Geometric Determinants of Electrocardiographic Left Ventricular Hypertrophy

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SUMMARY Experimental studies have suggested that electrocardiographic recognition of left ventricular hypertrophy depends on geometric relationships involving wall thickness and chamber size. To determine the clinical significance of these observations, we studied the effects of echocardiographic LV mass (LVM), posterior wall thickness (PWT), interventricular septal thickness (IVST) and internal dimension (LVID) on ECG voltage in 360 patients. Standard voltage and nonvoltage manifestations of LVH correlated modestly with LVM (r = 0.33-0.44, p < 0.001). Sokolow-Lyon precordial voltage (SLV) (SV1 + RV5 or VR) correlated moderately with LVM (r = 0.41, p < 0.001), but correlated less well with IVST (r = 0.26), PWT (r = 0.24) or LVID (r = 0.22). Stepwise regression revealed that there was no relation, independent of LVM, between SLV and IVST (r = −0.03), PWT (r = 0.03) or LVID (r = 0.01). The 90 patients with increased LVM (> 215 g) but without LVH by SLV (false negatives) were compared with the 48 identified by SLV (true positives). False negatives differed from true positives in LVM (298 ± 72 vs 339 ± 98 g, p < 0.01), age (55 ± 18 vs 44 ± 19 years, p < 0.001), weight (70 ± 16 vs 63 ± 14 kg, p < 0.02), and distance from skin to the interventricular septum (42 ± 10 vs 38 ± 8 mm, p < 0.02). Thus, for a given LVM, ECG voltage criteria of LVH are independent of LV chamber dilatation or other geometric variables, but depend on age, weight and LV depth in the chest, suggesting that stratification of subjects by clinical variables has promise for improved electrocardiographic recognition of LVH.

The ECG is a widely used, simple and inexpensive technique, but it is unreliable in the diagnosis of left ventricular hypertrophy (LVH). In support of this theory, a positive relationship has been found between LV volume and ECG voltage in several experimental studies and has been inferred in man on the basis of voltage changes during exercise. However, other studies in patients with chronic heart disease as well as experimental studies of acute changes in LV volume suggest that ECG voltage for any given LV mass (LVM) may decrease with LV enlargement.

Theoretical considerations suggest that other geometric factors may also be important. If the source of the recorded voltage is the potential difference across the myocardial cell membrane (which does not increase appreciably with LVH), then the voltage generated by a hypertrophied cell should be proportional to the cell’s surface area, i.e., to the square of its radius. If this theory is correct, the voltage detected by an electrode subtending a constant angle would be proportional to the square of the radius of individual myocardial cells, and thus, because increases in LVM during adult life occur primarily by cellular hypertrophy rather than hyperplasia, to LVH. According to another analysis derived from the solid-angle theory, ECG voltage should be proportional to the product of wall thickness (h) times chamber radius (r). Finally, it has been suggested that ECG voltage is influenced by the modulating effects of the inverse square law. Nevertheless, the relative importance of geometric variables and of clinical factors such as age and body habitus in modifying electrocardiographic manifestations of LVH remains controversial.

The present study was therefore designed to determine whether LV geometry and clinical variables significantly affect electrocardiographic voltage independent of LVM. Our findings in 360 patients suggest that age, weight and position of the heart in the chest are important determinants of electrocardiographic recognition of LVH, while specific patterns of LV geometry are not. As electrocardiography becomes more computerized, adjustment of ECG criteria to take these findings into account may become routinely feasible.

Methods

Patients

The study population included 360 consecutive patients with technically excellent echocardiograms at the New York Hospital–Cornell Medical Center who met the following criteria: (1) A technically satisfactory 12- or 15-lead ECG in a nonpaced rhythm was available within 30 days (mean 4 days) of the echocardiogram; (2) complete clinical records were available; and (3) there was no clinical, electrocardiographic or echocardiographic evidence of myocardial infarction, hypertrophic cardiomyopathy or bundle branch block. Clinical records, ECGs and echocardiograms were analyzed independently, without knowledge of the results of other studies. Cardiac diagnoses were estab-
lished independently by review of clinical records, including the recorded opinion of the cardiac consultant when available. Results of autopsy and cardiac catheterization were available in 11 and 50 patients, respectively. The patients were 15–88 years old (mean 46 years); 217 were female. Ninety patients had valvular heart disease, 81 hypertension, 29 mitral valve prolapse, 26 congestive cardiomyopathy, 21 pericardial disease and 17 coronary artery disease without myocardial infarction. A variety of other cardiovascular diseases were present in 17 subjects; 79 had no evidence of a cardiovascular abnormality.

**Echocardiographic Data**

M-mode echocardiograms were recorded with 2.25-MHz, focused transducers and Smith Kline 20, Picker 80C or Irex system 11 echographs on strip-chart paper at 50 mm/sec using Honeywell 1856 strip-chart recorders. They were analyzed independently and without knowledge of clinical data by two investigators. Differences were resolved by joint review of the coded echocardiograms. Standard echocardiographic measurements of end-diastolic and end-systolic LV dimension (LVIDd and LVIDs), wall thickness (PWT) and interventricular septum (IVS) were made according to the recommendations of the American Society of Echocardiography. Chest wall thickness, and the distance from the chest wall to the right side of the IVC and to the middle of the left ventricle were also measured. LV end-diastolic volume (LVEDV), end-systolic volume (LVESV) and ejection fraction were calculated by use of the cube function formula. To calculate LVM, an additional set of end-diastolic LV measurements were made in which the thickness of endocardial echoes were excluded from measurements of IVS and PWT, and the thickness of endocardial echoes from the left side of the septum and the posterior wall endocardium was included in the measurement of LVID. LVM was calculated by the autopsy-validated regression equation:

\[ \text{Echo LVM} = 1.04 \times (\text{LVIDp} + \text{PWTp} + \text{IVSp})^3 \]

\[ - (\text{LVIDp})^3 - 13.6g. \]

The necropsy population in which this method for echocardiographic estimation of LVM was validated resembled the present series in its spectrum of diagnoses, including patients with hypertensive, valvular and coronary heart disease, congestive cardiomyopathy and pericardial disease, and patients with noncardiac diseases. Further support for the validity of using this method is provided by our finding in a subsequent autopsy series of 22 patients that mean echocardiographic LVM was within 8% of autopsy LVM, and that the echocardiogram was 100% sensitive (seven of seven), and 93% specific (14 of 15) for LVH (Devereux RB, Alonso DR: unpublished data).

LVH was defined as echocardiographic LVM greater than 215 g. This is similar to the cutoff used in other autopsy, angiographic and echocardiographic studies. To take into account the influence of body size on normal LVM, we also classified patients alternately as having LVH if their LVM index exceeded 125 g/m².

To evaluate alternative geometric causes of ECG LVH, we also calculated the product of PWTd × LVIDp/2 (i.e., the h·r product), and LVM², which is proportional to the surface area of cardiac myocytes.

**Electrocardiography**

Standard 12-lead ECGs were analyzed blindly by two investigators and differences resolved by joint review of the tracing. Voltage of the R and S waves was measured in all leads. The precordial voltage combination of Sokolow and Lyon (SLV) was measured as SV₃ + larger R wave in V₃ or V₆. The amplitude of the R wave in aV₁ was also measured, and the Romhilt-Estes point score calculated based on the presence or absence of QRS amplitude criteria (3 points), as well as repolarization abnormalities of LVH (1 or 3 points, depending on whether digitalis was being taken), left atrial abnormality (3 points), QRS axis of – 30° or more (2 points), and QRS duration ≥ 0.09 second or intrinsocoid deflection ≥ 0.05 second (1 point each). We considered the ECG to inscribe LVH if SLV was greater than 35 mm, if RaV₁ was 11 mm or more, or if the Romhilt-Estes point score was 4 points or more.

**Statistical Methods**

Echocardiographic, ECG and clinical data for each patient were entered into an IBM 390 computer at the Rockefeller University Computer Facility. Analysis of quantitative variables by least-squares linear regression analysis, multivariable stepwise regression analysis, and t test were performed with assistance of the BMD software programs.

**Results**

Anatomic LVH was present in 138 patients (38%), in whom LVM ranged from 217 to 676 g. The remaining 222 patients had normal LVM, ranging from 35 to 214 g. A significant linear correlation was found between SLV and LVM (r = 0.41, p < 0.001). Weaker but still significant linear correlations also were found between SLV and interventricular septal thickness (r = 0.27, p < 0.01), posterior LV wall thickness (r = 0.32, p < 0.001) and LVID (r = 0.23, p < 0.05), which are the individual determinants of LVM (table 1).

In addition to SLV, similar correlations existed between LVM and several other ECG voltages, including RaV₁ (r = 0.33, p < 0.001), SV₁ (r = 0.32, p < 0.001), SV₃ (r = 0.43, p < 0.001) and RV₁ (r = 0.34, p < 0.001). The Romhilt-Estes point score, which incorporates nonvoltage criteria of LVH, did not yield a closer relationship with LVM (r = 0.44, p < 0.001). These correlations, which are similar to those reported by other investigators, were not significantly different from those found with SLV. Since SLV performed as well as other indexes of LVH, and because it is widely used by clinicians and investiga-
tors, it was used for the remainder of this analysis.

To determine whether septal thickness and PWT or chamber size contributed to the generation of electrocardiographic voltage independently of LVM, a stepwise regression analysis was performed. This revealed that, as might be expected, the effects of PWT and septal thickness are interdependent with either geometric factor, but not both, being a significant independent contributor to the generation of electrocardiographic voltage. LVID correlated with ECG voltage independently of wall thickness. However, when the contribution of LVM to the generation of SLV was first eliminated from the stepwise regression analysis, no independent contribution of wall thicknesses or chamber size to the generation of Sokolow-Lyon or other ECG voltages could be demonstrated (r = 0.01–0.04; NS).

The relationship of SLV with other geometric variables was also examined (table 1). A significant linear correlation existed between the product of wall thickness and chamber radius (PWT LVID/2 = h · r) and SLV (r = 0.40, p < 0.001). A similar relationship was also found between SLV and LVM23 (r = 0.40, p < 0.001). However, in both cases, the correlation was identical to that between SLV and LVM. Furthermore, no cutoff value of either of these variables performed better than did a SLV of greater than 35 mm in separating patients with and without LVH.

In addition, we examined the significance of the position of the heart in the chest cavity. As predicted by the inverse square law, ECG voltage varied inversely, but only slightly, with the distance from chest surface to IVS (r = −0.17, p > 0.05). However, the distance from the chest wall to the septum tended to increase marginally with increasing thickness of the septum (r = 0.18, p > 0.05) and posterior LV wall (r = 0.10, p > 0.05).

Geometric and demographic factors were analyzed separately in the subgroup of 138 patients with anatomic LVH to evaluate their effect on inscription of increased QRS voltage in patients with LVH (table 2). In this group, 48 had increased SLV (true positives) and 90 had normal voltage (false negatives). There was no significant difference in calculated LV volume between the ECG true positives and false negatives. Similarly, septal thickness was the same in both groups. Among the subjects with anatomic LVH, there was a relatively modest difference in LVM between those with ECG LVH (339 ± 98 g) and those without it (298 ± 72 g, p < 0.01).

In contrast to the marginal influence of geometric variables on ECG detection of LVH, the ECG true positives in this subgroup did differ significantly from the false negatives in that they were younger (44 ± 19 vs 55 ± 18 years, p < 0.001), leaner (63 ± 14 vs 70 ± 16 kg, p < 0.02) and had smaller body surface areas (1.72 ± 0.2 vs 1.80 ± 0.2 kg/m2, p < 0.05); the IVS was also closer to the anterior surface of the chest wall in these patients (3.8 ± 0.8 vs 4.2 ± 1.0 cm, p < 0.02). Substitution of the LVM index for LVM in this stepwise analysis renders insignificant the independent effects of body surface or distance of the septum from the chest surface on inscription of ECG LVH.

**Discussion**

The effect of intracardiac blood on the ECG has been the subject of extensive analysis and experimental study. Theoretically, the influence of a mass of highly conductive fluid within the myocardial shell...
should depend on whether depolarization is proceeding rapidly across the ventricular wall, or tangentially about the chamber. As pointed out by Brody and confirmed by others,31,32 intracardiac blood magnifies the voltage generated by radial excitation and diminishes that due to tangential excitation. Activation is normally primarily radial in the heart of most mammals, and it has been repeatedly shown that epicardial or body surface QRS voltage is increased by acute increases in either ventricular volume or blood conductivity.33,34 In addition, both theoretical analysis35 and clinical studies8 have shown a strong tendency for LVM to increase with enlarged LV volume. Thus, one would expect dramatic elevations of surface ECG voltage in the heart that is both dilated and hypertrophied.

In contrast to this prediction, our observations demonstrate that LV chamber size is only very weakly correlated with ECG voltage and that there is no significant difference in LV voltage in patients with and without ECG LVH. These data suggest that other factors must modify the relationship of increasing LV volume and ECG voltage. First, in the presence of LVH, the time of inscription of maximal QRS voltage may be delayed, as reflected by prolonged intrinsicoid deflection. This may shift vector forces into the latter portion of the QRS, when LV activation is predominantly tangential37 rather than radial.

The pattern of activation of dilated, diseased ventricles may also be altered so that the spread of the impulse is more tangential than normal. While this is well documented in the presence of bundle branch block,38 there are no pertinent data concerning the chronically dilated heart without conduction defects. Stretch of conduction fibers10 and their replacement by fibrous tissue in LVH39 would both retard the initial rapid spread of the impulse through the Purkinje network and might tend to enhance slower tangential excitation through the myocardium. The observation that QRS amplitude increases with exercise in person with poor LV function but decreases in normal persons11 is in accord with this explanation. However, reports from other laboratories have not confirmed a significant relationship between changes in LV volume and QRS voltage during exercise.39,40

Alternatively, generation and transmission of cardiac potentials might be more directly altered. Because LV dilatation often reflects myocardial failure, the dilated heart might have relatively fewer functional myocardi- cells than normal to generate electrical activity. However, the ejection fraction in our patients who inscribed ECG LVH was identical to that of those who did not. In addition, the presence of pericardial fluid might damp the ECG voltage detected in surface leads.

In another study,41 we have found that small to moderate-sized pericardial effusions cause only a modest reduction of the QRS voltage generated by a given LVM, but large pericardial effusions do significantly dampen ECG voltage so that all correlation with LVM is lost.

An alternative hypothesis has been advanced in recent years, that specific aspects of LV or thoracic geometry are more important than LVM itself in determining ECG LVH.6,17,18 Geometric features which have been advocated as having special importance include the product of LV wall thickness times chamber radius,6 the surface area of individual myocytes,19 and the distance of the outer surface of the LV myocardium from the ECG electrode.18 In the present study, each of these geometric variables was tested as a potential determinant of ECG LVH. Closer relationships were observed between LVM and standard ECG manifestations of LVH ($r = 0.41$–$0.44$) than between the alternative geometric measurements and ECG findings ($r = 0.18$–$0.40$). Furthermore, the cutoff proposed by Antman et al.6 of an $h$ product more than 2.6 performed no better than LVM more than 215 g in predicting the presence or absence of ECG LVH. Thus, neither the volume of blood within the left ventricle nor specific aspects of the chamber’s geometry are important determinants of ECG LVH independent of LVM.

However, the 12-lead surface ECG appears to be intrinsically limited in its correlation with LVM. Our correlation ($r = 0.41$) of LVM with ECG voltage is similar to that reported in other studies.5,6,41 The best estimate of the proportion of variation in a dependent variable due to the independent variable is provided by the coefficient of determination ($r^2$). Since $r$ equals 0.41 in this series, $r^2$ would be 0.17, implying that only about 17% of the variation in ECG voltage observed in a diverse clinical series can be attributed directly to differences in LVM. Attempts to improve the prediction of LVM from 12- or 15-lead ECG measurements of QRS voltage and other parameters by taking into account age, sex, body mass and disease states have been partially successful. The present study suggests that this approach may be fruitful, since it takes into account several factors that affect ECG manifestations of LVH, whereas further study of specific geometric determinants of ECG LVH is unlikely to be fruitful.

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