Echocardiographic Diagnosis of Left Ventricular Hypertrophy

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SUMMARY  Echocardiograms were obtained on 27 adults with electrocardiographic criteria of left ventricular hypertrophy (LVH) to determine how echocardiograms might best identify LVH. Both the left ventricular (LV) posterior wall thickness and interventricular septal thickness were found by echocardiography to be increased (≥ 12 mm) in only 13 of 27 patients (48%) with LVH. The LV was dilated (≥ 58 mm) in the absence of posterior wall thickening in 9 of 27 patients (33%). The LV mass, estimated from standardly measured dimensions, was increased (> 200 g) in 21 of 27 patients (78%) and when measurements were made by the Penn method, mass was increased in all patients. These observations indicate that the echocardiographic estimation of LV mass is a more sensitive indicator of LVH than LV posterior wall and septal thickness. Since LVH is defined as an increased mass of LV muscle, these observations are consistent with this fundamental definition of left ventricular hypertrophy.

CARDIAC HYPERTROPHY denotes an increase in the mass of the heart associated with an enlargement of the individual muscle fibers. In general, the heart responds to an increased afterload by an increase in wall thickness of the chamber, and this is referred to as "concentric hypertrophy." The ventricle usually responds to an increased preload by dilatation of the chamber, and this is referred to as "eccentric hypertrophy." The normal left ventricle of an adult male has been found to weigh 203 g or less. In females it weighs 141 g or less. Similar results were found utilizing a calculated value of left ventricular mass from measurements made by angiocardiography with their observed upper limit of normal for adult males being 221 g. The accuracy of the angiocardiographic calculations has been confirmed by autopsy correlation. Echocardiographic measurements of left ventricular wall thickness and internal dimensions have been shown to be similar to measurements made by left ventriculography.

Previous investigators have concluded that increases of both septal and left ventricular wall thickness are the primary echocardiographic correlates of left ventricular hypertrophy. However, since left ventricular hypertrophy indicates that the mass of the left ventricle has increased, it would seem that a direct estimation of left ventricular mass by echocardiography would be a more meaningful approach to the determination of cardiac hypertrophy. From equations which permit one to estimate left ventricular mass from the echocardiogram, it is apparent that mass is related to ventricular wall thickness and the internal dimension of the ventricle. The purpose of this study is to compare the sensitivity of measurements of ventricular wall thickness to the more direct estimation of left ventricular mass in order to determine the relative merits of each as an indicator of left ventricular hypertrophy.

Methods

Echocardiograms of 27 patients (22 male and five female) with hypertensive and/or valvular cardiac disease were studied. Each patient had electrocardiographic evidence of left ventricular hypertrophy based upon the maximal voltage of S1, or S2 plus the maximal voltage of Rv6 or Rv5 > 3.5 mV. Twenty-three of these patients showed an abnormal (> 50%) cardiothoracic ratio on chest roentgenograms. Sixteen patients with electrocardiographic evidence of hypertrophy had an S1 or S4 gallop, left ventricular thrust, or a laterally displaced ventricle on physical examination. Twenty-six patients had evidence of cardiac enlargement on chest roentgenograms or physical examination. The ages and diagnoses of these 27 patients are shown in Table 1. Echocardiograms were also obtained on 20 healthy subjects (19 male, one female) who served as controls. These subjects ranged in age between 25 and 47 years, the mean and standard error being 31 ± 1 years. All control subjects had normal chest roentgenograms (transverse diameter less than 14.5 cm and cardiothoracic ratios less than 50%). None had a gallop, ventricular thrust, or a laterally displaced cardiac impulse. All of the normal subjects showed normal ST segments and T waves on the electrocardiogram, and the voltage of the S in V1 or V2 plus R in V5 or V6 did not exceed 3.5 mV.

The electrocardiograms were analyzed for voltage and the presence of a strain pattern, and were also assessed by the point-score system for left ventricular hypertrophy suggested by Romhilt and Estes. A value of 5 points was found to be indicative of left ventricular hypertrophy and a score of 4 points indicated probable hypertrophy. The point-score for left ventricular hypertrophy was 5 or greater in 20 of the 27 patients in the hypertrophy group indicating a high probability of the presence of left ventricular hypertrophy in these patients. The score was 4 in four of the remaining seven patients (Table 1). Twenty-six of the 27 patients had a strain pattern. None of the normal subjects had a point-score greater than 1, indicating with high probability that they had no hypertrophy.

Echocardiograms were obtained with a Picker Echoview 19 using a 2.25 MHz transducer. Tracings were recorded on a Honeywell Visicorder, Model 1856, at a paper speed of 50 mm/sec with the patients in the left lateral position, between 20 and 45 degrees. The transducer was placed at the third to fifth left intercostal space and sweeps were made from the aortic root to the mitral valve and toward the apex of the left ventricle. The end-diastolic left ventricular posterior wall thickness (LVPWd), interventricular septal thickness (IVSd), and left ventricular internal dimension (LVIDd)
were measured at the level of the chordae of the mitral valve at the peak of the R wave of the electrocardiogram using standard methods (fig. 1). The standard measurement convention includes the thickness of the right and left septal endocardial echoes in the IVS₉ and includes the posterior wall endocardial echoes in the LVPW₉.

Normal left ventricular end-diastolic dimensions were considered to be the following: Left ventricular posterior wall (LVPW₉) = 6-11 mm; interventricular septal thickness (IVS₉) = 6-11 mm; left ventricular internal dimension (LVID₉) = 35-57 mm. Asymmetric septal hypertrophy (ASH) was defined as a ratio of the septal thickness to the posterior left ventricular wall thickness greater than 1.3.

Left ventricular mass was estimated from measurements using the standard convention as follows:

Mass (g) = 0.77 × 10⁻³ [(LVID₉ + LVPW₉ + IVS₉)³ - (LVID₉)³] + 2.4

where the dimensions are given in millimeters.

Left ventricular mass was also estimated from measurements using the Penn convention (fig. 1). Measurements with the Penn convention excluded the right and left septal endocardial echo thickness from the IVS₉ and this convention excludes the posterior wall endocardial echo thickness from the LVPW₉. Left septal endocardial echo thickness and posterior wall endocardial echo thickness are thus included in the LVID₉ by this method. Using the Penn convention, left ventricular mass is estimated as:

\[
\text{Mass (g)} = 1.04 \times 10^{-3} \left[ (\text{LVID}_d + \text{LVPW}_d + \text{IVS}_d)^3 - (\text{LVID}_d)^3 \right] - 13.6
\]

where the dimensions are given in millimeters.

These equations permit an estimate of left ventricular mass by assuming that the ventricle is ellipsoidal during end-diastole. The internal volume of the ventricle is subtracted from the external volume, which gives the volume of the ventricular muscle. Mass is estimated from the specific gravity of ventricular muscle, which is assumed to be 1.05 g/cm³.

**Results**

Among the patients with left ventricular hypertrophy, using standard methods for measurement, only 13 of 27 (48%) had both an increased posterior wall thickness and an increased interventricular septal thickness (table 2, fig. 2). Left ventricular dilatation, in the absence of thickening of the posterior wall, was present in 9 of 27 patients (33%). None of the normal subjects had enlargement of the posterior wall, interventricular septum, or left ventricular internal dimension (table 3). Left ventricular mass, estimated on the basis of measurements made by the standard convention, was increased (> 203 g) in 21 of 27 patients (78%) with left ventricular hypertrophy. Estimated left ventricular mass in patients with left ventricular hypertrophy (300 ± 20 g) (mean ± sem) in each instance exceeded the left ventricular mass estimated in control subjects (130 ± 4 g) (P < 0.001). None of the control subjects showed an elevated mass (table 3, fig. 2).
The left ventricular mass, estimated from measurements made with the Penn convention, was increased in all patients with left ventricular hypertrophy (370 ± 20 g) and the estimated mass was normal (150 ± 5 g) in all of the control subjects (P < 0.001) (fig. 3).

The voltage of the electrocardiogram (Sv1 or Sv2 + Rv5 or Rv6) and the estimated left ventricular mass, calculated by standard methods, showed an approximate correlation (r = 0.71) (fig. 4). The left ventricular posterior wall thickness correlated less closely with the combined voltages (r = 0.65). The left ventricular internal dimension showed a weak association with the combined voltages (r = 0.39). The left ventricular posterior wall thickness correlated approximately with left ventricular mass (r = 0.77) (fig. 5).

**Discussion**

The heart responds to an increased pressure load or increased volume load with an increased mass of muscle, which is recognized as cardiac hypertrophy. The mass of the myocardium increases through an enlargement of the individual muscle fibers. Since left ventricular hypertrophy implies an increase in left ventricular mass through an increased wall thickness and/or an increased internal dimension of the ventricle, it is likely that an improved determination of hypertrophy could be obtained by a direct estimation of left ventricular mass from echocardiographic measurements. Our data indicate that the assessment of hypertrophy using this concept rather than a measurement of wall thickness is the more accurate method.

The relation between the electrocardiographic voltage in
TABLE 3. Echocardiographic Measurements in Control Subjects (Standard Method)

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<tr>
<th>Patient number</th>
<th>LVIDd (mm)</th>
<th>LVPWd (mm)</th>
<th>IVSd (mm)</th>
<th>LVIDd/LVPWd</th>
<th>Calculated LV mass (g)</th>
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<td>8 ± .2</td>
<td>1.0 ± .02</td>
<td>130 ± 4</td>
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FIGURE 3. Left ventricular mass, estimated from measurements by the Penn convention, in patients with left ventricular hypertrophy (LVH) and normal subjects.

FIGURE 4. Relation between voltage on the electrocardiogram of $S_v$ or $S_v + R_v$ or $R_v$ and the left ventricular (LV) mass, estimated from measurements made by the standard method. The correlation coefficient and regression equation are shown.

FIGURE 5. Relation between left ventricular (LV) posterior wall thickness and left ventricular mass, estimated from measurements made by the standard method. The regression equation and correlation coefficient are shown.

left ventricular hypertrophy and left ventricular mass has been studied previously and diverse results have been reported. Bennett and Evans found a correlation between the electrocardiographic voltage ($S_v + R_v$ or $R_v$) and the left ventricular mass estimated from the echocardiogram; whereas Baxley and associates found a poor correlation between these same electrocardiographic voltages and left ventricular mass estimated from the ventriculogram. The data from this study showed an approximate correlation between $S_v$, $S_v + R_v$ or $R_v$ and the left
ventricular mass estimated by standard measurements from the echocardiogram. Only 48% of the patients with hypertrophy had a thickened left ventricular posterior wall. Others have shown that 75% of patients with left ventricular hypertrophy have thickening of the posterior left ventricular wall. Their conclusion was that the primary echocardiographic correlate of left ventricular hypertrophy was an increase in both septal and left ventricular posterior wall thickness. The significance of our study is that a greater percentage of patients with left ventricular hypertrophy can be identified by echocardiography if an estimation of left ventricular mass is used. The primary echocardiographic correlate of left ventricular hypertrophy is not an increase of wall thickness, but rather an increase of estimated left ventricular mass.

In summary, the data from this study show that left ventricular mass, estimated from the echocardiogram, is a better indicator of left ventricular hypertrophy than echocardiographic measurements of left ventricular posterior wall thickness and interventricular septal thickness.

References
15. Romhilt DW, Estes EH: A point-score system for the ECG diagnosis of left ventricular hypertrophy. Am Heart J 75: 752, 1968
18. Bennett DH, Evans DW: Correlation of left ventricular mass determined by echocardiography with vectorcardiographic and electrocardiographic voltage measurements. Br Heart J 36: 981, 1974

Echocardiographic Differentiation of Partial and Complete Atrioventricular Canal

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SUMMARY Retrospective examination of echocardiograms was performed in 34 patients with persistent atrioventricular (A-V) canal who had undergone cardiac catheterization. Characteristic findings in 16 patients with partial A-V canal were lack of continuity of mitral and tricuspid valves, paradoxical interventricular septal motion, definite E and A waves of the mitral valve anterior leaflet (MVAL) echoes and late systolic anterior motion of the mitral valve. Eighteen patients with persistent A-V canal had an interventricular communication and were classified as having the complete form. They characteristically had continuous mitral and tricuspid valves, normal interventricular septal motion, disorganized MVAL echoes, and normal systolic mitral valve motion.

Combining these findings allowed differentiation of 31 of the 34 patients (91%) as having partial or complete A-V canal. Determining the presence of a VSD from loss of interventricular septal echoes was unsatisfactory. The severity of mitral insufficiency could not be estimated from echocardiograms of our patients with partial A-V canal. The deficiency of the interventricular septum. Before echocardiography, noninvasive differentiation of the structures involved depended on findings on physical examination, electrocardiogram and chest X-ray. Estimation of mitral insufficiency, degree of deficiency of interventricular septum, and ventricular level shunts has required cardiac catheterization and angiography. Complete and accurate noninvasive evaluation of patients with persistent A-V canal defects may decrease patient morbidity and expense by improving the accuracy of precatheterization diagnosis, the

PERSISTENT ATRIOVENTRICAL (A-V) CANAL represents a broad anatomic spectrum including primum atrial septal defect (ASD), deficiency of septal leaflet of the tricuspid valve and anterior leaflet of the mitral valve, and
Echocardiographic diagnosis of left ventricular hypertrophy.
T M McFarland, M Alam, S Goldstein, S D Pickard and P D Stein

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