Hemodynamic Function at Rest, During Acute Stress, and in the Field

Predictors of Cardiac Structure and Function 2 Years Later in Youth

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Abstract—Left ventricular hypertrophy is an independent predictor of cardiovascular morbidity and mortality. However, predictors of cardiac structure and function in youth are not completely understood. On 2 occasions (2.3 years apart), we examined 146 youth aged initially 10 to 19 years (mean age, 14.2 ± 1.8 years). On the initial visit, hemodynamic function was assessed at rest, during laboratory stress (ie, orthostasis, car-driving simulation, video game, and forehead cold), and in the field (ie, ambulatory blood pressure). Quantitative M-mode echocardiograms were obtained on both visits. On both visits, black compared with white youth had higher resting laboratory systolic blood pressure ($P<0.02$), greater relative wall thickness ($P<0.003$), greater left ventricular mass indexed by either body surface area or height$^{2.7}$ ($P<0.01$ for both), and lower midwall fractional shortening ratio ($P<0.05$). Hierarchical stepwise regression analysis indicated that significant independent predictors of follow-up left ventricular mass/height$^{2.7}$ were the initial evaluation of left ventricular mass/height$^{2.7}$, body mass index, gender (males more than females), and supine resting total peripheral resistance (final model $R^2=0.53$). Left ventricular mass/body surface area was predicted by initial left ventricular mass/body surface area, weight, gender, mean supine resting total peripheral resistance, and systolic pressure response to car-driving simulation (final model $R^2=0.48$). Midwall fractional shortening was predicted by initial midwall fractional shortening, race (white more than black), and lower mean supine total peripheral resistance (final model $R^2=0.13$). The clinical significance of these findings and their implications for improved prevention of cardiovascular diseases are yet to be determined. (Hypertension. 1999;34:1026-1031.)

Key Words: blood pressure ■ left ventricular mass ■ ventricular function ■ youth

Left ventricular (LV) hypertrophy determined by either electrocardiography or echocardiography is a major risk factor for cardiovascular morbidity and mortality. Longitudinal epidemiological and necropsy studies have demonstrated that the pathogenesis of coronary artery disease and essential hypertension begins in childhood. Related to this, a number of cross-sectional and a few longitudinal studies with normotensive youth have indicated that gender (males more than females), race (blacks more than whites), adiposity, age, and hemodynamic function at rest and during acute laboratory stress are associated with increased LV mass (LVM) adjusted for body habitus (ie, LVM/height$^{2.7}$, LVM/body surface area [BSA]).

Ambulatory blood pressure (ABP) monitoring has been recognized to be superior to resting blood pressure (BP) in the prediction of cardiovascular morbidity. To the best of our knowledge, the value of ABP in predicting future LVM and LV function has not been evaluated in normotensive youth. Therefore, the purpose of this study was to evaluate comprehensively the individual and relative contributions of hemodynamic functioning at rest, during laboratory stress, and in the natural environment (ie, ABP monitoring) in the prediction of cardiac structure and function 2 years later in a sample of normotensive youth in whom the majority (ie, 96%) had a family history of cardiovascular disease. Individuals with a positive family history of essential hypertension and/or premature myocardial infarction are at increased risk for development of cardiovascular disease. Thus, identification of early predictors of cardiac structure and function is particularly important in such youth before manifestation of significant end-organ damage and cardiovascular disease.

Methods

Subjects

Subjects were 146 normotensive youth who participated in 2 laboratory evaluations separated by an average of 2.3 ± 0.5 years. They were initially aged 10 to 19 years (mean age, 14.2 ± 1.8 years). There were 59 males (32 white, 27 black) and 87 females (39 white, 48 black).
48 black). Subjects are participants in longitudinal studies of the development of cardiovascular risk factors in children with verified family histories of cardiovascular diseases (ie, essential hypertension and/or premature myocardial infarction).16,17

Procedure
The study was approved by the institutional review committee. On each visit after informed consent was obtained, with rare exception, all anthropometric and hemodynamic evaluations were conducted by a female research assistant of the same race as the subject. Subject’s height (in centimeters) and weight (in kilograms) were measured without shoes with a Health-O-Meter medical scale, which was calibrated daily. Waist circumferences were measured at the center of the umbilicus, and hip circumference was measured at the level of the greater trochanters. Two sets of readings were recorded and averaged. The subject was then escorted to a quiet temperature-controlled room (20°C to 22°C) and fitted with equipment for recording BP (Dinamap model 1846 SX, Critikon Inc) and cardiac output with the use of thoracic bioimpedance (NCCOM-3, Bo Med Medical Manufacturing Ltd), as previously described.16 Body mass index (BMI) was calculated as weight/height².

Hemodynamic Evaluations
Stroke volume and heart rate were measured and cardiac output was calculated (stroke volume×heart rate; L/min) every successive 12 QRS intervals while the Dinamap device was inflating and calculating pressure. Total peripheral resistance (TPR) was calculated with the use of concurrently derived systolic BP (SBP) and diastolic BP (DBP) and cardiac output as follows: [(SBP + 2×DBP)/3]/Cardiac Output, expressed in Wood units (mm Hg/L per minute). The subject was placed in a supine position and given standardized instructions to relax as completely as possible for 15 minutes. During minutes 11, 13, and 15 of the baseline rest period, hemodynamic measurements were obtained and averaged to provide 1 reading per 15 minute pressure evaluation. After the baseline evaluation, the subject engaged in 4 laboratory stressors (ie, orthostasis, video game, car-driving simulation, and forehead cold) in which standardized protocols were used.16–18 With the exception of the video game, all protocol instructions were conducted live by a female of the same race as the subject. The orthostasis test was administered first with hemodynamics measured at 1, 2, and 3 minutes after the subject assumed a standing position with the right arm relaxed at a 90-degree angle across the trunk. The remaining 3 stressors were presented in a counterbalanced order, with the subject in the supine position. A minimum 5-minute prestressor period preceded each stressor, during which all hemodynamic data were concomitantly obtained every other minute. Prestressor periods continued until the subject’s blood pressure returned to within ±5 mm Hg of the baseline values. The 5-minute video game stressor Break Out (Atari Inc) was presented under a monetary incentive challenge via videotaped instructions by a female of the same race as the subject. The BP cuff was placed on the nondominant arm, and the video game controller was secured at a position comfortable for use with the subject’s dominant hand. The game was presented on a 635-mm-diagonal color television, located 2000 mm from the subject at a position comfortable for use with the subject’s dominant hand. The game was presented on a 635-mm-diagonal color television, located 2000 mm from the subject at a position comfortable for use with the subject’s dominant hand. The game was presented under a monetary incentive challenge via videotaped instructions conducted live by a female of the same race as the subject.

Hemodynamic measures used in the analyses included both peak and change score (ie, peak—mean prestressor) responses. Initially, a series of 2 (race)×2 (gender)×2 (visit: initial, follow-up) repeated-measures ANOVAs were conducted on all demographic, anthropometric, resting, and echocardiographic parameters. A series of 2 (race)×2 (gender) ANOVAs were also conducted on 24-hour, daytime, and nighttime ABP.

Pearson product moment correlations were used to examine univariate relationships between all initial visit demographic, anthropometric, and hemodynamic data and echocardiographic data collected 2.3 years later. Variables found to be significantly related to echocardiographic variables (P<0.05) from this analysis were used

Echocardiographic Studies
Echocardiographic evaluations were performed after completion of the laboratory stressors and a brief rest. Sector-guided M-mode echocardiograms were performed with a Hewlett Packard Sonos 1500 echocardiograph. Left ventricular posterior wall in diastole (LVPWD), interventricular septum in diastole (IVSD), and LV internal diameter in diastole (LVIDD) were measured according to the American Society of Echocardiography convention. LVM was derived with the use of the following equation: LVM = (1.047×LVIDD²+0.413×LVIDS+6.34)/3, which has been validated for use in individuals with normal hearts. On the basis of the recommendation of de Simone et al,²¹ LVM was divided by height¹³ to adjust for normal growth without removing the effect of obesity.

A second index LVM/BSA was used to allow easy comparison with previous reports. Relative wall thickness (RWT) was calculated with the use of the following equation: RWT = (LVIDD/LVPWD)²/LVIDD².

To assess LV contractile function, circumferential end-systolic stress (cESS), midwall fractional shortening (MFS), and MFS ratio were calculated according to established formulas.²² Specifically, we calculated cESS at the midwall level of the left ventricle at an index of afterload using a cylindrical model, as follows:

\[
\text{cESS} = \frac{\text{SBP} \times \left(\frac{\text{LVIDS}}{2}\right)^2 \times \left(1 + \frac{\left(\text{LVIDS/2} + \text{LVPWS/2}\right)^2}{\left(\text{LVIDS/2} + \text{LVPWS}\right)^2}\right)^2}{\left(\text{LVIDS/2} + \text{LVPWS}\right)^2 - (\text{LVIDS/2})^2}
\]

where LVPWS is LV posterior wall in systole.

MFS was calculated following the method described by de Simone et al.²¹ as follows: MFS = (LVIDD + LVPWD/2/IVSD/2) – (LVIDS/2 + Hs/2)/LVIDD + LVPWD/2 + IVSD/2, where LVIDS is LV internal diameter in systole and Hs/2 is the assumed LV inner shell myocardial thickness at end systole, taking into account the epicardial migration of midwall during systole in a spheroidal model. To evaluate midwall LV performance independently of afterload, the ratio between MFS calculated from the echocardiographic measurement and the value predicted for a given level of cESS was calculated (ie, MFS ratio).

ABP Assessment
After completion of the laboratory evaluation, the subject was fitted with an ABP monitor cuff on the nondominant arm (model 90207, Space Labs, Inc). The subject wore the monitor for 24 hours, with measurements obtained every 20 minutes during the day (ie, 6 AM to 10 PM) and every 30 minutes at night (ie, 10 PM to 6 AM). The transitional periods from 6 AM to 8 AM and 10 PM to midnight were not included in the analyses by time period but were included in the 24-hour analyses. Measures of SBP and DBP nocturnal dip were calculated as the differences between average daytime minus nighttime readings, expressed as a percentage of the daytime average. Ambulatory data were edited according to established criteria described by Treiber et al.²²

Data Reduction and Statistical Analyses
All baseline and prestressor hemodynamic measurements were averaged. Hemodynamic stress measures used in the analyses included both peak and change score (ie, peak−mean prestressor) responses. Initially, a series of 2 (race)×2 (gender)×2 (visit: initial, follow-up) repeated-measures ANOVAs were conducted on all demographic, anthropometric, resting, and echocardiographic parameters. A series of 2 (race)×2 (gender) ANOVAs were also conducted on 24-hour, daytime, and nighttime ABP.

Pearson product moment correlations were used to examine univariate relationships between all initial visit demographic, anthropometric, and hemodynamic data and echocardiographic data collected 2.3 years later. Variables found to be significantly related to echocardiographic variables (P<0.05) from this analysis were used
in hierarchical stepwise multiple linear regression analyses using blocks of predictor variables. The blocks of possible predictor variables, in order, were as follows: (1) initial echocardiographic parameters (eg, LVM/BSA, LVM/height<sup>2.7</sup>); (2) demographics/anthropometrics (eg, gender, race, weight, BMI); (3) resting hemodynamics; (4) ABP measures; (5) peak hemodynamic stress responses; (6) hemodynamic change scores to stress; and (7) interactions of gender or race with resting hemodynamics, hemodynamic stress responses, and ABP variables. This form of regression is similar to analyzing a change score in that for all blocks of variables subsequent to the first block, the dependent variable is, in effect, the unexplained difference between the baseline and follow-up measure of interest.

Results

Descriptive Characteristics

Table 1 presents anthropometric data at the initial and follow-up visits. A significant visit by race by gender effect was found for weight and height, with white males increasing more than black males and black and white females increasing less. For BSA, a significant race by gender by visit interaction was due to no increase for white females, increase for black females, and a larger increase for black and white males. For WHR, males were higher than females at both visits. In addition, self-reported aerobic activity indicated that all baseline anthropometric parameters were related to follow-up LVM/height<sup>2.7</sup> (r range, 0.19 to 0.56; P<0.01 for both) and to LVM/BSA (r range, 0.25 to 0.33; P<0.01 for all). With respect to resting hemodynamic measures, both follow-up LVM indices were positively related to baseline SBP and TPR (r range, 0.20 to 0.27; P<0.05) and baseline heart rate (r=-0.29 for both; P<0.01).

With respect to hemodynamic stress responsivity, SBP responsivity to each of 4 stressors was significantly positively related to both indices of LVM 2.3 years later (r range, 0.22 to 0.33; P<0.01 for all). TPR peak and/or change score responsivity to car-driving simulation, video game, and cold stressors was positively related to both indices of LVM (r range, 0.19 to 0.23; P<0.05 for all). Finally, the DBP peak responsivity to postural change was positively related to follow-up LVM/BSA (r=0.18, P<0.05).

Ambulatory SBP measured during daytime, nighttime, or 24 hours was significantly positively related to all echocardiographic measurements except RWT (r range, 0.18 to 0.35; P<0.05 for all). RWT was positively correlated with nighttime ambulatory DBP (r=0.18; P<0.03). When partial correlations were tested, no gender- or race-related differences were found for the relationships between indices of follow-up LVM with hemodynamic stress responsivity and ambulatory BP. Of all nocturnal dip comparisons, only the SBP dip was

### Table 1. Anthropometric Characteristics of the Study Population by Race and Sex

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Sex</th>
<th>Black (n=75)</th>
<th>White (n=71)</th>
<th>P</th>
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<tr>
<td></td>
<td></td>
<td>Visit 1</td>
<td>Visit 2</td>
<td></td>
</tr>
<tr>
<td>Age, y</td>
<td>M</td>
<td>14.3±2.0</td>
<td>13.8±1.7</td>
<td>15.7±1.7</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>14.1±1.7</td>
<td>14.7±2.0</td>
<td>17.0±2.0</td>
</tr>
<tr>
<td>Height, cm</td>
<td>M</td>
<td>163.2±11.0</td>
<td>163.9±11.5</td>
<td>174.3±8.4</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>161.7±6.6</td>
<td>161.4±7.0</td>
<td>164.2±4.6</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>M</td>
<td>60.3±17.8</td>
<td>57.9±16.6</td>
<td>67.8±17.7</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>60.1±11.0</td>
<td>54.8±10.2</td>
<td>58.5±9.8</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>V×S×R&lt;0.03</td>
</tr>
<tr>
<td>WHR</td>
<td>M</td>
<td>0.82±0.06</td>
<td>0.83±0.06</td>
<td>0.81±0.05</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>0.76±0.05</td>
<td>0.75±0.05</td>
<td>0.74±0.04</td>
</tr>
<tr>
<td>BMI, kg/m&lt;sup&gt;2&lt;/sup&gt;</td>
<td>M</td>
<td>22.2±4.6</td>
<td>21.2±4.1</td>
<td>22.1±4.0</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>22.9±6.4</td>
<td>20.9±3.0</td>
<td>21.7±3.4</td>
</tr>
<tr>
<td>BSA, m&lt;sup&gt;2&lt;/sup&gt;</td>
<td>M</td>
<td>1.6±0.3</td>
<td>1.6±0.3</td>
<td>1.8±0.2</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>1.6±0.2</td>
<td>1.6±0.2</td>
<td>1.7±0.2</td>
</tr>
</tbody>
</table>

Values are mean±SD. WHR indicates waist-to-hip ratio; V, visit; S, sex; R, race; and ×, interaction.
Multivariate Relationships

The hierarchical stepwise regression analysis predicting future LVM/height$^{2,7}$ found initial LVM/height$^{2,7}$ to be the single best predictor ($R^2$=0.38). This was followed in the second block by BMI ($R^2$ increase=0.11) and gender ($R^2$ increase=0.02) and finally in the third block by the supine resting TPR readings from the initial evaluation ($R^2$ increase=0.02; total $R^2$=0.53; $P<0.001$). No other variables added significantly ($P<0.05$) to the model.

Follow-up LVM/BSA was predicted by baseline LVM/BSA ($R^2$=0.29), followed in the second block by weight ($R^2$ increase=0.06), gender ($R^2$ increase=0.11), and in the third block by mean supine resting TPR ($R^2$ increase=0.02). SBP change score to car-driving simulation was the only other parameter to enter the model in the final block ($R^2$ increase=0.03; total $R^2$=0.48). Follow-up MFS was predicted by baseline MFS ($R^2$=0.04), followed by race (white more than black; $R^2$ increase=0.04) and mean supine TPR (ie, negative relationship) in the second block ($R^2$ increase=0.06; total $R^2$=0.13).

Discussion

Few longitudinal studies have been conducted with normotensive youth evaluating the stability of LVM.$^{10,11,23}$ These studies involved white and black youth samples ranging in age from 10 to 17 years, with time intervals ranging from 2.5 to 6 years. All found relatively good stability whether LVM was indexed by body habitus or not ($r$ range, 0.40 to 0.83). The present findings in conjunction with these other recent findings suggest that the relative rank of LVM indexed by body habitus is a stable individual characteristic regardless of race or gender from late childhood through adolescence, which is marked by significant physical and social/emotional development. It should be noted that although Tanner staging was not conducted, it is unlikely that the outcome variables were influenced by differential pubertal status (ie, prepubertal versus pubertal). This is because at baseline the vast majority of subjects, regardless of race, were above the age at which puberty typically begins.

Prediction of Future LV Myocardial Function

To the best of our knowledge, this is the first study in youth to reveal an interaction between LV midwall performance and race. Blacks had relatively lower MFS ratio than white youth. The reason for this difference is not fully understood. This finding is, however, consistent with the known association of increased RWT, TPR, and LVM index on the one hand and lower MFS ratio on the other.$^{21}$ Although a direct clinical relevance is not implied, this finding in black youth may be a preclinical marker of adverse prognosis. In view of recent findings that subtle decreases in MFS are associated with increased cardiovascular risk profile in asymptomatic adults,$^{24}$ follow-up studies are needed to determine whether a
lower MFS ratio in normotensive youth predicts earlier onset of cardiovascular events.

**Relationship of ABP to Future LVM**

To our knowledge, only 1 study has examined the relationship between ABP and LVM in youth. Using a cross-sectional design, Belsha et al. found daytime and nighttime SBP but not DBP to be moderately significantly correlated with indexed LVM 2.3 years later.11–13 Our findings corroborate these studies in that SBP and/or TPR responsivity to video game challenge, forehead cold stimulation, and mirror tracing to be positively related to indexed and unindexed measures of LVM.7,9 Longitudinal studies have found the SBP, cardiac output, and/or TPR responsivity to physical (ie, dynamic exercise, orthostasis, cold pressor) and interpersonal behavioral stressors (ie, social stressor interview, car-driving simulation) to be related to increased aerobic fitness, which is associated with lower heart rate and increased LVM. The present findings lend support to this in that baseline resting heart rate was also correlated negatively with the LVIDD at follow-up (r = 0.37; P < 0.01).

Several recent pediatric studies have observed significant associations between hemodynamic responsivity to acute laboratory stressors and LVM. Cross-sectional studies have shown the SBP and/or TPR responsivity to video game challenge, forehead cold stimulation, and mirror tracing to be positively related to indexed and unindexed measures of LVM.7,9 Longitudinal studies have found the SBP, cardiac output, and/or TPR responsivity to physical (ie, dynamic exercise, orthostasis, cold pressor) and interpersonal behavioral stressors (ie, social stressor interview, car-driving simulation) to be related to increased indexed LVM 2 to 3 years later.11–13

**Relation of Hemodynamics at Rest and During Acute Stress to Future LVM**

The present results corroborate other recent longitudinal findings in youth in that resting SBP was correlated positively with follow-up LVM indexed either by height11 or BSA.10–12 Likewise, resting heart rate was negatively correlated to both follow-up measures of indexed LVM. It has been hypothesized that this inverse relationship may be partly related to increased aerobic fitness, which is associated with lower heart rate and increased LVM. The present findings lend support to this in that baseline resting heart rate was also correlated negatively with the LVIDD at follow-up (r = 0.37; P < 0.01).

Several recent pediatric studies have observed significant associations between hemodynamic responsivity to acute laboratory stressors and LVM. Cross-sectional studies have shown the SBP and/or TPR responsivity to video game challenge, forehead cold stimulation, and mirror tracing to be positively related to indexed and unindexed measures of LVM.7,9 Longitudinal studies have found the SBP, cardiac output, and/or TPR responsivity to physical (ie, dynamic exercise, orthostasis, cold pressor) and interpersonal behavioral stressors (ie, social stressor interview, car-driving simulation) to be related to increased indexed LVM 2 to 3 years later.11–13 Our findings corroborate these studies in that SBP and/or TPR responses to a variety of passive physical or challenging behavioral stressors were positively related to measures of indexed LVM 2.3 years later.

This is the first study to evaluate comprehensively the independent contributions of hemodynamic functioning at rest, during acute stress, and in the field (ABP) in the prediction of LVM. Similar to other recent longitudinal findings,11,12 the present results confirm that male gender, adiposity, vasoconstrictive tone, and hemodynamic stress responsivity are independent predictors of indexed LVM.

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**TABLE 3. Echocardiographic Data**

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Sex</th>
<th>Visit 1</th>
<th>Visit 2</th>
<th>Visit 1</th>
<th>Visit 2</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVIDD, mm</td>
<td>M</td>
<td>46.9±4.3</td>
<td>48.0±4.9</td>
<td>47.0±5.0</td>
<td>49.6±4.6</td>
<td>V&lt;0.001, S&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>45.5±3.7</td>
<td>44.8±4.0</td>
<td>43.6±4.1</td>
<td>44.5±3.8</td>
<td>V×S&lt;0.01</td>
</tr>
<tr>
<td>LVM, g</td>
<td>M</td>
<td>126.2±36.4</td>
<td>138.0±36.9</td>
<td>117.6±35.2</td>
<td>130.7±32.1</td>
<td>R&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>113.8±36.7</td>
<td>120.7±32.3</td>
<td>96.6±30.5</td>
<td>99.7±31.6</td>
<td>S&lt;0.001</td>
</tr>
<tr>
<td>LVM/BSA, g/cm²</td>
<td>M</td>
<td>76.7±16.9</td>
<td>77.2±15.0</td>
<td>71.6±12.1</td>
<td>72.0±12.0</td>
<td>R&lt;0.01</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>70.0±12.7</td>
<td>64.4±11.5</td>
<td>61.1±9.2</td>
<td>60.5±6.9</td>
<td>S&lt;0.001</td>
</tr>
<tr>
<td>LVM/height², g/m²</td>
<td>M</td>
<td>33.1±6.0</td>
<td>32.4±6.7</td>
<td>30.4±6.6</td>
<td>29.1±6.0</td>
<td>R&lt;0.001</td>
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<td></td>
<td>F</td>
<td>31.0±7.0</td>
<td>28.8±7.2</td>
<td>26.2±4.2</td>
<td>25.9±3.5</td>
<td>S&lt;0.001</td>
</tr>
<tr>
<td>RWT</td>
<td>M</td>
<td>0.35±0.04</td>
<td>0.35±0.04</td>
<td>0.33±0.04</td>
<td>0.32±0.04</td>
<td>R&lt;0.001</td>
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<td></td>
<td>F</td>
<td>0.34±0.05</td>
<td>0.34±0.04</td>
<td>0.34±0.06</td>
<td>0.33±0.04</td>
<td>R&lt;0.001</td>
</tr>
<tr>
<td>cESS, 10³dyne/cm²</td>
<td>M</td>
<td>129.0±33.9</td>
<td>158.0±28.3</td>
<td>144.6±29.0</td>
<td>163.0±25.6</td>
<td>V&lt;0.001</td>
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<td>127.8±21.8</td>
<td>140.6±20.1</td>
<td>126.0±25.3</td>
<td>139.4±23.3</td>
<td>S&lt;0.001</td>
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<tr>
<td>MFS, %</td>
<td>M</td>
<td>19.2±2.6</td>
<td>18.2±1.9</td>
<td>19.0±2.3</td>
<td>19.4±2.0</td>
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<td>F</td>
<td>19.3±2.2</td>
<td>19.0±1.8</td>
<td>20.0±2.9</td>
<td>20.0±1.7</td>
<td>R&lt;0.001</td>
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<tr>
<td>MFS ratio</td>
<td>M</td>
<td>112.3±13.3</td>
<td>110.1±10.8</td>
<td>114.7±14.3</td>
<td>118.0±13.0</td>
<td>R&lt;0.05</td>
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<td>F</td>
<td>113.4±11.5</td>
<td>112.4±9.4</td>
<td>113.7±16.7</td>
<td>115.2±10.1</td>
<td>R&lt;0.001</td>
</tr>
</tbody>
</table>

Values are mean±SD. Abbreviations are as defined in Table 1.
Unexpectedly, ABP was not found to be a significant independent predictor of indexed LVM. Findings concerning the relationship between ABP and LVM have been mixed. Some adult studies have reported positive correlations, while others failed to establish such associations.26–29 The lack of independent predictive power in the present study may have been due in part to the moderate correlations between ABP and measures of weight, adiposity, and resting TPR, all of which were independent predictors of both measures of indexed LVM.

In summary, baseline LVM index and MFS predicted cardiac structure and function 2.3 years later in these youth. Increased TPR consistently predicted future decreased MFS and increased LVM indexed by height or BSA. This would suggest that increased vascular tone leading to increased TPR plays a primary role in cardiac remodeling (increased mass and/or increased RWT) and function (lower MFS). The relative clustering of these findings (increased RWT, LVM index, TPR, and lower MFS ratio) in black youth with family histories of cardiovascular diseases may provide a possible explanation for the excess cardiovascular morbidity and mortality in black adults. It may be that these black youth with higher LVM index, resting SBP, and RWT exhibit an exaggerated BP responsivity to daily recurring psychosocial stress, which over time may elicit future increases in BP and LVM and lower midwall shortening. Future studies are necessary to evaluate these proposed mechanisms. Equally importantly, long-term follow-up is needed to determine whether the racial difference in MFS ratio observed herein is associated with significant racial differences in clinical outcomes.

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References

Hemodynamic Function at Rest, During Acute Stress, and in the Field: Predictors of Cardiac Structure and Function 2 Years Later in Youth
Gaston K. Kapuku, Frank A. Treiber, Harry C. Davis, Gregory A. Harshfield, Barton B. Cook and George A. Mensah

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