A Quantitative Angiocardiographic Study of Left Ventricular Hypertrophy and the Electrocardiogram

By William A. Baxley, M.D., Harold T. Dodge, M.D., and Harold Sandler, M.D.

SUMMARY

The QRS amplitude, duration, and mean axis direction of the standard 12-lead electrocardiogram were compared with left ventricular mass, volume, wall thickness, and stroke work in 112 adult patients. These patients had normal left ventricles or various degrees of left ventricular dilatation or hypertrophy. Left ventricular volumes and mass were determined by a quantitative angiocardiographic method. A significant but not a close correlation between left ventricular mass and ECG voltage was found. End-diastolic volume, stroke volume, stroke work, wall thickness, and "total left ventricular volume" (volume of left ventricular muscle and cavity at end-diastole) had generally lesser degrees of correlation with the QRS amplitude than did the mass alone. Subgrouping of patients into those with pressure overload on the left ventricle, volume overload, aortic valve disease, or anatomic left ventricular hypertrophy did not improve correlations. The left ventricular hypertrophy voltage criteria of Sokolow and of Grant were assessed; respectively, 68% and 67% of 75 patients with anatomic hypertrophy had hypervoltage. The direction of the anatomic long axis of the left ventricle was not significantly related to the mean QRS-vector direction.

Additional Indexing Words:

Angiocardiography  Electrocardiography  Hemodynamics
Left ventricular volume  Left ventricular mass

SINCE 1906 when Einthoven1 first described a relationship between heart size and the electrocardiogram, there has been widespread interest in and an increasing clinical use of electrocardiographic measurements for the diagnosis of cardiac hypertrophy. Various criteria for the diagnosis of left ventricular hypertrophy from the electrocardiogram have been suggested, most notably by Sokolow and co-workers,2 and by Grant.3 Subsequently, the accuracy of the different criteria has been studied by a number of investigators.4-15 Most of these studies of the relationship between the electrocardiogram and left ventricular size have consisted of comparisons of the electrocardiogram and various heart measurements made at autopsy, such as total heart weight5, 7, 11, 13 or ventricular wall thickness.4, 8 However, as pointed out by Levine, Rockoff, and Braunwald,16 there are disadvantages in relying on autopsy material for study of the heart: rigor mortis reduces ventricular volume and accordingly increases wall thickness, terminal-stage disease may introduce complicating anatomic variables, and autopsy studies do not permit satisfactory physiological-anatomic correlations.

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Another approach to the study of the electrocardiogram in left ventricular hypertrophy has been to relate the electrocardiogram to factors known to be associated with cardiac hypertrophy, such as the presence of systemic hypertension, an increased overall heart size as visualized on the chest x-ray, and an increased left ventricular wall thickness measured from angiograms uncorrected for x-ray distortion.

Recently, methods have been described for determining in-vivo left ventricular volume and left ventricular mass or weight from angiograms. Kennedy and co-workers have utilized these methods to establish the normal values for left ventricular volumes and mass in adults. In the present study, the electrocardiogram was compared to left ventricular mass, volume, stroke work and wall thickness, and the direction of the ventricular long axis as determined by angiocardiographic methods in groups of adult patients with and without enlarged left ventricles. It is the purpose of this study to define more clearly the relationship between the electrocardiogram and the anatomic and functional changes of the left ventricle associated with hypertrophy.

Methods

Data were analyzed from 112 adult patients who had left- or right-sided cardiac catheterization, or both, and angiocardiography at the Seattle Veterans Administration Hospital or the University Hospital at the University of Washington. There was no attempt at patient selection. All patients who had adequate electrocardiographic, pressure, and angiocardiographic data were included. There were 108 males and four females. The mean patient age was 47 (standard deviation = 12), with three under age 25. At cardiac catheterization, pressures were determined with strain gauges and recorded with a multichannel recorder. Angiocardiography was performed at 100-115 kv, 500 ma, and 1/30-second exposure time with power injection of 40 to 60 cc of contrast material into either the right- or left-sided cardiac chamber with most filming at a rate of 6-per-second with use of a Schonander or Elema biplane film changer. In general, the subjects in this study had heart diseases of varied etiologies, although most patients had valvular heart disease. Included were 19 patients without heart disease in whom angiocardiography was performed for clinical reasons other than suspected heart disease. All patients had a routine 12-lead electrocardiogram, with standardization at 1 cm = 1 millivolt, taken during the same hospitalization as that in which the catheterization and angiocardiography were performed. Five patients had left bundle-branch block as defined by a QRS duration of 0.12 second or greater, with the terminal 0.04 second of the QRS vector pointing leftward and posteriorly. Others had lesser degrees of QRS prolongation resembling left bundle-branch block apparently caused by long-standing hypertrophy; however, none was omitted because of particular electrocardiographic abnormalities. Only the QRS complex was studied because many patients were taking digitalis or had metabolic abnormalities causing ST- and T-wave changes similar to those observed with left ventricular hypertrophy. Furthermore, it has been shown in earlier studies that ST- and T-wave changes by themselves may lead to mistaken diagnoses of left ventricular hypertrophy.

The hemodynamic and electrocardiographic data listed below were tabulated for each patient on IBM punch cards and submitted to an IBM 7040-7094 electronic computer for statistical analysis.

**Hemodynamic Data**

1. Left ventricular volume. Volumes were calculated from the angiograms by the area-length method as previously described. In most cases angiocardiographic

![Figure 1](image-url)

A typical angiogram taken in the anteroposterior projection near end diastole. The left ventricular cavity is outlined, and the ventricular wall thickness is determined by planimetry of a 4-cm segment of anterolateral wall located perpendicular to the apex-aortic valve axis, one third of the way up from the apex.
filming extended over 3 or 4 cardiac cycles. Films were timed with respect to the ECG, and a composite left ventricular volume curve for one cardiac cycle was drawn. Left ventricular end-diastolic volume (LVEDV) and stroke volume (LVSV) were determined from the composite curves.

2. Left ventricular wall thickness. Because of the irregularity of the inner myocardial wall, an average value was obtained by planimetry of a 4-cm segment of the anterolateral ventricular wall on a film taken in the anteroposterior (AP) projection near end-diastole. The segment is located one third of the distance from the apex to the aortic valve as shown in figure 1 and as described in an earlier publication. This value was corrected for x-ray distortion due to nonparallel x-rays.17

3. "Total left ventricular volume." This was calculated as the volume of the left ventricular myocardium plus chamber at end-diastole. The left ventricular wall thickness, obtained as described above, is added to each of the major and minor axes of the left ventricular cavity determined for the calculation of ventricular volume, and volume of the cavity plus wall is then calculated.

4. Left ventricular mass (LVM). This was calculated from the films by the method of Rackley and co-workers. The "total left ventricular volume," as described above, minus the chamber volume equals the volume of left ventricular myocardium. The product of the volume of left ventricular myocardium and 1.050, the specific gravity of heart muscle, is the left ventricular mass.

5. Left ventricular mass per square meter of body surface area (LVM/BSA).

6. Left ventricular stroke work (LVSW). This was calculated in g-m utilizing the product of stroke volume determined from the angiocardiograms and mean systolic left ventricular pressure.

**Electrocardiographic Data**

<table>
<thead>
<tr>
<th>Voltage criteria of Sokolow$^2,10$</th>
<th>Maximum normal</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. Voltage criteria of Sokolow$^2,10$</td>
<td></td>
</tr>
<tr>
<td>1. $SV_1 + RV_5,6$ (S wave in lead $V_1$ + maximum R wave in lead $V_5$ or $V_6$)</td>
<td>35 mm</td>
</tr>
<tr>
<td>2. $RV_5,6$ (R wave in lead $V_5$ or $V_6$, whichever is greater)</td>
<td>26 mm</td>
</tr>
<tr>
<td>3. $R aV_L$ (R wave in lead $aV_L$)</td>
<td>11 mm</td>
</tr>
<tr>
<td>4. $R aV_R$ (R wave in lead $aV_R$)</td>
<td>20 mm</td>
</tr>
<tr>
<td>5. $R_3 + S_{III}$ (R wave in lead I + S wave in lead III)</td>
<td>25 mm</td>
</tr>
<tr>
<td>B. Voltage criteria of Grant$^3$</td>
<td></td>
</tr>
<tr>
<td>1. $SV_{1.5} + RV_6$ (maximum S wave in lead $V_1$ or $V_2 + R$ wave in lead $V_6$)</td>
<td>40 mm</td>
</tr>
<tr>
<td>2. Maximum $R + SV$ (maximum $R + S$ wave in any single V lead).</td>
<td>35 mm</td>
</tr>
<tr>
<td>C. Intrinsicoid deflection (maximum time from onset of QRS to peak positive deflection in lead $V_5$ or $V_6$)$^2$</td>
<td>0.5 sec</td>
</tr>
</tbody>
</table>

In addition to the total group of 112 patients, the following four subgroups were studied independently to determine if closer correlations among the above variables existed in patients who had similar hemodynamic abnormalities:

1. Twenty-one patients with only left ventricular volume overload (isolated aortic regurgitation or isolated mitral regurgitation, or both).
2. Nine patients with only left ventricular pressure overload (aortic valve stenosis, systemic hypertension, or aortic coartation).
3. Thirty patients with only aortic valve disease (stenosis or insufficiency, or both).
4. Seventy-five patients with left ventricular hypertrophy, regardless of etiology. Left ventricular hypertrophy is defined as left ventricular mass greater than 2 standard deviations above the mean adult normal as determined by Kennedy and co-workers$^{20}$ (greater than 125 g/m²).

The overall accuracy of the ECG criteria described by Grant for detecting ventricular hypertrophy was also assessed. Patients were classified as having left ventricular hypertrophy or not, according to the above-mentioned criteria. The electrocardiograms from patients with and without anatomic left ventricular hypertrophy were examined to determine the percentage with hypervoltage by any of the criteria of Sokolow or of Grant.

In 14 randomly selected patients with various types of heart disease, the direction of the major axis of the left ventricle as calculated from the angiocardiograms was compared with the direction of the mean QRS vector in the frontal plane. Methods used for calculating the length and spatial direction of the left ventricular major axis have been previously published.$^{17,21}$ The angle made by the major (apex-to-aortic valve) axis and the horizontal axis (x axis) on anteroposterior angiocardiograms as shown in figure 2 was determined

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*The R wave in lead I greater than 16 mm, a further voltage criterion of Sokolow, was of no help by itself in the diagnosis of left ventricular hypertrophy in the present group, and hence was not included in the statistical analysis.
after correction for distortion from nonparallel x-ray beams. The frontal-plane direction of the mean QRS vector was measured from leads I, II, and III of the conventional ECG by a triaxial reference figure as described by Grant.3 To determine the mean QRS axis position as accurately as possible, planimetry of the area beneath leads I, II, and III was utilized.

**Results and Discussion**

The relationships between the eight electrocardiographic and the seven anatomic or physiological variables for the total patient group are expressed as correlation coefficients and are given in table 1. As illustrated in this table, most of the correlations are significant at the 99% confidence level (P < 0.01 for all correlation coefficients greater than 0.24), although there is not a close correlation between any of these pairs of variables (highest r value present = 0.58). SV₁ + RV₅,₆ and SV₁,₂ + RV₆ in general gave the best correlations, in particular with left ventricular mass. It is of interest that SV₁ + RV₅,₆ as utilized by Sokolow's voltage criteria and SV₁,₂ + RV₆ as utilized in Grant's criteria correlate equally well with the variables studied. These correlations between the ECG and anatomic variables were similar to those found by Carter and Estes13 in an autopsy study of 319 subjects with and without hypertrophy. In this previous study, heart weight was significantly correlated with precordial voltages, expressed as P < 0.001.

Figure 3 illustrates the relationship between SV₁,₂ + RV₆ and left ventricular mass per square meter of body surface area, with each point representing observations on one patient. There is considerable scatter of points about the regression line. The regression equation is LVM/BSA = 71.3₁ + 2.42 (SV₁,₂ + RV₆), with s (the standard error of estimate) = 60.7 g/m². The dashed diagonal lines define the limits of standard error of estimate. The dotted horizontal and vertical lines represent the upper limits of normal for the two variables. Thus, points in the upper right quadrant have both increased left ventricular mass and hypervoltage in the electrocardiogram. Those in the lower left have normal mass and normal QRS voltage, those in the upper left are “false-positive” electrocardiograms with normal mass and hypervoltage, and those in the lower right are “false negatives” with increased mass but normal QRS voltage.

**Table 1**

<table>
<thead>
<tr>
<th>ECG variables</th>
<th>Wall thickness</th>
<th>Mass</th>
<th>Anatomic-physiologic variables of the left ventricle</th>
<th>Stroke volume</th>
<th>Systolic pressure</th>
<th>Stroke work</th>
</tr>
</thead>
<tbody>
<tr>
<td>SV₁ + RV₅,₆</td>
<td>0.40</td>
<td>0.54</td>
<td>0.56</td>
<td>0.36</td>
<td>0.49</td>
<td>0.39</td>
</tr>
<tr>
<td>RV₅,₆</td>
<td>0.22</td>
<td>0.34</td>
<td>0.36</td>
<td>0.31</td>
<td>0.33</td>
<td>0.36</td>
</tr>
<tr>
<td>R aVL</td>
<td>0.49</td>
<td>0.48</td>
<td>0.48</td>
<td>0.22</td>
<td>0.39</td>
<td>0.24</td>
</tr>
<tr>
<td>R aVF</td>
<td>0.00</td>
<td>-0.13</td>
<td>-0.09</td>
<td>-0.23</td>
<td>-0.18</td>
<td>-0.01</td>
</tr>
<tr>
<td>R₁ + S₁II</td>
<td>0.48</td>
<td>0.45</td>
<td>0.45</td>
<td>0.36</td>
<td>0.28</td>
<td>0.50</td>
</tr>
<tr>
<td>SV₁,₂ + RV₆</td>
<td>0.33</td>
<td>0.55</td>
<td>0.58</td>
<td>0.44</td>
<td>0.38</td>
<td>0.41</td>
</tr>
<tr>
<td>Max R + Sᵥ</td>
<td>0.41</td>
<td>0.44</td>
<td>0.46</td>
<td>0.20</td>
<td>0.38</td>
<td>0.38</td>
</tr>
<tr>
<td>Intrinsic deflection</td>
<td>0.19</td>
<td>0.50</td>
<td>0.48</td>
<td>0.57</td>
<td>0.58</td>
<td>0.40</td>
</tr>
</tbody>
</table>

**Figure 2**

An angiocardiogram in the anteroposterior projection. The mid-aortic valve point (A) and apex point (B) are corrected for nonparallel x-rays to A' and B', respectively. A'–B' then represents the true major axis of the left ventricle in the frontal plane, and the angle α that this axis makes with the horizontal axis is measured directly.17, 21

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Comparison of the ECG voltage \( SV_1, 2 + RV_6 \) with left ventricular mass per square meter of body surface area, utilizing the total patient group. The horizontal and vertical dotted lines define the upper limits of normal for voltage and mass, respectively. The solid line represents the line of regression and the parallel dashed lines define the limits of the standard error.

Relationships generally similar to those shown in figure 3 also existed between \( SV_1 + RV_5, 6 \) and LVM/BSA: LVM/BSA = 77.10 + 2.47 \((SV_1 + RV_5, 6)\); \( s = 61.8 \text{ g/m}^2 \) and between the maximum \( R + S_V \) and LVM/BSA: LVM/BSA = 71.90 + 2.65 \((R + S_V)\); \( s = 66.1 \text{ g/m}^2 \).

It is of interest that \( SV_1 + RV_5, 6 \) and \( SV_1, 2 + RV_6 \) correlated as well with left ventricular stroke work as with mass. This is probably due to the relatively close relationship of stroke work to left ventricular mass as illustrated in figure 4, which shows LVM versus LVSW \((r = 0.64, P < 0.01)\). These voltages correlated less well with components of stroke work, that is, left ventricular systolic pressure \((r = 0.47 \text{ and } 0.41)\) and stroke volume \((r = 0.30 \text{ and } 0.38)\), possibly because each component influences cardiac dynamics and hypertrophy in a different manner.

Significant correlations between left ventricular wall thickness and QRS voltage were reported by Carter and Estes\(^{13}\) from a post-mortem study of adults; however, Hatam and associates\(^{15}\) in an angiocardiographic study in children, found no significant correlation between wall thickness and QRS voltage. As shown in table 1, left ventricular wall thickness in the present study has a significant correlation with precordial voltages \((P < 0.01)\), but not as close correlation as has mass. Wall thickness is but one determinant of left ventricular weight, the other major factor being chamber dimensions. It has been shown previously that pressure overloading of the left ventricle tends to increase wall thickness, with chamber volume remaining relatively normal ("concentric hypertrophy"); volume overloading tends to increase chamber size ("eccentric hypertrophy") with less marked increase in wall thickness.\(^{16, 22}\)

Figure 4

The relationship between left ventricular stroke work and left ventricular mass for the total patient population studied.

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Left ventricular end-diastolic volume may be increased by two general mechanisms sometimes occurring together in patients with heart disease: by enlargement secondarily to increased stroke volume from volume overload, or by dilatation in association with heart failure. In this context it was of interest to compare left ventricular end-diastolic volume to left ventricular mass for the total patient group. The relationship of these two variables, expressed as per square meter of body surface area, is shown in figure 5. Nineteen patients had hypertrophied ventricles without increased volume (upper left portion of the illustration), but only five patients had dilated left ventricles without significant hypertrophy (lower right portion). Four of these five had predominant mitral stenosis with some insufficiency of either the aortic or the mitral valve, and the EDV/BSA of these five was only slightly increased (mean 110 ml/m² as compared to upper normal of 102 ml/m²). These data suggest that, in patients with chronic heart disease, left ventricular hypertrophy nearly always accompanies an increased left ventricular end-diastolic volume.

It had been suggested that increasing left ventricular volume influences the ECG precordial voltage by decreasing the distance from the electrodes to the heart and by increasing the “solid angle” subtended by the ventricle at the precordial electrode. However, as seen in table 1, neither left ventricular end-diastolic volume nor the sum of end-diastolic volume and mass (“total left ventricular volume”) correlated better with the ECG voltages than did mass per se. Neither was the correlation improved by adjusting left ventricular mass for body surface area (LVM/BSA).

Comparison of the electrocardiographic with the anatomic or physiological variables in the four subgroups of patients (volume overload, pressure overload, aortic valve disease, left ventricular hypertrophy) revealed correlations similar for each subgroup (table 2) and slightly lower than that observed for the total group of patients as was shown in table 1. It was anticipated that separation of the patients into these more homogeneous subgroups would improve the correlations, since pressure overload and volume overload influence hypertrophy differently. However, the correlation between left ventricular mass and ECG voltages was not improved by including an expression of volume or by subdividing the patients according to the type of load stimulating hypertrophy. These results are consistent with those of Carter and Estes who found a significant correlation (P < 0.001) between precordial voltages and total

**Table 2**

<table>
<thead>
<tr>
<th>Correlation coefficients (r) for LVM/BSA versus SV₁ + RV₅₆</th>
<th>(n = 21) r = 0.37</th>
<th>(n = 9) r = 0.41</th>
<th>(n = 30) r = 0.33</th>
<th>(n = 75) r = 0.41</th>
</tr>
</thead>
<tbody>
<tr>
<td>Volume overload</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pressure overload</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aortic valve disease</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Increased left ventricular mass</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Table 3

Accuracy of Various ECG Voltage Criteria for Left Ventricular Hypertrophy

<table>
<thead>
<tr>
<th></th>
<th>Increased LVM (75 pts.)</th>
<th>Normal LVM (37 pts.)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>A. Grant’s criteria</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypervoltage</td>
<td>67</td>
<td>27</td>
</tr>
<tr>
<td>Normal voltage</td>
<td>33</td>
<td>73</td>
</tr>
<tr>
<td><strong>B. Sokolow's criteria</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypervoltage</td>
<td>68</td>
<td>27</td>
</tr>
<tr>
<td>Normal voltage</td>
<td>32</td>
<td>73</td>
</tr>
<tr>
<td><strong>C. Sokolow's criteria, substituting</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>40 mm for upper normal SV1 + RV5,6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypervoltage</td>
<td>57</td>
<td>16</td>
</tr>
<tr>
<td>Normal voltage</td>
<td>43</td>
<td>84</td>
</tr>
</tbody>
</table>

heart weight in an unselected autopsy study, but in a subgroup of hypertrophied hearts they found a lesser degree of correlation.

The intrinsicoid deflection or ventricular activation time (the time from onset of the QRS to peak positive deflection in lead V5 or V6) was described by Sokolow as prolonged past 0.05 sec in patients with left ventricular hypertrophy.² This is generally thought to be a result of a conduction defect from long-standing hypertrophy,¹⁰ ²⁵ and has not by itself been regarded as particularly useful in the diagnosis of left ventricular hypertrophy.⁵ ⁹ However, as shown in table 1, there is a positive correlation between the time of the intrinsicoid deflection and left ventricular mass (r = 0.50, P < 0.01). The time of the intrinsicoid deflection correlates most closely with “total left ventricular volume” (r = 0.58). It is of interest that Carter and Estes¹³ found the ventricular activation time prolonged in patients with congestive failure. Chronic failure results in enlarged end-diastolic volume plus increased left ventricular mass, the two components of “total left ventricular volume.”

The data in this study have also been analyzed to determine the accuracy of currently used methods for detecting left ventricular hypertrophy from the QRS voltage of the conventional electrocardiogram and to determine if more reliable electrocardiographic criteria might be developed. The wide scatter of values about the regression lines shown in figure 3 demonstrates that, although there is a significant correlation between the ECG voltages and left ventricular weight, this correlation is not close and one could not with
any degree of accuracy predict the amount of hypertrophy from the conventional electrocardiogram. In Table 3 are shown the percentages of patients having hypervoltage and normal voltage according to any of the criteria of Grant and of Sokolow for the group with normal values for left ventricular mass and for the group with left ventricular hypertrophy. Grant's criteria (A) and Sokolow's criteria (B), give virtually identical results. The 33% and 32%, respectively, of patients with increased left ventricular mass and normal voltage represent false-negative electrocardiograms; the 27% with hypervoltage despite normal left ventricular mass are false positives.

It has been suggested that age affects the accuracy of the ECG diagnosis of left ventricular hypertrophy and that voltage criteria are less accurate for adults under age 25. Only three patients in this study were under 25. However, 11 patients were under age 35; seven of these (64%) were correctly classified by Sokolow's criteria, and three (27%) had false positive ECGs. These relationships are not significantly different from those of the entire group.

If one substitutes 40 mm as the upper normal for \( SV_1 + RV_a \) rather than the 35 mm described by Sokolow (part C of Table 3), there were fewer false positives (16 versus 27%), but there are also fewer hypertrophied ventricles correctly classified by hypervoltage (57 versus 68%).

Of the 75 patients with anatomic hypertrophy, 68% had hypervoltage by at least one of Sokolow's criteria as listed previously. These findings are similar to those of Heine and associates, who reported these ECG criteria positive in 71% of 261 hearts classified as hypertrophied by x-ray. Allenstein and Mori found 71% of 17 patients with wall thickness greater than 10 mm at autopsy to have hypervoltage by Sokolow's criteria. Scott and co-workers found 85% of ECGs positive for these criteria in an autopsy study of 100 hearts with left ventricular wall thickness greater than 13 mm. Griepp found only 22% "Sokolow positive" ECGs in 300 hearts with left ventricular hypertrophy at autopsy. Selzer and co-workers described 261 patients with left ventricular hypertrophy at autopsy; 51% had a correct ECG diagnosis of left ventricular hypertrophy. In other studies the incidence of anatomic hypertrophy has been determined in patients with hypervoltage in the ECG. Selzer and associates found hypervoltage at autopsy in 69% of 105 patients with ECG hypervoltage. Grubhschmidt and Sokolow studied 101 patients with hypervoltage; 95% had clinical heart disease placing a strain on the left ventricle, and 70% had left ventricular hypertrophy apparent on plain chest x-rays. This range of figures reflects the difficulty in assessing hypertrophy of the left ventricle in the different patient populations studied.

No significant correlation could be found between the position of the long axis of the left ventricle at end-diastole and the mean QRS vector direction in the frontal plane \((P > 0.05)\). As seen in Figure 6, there was a wide range of mean QRS vector directions extending from right axis deviation to left axis deviation, with a relatively narrow range of anatomic axis direction \((31° \text{ to } 62°)\). These findings are similar to those of Grant, who found no correlation between the QRS vector direction and the direction of the anatomic left ventricular axis by utilizing angular measurements made at autopsy.

References


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