Predicting Heart Growth During Puberty: The Muscatine Study

Kathleen F. Janz, EdD*; Jeffrey D. Dawson, ScD‡; and Larry T. Mahoney, MD§

ABSTRACT. Objectives. During childhood, heart growth is closely associated with somatic growth including increases in body weight, fat-free body mass (FFM), and height. However, with age, greater variability in heart size in relationship to body size is observed, presumably attributable to the increased effect of cardiac workload. At this time, little is known as to what functional attributes (e.g., aerobic fitness) contribute to cardiac workload and the relative contribution of these attributes to heart growth during childhood and adolescence. In this article, we report cross-sectional and longitudinal relationships among aerobic fitness, body size, blood pressure (BP), and left ventricular mass (LVM) through puberty including the predictors of heart growth during puberty and the tracking of LVM from pre-puberty to late and post-puberty. Describing the predictors of heart size and heart growth and establishing the likelihood that a large heart, relative to peers, may (or may not) remain a large heart should aid pediatricians in discerning between normal developmental increases in LVM and increases in LVM suggestive of excessive heart growth (left ventricular hypertrophy).

Methodology. Using a repeated-measures design, we assessed aerobic fitness, FFM, fatness, weight, height, sexual maturation, resting BP, peak exercise BP, and LVM in 125 healthy children (mean baseline age: 10.5 years) for a period of 5 years. All subjects were either in prepuberty or early puberty at the beginning of the study. At follow-up, 110 subjects attempted all research procedures (87% of the initial cohort). Using anthropometry and bioelectrical impedance, we measured FFM, fatness, weight, and height quarterly (once every 3 months) for a total of 20 examinations. Resting BP and LVM (2-dimensional echocardiography) were also assessed quarterly. Aerobic fitness, peak exercise BP, and sexual maturation (staging of secondary sex characteristics and, for boys, serum testosterone) were measured annually (5 examinations). The same field staff conducted all examinations.

Statistical methods included Spearman rank correlation coefficients (r_s) calculated to estimate how well the year 5 LVM was predicted by LVM at earlier years. We also categorized the LVM data into tertiles and reported year 5 LVM was predicted by LVM at earlier years. We also categorized the LVM data into tertiles and reported predictor variables, also quantified by subject-specific slopes.

Results. At baseline and at follow-up, boys tended to be taller, leaner, more aerobically fit, and had greater LVM than girls. Rate of change for these variables was also greater in boys than girls. For example, LVM increased 62% in boys and 48% in girls. At year 5, subjects had advanced at least 1 stage in genital or breast development and over 80% of the subjects were in late- or post-puberty. Significant and strong tracking of heart size (r_s = .65–.87) was observed. The likelihood that a subject would be in an extreme tertile for heart size at follow-up was approximately doubled if he or she started there at baseline.

In boys, baseline FFM explained 54% of the variability in follow-up LVM. Change in aerobic fitness and change in FFM explained 55% of the variability in change in LVM. In girls, baseline aerobic fitness and fatness explained 45% of the variability in follow-up LVM. Because FFM did not enter in this model, we constructed an alternative model in which baseline aerobic fitness adjusted for FFM was entered. Using this approach, 43% of the variability in follow-up LVM was explained by baseline FFM, fatness, and adjusted aerobic fitness. Change in FFM explained 58% of the variability in change in LVM. For both boys and girls, all statistically significant variables entered as positive regression coefficients.

Conclusions. Our tracking results suggest predictability in LVM, most likely attributable to normal growth and regulated by the genetic and hormonal influences that affect FFM as well as LVM. Our multiple regression results indicate that during adolescence FFM is an important determinant of heart size and heart growth for both boys and girls but changes in aerobic fitness, presumably attributable to improvements in cardiac function, also affect heart growth in boys. This latter finding indicates that the known age-related decrease in the ability of body size to fully predict heart size begins sooner in males than females. It also suggests that changes in cardiac functioning as well as morphologic increases in lean tissue cause the “athletic heart syndrome.”

ABBREVIATIONS. FFM, fat-free body mass; LVM, left ventricular mass; BP, blood pressure; VO2, volume of oxygen uptake; HR, heart rate; SKF, skinfolds; SBP, systolic blood pressure; DBP, diastolic blood pressure.

During childhood, heart growth is closely associated with somatic growth including increases in body weight, fat-free body mass (FFM), and height.1,2 However, with age, greater...
variability in heart size in relationship to body size is observed, presumably attributable to the increased effect of cardiac workload.\textsuperscript{3,4} For example, using a sample of 766 subjects ranging from 0 to 85 years of age, de Simone and colleagues\textsuperscript{5} have shown an age-related decrease in the value of body size and an age-related increase in the value of cardiac workload for predicting heart size. At this time, little is known as to what functional attributes (eg, aerobic fitness) contribute to cardiac workload and the relative contribution of these attributes to heart growth during childhood and adolescence.

Previously, we reported cross-sectional and 2-year follow-up relationships among aerobic fitness, body composition, and left ventricular mass (LVM) in pre-pubescent and early pubescent children (mean baseline age: 10.5 years) and found FFM to be the most important predictor of LVM (heart size)\textsuperscript{6} and increased FFM to be the most important predictor of increased LVM (heart growth).\textsuperscript{7} In these studies, FFM explained 55\% of the variability in LVM, while increased FFM explained 18\% of the variability in increased LVM. In this article, using the same cohort, we report cross-sectional and longitudinal relationships among aerobic fitness, body size, blood pressure (BP), and LVM through puberty including the predictors of heart growth during puberty and the tracking of LVM from prepuberty to late and post-puberty. Describing the predictors of heart size and heart growth and establishing the likelihood that a large heart, relative to peers, may (or may not) remain a large heart should aid pediatricians in discerning between normal developmental increases in LVM and increases in LVM suggestive of excessive heart growth (left ventricular hypertrophy).

METHODS

Subjects

The Muscatine Study is a longitudinal, population-based investigation of cardiovascular disease risk factors in children, young adults, and selected family groups from Muscatine, Iowa. From a sample of 766 subjects ranging from 0 to 85 years of age, de Simone and colleagues\textsuperscript{5} have shown an age-related decrease in the value of body size and an age-related increase in the value of cardiac workload for predicting heart size. At this time, little is known as to what functional attributes (eg, aerobic fitness) contribute to cardiac workload and the relative contribution of these attributes to heart growth during childhood and adolescence.

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shoulder and the olecranon.14 The mean of the 3 measures of the first (systolic BP [SBP]) and fourth (diastolic BP [DBP]) Korotkoff sounds were recorded as the resting BP. We recorded the fifth Korotkoff sound in the very rare instances that the fourth sound was not discernable (and in these instances set any value <30 mm Hg to missing).

Echocardiographic Determination of LVM

Image Acquisition

Two-dimensional echocardiograms were obtained with a Hewlett-Packard Ultrasound System (Andover, MA) with transducer selection determined by the child’s body size and image quality. The transducer was positioned according to the standards for transducer positions and imaging planes.5,16 A simultaneous electrocardiogram was recorded for reference. A short-axis parasternal view of the heart was obtained by rotating the transducer 90 degrees from the parasternal long-axis view. In this view, the tips of the papillary muscles can be identified by a superior–inferior sweep until the chordae disappear from the papillary muscles. To assure acquisition of the maximum major axis, an apical 4-chamber view was obtained with the left ventricle imaged as parallel as possible to the ultrasound beam. End-systole was defined by setting end-systole to trigger from the peak of the T wave. Multiple gated images were recorded on videotape for later measurement.

Measurements

All measurements were made off-line from videotape on a Microsonics CAD 886 system (Indianapolis, IN). Accurate calibration of this system and the echocardiographic unit was confirmed with an echocardiographic phantom. In the short-axis view, the left ventricular epicardium at end-systole was traced using the midinterface convention.15 The papillary muscles were excluded from the tracing of the endocardium. The resultant planimetered values were recorded as epicardial and endocardial areas. The major axis, or length, of the left ventricle was measured in the apical 4-chamber view at end-systole from the mitral valve annulus to both the endocardium and epicardium. If either interface was not clearly visualized, the missing length was calculated by measuring the left ventricular posterior wall at end-systole in the short-axis view and the appropriate value was either added to the endocardial measurement to equal the epicardial value or, conversely, subtracted from the epicardial measurement to equal the endocardial value. Measurements were made on 3 cardiac cycles and averaged. Epicardial and endocardial volumes and the derived LVM were calculated during systole as follows17:

\[
\text{Volume} = \frac{2}{3} \times (\text{area} \times \text{length})
\]

\[
\text{LVM} = 1.05 \times (\text{epicardial volume} - \text{endocardial volume}).
\]

In our pediatric laboratory, intraclass correlations for 2-dimensional echocardiographic measures and derived variables range from .89 to .96, with a precision of 7.8 g for LVM measured at end-systole (95% confidence interval: 6.9–9.6 g).18

Statistical Analysis

The 4 quarterly measures of body composition and LVM were converted to yearly averages. Means and standard deviations were then calculated for all variables. Variables were stratified by gender and the sex for distributional properties. Wilcoxon rank sum tests were used to examine gender differences. Spearman rank correlation coefficients (r) were calculated to estimate how well the year 5 LVM was predicted by LVM at earlier years. We also categorized the LVM data into tertiles and reported the percentage who remained in the extreme tertile at follow-up. The statistical significance of the tracking within tertiles was assessed using Kendall’s Tau-b.

Predictors of LVM and predictors of changes in LVM were tested using the following approaches. First, we calculated the Spearman correlation coefficients between follow-up LVM and each potential predictor (year 1) and follow-up (year 5) predictor. Next, we calculated subject-specific slopes for LVM and each predictor, and then used the Spearman correlation to assess whether the changes in LVM, as quantified by the slopes, were associated with the changes in potential predictor variables. Finally, we used multiple linear regression to fit models to predict follow-up LVM and changes in LVM. Predictor variables considered for these models included age, FFMI, height, peak VO2, peak SBP, peak DBP, sum of SKF, and maturity (Tanner and, for boys, testosterone). Variables were allowed to enter the models if significant at the <.05 probability level.

RESULTS

Table 1 characterizes, by gender, the study subjects and includes the yearly rate of change (slope) for variables. At baseline and at follow-up, boys tended to be taller, leaner, more aerobically fit, and had greater LVM than girls. Rate of change for these variables was also greater in boys than girls. For example, LVM increased 62% in boys and 48% in girls. At the beginning of the study, all boys and girls were either in prepuberty or early puberty. At year 5, subjects had advanced at least 1 stage in genital or breast development and over 80% of the subjects were in late- or post-puberty. The proportion of boys and girls within specific stages for pubic hair development was similar to the proportion in specific stages for genital and breast development.

Tracking of LVM

Table 2 presents the Spearman correlation coefficients between follow-up LVM and preceding years. The table also shows the likelihood of remaining in an extreme tertile at follow-up given the subject was in the same extreme tertile at baseline. The tracking correlation from baseline (year 1) to follow-up (year 5) was .66 for boys and .78 for girls. There was an increased likelihood that a subject would be in an extreme tertile at follow-up if he or she was in the same tertile at baseline. The relative chance of a boy or girl being in the upper tertile for LVM at the end of the study was approximately doubled if he or she started there at baseline. The relative chance of a subject being in the lower tertile for LVM at follow-up was 2.6 times as great for a girl and 1.6 times as great for a boy if he or she started there at baseline.

Bivariate Results

Relationships among potential predictor variables and LVM were examined using Spearman correlation coefficients (Table 3). Baseline and follow-up body size variables, SBP, peak SBP, and peak VO2 were moderately to strongly associated with follow-up LVM. Overall, associations between baseline variables and follow-up LVM were similar in direction and magnitude to associations between follow-up variables and follow-up LVM. However, baseline Tanner index was not associated with follow-up LVM, but follow-up maturation was associated with follow-up LVM (r = .40-.43). Baseline DBP in girls was not associated with follow-up LVM (r = .20), but follow-up DBP was associated with follow-up LVM (r = .36).

Spearman correlation coefficients describing associations among change in potential predictor variables and increases in LVM are presented in Table 4. In boys, change in testosterone, change in maturation, change in height, change in FFMI, and change in
peak V\textsubscript{o}\textsubscript{2} were strongly associated with change in LVM ($r_s = .60-.77$), whereas, in girls, change in height, change in weight, and change in FFM were strongly associated with change in LVM ($r_s = .61-.75$). Moderate associations existed between change in LVM and change in SBP (resting SBP and peak SBP) for both boys and girls, with correlation coefficients ranging from .32 to .43. Change in DBP was not significantly associated with change in LVM but was barely significant in boys ($r_s = .26; P = .043$). Change in sum of SKF (our measure of body fatness) was inversely associated with change in LVM ($r_s = -.28$) in boys but was positively associated with change in LVM in girls ($r_s = .38$).

### Regression Results

Stepwise multivariate analysis was used to evaluate predictors of follow-up LVM and predictors of changes in LVM. Three gender-specific models were considered. The first model used predictor variables measured at follow-up to examine variability in follow-up LVM (a cross-sectional approach). The second model examined predictors of follow-up LVM using baseline variables (prospective follow-up approach). The third model examined whether the variability in the changes in LVM, as quantified by subject-specific slopes, could be explained by changes in predictor variables, also quantified by subject-specific slopes.

In boys, follow-up peak V\textsubscript{o}\textsubscript{2}, sum of SKF, peak SBP, and age explained 82% of the variability in follow-up LVM (Fig 1). In this cross-sectional model, FFM did not enter; therefore, we could not be sure if peak V\textsubscript{o}\textsubscript{2} was independent of its effects, so we constructed an alternative model in which peak V\textsubscript{o}\textsubscript{2} adjusted for FFM was entered. Using this approach, 82% of the variability in follow-up LVM was explained by follow-up FFM, peak SBP, sum of SKF, and adjusted peak V\textsubscript{o}\textsubscript{2} (data not shown). When baseline variables were entered (prospective follow-up approach), baseline FFM explained 54% of the variability in follow-up LVM (Fig 2). Change in peak V\textsubscript{o}\textsubscript{2} and change in FFM explained 55% of the variability in change in LVM (Fig 3).

In girls, follow-up FFM, peak V\textsubscript{o}\textsubscript{2}, and sum of SKF explained 61% of the variability in follow-up LVM (Fig 1). Baseline peak V\textsubscript{o}\textsubscript{2} and sum of SKF explained

### TABLE 1. Description of Subjects

<table>
<thead>
<tr>
<th>Subject</th>
<th>Baseline</th>
<th>Follow-up</th>
<th>Slope (Change/Year)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Boys</td>
<td>10.8 (1.0)*</td>
<td>14.6 (1.0)*</td>
<td>1.0 (0.1)</td>
</tr>
<tr>
<td>Girls</td>
<td>10.3 (1.0)</td>
<td>14.2 (1.0)</td>
<td>1.0 (0.1)</td>
</tr>
<tr>
<td>Height (cm)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Boys</td>
<td>143.9 (7.2)*</td>
<td>169.4 (8.3)*</td>
<td>6.3 (1.2)*</td>
</tr>
<tr>
<td>Girls</td>
<td>140.6 (8.4)</td>
<td>160.0 (5.8)</td>
<td>4.9 (1.5)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Boys</td>
<td>39.2 (10.6)</td>
<td>62.4 (16.0)</td>
<td>5.6 (1.0)</td>
</tr>
<tr>
<td>Girls</td>
<td>37.9 (9.5)</td>
<td>58.8 (13.5)</td>
<td>5.2 (2.0)</td>
</tr>
<tr>
<td>FFM (kg)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Boys</td>
<td>30.1 (5.1)*</td>
<td>49.3 (9.4)*</td>
<td>4.7 (1.6)*</td>
</tr>
<tr>
<td>Girls</td>
<td>27.8 (5.4)</td>
<td>40.8 (6.4)</td>
<td>3.3 (1.1)</td>
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<tr>
<td>Sum of SKF (mm)</td>
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<td></td>
</tr>
<tr>
<td>Boys</td>
<td>72.9 (41.5)*</td>
<td>78.1 (44.5)*</td>
<td>1.6 (7.1)*</td>
</tr>
<tr>
<td>Girls</td>
<td>88.1 (34.2)</td>
<td>111.0 (43.1)</td>
<td>5.3 (6.7)</td>
</tr>
<tr>
<td>SBP (mm Hg)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Boys</td>
<td>98.0 (7.5)</td>
<td>106.8 (11.2)</td>
<td>2.2 (2.4)</td>
</tr>
<tr>
<td>Girls</td>
<td>98.3 (6.8)</td>
<td>105.1 (8.1)</td>
<td>1.6 (1.6)</td>
</tr>
<tr>
<td>DBP (mm Hg)</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Boys</td>
<td>60.9 (6.8)*</td>
<td>63.2 (6.3)</td>
<td>.7 (2.3)</td>
</tr>
<tr>
<td>Girls</td>
<td>63.3 (6.7)</td>
<td>63.1 (6.3)</td>
<td>.1 (1.6)</td>
</tr>
<tr>
<td>Peak SBP (mm Hg)</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Boys</td>
<td>166.8 (19.3)</td>
<td>187.9 (20.2)*</td>
<td>5.1 (5.0)*</td>
</tr>
<tr>
<td>Girls</td>
<td>163.0 (19.8)</td>
<td>174.4 (14.8)</td>
<td>2.9 (5.7)</td>
</tr>
<tr>
<td>Peak V\textsubscript{o}\textsubscript{2} (mL/min)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Boys</td>
<td>1860 (299)*</td>
<td>2729 (490)*</td>
<td>223 (122)*</td>
</tr>
<tr>
<td>Girls</td>
<td>1479 (345)</td>
<td>1907 (293)</td>
<td>133 (80)</td>
</tr>
<tr>
<td>LVM (g)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Boys</td>
<td>91.4 (19.8)*</td>
<td>148.4 (39.7)*</td>
<td>13.5 (8.1)*</td>
</tr>
<tr>
<td>Girls</td>
<td>83.1 (18.3)</td>
<td>122.8 (22.3)</td>
<td>10.5 (3.9)</td>
</tr>
<tr>
<td>Testosterone (mg/dL)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Boys</td>
<td>15.8 (15.1)</td>
<td>484.9 (174.4)</td>
<td>112.5 (61.7)</td>
</tr>
</tbody>
</table>

* Boys significantly different than girls ($P < .05$).

### TABLE 2. Spearman Correlation Coefficients Tracking LVM Between Follow-up (Year 5) and Preceding Years (4 to 1) and Percentage of Subjects Remaining in Lower and Upper Tertiles for LVM at Follow-up Who Were in These Same Tertiles at Baseline

<table>
<thead>
<tr>
<th>Year</th>
<th>Year 4</th>
<th>Year 3</th>
<th>Year 2</th>
<th>Year 1</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVM (g)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Boys</td>
<td>.87</td>
<td>.81</td>
<td>.66</td>
<td>.66</td>
</tr>
<tr>
<td>Girls</td>
<td>.85</td>
<td>.73</td>
<td>.65</td>
<td>.78</td>
</tr>
<tr>
<td>Percentage of Subjects Remaining in Tertiles*</td>
<td>Lower</td>
<td>Upper</td>
<td>(P)</td>
<td></td>
</tr>
<tr>
<td>LVM (g)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Boys</td>
<td>53</td>
<td>65</td>
<td>&lt;.001</td>
<td></td>
</tr>
<tr>
<td>Girls</td>
<td>84</td>
<td>68</td>
<td>&lt;.001</td>
<td></td>
</tr>
</tbody>
</table>

All correlations were highly significant ($P < .001$). * Thirty-three percent would be expected by chance alone, while 100% would indicate perfect tracking.
45% of the variability in follow-up LVM (Fig 2). Again, because FFM did not enter into this model, we constructed an alternative model in which baseline peak VO2 adjusted for FFM was entered. Using this approach, 45% of the variability in follow-up LVM was explained by baseline FFM, sum of SKF, and adjusted peak VO2 (data not shown). Change in FFM explained 58% of the variability in change in LVM (Fig 3). For both boys and girls, all statistically significant variables entered as positive regression coefficients.

**DISCUSSION**

Aerobic fitness, body size, BP, maturation, and LVM were measured in a representative sample of children in prepuberty and early puberty. At follow-up almost all study subjects were in late or post-puberty and heart size had increased 62% in boys and 48% in girls. Throughout the study, we found significant and strong tracking of heart size. Results also indicate that during this period of rapid heart growth, FFM is an important determinant of heart growth for both boys and girls but changes in aerobic fitness, presumably attributable to improvements in cardiac function, also affect heart growth in boys. Although, we detected several moderate bivariate associations among BP and LVM, in our multivariate analyses, only follow-up peak SBP predicted follow-up LVM (in boys) and its contribution was relatively weak, explaining 3.5% of the variability in follow-up LVM.

There was a strong tendency for boys ($r_s = .66$) and an even stronger tendency for girls ($r_s = .78$) to maintain rank order LVM across the observational period. Perhaps attributable to our ability to reduce the imprecision associated with echocardiography by using multiple (quarterly) measures for each observational year, these tracking results are greater in magnitude than previous reports. For example, within the Medical College of Virginia Twin Study, Schieken and colleagues noted tracking correlation coefficients of .48 for a 3-year interval and .46 for a 4.5-year interval for 231 subject, 11 years of age at baseline. Gender differences in the magnitude of tracking were not observed in their study. After adjusting for weight and height, Mahoney and colleagues reported a tracking correlation of .36 for a 3.4-year interval for 274 children and adolescents with baseline ages ranging from 6 to 15 years. Gender differences in the magnitude of tracking were also not reported in the study by Mahoney et al.

Our results, when coupled with the work of others, suggest predictability in LVM, most likely attributable to normal growth and regulated by the genetic and hormonal influences that affect FFM as well as LVM. In addition, our results indicate that LVM tracks better in girls than boys. The poorer tracking in boys may be associated with their wider variation during pubertal growth spurts (compared with girls), different patterns of growth between boys and girls (20–22), and possibly an earlier effect of cardiac function to myocardial development in boys.

In our bivariate analysis, follow-up sexual maturation was associated with follow-up LVM and change in sexual maturation was associated with change in LVM in both boys and girls. Change in testosterone, in boys, was the strongest maturation-related association with change in LVM ($r_s = .64$). However, in multivariate analysis, neither Tanner staging nor testosterone entered as an independent predictor. Similar findings have been reported by others and suggest that the effect of sexual maturation on LVM is mediated through hormonal changes that also influence body size, particularly acquisition of FFM.
In our regression analysis, changes in FFM explained approximately half of the observed heart growth for boys and girls, ie, on average, the boys and girls who were gaining the most lean tissue were also the same children experiencing the greatest increases in heart size. This finding parallels the cross-sectional work of Daniels and colleagues.22,23 Interestingly, changes in height and sum of SKF were not independent predictors of heart growth in our study. These results indicate that during puberty, increased FFM is the primary body size determinant of heart growth, whereas increased fat mass is of substantially less importance. However, because lean tissue is needed to support additional fat tissue, FFM may increase in response to increased fat mass23 suggesting that for some of our heavier subjects, the observed link between changing FFM and changing LVM was associated with their increasing obesity.

Fig 1. Explained variance (%) by gender for follow-up LVM using predictor variables measured at follow-up.

Fig 2. Explained variance (%) by gender for follow-up LVM using predictor variables measured at baseline.
Aerobic fitness (peak Vo2) was associated with heart size and heart growth in both boys and girls. However, improvements in aerobic physical fitness independently predicted heart growth only in boys. These data indicate that as boys mature, changes in cardiac function associated with aerobic fitness influence myocardial development. Other investigators have also observed gender-specific associations between cardiac function and LVM. Goble and colleagues studied 11-year-old children and reported inverse associations between resting HR and LVM. They attributed this relationship to physical conditioning and speculated that the boys were more physically fit than were girls and that this difference might contribute to gender differences in LVM. De Simone and colleagues have shown that while the affect of body size to heart size decreases with age, the influence of gender to heart size increases with age, and the influence of cardiac workload increases with age. In Native American adults, Bella and colleagues have reported that FFM, age, gender, stroke volume, and cardiac midwall shortening (an index of afterload) explain 72% of the variability in LVM. Because the ability to increase stroke volume during exercise is one of the most critical factors in determining aerobic fitness, their results support our contention that changes in cardiac function associated with aerobic fitness influence myocardial development. Similarly, in healthy normotensive men, Molina and colleagues have observed positive associations between physical activity and LVM and no associations between physical activity and left ventricular hypertrophy.

CONCLUSION

In summary, by considering aerobic fitness and indices of body size, particularly FFM, we were able to explain an appreciable proportion of the variability in heart growth in normal children and adolescents. We have also shown strong tracking of heart size through puberty and gender-specific differences in the effect of changes in aerobic fitness to heart growth. The latter suggests that the known age-related decrease in the ability of body size to fully predict heart size may begin sooner in males than in females. Finally, our work indicates that changes in sexual maturation and changes in BP are not important in determining heart growth in normal children and adolescents. The biological mechanisms in which FFM and aerobic fitness affect myocardial development and their potential value for indexing normal, healthy heart growth warrant further inquiry.

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