A Theoretic Model of the T Wave

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Advances in understanding the electrocardiogram may be considered steps in the development of a theoretic model. Notable progress has been made in defining the membrane phenomena concerned in the cellular action potential. The sequence of cardiac excitation has been defined in reasonable detail and this information has been applied in explanation of the QRS complex. There has been relatively little progress, however, in explaining the T wave of the electrocardiogram.

One of the most informative analyses of the T wave was that of Macleod in 1938. That analysis was based on the observed potential difference between active and resting muscle and consisted of a graphic derivation of the ECG from graded stages of activity. The analysis was applied only to a simple muscle strip.

Since that time, cellular action potentials have been recorded, and their form provides a more exact description of the graded activity of cardiac muscle. The sequence of activation has been defined and can also be taken as the sequence of the onset of recovery. Finally, limited data concerning the duration of the refractory period have been obtained. Present information concerning the form of ventricular action potentials, the sequence with which recovery begins, and the pattern of refractory periods permits more detailed analysis of the T wave than previously.

Introduction of a Repolarization Model

It is evident that the T wave of the body surface electrocardiogram is related to the downstroke of the action potential in individual cardiac cells. In comparison to excitation, as reflected by the upstroke of action potentials, the downstroke takes place over a prolonged time. A useful model of the T wave will, therefore, need to consider a sufficient number of moments to define the form of the action potential downstroke as well as a sufficient number of action potentials in appropriate time phase to define the sequence of ventricular recovery. An example of a limited number of action potentials considered in this manner has been presented by Hecht.

In the model the form of a representative action potential from ventricular muscle has been employed (fig. 1). It has been divided into segments, each representing a time unit. The difference in the height of the action

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Figure 1

A diagrammatic ventricular action potential modified from Hoffman and Cranefield is shown. $E$ represents the potential difference during depolarization and $e_1, \ldots, e_m$ represent potential differences during successive time units of repolarization. In the model to be described the action potential was divided into 54 such units.
potential at the beginning and end of each time unit represents the potential difference during that time unit of repolarization \( (e_n) \). The sum of all potential differences during repolarization is equal to the potential difference during depolarization \( (E) \).

\[
E = \sum_{n=1}^{m} e_n
\]

Figure 2 shows a diagram of a circular sheet of excitable tissue. Three moments in the excitatory process have been represented by wavefronts \( (\hat{A}, \hat{B}, \text{and} \hat{C}) \). Each front corresponds to the upstroke of action potentials in that area. Maximum negativity occurs when activation has been completed. The relation of activation fronts to a vertical ECG lead is illustrated. The magnitude of instantaneous depolarization vectors has been expressed as the product of the electromotive force in two adjacent areas \( (E) \) and the length of the lines \( (\vec{A}, \vec{B}, \text{and} \vec{C}) \) which close the activation fronts. The vectors have been drawn perpendicular to these lines and directed toward areas not yet activated.

![Figure 2](image)

**Figure 2**

Diagrammatic representation of a circular sheet of excitable tissue illustrating the relation of activation fronts to the ECG. Parts a through d show successive moments during excitation beginning in area A. \( \hat{A}, \hat{B}, \text{and} \hat{C} \) represent activation fronts and \( \vec{A}, \vec{B}, \text{and} \vec{C} \) are lines closing these fronts. The electrocardiographic effects of the fronts can be represented by the vectors shown which have been made perpendicular to lines \( \vec{A}, \vec{B}, \text{and} \vec{C} \) and given magnitudes proportional to the length of these lines. The ECG resulting from projection of these vectors on a vertical axis is shown. Complete labeling given in part a only.

Figure 3

Three coexisting action potentials are shown. They share a common plateau phase in which no potential differences exist between them. During the downstrokes, potential differences exist between \( A \) and \( B \) (heavy vertical lines) and between \( B \) and \( C \) (broken vertical lines).

The wavefronts \( (\hat{A}, \hat{B}, \text{and} \hat{C}) \) have also been taken to represent the sequence of onset of the recovery process. This process has been considered to consist of a gradual increase in positivity as reflected by the downstroke of the action potential. Because the increase is gradual, multiple action potential downstrokes coexist. Potential differences in multiple areas must be considered in the construction of the instantaneous T vector. Figure 3 shows three coexisting action potentials. After completion of activation, the common plateau represents a state in which no potential differences exist, and the downstrokes represent a state in which multiple potential differences exist.

Figure 4 shows the same excitable tissue illustrated in figure 2, with the fronts \( \hat{A}, \hat{B}, \text{and} \hat{C} \), now enclosing areas in different stages of repolarization. At the completion of the plateau phase of the action potential in area A (fig. 4a) a potential difference \( (e_1) \) exists between area A and area B. The magnitude of the repolarization vector \( (\vec{T}_1) \) during this time unit has been made equal to the product of the potential difference between area A and area B \( (e_1) \) and the line \( (\vec{A}) \) which closes the open ends of the boundary of this repolarizing area. A small vector representing \( \vec{T}_1 \) has been drawn perpendicular to \( \vec{A} \) and directed...
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Diagrams of the same excitable tissue previously illustrated in figure 2 during four moments of repolarization. The wavefronts A, B, and C and the lines closing these fronts A, B, and C are labeled in figure 4a only. Vectors have been drawn perpendicular to the lines closing boundaries between areas in different states of repolarization. These vectors are equal to the product of A, B, and C and the potential differences \( e_{\alpha} \) across boundaries and directed opposite to depolarization vectors. The relationship of repolarization vectors to a vertical ECG lead is shown.

opposite to the direction of the depolarization vector. Similarly during the next time unit, when the plateau phase of the action potential has been completed in area B (fig. 4b), a potential difference \( (e_1) \) exists between areas B and C and a second potential difference exists between areas A and B \( (e_2) \). The values of \( e_1 \) and \( e_2 \) are not equal because the slopes of the downstrokes of the action potentials are different in the first and second time units after the completion of the action potential plateau. The vector during this time unit is equal to the sum of the products of the lines closing areas in different states of repolarization and the potential differences across these boundaries. During each succeeding moment of repolarization the sum of the vectors present at each boundary where potential differences exist must be taken to determine instantaneous T vectors. The sum of all instantaneous T vectors is equal to the T area \( (\vec{AT}) \). If a homogeneous sheet of excitable tissue is considered, the QRS area is equal to the T area because the sum of potential differences during depolarization is equal to the sum of potential differences during repolarization. The QRS and T are of opposite polarity because the QRS represents the upstroke, and the T the downstroke of the action potential. The mathematical expressions for these relationships are presented in the appendix.

These same methods have been applied to a schematic diagram of the heart (fig. 5a to c). Wavefronts have been represented to approximate known information concerning the normal sequence of depolarization. These same wavefronts have also been taken to represent the onset of repolarization. At the completion of the plateau phase of the action potential in area A, a potential difference \( (e_1) \) exists between area A and the remainder of the heart. The magnitude of \( \vec{T_1} \) has been made equal to the product of the potential difference

A diagrammatic representation of the heart showing three moments (a, b, and c) in the repolarization process. The same methods used to determine repolarization vectors which were illustrated in figure 4 are here applied to the more complex geometry of the heart and its activation sequence. The relation of repolarization vectors to a horizontal ECG lead is shown.
between area A and the rest of the heart (e₁) and the length of the line (A) which closes the open ends of the boundary of this repolarizing area. A representation of \( \overrightarrow{T_1} \) has been drawn perpendicular to A and opposite to the direction of the depolarization vector. \( \overrightarrow{T_1} \) has been projected on a horizontal axis as a small positive deflection as shown in the diagrammatic ECG in figure 5a. During the next time unit (fig. 5b) the downstroke of the action potential begins in areas B₁ and B₂. Potential differences now exist between areas A and B₁ (e₂), and areas B₁ and B₂ and the rest of the heart (e₁). To find \( \overrightarrow{T_2} \), each area in a different state of repolarization has been closed by the lines A, B₁, and B₂ and vectors have been drawn to each of these lines. The magnitude of these vectors was found by multiplying the length of the line which closes each repolarizing area and the potential differences in adjacent areas. Representations of each of these vectors are shown perpendicular to the lines which close the repolarizing areas and opposite to the direction of the depolarization vectors. When all of these vectors were summed, \( \overrightarrow{T_2} \) was obtained and was projected on a horizontal axis as a downward deflection. Similarly when the downstroke of the action potential begins in area C, all of the repolarizing areas must be considered (fig. 5c). Vectors of appropriate magnitude and direction were drawn at each boundary and their sum taken to give \( \overrightarrow{T_3} \). The projection of this vector on a horizontal ECG lead is shown.

**Application of the Model to Records from Experimental Animals**

In this application the T vectors have been derived by using instantaneous QRS vectors rather than activation sequence data. It should be noted that neither the sequence of depolarization nor the sequence of repolarization may be inferred from instantaneous QRS or T vectors. Many different combinations of simultaneous depolarization fronts may produce the same instantaneous QRS vector. In the same way instantaneous T vectors may also be the result of repolarization proceeding in several possible combinations of areas. To apply this model to the derivation of T waves from experimental animals, instantaneous QRS vectors were taken to represent the sum of the vectors of all areas depolarizing in one unit of time. A state in which action potentials were of the same shape and duration in all areas of the ventricles was first assumed. In such a heart the sequence in which each area of the ventricles reaches the excitable state is the same as the sequence of depolarization. No gradient exists and the area of a derived T wave equals the QRS area and is of opposite direction.

In figure 6a, a vectorcardiogram of a dog is shown. These records were taken using the triaxial dog lead system. From this QRS loop, a T loop was derived without consideration of the gradient. The maximum vectors of QRS and T loops are in approximately opposite directions. In the derivation of this T loop the previously illustrated diagrammatic action potential was considered to have a duration of 216 msec. This was divided into 54 time units of 4-msec duration each. The height of the action potential was measured at the beginning and end of each time unit. The difference between these two measurements represents the EMF (eₙ) during that time unit of repolarization. Spatial instantaneous QRS vectors were obtained from three orthogonal leads at 4-msec intervals, which correspond to the time units used to divide the action potential. The magnitude and direction of the instantaneous QRS vectors were measured and used to derive instantaneous T vectors. The direction of the instantaneous T vectors was made opposite the QRS vectors. From these values the T loop shown in figure 6b was constructed. Figure 6c shows a tracing of the recorded ECG with the derived T waves with and without a gradient superimposed. T loops derived with a gradient are shown in figure 6d. In the next section the methods used to derive T waves and loops with a gradient will be considered.

In this consideration the action potential
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The frontal and horizontal plane projections of the vectorcardiogram of a dog are shown in a. The triaxial dog lead system was used in making these tracings. The QRS loop which has been plotted from instantaneous QRS vectors measured from the recorded loop is shown in b. The T loop shown was derived from these instantaneous QRS vectors and the downstroke of the action potential using methods described in the text. In the derivation of this loop action potentials were considered to be of uniform duration. The maximal QRS and T vectors are in approximately opposite directions. Tracings of the scalar ECG of this dog are shown in c. T waves derived with and without a gradient are shown. Part d shows QRS loops plotted from the recorded loops and the T waves derived when the action potentials of nonuniform duration were considered.

plateau in certain areas of the ventricles was of longer duration than in others. The first area to be depolarized was then no longer the first to reach the excitable state. In ventricles in which the action potentials are of nonuniform duration, the T-wave area is not equal to the area of the QRS, that is, a gradient exists. Wilson and associates\(^7\) have defined the ventricular gradient as the area of the QRST deflections. They expressed this value as a vector and concluded that “the sum of the areas of the initial and final deflections of any curve which represents the excitation of the ventricular muscle is a measure of the effects produced by local variations in the excitatory process, and particularly by local variations in the duration of the excited state.”

In the present study it was postulated that, just as the mean QRS vector is the vectorial summation of a series of instantaneous QRS vectors, the ventricular gradient vector (\(\mathbf{G}\)) may also be considered to be the summation
of a series of component gradient vectors \( \vec{G}_c \). Component gradient vectors were defined as those vectors produced by variations in the shape or duration of coexisting action potentials. The greater the disparity in the shape or duration of coexisting action potentials the greater the magnitude of the component gradient vector. The value of each component gradient vector must be added to the instantaneous \( T \) vectors \( \vec{T}_n \) as derived without considering the gradient to determine the instantaneous \( T \) vectors \( \vec{T}_e \) when there is a gradient. Further theoretic considerations of this concept are presented in the appendix.

If the shape and duration of the action potentials in all parts of the ventricle were known, the magnitude of \( T \) vectors could be obtained by using instantaneous QRS vectors and potential differences measured from action potentials. By comparing the sequence of depolarization with the sequence with which the excitable state was reached, the direction of these vectors could be obtained. Since in-vivo action potential configuration in multiple areas of the ventricle is not readily available, indirect means were used to infer action potential duration. Van Dam and Durrer have found that the functional recovery of the myocardium follows a regular sequence across the wall of the ventricle. In the innermost layers the functional refractory period is approximately 15 msec longer than in the middle and subepicardial layers. The sequence of functional recovery closely follows the sequence of depolarization in the middle and outer thirds of the ventricle. Action potentials of the same duration may then be considered to occur in concentric rings, or in a cup shape. Since the component gradient vectors are produced by a disparity in the duration of the action potentials, the gradient, in this sense, is also cup-shaped. Scher and Young's data on the normal sequence of depolarization indicate that the activation wavefronts also assume a cup shape. Insofar as these generalizations are true, the instantaneous depolarization vectors may be used together with the gradient vector \( \vec{G} \) to derive simultaneous equations to infer the duration of the action potential plateau. This relationship is discussed further in the appendix.

Changes in the relationship of the time sequence of depolarization and recovery are shown in figure 7. Figure 7a shows a diagram of a strip of excitable tissue. Figure 7b shows the order of recovery in a case in which there is no gradient. Figure 7c shows a case in which the plateau phase of the action potential in area A is prolonged by 1 time unit. In this case area A and area B complete the action potential plateaus at the same time and there is no potential difference between these areas.

![Figure 7](http://circ.ahajournals.org/)

Illustration of the relationship of activation and recovery with various action potential durations. Part a represents a ventricular muscle strip with activation proceeding toward the epicardium. In parts b through e the upper margins of the shaded areas represent the depolarization sequence. The duration of the action potential plateaus is represented by the height of the shaded areas. The potential differences between areas during repolarization are represented by the steps on the lower margins of the shaded areas. In b, the duration of the action potential plateau is equal in all areas. In c, the action potential plateau is considered to be 1 time unit longer in area A than in the rest of the strip. Prolongation of the action potential in area A by 2 and 3 time units is illustrated in d and e.
areas. The potential differences that exist between all of the other areas are the same as they were in the case without a gradient.

In Figure 7d the plateau phase of action potentials in area A has been prolonged by 2 time units. Area B now completes the plateau phase 1 time unit before area A, and area A and area C complete the plateau phase at the same time. The potential difference between area A and area B is now the same as it was in the case without a gradient but is now a positive value. The potential differences between area B and area C and all other areas are again the same as they were in the case without a gradient. The effect of prolongation of the plateau phase of the action potential in area A by 3 time units is shown in figure 7e.

Figure 8 shows the sequence in which the plateau phase of action potentials was completed in the dog whose records were shown in figure 6. This sequence of repolarization was determined by methods described in the appendix. Formulas for instantaneous T vectors for the pattern of recovery were derived and these are also presented in the appendix. The T waves and T loops derived in this way were shown in figure 6c and d.

T waves derived in two additional dogs are shown in figure 9. Patterns of refractoriness in all three animals closely resembled the pattern of refractoriness found experimentally by van Dam and Durrer.

Discussion

The model presented in this paper represents a step in relating the form of the intracellular action potential to the configuration of T waves. If the sequence of activation is known and if the shape and duration of action potentials in each area enclosed by an activation front are also known, T waves may be derived using the methods described. However, in experimental animals the information required to derive T waves is not known in precise terms and certain simplifying assumptions were made: (1) Depolarization vectors were taken to represent the lines closing activation fronts and it was assumed that depolarization proceeded from endocardium to epicardium in cup-shaped wavefronts. (2) It was assumed that variations in the configuration of ventricular action potentials were limited to variations in plateau duration. (3) It was assumed that action potentials in individual layers of the ventricles tend to have plateaus of the same duration. (4) It was assumed that action potentials of similar durations could, in an approximate way, be assigned to areas represented by instantaneous QRS vectors.

For the purposes of the model a special view of the gradient was developed. It was considered that just as the mean QRS vector

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**Figure 8**

The relationship of the sequence of activation and recovery in an experimental animal. The sequence of instantaneous QRS vectors at 4-msec intervals is represented by the steps on the upper margin of the shaded area. The sequence of completion of action potential plateaus in the areas represented by instantaneous QRS vectors is shown by the steps along the lower margin. The height of the shaded area represents the relative duration of the action potential plateau. The upper and lower sections of the shaded area are separated because the absolute value of the action potential duration was not known. Those areas represented by the vectors V₁ to V₇ have action potential plateaus of the shortest duration. Areas represented by the vectors V₃, V₇, and V₁₀ have action potential plateaus of longer duration, and those areas represented by the vector V₉ have the longest action potential plateaus. If it is assumed that depolarization vectors represent cup-shaped activation fronts that proceed from endocardium to epicardium, the order of action potential plateau completion closely approximates the pattern of refractoriness found experimentally by van Dam and Durrer; that is, action potential plateaus of longest duration were found in endocardial areas; those of shortest duration, in the middle layers, and those of intermediate duration, in the epicardial layers.
Figure 9

Scalar ECGs of two dogs (a and b). T waves derived with the computer model are shown as dotted lines superimposed on tracings of recorded T waves.

is the sum of a series of instantaneous QRS vectors, the gradient vector is the sum of a series of component gradient vectors. Component gradient vectors were defined as those vectors produced by a disparity in the shape or duration of coexisting action potentials. By use of these assumptions and the concept of the component gradient vector, simultaneous equations were derived which related the gradient measured from three orthogonal leads to the sum of the product of the k value (the disparity in the durations of the plateau phases in areas represented by instantaneous QRS vectors) and the magnitude of instantaneous QRS vectors in these three leads. For three animals the derived simultaneous equations were solved with a computer. The pattern of ventricular recovery determined by these means closely resembled that found by van Dam and Durrer,8 and the T waves derived from these patterns closely resembled the T waves recorded from the experimental animals. The values of ventricular refractory periods found by van Dam and Durrer could also have been used to assign appropriate durations to the plateau phase of action potentials in layers of the myocardium. This would have required average values which would not necessarily apply to the dogs studied. The indirect means used in this study to infer action potential duration were limited to use in deriving T waves that followed a normal sequence of excitation. The T waves that follow abnormal activation patterns present a different problem. In the case of premature ventricular contractions, for example, the gradient probably retains its cup shape, that is, action potentials in individual layers
of the myocardium tend to be of similar duration. The sequence of depolarization, however, is abnormal and depolarization vectors can no longer be assumed to represent myocardial layers with action potentials of similar durations. Therefore, component gradient vectors can no longer be correlated with depolarization vectors.

As further information is accumulated concerning the shape and duration of action potentials of the in-vivo ventricle, the model may be used with greater precision. The work of Toyoshima and co-workers in which action potentials were recorded from the ventricles of dogs during warming and cooling of the heart represents a step in the systematic recording of in-vivo ventricular action potentials.

No attempt has been made to predict the form of the S-T segment. In fact, a diagrammatic action potential with a long flat plateau phase was purposely chosen so that coexisting action potentials shared some period of time during which there was no potential difference and an isoelectric S-T segment was derived. To account for abnormalities of the S-T segment, it would have been necessary to consider several additional factors: (1) If the slope of the plateau phase of action potentials in one area of the heart differed from the slope of plateaus in other areas, a potential difference would exist and be represented on the body surface ECG as a deviation from the isoelectric line. (2) If the plateau phase was steep in all areas, the S-T segment would not have been isoelectric. (3) Local changes in the resting potential without changes in the shape or duration of the action potential would also have produced S-T segment displacements. At present, it would be difficult to determine which of these factors was responsible for S-T segment displacement in a given electrocardiogram.

In spite of its shortcomings, the analysis presented is a versatile model of ventricular repolarization that should prove to be useful in predicting the form of T waves and loops both in normal hearts and eventually in predicting T-wave form in the presence of drugs and anatomic lesions.

Summary

A theoretical model of the T wave based on the sequence of depolarization and multiple action potentials in appropriate time phase has been presented. The methods used to derive T waves and loops from the QRS complex of experimental animals have been described, and derived T waves were closely similar to the T waves recorded from these animals. The findings indicate utility of the model in understanding normal T waves and suggest it may be useful in accounting for abnormal T waves.

Appendix

An instantaneous repolarization vector may be expressed as the sum of the products of the line closing the uncanceled portion of a boundary between areas in different states of repolarization and the potential difference across this boundary. It is directed opposite to the depolarization vector. For the homogeneous excitable tissue diagrammed in figure 4, the first repolarization vector occurs when area A completes its action potential plateau (fig. 4a) and may be expressed:

\[ \mathbf{T}_1 = \mathbf{A} \times \mathbf{e}_1 \]

\( \mathbf{A} \) represents the line closing the boundary between area A and the remainder of the excitable tissue shown, and \( \mathbf{e}_1 \) represents the potential difference across the boundary A. The potential difference \( \mathbf{e}_1 \) was found by measuring the difference in the height of a representative action potential at the beginning and end of the first time unit after completion of the plateau. During subsequent moments of repolarization potential differences are present at several boundaries. The vectors at each boundary must be summed to obtain an instantaneous repolarization vector.

The expressions for \( \mathbf{T}_2 \), \( \mathbf{T}_3 \), and \( \mathbf{T}_n \) are shown below:

\[ \mathbf{T}_2 = \mathbf{A} \times \mathbf{e}_2 + \mathbf{B} \times \mathbf{e}_1 \]
\[ \mathbf{T}_3 = \mathbf{A} \times \mathbf{e}_2 + \mathbf{B} \times \mathbf{e}_2 + \mathbf{C} \times \mathbf{e}_1 \]
\[ \mathbf{T}_n = \mathbf{A} \times \mathbf{e}_n + \mathbf{B} \times \mathbf{e}_{n-1} + \mathbf{C} \times \mathbf{e}_{n-2} \]

Since the sum of the potential differences during the downstroke \( (\Sigma \mathbf{e}_n) \) is equal to the potential difference during the upstroke \( (\mathbf{E}) \) the area of the QRS is equal to the area of the T:

\[ \mathbf{T} = \frac{m+2}{n} \sum_{n=1}^{m+2} \mathbf{A} \times \mathbf{e}_n + \mathbf{B} \times \mathbf{e}_n + \mathbf{C} \times \mathbf{e}_n \]

To derive T waves from excitable tissue with action potentials of nonuniform duration, a special
A diagram of a circular sheet of excitable tissue is shown during a moment of repolarization. \( \vec{A} \), \( \vec{B} \), and \( \vec{C} \) represent the activation fronts that have passed through this tissue. The tissue below \( \vec{C} \) is considered to have an action potential plateau duration 2 time units longer than the tissue above \( \vec{C} \). \( \vec{A}, \vec{B}_1, \vec{B}_2, \vec{B}_3, \vec{C}_1, \vec{C}_2, \) and \( \vec{C}_3 \) represent the lines closing boundaries where potential differences exist. Vectors must be determined at each boundary and their sum taken to determine an instantaneous \( T \) vector. The potential differences at each boundary are listed in the fourth column of table 1 and the signs of these potential differences are in the fifth column of table 1. See text for further details.

Figure 10 shows a situation in which a gradient exists. For the purposes of illustration the action potentials below the dotted arc (\( \vec{C} \)) were considered to be 2 time units longer than those above \( \vec{C} \). A line \( \vec{C} \) was drawn to close the open ends of \( \vec{C} \). A line perpendicular to \( \vec{C} \) and equal to the product of \( \vec{C} \) and \( \Sigma e_n \), would represent the component gradient vector. In this case \( \Sigma e_n \) represents potential differences across \( \vec{C} \) due to variations in action potential configuration.

\[
\vec{C}_e = \vec{C} \times k \Sigma e_n
\]

The value \( k \) is equal to the degree to which the duration of the action potential plateaus differ across \( \vec{C} \), and in this case is equal to 2. Since action potentials below \( \vec{C} \) were considered to be of longer duration than those above \( \vec{C} \), this is an area which is relatively negative throughout repolarization. The component gradient vector is therefore opposite to the direction of the \( T \) vector. If the action potentials were shortened below \( \vec{C} \), \( \vec{C}_e \) would be in the same direction as the \( T \) vector, that is toward area \( A \).

Instantaneous \( T \) vectors may be calculated for the excitable tissue shown in figure 10 either by taking the sum of the products of the lines closing each boundary where a potential difference exists and the potential differences across these boundaries, or by calculating the \( T \) vectors as if there were no gradient and then adding the value of the component gradient vectors. Potential differences that are present when the gradient is considered are listed in column 4 of table 1, and potential differences present when there is no gradient are listed in column 2 of table 1. Using the first method the instantaneous \( T \) vector at time \( (T_{Gn}) \) may be stated:

\[
T_{Gn} = -\vec{A} \times e_{n-1} + \vec{B}_2 \times (e_n + e_{n-1})
- (\vec{B}_1 + \vec{B}_3) \times e_{n-2} - \vec{B}_2 \times e_n
+ (\vec{C}_1 + \vec{C}_2) \times (e_{n-1} + e_{n-2})
- \vec{C}_2 \times e_{n-1}
\]

If the value \( \vec{B}_2 \times e_{n-2} \) is added and subtracted to this equation the terms may be collected to give the formula:

\[
T_{Gn} = -\vec{A} \times e_{n-1} - (\vec{B}_1 + \vec{B}_2 + \vec{B}_3) \times e_{n-2}
- \vec{C}_2 \times e_{n-1}
+ (\vec{C}_1 + \vec{C}_2 + \vec{B}_2) \times (e_{n-1} + e_{n-2})
\]

The sum of the vectors drawn to \( \vec{B}_1, \vec{B}_2, \) and \( \vec{B}_3 \) is equal to the vector drawn to \( \vec{B} \), and the sum of the vectors drawn to \( \vec{C}_1, \vec{C}_3, \) and \( \vec{B}_2 \) is equal to the vector drawn to \( \vec{C} \). The last formula may therefore be rewritten for \( \vec{A} \):

\[
\vec{A}T_{G} = -\vec{A} \Sigma e_n - \vec{B} \Sigma e_n - \vec{C}_2 \Sigma e_n + 2 \vec{C} \Sigma e_n
\]

which is the equivalent of calculating the repolarization vectors without a gradient and adding the value of the component gradient vectors. For convenience, in this application \( e_n \) was considered a positive value and each term of the equation was multiplied by the appropriate sign. \( \vec{C} \Sigma e_n \) has been multiplied by 2 because the sum of potential differences have been taken at 2 time units, \( n-1 \) and \( n-2 \). From this formula it can be seen that the value of vectors produced at
Table 1

Information Concerning the Tissue Diagrammed in Figure 10

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<th>Without gradient</th>
<th>With gradient</th>
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<td>( e_{n-1} )</td>
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<td>( e_{n-1} )</td>
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</table>

Explanation of table: The potential differences and the signs of the potential differences when the tissue in figure 10 is considered to have action potentials of uniform duration are listed under the heading, “without gradient.” The potential differences and their signs when tissue below \( \bar{C} \) has action potentials 2 time units longer than the tissue above \( \bar{C} \) are listed under the heading, “with gradient.” The lines closing each boundary are listed in the first column. An instantaneous T vector is equal to the sum of the products of the length of the lines closing each boundary and the potential differences across each boundary.

*\( \bar{B}_2 \) is listed twice because it closes both the middle section of \( \bar{C} \) and the middle section of \( \bar{B} \).

boundaries of areas with action potentials of different configurations is independent of the sequence of depolarization.\(^10\)

To derive T waves from recorded QRS complexes, the magnitudes of instantaneous QRS vectors were considered to represent those of lines closing activation fronts. The downstrokes of action potentials were considered to be of the same duration in all areas, but the duration of the plateaux varied. Action potentials in each area enclosed by an activation front were considered to be of the same duration. The gradient, therefore, may be considered to equal the sum of the products of \( k \), the degree to which plateau durations varied in adjacent areas, and \( V_n \), the lines closing boundaries between areas with action potentials of different configurations:

\[
\bar{G} = k_1 V_1 + k_2 V_2 + \ldots + k_n V_n
\]

If the plateau phase was prolonged 2 time units in those areas expressed by \( V_2 \) and there was no prolongation of plateaux in other areas, the gradient vector would be:

\[
\bar{G} = 2 V_2 + 2 V_2 + \ldots + 0 V_n
\]

In tracings taken on experimental animals, the gradient and 10 instantaneous QRS vectors were measured in the \( X \), \( Y \), and \( Z \) leads and simultaneous equations were written:

\[
\begin{align*}
G_X &= k_1 V_1 + k_2 V_2 + \ldots + k_{10} V_{10} \\
G_Y &= k_1 V_1 + k_2 V_2 + \ldots + k_{10} V_{10} \\
G_Z &= k_1 V_1 + k_2 V_2 + \ldots + k_{10} V_{10}
\end{align*}
\]

An IBM 1620 computer was used to solve these simultaneous equations for the values \( k_1 \) \ldots \( k_{10} \). Those \( k \) values were used which gave values for \( G_X \), \( G_Y \), \( G_Z \) that most closely corresponded to the gradients measured from records of experimental animals.

Figure 8 shows a diagram of the pattern of refractoriness in the dog whose records were shown in figure 6. The \( k \) values in this dog were as follows:

\[
\begin{align*}
k_1 &= 0, \quad k_2 = 2, \quad k_3 = 4, \quad k_4 = 1, \quad k_5 = 1, \quad k_6 = 1, \\
k_7 &= 1, \quad k_8 = 0, \quad k_9 = 0, \quad k_{10} = 1
\end{align*}
\]

If the \( k \) values were 0, action potential plateaux were considered to be of the same duration in two adjacent areas. Since the upstrokes in adjacent areas were considered to be separated by a time unit, the completion of plateaux in adjacent areas was also considered to be separated by a time unit. When the \( k \) values equalled 1, plateaux in the first area to be depolarized were 1 time unit longer than those in the next area to depolarize and their action potential plateaux were completed at the same time. In figure 8, areas represented by vectors \( V_4 \) through \( V_8 \) complete their action potentials at the same time and no potential differences exist between them. These areas are the first to reach the end of the plateau phase. At the end of the next time unit, area I completes its plateau, and at that moment a potential difference exists between areas H and I, the boundary that gave rise to \( V_8 \), and also at the boundary between C and D, the boundary that gave rise to \( V_3 \).

\[
T_{G_1} = V_3 \times e_1 - V_8 \times e_1
\]

In this animal repolarization vectors existed only at the boundaries where the vectors \( V_1 \), \( V_2 \), \( V_3 \),...
V₈ and V₉ arose. The general formula for T vectors in this animal may be expressed:

\[ \mathbf{T}_{\alpha_n} = -V_1 \times e_{n-3} + V_2 \times e_{n-1} + V_3 (e_n + e_{n-1} + e_{n-2}) - V_8 \times e_n - V_9 \times e_{n-1} \]

The T waves and loops derived with this formula were shown in figure 6c and d.

References


Emerson on Scholarship

There is then creative reading as well as creative writing. When the mind is braced by labor and invention, the page of whatever book we read becomes luminous with manifold allusion. Every sentence is doubly significant, and the sense of our author is as broad as the world. We then see, what is always true, that as the seer's hour of vision is short and rare among heavy days and months, so is its record, perchance, the least part of his volume.—Brooks Atkinson (Ed.): The Complete Essays and Other Writings of Ralph Waldo Emerson. In The Modern Library, New York, Random House, 1950, p. 51.
A Theoretic Model of the T Wave
KENICHI HARUMI, MARY JO BURGESS and J. A. ABILDSKOV

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