Serial Electrocardiographic Changes in Treated Hypertensive Patients with Reference to Voltage Criteria, Mean QRS Vectors, and the QRS-T Angle

By Philip L. Dern, M.D., Ray Pryor, M.D., Strother H. Walker, Ph.D., and Donald T. Searls, Ph.D.

SUMMARY
Serial changes in voltage, QRS vectors, and QRS-T angles were studied in 44 patients who had shown improvement in their electrocardiograms during treatment for hypertension. Changes were observed in a number of electrocardiographic voltage criteria often used in the study of left ventricular hypertrophy. This is consistent with a change in the magnitude of the mean QRS vector and its reflection on a variety of ECG leads even if pretreatment records were normal. Improvement also occurred in the mean T vectors and the QRS-T angle. It is proposed that serial changes in the ECG might be of greater value than pretreatment abnormalities in diagnosing the disorder of cardiac structure or function that occurs in hypertension.

Additional Indexing Words:
Antihypertensive agents Ventricular hypertrophy
Heart enlargement Electrocardiography

It seems possible that study of the electrocardiogram during treatment of hypertension might show whether a diseased myocardium existed before treatment began. If patients with left ventricular hypertrophy or some associated abnormal functional cardiac state have favorable changes in the ECG during antihypertensive therapy, then these serial improvements would have diagnostic value even if the pretreatment tracings were within normal limits. These considerations led to the study of 44 patients having electrocardiographic improvement during treatment of hypertension.

Methods
Records of patients coming to the hypertension clinic for treatment were examined and those selected were ones in which at least two electrocardiograms were available and in which post-treatment ECGs were thought to show improvement in the standard voltage criteria for left ventricular enlargement. The “control” ECG was considered to be the record showing the most voltage criteria of greatest magnitude. The blood pressure taken on the date nearest to that of the control ECG was called the “control” blood pressure. If a number of blood pressures were recorded on this date, the mean of the readings was used. The “post-treatment” ECG was considered to be the record taken subsequent to the control ECG that showed the most voltage criteria of smallest magnitude. The “post-treatment” blood pressure was the pressure recorded nearest the date of the post-treatment ECG. Properly standardized electrocardiograms were used in all cases. The following voltages were used as criteria for left ventricular enlargement: height of $R_1 + \text{depth of } S_{111} > 25 \text{ mm}^1$; height of $R$ in $aV_L > 11 \text{ mm}^2$; height of $R$ in $aV_r > 20 \text{ mm}^2$; depth of $S$ in $V_1$ more than $26 \text{ mm}^4$; depth of $S$ in $V_1$ + height of $R$ in $V_5$ (or $V_6$ if taller) above $35 \text{ mm}^2$; height of

From the Departments of Medicine (Division of Cardiology) and Preventive Medicine of the University of Colorado School of Medicine, Denver, Colorado.
tallest R + depth of deepest S in precordial leads (Rmax + Smax) above 45 mm² and tallest R + deepest S in any one precordial lead (R + Smax) greater than 35 mm.° The directions of the mean QRS and the T vectors were determined in the frontal and horizontal planes. The angle between the mean QRS and T vector was also recorded for each plane. The direction of the mean QRS or T vector was assumed to be 90° from the lead showing transitional complexes for either item. The standard frontal plane triaxial reference diagram was used with the positive side of limb lead I listed as zero degrees. The positive side of V₄ was put at zero degrees for horizontal plane measurements. The height, weight, and ponderal index were determined on the dates closest to those of the control and post-treatment ECGs. Ponderal index, which is the height in inches divided by the cube root of the weight in pounds, was used as a measure of body build.°

Results

Relation of Pretreatment Voltage Criteria and Blood Pressure

Before treatment the systolic blood pressure was significantly correlated with the magnitude of R₁ + SIII; S₁ + Rv₅₋₆ and RavL; the last was the criterion giving the best correlation (r = 0.40, P < 0.01).

In addition to the above criteria, the control diastolic blood pressures correlated significantly with the levels of Rmax + Smax and R + Smax. The strongest correlations for diastolic blood pressure were with S₁ and S₁ + Rv₅₋₆ (r = 0.48, P < 0.01 for both).

Voltages were within normal limits for many of the criteria which correlated with pretreatment blood pressures.

Changes in Voltage Criteria with Treatment

A fall in systolic blood pressure significantly correlated with a decrease in voltage of RavL, the criterion which related best with the control systolic pressure, even though the majority of values for RavL were within normal limits. A decrease in diastolic pressure was significantly correlated with a decrease in S₁ + Rv₅₋₆, one of the criteria relating best to control diastolic blood pressure.

Significant correlations did not exist between post-treatment values for voltage criteria and post-treatment diastolic blood pressure. However, if the post-treatment systolic pressure remained relatively high, voltages were significantly higher in R₁ + SIII and RavL but still usually well within normal limits.

Relation Between Changes in Precordial Voltage Criteria and the Standard Limb and Unipolar Lead Criteria

A change in a precordial voltage criterion during treatment of hypertension could be due to changes in chest thickness or to improvement in the heart. The latter seemed more likely because as the precordial criterion S₁ + Rv₅₋₆ changed, a significant improvement occurred in R₁ + SIII and RavL criteria not expected to be altered by variations in chest wall thickness.

Ponderal Index, Changes in Blood Pressure, and Interaction with a Precordial Voltage Criterion

Changes in blood pressure during treatment were not significantly related to changes in body build as measured by ponderal index. Before treatment the correlation between diastolic blood pressure and the magnitude of S₁ + Rv₅₋₆ was exactly the same (r = 0.48, P < 0.01) before and after removing the effect of ponderal index by partial correlation analysis. Therefore, pretreatment diastolic blood pressure seemed to be a more important factor in influencing the control values S₁ + Rv₅₋₆ than body build was in this group of patients.

Individual Control Voltage Criteria Versus Post-treatment Changes

Since the basis for inclusion of a patient in this study rested upon an improvement in the ECG during treatment, demonstration of improvement in the ECG was not surprising. The notable feature was that so many control tracings had normal voltage criteria. Pretreatment values for limb lead and unipolar lead criteria, R₁ + SIII, RavL, and RavF, were usually normal despite the fact that improvements occurred during therapy. Control precordial voltage criteria were more frequently abnormal. Post-treatment improvements in voltage criteria were more common than pretreatment abnormalities suggesting that a retrospective evaluation of the effect of treatment upon the ECG might best be done...
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Figure 1

Upper portion of this diagram shows the number of patients having various values for $R_1 + S_2$ before treatment. (Sum of the voltage of R in standard lead I and S in standard limb lead III). In the bottom portion of the diagram are listed the number of patients having various increases or decreases in voltage of $R_1 + S_3$ after therapy.

before deciding whether a given patient had cardiac involvement. The contrast between the scarcity of pretreatment abnormality and the frequency of post-treatment improvement is illustrated in figures 1 to 4. The frequency of abnormal pretreatment values for each of the seven voltage criteria is given in table 1.

Control Versus Post-treatment Combinations of Voltage Criteria

The diagnosis of left ventricular hypertrophy might be stronger if a number of positive voltage criteria exist rather than a few. With this possibility in mind it was interesting to see that the study of improving post-treatment ECGs allowed more criteria to be of use in individual patients than if only the pretreatment records were evaluated. The distribution of patients having specific numbers of abnormal voltage criteria on control ECGs is shown in table 2, which shows that no patient had all seven abnormal voltage criteria before treatment. However, after treatment almost all patients had five or more improving criteria and 13 patients had shown improvement in all seven (table 3). If worsening criteria, and there were a few, are subtracted from improving criteria, the number of patients with multiple improving criteria remains striking (table 4).

The specific combinations of abnormal voltage criteria in the control ECGs are listed in table 5. Table 6 shows the specific combinations of improving criteria after therapy. The number of patients having favorable changes in many voltage criteria is notable.

Mean QRS Vectors and Blood Pressure

The higher the control value for the systolic blood pressure, the more leftward was the direction of the mean frontal QRS vector ($r = 0.37, P < 0.05$). The higher the pretreatment diastolic blood pressure, the more posterior was
Distribution of pretreatment values and post-treatment changes in \( R_{AVF} \) (height of \( R \) wave in lead \( aVF \)) are listed as in figure 1.

The direction of the mean horizontal QRS vector \((r = 0.34, P < 0.05)\).

**Changes in the Mean QRS Vectors with Treatment**

The mean QRS vector in the frontal plane did not change direction significantly during treatment. Unlike the behavior of the frontal plane vector, the horizontal mean QRS vector significantly changed during therapy. Pretreatment and post-treatment values for this vector were correlated \((r = 0.51, P < 0.005)\) and the regression slope of post-treatment on pretreatment values indicated a significant anterior

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**Table 1**

*Frequency of Individual Abnormal Voltage Criteria (Controls)*

<table>
<thead>
<tr>
<th>( R_{III} &gt; 25 )</th>
<th>( R_{AVL} &gt; 11 )</th>
<th>( R_{AVP} &gt; 20 )</th>
<th>( S_{V1} &gt; 26 )</th>
<th>( S_{V1} + R_{V5-6} &gt; 35 )</th>
<th>( R_{max} + S_{max} &gt; 45 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>( N ) patients*</td>
<td>4</td>
<td>6</td>
<td>1</td>
<td>13</td>
<td>36</td>
</tr>
</tbody>
</table>

*The number of patients having certain abnormal voltage criteria before treatment are listed.

**Table 2**

*Combinations of Abnormal Voltage Criteria (Control Electrocardiograms)*

<table>
<thead>
<tr>
<th>Number of abnormal criteria</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
</tr>
</thead>
<tbody>
<tr>
<td>( N ) patients*</td>
<td>5</td>
<td>6</td>
<td>11</td>
<td>8</td>
<td>12</td>
<td>1</td>
<td>1</td>
<td>0</td>
</tr>
</tbody>
</table>

*The number of patients having varying numbers of abnormal voltage criteria before treatment are listed.
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Table 3

Combinations of Improving Voltage Criteria
(Post-treatment Electrocardiograms)

<table>
<thead>
<tr>
<th>Number of improving criteria</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
</tr>
</thead>
<tbody>
<tr>
<td>N patients*</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>10</td>
<td>20</td>
<td>13</td>
</tr>
</tbody>
</table>

*The number of patients with varying numbers of improving voltage criteria are listed. Any decrease in voltage is considered an improvement and a worsening voltage is considered to be no change.

Table 4

Combinations of Improving Voltage Criteria
(Post-treatment Minus Worsening)

<table>
<thead>
<tr>
<th>Number of improving criteria</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
</tr>
</thead>
<tbody>
<tr>
<td>N patients*</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>5</td>
<td>10</td>
<td>13</td>
<td>13</td>
</tr>
</tbody>
</table>

*In this table the number of patients having varying numbers of improving voltage criteria after treatment are listed. Worsening criteria are subtracted from improving criteria.

Table 5

Frequency of Combinations of Abnormal Voltage Criteria in Control Electrocardiograms*

<table>
<thead>
<tr>
<th>Combinations</th>
<th>Patients</th>
<th>Key to combinations</th>
</tr>
</thead>
<tbody>
<tr>
<td>1,2,4,5,6,7</td>
<td>1</td>
<td>None = none abnormal</td>
</tr>
<tr>
<td>2,5,6,7</td>
<td>1</td>
<td>1 = R1 + SIII abnormal</td>
</tr>
<tr>
<td>3,4,5,7</td>
<td>1</td>
<td>2 = RL abnormal</td>
</tr>
<tr>
<td>1,2,5,6</td>
<td>1</td>
<td>3 = RavP abnormal</td>
</tr>
<tr>
<td>4,5,6</td>
<td>1</td>
<td>4 = SvI abnormal</td>
</tr>
<tr>
<td>2,5</td>
<td>1</td>
<td>5 = SvI + Rv5-6 abnormal</td>
</tr>
<tr>
<td>1,2,4,5,6,7</td>
<td>2</td>
<td>6 = Rmax + Smax abnormal</td>
</tr>
<tr>
<td>None</td>
<td>5</td>
<td>7 = R + Smax</td>
</tr>
<tr>
<td>5</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>5,6,7</td>
<td>7</td>
<td></td>
</tr>
<tr>
<td>4,5,6,7</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>5,6</td>
<td>10</td>
<td></td>
</tr>
</tbody>
</table>

*The number of patients with specific combinations of abnormal voltage criteria before treatment are listed. For example, 10 patients had abnormalities both SvI + Rv5-6 and Rmax + Smax.

movement of this vector during treatment. This implies to the authors that the control values for the mean horizontal QRS vector were abnormally posterior for these particular patients.

The QRS-T Angles and Blood Pressure

Before treatment the horizontal and frontal plane QRS-T angles did not correlate with the level of the blood pressure although a number of patients had abnormally wide angles.

Changes in the QRS-T Angle with Treatment

The QRS-T angle decreased significantly with falls in the systolic blood pressure. (Decrease in systolic BP and frontal QRS-T angle: r = 0.48; decrease in systolic BP and decrease in horizontal QRS-T angle: r = 0.39, P < 0.01 for both.) A fall in diastolic blood pressure correlated significantly only with a decrease in the frontal QRS-T angle (r = 0.35, P < 0.05).

In some patients the decrease in the QRS-T angle associated with therapy was largely due

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to movement of the mean QRS vector. In other patients a predominant alteration in the direction of the mean T vector explained a decrease in the angle. Combined effects accounted for changes in the remainder of the patients. QRS-T angle changes during treatment must be examined, therefore, as to whether they are largely due to T-vector alteration, and hence an implied change in "ischemia," or whether the angle change is due to movement of the QRS vector, in which case hypertrophy might be the more important variable.

Changes in the Mean Horizontal QRS Vector and QRS-T Angle During Therapy in Patients Having Initial Normal Voltage Criteria

Five patients were found in whom all standard voltage criteria for left ventricular hypertrophy were normal before treatment. A wide horizontal plane QRS-T angle (greater than 60°) was present in four of these patients before treatment and improved after therapy in three. One notable patient had normal values for all criteria before treatment but showed marked improvement in voltage criteria, QRS-T angles, and direction of the mean horizontal QRS vector after therapy. The horizontal QRS-T angle changed well within the normal zone in these patients and post-treatment values of 0 to 40° occurred.

Time Relationships Between Maximal Change in Voltage Criteria and Maximal Change in QRS-T Angles

Twenty-five patients had a sufficient number of ECGs taken during the period between maximal and minimal values for voltage criteria for a study of the possibility of a discordant relationship in the time of maximal voltage and QRS-T angle change. Of these patients, seven had QRS-T angles in frontal or horizontal plane which decreased maximally 3 to 26 months before maximal improvement in voltage criteria. The early QRS-T angle changes were not transient. Study of the components of the QRS-T angle showed that the early improvement was usually due to T-vector movement. This was consistent with an early decrease in "ischemia."

Discussion

While certain electrocardiographic voltage magnitudes tend to be elevated in the presence of hypertensive heart disease,1-8 the possibility that a pathological state exists before the upper limits of normal are exceeded is often neglected. The results of this study suggest that the serial changes in the electrocardiogram during antihypertensive therapy may be more striking than the abnormalities present in the same patients' records before treatment. Serial improvements occur in individual voltage criteria, combinations of these criteria, direction of the mean horizontal QRS vector, and the QRS-T angles even though values for these are within normal limits before treatment.

Numerous difficulties beset one who would like to assign a diagnostic limit for ECG criteria in left ventricular enlargement. False positive diagnoses occur in emaciated subjects,9 and obesity may increase the distance from electrode to heart or otherwise alter voltages in the precordial leads. An attempt to correct ECG voltage criteria for body build has been made by Kilty and Lepeschkin,10 but a notable variation of voltages exists in persons of similar body build. Young persons may have high precordial voltages in the absence of left ventricular enlargement. If, as we believe, the mean QRS vector reflects many ECG leads and if the magnitude of this vector, or the direction of it, changes as left ventricular enlargement recedes, then serial changes in a variety of ECG leads ought to occur regardless of such factors as body build. Symons and Wahl11 found that the diagnosis of left ventricular enlargement was difficult in greatly enlarged or transverse hearts, and Evans12 used a special lead system to avoid this. We wonder if these situations need give difficulty if serial post-treatment changes in the electrocardiogram are studied before a diagnosis is made.

We call attention to a decrease in the QRS-T angle with therapy, even in the absence of initial abnormal voltage criteria, and suggest that, before ascribing a QRS-T angle
change to ischemia or hypertrophy, one determines whether this change is due to movement of the QRS or the T vector. It is also apparent that changes in the T vector are not simply directly related to QRS changes since a number of patients are known to have improvements in T vectors which antedate QRS changes.

In the best of all possible circumstances a decrease in a voltage criterion, direction of the horizontal QRS vector, or magnitude of the QRS-T angles would be directly equivalent to a decrease in anatomic left ventricular hypertrophy. Unfortunately, how often this is actually true is not known and some disturbing exceptions exist. We have seen the voltage criteria for left ventricular enlargement appear within hours after the onset of hypertension from renal artery embolus, making it difficult to believe that anatomic thickening of the left ventricle could be present. Rapid lowering of the blood pressure is said to be associated with immediate lowering of QRS voltages. In certain cases conduction disturbances may be the basis for the electrocardiographic manifestation of left ventricular hypertrophy. Even so, perhaps one should reserve opinion as to whether a given hypertensive patient has left ventricular disease until serial post-treatment graphs have been examined. These serial improvements, which may be well within the accepted normal zone for diagnostic criteria, very likely relate to the opposite changes in the ECG which occur during the development of hypertensive heart disease. If so, justification for routine electrocardiography as part of periodic multiphasic health screening programs would be strong.

Acknowledgment

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References

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