The Spatial Ventricular Gradient: Intermittent Wolff-Parkinson-White Syndrome, Intermittent Left Bundle Branch Block and Ventricular Premature Contractions

By Mordecai A. Berkun, M.D., Russell H. Kesselman, M.D., Ephraim Donoso, M.D. and Arthur Grishman, M.D.

Criteria are given for testing the ventricular gradient concept. The spatial ventricular gradient is then determined in three types of intraventricular conduction disturbances using the cube system of orthogonal bipolar leads. Correction of the isoelectric baseline for the effect of $T_p$ on the QRS and $T$ areas is made. The spatial ventricular gradient remains essentially constant both in magnitude and direction in intermittent Wolff-Parkinson-White syndrome and intermittent left bundle branch block despite marked changes in body surface electrocardiograms. Ventricular premature contractions produce marked deviations in the spatial ventricular gradient. The spatial ventricular gradient merits further study as one of the fundamental quantities in electrocardiography.

In 1921, F. N. Wilson found a relationship between the forms of the QRS and $T$ complexes in electrocardiographic curves. He noted that changes in the QRS may be accompanied by corresponding changes in $T$ of opposite sign. He concluded that the form of $T$ depends on the order in which the various regions of the ventricles complete their electric activity. Changes in the duration of the electric response of particular regions of the ventricles, as seen with digitalis, tachycardia, exertion, and myocardial ischemia, were defined as primary $T$ changes. On the other hand, changes in $T$ accompanying changes in QRS, as in bundle branch block, were defined as secondary $T$ changes. In 1931, Wilson introduced the term ventricular gradient for a vector defined by the electrical axis and the manifest area of QRST.

The mean electric axis of QRS gives the mean direction in which the excitatory process spreads over the ventricles. Since recovery produces electric forces opposite in polarity to those produced by excitation, the mean electric axis of $T$ must give the inverse of the mean direction in which the recovery process spreads over the ventricles. The ventricular gradient, which is the vector sum of the mean QRS and $T$ vectors, therefore, represents the magnitude and direction of the electrical forces resulting from the lack of uniformity in the duration of the excited state in different portions of the ventricles. It points from the region in which the average duration of electrical systole is greatest toward the region in which it is least. The ventricular gradient concept neglects the effect of ventricular contraction upon the position and orientation of component cardiac muscle units with respect to each other and to the leads employed.

Wilson limited his measurements to the frontal plane, using the areas of QRS and $T$ in any two of the standard limb leads. S-T displacements were considered part of the recovery process except when due to injury currents.

The most important applications of the frontal plane ventricular gradient to clinical electrocardiography were made by Ashman, Byer, Gardberg and associates. They believed that an increase in stroke volume causes an increase in the magnitude of the frontal
planar ventricular gradient, while an increase in heart rate or the use of digitalis\textsuperscript{11} causes a decrease.

Ashman mentioned that the ventricular gradient might result from intramyocardial pressure differences,\textsuperscript{12} while Lepeschkin felt that intramyocardial temperature variations were more important.\textsuperscript{13}

Ashman never actually measured the spatial ventricular gradient although he devised a method of determining its direction in space.\textsuperscript{16} Burch and his coworkers determined the spatial ventricular gradient, using leads I, V \textsubscript{F} and V \textsubscript{B} in a group of normal women during pregnancy and after delivery.\textsuperscript{14} Jouvene and associates determined the directions of the three-plane projections of the spatial ventricular gradient, using their lead system.\textsuperscript{15} Ivancic and Mikulicic, using a different system of orthogonal bipolar leads, calculated the magnitude and direction of the spatial ventricular gradient in 100 normal subjects.\textsuperscript{16} Simonson, Schmitt and co-workers were the first to report on the use of an electronic integrating circuit to obtain the spatial ventricular gradient.\textsuperscript{17}

**Objectives of this Investigation**

Several authors have attempted to test the validity of the ventricular gradient concept in induced and spontaneous intermittent or transient intraventricular conduction disturbances, using frontal plane leads alone and the spatial ventricular gradient was not determined.

Wilson and his co-workers produced right bundle branch block in a dog and then caused varying fusion complexes by artificial stimulation of a single right ventricular focus. The frontal plane ventricular gradient showed a relatively small average variation.\textsuperscript{4} Wilson also found fair agreement between the normal and aberrant ventricular conduction when he measured the frontal plane ventricular gradient in one patient with intermittent left bundle branch block.\textsuperscript{5} Eckey and Schäfer subtracted the normal complexes in leads I, II and III from the abnormal complexes of intermittent Wolff-Parkinson-White syndrome in one case and obtained diphasic curves having approximately equal positive and negative areas.\textsuperscript{18} They did not, however, discuss their findings in terms of the ventricular gradient concept. Fowler and Westcott reported six examples of right bundle branch block induced by cardiac catheterization and determined the frontal plane ventricular gradient before, during, and after the block.\textsuperscript{19} Simonson and associates also studied one case with right bundle branch block and idioventricular rhythm in which the frontal plane ventricular gradient did not change appreciably.\textsuperscript{17} Segers and Boyadjian calculated the frontal plane ventricular gradient in intermittent right and left bundle branch block and concluded that determination of the ventricular gradient projection on another plane was desirable.\textsuperscript{20}

We have given consideration to the criteria for an ideal, and consequently crucial, test for the ventricular gradient concept in human beings. They are listed as follows:

1. The spatial ventricular gradient is measured before or after a spontaneous or induced intermittent or transient intraventricular conduction disturbance and during the abnormal conduction.

2. The measurements are made within a few seconds before and after the change in conduction to minimize the effect of alterations in other physiologic variables.

3. Three noncollinear component leads are obtained simultaneously.

4. The electrocardiographic tracings used are technically correct with minimum baseline wander.

5. A variety of types of intraventricular conduction disturbances is studied.

6. Correction is made for the effect of T\textsubscript{p} on the baseline for the measurement of QRS and T areas.

7. The cardiac rate remains essentially unchanged throughout the determinations.

8. Marked tachycardia sufficient to cause overlapping of P and the preceding T is absent because it would then be impossible to construct any isoelectric baseline.

9. The electrocardiographic records are obtained during the same phase of respiration to eliminate the effects of changes in cardiac position on the direction of the spatial ventricular gradient.

The studies to be described satisfy these criteria with the few exceptions to be noted.
The component leads used are the A, B, and C leads previously described. They make up an orthogonal lead system presenting right-left, posterior-anterior, and superior-inferior components. The electrodes are placed approximately equidistant from the electrical center of the heart (fig. 1). The sphere is an ideal geometric figure for the vectorial study of an electric field. This is true because the lines connecting points equal distances along the surface of a sphere with the center of the sphere subtend equal angles and, therefore, indicate comparable quantities of the electric field in each lead. The eight corners of a cube represent eight equally distant points on a sphere's surface. Each electrode is sufficiently distant from the heart in the cube arrangement for the exploration of its field. The interposed tissue between the heart and the four electrodes is predominantly lung tissue. With the cube arrangement there is close correlation of the horizontal plane projection of the spatial vectorcardiographic loop and the chest leads.

### TABLE 1.—Clinical Material

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Initials</th>
<th>Age</th>
<th>Sex</th>
<th>Clinical Heart Disease</th>
<th>Types of Intraventricular Conduction</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>P. C.</td>
<td>59</td>
<td>F</td>
<td>Arteriosclerotic</td>
<td>Normal, Wolff-Parkinson-White</td>
</tr>
<tr>
<td>2</td>
<td>J. C.</td>
<td>20</td>
<td>M</td>
<td>Congenital (coarctation of the aorta)</td>
<td>Normal, Wolff-Parkinson-White</td>
</tr>
<tr>
<td>3</td>
<td>A. K.</td>
<td>37</td>
<td>M</td>
<td>None</td>
<td>Normal, Wolff-Parkinson-White</td>
</tr>
<tr>
<td>4</td>
<td>A. E.</td>
<td>67</td>
<td>F</td>
<td>Arteriosclerotic</td>
<td>Normal, Wolff-Parkinson-White, left bundle branch block</td>
</tr>
<tr>
<td>5</td>
<td>F. R.</td>
<td>62</td>
<td>F</td>
<td>Arteriosclerotic</td>
<td>Normal, left bundle branch block</td>
</tr>
<tr>
<td>6</td>
<td>W. P.</td>
<td>47</td>
<td>M</td>
<td>Hypertensive</td>
<td>Normal, left bundle branch block</td>
</tr>
<tr>
<td>7</td>
<td>D. D.</td>
<td>72</td>
<td>F</td>
<td>Arteriosclerotic</td>
<td>Normal, left bundle branch block</td>
</tr>
<tr>
<td>8</td>
<td>M. L.</td>
<td>45</td>
<td>F</td>
<td>Arteriosclerotic</td>
<td>Normal, incomplete and complete left bundle branch block</td>
</tr>
<tr>
<td>9</td>
<td>P. H.</td>
<td>54</td>
<td>M</td>
<td>Hypertensive</td>
<td>Normal, left bundle branch block</td>
</tr>
<tr>
<td>10</td>
<td>S. K.</td>
<td>53</td>
<td>M</td>
<td>Arteriosclerotic</td>
<td>Left bundle branch block, ventricular premature contractions from two foci</td>
</tr>
<tr>
<td>11</td>
<td>G. C.</td>
<td>59</td>
<td>M</td>
<td>Arteriosclerotic</td>
<td>Normal, ventricular premature contraction</td>
</tr>
<tr>
<td>12</td>
<td>C. R.</td>
<td>37</td>
<td>M</td>
<td>None</td>
<td>Normal, ventricular premature contraction</td>
</tr>
<tr>
<td>13</td>
<td>I. B.</td>
<td>84</td>
<td>M</td>
<td>Arteriosclerotic</td>
<td>Right bundle branch block, ventricular premature contraction, atrial fibrillation</td>
</tr>
<tr>
<td>14</td>
<td>M. V.</td>
<td>45</td>
<td>M</td>
<td>Rheumatic</td>
<td>Normal, ventricular premature contraction</td>
</tr>
<tr>
<td>15</td>
<td>I. G.</td>
<td>64</td>
<td>M</td>
<td>Arteriosclerotic</td>
<td>Left bundle branch block, ventricular premature contraction</td>
</tr>
<tr>
<td>16</td>
<td>G. P.</td>
<td>54</td>
<td>M</td>
<td>Aortic aneurysm</td>
<td>Normal, ventricular premature contraction</td>
</tr>
<tr>
<td>17</td>
<td>F. B.</td>
<td>41</td>
<td>F</td>
<td>Congenital</td>
<td>Normal, ventricular premature contraction</td>
</tr>
</tbody>
</table>
The duration of atrial electrical activity (P + Tp) and its relationship to the P-P interval and the atrial rate.

The magnitude of the spatial ventricular gradient and its projections on any plane are usually expressed in microvolt-seconds (μ vs.), or in Ashman units (A.U.), each of which equals 4μ vs.22

MATERIALS AND METHODS

The records of 17 patients from the files of the Vectorcardiographic Laboratory of The Mount Sinai Hospital were suitable for this study (table 1).

All of the electrocardiograms were taken with the patient supine and with respiration temporarily held. A three-channel, direct-writing Technicon Cardiograph was used for all the records, and the determinations were made from simultaneous leads. Each patient had a standard 12-lead electrocardiogram and then simultaneous A, B and C leads were obtained. In most of the records the paper speed was 50 mm. per second. The standardization was adjusted so that a 1 mv. input gave a 15 mm. upright deflection. Occasionally, it was necessary to apply appropriate corrections for slight standardization differences.

The A, B and C component leads were then projected by means of an opaque projector against a wall resulting in a magnification of 8.1 diameters. The projected curves were traced along their upper margins and the time of onset of QRS, end of QRS and end of T were marked in the simultaneous leads using the most clearly outlined and usually the longest intervals for timing purposes. The U-P segment was taken as the true isoelectric baseline, and, where this was clearly identifiable, a straight line was drawn from the U-P segment preceding the cycle used to the U-P segment immediately following it. Where no clear U-P segment was found, or the baseline wandered appreciably, a line drawn from the start of P to the end of T (or U, if present), was used as the isoelectric baseline. In a few instances baseline wander was in more than one direction during a single beat so that any baseline constructed was subject to considerable error.

One of the difficulties encountered in any direct measurement of QRS and T areas is the magnitude

FIG. 2. The duration of atrial electrical activity (P + Tp) and its relationship to the P-P interval and the atrial rate.

FIG. 3. Method of measuring areas.

FIG. 4. Definitions of GF and angle α.

FIG. 5. Definitions of GH and angle β.
of T_P (representing atrial recovery). T_P is directed opposite to P and its enclosed area is approximately equal to that of P.\textsuperscript{23} When P is small or isoelectric, T_P causes no discernible deviation of the baseline and no correction is necessary. When P is large and positive, T_P depresses the baseline for QRS and T considerably (particularly evident on the magnified tracings), and a baseline correction must be introduced or the measured areas of QRS and T will both be too small. Where P is large and negative, T_P is large and positive, and an opposite correction must be made or the measured areas will be too large.

Ashman\textsuperscript{7} used an implied partial correction of the baseline for T_P in ventricular gradient determinations. Lepeschkin\textsuperscript{13} suggested the use of a straight line from the start of QRS to the end of U as the baseline and set the notch between T and U as the end of T.

We approached this problem in a more direct fashion. The duration of atrial electric systole (P + T_P) was determined from 74 electrocardiograms with complete or incomplete heart block and this was plotted against the P-P interval. It was found that the duration of atrial electric systole decreases as the atrial rate increases and that there is a linear relationship when the P + T_P duration is plotted against the P-P interval\textsuperscript{34} (fig. 2).

The duration of P + T_P for the atrial rates of each case was derived from the graph. This was measured off along the isoelectric baseline from the start of P. Then a line was drawn from the onset of QRS to this point and this corrected baseline was used to measure the areas of QRS and T (fig. 3).

The tracings were attached to a flat board and the areas of QRS and T were measured with a planimeter. Areas above the corrected baseline were considered positive and areas below were considered negative. Each tracing was measured twice and the results averaged and then converted into Ashman units. The magnitude of the spatial ventricular gradient and the magnitudes and directions of its frontal and horizontal plane projections were calculated.

A series of symbols is defined and they will be used throughout the remainder of this presentation as well as in our future publications. Scalar quantities appear in ordinary type. Vector quantities appear in bold face type. (Modern publications in physics and mathematics universally use ordinary type for scalars and bold face type for vectors).

\( \mathbf{G} \) is the spatial ventricular gradient, \( \mathbf{G} \) is the magnitude of \( \mathbf{G} \), \( \mathbf{G}_F \) is the frontal plane projection of \( \mathbf{G} \), \( \mathbf{G}_H \) is the magnitude of \( \mathbf{G}_F \), \( \mathbf{G}_H \) is the horizontal plane projection of \( \mathbf{G} \), \( \mathbf{G}_H \) is the magnitude of \( \mathbf{G}_H \), \( \mathbf{G}_A \) is the magnitude of the projection of \( \mathbf{G} \) on the \( A \) axis, \( \mathbf{G}_B \) is the magnitude of the projection of \( \mathbf{G} \) on the \( B \) axis, and \( \mathbf{G}_C \) is the magnitude of the projection of \( \mathbf{G} \) on the \( C \) axis.

Angle \( \alpha \) is the smaller angle between \( \mathbf{G}_F \) and the positive \( A \) axis and angle \( \beta \) is the smaller angle between \( \mathbf{G}_H \) and the positive \( A \) axis.

Angle \( \alpha \) is given a positive sign if \( \mathbf{G}_F \) is inferior, and a negative sign if \( \mathbf{G}_F \) is superior to the \( A \) axis. Similarly, angle \( \beta \) is called positive if \( \mathbf{G}_H \) is anterior and negative if \( \mathbf{G}_H \) is posterior to the \( A \) axis.

From the Pythagorean Theorem

\[
G = \sqrt{G_A^2 + G_H^2 + G_C^2}
\]

(1)

In the frontal plane (fig. 4)

\[
G_F = \sqrt{G_A^2 + G_C^2}
\]

(2)

and

\[
\sin \alpha = \frac{G_C}{G_F}.
\]

(3)

In the horizontal plane (fig. 5)

\[
G_H = \sqrt{G_A^2 + G_B^2}
\]

(4)

and

\[
\sin \beta = \frac{G_B}{G_H}.
\]

(5)

| Table 3.—Calculated Variations in \( \mathbf{G}_F \) and \( \mathbf{G}_H \) and the Changes in Angles \( \alpha \) and \( \beta \) for the Various Types of Conduction |
|-----------------|-------------|-------------|-------------|-------------|-------------|
| Conduction      | Case No. 1 | \( \mathbf{G} \) (%) | \( \mathbf{G}_F \) (%) | \( \mathbf{G}_H \) (%) | \( \alpha \) (Degrees) | \( \beta \) (Degrees) |
| Disturbance     |            |             |             |             |             |                 |
| Wolff-          | 1          | 8           | 5           | 25          | 9           | 4                |
| Parkinson-      | 2          | 1           | 1           | 8           | 8           | 1                |
| White Syndrome | 3          | 23          | 11          | 25          | 1           | 39               |
|                 | 4          | 9           | 23          | 18          | 10          | 13               |
| Average         |            | 10          | 10          | 19          | 7           | 14               |
| Left bundle     | 4          | 1           | 10          | 19          | 1           | 6                |
| branch block    | 5          | 0           | 0           | 3           | 8           | 2                |
|                 | 6          | 2           | 19          | 6           | 32          | 38               |
|                 | 7          | 48          | 57          | 15          | 18          | 8                |
|                 | 8          | 24          | 58          | 1           | 15          | 17               |
|                 | 9          | 7           | 17          | 3           | 4           | 13               |
| Average         |            | 14          | 25          | 14          | 13          | 14               |
| Ventricular     | 10         | 39          | 83          | 59          | 56          | 35               |
| premature       | 10         | 16          | 32          | 18          | 16          | 2                |
| contraction     | 11         | 65          | 78          | 68          | 131         | 110              |
|                 | 12         | 58          | 49          | 63          | 19          | 35               |
|                 | 13         | 40          | 126         | 20          | 34          | 95               |
|                 | 14         | 72          | 33          | 151         | 64          | 20               |
|                 | 15         | 48          | 58          | 95          | 53          | 49               |
|                 | 16         | 9           | 7           | 11          | 14          | 2                |
|                 | 17         | 99          | 142         | 63          | 76          | 74               |
| Average         |            | 55          | 74          | 59          | 51          | 47               |

Case 8 was included in these calculations and in this table only for the change in conduction from incomplete to complete left bundle branch block.
RESULTS

The results are recorded in table 2. This contains the findings in four patients with intermittent Wolff-Parkinson-White syndrome, six patients with intermittent left bundle branch block, and eight patients with premature ventricular contractions.

For each case the table shows the atrial rate in cycles per minute, the derived P + T duration in seconds (from fig. 1), the types of intraventricular conduction, the magnitude of QRS and T areas, $G_A$, $G_B$, $G_C$, $G$, $G_F$, $G_H$, $\alpha$ and $\beta$. All magnitudes are in Ashman Units and all angles are in degrees.

Table 3 contains all the calculated variations in $G$, $G_F$, and $G_H$,* and the changes in angles $\alpha$ and $\beta$ for the various types of conduction.†

* Let $G$ be $G_1$ during control conduction, let $G$ be $G_2$ during altered conduction, and let $v_G$ be the percent variation in $G$. Then

$$v_G = \frac{2\sqrt{(G_2 - G_1)^2}}{G_1 + G_2} \times 100\%$$

The symbols $v_F$ and $v_H$ are similarly defined for the percent variation in $G_F$ and $G_H$.

† The absolute values of the angle changes in degrees are used and these are indicated by the symbols $|\Delta \alpha|$ and $|\Delta \beta|$.

Figure 6 contains representative A, B, and C component leads during control and abnormal ventricular conduction for cases 2, 4 and 5.

DISCUSSION

Four patients with intermittent Wolff-Parkinson-White syndrome were studied (cases 1, 2, 3 and 4). The average variation in $G$ was 10 per cent (range 1 to 23 per cent); the average variation in $G_F$ was 10 per cent (range 1 to 23 per cent); and the average variation in $G_H$ was 19 per cent (range 8 per cent to 25 per cent). Angle $\alpha$ changed an average of 7 degrees (range 1 degree to 10 degrees), and angle $\beta$ changed an average of 14 degrees (range 1 to 39 degrees). This is the first series of patients with intermittent Wolff-Parkinson-White syndrome in which $G$, $G_F$, and $G_H$ have been determined. Within the limits of experimental error, $G$, $G_F$, and $G_H$ did not change in magnitude or direction from that accompanying normal intraventricular conduction.

There were six patients with intermittent left bundle branch block (cases 4, 5, 6, 7, 8, and 9). The average variation in $G$ was 14 per cent (range 0 to 48 per cent); the average variation in $G_F$ was 25 per cent (range 0 to 58
Fig. 6. Illustrative cases, A, Case 2, intermittent Wolff-Parkinson-White syndrome; B, case 4, normal conduction, Wolff-Parkinson-White syndrome and left bundle branch block; C, case 5, intermittent left bundle branch block.

RESULTS

The results are recorded in table 2. This contains the findings in four patients with intermittent Wolff-Parkinson-White syndrome, six patients with intermittent left bundle branch block, and eight patients with premature ventricular contractions.

For each case the table shows the atrial rate in cycles per minute, the derived P + Tₚ duration in seconds (from fig. 1), the types of intraventricular conduction, the magnitude of QRS and T areas, Gₓ, Gₓ, Gₓ, G, Gₓ, Gₓ, α and β. All magnitudes are in Ashman Units and all angles are in degrees.

Table 3 contains all the calculated variations in G, Gₓ, and Gₓ, * and the changes in angles α and β for the various types of conduction.†

* Let G be Gₓ during control conduction, let G be Gₓ during altered conduction, and let vₓ be the percent variation in G. Then

\[ vₓ = \frac{2 \sqrt{(Gₓ - Gₓ)^2}}{Gₓ + Gₓ} \times 100\% \]

The symbols vₓ and vₓ are similarly defined for the percent variation in Gₓ and Gₓ.

† The absolute values of the angle changes in degrees are used and these are indicated by the symbols |DA| and |DB|.

Figure 6 contains representative A, B, and C component leads during control and abnormal ventricular conduction for cases 2, 4 and 5.

DISCUSSION

Four patients with intermittent Wolff-Parkinson-White syndrome were studied (cases 1, 2, 3 and 4). The average variation in G was 10 per cent (range 1 to 23 per cent); the average variation in Gₓ was 10 per cent (range 1 to 23 per cent); and the average variation in Gₓ was 19 per cent (range 8 per cent to 25 per cent). Angle α changed an average of 7 degrees (range 1 degree to 10 degrees), and angle β changed an average of 14 degrees (range 1 to 39 degrees). This is the first series of patients with intermittent Wolff-Parkinson-White syndrome in which Gₓ, Gₓ, and Gₓ have been determined. Within the limits of experimental error, G, Gₓ, and Gₓ did not change in magnitude or direction from that accompanying normal intraventricular conduction.

There were six patients with intermittent left bundle branch block (cases 4, 5, 6, 7, 8, and 9). The average variation in G was 14 per cent (range 0 to 48 per cent); the average variation in Gₓ was 25 per cent (range 0 to 58
per cent); and the average variation in \( G_\alpha \) was 14 per cent (1 to 50 per cent). There was an average change in angle \( \alpha \) of 13 degrees (range 1 to 32 degrees), and in angle \( \beta \) of 14 (2 to 38 degrees). The changes in magnitude and direction of \( G, G_\alpha \) and \( G_\beta \) are within the error of the determination and this indicates that \( G \) does not change in left bundle branch block. Unfortunately, our series did not contain any instances of intermittent right bundle branch block and we can give no confirmation of the ventricular gradient concept in that conduction disturbance, though we assume that it will be proved valid here, too.

Case 4 (see fig. 6), which showed normal conduction, Wolff-Parkinson-White conduction, and left bundle branch block is of particular interest not only because of the rarity of this combination but that the records were obtained at a single session and they can, therefore, be compared with each other. As can be seen from table 2 and figure 6, \( G, G_\alpha \) and \( G_\beta \) were almost the same whereas the forms of the electrocardiographic complexes in the component leads were quite different from each other.

Eight cases with ventricular premature contractions were in the studied group (cases 10, 11, 12, 13, 14, 15, 16 and 17). The average variation in \( G \) was 55 per cent (range 9 to 72 per cent); the average variation in \( G_\alpha \) was 74 per cent (range 7 to 142 per cent); and the average variation in \( G_\beta \) was 59 per cent (range 11 to 151 per cent). The change in angle \( \alpha \) averaged 51 degrees (range 16 to 131 degrees), and in angle \( \beta \) averaged 47 degrees (range 2 to 110 degrees). These changes in \( G, G_\alpha \) and \( G_\beta \) in ventricular premature contractions are about three to four times larger than the changes found in the same quantities in the cases with Wolff-Parkinson-White conduction and in the cases with left bundle branch block and are definitely outside the limits of observational error. \( G \), therefore, changes both in magnitude and direction during ventricular premature contractions. Despite the divergent results obtained with ventricular premature contractions some positive correlation of lower magnitude appears to be present, but a much larger series of similarly studied patients would be required in order to reliably determine the degree of this correlation.

In the fifteen types of ventricular fusion complexes produced by Wilson and his associates in their dog experiment,\(^4\) there was a 19 per cent average variation in \( G_\alpha \) as determined from the Einthoven limb leads (range 4 to 46 per cent).\(^*\) This experiment is not quite analogous to spontaneously occurring ventricular premature contractions in human hearts, some of them diseased. Burger and associates studied vectorcardiograms simultaneously obtained, using two different lead systems.\(^5\) For normal conduction there was good correlation between the two vectorcardiograms, but for the ventricular premature contractions the correlation was poor. Their explanation was that the excitation spreads differently over the heart in the two types of conduction, and, because of the uneven distribution of insulating fatty tissue on the surface of or around the myocardium, the current field in the thorax is affected differently in normal and premature beats. Because of the abnormal pathway of the excitation wave in extrasystoles, the sequence of stimulation of different portions of the myocardium may vary greatly. This, in turn, may cause considerable shifts of the dipole center during the spread of the excitation wave accompanying an extrasystole which may affect the two lead systems used by Burger and his associates to a varying degree without having any additional significance. Another possibility is a basic difference between the excitatory and recovery processes in ventricular premature contractions as compared with normal conduction, Wolff-Parkinson-White conduction, and left bundle branch block. In ventricular premature contractions excitation takes place at differing times following the preceding beat and thus may occur during the relative refractory period or supranormal conduction phase of the ventricular muscle. Our findings strongly suggest that ventricular premature contractions are poorly suited to the investigation or confirmation of the ventricular gradient concept.

To compare \( G \) and \( G_\alpha \) obtained by others,

\(^*\) This calculation was made by us using Wilson's published data.\(^2\)
using different lead systems, with our results obtained with the cube arrangement, it is necessary to relatively standardize the systems with respect to the actual electric field strength of the cardiac vector. To compare the cube with a system based on the Einthoven limb leads one may assume that the heart is immersed in a homogeneous volume conductor of infinite extent and that the lead electrodes are equidistant from the heart. Making the above assumptions, which are only approximations for any given subject, it has been calculated that the deflections in the standard limb leads of Einthoven have a 1.5 times greater amplitude than those in the cube system for the same magnitude of heart vector.\textsuperscript{26} Consequently, to compare our values of $G$ with those of Simonson and co-workers,\textsuperscript{17} or our values of $G_r$ with those of Ashman and his colleagues,\textsuperscript{6-10} or others using the Einthoven limb leads, our gradient magnitudes must be multiplied by the factor 1.5. However, the lead system of Ivancic and Mikulicic\textsuperscript{16} utilizes bipolar orthogonal leads and no standardization factor is required for comparison.

The consistency of the values of $G$ both as to magnitude and direction in intermittent Wolff-Parkinson-White syndrome and intermittent left bundle branch block is indicative of the validity of the A, B, and C orthogonal lead system in electrocardiographic and vectorcardiographic practice.

Sources of Error

There are variations in the vertical thickness (along the voltage axis), of the electrocardiographic curve, depending on the shape of a deflection. This is due to incomplete burning of the heat-sensitive paper coating by the stylus at high speeds of deflection. The finite width of the stylus in the horizontal direction (along the time axis), affects the area under any curve if the rate of ascent is different from the rate of descent. By consistently making all tracings along the upper edge of the projected curves this error was kept to a minimum. In case 7, the upper edge of the original electrocardiograms was frequently poorly visible and the error in tracing the projected curves was probably large.

Baseline wander introduces an error of variable extent, particularly when the wander is in more than one direction. Case 13 had an irregular baseline due to atrial fibrillation.

The $T_r$ correction is only an approximation for any individual record but the error introduced is probably relatively small.

Other minor errors resulted from 60 cycle per second alternating current electric interference, limitation of planimeter accuracy, and the human error in tracing the projected curves and using the planimeter.

Cases 7 and 8 were included in this study even though our criteria were not completely satisfied. In each, the intermittent left bundle branch block was produced by exercise. In case 7, the heart rate remained unchanged at 75 per minute, but in case 8, the heart rate increased from 75 per minute during normal conduction to 100 per minute during the two degrees of left bundle branch block. $G$, $G_r$, and $G_i$ decreased in both cases. Diminution of the ventricular gradient occurs with any increase in heart rate.\textsuperscript{7} Case 7 is included in the calculations of average changes and all subsequent discussion of results, but in case 8 normal conduction is excluded and only the results in the two degrees of left bundle branch block are referred to in Table 4 and subsequently. Case 8, as far as we know, is the first demonstration that $G$ decreases on increase in heart rate, all previous conclusions being based on the observed decrease in $G_r$.

Conclusions

Our observations show that the spatial ventricular gradient can be readily determined, using the bipolar orthogonal component leads A, B and C of the cube system of vectorcardiography. The measurement of the spatial ventricular gradient is a significant addition to the understanding and also to the clinical analysis of the electric events of the cardiac cycle. The constancy of the magnitude and direction of the spatial ventricular gradient during normal intraventricular conduction, Wolff-Parkinson-White syndrome, and left bundle branch block emphasizes the value and significance of the gradient concept. When Wolff-Parkinson-White conduction or left
bundle branch block (and frequently left or right ventricular hypertrophy), are found in an electrocardiographic record, other changes due to myocardial damage or infarction are obscured. The spatial ventricular gradient may then be useful in diagnosis under these conditions by comparison with either a previous ventricular gradient determination made on an earlier record of the same patient or a set of normal standards.

It is apparent that the technics employed in this study are too time-consuming to be used in a very large series of cases. However, this project is a necessary preliminary to the automatic electronic recording of the spatial ventricular gradient.

Problems that we intend to investigate, using electronic apparatus and technics, are the determination of the range of the magnitude and the direction of the spatial ventricular gradient in normal individuals as well as similar measurements in ventricular hypertrophy, myocardial damage, myocardial infarction, and electrolyte disturbances, and the evaluation of the extent of digitalization.

**SUMMARY**

(1) The spatial ventricular gradient and its projections on the frontal and horizontal planes are measured both in magnitude and direction, using an orthogonal cube lead system consisting of right-left, posterior-anterior and superior-inferior leads (A, B and C leads).

(2) A method of correcting for the effect of Tp on the areas of QRS and T is described.

(3) The spatial ventricular gradient is determined in four patients with intermittent Wolff-Parkinson-White syndrome during normal and abnormal intraventricular conduction. The spatial ventricular gradient did not change significantly in either magnitude or direction.

(4) The spatial ventricular gradient is determined in six cases with intermittent left bundle branch block and there are no significant changes in its magnitude or direction in the two types of conduction.

(5) The spatial ventricular gradient is measured in eight cases with ventricular premature contractions and there are significant changes both in magnitude and direction when compared with the control conduction. The results suggest that some positive correlation of lesser degree would be found if a larger series of cases is studied.

(6) A single case showing normal conduction, Wolff-Parkinson-White conduction and left bundle branch block is reported. There is no significant change in the magnitude or direction of the spatial ventricular gradient.

(7) In a single case, the spatial ventricular gradient is found to decrease in magnitude with an increase in heart rate.

(8) Criteria for testing Wilson’s concept of the ventricular gradient are given and this concept is found to be valid.

(9) The results indicate that the orthogonal bipolar cube A, B and C lead system present reliable projections of the cardiac vector.

**SUMMARIO IN INTERLINGUA**

(1) Le spatial gradient ventricular e su projectiones super le planos frontal e horizontal es mesurate in magnitude e direction per medio de un orthogene systema cubic de derivationes, consistente de derivationes dextero-sinistre, postero-anterior, e supero-inferior (A, B, e C).

(2) Es describite un metodo que rectifica le effecto de Tp super le areas de QRS e T.

(3) Le spatial gradient ventricular es determinate in quatro patientes con intermittente syndrome Wolff-Parkinson-White durante normal e anormal conduction intraventricular. Le spatial gradient ventricular non eseva significativamente alterate in o magnitude o direction.

(4) Le spatial gradient ventricular es determinate in sex casos de intermittente bloco de branca sinistre. Il non ha significative alterationes in direction o magnitude del gradiente in le duo typos de conduction.

(5) Le spatial gradient ventricular es mesurate in octo casos de prematur contractiones ventricular. Il occurre significative alterationes in e magnitude e direction in comparation con le conduction de controlo. Le resultatos rende probabile que un correlation positive de grados minus marcate esserea constatate si un plus extense serie de casos esseva studiate.
REFERENCES


The Spatial Ventricular Gradient: Intermittent Wolff-Parkinson-White Syndrome, Intermittent Left Bundle Branch Block and Ventricular Premature Contractions

MORDECAI A. BERKUN, RUSSELL H. KESSELMAN, EPHRAIM DONOSO and ARTHUR GRISHMAN

_Circulation_. 1956;13;562-572
doi: 10.1161/01.CIR.13.4.562
_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1956 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/13/4/562

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in _Circulation_ can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to _Circulation_ is online at:
http://circ.ahajournals.org/subscriptions/