Frontal and Sagittal Electrocardiograms of Normal and Hypertensive Subjects during an Experimentally Produced Phase of Lowered Blood Pressure

By Joseph Brumlík, M.D., and Charles E. Koosmann, M.D.

Reduction of the blood pressure in normal and hypertensive subjects by intravenous pyrogen caused the electrocardiograms to assume a more abnormal configuration as judged by present criteria of electrocardiographic normality. A temporary reduction of the blood pressure by the intravenous administration of tetraethylammonium chloride caused only minor and variable modifications of the electrocardiogram. The results of the experiments as done do not support the concept of strain as applied to certain electrocardiographic abnormalities which occur in the course of hypertension.

Patients with hypertension often have electrocardiograms which are characterized by a high voltage of QRS and deviation of its mean axis to the left, a depression of the S-T segment in leads I, II and V₅, and lowering or inversion of the T wave in leads I, V₅, and sometimes lead II.⁹,¹⁻⁻¹ Similar abnormalities and a late RS deflection may be encountered in leads from the left side of the precordium. The changes, particularly in the final ventricular deflections, have been ascribed to cardiac "strain."¹⁰⁻⁻¹² This mechanical interpretation is strengthened by the observation that the abnormalities, particularly in the T wave, sometimes disappear after sympathectomy, or after therapeutic measures which presumably cause a prolonged fall in blood pressure.⁴⁻⁻⁶

It seemed proper to investigate the possibility of reversing the electrocardiographic abnormalities found in certain hypertensive patients by lowering the blood pressure acutely. With this in mind, the blood pressure was reduced in hypertensive patients and in normal control subjects by the intravenous injection of typhoid vaccine or of tetraethylammonium chloride (Etamon).⁶,⁹

Technic

In order to prevent the febrile phase of the pyrogenic reaction, the patients who were to receive the vaccine were given 2.4 to 3.6 Gm. of amidopyrine during the preceding 12 hours. Control electrocardiograms were obtained before and after amidopyrine. Triple typhoid vaccine,† 0.1 cc., was given intravenously in the morning with the patient in a basal state. Pulse rate, rectal temperature and blood pressure measured with the ordinary mercury sphygmomanometer were recorded before and for every hour of the first day after the injection of the vaccine with the patient recumbent. Tracings were made when a significant fall in blood pressure occurred, which was usually two to five hours after the injection. The blood pressure generally returned to its control level the next day; occasionally the process took several days. As long as the blood pressure remained low, additional electrocardiograms were obtained, and as a rule a tracing was taken when the blood pressure finally returned to its control level.

In addition, in some patients not under basal conditions the blood pressure was reduced by the intravenous injection of 4 cc. of tetraethylammonium chloride (400 mg.).

Observations using triple typhoid vaccine were made on 15 patients (11 women and 4 men), and on five men with normal blood pressure (tables 1

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* Kindly supplied to us by Parke, Davis, and Co., Detroit, Mich.
† New York City Department of Health Standard Triple Typhoid Vaccine.

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and 2, figs. 1, 2, and 3). Observations using tetraethylammonium chloride were made on six patients (four women and two men).

Electrocardiograms of the patients receiving vaccine were recorded with a Hindle all-electric electrocardiograph. Electrocardiograms of patients receiving tetraethylammonium chloride were made with a Technicon cardiograph. Changes in blood pressure with the latter drug are so evanescent that multiple leads must be recorded rapidly, a purpose for which the Technicon instrument is well suited. Even with it, however, it was not possible to make as complete an electrocardiographic survey as in the patients receiving the longer acting pyrogen.

The electrocardiograms recorded were: the standard, the unipolar limb, and the usual six unipolar precordial leads; Arrighi's leads* in the sagittal plane; and the potential of the posterior angle of his triangle. In the experiments with tetraethylammonium chloride only the standard leads and the extremity and precordial potentials could be recorded during the brief action of the drug.

The position of the patient when records were made was either recumbent or sitting in bed, but in any one series was constant. None of the patients was taking digitalis or any other drugs known to affect the electrocardiogram, and none was in cardiac failure.

The areas of electrocardiographic deflections were measured by a special technic. The base line just preceding the P wave of the record was regarded as the reference level. Frequent, minute perforations were made in the record itself, the frequency being dictated by the rapidity of change of contour. With the aid of an Elliott comparator the exact time and height or depth of the perforations were determined, corrections made for inaccuracies of standardization, and the curve redrawn on an enlargement of approximately five times. This method obviated the necessity for making corrections for varying speeds of the electrocardiographic camera. Areas were calculated from simple geometric relationships in the reconstructed curve, each deflection being reduced to triangles and quadrangles. Difficulties were encountered as a result of displacement of the P-R segment which was almost always present, and certain assumptions had to be made occasionally with regard to termination of the T (T wave) wave. All QRS deflections were measured from the P-R segment as a base line; the levels of the S-T junction and segment, and of the T wave, were referred to the true isoelectric level (U-P interval).

The angle alpha in the frontal (α) and sagittal (α') planes and the length of the vectors concerned were determined with the aid of Goodman's 42 mechanical adaptation of Carter, Richter, and Greene's chart. 40 The two leads in each plane used for calculations were those which displayed well defined deflections easy to measure.

**Results**

**With Vaccine**

A. Normal Subjects (Tables 1 and 2, Fig. 1). In the five normal control subjects there was a mean decrease in systolic pressure of 24 mm. Hg (110 mm. to 86 mm.), and of 15 mm. Hg in diastolic pressure (69 mm. to 54 mm.).

In the frontal plane the length of the QRS vector shortened on the average 18.7 per cent [4.8 microvolt-seconds (μvs.)] but changes in direction were slight (fig. 1, open circles and dashed arrows), usually counterclockwise (leftward) with one exception, and the greatest deviation was 13.4 degrees.† In the exception

* See footnote, left column, this page.

† In the coordinate system used in electrocardiography it is well known that the sign of the y axis is the reverse of usual mathematical procedure. Further, an electrical axis with an angle alpha of −10 degrees is regarded as being deviated farther to the left than one with an angle of 0 degrees. This, too, is contrary to the usual terminology of polar coordinates in which the greatest deviation to the left is at 0 degrees. Geometrically a change from 0 in either a clockwise or counterclockwise direction through any part of 180 degrees is really a deviation of the axis concerned to the right of its original position. The electrocardiographic method of nomenclature with regard to the frontal plane, including the unfortunate use of "deviation" for "direction," is so universally used that a change at this time seems undesirable. However, when a mean manifest potential changes its direction in experiments such as reported, the nomenclature often breaks down. For example, it is difficult to say in which direction deviation has occurred, right or left, when the angle α changes from −160 to −120 degrees. The least confusing adjectives to describe deviation in the frontal
FRONTAL AND SAGITALL ELECTROCARDIOGRAMS

(subject Zuc), the angle underwent a change but the vector was small and the direction of plane are clockwise and counterclockwise, and these are used throughout this paper. In a paper of a similar kind, deviations of the mean manifest potential in the frontal plane were determined from the observer's point of view, that is, to his right or to his left.

With regard to the sagittal plane, any nomenclature adopted is unhindered by precedent or previous usage. In this paper a horizontal axis directed posteriorly as viewed from the left side of the subject is said to display a sagittal angle alpha (α) of 0 degrees; when directed anteriorly it is 180 degrees. Deviations away from the former in either change was uncertain. Excluding this case, the average counterclockwise deviation was 4.8 degrees. In four of the five normal subjects the shortening of the QRS vector in the sagittal plane averaged 11.8 per cent (3.5 microvolt-seconds). Deviation in this plane was consistently posterior (counterclockwise) and averaged direction are forward; deviations away from the latter in either direction are backward. When the deviation of an axis, compared to its original position, is counterclockwise in the sagittal plane viewed from the left the angle of change is preceded by a minus sign (table 1). It is realized that this is also contrary to mathematical usage.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Blood Pressure (mm Hg)</th>
<th>Heart Rate (beats per min)</th>
<th>Frontal (xy)</th>
<th>Sagittal (yz)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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<td>QRS</td>
<td>T</td>
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<td>A   a</td>
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<td>5 Normal Subjects (before pyrogen)</td>
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<tr>
<td>Car.</td>
<td>100 60 78</td>
<td>34.6 55.6 32.0</td>
<td>25.0 64.4 41.0</td>
<td>27.6 77.8 37.4</td>
</tr>
<tr>
<td>McE</td>
<td>110 78 76</td>
<td>18.6 70.2 27.2</td>
<td>57.2 45.2 62.4</td>
<td>33.6 69.6 19.5</td>
</tr>
<tr>
<td>Mos.</td>
<td>110 70 82</td>
<td>29.6 68.8 41.2</td>
<td>54.2 70.0 60.1</td>
<td>48.8 103.0 30.8</td>
</tr>
<tr>
<td>Mul.</td>
<td>120 70 66</td>
<td>39.0 78.8 35.0</td>
<td>53.0 72.2 66.4</td>
<td>8.8 -22.1 41.0</td>
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<tr>
<td>Zuc.</td>
<td>110 65 61</td>
<td>6.6 104.7 27.0</td>
<td>30.2 29.5 42.6</td>
<td>31.8 41.2 54.4</td>
</tr>
<tr>
<td>Mean</td>
<td>110 69 73</td>
<td>25.7 75.5 32.5</td>
<td>43.3 56.3 54.4</td>
<td>29.7 63.4 32.2</td>
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15 Hypertensive Patients (before pyrogen)

<table>
<thead>
<tr>
<th>Patient</th>
<th>Blood Pressure (mm Hg)</th>
<th>Heart Rate (beats per min)</th>
<th>Frontal (xy)</th>
<th>Sagittal (yz)</th>
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<td>QRS</td>
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<tr>
<td>Bry.</td>
<td>114 110 80</td>
<td>44.4 3.0 13.2</td>
<td>58.7 53.0 15.2</td>
<td>33.6 19.0 37.4</td>
</tr>
<tr>
<td>Byr.</td>
<td>110 115 72</td>
<td>3.0 46.2 3.5</td>
<td>123.2 5.1 88.2</td>
<td>18.0 73.0 10.0</td>
</tr>
<tr>
<td>Cham.</td>
<td>255 170 108</td>
<td>59.0 10.0 42.4</td>
<td>143.0 43.3 55.9</td>
<td>25.2 68.2 15.7</td>
</tr>
<tr>
<td>Chap.</td>
<td>275 130 73</td>
<td>62.4 8.8 23.7</td>
<td>-177.0 39.1 12.7</td>
<td>15.6 -32.8 5.5</td>
</tr>
<tr>
<td>Del.</td>
<td>222 136 83</td>
<td>53.0 -3.8 73.6</td>
<td>169.5 23.6 154.5</td>
<td>18.0 73.0 10.0</td>
</tr>
<tr>
<td>Did.</td>
<td>188 95 35</td>
<td>30.0 32.2 51.8</td>
<td>51.1 -9.0</td>
<td>25.2 68.2 15.7</td>
</tr>
<tr>
<td>Get.</td>
<td>210 115 69</td>
<td>44.8 -12.6 8.5</td>
<td>112.5 40.3 -1.7</td>
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</tr>
<tr>
<td>Hol.</td>
<td>160 125 70</td>
<td>48.6 1.7 3.0</td>
<td>-10.0 51.7 1.0</td>
<td>15.6 -32.8 5.5</td>
</tr>
<tr>
<td>Jon.</td>
<td>160 90 84</td>
<td>30.0 10.4 25.4</td>
<td>-97.0 32.8 -36.5</td>
<td>18.0 73.0 10.0</td>
</tr>
<tr>
<td>McO.</td>
<td>162 92 79</td>
<td>26.3 -26.4 4.4</td>
<td>86.3 25.0 -16.6</td>
<td>15.6 -32.8 5.5</td>
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<tr>
<td>Orr.</td>
<td>244 180 96</td>
<td>41.0 23.5 24.5</td>
<td>96.2 53.7 49.3</td>
<td>15.6 -32.8 5.5</td>
</tr>
<tr>
<td>Pae.</td>
<td>155 110 69</td>
<td>23.0 68.2 19.9</td>
<td>9.2 37.6 41.9</td>
<td>39.6 62.2 42.0</td>
</tr>
<tr>
<td>Pas.</td>
<td>160 110 73</td>
<td>31.0 61.3 22.7</td>
<td>133.2 43.7 91.2</td>
<td>15.6 -32.8 5.5</td>
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<tr>
<td>Poe.</td>
<td>188 98 78</td>
<td>10.3 64.0 10.5</td>
<td>-3.2 17.5 30.0</td>
<td>15.6 -32.8 5.5</td>
</tr>
<tr>
<td>Roo.</td>
<td>180 120 74</td>
<td>80.3 13.2 95.2</td>
<td>-172.2 17.0 162.1</td>
<td>52.8 85.8 22.4</td>
</tr>
<tr>
<td>Mean</td>
<td>191 120 78</td>
<td>39.5 19.4 26.8</td>
<td>109.7 35.6 27.9</td>
<td>30.8 40.1 22.2</td>
</tr>
</tbody>
</table>

A is the mean manifest area in microvolt-seconds.

α in degrees. The mean values for α were determined by obtaining the mean of the ratios of the sines to the cosines of the angles.
24.1 degrees.* Changes in length and direction of the QRS vector in either plane were not statistically significant (table 2). Little change occurred in the QRS of the precordial leads.

The gradient (QRS-T) in the frontal plane shortened in all, averaging approximately half its control size (fig. 2). It deviated consistently in a counterclockwise direction (mean deviation 28.7 degrees). In the sagittal plane shortening also occurred, averaged 26.5 microvolt-seconds, or slightly less than half its average control value. It also deviated posteriorly (counterclockwise) on the average by 33.1 degrees.

As expected under such circumstances the mean T vector in the frontal plane shortened by 53.5 per cent, and also deviated in a counterclockwise direction by 61.7 degrees (fig. 3). In the sagittal plane the shortening was 75.5 per cent, and the deviation posteriorly (counterclockwise) was 18.7 degrees.

These modifications were all statistically significant ($p < 0.01$) or probably significant ($p < 0.05$) with the exception of the change in direction of the gradient (QRS-T) in the frontal plane (table 2).

These changes in vectors manifested themselves most clearly in the usual clinical leads as

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* This mean difference was obtained by calculating the means of the ratios of the sines and cosines of the angles before and after pyrogen and measuring the difference. This is the figure shown in table 1. To calculate standard deviations, however, the ordinary mean of individual differences in the angle was used (table 2). If the vector remained in the same quadrant after the pyrogen, the mean difference obtained by the two methods was identical (table 2).
modifications in the T wave. These consisted usually of a lowering of this deflection in leads II and III with or without a depression of the S-T junction. In the extremity leads a reciprocal change occurred in the T wave in leads aVL and aVF with an increase in positivity of the former and a decrease in positivity of the latter during the inscription of this deflection. In the precordial leads there was most often little change in the T wave, but in the exceptions (figs. 4 and 5) the T wave became deeper or inverted in leads further to the right than was the case before the pyrogen was given.

It was deduced from these observations in normal subjects that the spatial QRS vector was changed insignificantly in size following the use of pyrogen. The decrease in length of its frontal projection was in part the result of its posterior deviation. The shortening of the spatial vector representing the ventricular gradient was distinctly not on the basis of a change in position alone since a decrease in its size occurred in convincing degree in both the frontal and sagittal planes. The conclusion seemed justified, in accordance with current theory, that the differences in the duration of the excited state in different parts of the normal ventricular muscle were reduced by intravenous pyrogen as given. These conclusions seemed to be valid even when allowances were made for the probable inaccuracies which result from the use of Arrighi’s triangle for measurement of the sagittal components of the vectors measured.
B. Hypertensive Patients (tables 1 and 2). In the 15 patients there was a considerable fall in blood pressure, following the pyrogen, which averaged 58 mm. Hg systolic (191 mm. to 133 mm.), and 33 mm. Hg diastolic (120 mm. to 87 mm.).

In the frontal plane the length of the QRS vector varied greatly after pyrogen (fig. 1, solid circles and arrows). The range was between an increase of 27.0 microvolt-seconds and a decrease of 19.4 microvolt-seconds (table 1). The average change was an increase of 5.0 microvolt-seconds or 12.7 per cent. The angle alpha in control records ranged from +68.2 degrees to -26.4 degrees; only three patients displayed an angle alpha less than 0 degrees. After pyrogen the change in direction was quite variable but in 11 of the 15 it was clockwise (to the right); but in all the average change in this direction was only 3.0 degrees. However, in one instance it changed in the opposite direction by 53.4 degrees.

![Fig. 1. Mean manifest area of QRS (AQRS) in the frontal (Einthoven) and sagittal (Arrighi) planes before and after reduction of the blood pressure by intravenous pyrogen in normal and hypertensive subjects. In this and the following two figures (figs. 2 and 3) the heads of the individual vectors are designated by an open circle for the normal subjects and by a solid circle for the hypertensive patients. The mean size and direction of all the vectors of the normal subjects is represented by a broken arrow; of the hypertensive patients by a solid arrow. The mean of the angle alpha was obtained in each group by taking the mean of sines and of the cosines of each angle. The ratio of these means gave the tangent of the mean angle alpha. Insignificant changes in size and direction are observed to occur in both planes (see tables 1 and 2).](http://circ.ahajournals.org/)

![Fig. 2. Mean manifest area of QRS-T (A \(_G\)) in the frontal and sagittal planes before and after reduction of the blood pressure by intravenous pyrogen in normal and hypertensive subjects. In both groups and in both planes the mean vector is shorter after pyrogen. In the frontal plane both rotate slightly in a counterclockwise manner; in the sagittal plane the mean vector of the normal subject (dashed arrow) rotates counterclockwise (backward viewed from the left) but the mean vector of the hypertensive patients (solid arrow) changed its direction in the opposite way, that is, forward. Changes in the length and direction of the mean normal vector are probably significant (p < 0.05) with the exception of the change in direction in the frontal plane (table 2). Changes in the length and direction of the abnormal (hypertensive) mean vector are statistically insignificant.](http://circ.ahajournals.org/)
None of the changes in the length or direction of the QRS vector was statistically significant (table 2).

A moment’s reflection on these mean values of QRS length and direction indicates that they are not likely, assuming that the true frontal and sagittal values of the vectors are given by

![Diagram of QRS vectors before and after pyrogen]

Fig. 3. Mean manifest area of $T (\hat{A}_r)$ in the frontal and sagittal planes before and after reduction of the blood pressure in normal (open circles, dashed arrow) and hypertensive (solid circles, solid arrow) subjects. The mean vector of the hypertensive patients changed little in size in either plane after pyrogen. The normal mean vector shortened in both. Rotation of the two vectors after pyrogen was in opposite directions in the two planes—counterclockwise for the normal, clockwise for the hypertensive. Only the changes in size and direction of the mean normal vector are probably significant ($p < 0.05$). Failure of a single spatial vector to account for the frontal and sagittal projections can be seen best in this figure (dashed arrow, after pyrogen). Poorest fits were obtained in individual cases when the vector was of small size as in this instance of the mean normal vector (see text).

Einthoven’s and Arrighi’s triangles. The reason is as follows: An assumed decrease in length of the spatial QRS vector will cause an increase in the length of its frontal projection only if its change in direction in the sagittal projection is toward the vertical. In this instance the mean change in sagittal direction was the reverse, that is, toward the horizontal. A further analysis of the records of the six patients under consideration revealed that only two displayed changes in the frontal and sagittal QRS vectors which could fit a single change in the spatial vector. This was in contrast to what was observed in the normal group. The observation is significant in that conclusions with regard to the spatial vectors in hypertensive patients must be tentative, and absolute statements can be made with regard to these vectors only as studied by Einthoven’s and Arrighi’s triangles. A final solution of the problem revolves about the choice of a reference system which will yield the frontal and sagittal components of the mean manifest potential of any deflection accurately.

The gradient in the frontal plane showed an average shortening of 8.5 microvolt-seconds (23.9 per cent). There was considerable scatter in the distribution (fig. 2) although 11 of the 15 showed shortening. In the sagittal plane the change in direction in both planes after pyrogen was so variable as to make mean values in this small series relatively meaningless. However, the trend (fig. 2) was counterclockwise in the frontal plane and clockwise in the sagittal plane, the latter being the opposite of what occurred in the normal subjects. In terms of the direction of the spatial gradient the average change was from a direction downward, to the left, and backward to a direction horizontal, to the left, and forward. It is to be noted in figure 2 that the average vectors in the two planes either before or after pyrogen cannot have their origin from a single spatial vector as in the case of the QRS vector.

With regard to the length of the spatial gradient the trend was, as in the normal subjects, toward a reduction but to a lesser degree (fig. 2, table 2).

The $T$ vector in the frontal plane, as could be deduced from the change in the gradient, was quite variable but the mean change in length was small (+1.1 microvolt-seconds, +4.1 per cent). In the sagittal plane the mean change in length was also slight (−1.3 microvolt-seconds, −5.9 per cent). Little can be said about the change in direction in either plane because this varied widely, but the trend of the spatial T vector after pyrogen was from a direction down-
ward, to the right, and forward, to upward, to the right and forward (fig. 3). None of these changes in length or direction was statistically significant (table 2).

These changes in vectors manifested themselves most clearly in the usual clinical leads as electrocardiograms which were the least abnormal. In the so-called “typical” records of hypertension the changes during the hypotensive phase were not so obvious (figs. 4 and 5), although measurements of areas proved that they had occurred (table 1).

From the extremity potentials it was evident that clockwise rotation of the QRS axis in the frontal plane, when it occurred, caused an increase in the size of the R wave and a decrease in the size of the S wave in lead aVF, and the reverse events in lead aVL. The usual depression of the S-T segment and decrease in

![Fig. 4. Patient, D. R., essential hypertension. Before pyrogen. The standard leads (I, II, III), the augmented extremity potentials (aV_R, aV_L, aV_F), the sagittal leads of Arrighi (Ts, II_s, III_s), and the potential of the point on the back (V_b) used in recording the sagittal leads were made with the string sensitivity normal (1 mv. = 1 cm.). The precordial leads (V_1, V_2, V_3, V_4, V_5, V_6) were recorded with the string sensitivity at half normal (1 mv. = 0.5 cm). Time lines occur every 0.04 second. Ventricular rate is 73 beats per minute.](http://circ.ahajournals.org/)

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size or inversion of the T wave in lead I was produced by an increase in potential of the right arm and a simultaneous decrease in the potential of the left arm during the inscription of these deflections.

The precordial leads usually displayed no wave in precordial leads farther to the right than observed in the control records.

**With Tetraethylammonium Chloride**

The results of injecting tetraethylammonium chloride intravenously in six hypertensive sub-

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**Fig. 5. Patient D. R., essential hypertension. After pyrogen.** The ventricular rate is 91 beats per minute. The most obvious gross change is the deep inversion of the T wave which has occurred in leads V₃ and V₄, and the change to upright of the T wave in lead V₆. Symbols and time lines are as in figure 4.
tients the S-T segment in leads I and II was slightly depressed and in two it was slightly elevated during the hypotensive phase.

The impression gained was that the drug used had no distinct or consistent effect on the form of the electrocardiogram although the blood pressure was always reduced appreciably (mean change from 176 to 127 mm. Hg systolic and 100 to 74 mm. Hg diastolic).

**DISCUSSION**

The electrocardiographic changes observed during the phase of hypotension following triple typhoid vaccine in normal and hypertensive subjects are similar to those described by Freedberg, McManus and Altschule during fever induced in normal subjects. Although fever was not always prevented entirely by premedication with amidopyrine, the temperature never rose to levels which are usually attained with the dosage of vaccine employed. Furthermore, there was no correlation between the extent of the electrocardiographic changes and the extent of the fever, and the same type of changes occurred whether fever was entirely prevented or not. Often the electrocardiographic modifications persisted after the temperature subsided. For all these reasons it is believed that the changes reported were not due to fever, or at least not to fever alone.

The injection of the vaccine was followed invariably by a moderate but statistically significant rise in the heart rate (tables 1 and 2). In spite of the fact that an increase in the sinus rate has, as Ashman has shown, a bearing upon the form of the electrocardiogram, the changes observed in our series by far exceeded those usually attributed to higher rates. They also persisted, or were even greater, after the rate returned to control values.

An explanation for the electrophysiologic phenomena observed is difficult. The pyrogen causes such changes as a decrease in peripheral resistance, a fall in intraventricular and intramural ventricular pressure, a decrease in tension in the aortic wall with possible modification of suspension of the heart in the thorax, a change in coronary blood flow, and a variety of alterations in the physicochemical composition of the blood. Although a majority of these depend on a fall in blood pressure, it cannot be stated with certainty that the alterations observed in the electrocardiograms were caused exclusively by this variable. However, as long as the blood pressure remained low, the deviations persisted.

Since the electrocardiogram in the hypertensive patients was modified by a relatively sudden and brief fall in blood pressure in a direction which by present standards may be regarded as more "abnormal," the question arose whether a reduction of blood pressure is the factor responsible for the reverse modifications which have been reported to occur after sympathectomy. A good many records have been

![Fig. 6. Electrocardiograms (I, II, III, aV_R, aV_L, aV_F, V_1, V_2, V_3). before (A) and 6 months after (B) bilateral lumbosacral sympathectomy. Although the blood pressure is almost identical there is a complete reversion of the T waves to what is regarded as normal.](http://circ.ahajournals.org/asset/1702x688.png)
published showing a return of T waves to "normal" after sympathectomy,12–28 but none, except those quoted by Raab and Lepeschkin,49 have demonstrated an "improvement" in the electrocardiogram after sympathectomy without a fall in blood pressure. We have had the opportunity of observing* one such case which is summarized in figure 6. A record made on July 2, 1948 displayed distinct abnormalities of the T wave in leads I, II, aVL, V5, and V6 (not shown) at a time when the blood pressure was 233/150. A sympathectomy was done in two stages on July 28 and August 4. Another record made on Aug. 21, 1948 (not shown) when the blood pressure was 150/100 showed a partial regression of these T-wave abnormalities. A record made on Feb. 2, 1949 (fig. 6) displayed complete disappearance of the abnormal T waves but the blood pressure was 240/140, its approximate preoperative level. The ventricular rates in the first and last records were 60 and 72 respectively, and the frontal axis and area of QRS was similar in both.

The case is cited in support of the experimental data given. Together they seem to indicate that variables other than the fall in blood pressure can be responsible for those alterations in ventricular regression which result in more "normal" T waves in the electrocardiogram of certain hypertensive subjects when the hypotension is induced by sympathectomy,12–28 diet,29–30 or other means. Further, certain theoretic considerations permit the conclusion that an upright T wave in lead I, or lead V1, when there is considerable hypertrophy of the left ventricle, may actually represent a more abnormal electrophysiologic state than an inverted T wave in these leads.

The trend of the frontal QRS-T in our experiments was to shorten and rotate to the left (counterclockwise) in the frontal plane (fig. 2). These findings are somewhat at variance with the observations made on preoperative and postoperative electrocardiograms by Boyer and Hewitt56 in 106 hypertensive patients subjected to sympathectomy (Smithwick). In their series the mean manifest potential of QRS and of QRS-T shifted to the right (clockwise) but only in the former was the change statistically significant.

**Summary and Conclusions**

1. Regarding QRS, T, and QRS-T as vectors studied in two planes by means of Einthoven's and Arrighi's triangles, the specific modifications which occurred after intravenous triple typhoid vaccine were:

   (a) In five normal subjects the QRS-T shortened and rotated in a counterclockwise manner in the frontal plane, and shortened and rotated posteriorly (counterclockwise viewed from the left) in the sagittal plane. These modifications in the ventricular gradient were reflected best in the similar behavior of the T vector; the QRS vector was modified similarly but to an insignificant degree.

   (b) In 15 hypertensive patients the QRS-T vector also shortened in both planes but changes in direction were quite variable. On the average, the change in direction was counterclockwise in the frontal plane but clockwise (viewed from the left) in the sagittal plane. The mean changes in size and direction of all vectors in these 15 patients were statistically insignificant.

2. Intravenous pyrogen reduces the size of the differences in the duration of the excited state in the ventricular muscle both in normal subjects and in hypertensive patients but to a lesser degree in the latter. The fall in blood pressure caused by the pyrogen is probably but not absolutely the important variable concerned with this change.

3. The modifications in the ventricular gradient produced by intravenous triple typhoid vaccine resulted in electrocardiograms with a more "abnormal" configuration, particularly of the T wave.

4. The electrocardiographic response of six hypertensive subjects to temporary hypotension produced by tetraethylammonium chloride given intravenously was minor and variable, except for the consistent shortening of the QRS vector as measured in the frontal plane only.

5. Attention is called to the tentative va-

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* Initial observations were made by Dr. Ignacio Chavez of Mexico City, to whom the authors are deeply indebted for permission to use his data.
lidity of some of the data by virtue of the disclosed unreliability of the methods used to obtain the exact spatial vector.

6. Electrocardiograms of a hypertensive patient are presented in which the T waves returned to "normal" following a sympathectomy only after the blood pressure had returned to preoperative hypertensive levels.

7. The experimental data presented and the case cited do not support the concept of "strain" as applied to certain electrocardiographic abnormalities in hypertension which are sometimes reversed by agents or procedures which decrease the blood pressure.

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