
<td>8.3±0.7</td>
<td>NS</td>
</tr>
<tr>
<td>DBP, mm Hg</td>
<td>85±14</td>
<td>84±14</td>
<td>NS</td>
</tr>
<tr>
<td>ESP, mm Hg</td>
<td>118±18</td>
<td>120±22</td>
<td>NS</td>
</tr>
<tr>
<td>MBP, mm Hg</td>
<td>101±15</td>
<td>102±17</td>
<td>NS</td>
</tr>
<tr>
<td>PP, mm Hg</td>
<td>50±15</td>
<td>54±18</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>SBP, mm Hg</td>
<td>135±22</td>
<td>138±27</td>
<td>0.013</td>
</tr>
<tr>
<td>PWV, cm/s</td>
<td>950±230</td>
<td>948±252</td>
<td>NS</td>
</tr>
<tr>
<td>TSVR, mm Hg · min/mL</td>
<td>0.029±0.01</td>
<td>0.030±0.01</td>
<td>0.07</td>
</tr>
<tr>
<td>Wall thickness, cm</td>
<td>0.96±0.14</td>
<td>0.93±0.16</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td><strong>Noncardiovascular parameters</strong></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Age, y</td>
<td>52.4±12.9</td>
<td>52.1±12.7</td>
<td>NS</td>
</tr>
<tr>
<td>C-peptide, nmol/L</td>
<td>0.51±0.29</td>
<td>0.51±0.26</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Lifestyle</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Urban</td>
<td>194</td>
<td>193</td>
<td>NS</td>
</tr>
<tr>
<td>Rural</td>
<td>504</td>
<td>424</td>
<td></td>
</tr>
<tr>
<td>Physical activity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>High</td>
<td>321</td>
<td>193</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Low</td>
<td>377</td>
<td>434</td>
<td></td>
</tr>
<tr>
<td>Urine Na content, mmol/L</td>
<td>54±29</td>
<td>47±31</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

IMT indicates intimal-medial thickness; lnE, natural logarithm of E.

Additional regression analyses were performed with either the BP variables, LVEDV, or ESSV removed and the derived parameters substituted in their place. Significance in any model was accepted at the \(P<0.05\) level.

**Results**

The mean values of most parameters differed by sex (Table 1). Table 2 summarizes the univariate results. Every measured variable, with the exception of activity level, hematocrit, and TSVR, was significantly correlated with LVM, with the 4 strongest positive correlations being with LVEDV, LV load, SW, and SW and the strongest negative correlation with ESSV. Table 3 lists correlation coefficients among the anthropometric, cardiac, and vascular parameters.

Table 4 depicts four different main effects multiple regression models. LV load could not be retained because it was colinear with another variable. The first column lists the results of the optimal model with 9 main independent predictors of LVM (adjusted \(\hat{r}^2=0.740\)). Although many variables differed by sex, sex was itself not an independent predictor of LVM. Likewise, fasting serum C-peptide, urine sodium content, physical activity, and lifestyle were not independent predictors. Except for ESSV, all other variables...
had positive partial regression coefficients. The relative importance of each of the independent determinants of LVM is shown in Figure 1. By far, the most important predictor of LVM is SV. Together with SBP, aortic diameter, ESSV, and BMI, these 5 variables accounted for 98% of the model variance. The only interaction that added significantly to the overall variance was SV
\(^3\)ESSV. Adding this term, however, only increased the adjusted variance by 3% (from 0.740 to 0.764). Figure 2 is a 3-dimensional plot illustrating the interacting effects of these two determinants of LVM.

Substituting the other BP variables for SBP lowered the adjusted variance, so SBP was used in all models. Although most of the derived vascular variables were significantly correlated with LVM in the univariate analyses, none were significant independent predictors of LVM.

We repeated the multiple regression procedure to see whether determinants of LV wall thickness (a direct rather than a derived measure of LV hypertrophy) or relative wall thickness (wall thickness/end-diastolic diameter) were similar to those for LVM. The optimal models each contained the same 9 predictors as for LVM from the M-mode and the same top 5 predictors (accounting for 93% of the total model variance), that is, SBP (37%), ESSV (33%), SV (12%), BMI (6%), and aortic diameter (5%). The adjusted model variances for wall thickness and relative wall thickness were,
LVEDV and ESSV. Although this study did not examine the determinants of LVM in an occidental population of patients on hemodialysis found M-mode– derived LVM to depend on determinants of LVM in this population. A recent echocardiographic study of the determinants of LVM accounting for 98% of the total variance (in order of partial $r^2$/model $r^2$): SV (63%), BMI (13%), ESSV (9%), aortic diameter (7%), and SBP (6%). This result, combined with the LV wall thickness results presented above, provides confidence that our results present a realistic picture of the determinants of LV mass.

Discussion

Because the M-mode formula for LVM includes parameters, for example, LV diameters, used to calculate other parameters, there may be some concern about independence. To verify the reliability of the predictors of LVM only if SV, ESSV, or SBP were deleted. For example, when SV was omitted, LVEDV become a predictor, but with lower overall model variance. Omitting ESSV did not allow PP, AC, or Ea to become significant predictors when SV was in the model. AGI, PWV, or E became significant predictors only when SBP was deleted. However, with these three parameters, not only was the adjusted model $r^2$ decreased to 0.658 and 0.661 for the SV and LVEDV models, respectively, but PWV, AGI, and E together accounted for <4% of the total model variance.

As shown in the remaining columns in Table 4, some of the other parameters became significant predictors of LVM only if SV, ESSV, or SBP were deleted. For example, when SV was omitted, LVEDV become a predictor, but with lower overall model variance. Omitting ESSV did not allow PP, AC, or Ea to become significant predictors when SV was in the model. AGI, PWV, or E became significant predictors only when SBP was deleted. However, with these three parameters, not only was the adjusted model $r^2$ decreased to 0.658 and 0.661 for the SV and LVEDV models, respectively, but PWV, AGI, and E together accounted for <4% of the total model variance.

As for noncardiovascular modulators of LVM, our results confirmed the independent relation between body mass and LVM previously reported. In all our models BMI was consistently among the 5 strongest predictors. There was also a small independent effect of BSA as reported by others. The BSA effect was only about one-fifth that of BMI. Previous studies showing a relation between BSA and LVM, however, did not account for the effects of either SV or LVEDV. We also found an independent age-related effect on LVM as previously reported. However, the age effect was very minor, accounting for only ~0.5% of the model variance. Apparently, most of the age-related effects are accounted for by the other parameters in the model. Despite the significant sex differences in many parameters, sex itself was not an independent predictor of LVM, which suggests that essentially all of the previously reported sex influences on LVM can be accounted for by body size, weight, and so on.

An increase in the vascular load—because it affects the internal and external LV loads—must cause the LV to remodel. Despite many studies demonstrating a relation between one or more vascular parameters and LVM, it is still not clear which aspect(s) of cardiac load many of these indices affect. These parameters can be categorized as affecting BP, arterial size, arterial wall property, and integrated load. SBP is an integrated but nonspecific external load on the left ventricle during ejection because it is affected by wall stiffness, wave reflections, and transmission speed and is itself affected by LV ejection. Despite its nonspecificity, its integrative power conveys sufficient information about the arterial system such that none of the more specific vascular parameters, the determinants of LVM they found were two of the strongest predictors we observed. The surprising lack of dependence on BP in that population was attributed to the high proportion of patients with myocardial dysfunction. Additionally, we found another 199 separate individuals (90 men and 109 women) in the same age range as our study population (mean age 59 years) with mean SBP and DBP of 152 and 91 mm Hg, respectively, who were being treated for hypertension. Because this population was not homogeneous in terms of duration of hypertension or duration and type of treatment, we did not include this group in our main study. Nevertheless, they were examined in the same manner. With LVM assessed by the M-mode formula, the overall model variance was 0.55 and the top predictors were SV (45%), ESSV (31%), SBP (10%), and BMI (7%). Aortic diameter was not a significant predictor. With the use of the 2-D formula for LVM, the overall model variance was 0.52 and the top predictors were SV (69%), SBP (19%), and ESSV (6%), with BMI and aortic diameter not significant predictors. Thus even in these two widely differing populations, the most important independent predictors of LV mass were essentially the same as in our main population. These results suggest that our findings may be generalizable.
itself is a weaker determinant than SBP. This is not too surprising because DBP does not directly affect ejection. MBP is, likewise, only a steady load component and is not as strong a predictor as SBP. One might have thought that PP would be as strong a predictor as SBP because it manifests the pulsatile load. This, however, was not the case. PP appeared to index contractile function to some extent because it could replace ESSV in the model (albeit with lower power and still required SBP). By itself, PP led to a slightly weaker model than SBP. Thus SBP is the strongest single BP determinant of LVM, although the other pressure terms carry much of the same information.

One surprising and novel finding is the importance of the aortic diameter. In all the models we found aortic root diameter to be among the 5 strongest predictors and as potent as BMI. This loading probably arises from the inertia of the blood in the ascending aorta on ejection. Previous studies addressing this issue by measuring blood acceleration have been inconclusive. Recent modeling studies of the arterial system have, however, shown that including an inertance term more completely characterizes arterial function than models without inertance. The present study is the first to show an independent effect of an inertial term on LV remodeling. Our data confirm that carotid artery diameter is also an independent albeit fairly weak determinant of LVM, although why this is so is not clear. It is possible that even the smaller mass of blood in these very proximal vessels imposes sufficient load to induce LVM—although further study is needed to verify this.

Carotid wall thickness, elastic modulus, and PWV (which is directly related to wall stiffness) all affect the regression models differently, confirming that they do not all convey the same information about arterial wall properties. The two most direct measures of stiffness, E and PWV, are not important predictors, except in the absence of the BP terms. This indicates that wall stiffness effects can essentially all be accounted for by BP. This may reflect the direct relation between aortic pressure and wall stiffness. Carotid wall thickness is an independent but relatively minor predictor of LVM. Even though a thicker wall is likely to be stiffer, the fact that E is not a predictor suggests that the wall thickness effect is not attributable to stiffness. Rather, a thicker carotid wall may be a manifestation of a more generalized alteration in the arterial system that loads the left ventricle. Regardless, the combined effects of aortic and carotid artery diameter and wall thickness may explain, in part, the higher fraction of LV variability accounted for by our study than a comparable recent study. The lack of effect of urine sodium suggests that the effects of salt intake arise through SBP or other parameters.

The integrated indices of arterial function (TSVR, AGI, AC, and Ea) were not independent predictors of LVM in the presence of SBP and ESSV. TSVR, despite comprising >90% of the impedance, was not a predictor of LVM in either the univariate or multivariate analyses, thereby indicating that it has little influence on cardiac remodeling. This is concordant with studies showing resistance is not a good index of hemodynamic loading. AGI is a useful, noninvasive index of the timing and amplitude of peripheral reflections and hence external load on the heart. Our results show, however, that it provides no additional predictive power over that of blood pressure for LVM.

AC enters into the models differently than either PWV or E, which indicates that wall stiffness and compliance do not have a direct relation. This is not surprising because AC embodies arterial size as well as stiffness. Because AC and Ea were predictors only if ESSV was eliminated, this suggests that they exert their influence on LVM by contractility rather than vascular loading. For AC this may be due to the dominant effect of SV. The relation between Ea and contractility is not surprising because Ea has been shown to be closely related to LV systolic function with aging, coronary artery disease, and heart failure. Both AC and Ea, however, entered the multiple regression models with signs we considered inappropriate, which suggests that they were collinear with other parameters in the model. In fact, the coefficient for Ea in either the univariate or the multiple regression models had a negative sign. Why this is so is unclear but may be due to the effect of Ea being dominated by the SV term in the denominator. The fact that it enters into the regression models differently than the wall stiffness indexes, and only if SV is omitted, further confirms that it is a different manifestation of arterial loading than wall stiffness.

The importance of SV (or LVEDV, with which SV is highly correlated) and ESSV in determining LVM confirms previous findings. Their dominance in the presence of the many other potential predictors, especially the arterial load parameters, is impressive. The relation between LV cavity size and LVM is understandable and expected. Myocyte stretch is a powerful stimulus leading to a variety of responses including changes in tonic homeostasis, activation of intracellular messenger systems, increases in various proteins and genes, and elaboration of growth factors. All of these, acting singly or in combination, can lead to cardiac hypertrophy. The strong, independent role of reduced contractility—even in our population with no clinical evidence of heart failure—suggests that although cell stretch may be a unifying explanation for the effects of increased LV size and decreased contractility, they each elicit different stimuli at the cellular/molecular level.

In summary, this comprehensive study in a large, racially homogeneous population found that the most important independent determinants of LVM were LV size, SBP, LV contractility, aortic size, and BMI. Independent predictors of lesser importance were carotid artery diameter and wall thickness, BSA, age, and heart rate. Many of the popular derived vascular load parameters were examined, and none of them added additional predictive power over and above the more direct parameters listed above. The 9 independent factors identified together thus constitute the most potent description of the impact of mechanical loading factors, probably signaling in concert with molecular growth factors, as determinants of LVM.

Acknowledgments

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