Application of a Theoretic T-Wave Model to Experimentally Induced T-Wave Abnormalities

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A THEORETIC MODEL of the T wave is presented in another paper in this issue. Action potentials of appropriate duration were assigned to each area of the ventricle represented by an activation front. The onset of action potentials was made to correspond to the normal sequence of depolarization. Action potentials were divided into time units, and the difference in the height of the action potential at the beginning and end of each time unit was taken as the potential difference during that time unit of repolarization. The direction of repolarization vectors during each time unit was determined by relating the time phase of action potential upstrokes to that of downstrokes. With this model it was possible to account for the polarity and form of T waves recorded from animals. The present paper deals with further applications of this model.

Concept of the Base-Line and Local Gradient Vectors and Its Relation to the T Wave

In the previous paper the concept of the component gradient vector \( \mathbf{G}_c \) was introduced and was defined as those vectors produced by variations in the shape or duration of coexisting action potentials:

\[
\mathbf{G}_c = \mathbf{T} k \sum e_n
\]

In this formula, \( \mathbf{T} \) represents the line which closes the open ends of a boundary between two areas with action potentials of different configurations, \( k \) represents the degree to which the duration of the plateau phases differ in two adjacent areas, and \( e_n \) represents the potential differences during any 1 unit of time of the downstroke that are due to variations of action potential configuration.

Normally the duration of the ventricular action potential plateau, measured indirectly by the functional refractory period (FRP) is not equal in all layers of the myocardium. Van Dam and Durrer found that the FRPs of the innermost layer of the canine heart were approximately 15 msec longer than those in the middle and subepicardial layers and that there was a slight progressive prolongation of the FRPs from the middle third of the myocardium to the epicardial surface. It should be recognized that in individual layers of the ventricles there is a range of action potential durations that may be considered to be within normal limits. For the purposes of the model, a set of values that is within the normal range was arbitrarily chosen to represent the base-line state. The vectors produced by this disparity in the shape or duration of action potentials will be referred to as base-line gradient vectors \( \mathbf{G}_B \). The vectors produced by any change in this arbitrarily defined base-line state will be referred to as local gradient vectors \( \mathbf{G}_L \). Local gradient vectors may be the result of normal variations in heart rate or autonomic tone and do not necessarily imply an abnormal condition. On the other hand, local gradient vectors may result from anoxemia, electrolyte abnormalities, drugs, or other factors that produce an abnormal disparity.
in the configuration of action potentials either in local areas or diffusely throughout the ventricles. The component gradient vectors \( \vec{G}_c \) are equal to the sum of the base-line gradient vectors \( \vec{G}_n \) and the local gradient vectors \( \vec{G}_l \). The sum of all the component gradient vectors is in turn equal to the ventricular gradient \( \vec{G} \).

\[
\vec{G} = \vec{G}_c = \vec{G}_n + \vec{G}_l.
\]

Since \( \vec{G}_n \) and \( \vec{G}_l \) are component gradient vectors, they may also be expressed by the formula for \( \vec{G}_c \). Local gradient vectors fall along the same axis as the depolarization vectors in the area being considered. To determine the direction of \( \vec{G}_l \) along this axis, it is necessary to know the sequence of depolarization in the area being considered and the relationship of the depolarization sequence to the sequence of recovery. That is, it is necessary to know if the time phase of coexisting action potentials is the same during a given moment of repolarization as it was during depolarization.

Figure 1a shows a diagram of a heart in which the dotted lines represent boundaries between areas with action potentials of different configurations. The normal sequence of depolarization begins on the endocardial surface and proceeds to the epicardial surface. Depolarization vectors, arising from the shaded area in figure 1a, are directed toward the epicardium and are approximately parallel to a horizontal axis and perpendicular to a vertical axis. Diagrams of the shaded area shown (fig. 1a) are shown in figure 1b to d. The relationship of action potential onset and completion and the direction of repolarization vectors when action potentials of different durations are assigned to area C are shown. The base-line state is represented in figure 1b. The action potentials are of shortest duration in area B and are completed first in this area. The action potentials in area C are completed next, and area A, whose action potentials are of the longest duration, is the last to reach the excitable state. The temporal relationship of the action potential onset and completion in these three areas is shown. If two adjacent areas complete their action potentials in the same sequence as the sequence of depolarization, the sign of the potential difference across their boundary during repolarization is opposite to the sign across their boundary during depolarization. The component gradient vector is, therefore, opposite to the direction of the depolarization vector. If the first area to be depolarized has a plateau duration longer than the duration of the plateau in the next area to be depolarized, the sequence of plateau completion in these two areas will be opposite to the sequence of depolarization. The first area to be depolarized will be relatively negative with respect to its adjacent area throughout the repolarization process. The sign of the potential difference across the boundary of these two areas will, therefore, be the same as it was during depolarization, and the component gradient vector will be directed in the same direction as the depolarization vector. The directions of the component gradient vectors at the boundaries A-B and B-C are shown. A prolongation of the plateau phase of the action potential in area C, to the degree illustrated in figure 1c, does not alter the sequence of action potential plateau completion. The component gradient vectors at A-B and B-C are in the same direction as they were in the base-line state, but the magnitude of the vector at B-C has changed. The change in the magnitude of the component gradient vector from the base line to this new state is a measure of the local gradient vector. The effect of shortening the action potential plateau in area C is shown in figure 1d. The action potential plateau from area C is completed before that in area B, and area A is still the last to complete its action potential plateau. The temporal relationship of the action potentials from these three areas and the component gradient vectors at the boundaries A-B and B-C have the directions indicated in the illustration. Progressive prolongation of the plateau phase of the action potential in area C would cause a progressive increase in the magnitude of the local
A diagram of a heart in which the ventricles are divided by broken lines into inner, middle, and outer thirds (la). The shaded area is shown again in parts Ib to d to demonstrate the relation of action potential onset and completion to alteration of the duration of the plateau in area C. The direction of repolarization vectors at the boundaries A-B and B-C are also shown. Diagrams in Ib show a base-line state. The action potential is of the shortest duration in area B, is longer in area C, and longest in area A. The sequence of the upstrokes, plateau completion, and downstroke completion is shown. The repolarization vectors at the boundary A-B are directed toward the epicardium because A depolarizes before B and completes its plateau after B. The repolarization vectors at the boundary B-C are directed toward the endocardium because B depolarizes and completes its plateau before C. In 1c the action potential has been prolonged in area C. The sequence of action potential onset and completion is shown and is the same as it was in the base-line state shown in 1b, and the direction of repolarization vectors at the boundaries A-B and B-C are also the same as those in 1b. However, the magnitude of the repolarization vectors at B-C is increased compared to that in the base-line state because the potential differences at this boundary have been increased by prolonging the action potential in C. In 1d, the duration of the action potential in area C has been made shorter than that in area B. The action potentials in areas A and B are the same as those in the base-line state shown in 1b. The repolarization vectors at the boundary B-C are now directed toward the epicardium because B depolarizes before C and completes its action potential after C.

As mentioned previously, the gradient \( \vec{G} \) measured from the body surface electrocar-diogram is the sum of the base-line gradient vectors and the local gradient vectors; that is, the gradient is the sum of the vectors produced by the base-line disparity in the duration of co-existing action potentials, and the vectors produced by any deviation from this state. If the heart is in a base-line state, no local gradient vectors will exist. \( \vec{G}_L \) will be equal to 0, and \( \vec{G} \) will be equal to \( \vec{G}_B \). If \( \vec{G} \)
is considered to be the sum of $\mathbf{G}_B$ plus $\mathbf{G}_L$, $\mathbf{G}_B$ plus $\mathbf{G}_L$ are also equal to AQRS:

$$\mathbf{G}_B + \mathbf{G}_L = \text{AQRS} + \mathbf{AT}$$

and:

$$\mathbf{AT} = \mathbf{G}_B + \mathbf{G}_L - \text{AQRS}$$

The introduction of a local gradient will, therefore, produce changes in the configuration of the T waves. In figure 2 the heavy arrows represent the mean QRS, T, and gradient vectors in a heart in the base-line state. It can be seen that the addition of the local gradient vectors, $\mathbf{G}_{l1}$ and $\mathbf{G}_{l2}$, moves the mean T vector along a line that is parallel to the local gradient vectors. If these relationships are correct, it should be possible to alter experimentally the duration of the action potential plateau in a localized area of the heart and produce predictable T-wave changes. A series of experiments were designed to test the theoretic T-wave model.

**Design of Experimental Study**

Experiments were performed on nine mongrel dogs ranging in weight from 13.5 to 16 kg. Pentobarbital, 30 mg/kg, was used for anesthesia, and succinylcholine (Anectine), 1.5 to 2 mg/kg, was used to decrease the effects of muscle tremors on the ECG. The chest was opened with a sternal-splitting incision while respirations were maintained with a Harvard pump respirator or an Enesco respirator, that could be stopped at a fixed point in the respiratory cycle. The pericardium was opened, and a bipolar stimulating electrode was placed on the right atrium. The sinus node was crushed so that a constant rate could be maintained by electrical stimulation. A rate at which there was no overlapping of the T and P waves was used.

Qualitative changes in the T wave were observed in four dogs. Glass tubing was bent in the middle to form a circle with a diameter of 2.5 cm that was at right angles to the rest of the tubing. This flat circular surface could then be placed on the heart. Water of varying temperatures was circulated through this system. The glass tubing was supported on the surface of the ventricles, and the pericardium was loosely reapproximated. The chest cavity was packed with saline-soaked polyurethane foam that was warmed to body temperature. This packing was used to restore a portion of the volume conductor properties of the chest. The chest was closed, and a scalar ECG and vectorcardiogram were recorded with the triaxial dog lead system. Water at 45, 25, and 5°C was run through the glass tubing, and after the ECG form had stabilized, electrocardiograms and vectorcardiograms were recorded. The scalar ECGs were taken at paper speeds of 200 and 300 mm/sec. The chest was then carefully opened, and the position of the glass tubing was checked. The procedure was repeated by cooling and warming areas on the anterior surface of the heart, the lateral wall of the right ventricle, the lateral wall of the left ventricle, and the apex. The qualitative changes in T waves were analyzed in terms of the theoretic model.

In five dogs the functional refractory period (FRP) was measured in an area that was warmed and cooled. A rubber bladder with seven stainless steel unipolar electrodes was designed. It was made so that it could be sutured on the epicardium and had rubber tubing connections for circulating water through it. The bladder measured 2.5 cm² and the electrodes were arranged in a circle with one central electrode. The electrodes were insulated except at the tips, and they could be used for either recording or stimulating. The leads from these electrodes were connected to a selector switch. A bipolar reference electrode was placed on the ventricle at a distance from the testing electrodes to detect propagated ventricular responses. The atrium was stimulated at a rate of 144 per minute. This rate was chosen because it was usually possible to maintain it throughout an experiment without escape of the intrinsic rhythm, and at this rate there was no overlapping of the T and P waves. The response to the atrial stimulus at the bipolar reference

![Figure 2](attachment:image.png)

**Figure 2**

Mean QRS, T, and G vectors are shown as heavy arrows. The addition of the local gradient vectors, $\mathbf{G}_{l1}$ and $\mathbf{G}_{l2}$ to $\mathbf{G}_B$ moves the mean T vector along an axis parallel to the axis of the local gradient vector. The distance that the mean T vector moves is equal to the magnitude of the local gradient vectors.
electrode was displayed on one channel of an oscilloscope. The response at the unipolar testing electrode was displayed on the second channel of the oscilloscope and was horizontally adjusted so that the intrinsic deflection was aligned on the vertical axis of the oscilloscope with the intrinsic deflection recorded from the reference electrode. This was done so that the conduction time between the testing electrode and the reference electrode was not included in the FRP. The sweep speed was calibrated at 1 cm/50 msec. Stimuli from the atrial stimulus generator were passed through a counter so that every sixth stimulus triggered the sweep of the oscilloscope. A second stimulator was used to deliver cathodal stimuli of 2-msec duration and 1.5 to 2 times stimulus threshold intensity. These stimuli were passed through the counter and delivered to the testing electrodes at adjusted intervals after every sixth basic driving stimulus. The time interval between the response to the basic driving stimulus and the earliest testing stimulus that produced a propagated ventricular response was taken as the FRP. In the animals in which the FRP was determined, the bladder and electrode system were sutured on a relatively avascular area of the free wall of the right ventricle. The chest cavity was packed with saline-soaked polyurethane foam, and the chest was closed. Scalar ECGs and vectorcardiograms were recorded, and the FRPs were measured while water at 40, 30, 20, and 10 C was run through the bladder.

The T area and the QRS-T area were measured with a planimeter on records taken at a paper speed of 300 mm/sec, and the relationship between the change in T area and gradient and the change in the FRPs was analyzed in terms of the model.

Results
Qualitative Analysis of Changes in T Waves
As previously stated, local gradient vectors are directed along the same axis as the depolarization vectors in the area being considered. Figure 2 shows that the introduction of a local gradient vector causes displacement of the mean T vector along an axis parallel to that of the local gradient vector and perpendicular to the line closing activation vector fronts in the area where the local gradient vector was introduced. The distance between the termini of mean T vectors before and after the introduction of a local gradient vector is equal to the magnitude of the local gradient vector.

Since the T loops recorded in these experi-ments were narrow, maximal T vectors were used as approximations of mean T vectors in the qualitative analysis. Figure 3 shows a diagram of a dog’s heart in which the shading indicates the apical area that was warmed and cooled. Tracings of the T loops that were recorded in the frontal plane under control conditions and when water at 45, 25, and 5 C was run through the glass tubing are shown. If depolarization is assumed to proceed through the ventricles from endocardium to

\[
\begin{align*}
5^\circ & \\
25^\circ & \\
45^\circ & \\
\text{CONTROL} & \\
\end{align*}
\]

Figure 3

The shading indicates the area of the heart where local gradients were introduced by warming and cooling the epicardial surface. Tracings of T loops recorded in the frontal plane under each condition of temperature are shown with their isoelectric points superimposed. The maximal T vectors of these loops tend to fall along an axis that is perpendicular to an imaginary line drawn across the apex of the heart closing normal activation fronts in this area.
epicardium in concentric wavefronts, an imaginary horizontal line drawn across the apex closes these cup-shaped depolarization fronts. Another line drawn through the ends of the maximal T vectors is approximately perpendicular to the imaginary line closing the open ends of the depolarization fronts. In these experiments the area warmed and cooled was kept constant, and the maximal T vector moved along one axis in the manner predicted by the model. It was assumed that warming and cooling the myocardium in the manner employed produced changes in the duration of the action potentials only in the outer third of the ventricle and only in the area covered by the glass tubing. Since warming causes the ventricular action potential to shorten, water at 45°C was assumed to decrease the duration of action potentials in the outer third of the ventricle. The outer third of the ventricle was then assumed to reach the excitatory state before the middle third. This recovery order was opposite to the normal sequence of depolarization so that the repolarization vectors arising at the boundary of the middle and outer third of the ventricle in this warmed area were directed toward the epicardium, that is, slightly leftward and inferiorly, and would be expected to move the maximal T vector in this direction. Cooling which prolongs the duration of the action potential would not change the direction of repolarization vectors in this cooled area in comparison to their direction in the base-line state. As in the base-line state the middle third of this section of the ventricle would complete its action potentials before the inner and outer thirds. If the plateau phase of the action potential is prolonged in the outer third of the ventricle and there is no prolongation of the plateau phase in the middle third of the ventricle, the magnitude of the potential difference at this boundary during any one moment of repolarization is increased. Repolarization vectors that arise at the boundary of the middle and outer thirds of the ventricle would be directed toward the endocardium as they were in the base-line state. That is, they would be directed rightward and superiorly, but their magnitude would be increased, and the maximal T vector would move in this direction.

Comparable changes were observed in all four dogs when apical areas were warmed and cooled. In these same dogs the T-wave changes, produced by alterations in temperature in localized areas of the anterior surface of the ventricle and the lateral surfaces of the right and left ventricle, were also explicable by the model. Figure 4 shows the scalar ECG of a dog under control conditions and when water at 45, 25, and 5°C was run through the glass tubing. Under each condition of temperature the maximal T vector moved along an axis that was approximately perpendicular to an imaginary line closing normal activation fronts in the area warmed and cooled. Warming caused the maximal T vectors to be directed more toward the epicardium and cooling caused the maximal T vectors to be directed more toward the endocardium. As an example, temperature alterations of the lateral wall of the right ventricle were largely manifest in the X lead. Warming resulted in inversion of the T waves in this lead, that is, a change in the direction of the T vector toward the right or epicardial surface of the right ventricle. With cooling, the amplitude of T waves increased in the X lead, that is, a leftward change of T-vector direction toward the endocardium of the right ventricle.

**Quantitative Analysis of the Relationship of T-Wave Changes to Changes in FRP**

Measurements of the FRP were made at the seven testing electrode sites. Figure 5 shows a graph of the values obtained in one animal when water at 40, 30, 20, and 10°C was run through the test bladder. Cooling caused the FRP to prolong. The greater the degree of cooling the greater was the disparity in the duration of the FRP at the seven test sites. The values of the FRP at the test sites varied from one another by as much as 15 msec at the warmest temperature, and 50 msec at the coldest temperature. The values of the FRP at each test site were frequently checked, and repeated measurements at any one site...
Scalar ECG of a dog. Records shown were taken under control conditions and when water at 45, 25, and 5°C was run through glass tubing that was consecutively placed on the anterior surface of the ventricle, the apex, the lateral surface of the right ventricle, and lateral surface of the left ventricle. Warming caused the T vectors to be directed more toward the epicardium of the warmed area, and cooling caused the T vectors to be directed more toward the endocardium of the cooled area. The * indicates that the driving rate which was at an interval of 0.42 sec is temporarily replaced by the intrinsic rate of this animal with an R-R interval of 0.41 sec.

Table 1

<table>
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<tr>
<th>Water (°C)</th>
<th>FRP (msec) Dog 1</th>
<th>FRP (msec) Dog 2</th>
<th>FRP (msec) Dog 3</th>
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In each experiment an average of the FRPs at the seven test sites was taken under each condition of temperature and was used as in indirect measure of the degree to which the plateau phase of the action potential was altered. In table 1 the average FRPs at 40, 30, 20, and 10°C in the five experimental animals are shown to indicate the magnitude of the changes produced.
Temperature is plotted on the abscissa and the duration of the FRP at seven test sites on the ordinate. At the warmest temperature the FRPs at the seven separate test sites were within 10 msec of one another, while at the coldest temperature the FRPs at these seven sites extended over a range of 50 msec.

Figure 6a and b show the orientation of the mean T vector on the horizontal and frontal planes when water at 40, 30, 20, and 10 C was run through the test bladder. The prediction was that the mean T vectors should tend to fall along a straight line that is approximately perpendicular to an imaginary line that closes normal activation fronts in the area of the right ventricle that was warmed and cooled. The mean T vectors shown in figure 6a and b correspond to the predicted results and are comparable to the changes observed in the maximal T vectors in the initial qualitative analysis. The sequence of depolarization remained relatively constant during the course of an experiment and the base-line gradient vector as defined is a constant value. The local gradient, therefore, could be measured by measuring the change in $\overrightarrow{AQRST}$ that occurred under each test condition. The changes in QRS-T area in each lead ($\Delta G_X, \Delta G_Y, \Delta G_Z$) were considered projections of the local gradient in that lead, and the spatial local gradient ($\overrightarrow{SG_L}$) was calculated using the formula:

$$\overrightarrow{SG_L} = \sqrt{\Delta G_X^2 + \Delta G_Y^2 + \Delta G_Z^2}$$

A composite graph showing the relationship between the $\overrightarrow{SG_L}$ and the FRP in five experimental animals is shown in figure 7. The significance of this relationship will be discussed below.

Correlation of the Experimental Findings and the Theoretic Model of the T Wave

The experiments described above were designed to test the accuracy of the T-wave model. As previously discussed:
A composite graph from five dogs in which the changes in the FRP are plotted on the abscissa and changes in the T area are plotted on the ordinate. The first point plotted on each line represents the change in temperature from 40 to 30, the second point, the change from 30 to 20, and the last point, the change from 20 to 10 C.

\[ \Delta T = \mathbf{G}_B + \mathbf{G}_L - \mathbf{QRS} \]

and

\[ \mathbf{G}_L = \mathbf{G} k \Sigma e_a \]

\( \mathbf{G}_B \) was defined as the vectors produced by the variations in the shape or duration of co-existing action potentials in ventricles in a base-line state. For any given heart \( \mathbf{G}_B \) may be considered a constant value. If the sequence of depolarization remains constant, any change that occurs in \( \Delta T \) must be secondary to the introduction of a local gradient vector. That is, a change in \( \Delta T \) without a change in the sequence of depolarization is secondary to a change in the shape or duration of ventricular action potentials.

In the dogs studied, the QRS form was constant. It was assumed that the shape and duration of the ventricular action potentials remained constant in all areas except those warmed and cooled. Changes in \( \Delta T \) could, therefore, be considered to be secondary to the introduction of local gradient vectors produced by temperature alteration. Since the sequence of depolarization remained constant, the line along which \( \mathbf{G}_L \) fell also remained constant. The magnitude of \( \mathbf{G}_L \) is dependent on the degree to which the durations of the pla-
teaus in two areas vary, and the degree to which the downstrokes of the action potentials in two areas vary. Intracellular action potentials that have been recorded from cooled ventricular tissue have shown a marked prolongation of the plateau phase of the action potential. Compared to the marked changes in the duration of the plateau phase, the changes in the downstroke are less pronounced. The only other factor that must be considered is the resting potential. A change in the resting potential could produce potential differences without changing the duration or shape of the action potential. If the resting potential changed, potential differences would be present during the plateau phases as well as during the downstrokes and would be reflected by displacement of the S-T segment. With careful placement of the test bladder on a relatively avascular area on the lateral wall of the right ventricle the S-T segment remained isoelectric. If the simplifying assumptions are made that under the conditions of this experiment there was no change in the resting potential and that warming and cooling changed the duration of the plateau phase without changing the downstroke of the action potential, the values of \( e_a \) could also be considered to remain constant. The only factor in the formula for \( \mathbf{G}_L \) that was varied was the \( k \) value, representing the duration of the plateau, and this was indirectly measured by the FRP. If the proposed hypotheses are correct, the degree to which the FRP is changed should under the conditions of these experiments determine the magnitude of \( \mathbf{G}_L \), and there should be a linear relationship between the changes in area of the T wave, which reflect \( \mathbf{G}_L \), and the changes in FRP. Figure 7, which shows a composite graph of the five experimental animals, demonstrates this linear relationship. In all five dogs each change in the duration of the FRP resulted in a proportional change in the T area.

The model presented has been useful in relating action potential form to the T wave of the body surface ECG, and with it, normal T waves could be derived from normal QRS.
complexes. The study reported in this paper indicates that the assumptions utilized in the model are sufficiently valid for the model to be useful. The concept of the local gradient vector was introduced to permit application of the model to abnormal T waves. With this concept it was possible to predict the form of the T wave when changes in the duration of the action potential were induced in localized areas of the ventricles. It should be possible also to account for T-wave abnormalities resulting from disease states in which changes in action potential configurations occur, provided the form of the action potential has been defined. The concept of the local gradient should be helpful also in determining the site of a localized area in which action potential configuration has changed.

Summary

A series of experiments was performed to test the validity of a theoretic T-wave model. This model was based on the sequence of depolarization and on multiple action potentials taken in appropriate time phase. The previously introduced concept of the component gradient vector was extended so that this quantity included base-line and local gradient vectors. Component gradient vectors were defined as those vectors produced by a disparity in the configuration of coexisting action potentials. They were directed along the same axis as the depolarization vectors in the area where the component gradient existed. The magnitude of the component gradient vectors was determined by the degree to which the plateau durations and downstrokes of coexisting action potentials differed. Those component gradient vectors that arose from the normal disparity in configuration of ventricular action potentials were defined as base-line gradient vectors, while vectors that arose from a deviation from that state were defined as local gradient vectors. In the experiments reported, the predicted T-wave changes occurred. The maximal T vector tended to move along an imaginary line that was perpendicular to the line closing normal activation fronts in the area in which the local gradient was introduced. In a quantitative test of the model, FRP was measured and used as an indirect measure of the duration of the action potential plateau. The observed changes in T area were proportional to the magnitude of changes in functional refractory period (FRP). The results of these limited experiments indicate that the model should be useful in accounting for the configuration of T waves in states in which localized alterations of action potential configurations occur.

References

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