Measurement of Left Ventricular Wall Mass in Pediatric Populations

Richard M. Schieken

SUMMARY This paper reviews the techniques for obtaining technically adequate echocardiograms for epidemiologic studies. When these techniques were applied to studies of pediatric populations the following objectives were achieved: 1) reproducible echocardiograms, 2) observations about the relationship of echocardiographic variables to cardiovascular variables, and 3) information about the relationship of echocardiographic and electrocardiographic variables to each other and to blood pressure. We documented the precision of M-mode left ventricular chambers and dimensions, interobserver and intraobserver variability, and the day-to-day variability of these measures. Left ventricular wall mass was significantly larger than expected for age and body size in children with persistently elevated blood pressure. The relationship between the echocardiographic and electrocardiographic variables was poor. Moreover, the electrocardiographic measures of ventricular hypertrophy did not correlate with blood pressure. (Hypertension 9 [Suppl II]: 11-47—11-52, 1987)

KEY WORDS • hypertension • left ventricular mass • children • echocardiography

ALTHOUGH echocardiograms are frequently used to diagnose anatomic and functional heart disease in children, the usefulness of the echocardiogram in cardiovascular population studies had not been established when our study began. Before the echocardiogram could be employed in cardiovascular epidemiologic studies, the sources and magnitude of variability had to be identified and, if possible, reduced to acceptable levels. In clinical situations, the agreement between two independent readers measuring randomly selected echocardiograms was low, with the interclass correlation coefficient averaging 0.73 for most dimensions of the left side of the heart. To increase the agreement between independent readers, we developed a set of reading criteria. These reading criteria defined technically acceptable tracings and standardized measurement techniques.

After documenting the precision and reproducibility of the echocardiographic as an epidemiologic tool, we sought to study children whose blood pressure level persisted in the low, middle, and upper parts of the distribution for age and sex, to determine whether echocardiographic differences existed in cardiac output and left ventricular wall mass. Because of the wide availability of the electrocardiogram, we undertook to determine whether the electrocardiogram could detect a difference in left ventricular wall mass among children with high, middle, or low blood pressure.

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Methods

Echocardiographic Reproducibility

Our study subjects were healthy volunteer children of the community, aged 6–16 years with a mean age of 8.9 years. Studies were performed beginning at 1400 each day. The echocardiograms were recorded using an Echoline 20A ultrasonoscope (SKI Industries) with a 3.5-mHz transducer interfaced with a Honeywell (Minneapolis, MN, USA) 1856 strip chart recorder.

The criteria for defining an acceptable line for measurement were 1) a single dominant line, 2) continuous lines at the point of measurement of at least 5 mm in length, and 3) an interface with motion characteristics for the specific cardiac structure.

Tracings that did not meet these criteria could not be measured reproducibly.

The standard interspace technique for transducer position was used. The transducer was placed perpendicular to the chest wall to record the anterior mitral leaflet. The ultrasonic beam was angulated toward the apex of the left ventricle to record the maximal dimension showing both rapid systolic anterior left ventricular posterior wall motion and rapid systolic posterior interventricular septal motion. The chordae tendinae were delineated.

The electrocardiogram was clearly defined with a discernible QRS complex demonstrating a normal sinus rhythm. If sinus arrhythmia was present, the cardiac cycles with the shortest R-R interval were selected for analysis. The calibration signal for 1 cm was clearly defined, and the recording paper was run at a speed of 50 mm/sec. Dimensions were measured as intercepts perpendicular to the main bang on the initial rapid deflection of the QRS complex of the electrocardiogram.

The interventricular septal dimension was measured from the right to the left septal surface. The right interventricular septal
surface was the dominant interface moving synchronously with the left septal surface. This measurement was taken from the most anterior aspect of the line thickness (leading edge). If more than one dominant line was present, the most anterior line was measured. This structure was unacceptable for measurement if the interface moved in opposite direction to the left septal surface (e.g., the tricuspid valve annulus). The left ventricular septal measurement was made at the most posterior aspect of this structure. If multiple lines were present, the most posterior aspect of the posterior line was selected. For adequate measurement, the septum must move posteriorly and increase in thickness during systole.

The left ventricular diastolic dimension (LVDd) was measured from the posterior left septal surface to the anterior aspect of the left ventricular posterior wall (LVPW) endocardium, synchronously with the initial deflection of the QRS of the electrocardiogram. The left ventricular systolic dimension (LVDs) was measured as the intercept perpendicular to the main bang, which intersected the interventricular septum (IVS) and the LVPW. The dimension was taken from the most anterior excursion of the LVPW endocardium to the left septal surface. The LVPW dimension was taken from the LVPW endocardial surface to the epicardial-pericardial interface. The LVPW endocardial surface was defined as the dominant endocardial interface that moved anteriorly during systole and was continuous with the apex of this anterior systolic excursion. Because of the difficulty of identifying clearly the endocardial-myocardial interface, the epicardial-pericardial interface was taken as the dominant posterior interface moving in concert with the endocardial interface. Similar guidelines have been developed for measurement of the aortic and left atrial dimensions.

We calculated the echocardiographic left ventricular mass by the formula:

\[
LV\ Mass\ (g) = 0.77 \times [LVDd + LVPW + IVS] - (LVDd)^2 = 2.4
\]

### Blood Pressure Studies

School children of Muscatine, Iowa have participated in a screening program for coronary risk factors since 1970. Most are white (96.5%). Based on consecutive school screening measurements in the years 1975 and 1977, we identified students whose systolic blood pressure (SBP) as in the high, middle, or low quintile for age. In 1979 the children were in grades 4 to 12 and ranged in age from 9 to 18 years. We selected a 70% stratified random sample of those children whose SBP was initially (1975) in the high (fifth), middle (third), and low (first) quintiles and persisted (1977) in the same quintile. A total of 264 students, 140 males and 124 females, participated.

All studies were performed in a trailer parked next to the school building. Repeat casual blood pressure was measured in the right arm of seated subjects using a mercury sphygmomanometer. Blood pressures at the first, fourth, and fifth Korotkoff phase were recorded. Diastolic blood pressure (DBP) measurements were taken at the fourth Korotkoff phase.

Echocardiograms were performed using our previously described techniques. Additionally, standard lead electrocardiograms were recorded by a Health-tek cart transmitted over telephone lines to a Marquette computer system, housed at the University of Iowa. The measurement matrix of the Bonner program resembled the previous screening blood pressures and continued to divide into three distinct groups.

Echocardiograms were performed using our previously described techniques. Additionally, standard lead electrocardiograms were recorded by a Health-tek cart transmitted over telephone lines to a Marquette computer system, housed at the University of Iowa. The measurement matrix of the Bonner program was printed and used for analysis. The diagnosis generated by the Bonner program was not used because it was not applicable to pediatric subjects. The amplitudes of the standard electrocardiogram were measured both by analyzing matrix voltages and by visual measurement of the electrocardiograms. Duplicate visual measurements were made by technicians blinded to the blood pressure classification, with adjudication of any differences of 5% or greater. The second to last beat in each appropriate lead was measured. The voltage calibration of the electrocardiogram was checked from an external signal box checked regularly with a voltmeter. Repeated tests at the end of the screening showed a response of 1 cm/mV ± 0.3%. The maximal R-wave amplitudes in leads I, II, III, V4, V5, V6; the maximal RS amplitudes in leads I, II, III; the sum of the maximal R-wave amplitudes in V4, V5, and V6 plus maximal S amplitude in V1, V5, and V6; and the maximal S-wave amplitude in V1, V5, and V6; were used as echocardiographic measurements of left ventricular hypertrophy. We obtained correlations between the voltages measured by the computer matrix and those made by visual measurement.

### Results

**Echocardiographic Reproducibility**

Measurements for most left ventricular dimensions, when the criteria for interfaces were utilized, showed extremely high agreement, both within a single observer and between two observers reading the same tracings (Table 1). The reproducibility for all echocardiographic dimensions, when measured by a single observer, was high, with interclass correlation coefficients ranging from 0.87 to 0.98. The between-observer interclass correlation coefficients were also high, ranging from 0.86 to 0.98 with one exception, measurement of the left ventricular posterior wall.

Day-to-day variability within single subjects of echocardiographically determined cardiac dimensions showed close agreement for all measurements (Table 2). Stage two was performed approximately 2 months after the initial between-observer reproducibility study. A noticeable improvement in agreement was achieved for all left ventricular dimensions measured, particularly for the single observer correlation coefficient for the left ventricular posterior wall, which rose from 0.87 to 0.99. In this study the standard deviation of error for all variables was decreased to no more than 0.3 mm.

The lowest within-observer agreement was found in measurement of the left ventricular posterior wall. Agreement between two observers measuring this structure was also poor. One factor possibly responsible for this variance was the thickness of the epicardial-pericardial interface. In Stage three, we evaluated the usefulness of switched gain to reduce the measurement error (Table 3). One reader measured the duplicate tracings. No significant difference was observed between means for gain-on and gain-off readings. Utilizing the switch gain, no significant improvement in measurement error was observed.

**Blood Pressure**

After correction for age and sex, children with the high blood pressure levels were noted to be significantly taller, heavier, and more obese (Table 4) than children in the other groups. The SBP measured at the time of this study with the mercury sphygmomanometer resembled the previous screening blood pressures and continued to divide into three distinct groups.

Left ventricular hypertrophy in the high blood pressure group was documented by an increased echocardiographic left ventricular wall mass (Table 5). Before correction for age, sex, height, weight, and triceps skinfold thickness, the interventricular septum, the left ventricular posterior wall, and left ventricular wall mass were all increased significantly in the high blood pressure group. After correction for the large measurement variability, however, the left ventricular posterior wall mass was no longer significantly increased although the group mean remained larger. After correction, the increases in interventricular septum, left atrium, and left ventricular wall mass persisted in the high blood pressure group. No group differences were observed for the ratio of interventricular septum to left ventricular posterior wall.

The corrected heart rate was higher in the high blood pressure group than in the low blood pressure group (Table 6). No group
differences were observed in the left ventricular prejection period (LVPEP), left ventricular ejection time (LVET), the ratio of left ventricular prejection to ejection time, or in the interval of the electrocardiographic Q wave to mitral valve closure. The echocardiographically derived estimates of stroke volume in cardiac output showed no significant group differences (Table 7).

Because the children in the high blood pressure group were taller, heavier, and more obese, we estimated the proportion of observed differences in the echocardiographic measurements in this group explained by measures of body mass. After correcting for age, sex, and height, 4 to 14% of the remaining variability was explained by measures of body mass. Simple correlations were generally small. In all cases the majority of this contribution was explained by skinfold thickness or Quetelet index. The largest overall multiple correlations observed were for left ventricular posterior wall and left ventricular wall mass, where 11% and 14%, respectively, were explained by skinfold thickness and Quetelet index. In the high blood pressure group there was a small but significant relationship between obesity and left ventricular hypertrophy.

Within the high blood pressure group, calculated cardiac output ranged from 1.0 to 5.0 L/min/m², suggesting a wide range of systemic vascular resistance. We reexamined the relationship of cardiac output to SBP and DBP, holding age, sex, height, weight, and tricep skinfold thickness constant. In the children with blood pressure levels in the highest quintile, the highest DBP was found in those with the lowest cardiac output (partial correlation, \( r = 0.6, p<0.001 \)). Both the left ventricular diastolic dimension (\( r = 0.5, p<0.001 \)) and the left ventricular wall mass (\( r = 0.25, p<0.001 \)) were greatest in those children whose blood pressures were elevated on the basis of increased cardiac output.

The correlation between computer matrix voltage measurements and visual measurements was good (Table 8), verifying the validity of the computer measurements. No ST-segment or T-wave abnormalities were found. We found no significant relationship between the partial correlations of computer-measured electrocardiographic voltages with the echocardiographic measurements of ventricular wall and chamber size while holding age, sex, height, weight, and tricep skinfold thickness constant (Table 9). Also, no electrocardiographic correlations were found with the derived echocardiographic measures of cardiac index or left ventricular wall mass (Table 10).

The sum of the maximal R wave and S wave in standard leads I, II, and III of the computer-measured voltages correlated with SBP (Table 11). Weaker correlations were found with both DBP and heart rate. Although we observed the correlation for an electrocardiographic measure of left ventricular hypertrophy with SBP, this relationship explained less than 3% of the variability. Although body weight explained a large proportion of the variability of SBP (15%), most of the variability (82%) was unexplained.

Significant negative electrocardiographic correlations were observed with weight and tricep skinfold thickness. The weak Spearman rank correlations of the electrocardiogram with SBP contrasts with the stronger negative correlations to skinfold thickness and body weight (Table 12).

### Discussion

Three distinct factors were responsible for achieving the close agreement between two observers for all structures of the left side of the heart. These were recording techniques, screening criteria for acceptability, and measurement guidelines. Screening criteria improved reproducibility by rejecting tracings that were difficult to measure. We insisted that the interface record be dark enough to be seen clearly. We did not recommend a change in electronic enhancement of the signal but rather the proper selection of transducers and gain settings to allow measurement of a dark, thin line. Discontinuity of the line, commonly called hash, can often be minimized by proper use of the damping and reject controls. If it is not possible to record a single continuous line, the agreement between two leaders measuring that interface is poor. Lastly, we required that the interfaces demonstrate the characteristic motion of the cardiac structure imaged.

Restriction of the measurement within areas of maximum excursion of the interventricular septum and left ventricular posterior wall, coupled with the largest internal dimension of the left ventricle during diastole and systole, reduced the selection of beats that were suitable for analysis. The pure error, or the error introduced by measuring line segments with a hand-held instrument, was small. No significant differences in pure error were found when comparing hand-measured line segments to those measured with a sonic digitizer. The calculated precision of measurements for left

### Table 1. Echocardiograms Obtained in 20 Children Aged 6 to 16 Years (11 Boys and 9 Girls), Division of Pediatric Cardiology Graphics Laboratory during 1977-1978

<table>
<thead>
<tr>
<th>Variable</th>
<th>Intraclass correlation</th>
<th>Error (cm)</th>
<th>Precision (cm)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Within observer</td>
<td>Between observers</td>
<td></td>
</tr>
<tr>
<td>IVS</td>
<td>0.95</td>
<td>0.87</td>
<td>0.06</td>
</tr>
<tr>
<td>LVDd</td>
<td>0.96</td>
<td>0.88</td>
<td>0.13</td>
</tr>
<tr>
<td>LVDs</td>
<td>0.95</td>
<td>0.86</td>
<td>0.10</td>
</tr>
<tr>
<td>LV PW</td>
<td>0.87</td>
<td>0.59</td>
<td>0.06</td>
</tr>
<tr>
<td>LVET (n=17)</td>
<td>0.98</td>
<td>0.98</td>
<td>0.01</td>
</tr>
<tr>
<td>LAD</td>
<td>0.98</td>
<td>0.98</td>
<td>0.06</td>
</tr>
<tr>
<td>AO</td>
<td>0.96</td>
<td>0.96</td>
<td>0.05</td>
</tr>
</tbody>
</table>

IVS = interventricular septum; LVDd = left ventricular diastolic dimension; LVDs = left ventricular systolic dimension; LV PW = left ventricular ejection time; LAD = left atrial dimension; AO = aortic root.

*Square root of estimated error variance.
†Square root of the sum of estimated variances for error and observer of subject interaction.

Adapted from Schieken et al.²

### Table 2. Echocardiographic Studies of 10 Children

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean ± SD (cm)</th>
<th>Intraclass correlation coefficient</th>
<th>Precision (cm)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Day 1</td>
<td>Day 2</td>
<td></td>
</tr>
<tr>
<td>IVS</td>
<td>0.66±0.15</td>
<td>0.68±0.15</td>
<td>0.98</td>
</tr>
<tr>
<td>LVDd</td>
<td>3.19±0.33</td>
<td>3.20±0.34</td>
<td>0.99</td>
</tr>
<tr>
<td>LVDs</td>
<td>2.01±0.34</td>
<td>2.00±0.34</td>
<td>0.99</td>
</tr>
<tr>
<td>LV PW</td>
<td>0.80±0.22</td>
<td>0.79±0.21</td>
<td>0.99</td>
</tr>
</tbody>
</table>

Measurements were made at 1400, 24 hours apart. Abbreviations as in Table 1.

Adapted from Schieken et al.²

### Table 3. Echocardiographic Studies of 12 Additional Children

<table>
<thead>
<tr>
<th>Gain on</th>
<th>Gain off</th>
<th>F*</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean (n=11)†</td>
<td>0.90</td>
<td>0.89</td>
<td>1.16</td>
</tr>
<tr>
<td>Measurement error‡</td>
<td>0.03</td>
<td>0.04</td>
<td>1.25</td>
</tr>
</tbody>
</table>

Echocardiograms obtained approximately 6 months after the 20 subjects shown in Table 1.

*F statistic from analysis of variance.
†Tracings of 11 of 12 children met initial screening criteria.
‡Square root of pooled error variance.

Adapted from Schieken et al.²
### Table 4. Comparison of Baseline Data in Subjects from the Low, Middle, and High Quintiles of Blood Pressure

<table>
<thead>
<tr>
<th>Variable</th>
<th>Uncorrected means</th>
<th>Corrected means*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>F&lt;sup&gt;1&lt;/sup&gt; (p&lt;)</td>
<td>SD</td>
</tr>
<tr>
<td>Age</td>
<td>1.20</td>
<td>2.61</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>1.05</td>
<td>14.27</td>
</tr>
<tr>
<td>Weight (lbs)</td>
<td>11.33</td>
<td>34.65</td>
</tr>
<tr>
<td>Triceps skinfold thickness</td>
<td>17.71</td>
<td>5.54</td>
</tr>
<tr>
<td>Ponderal index</td>
<td>27.80</td>
<td>0.65</td>
</tr>
<tr>
<td>Body surface area (m&lt;sup&gt;2&lt;/sup&gt;)</td>
<td>5258</td>
<td>0.01</td>
</tr>
</tbody>
</table>

**SD = pooled standard deviation from analysis of variance. Brackets indicate no significant differences.**

*Corrected for age and sex.
†F from comparison of three group means (analysis of variance).

Adapted from Schieken et al.

### Table 5. Comparison of Echocardiographic Dimensions of Left Side of Heart in Subjects in Low, Middle and High Quintiles of Blood Pressure

<table>
<thead>
<tr>
<th>Variable</th>
<th>Uncorrected means</th>
<th>Corrected means*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>F&lt;sup&gt;†&lt;/sup&gt; (p&lt;)</td>
<td>SD</td>
</tr>
<tr>
<td>LVDd (cm)</td>
<td>1.41</td>
<td>0.47</td>
</tr>
<tr>
<td>LVDs (cm)</td>
<td>3.80</td>
<td>0.02</td>
</tr>
<tr>
<td>LVFW (cm)</td>
<td>1.12</td>
<td>0.03</td>
</tr>
<tr>
<td>IVS (cm)</td>
<td>11.12</td>
<td>0.38</td>
</tr>
<tr>
<td>Aorta (cm)</td>
<td>0.11</td>
<td>0.34</td>
</tr>
<tr>
<td>LV wall mass (g)</td>
<td>5.62</td>
<td>39.9</td>
</tr>
</tbody>
</table>

**SD = pooled standard deviation from analysis of variance. Brackets indicate no significant differences.**

*Corrected for age, sex, height, weight, and triceps skinfold thickness.
†F from comparison of three group means (analysis of variance).

Adapted from Schieken et al.

### Table 6. Comparison of Systolic Time Intervals in Subjects from the Low, Middle, and High Quintiles of Blood Pressure

<table>
<thead>
<tr>
<th>Variable</th>
<th>Uncorrected means</th>
<th>Corrected means*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>F&lt;sup&gt;†&lt;/sup&gt; (p&lt;)</td>
<td>SD</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>3.18</td>
<td>13.2</td>
</tr>
<tr>
<td>LVET (sec)</td>
<td>0.62</td>
<td>0.025</td>
</tr>
<tr>
<td>LVPEP (sec)</td>
<td>0.86</td>
<td>0.0157</td>
</tr>
<tr>
<td>QMVC (sec)</td>
<td>2.20</td>
<td>0.0113</td>
</tr>
<tr>
<td>LPEP/LVET</td>
<td>0.39</td>
<td>0.06</td>
</tr>
</tbody>
</table>

**SD = pooled standard deviation from analysis of variance. Brackets indicate no significant differences.**

LVPEP = left ventricular preejection period; QMVC = Q wave to mitral valve closure. Other abbreviations as in Table 1.

*Corrected for age, sex, height, weight, and triceps skinfold thickness.
†F from comparison of three group means (analysis of variance).

Adapted from Schieken et al.
ventricular structures was 0.3 mm. This source of error did not significantly influence reproducibility.

In 1978, the American Society of Echocardiography published guidelines for the measurement of echocardiograms of both adults and children. These measurement techniques, when applied to technically satisfactory tracings, should provide a uniformity sufficient to achieve reproducible echocardiograms. Nonetheless, epidemiologic studies require each echocardiographic laboratory to document the variability of their measurements and the reproducibility of their echocardiographic dimensions.

Two results emerged from the blood pressure study: 1) left ventricular wall mass was significantly larger than expected for age and body size in children in the upper quintile of blood pressure, and 2) cardiac output values were widely distributed in children having persistently elevated blood pressure. Children whose blood pressure was elevated on the basis of high cardiac output tended to have larger increases in left ventricular diastolic dimension and left ventricular wall mass than those with low
cardiac output. Children whose blood pressure was at the upper part of the distribution were significantly taller and heavier and had thicker tricep skinfolds. Other investigators have recognized the strong association of obesity and hypertension in children. Our data corroborate these findings.

An important unanswered question is whether children with elevated blood pressure and high cardiac output or elevated systemic vascular resistance are more likely to develop fixed hypertension as adults. School children with persistently elevated blood pressure from elevated cardiac output may be in a so-called hyperkinetic phase. These children with elevated cardiac output tend to have the largest left ventricular wall mass. These observations are consistent with the experimental observations in young spontaneously hypertensive rats with left ventricular hypertrophy. Whether these children may normalize their cardiac output, increase their systemic vascular resistance, and further increase their left ventricular wall mass is not known.

Other studies using M-mode echocardiography have confirmed the findings of left ventricular hypertrophy in children with persistently elevated blood pressure. Zahka et al. not only found greater interventricular septal and posterior wall thicknesses in hypertensive children but also found increases in radius-to-wall thickness ratios, cross-sectional muscle area, and left ventricular mass. Johnson et al. studying high school children with persistently elevated blood pressure, found a correlation between left ventricular wall mass and blood pressure in children in the low and intermediate ranges of SBP, but after correction for body size differences they failed to demonstrate increased wall mass in the high blood pressure group.

When anatomic left ventricular hypertrophy is marked, electrocardiographic and echocardiographic left ventricular measurements are related. Patients with electrocardiographic evidence of left ventricular hypertrophy frequently have echocardiographic evidence of concentric hypertrophy. When the electrocardiographic voltage measurements are correlated with the left ventricular mass derived from echocardiographic measurements, however, the relationship is weak. In normal children, the electrocardiographic voltage measurements relate poorly to blood pressure level. Confounding the relationship between an increase in precordial voltages and increasing heart size is the negative correlation of precordial voltages with both body size and body fat. The echocardiogram, a more direct measure of chamber size and heart wall thickness, is not influenced by body fat and detects increases in left ventricular mass even after correction for body size differences in children with high blood pressure.

The significance of increased left ventricular wall mass in children in the upper quintile of blood pressure is not clear. Whether it is a separate event in the hypertensive process or related to increased work of the heart is not known. Longitudinal echocardiographic studies in children may provide useful insights above the relationship of left ventricular wall mass and the development of hypertension.

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