Physiologic Determinants of the Electrocardiographic Diagnosis of Left Ventricular Hypertrophy

Elliott M. Antman, M.D., Laurence H. Green, M.D., and William Grossman, M.D.

SUMMARY The relative importance of left ventricular (LV) geometry, wall thickness, and mass on the inscription of left ventricular hypertrophy (LVH) on the ECG was examined in 93 patients; 36 had no LVH on the ECG, 10 had borderline voltage for LVH, and 47 had LVH on the ECG. LV cineangiograms in the right anterior oblique projection were analyzed for LV end-diastolic volume, free wall thickness (h), semiminor radius (R), LV mass index (LVMI), and the geometric relations described by h/R, mass/volume, and h x R. Although mean LVMI was greater in patients whose ECG voltage was either borderline or diagnostic of LVH, increased LVMI also occurred without LVH on the ECG. None of the variables (h, R, h/R or mass/volume) reliably indicated when LVH would be inscribed on the ECG. The product of h x R, however, defined three electrocardiographic groups; all patients with LVH on their ECG had h x R > 2.6 cm². Wall thickening sufficient to result in an increased LV mass did not result in LVH on the ECG unless sufficient concurrent chamber dilatation was present. Thus, a critical geometric relationship resulting from the interplay of wall thickness and chamber dilatation is necessary for LVH to appear on the ECG. This finding is consistent with the solid-angle theory of electrocardiography as it relates to ventricular hypertrophy.
myocardial, valvular or coronary artery disease or systemic hypertension by diagnostic cardiac catheterization. The indication for catheterization in the control group was evaluation of a chest pain syndrome. Patients were included in the study if: 1) there was no concomitant coronary artery disease (>50% luminal narrowing in a major coronary artery) or ventricular dysnergy on ventriculography; 2) they were older than 35 years of age; 3) atrial fibrillation, right ventricular hypertrophy, right bundle branch block, left bundle branch block, or the preexcitation syndrome were not present on the ECG at the time of the study; 4) high-quality LV cineangiograms adequate for analysis of chamber and free wall dimensions were available; and 5) they were not obese (>20% above ideal body weight for their height).10

As part of routine diagnostic left-heart catheterization, all patients underwent left ventriculography in the right anterior oblique projection. LVEDV was obtained by planimetry of an end-diastolic silhouette using a sonic digitizer interfaced with a small computer, a calibrated grid system to correct for magnification, and the area-length formula for a prolate ellipsoid.20 LVEDVs were corrected according to a regression equation derived in our laboratory21 and then indexed for body surface area (LVEDVI). LV wall thickness (h) was determined by the method described by Kennedy et al.22 In brief, from the right anterior oblique end-diastolic silhouette, an average LV free wall thickness was calculated as the length of a perpendicular line drawn between the chamber silhouette and the outer edge of the free wall trace at a point approximately midway between the apex and base, so as to avoid the right ventricular outflow tract. Values were adjusted by means of the same magnification correction factor used in the volume calculations. The corrected wall thickness was added to the semidiameter used in calculating the chamber volume and thereby the volume of the chamber plus muscle wall was obtained. LVM was then calculated as: 1.050 (Vc + w - Vc), where Vc + w is the volume of the chamber plus the wall and Vc is the volume of the chamber, and the values were indexed for body surface area (LVMI).22

Because electrocardiographic voltage abnormalities are easily and accurately measured, we defined LVH as being present on the ECG when any of the following criteria were met (with standard instrument sensitivity 10 mm = 1 mV): R1 + S11 >25 mm28 (with the requirement that S11 was greater than 15 mm when left anterior hemiblock was present24); R4 >13 mm,26 R4 >20 mm,25 S1 + R5 or R6 >35 mm,29 or R5 or R6 >26 mm.29 ECGs were designated borderline for LVH if the above frontal plane criteria were absent, with S1 + R5 or R6 ≥30 mm,27 but ≤34 mm. ECGs were considered negative for LVH if the above frontal plane criteria were absent, with S1 + R5 or R6 <30 mm. Because of the limited sensitivity of intrinscoid delay8 and the limited specificity of left-axis deviation24,28 or ST-T wave abnormalities,19 we did not include these criteria for LVH in our analysis.

In order to assess the relative roles of LV geometry and wall thickness in the inscription of LVH on the ECG, the following calculations were made for each patient: LVMI, LVEDVI, LV free wall thickness (h), the semimajor radius of the left ventricle in the right anterior oblique projection (R), as well as LVMI/LVEDVI, h/R, and h + R.

Statistical analysis of the data included the t test for independent variables and linear regression analysis by the least squares method.

Results

Clinical and ventriculographic data for each patient are shown in table 1, and measured voltages in the leads used to diagnose LVH are shown in table 2.

The mean LVMI in the group with LVH on the ECG was significantly higher than in either the normal (p < 0.001) or borderline (p < 0.005) groups. Although the mean LVMI was different among the three groups (fig. 1), there was considerable overlap of patients, and increased LVMI was seen in cases without LVH on the ECG. The correlation by linear regression analysis of the sum of voltages S1 + R5 or R6 with LVMI in patients with LVH on the ECG was poor (r = 0.40).

Although the individual variables that contribute to the LVM calculation (that is, volume or radius and wall thickness) also were significantly higher in patients with LVH than in those with borderline voltage or no LVH, even greater overlap in values occurred among the groups (fig. 2). Thus, no single characteristic of the ventricle itself reliably identified when LVH would be inscribed on the ECG.

The geometric relationship described by the ratio h/R was significantly different between LVH and normal patients (p < 0.001) (fig. 3). However, differences between mean h/R in LVH vs borderline and borderline vs normal patients were not significant. The LVMI/LVEDVI ratio also did not prove to be a useful discriminator among patients.

In contrast, the geometric parameter described by the product of h · R separated patients into the three electrocardiographic groups (fig. 4). Minimal overlap of groups occurred. Values from patients with LVH on the ECG were above the line described by the hyperbolic function, h · R = 2.6, while patients without LVH were on or below this line. Values from borderline LVH patients clustered on either side of the line, as shown. Patients who had increased LVM and did not record LVH on the ECG were distributed below and to the left of the LVH patients on the graph because of a lower product of h · R.

Discussion

As has been noted by other investigators,7,8,14 we have shown that patients with electrocardiographic evidence for LVH have increased LVM. Nevertheless, some patients in this study with an increased LVM had ECGs that showed either no LVH or only borderline voltage for LVH. We therefore postulate that additional geometric factors must be considered to explain this finding.
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II. Borderline voltage for LVH on ECG

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**Table 1. (Continued)**

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<th>LVEDVI (ml/m²)</th>
<th>h/R</th>
<th>h · R (cm²)</th>
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**Pressure overload**

| 47      | AS  | 203         | 81             | 1.4    | 2.7    | 2.5         | 0.51            | 3.8 |           |
| 48      | AS  | 206         | 171            | 1.2    | 3.7    | 1.2         | 0.31            | 4.4 |           |
| 49      | AS  | 273         | 104            | 1.6    | 2.9    | 2.6         | 0.55            | 4.6 |           |
| 50      | AS  | 205         | 78             | 1.6    | 3.0    | 2.6         | 0.54            | 4.8 |           |
| 51      | AS  | 162         | 117            | 1.1    | 3.4    | 1.4         | 0.31            | 3.7 |           |
| 52      | AS  | 138         | 103            | 1.0    | 3.3    | 1.6         | 0.50            | 3.3 |           |
| 53      | AS  | 182         | 84             | 1.3    | 2.8    | 2.7         | 0.46            | 3.6 |           |
| 54      | AS  | 127         | 65             | 1.1    | 2.6    | 2.0         | 0.42            | 2.9 |           |
| 55      | AS  | 160         | 81             | 1.3    | 3.0    | 2.0         | 0.43            | 3.9 |           |
| 56      | AS  | 173         | 97             | 1.4    | 2.5    | 1.8         | 0.57            | 3.5 |           |
| 57      | AS  | 231         | 139            | 1.3    | 3.6    | 1.7         | 0.38            | 4.7 |           |
| 58      | AS  | 250         | 136            | 1.6    | 3.6    | 2.0         | 0.43            | 5.8 |           |
| 59      | AS  | 120         | 53             | 1.2    | 2.3    | 2.3         | 0.48            | 2.5 |           |
| 60      | AS  | 216         | 103            | 1.3    | 2.9    | 2.1         | 0.46            | 3.8 |           |
| 61      | AS  | 114         | 78             | 1.0    | 1.5    | 1.5         | 0.34            | 2.8 |           |
| 62      | AS  | 174         | 106            | 1.2    | 1.6    | 1.3         | 0.37            | 4.0 |           |
| 63      | AS  | 113         | 109            | 0.9    | 1.0    | 1.0         | 0.27            | 3.0 |           |
| 64      | AS  | 149         | 61             | 1.2    | 2.3    | 2.4         | 0.52            | 2.8 |           |
| 65      | AS  | 200         | 139            | 1.2    | 3.4    | 1.4         | 0.35            | 4.1 |           |
| 66      | AS  | 147         | 107            | 1.0    | 1.4    | 1.4         | 0.29            | 3.4 |           |
| 67      | TBP | 159         | 78             | 1.3    | 2.5    | 2.0         | 0.57            | 3.0 |           |
| 68      | TBP | 148         | 64             | 1.3    | 2.6    | 2.3         | 0.50            | 3.4 |           |
| 69      | TBP | 206         | 154            | 1.1    | 3.8    | 1.3         | 0.29            | 4.2 |           |

**Volume overload**

| 70      | MR  | 160         | 118            | 1.0    | 3.2    | 1.4         | 0.31            | 3.2 |           |
| 71      | MR  | 131         | 130            | 0.8    | 3.5    | 1.0         | 0.23            | 2.8 |           |
| 72      | MR  | 143         | 85             | 1.1    | 2.9    | 1.7         | 0.38            | 3.3 |           |
| 73      | AR  | 130         | 106            | 1.0    | 3.3    | 1.2         | 0.30            | 3.3 |           |
| 74      | AR  | 184         | 143            | 1.2    | 3.3    | 1.3         | 0.36            | 4.0 |           |
| 75      | AR  | 154         | 117            | 1.0    | 3.4    | 1.3         | 0.30            | 3.4 |           |
| 76      | AR  | 241         | 192            | 1.2    | 3.7    | 1.3         | 0.32            | 4.4 |           |
| 77      | AR  | 173         | 98             | 1.2    | 3.0    | 1.8         | 0.40            | 3.6 |           |
| 78      | AR  | 202         | 162            | 1.2    | 3.8    | 1.3         | 0.32            | 4.6 |           |
| 79      | AR  | 335         | 201            | 1.6    | 4.0    | 1.7         | 0.40            | 6.4 |           |
| 80      | AR  | 191         | 185            | 1.0    | 4.0    | 1.4         | 0.29            | 4.0 |           |
| 81      | AR  | 244         | 226            | 1.2    | 3.9    | 1.1         | 0.31            | 4.7 |           |
| 82      | AR  | 385         | 219            | 1.7    | 4.0    | 1.8         | 0.43            | 6.8 |           |
| 83      | AR  | 186         | 131            | 1.2    | 3.3    | 1.4         | 0.30            | 4.0 |           |
| 84      | AR  | 411         | 331            | 1.4    | 4.6    | 1.2         | 0.30            | 6.4 |           |
| 85      | AR  | 263         | 256            | 1.0    | 4.0    | 1.0         | 0.25            | 4.0 |           |
| 86      | AR/MR | 270        | 166            | 1.3    | 3.6    | 1.6         | 0.37            | 4.7 |           |
| 87      | AR/MR | 174        | 133            | 1.0    | 3.1    | 1.3         | 0.32            | 3.1 |           |

**Combined overload**

| 88      | AS/AR | 122      | 75             | 1.1    | 2.9    | 1.6         | 0.36            | 3.2 |           |
| 89      | AS/AR | 221      | 92             | 1.5    | 3.0    | 2.4         | 0.50            | 4.5 |           |
| 90      | AS/AR | 246      | 162            | 1.3    | 3.5    | 1.5         | 0.35            | 4.6 |           |
| 91      | AS/AR | 121      | 70             | 1.0    | 2.8    | 1.7         | 0.36            | 2.8 |           |

**Myopathy**

| 92      | CMP-C | 110   | 79             | 0.9    | 3.0    | 1.4         | 0.30            | 2.7 |           |
| 93      | CMP-C | 262   | 261            | 1.1    | 4.6    | 1.0         | 0.24            | 5.1 |           |

**Mean ± SEM**

|                     | 194 ± 9.8* | 128 ± 8.7* | 1.2 ± 0.03* | 3.3 ± 0.1* | 1.6 ± 0.1* | 0.38 ± 0.01* | 3.9 ± 0.1* |

*Statistical analysis compares values from patients without LVH on the ECG to patients with either borderline voltage or LVH on the ECG.

*p < 0.001.

**Abbreviations:** LVMI = left ventricular mass index; Dx = diagnosis; LVH = left ventricular hypertrophy; LVEDVI = left ventricular end-diastolic volume index; h = left ventricular wall thickness; R = left ventricular semiminor radius in the right anterior oblique projection; AS = aortic stenosis; AR = aortic regurgitation; AS/AR = combined aortic stenosis and regurgitation; MR = mitral regurgitation; CMP-C = congestive cardiomyopathy; CMP-R = restrictive cardiomyopathy; †BP = hypertensive heart disease; S/P AVR = status post aortic valve replacement.
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| Pressure overload | | | | | | | | | | |
| 25 AS | 5 | 0 | 1 | 10 | 10 | 16 | 12 | 26 | |
| 26 AS | 9 | 3 | 5 | 5 | 12 | 14 | 10 | 26 | |
| 27 AS | 3 | 0 | 1 | 10 | 14 | 15 | 10 | 29 | |
| 28 ↑BP | 10 | 4 | 6 | 2 | 7 | 12 | 8 | 19 | |
| 29 ↑BP | 8 | 0 | 4 | 7 | 5 | 11 | 9 | 16 | |

| Volume overload | | | | | | | | | | |
| 30 MR | 10 | 10 | 11 | 0 | 13 | 16 | 10 | 29 | |
| 31 MR | 4 | 10 | 6 | 1 | 8 | 15 | 9 | 23 | |
| 32 MR | 8 | 4 | 5 | 2 | 11 | 18 | 7 | 29 | |

| Myopathy | | | | | | | | | | |
| 33 CMP-R | 4 | 3 | 2 | 4 | 7 | 7 | 9 | 16 | |
| 34 CMP-R | 5 | 2 | 2 | 5 | 8 | 10 | 12 | 20 | |
| 35 CMP-R | 7 | 3 | 3 | 3 | 12 | 13 | 10 | 25 | |
| 36 CMP-C | 3 | 7 | 5 | 2 | 8 | 12 | 13 | 21 | |

| II. Borderline Voltage for LVH on ECG | | | | | | | | | | |
| No overload | | | | | | | | | | |
| 37 Normal | 7 | 4 | 5 | 13 | 16 | 18 | 16 | 34 | |

| Pressure overload | | | | | | | | | | |
| 38 AS | 8 | 1 | 4 | 6 | 21 | 10 | 10 | 31 | |
| 39 AS | 7 | 3 | 6 | 9 | 19 | 13 | 12 | 32 | |
| 40 AS | 13 | 3 | 5 | 7 | 13 | 17 | 16 | 30 | |
| 41 ↑BP | 6 | 7 | 6 | 1 | 17 | 15 | 11 | 32 | |
| 42 ↑BP | 12 | 8 | 9 | 1 | 15 | 16 | 15 | 31 | |

| Volume overload | | | | | | | | | | |
| 43 MR | 7 | 6 | 7 | 5 | 11 | 19 | 13 | 30 | |
| 44 AR/MR | 13 | 0 | 6 | 7 | 10 | 22 | 21 | 32 | |

| Combined overload | | | | | | | | | | |
| 45 AS/AR | 13 | 11 | 12 | 3 | 11 | 21 | 19 | 32 | |
| 46 AS/AR | 8 | 2 | 7 | 3 | 12 | 20 | 17 | 32 | |
It has been suggested that a dilated ventricle alone will augment potentials more than a thickened myocardium will, or carry the heart closer to the precordial electrodes, thereby increasing QRS voltage by a proximity effect. Brody has reasoned that a dilated ventricle will more effectively augment radially directed dipoles because of the increase in intracavitary blood mass and thereby increase the voltage...
has largely been used to analyze TQ-ST-segment mapping techniques, the principles involved are applicable to LVH.

Craib has suggested that current flow is established across boundaries in isolated muscle strips by adjacent dipole sources with different electrical potentials. Bayley has shown that the electrical potential at any point in a conducting medium surrounding a closed-surface, double-layer dipole source is directly related to the dipole moment of each element of the surface and the solid angle, \( \Omega \), subtended by the boundaries of the surface at a point \( P \). The solid angle, \( \Omega \), is defined (fig. 5) as the area of spherical surface cut off a unit sphere (inscribed about a point \( P \)) by a cone whose base is defined by the boundary of interest and whose apex is at point \( P \). For a region of the in situ heart with different transmembrane voltages, \( V_m \), and \( V_{m_2} \) (fig. 5), the electrical potential, \( \epsilon \), recorded at a point \( P \) (on the body surface) can be given by:

\[
\epsilon = \frac{K \Delta V_m \Omega}{4\pi}
\]  

where \( \Omega \) is the solid angle subtended by the surface described by the limits of the region being analyzed, \( \Delta V_m \) is the difference between transmembrane potentials \( V_m \) and \( V_{m_2} \), and \( K \) is a constant that relates to the difference in conductivity and inhomogeneities within cardiac muscle. As stressed by Holland and Arnsdorf: “Since the solid angle depends only on the position of the recording electrode and the geometry of the boundary, it reflects spatial influences on the magnitude of \( \epsilon \). The difference in transmembrane potential between two regions does not, however, depend on geometry and thus reflects nonspatial influences on the magnitude of \( \epsilon \).” Because there is no change in the transmembrane action potentials in myocytes of chronically hypertrophied hearts, Witham has deduced that the increased voltage in cases of LVH on the ECG cannot be ascribed to the “simple product of the number of cells and their individual voltage.” Therefore, the geometry of the ventricle must play an important role in determining the increased voltage.

The solid-angle theory is limited by the fact that the angle \( \Omega \) can be easily constructed only for simple geometric figures. Holland and Arnsdorf have shown that the solid angle subtended by a circular disc at a point directly overlying its center varies inversely with the distance between the disc and the recording electrode site. Because an elliptical model of the left ventricle is commonly used in analysis of ventricular geometry, it would be useful to know if a similar relationship holds for an ellipsoidal disc. The solid angle subtended by an ellipsoidal disc with a recording electrode directly over its center at a distance \( z \) is given by the following equation:

\[
\Omega (a, b, z) = 2\pi - 4z^{n/2} \int_0^{2\pi} \left[ \frac{b^2 + (a^2 - b^2) \sin^2 \phi}{b^2 (a^2 + z^2) + z^2 (a^2 - b^2) \sin^2 \phi} \right] d\phi
\]  

FIGURE 1. A plot of angiographically determined left ventricular mass index (LVMI) in three electrocardiographic groups: no left ventricular hypertrophy (LVH) on the ECG, borderline LVH on the ECG, and LVH on the ECG. There was some overlap of values between groups and some patients had increased LVMI without LVH on the ECG.
where a is the semimajor axis, b the semiminor axis, and $\phi$ an angle describing the relationship between a point projected onto an ellipse from a recording electrode and the center of the ellipse.

Inspection of equation (2) shows that $\Omega$ varies inversely with the distance $z$, and thus the solid angle subtended when the surface of the left ventricle is carried closer to the precordial recording electrodes would be greater. Greater proximity to the recording electrodes can be accomplished by either an increase in wall thickness or LV chamber dilatation with an increase in radius, or a combination of the two.

We therefore plotted wall thickness vs radius in all patients studied and found this to delineate more accurately the ventricular geometry leading to the inscription of LVH on the ECG than LVM alone. The relationship of $h \cdot R$ not only identifies patients with LVH, but also explains why certain patients with increased LVM do not show LVH on the ECG (fig. 4). For example, a patient with aortic stenosis and...
sufficient thickening of the free wall to increase LVM would not show LVH on the ECG unless sufficient chamber dilatation (i.e., increased radius) had also occurred.

It can readily be seen that patients with LVH on ECG are distributed in the upper right hand portion of the plot (fig. 4), while patients without LVH are at the lower left portion. Thus, a critical geometric relationship must be achieved by the hypertrophied left ventricle, involving a combination of a thickened free wall and dilated chamber, to yield a solid angle sufficiently great that LVH would be recorded on the ECG.

This relationship is seen on the one extreme in patients with ventricles markedly dilated from volume overload or cardiomyopathy and only a slightly thickened free wall. On the other extreme are patients who have pressure overload of the left ventricle, with a relatively small chamber volume (normal R) and a markedly thickened free wall. Various combinations of hemodynamic lesions will lead to a variety of combinations of alterations in h or R or both.

The relationship h · R appears to define the geometry better than mass alone, even though h and R are the major determinants of LVM. Although theoretically it should be possible to have electrocardiographic LVH from a massively dilated ventricle with a thin wall and a normal LVM, such a situation would be physiologically improbable on a chronic basis because of intolerably high resultant myocardial wall stress. Most patients with chronic LV volume overload have an increase in h commensurate with

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**Figure 4.** Top) The wall thickness (h) is plotted on the vertical axis vs the semiminor radius (R) plotted on the horizontal axis. The hyperbolic function described by h · R = 2.6 is shown in dashed lines. Bottom) This relationship of h and R is represented schematically and shows that when h · R < 2.6 no left ventricular hypertrophy (LVH) is inscribed on the ECG; LVH is inscribed when h · R > 2.6. Patients with h · R close to 2.6 have borderline voltage for LVH on the ECG.

**Figure 5.** Mathematical and pictorial characterization of the solid-angle theory. P represents a recording electrode on the body surface with a unit sphere inscribed about it. Ω represents a solid angle defined by the area of spherical surface cut off the unit sphere by a cone drawn as shown with its apex at P. Vm1 and Vm2 are adjacent regions of myocardium with different transmembrane voltages (see text). (Reproduced from Holland and Arnsworf with the permission of the authors and the publisher.)
their increased R, so that h/R remains normal.41

Thus, the relative interplay between wall thickness and chamber dilatation determines whether LVH will be recorded on the ECG (fig. 4). As predicted by the mathematical model of Rudy and co-workers,28 and schematically represented in figure 6, an enlarged chamber plays a greater role than a thickened myocardium in augmenting potentials. Electrocardiographic LVH seems to be a manifestation of LV enlargement and may not be present when wall thickening alone, without sufficient dilatation, produces an increase in LVM without subtending a large enough solid angle at the body surface.

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![Figure 6. The influence of chamber dilatation and wall thickness in increasing the solid angle subtended at the recording electrode. With normal growth from childhood to adulthood, heart mass and torso mass increase proportionately, and the solid angle subtended remains constant. With volume overload of the left ventricle, total left ventricular (LV) mass is increased largely through chamber dilatation, and the solid angle subtended at the recording electrode is increased markedly. Pressure overload of the left ventricle, although leading to an increase in LV mass through wall thickening, will not subdivide a markedly enlarged solid angle unless chamber dilatation occurs as well. The distance between the heart and the recording electrode may influence the solid angle subtended. Such potential variability in the solid angle may explain why certain normal young subjects with a thin chest and narrow anteroposterior diameter have increased QRS voltage. (Reproduced from Holland and Arnsdorf with the permission of the authors and the publisher.)](http://circ.ahajournals.org/Download/access redistribute rights)
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E M Antman, L H Green and W Grossman

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