Analysis of T-Wave Abnormalities Associated with Myocardial Infarction Using a Theoretic Model

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SUMMARY
The physiological basis of serial T-wave abnormalities associated with myocardial infarction was studied. After coronary artery ligation in dogs, ventricular functional refractory periods (FRPs) were measured at five to eight epicardial, intramural, and endocardial sites. FRPs measured during acute ischemia shortened an average of 26 msec. FRPs measured 24 to 72 hr after coronary ligation were 24 msec longer at ischemic than at nonischemic sites. Alterations of recovery times were analyzed in terms of a repolarization model which related the form of the action potential downstroke to the form of the T wave of the body surface ECG. Observed FRPs were used to infer action potential duration and T waves were derived. Shortening FRPs in the anterolateral wall of the ventricle increased the amplitude of derived T waves in the X lead and caused T-wave inversion in the Z lead. Prolonging FRPs in the same area caused T-wave inversion in lead X and increased T amplitude in lead Z. The form of derived T waves qualitatively corresponded to serial T-wave abnormalities seen in patients with myocardial infarction.

Additional Indexing Words:
T-wave model
Transmembrane action potential form
Ventricular refractory period
Coronary ligation

The recognition of myocardial infarction is one of the major diagnostic applications of the electrocardiogram. The physiological mechanisms of QRS abnormalities resulting from destruction of excitable tissue and alteration of intraventricular conduction have been reasonably well defined, but the mechanism of ST-T-wave abnormalities is less completely understood. The recent development of a theoretic model of repolarization relating the downstroke of the transmembrane action potential to the T wave of the body surface ECG permits a more detailed analysis of T-wave abnormalities than was previously possible. The present study was undertaken to analyze in terms of that model the early increase in amplitude of later T-wave inversion associated with myocardial infarction. Electrocardiograms of two patients are shown in figure 1 to illustrate these abnormalities. The first tracing of each series was taken within 2 hr of the onset of substernal pain. T waves were tall and peaked in leads I, aV_L, and V_2 to V_5. In later tracings QRS abnormalities, characteristic of infarction, were present, and T waves were inverted in leads in which they were initially tall and peaked.

Since an explanation of T-wave abnormalities associated with myocardial infarction...
must account for both the early peaking and later inversion of T waves, the effects of ischemia on ventricular recovery times were studied in both acute and chronic experimental animal preparations. As has been repeatedly observed, functional refractory periods (FRPs) were shortened with acute ischemia, and as reported by Wilson and associates, the FRPs were prolonged with chronic ischemia. FRPs were used to infer action potential duration and T waves were derived using the model. The derived T waves qualita-

tively corresponded to T-wave abnormalities that have been observed both in experimental animals and in patients with myocardial infarction.

Methods
Experiments were performed on 10 mongrel dogs ranging in weight from 14.5 to 24.5 kg. Pentobarbital, 30 mg/kg intravenously, was used for anesthesia. Five dogs were studied acutely. In these animals the chest was opened with a sternal splitting incision, while respirations were maintained with a Harvard pump respirator. The pericardium was opened and a pericardial cradle...
was fashioned. The sinus node was crushed, and a bipolar electrode was attached to the right atrium so that a constant rate could be maintained by electrical stimulation. Small unipolar hook electrodes, insulated except at their tips, were used for recording and stimulating in the measurement of FRPs at multiple epicardial and endocardial sites. A bipolar reference electrode was placed on the ventricle at a distance from the testing electrodes to detect propagated ventricular responses. The response to the atrial stimulus at the bipolar reference electrode was displayed on one channel of an oscilloscope. The response at the unipolar testing electrode was displayed on the other channel and aligned on the vertical axis, with the intrinsic deflection recorded from the reference electrode. With this procedure, conduction time between testing electrode and reference electrode was not included in the measurement of FRP. The sweep speed of the oscilloscope was calibrated at 1 cm/50 msec. Stimuli from the atrial stimulus generator were passed through a counter, and every sixth stimulus triggered the sweep of the oscilloscope. A second stimulator was used to deliver cathodal stimuli of 2-msec duration and one and a half to two times 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.

A branch of the anterior descending coronary artery was isolated and a loose ligature placed around it. Base-line FRPs were measured at five to eight endocardial and epicardial sites within the area of potential ischemia, and at two to three additional epicardial sites distant from the area of potential ischemia. The sites distant from the area of ischemia served as controls after coronary ligation. FRPs were measured immediately after ligation and at 15-min intervals for periods up to 4 hr. The hearts were preserved in Formalin and later sectioned to confirm the site and extent of lesions.

Five dogs were studied chronically. These animals were anesthetized with 30 mg/kg intravenous pentobarbital; a left lateral thoracotomy was performed, the pericardium opened, and a branch of the anterior descending coronary artery isolated and ligated. The pericardium was loosely approximated and the chest incision closed. The triaxial dog lead system was used to take daily body surface electrocardiograms. After 24 to 72 hr, when QRS and T abnormalities were present, each dog was again anesthetized and the chest opened with a sternal splitting incision. The pericardium was incised and a pericardial cradle formed. Intramural ventricular FRPs were measured with a multi-electrode needle (DISA electrode 13 K 95). The multi-electrode needle had eight 1.0 by 0.1-mm platinum unipolar electrodes placed at regular intervals over an 11-mm length of the needle shaft, which had a diameter of 0.65 mm. FRP measurements were made at multiple control and ischemic sites. At the end of the experiment the animals were killed and the hearts removed for pathological examination.

FRP values were used to infer action potential duration and T waves derived. The model used in these derivations has previously been reported in detail and only those points pertinent to understanding the present study will be summarized here. In the model, the T wave of the body surface ECG was considered to have the same relationship to the downstroke of the transmembrane action potential that the QRS has to the action potential upstroke. Depolarization is a rapid process, and an instantaneous QRS vector may be considered to equal the product of the length of the line closing an activation front and the potential difference across the front, with the action potential upstroke representing this potential difference. A depolarization vector was considered to be perpendicular to the line closing the activation front and directed toward areas not yet activated (fig. 2). Instantaneous repolarization vectors may be expressed in a similar manner; that is, they may be considered to equal the sum of the products of the lengths of the lines closing repolarizing boundaries and the potential differences across them. Since repolarization is a slow process, multiple action potentials coexist and potential differences at several repolarizing boundaries must be considered in determining an instantaneous repolarization vector. In applications of the model, a diagrammatic ventricular transmembrane action potential adapted from Hoffman and Cranefield was used. The action potential was divided into 54 time units, and the difference in its height from the beginning to the end of each time unit was taken as the potential difference during a given moment of repolarization. Action potentials of uniform duration were assigned to each ventricular area included in a single activation front. Therefore, only boundaries considered during activation were considered during recovery, and the lines closing activation fronts also closed repolarizing boundaries. Van Dam and Durrer's data on normal canine intramural ventricular refractory periods were used to define the duration of ventricular action potentials. As illustrated in figure 2, the direction of repolarization vectors was determined by
comparing the sequence of activation in areas adjacent to those of recovery. If an area depolarized and completed its action potential plateau before its adjacent area, the sign of the potential difference across the boundary of these two areas during recovery was opposite to the sign of the potential difference across their boundary during activation, and the repolarization vector was opposite to the direction of the depolarization vector (fig. 2a). If the first area to be depolarized was not the first to complete its action potential plateau, the sign of the potential difference across the boundary of these two areas was the same during repolarization as during depolarization and the repolarization vector was in the same direction as the depolarization vector (fig. 2b).

Ventricular activation patterns were not determined in these experiments, and one set of Scher’s horizontal plane diagrams of the normal ventricular activation sequence of the dog was used.

Four diagrams parallel to the frontal plane were constructed from the horizontal plane diagrams and instantaneous depolarization vectors were derived for the six moments of activation represented. Each simultaneously occurring activation front was closed by a line and a vector drawn perpendicular to it. The magnitude of the vector was made proportional to the line closing the activation front and to the thickness of the slice of ventricle in which it occurred. Depolarization vectors were directed toward areas not yet activated. The sum of all simultaneously occurring vectors was taken and projected on an orthogonal lead system. Horizontal plane vectors were used for the X and Z projections, and frontal plane vectors were used for the Y projection. Van Dam and Durrer’s data on normal intramural ventricular functional refractory periods were used to infer action potential duration. Action potentials of the shortest duration were assigned to the middle layers of the myocardium, those of intermediate duration were assigned to the outer layers, and those of longest duration were assigned to endocardial layers. T waves representing the control state were derived. To derive T waves representative of acute ischemia, the area of ischemia was assigned action potentials of shorter duration than nonischemic areas were. This assignment of action potentials corresponded to the FRPs measured in experimental animals. To derive T waves representative of chronic ischemia, action potentials of normal duration were assigned to nonischemic areas, and action potentials of longer duration were assigned to the area of chronic ischemia. This assignment of action potential duration also corresponded to the experimental findings.

Figure 2

Diagrams of a section of ventricle which has been divided by a broken line into an inner and outer layer are shown to illustrate the assignment of T vector direction for the theoretic model. In part a, the action potentials assigned to the inner layer are of shorter duration than those assigned to the outer layer, and in part b, the action potentials assigned to the inner layer are longer than those assigned to the outer layer. The two upper diagrams represent a moment of activation when the inner layer of the ventricle has depolarized. The upstroke of the action potential in this layer is shown as a solid line to indicate that it has been completed. The remainder of the action potential in the inner layer and the action potential in the outer layer are shown as broken lines to indicate that they have not yet been completed. When the upstroke is completed in the inner portion of the ventricle, this layer is relatively negative with respect to the outer portion of the ventricle, and the QRS vector has been directed from the area of relative negativity to the area of relative positivity, that is toward the epicardium. The two lower diagrams represent a moment of repolarization. In part a, the action potential in the outer layer is still in the plateau phase during the downstroke of the action potential in the inner layer. With this circumstance the inner layer is relatively positive with respect to the outer layer. The sign of the potential difference across the boundary of the inner and outer layers is opposite to the sign of the potential difference across their boundary during depolarization, and the repolarization vector was assigned a direction opposite to that of the QRS vector. In part b, the action potential in the inner layer is still in the plateau phase during the downstroke of the action potential in the outer layer. With this circumstance the inner layer of the ventricle is relatively negative with respect to the outer layer, the sign of the potential difference across the boundary of the inner and outer layers is the same during repolarization as it was during depolarization, and the T vector has been assigned the same direction as the depolarization vector.

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Table 1
Average Functional Refractory Period Before and After Coronary Artery Ligation

<table>
<thead>
<tr>
<th>Exp. no.</th>
<th>Average control FRPs (msec)</th>
<th>Average shortening at time of maximal shortening (msec)</th>
<th>Average shortening at time of maximal shortening (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>226</td>
<td>19</td>
<td>8</td>
</tr>
<tr>
<td>2</td>
<td>211</td>
<td>6</td>
<td>3</td>
</tr>
<tr>
<td>3</td>
<td>237</td>
<td>36</td>
<td>15</td>
</tr>
<tr>
<td>4</td>
<td>220</td>
<td>25</td>
<td>12</td>
</tr>
<tr>
<td>5</td>
<td>200</td>
<td>46</td>
<td>23</td>
</tr>
<tr>
<td>Average</td>
<td>219 msec</td>
<td>26 msec</td>
<td>12%</td>
</tr>
</tbody>
</table>

Results

Acute Studies

Stimulation threshold increased after coronary artery ligation at many of the ischemic sites, and some sites were transiently or permanently inexcitable. In each of the experimental animals, FRPs shortened at all excitable ischemic test sites, and most endocardial sites shortened more than epicardial sites. In four animals, shortening of FRPs at all test sites occurred within 2 min of coronary artery ligation. In the remaining animal, shortening of FRPs occurred within 2 min of coronary ligation at all but one of the test sites. In two animals, maximal shortening occurred within 2 min of coronary artery ligation; in the remaining three animals, maximal shortening occurred 1 hr 15 min to 1 hr 40 min after ligation. Table 1 lists the average values of FRPs prior to coronary artery ligation, and the average values of FRPs after ligation, at the time when maximal shortening occurred. Considering all experiments, the average of FRPs at the time of maximal shortening was 26 msec or 12% less than the average preligation value.

Graphs of the time course of average change in FRPs at ischemic epicardial, ischemic endocardial, and nonischemic epicardial sites are shown in Figure 3. As shown in the graphs, endocardial test sites shortened more than epicardial test sites in all animals. Considering all animals, the average shortening at the endocardial sites was 42 msec or 20%, and the average shortening at epicardial sites was 20 msec or 9%. The difference in magnitude of shortening of FRPs at epicardial and endocardial sites may be due, in part, to epicardial cooling in these open-chest dog preparations. However, FRPs were measured at nonischemic epicardial control sites and, at the time when maximal shortening occurred at ischemic sites, FRPs at nonischemic sites were an average of only 4 msec longer than preligation base-line values. This prolongation at control sites was not sufficient to

![Figure 3](http://circ.ahajournals.org/)

Graphs showing the changes in FRPs after coronary artery ligation in the five acute experiments. Averages of the change in FRP at nonischemic control sites, ischemic endocardial sites, and ischemic epicardial sites are shown. There are no control values in graph a, because in this experiment all the testing electrodes were within the area of ischemia. In each of the experiments FRPs shortened more at endocardial than at epicardial sites. FRP shortening at both endocardial and epicardial sites lasted less than 30 minutes in two of the experiments. In the other experiments FRP shortening at endocardial sites persisted throughout the course of experiments but at epicardial sites prolongation of FRPs followed the initial shortening in all but one of the experimental animals.
Analyses of T-Wave Abnormalities

Electrocardiograms of a dog taken prior to and 72 hours after coronary ligation. These tracings were taken with the triaxial dog lead system. In this lead system the anterior chest electrodes are negative with respect to the posterior chest electrode. In the control tracing the T waves are biphasic in the X lead, upright in the Y lead, and inverted in the Z lead. In the tracing taken 72 hours after coronary ligation the T waves are deeply inverted in the X and Y leads, and QRS changes characteristic of infarction are present and representative of the kind of changes present when FRPs were measured in the chronic experiments.

Interestingly, in the control tracing the T waves were biphasic in the X lead, upright in the Y lead, and inverted in the Z lead. In the tracing taken 72 hours after coronary ligation the T waves were deeply inverted in the X and Y leads, and QRS changes characteristic of myocardial infarction were present. When such characteristic changes occurred, the animals were anesthetized, the chests opened, and intramural FRPs measured. For each dog averages were taken of FRPs at ischemic and nonischemic sites and are shown in table 2. In every animal FRPs were longer at ischemic than at nonischemic sites. Considering all animals, FRPs at ischemic sites averaged 24 msec or 13% longer than FRPs at nonischemic sites.

Intramural FRPs at ischemic and nonischemic sites of one representative dog are shown in figure 4. Averages of FRPs measured at the same level of the ventricular wall at each ischemic site were taken and are shown as a broken line on the graph. Averages of FRPs from nonischemic sites are shown as a solid line. FRPs were prolonged in all layers of the ischemic myocardium, and the usual relationship of FRP durations in endocardial, intramural, and epicardial layers was preserved. That is, FRPs were of the longest duration at endocardial sites, of intermediate duration at epicardial sites, and

**Table 2**

*Average Functional Refractory Periods in Chronic Ischemia*

<table>
<thead>
<tr>
<th>Exp. no.</th>
<th>Nonischemic sites</th>
<th>Ischemic sites</th>
<th>Average FRP duration</th>
<th>Average FRP prolongation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>167</td>
<td>190</td>
<td>23</td>
<td>14</td>
</tr>
<tr>
<td>2</td>
<td>212</td>
<td>235</td>
<td>23</td>
<td>11</td>
</tr>
<tr>
<td>3</td>
<td>197</td>
<td>221</td>
<td>24</td>
<td>12</td>
</tr>
<tr>
<td>4</td>
<td>197</td>
<td>230</td>
<td>33</td>
<td>17</td>
</tr>
<tr>
<td>5</td>
<td>181</td>
<td>197</td>
<td>16</td>
<td>9</td>
</tr>
<tr>
<td>Average</td>
<td>191 msec</td>
<td>215 msec</td>
<td>24 m sec</td>
<td>13%</td>
</tr>
</tbody>
</table>

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of shortest duration at intramural sites. In nonischemic areas, however, the FRPs were longer at epicardial than at endocardial sites. This finding was probably due to epicardial cooling in these open-chest preparations. However, in spite of epicardial cooling, the prolongation of FRPs at endocardial ischemic sites was greater than the prolongation of FRPs at epicardial ischemic sites in all but one animal (table 3). When all experiments were considered, FRPs at epicardial ischemic sites averaged 19 msec longer than those at nonischemic epicardial sites, and FRPs at endocardial ischemic sites averaged 31 msec longer than those at nonischemic endocardial sites.

Derived T Waves
The theoretic model of repolarization described in the "Section on Methods" was used in the derivation of T waves. ECGs derived for a control state, and states of acute and chronic anterolateral wall ischemia are shown in figure 6. In the derivation of T waves representative of acute ischemia, the action potentials in the ischemic area were shortened by an amount that corresponded to the FRPs experimentally observed. When T waves were derived for chronic ischemia, action potentials in the ischemic area were prolonged by an amount corresponding to experimental observations. In the tracing representative of the control state, the derived T waves were inverted in the X and Z leads, and were isoelectric in the Y lead. In the tracing representative of acute ischemia the derived T waves were taller in the X lead and more deeply inverted in the Z lead, and in the tracing representative of chronic ischemia the T waves were more deeply inverted in the X lead than in the control tracing and were upright in the Z lead. The

Table 3
Comparison of Endocardial and Epicardial Functional Refractory Period Prolongation in Chronic Ischemia

<table>
<thead>
<tr>
<th>Dog number</th>
<th>Endocardial FRPs</th>
<th>Epicardial FRPs</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Avg. of FRPs nonischemic sites</td>
<td>Avg. of FRPs ischemic sites</td>
</tr>
<tr>
<td>1</td>
<td>163</td>
<td>189</td>
</tr>
<tr>
<td>2</td>
<td>204</td>
<td>241</td>
</tr>
<tr>
<td>3</td>
<td>198</td>
<td>230</td>
</tr>
<tr>
<td>4</td>
<td>194</td>
<td>228</td>
</tr>
<tr>
<td>5</td>
<td>175</td>
<td>201</td>
</tr>
<tr>
<td>Average</td>
<td>187 msec</td>
<td>218 msec</td>
</tr>
</tbody>
</table>
ECGs derived for a control state and states of acute and chronic anterolateral wall ischemia. Scher's excitation-sequence diagrams were used to derive QRS vectors for the control tracing and for the tracing representative of acute ischemia. In the derivation of QRS vectors in the tracing representative of chronic ischemia, the effects of a destructive lesion in the anterolateral wall of the ventricle were taken into account. In the derivation of T waves for the control tracing, Van Dam and Durrer's data on intramural ventricular refractory periods were used in assigning action potential duration. In the derivation of T waves representative of acute ischemia action potential duration was shortened in the ischemic area by an amount corresponding to the FRP shortening measured in the acute experimental animals, and in the derivation of T waves representative of chronic ischemia action potentials were prolonged in the ischemic area by an amount corresponding to the FRP prolongation measured in the chronic experimental animals. In the control tracing the derived T waves are inverted in the X and Z leads and isoelectric in the Y lead. In the tracing representative of acute ischemia the T waves are increased in amplitude in the X lead and are more deeply inverted in the Z lead. In the tracing representative of chronic ischemia the T waves are more deeply inverted in the X lead than in the control tracing and are upright in the Z lead. The configuration of the derived T waves qualitatively corresponds to the T-wave abnormalities seen in patients with myocardial infarction.

Figure 6

Configuration of derived T waves qualitatively corresponds to the T-wave abnormalities seen in patients with acute myocardial infarction. Examples of tracings taken on patients with these abnormalities were shown in figure 1.

Since QRS configuration is often normal early in the course of infarction, a normal sequence of activation was assumed in the derivation of T waves representative of acute ischemia. In the derivation of T waves representative of chronic ischemia, the effect of the lesion on activation wavefronts was taken into account. Therefore, with chronic ischemia the configuration of derived T waves was influenced by both the change in activation sequence (secondary T-wave changes) and the change in recovery time (primary T-wave changes). To determine how much of the change in T-wave form was due to the change in activation sequence and how much was due to the change in recovery time, T waves were derived for a situation in which the activation sequence was altered by an anterolateral wall infarction and recovery times were normal. The tracing derived under these conditions is shown in figure 7, and for purposes of comparison the tracing derived with both abnormal activation and recovery sequences is also shown. Although T waves were inverted in the X lead in both tracings,

Figure 7

Derived ECGs showing the form of T waves with chronic ischemia. The configuration of T waves resulting from a change in the activation sequence (secondary T-wave changes) is shown on the left, and the configuration of T waves resulting from both the change in activation sequence and change in action potential duration (primary and secondary T-wave changes) is shown on the right of the figure. Although the T waves are inverted in the X lead in both tracings, the T inversion was greater when both the change in activation sequence and prolongation of recovery time were considered in the derivation.
the T inversion was greater when prolongation of recovery time was considered in the derivation.

Discussion

The physiological basis of the T wave has been more difficult to define than that of the QRS. Activation is accomplished quickly in individual cells and is propagated to neighboring cells. The occurrence of activation can be detected with electrograms from multiple sites to determine propagation sequence which can be used in explanation of the QRS complex. Recovery in individual cells follows activation and is not dependent on the occurrence of recovery in neighboring cells, that is, recovery is not propagated. Recovery is further unlike activation in requiring a much longer time for completion. These features of the recovery process mean its sequence cannot be mapped with the same techniques used to define propagation of excitation, and its relation to the T wave is more complex than that of activation to the QRS.

The theoretic model of the T wave provides a new approach to improved understanding of the physiological basis of repolarization. In this model the sequence of cardiac excitation was also taken as the sequence of onset of recovery. The sequence of completion of recovery was taken from measurements of refractory period duration and available information concerning action potential duration at various sites. The sequence with which intermediate stages of recovery occurred was defined using the shape of the downstroke of a diagrammatic ventricular transmembrane action potential and adjusting the length of the plateau to give the desired pattern of completion of recovery. With this model, patterns of potential difference in the heart at various stages of recovery can be related to the T wave of the body surface electrocardiogram in the same way patterns of potential difference during excitation can be related to the QRS complex. In previous studies the model has been used to derive T waves from the ECGs of dogs with normal sequences of activation and recovery, and has also been used to predict the form of experimentally induced T-wave abnormalities. In a quantitative test of the model, refractory periods were measured in ventricular areas that were warmed and cooled and, as predicted by the model, the observed changes in T area were proportional to the magnitude of refractory period changes. These findings suggested that the model was sufficiently valid to be helpful in explaining T-wave alterations associated with disease.

The present study was undertaken to analyze, in terms of the model, the serial T-wave abnormalities associated with myocardial infarction. To permit this application, an experimental study of the effects of acute and chronic ischemia on ventricular recovery time was carried out. It was found that during acute ischemia FRPs shortened. If action potential duration is shortened in a localized area, the area in which the shortening has occurred is relatively positive during repolarization with respect to its surrounding areas. The model predicts that repolarization vectors are directed from areas that are relatively negative to areas that are relatively positive, and if action potentials are shortened, the mean repolarization vector should move toward the area in which the shortening has occurred (fig. 2a). Therefore, during acute ischemia, the T vector should move toward the area of ischemia. For example, T waves would be expected to increase in amplitude in the anterior precordial leads with an acute anterior wall infarction, and with a diaphragmatic infarction the T waves would be expected to increase in amplitude in leads II, III, and aVF. Comparable changes in T-wave form were present in T waves derived for a state of acute ischemia. Since FRP shortening following coronary occlusion is relatively transient, these findings are not always seen in the clinical electrocardiogram. During chronic ischemia, FRPs measured in ischemic areas of the ventricle were longer than those in surrounding areas. If action potential duration is prolonged in a localized area, the area in which the prolongation has occurred
ANALYSIS OF T-WAVE ABNORMALITIES

is relatively negative during repolarization with respect to surrounding areas. The model predicts that repolarization vectors are directed from areas that are relatively negative to areas that are relatively positive, and the mean repolarization vector should be directed away from areas in which action potentials have been prolonged (fig. 2b). Therefore, during chronic ischemia, the T vector should move away from the area of ischemia, and T waves would be expected to be inverted in the anterior precordial leads with an anterior wall infarction, and inverted in II, III, and aVF with a diaphragmatic infarction. These findings are frequently seen in clinical electrocardiograms, and were present in the T waves derived for a state of chronic ischemia.

Any study of this type is limited by the lack of information available concerning ventricular repolarization. Van Dam and Durrer’s studies on intramural refractory periods of the normal dog ventricle have contributed the most detailed data available. Since very little information concerning the form of in vivo action potentials is available, a diagrammatic ventricular action potential adapted from Hoffman and associates11 was used to represent the form of action potential downstrokes, and FRPs used to infer action potential duration. Although refractory periods in these experiments were measured in some detail in ischemic areas, only a limited number of measurements were made in non-ischemic sites. Van Dam and Durrer’s data were used in assigning action potential duration in nonischemic areas. In addition, excitation sequence was not mapped in the experimental animals in this study. Scher’s diagrams were taken as representative of the excitation sequence of control states and states of acute ischemia. For states of chronic ischemia, the change in the form of activation fronts produced by a destructive lesion in the anterolateral wall of the left ventricle was considered in the derivation of QRS and T vectors.

In spite of limitations, the theoretic T-wave model has been useful in providing insights into the physiological mechanisms of serial T-wave abnormalities associated with myocardial infarction. With the model it has been possible to derive T waves that corresponded qualitatively to both the increase in T amplitude seen early in the course of infarction and the T-wave inversion seen later in the course of infarction. More exact predictions could be made if the sequence of depolarization and form of intramural action potentials in individual animals were known. The repolarization model could also be used in a study of ST-segment abnormalities if information were available concerning both action potential configuration and resting membrane potential. An approach to this problem has been reported by Samson and Scher.12 They recorded a limited number of in vivo action potentials from ischemic areas of the ventricle and related the change in the configuration of the action potential and change in resting potential to the configuration of the ST segment of the body surface ECG. Further studies in these areas are needed for a more complete understanding of the repolarization process and its relation to the ST-T deflection.

References

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Inscrutable Judgments

And if, whether in real life or even in chess, you begin to tamper with basic notions—continuity of space, divisibility of time and the like, you will soon reach a stage in which the symbols fail to function. Your thoughts become confused and paralysed. Consequently the fuller our knowledge of facts and of their connections the more difficult to conceive alternatives; the clearer and more exact the terms—or the categories—in which we conceive and describe the world, the more fixed our world structure, the less 'free' acts seem. To know these limits, both of imagination and, ultimately, of thought itself, is to come face to face with the 'inexorable' unifying pattern of the world; to realize our identity with it, to submit to it, is to find truth and peace. This is not... a yearning for mystical illumination or integration. It is scrupulously empirical, rational, tough-minded and realistic. But its emotional cause is a passionate desire for a monistic vision of life on the part of a fox bitterly intent upon seeing in the manner of a hedgehog.—Isaiah Berlin: The Hedgehog and the Fox. New York, A Mentor Book. New American Library of World Literature, Inc., 1957, p. 113.
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