Relative Efficiency of Depolarization and Repolarization of the Myocardium Determined from the Spatial Vectorcardiogram

By Charles E. Kossmann, M.D., Stanley A. Biiller, M.D. and Nathan Marchand, M.S.

Utilizing differential vectorcardiography and certain calculations, a method is presented by which the relative quantities of electrical energy expended in depolarization and repolarization of the myocardium in man can be determined.

The numerous investigations now being conducted in vectorcardiography will undoubtedly clarify many nebulous aspects of clinical electrocardiography and will improve present methods of teaching. Yet, with the exception of superiority in demonstrating subtle differences in the phase of electrical events occurring in the heart, the method in its present state of development does not appear to present important clinical advantages over older methods of making electrocardiographic leads. On the other hand, it occurred to us sometime ago that determination from the spatial vectorcardiogram of the manifest electrical energy involved in depolarization and repolarization of atrial and ventricular muscle, but particularly the latter, might prove to be useful clinically, especially if a correlation with the mechanical energetics of the myocardium can eventually be established.

With this concept in mind, the initial steps in instrumentation were undertaken. The first of these, the recording of the ventricular QRS and T loops, or any part of each separately, was accomplished with the differential vectorcardiograph designed and constructed in this laboratory. An instrument for integrating areas of the spatial loops is under construction. However, this aspect of the problem promises to take a good deal of time. Further, there are other problems to be solved before a complete investigative or clinical use of an energy concept of the basic electrical processes in the heart will be possible. These are concerned principally with an exact determination of the orthogonal components of the spatial vector free from the distortion caused by inhomogeneity, anisotropism, and variable contours of the conducting medium, and by eccentricity of the source of potential. In the foreseeable future these variables will be quantitated adequately in man but there are others, such as the change in locus of the source of potential during systole, which may not be. Nevertheless it seemed desirable to report on the concept in further detail with the full realization that the data in support of its validity will require modification as techniques are refined.

Basic Considerations

The fundamental consideration is that the vectorcardiogram supplies the necessary components for determining the manifest electrical energy resulting from myocardial activity, namely, voltage and time. The formula which defines rate of electrical work is:

\[ W = E \cdot I \cdot t \]  

where \( W \) means work in wattseconds or joules, \( E \) is the electromotive force in volts, \( I \) the current in amperes, and \( t \) the time, usually in seconds. In applying the formula to the vectorcardiogram, \( I \) is converted by Ohm's law to \( \frac{E}{R} \) whence the equation becomes:

\[ W = \frac{E^2}{R} \cdot t \]
where $R$ is the resistance. It can be shown that equation (2) applies to a complicated resistive mesh as is encountered in the body. Since the resistance is assumed to be constant or almost constant, it cancels out in the ratio of the electrical energy involved in depolarization and repolarization as follows:

$$\eta = \frac{W_1}{W_2} = \frac{E_1^2 \cdot t_1}{E_2^2 \cdot t_2} \quad (3)$$

As applied to the vectorcardiogram, $E$ is voltage, $t$ is time, and $W$ is work. Since the resistance and the generator potential in the corporeal circuit are unknown, actual expression of the work done in joules is not possible, but this does not change the validity of the ratio. It is this value, which we have designated as the electrical merit, or $D-R$ (depolarization-repolarization) energy ratio* of the heart.

Instead of a ratio, the manifest work of depolarization and repolarization can be expressed as a sum; but since the value, $E$, varies from moment to moment, this summation must be by integration. For the ventricles this relationship may be expressed as follows:

$$\int_Q E^2 dt + \int_J E^2 dt = \int_T E^2 dt \quad (4)$$

The limit $Q$ represents the beginning of ventricular depolarization; the limit $J$ the end of depolarization and the beginning of repolarization; and the limit $T$ the end of repolarization. Again, since the equivalent circuit of the body is unknown, the equation yields a value which is proportional to the total work expended in one complete electrical cycle of the heart. It is a scalar which will always have a positive value. This measurement, which may be called the manifest cyclical energy consumption (CEC), will have a value proportional to the total transfer of electrical energy within the ventricles with each heart beat. It is possible that the proportionality constant which depends on certain electrical characteristics of the body may show interindividual variations, and possibly variations within individuals as well. Work in progress makes it likely that this constant can be resolved.

In obtaining such a value it is necessary to admit a small error in so far as the $S-T$ junction or $J$ does not define the exact dividing line between the end of depolarization and the beginning of repolarization of ventricular muscle.

**Practical Considerations**

At any particular moment the mean manifest electromotive force in space may be defined in terms of the orthogonal coordinates:

$$E^2 = (E_x^2) + (E_y^2) + (E_z^2) \quad (5)$$

and

$$E = \sqrt{(E_x^2) + (E_y^2) + (E_z^2)} \quad (6)$$

The problem of which reference system to use in obtaining the true values of $x$, $y$, and $z$ in the body has not been solved. The approach we have made is by way of the isosceles tetrahedron* in which the values $x$, $y$, and $z$ are multiplied by the factors 1, 1.7 and 1.7 respectively.

Knowing the factors to be used, an instrumental determination of the areas of the spatial vectorcardiographic loops will be a reality in the foreseeable future, and, as noted, an instrument to do this is under construction in this laboratory.

Calculation of the spatial areas from any simultaneously recorded 2 of the 3 planar vectorcardiograms is rather laborious. It has been our custom to record the frontal ($xyz$) with either the sagittal ($yx$) or transverse ($zx$) vectorcardiogram simultaneously. The time intervals, $t$, are 0.0025 sec. Any instantaneous spatial electromotive force ($E^{xyz}$, or simply $E$) may be calculated in a variety of ways depending upon the geometric and trigonometric relationships which may be visualized in the accompanying line drawing (fig. 1).

$$E^{xyz} = (E_x^2) + (E_y^2) + (E_z^2) \quad (7)$$

and

$$E = \sqrt{(E_x^2) + (E_y^2) + (E_z^2)} \quad (8)$$
FIG. 1. Diagram to illustrate the nomenclature of the rectilinear axes \((x, y, z)\) and planes \((xy, xz, yz)\) of the body as viewed from the front, and the angles \(\alpha, \beta, \gamma\) relative to these created by the instantaneous electromotive forces in the frontal plane \((E^x)\), the sagittal plane \((E^y)\), and in space \((E^z)\) or simply \(E\). The force in the transverse plane \((E^z)\) and the angle made by it on the \(z\) axis \((\alpha^z)\) is not shown. Calculation of \(E^z\) from the instantaneous electrical axes in any two of the planes is done by triangulation.

\[
E^2 = h_N
\]

\[
\theta = 0.0025 \text{ SEC.}
\]

Measurement of the various angles and lines is made on a greatly enlarged (approximately 12X) projection of the original vectorcardiogram. Once the magnitude of \(E\) is obtained for each of the 40 radii of the QRS loop which occur in 0.10 sec., and of the approximately 70 to 80 radii of the slower T loop, it is squared and then becomes a value \((h_n)\) in a series of isosceles triangles (fig. 2) with relations as follows:

\[
S, = \frac{1}{2} h_n a
\]  

\[
S_n = \frac{h_n \times 0.0025}{2}
\]  

\[
S_1 = S = (h_n)(0.00125)
\]  

\[
\eta = \frac{W_1}{W_T} = \frac{S_{QRS}}{S_T}
\]

The equation relates the work done in depolarization to the work done in repolarization and is therefore an index of the relative effi-
Table 1.—Experimental Data

<table>
<thead>
<tr>
<th>Patient</th>
<th>( \frac{\Sigma \Delta T}{\Delta T} )</th>
<th>( \frac{\Sigma \Delta T}{\Delta T} )</th>
<th>( \frac{\Sigma \Delta T}{\Delta T} )</th>
<th>( \frac{\Sigma \Delta T}{\Delta T} )</th>
</tr>
</thead>
<tbody>
<tr>
<td>E. O'C.</td>
<td>87.60</td>
<td>0.50</td>
<td>221.60</td>
<td>0.50</td>
</tr>
<tr>
<td>A. R.</td>
<td>97.24</td>
<td>0.50</td>
<td>263.04</td>
<td>0.50</td>
</tr>
<tr>
<td>J. D.</td>
<td>77.28</td>
<td>0.50</td>
<td>381.90</td>
<td>0.50</td>
</tr>
<tr>
<td>S. L.</td>
<td>40.40</td>
<td>0.50</td>
<td>54.10</td>
<td>0.50</td>
</tr>
</tbody>
</table>

* microvolt seconds.
† (millivolt)* milliseconds.
‡ \( \Sigma \Delta T \) or \( \Sigma \Delta T \) × 0.0032.

Fig. 3. Electrocardiograms of patient E. O'C., white male, age 36 years. Shown are the bipolar extremity leads (I, II, III), the augmented unipolar extremity leads (aVR, aVL, aVF), and the unipolar precordial leads (V1, V5, V6). The last were recorded with the tension of the string adjusted so that 1 mv = 0.5 cm. Time lines occur every 0.04 sec.

Note the high voltage of QRS in the frontal plane, the width of the QRS interval (0.16 sec.), and the lateness of the intrinsicoid (RS) deflection in the precordial leads measured from the beginning of the QRS interval in those leads. In lead V1, this QR interval is approximately 0.10 sec. and suggests block of the two electrical processes. A possible source of confusion in absolute values, but not in ratios, arises from the fact that a denominator of 2 appears in equation (12) but not in equation (2). This means that the area of a loop, as calculated, gives only half of the electrical area or electrical work actually done, a mathematical relationship of areas geometrically calculated in vector space. For consistency all values for areas given in Table 1 represent time corrected areas as measured geometrically, and accordingly are indicated by half units (i.e., \( \mu V s / 2 \)).

Calculation from a Spatial Vectorcardiogram

To make the necessary calculations manually with any degree of accuracy, it is necessary to select vectorcardiograms displaying a considerable area of both the QRS and T loops. A male patient, age 36, with an atypical right bundle-branch block (fig. 3) but no evidence of other organic disease was studied. By means of the differential vectorcardiograph, photographs of the desirably large QRS loop and T loop were obtained separately (fig. 4), and the direction of rotation determined by photographing only part of each. A check on what was photographed was possible from the simultaneously recorded duration of exposure of the right bundle branch. However, in leads V5 and V6, and in lead aVF, it is approximately 0.08 sec. Since there was no roentgenographic evidence of hypertrophy, the inference is that a block of some type existed also on the left side of the heart.
loops ($L$), and the areas of the loops obtained after squaring the instantaneous vectors ($W$) during depolarization ($QRS$) and repolarization ($T$) of the ventricular muscle are shown for this patient and for others in Table I. The last four columns of the table also show the ratio of the area of the two processes ($L_{QRS}/L_T$ or $D/R$ area ratio), the ratio of the energy areas ($W_{QRS}/W_T$ or $D/R$ energy ratio), and the sums of $L_{QRS}$ and $L_T$, and $W_{QRS}$ and $W_T$ (cyclical energy consumption or CEC).

The series is too small and the methods thus far too gross to draw any but the most tentative of clinical conclusions from the data. Further, the vectorcardiograms were selected for large or easily measured loops. Three of the patients (Table 2, E. O'C., A. R., and J. D.) displayed QRS intervals in excess of 0.12 sec. Only one subject had an unequivocally normal heart as determined clinically (S. L.). Nevertheless the calculation of immediate interest, the $D/R$ energy ratio ($W_{QRS}/W_T$) did show some interesting random clinical correlations,

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**Fig. 4.** Vectorcardiogram of patient E. O'C., white male, age 36 years, whose electrocardiograms are shown in Fig. 3. The frontal ($xy$, the left of each pair), and the sagittal ($yz$, the right of each pair) views were recorded simultaneously with a twin-beam oscilloscope using the isocèle tetrahedron as the reference system, and the central terminal connected through resistances of 5000 ohms to each of the three extremities. Calibrations are shown, and the time intervals are 0.0025 sec.

The frame on the left shows the loops of QRS and T ($VCQ_{QRS}$, $VCQ_{T}$). The middle frame shows the loops of QRS ($VCQ_{QRS}$, $VCQ_{T}$) separately, and the right frame the loops of the T wave ($VCQ_{T}$) separately, the latter at increased gain of the instrument.

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**Fig. 5.** Exposure intervals made simultaneously with the vectorcardiograms shown in Fig. 4 by means of a three-channel direct-writing recorder (Technicon). When the upper trace is depressed, the oscillographic record is being photographed. The middle and lower traces in this instance were a unipolar lead from the left leg (lead $VF$) which in the reference frame used is 0.623 of the $y$ component, and bipolar lead I (Lead $I$) which is regarded as the $x$ component.

The 3 sets of records correspond to the exposures of QRS, QRS, and T (the latter at an increased gain) shown in the 3 sets of Fig. 4.
DEPOLARIZATION AND REPOLARIZATION OF MYOCARDIUM

Table 2—Clinical Data

<table>
<thead>
<tr>
<th>Patient, Age, Sex</th>
<th>Diagnosis</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>E. O'C., 35, M.</td>
<td>1. Cardiac (a) Unknown (b) Undiagnosed (c) NSR, paroxysmal atrial fibrillation, right bundle branch block, terminal complete heart block with asystole (d) IIC–IVE 2. Chronic otitis media, left 3. Chronic pulmonary tbc, arrested, right with re-expanded pneumothorax</td>
<td>Repeated episodes of unconsciousness for 1½ years. Neurologic studies negative. Complete heart block noted terminally only.</td>
</tr>
<tr>
<td></td>
<td>1. Cardiac (a) Congenital (b) Enlarged heart, isolated dextrocardia, undiagnosed right to left shunt (c) NSR, incomplete A-V block, bundle-branch block unclassified (d) IIC 2. Acute diffuse glomerulonephritis 3. Heroin intoxication</td>
<td>Cyanosis and clubbed fingers since birth. Polycythemia. (Angiocardiograms in another hospital said to reveal single atrium, small right ventricle).</td>
</tr>
<tr>
<td>A. R., 20, M.</td>
<td>1. Undiagnosed manifestation (possible heart disease) 2. Cardiac neurosis 3. Osteoarthritis of thoracolumbar spine</td>
<td>Abnormal electrocardiogram, constant for 7 years, with deep inversion of T wave in leads I, II, aV_{1}, and aV_{F} and precordial leads V_{5} to V_{6}. Heart normal size.</td>
</tr>
<tr>
<td>I. G., 57, F.</td>
<td>Gout</td>
<td>Left bundle-branch block and incomplete A-V block which disappeared while receiving ACTH. Normal sized heart. No clinical evidence of heart disease.</td>
</tr>
<tr>
<td>J. D., 62, M.</td>
<td>Normal</td>
<td></td>
</tr>
<tr>
<td>S. L., 26, M.</td>
<td>Normal</td>
<td>—</td>
</tr>
</tbody>
</table>

the validity of which must be established by further studies.

Theoretically the \( D/R \) energy ratio should be close to one. In the normal subject (S. L.) it was 0.59. In patient I. G. it was 1.07. This 57-year-old white widow had a strikingly abnormal electrocardiogram characterized by a high voltage of QRS and deeply inverted T waves in all of the usually recorded twelve leads except in leads III, aV_{R}, and V_{1}. Although she was regarded as having arteriosclerotic heart disease she was observed for seven years without any change either in the electrocardiogram or in the normal size of the heart. She has had no diminution of cardiac reserve but continues to have rather uncharacteristic pains in the chest to which the rubric "anginal syndrome" has been assigned as a result of conventional interpretation of the electrocardiograms. In contrast, the ratio in patient E. O'C. was 23.98. Although his records were quite abnormal (figs. 3 and 4), no clinical evidence of cardiac disease was apparent. However, three months after the special studies were done he developed complete heart block with recurrent ventricular asystole and died within three days. Patient A. R. and patient J. D., both with intraventricular block, intermittent in the latter, revealed values of 3.61 and 8.20. The first had cyanotic congenital heart disease. The second had no clinical evidence of heart disease other than the block (Table 2).

The significance of different values of the \( D/R \) energy ratio in terms of the cellular func-
tions involved must wait upon correlations being attempted in this laboratory between the intracellular myocardial potential and the mechanical behavior of the cell as modified by various chemical agents. In theory, a high $D/R$ energy ratio would seem to indicate an inefficient generating system and suggests that the excess electrical energy expended in depolarization must be restored externally by chemical means. The facts to support the theory, as applied to the myocardial cell, remain to be collected.

**SUMMARY**

By means of differential vectorcardiography and certain calculations it is possible to determine the relative amounts of electrical energy expended in depolarization and repolarization of the myocardium in man. The fraction relating these, the $D/R$ energy ratio, was found to have for the ventricular muscle a value of 0.59 in one normal subject, and varied from 1.07 to 2.98 in four patients. Although the ratio has suggestive clinical value per se, it is anticipated that correlations with other myocardial functions at the cellular level may make it possible to estimate these from a clinical determination of the ratio.

**REFERENCES**


**Circulatory Factors as Cause of “Dead Point” and “Second Wind”**

Vigorous exercise can lead to a state in which the muscles continue to respond only through exertion of greatest will power. This so-called “dead point” is characterized by distressing sensations in the head and chest, while the intensity of muscular effort diminishes. If, however, work is continued, this crisis passes off and the symptoms disappear quite suddenly; the muscles seem to regain their vigor, the heart rate slows, and breathing, in particular, becomes easier; hence the condition has been called “second wind” among athletes.

Explanations for the appearance and disappearance of the “dead point” have not been wholly satisfactory. Recent work from Helsinki, Finland, suggests that the sensation of collapse may be due to “the adaptations of the circulatory system to new conditions of heat loss when the body temperature during work stabilizes at its final level”.

In experiments carried out on a bicycle ergometer it was found that ventilation volume and oxygen uptake are both maximum at the “dead point” but that the ratio of increase (i.e. the ventilation equivalent for O$_2$) rose. Pulse rate, cutaneous and muscular blood flows in the arm also reached their maxima at the dead point. However, with the development of “second wind” systolic arterial pressure fell. This is attributed to a reduction of peripheral resistance due to opening of new skin vessels, which, in turn, is caused by heat regulating mechanisms.

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