Electrocardiographic Measures of Left Ventricular Hypertrophy in Children Across the Distribution of Blood Pressure: The Muscatine Study

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SUMMARY We sought to test the effectiveness of the ECG as a measure of increased left ventricular wall mass in children with high blood pressure. One hundred eighty-one children, ages 9–18 years, were selected from the lowest, middle and highest quintile of systolic blood pressure from the Muscatine Study, based upon two biennial school screenings. After correction for age, sex, height, weight and skinfold thickness, children with the highest blood pressure had increased echocardiographic left ventricular wall mass (p < 0.02). Voltage measurements of maximum R and S waves in the standard and precordial leads were measured by computer. We correlated blood pressure and echocardiographic measurements of the interventricular septum, left ventricular posterior wall and left ventricular wall mass to echocardiographic combinations used to predict left ventricular hypertrophy in both children and adults. The electrocardiographic correlations ranged from -0.01 to +0.17. Poor correlations were found between electrocardiographic measures and blood pressure, left ventricular wall thickness or left ventricular wall mass. Skinfold thickness and weight had negative correlations, suggesting a damping effect upon measured voltage. We conclude that the echocardiogram is a more sensitive measurement of increased left ventricular mass than the ECG in children with elevated blood pressure.

ADULTS at high risk of cardiovascular disease can be identified by measuring serum cholesterol, blood pressure, smoking history, glucose tolerance and the presence or absence of electrocardiographic left ventricular hypertrophy.1 Electrocardiographic evidence of left ventricular hypertrophy in adults is predictive of coronary heart disease.2 Children with persistently increased systolic blood pressure have significantly greater left ventricular wall mass by echocardiography after correction for body size than those with pressures in the middle or lower range.3 This study was undertaken to determine if the ECG could also detect a difference in left ventricular wall mass present between children with high-, middle- or low-range blood pressure.

Methods

Population
Since 1970, the school children of Muscatine, Iowa, have participated in a screening program for coronary risk factors. Most of the children in the study (96.4%) are white. Based on consecutive school screening measurements in the years 1975 and 1977, we identified students whose systolic blood pressure was in the low (first), middle (third) or high (fifth) quintile for age. In
1979, the children were 9–18 years old. We selected a 70% stratified random sample of children whose systolic blood pressure was initially (in 1975) in the low, middle and high quintiles and remained (until 1977) in the same quintile (Table 1). The three groups were not significantly different in age and height. However, the subjects in the high-blood-pressure group weighed significantly more and had larger skinfold thicknesses ($p < 0.01$).

All studies were performed in a trailer parked next to the school. The school authorities assisted in selecting the time of study for each child. After obtaining both the parent’s and the child’s consent, we measured height, weight and triceps skinfold thickness (a measure of obesity) and right arm casual blood pressure according to a previously described protocol.4

Standard 12-lead ECGs were recorded by a Healthtek cart transmitting 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 analyses. The diagnosis generated by the Bonner program was not used because of its inapplicability to pediatric subjects. The amplitudes of the standard ECG were measured both by analyzing the matrix voltages and by visual measurement of ECGs. 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 ECG was checked from an external signal box checked regularly with a voltmeter. Repeated tests at the end of screening showed a response of 1 cm/mV $\pm$ 0.3%.

The maximal R-wave amplitudes in leads I, II, III, V4, V5, and V6; the maximal R + S amplitudes in leads I, II, III; the sum of the maximal R-wave amplitude in V4, V5, and V6 plus maximal S amplitude in V1, V3, and V6; and the maximal S-wave amplitude in V1, V3, and V5 were used as electrocardiographic measurements of left ventricular hypertrophy. We obtained correlations between the voltages measured by the computer matrix and those made by visual measurement.

With the patient recumbent, echocardiograms were performed using an SKI ultrasonoscope 20-A with a 3.5-MHz transducer and a Honeywell 1856 strip-chart recorder. The standard interspace technique for transducer position was used.5

### Statistical Analysis

Pearson correlation coefficients were used to examine the association of the electrocardiographic and echocardiographic measurements of left ventricular hypertrophy. Adjustments were made using partial correlations, holding age, sex, height, weight and triceps skinfold thickness constant.

The following dimensions were measured as intercepts perpendicular to the transducer artifact on the initial rapid deflection of the QRS complex of the ECG: interventricular septum (IVS), left ventricular diastolic dimension (LVDd), left ventricular systolic dimension (LVDs) and left ventricular posterior wall (LVPW).

The left ventricular diastolic and systolic volumes (LVDV and LVSV) were calculated from the echocardiographic left ventricular end-diastolic and end-systolic dimensions (LVDD and LVDs) by the method of Meyer et al.:6

$$\begin{align*}
\text{LVDV} &= 19.1 + 14.6 \text{LVDd} + 0.62 \text{LVDd}^3 \\
\text{LVSV} &= 19.1 + 14.6 \text{LVDs} + 0.62 \text{LVDs}^3
\end{align*}$$

The echocardiographic stroke volume was calculated by the difference between diastolic and systolic volumes. The cardiac output was calculated as stroke volume $\times$ the heart rate, and the cardiac index was determined by dividing by the body surface area.

We calculated the echocardiographic left ventricular mass by the formula7

$$0.77 \{\text{LVDd} + \text{LVVPW} + \text{IVS}\} - \text{LVDd}^3 + 2.4.$$ 

We have reported the reproducibility of the left-heart echocardiographic dimensions.8 Only echocardiographic tracings that met our measurement criteria for group echocardiographic studies were analyzed.

### Results

The echocardiographic left ventricular heart dimensions after age, sex and body size corrections showed larger interventricular septal measurements in the high blood pressure group (Table 2). Although the left ventricular posterior wall was larger in the high group,
large variability prevented a statistical separation of the group means. However, the left ventricular wall mass was significantly greater in the high group.

The correlation between the computer matrix voltage measurements and the visual measurements was good (table 3), 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 measures of ventricular wall and chamber size while holding age, sex, height, weight and triceps skinfold thickness constant (table 4). Also, no electrocardiographic correlations were found with the derived echocardiographic measures of cardiac index or left ventricular wall mass (table 5).

The sum of the maximal R wave and S wave in standard leads I, II and III of the computer-measured voltages correlated with systolic blood pressure (table 6). Weaker correlations were found with both diastolic blood pressure and heart rate. Although we observed a correlation for an electrocardiographic measure of left ventricular hypertrophy with systolic blood pressure, this relationship explained less than 3% of the variability. Although body weight explained a large portion of the variability of systolic blood pressure (15%), most of the variability (82%) was unexplained.

Significant negative electrocardiographic correlations were observed with weight and triceps skinfold thickness. The weak Spearman rank correlations of the ECG to systolic blood pressures contrast with the stronger negative correlations to skinfold thickness and body weight (table 7).

**Discussion**

Electrocardiographic voltage amplitudes did not correlate strongly with increases in systolic blood pressure within the range found in normal children. In the Evans County study,7 electrocardiographic voltage criteria for left ventricular hypertrophy were not present in more young adult hypertensives than normotensive controls. In adults with longstanding hypertension, the Framingham Study documented that patients with electrocardiographic left ventricular hypertrophy have 10 times the risk of developing congestive heart failure.10 Thus, in adults, electrocardiographic left ventricular hypertrophy appears to be a late, but important, predictor of severe disease.

In this study, we have shown that as children grow older and develop increasing body size, their electrocardiographic precordial voltages decrease. Walker et al.,11 who made a similar observation in children and adolescents, pointed out the importance of developing age-adjusted electrocardiographic criteria for left ventricular hypertrophy. Moreover, body fat reduces the amplitude of voltages over the precordium.12 This observation is consistent with our findings of the negative correlation of electrocardiographic precordial voltages with triceps skinfold thickness.

In adults, increased electrocardiographic voltages are used as clinical evidence of left ventricular hyper-
trichrome. Anatomic validation of these electrocardiographic measures have both confirmed\textsuperscript{13, 14} and questioned\textsuperscript{15} the value of the ECG in detecting left ventricular hypertrophy. A commonly used electrocardiographic estimate of left ventricular hypertrophy is the arithmetic sum of the depth of the S wave in a right precordial lead and the height of an R wave in a left precordial lead.\textsuperscript{16}

The echocardiogram provides a more direct measure of the size of the left heart than the ECG. The echocardiographic measures of left ventricular wall thickness and internal chamber dimensions are similar to angiographic measures.\textsuperscript{17, 18} Anatomic validation has verified that echocardiographic measures can be used to estimate left ventricular wall mass.\textsuperscript{19}

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.\textsuperscript{20} However, when electrocardiographic voltage measurements are correlated with the left ventricular mass derived from echocardiographic measures, the relationship is weak.\textsuperscript{21}

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.\textsuperscript{3}

The ECG and the echocardiogram measure different phenomena. The ECG measures an electrical voltage and the echocardiogram measures ultrasonic left ventricular dimensions. Thus, it is not surprising that they do not correlate across the distribution of left ventricular wall mass. Despite this poor correlation, electrocardiographic evidence of left ventricular hypertrophy is consistently observed in those with severe echocardiographic concentric hypertrophy.\textsuperscript{20} However, in milder forms of increased echocardiographically measured left ventricular wall mass, the ECG does not appear abnormal.

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. In the spontaneously hypertensive rat, Cutilletta et al.\textsuperscript{22} showed that increased left ventricular wall mass results independently of the development of the hypertensive process. Longitudinal echocardiographic studies in children may provide useful insights about the relationship of left ventricular wall mass and the development of hypertension.

References

Comparison of Techniques for Measuring Baroreflex Sensitivity in Man

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SUMMARY Because discrepancies about baroreflex sensitivity in essential hypertension may have resulted from the use of different measurement techniques, we assessed the extent to which the results of different techniques agree in the same subjects. The eight techniques studied were the change in RR interval per unit change in systolic pressure during the Valsalva maneuver, upon release of the Valsalva maneuver, after injection of phenylephrine and after injection of nitroglycerin; the changes in RR interval and in systolic pressure per mm Hg externally applied neck suction; and the changes in RR interval and systolic pressure per mm Hg externally applied neck pressure.

The average intercorrelation among these measures in 30 subjects was statistically significant (r = 0.36, p < 0.01), but suggests that variance in one measure accounted for an average of about 13% of the variance in other measures. Standard deviations among subjects were often as large as the mean, indicating important interindividual variability as well.

These findings demonstrate that baroreflex sensitivity varies widely among subjects and that different techniques for measuring baroreflex sensitivity probably measure different aspects of baroreflex function.

THE POSSIBLE ROLE of altered baroreflex function in producing or maintaining high blood pressure in man has been controversial for many years.1 When the extent of the bradycardiac response to vasoconstrictor-induced increases in blood pressure has been used as an index of baroreflex sensitivity, patients with essential hypertension have shown decreased baroreflex sensitivity compared with normotensive controls.2,3 Similar results have been obtained when the extent of bradycardia after release of the Valsalva maneuver is used to measure baroreflex sensitivity.4,5 When heart rate or blood pressure responses to externally applied neck pressure or suction have been used as an index of baroreflex sensitivity, investigators have disagreed about whether hypertensives show abnormal baroreflex function.6-8

No study has dealt with the extent to which these

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