High-Voltage QRS Complexes in the Absence of Left Ventricular Hypertrophy

By Gordon R. Cumming, M.D., and William L. Proudfit, M.D.

Of an unselected group of otherwise normal electrocardiograms in which the sum of S in V₁ and R in V₅ exceeded 35 mm, 39 per cent came from patients with no clinical evidence of cardiac disease. Of electrocardiograms in which R in aVL exceeded 11 mm, 29 per cent were from patients without cardiac disease. All of the cases in which the voltage was abnormal in both lead aVL and the precordial leads had evident or possible heart disease. Thus, while the voltage indexes for left ventricular hypertrophy of Sokolow and Lyon are useful and easy to apply, the lack of specificity of voltage changes limits clinical application.

Electrocardiograms of patients who have left ventricular hypertrophy frequently demonstrate increased voltage in the left precordial leads. Scott and associates reported correlated necropsy data from cases of left ventricular hypertrophy with the various electrocardiographic criteria used for diagnosis of the condition, and concluded that those of Sokolow and Lyon were the most inclusive, indicating the abnormality in 80 per cent of cases. The criteria of Sokolow and Lyon differed from those of other authors chiefly with respect to the inclusion of defined voltage limits in the precordial leads. Although the sensitivity of the criteria was satisfactory, the important question of specificity was not investigated. Subsequently Seltzer and associates reported that when 108 electrocardiographic tracings indicative of left ventricular hypertrophy were correlated with the postmortem findings of each case, there were 17 false-positive and 16 questionable results. In 14 of the 17 false-positive findings the voltage index of the precordial leads was abnormal and 12 of the 16 borderline cases had abnormal indices. The results of study of postmortem material may not be strictly applicable to clinical experience for various reasons, but it is evident that false-positive results are common when precordial voltage changes are used as a criterion of left ventricular hypertrophy. Recently Grubschmidt and Sokolow reported that in from 95 to 98 per cent of patients whose electrocardiograms were abnormal only with respect to the RS voltages in various leads there was clinical evidence of left ventricular hypertrophy. Prior to the above publication a similar analysis at the Cleveland Clinic disclosed that as high as 40 per cent of such electrocardiograms come from normal patients. The disparity in findings seems worthy of note.

Method of Selection

One hundred and forty-three electrocardiograms, selected because of voltage changes only, were divided into 3 groups.

Group 1. Electrocardiograms (94) in which the sum of S in lead V₁ plus R in lead V₅ exceeded 35 mm. The records were normal in all other respects, save that in 7 cases the intrinsicoid deflection was inscribed between 0.05 and 0.06 second after the onset of the QRS complex. In none of these cases did the voltage of R on aVL exceed 11 mm.

Group 2. Electrocardiograms (34) in which the voltage of R in lead aVL exceeded 11 mm. In no case did the onset of the intrinsicoid deflection exceed 0.05 second, nor did the sum of S and R in leads V₁ and V₅ exceed 35 mm.

Group 3. Electrocardiograms (15) in which the sum of the S in V₁ and the R in V₅ exceeded 35 mm, and the R in aVL exceeded 11 mm, but which otherwise were normal.

The charts of the 143 patients were reviewed for clinical causes of left ventricular hypertrophy, and the radiologists' reports concerning heart size on 6-foot roentgenograms were noted. These patients were further divided into 3 clinical subgroups.
HIGH-VOLTAGE QRS COMPLEXES

TABLE 1.—Correlation of Voltage Value and Clinical Cardiovascular Status, from 143 Electrocardiograms*

<table>
<thead>
<tr>
<th>Group</th>
<th>Criteria</th>
<th>Clinical cardiovascular status (subgroup)</th>
<th>No. of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>A</td>
<td>B</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Normal (%)</td>
<td>Definite left ventricular hypertrophy (%)</td>
</tr>
<tr>
<td>1</td>
<td>S V₁+R V₅ &gt; 35 mm.</td>
<td>94</td>
<td>39</td>
</tr>
<tr>
<td>2</td>
<td>R aV₅ &gt; 11 mm.</td>
<td>34</td>
<td>29</td>
</tr>
<tr>
<td>3</td>
<td>S V₁+R V₂ &gt; 35 mm.</td>
<td>15</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>R aV₁ &gt; mm.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Selected because of voltage changes.

Subgroup A. No clinical evidence of cardiovascular disease.

Subgroup B. Definite clinical evidence of cardiovascular disease or left ventricular hypertrophy.

Subgroup C. Questionable evidence of cardiovascular disease. This group had no evidence for the diagnosis of left ventricular hypertrophy other than transient elevations of blood pressure of not greater than 180/100 mm. Hg. Normal pressures were obtained on 2 subsequent readings without further observations of the hypertension.

All patients were more than 25 years of age; the majority were between 40 and 60 years of age.

RESULTS

Clinical Evidence of Left Ventricular Hypertrophy. These data are given in table 1. In group 1, 39 per cent of patients, in group 2, 29 per cent, and in group 3, no cases, were in subgroup A (showed no clinical evidence of cardiovascular disease). In group 3, 93 per cent of patients were in subgroup B (had definite clinical evidence of left ventricular hypertrophy).

Voltages in Group 1. The ranges and average values for this index are given in table 2. While patients in group 1 had higher voltages than did those in group 2 and 3, there is considerable overlapping. If all voltages under 40 mm. were to be considered normal, then 19 per cent of the electrocardiograms still would come from normal patients and the evidence for left ventricular hypertrophy would be questionable in an additional 6 per cent. At the same time, this extension of the normal range would exclude 25 per cent of subgroup B.

Voltages of R in V₅ Exceeding 26 mm. In group 1, 44 patients were in this category, 30 per cent of whom were in subgroup A, and 15 per cent in subgroup C. This index, then, is only slightly more selective, and at the same time is absent in many cases in which there is left ventricular hypertrophy.

Roentgenograms. Of group 2, 22 per cent, and of group 3, 60 per cent, had radiologic evidence of left ventricular hypertrophy. All of these cases belonged to subgroup B.

DISCUSSION

There is as yet no large published series of normal values for the precordial leads, so that the frequency distribution of the lead voltage is not known. One possible explanation for the considerable difference in results between the series of Grubschmidt and Sokolow and this series is that the latter was derived from clinic as well as hospital cases, and many patients in the clinic group are normal adults seen for regular periodic examinations. Furthermore, the cases in the report of Grubschmidt and Sokolow were not further divided into subgroups, and more of their patients may have fitted into group 3 of this present series (abnormal voltages in the precordial leads as well as lead aV₁), in which the incidence of clinical left ventricular hypertrophy was very high. Some of the patients of these authors would be placed in the questionable group in this series. Whether patients with transient mild elevations in blood pressure may be said to have left ventricular hypertrophy is highly debatable. It has been observed that the infusion of norepinephrine to
raise the blood pressure from normal to 180/90 mm. Hg, or of epinephrine to raise cardiac rates from about 68 to about 100, may cause an increase in the R and S voltages in leads V₁ and V₅ of up to 20 per cent. Similarly, electrocardiographic voltages might be elevated from normal to "abnormal" by emotional strain.

Cases in which both the precordial and the left arm leads showed increased voltage had a higher incidence of clinically evident left ventricular hypertrophy. It would seem that in the presence of left axis deviation the increase of the sum of S in V₁ and R in V₅ to more than 35 mm. is of much greater significance than an electric axis with a value greater than zero.

This lack of exact correlation between RS voltage and left ventricular hypertrophy is not surprising: the position of the electrode is independent of the position of the heart; the thickness of the thoracic wall and the proximity of the heart to the thoracic wall vary; only the outer half of the ventricular wall is said to be responsible for the RS complex; the area under the deflection is more indicative of the forces generated than is the height of the deflection alone; and, finally, it remains to be proved that the electric force increases in proportion to the increase in muscular mass.

**Summary**

Of an unselected group of "otherwise normal" electrocardiograms in which the sum of S in V₁ and R in V₅ exceeded 35 mm., 39 percent came from patients with no clinical evidence of cardiac disease. Similarly, 29 percent of electrocardiograms in which R in aV₅ exceeded 11 mm. were from patients without cardiac disease. All of the patients in whom the voltage was abnormal in lead aV₅ and the precordial leads had evident or possible heart disease. Thus, while the voltage indexes for left ventricular hypertrophy presented by Sokolow and Lyon³ are useful and easy to apply, the lack of specificity of voltage changes seriously limits the clinical application.

**Summario in Interlingua**

In un non-seligite gruppo de "alteremente normal" electrocardiagrammas in que le summa de S in V₁ e R in V₅ exceedeva 35 mm, 39 pro cento pertineva a patientes sin evidentia clinica de morbo cardiac. Similmente, 29 pro cento del electrocardiagrammas in que R de aV₅ exceedeva 11 mm pertineva a patientes sin morbo cardiac. Omne le patientes in qui le voltage esseva anormal in aV₅ e in le derivaciones precordial esseva subjectos con evidente o possibile morbo cardiac. Assi, ben que le indices de voltage pro hypertrophia sinistro-ventricular presentate per Sokolow e Lyon³ es utile e facile a applicar, le manco de specificitate del alterationes de voltage restrinche seriemente lor application clinica.

**REFERENCES**

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