Precordial and Epicardial Surface Potentials during Myocardial Ischemia in the Pig

A THEORETICAL AND EXPERIMENTAL ANALYSIS OF THE TQ AND ST SEGMENTS

By Roger P. Holland and Harold Brooks

ABSTRACT

The solid angle theorem was used to analyze the relationships between TQ and ST segment deflections recorded from precordial and epicardial locations and the time course, size, shape, and transmural location of the ischemic process in the ventricular myocardium. Mathematical predictions were compared with experimental data from the intact heart. Precordial electrograms obtained in anesthetized close-chest pigs were compared with epicardial electrograms recorded directly from the heart’s surface. Various areas of ischemia were produced by occluding large and small coronary artery branches, and the resultant changes in ischemic shape were delineated with Thioflavin S injections and postmortem ultraviolet photography. Formally derived equations and cumulative experimental data were in close agreement, suggesting that in the ischemic ventricle (1) TQ depression always accompanies ST elevation, (2) TQ and ST segment changes in magnitude and polarity are complex functions of ischemic size, shape, and transmural location; (3) precordial electrocardiogram (ECG) ST segment elevation is directly related to ischemic size; and (4) epicardial ECG ST segment elevation is inversely related to ischemic size. It is thus concluded that precordial and epicardial ECG TQ and ST segment deflections are complex functions of ischemic geometry and that their accurate interpretation with respect to ischemic size and shape and in the presence of pharmacological interventions is often difficult and may be misleading.

In 1920 Pardee (1) demonstrated that ST segment changes in the peripheral electrocardiogram (ECG) are indicative of myocardial injury in a patient with coronary heart disease. Later Wilson et al. (2) placed an electrode on the epicardial surface of the dog heart and confirmed that ST segment alterations occur with coronary artery ligation. More recently, the epicardial surface electrogram has had wide application in a variety of experimental situations designed both to confirm and to quantify regional myocardial ischemia (3). In all of these studies, the unproven assumption has been made that previous clinically and empirically derived relationships between alterations in the ST segment of the precordial electrogram and the presence, duration, size, and configuration of the ischemic process in the ventricular wall are equally applicable to interpretation of similar changes recorded from the epicardial surface of the heart.

The purpose of this investigation was to examine the precordial and the epicardial surface electrograms from both a theoretical and an experimental viewpoint. The magnitude of the ST segment deflection as a function of ischemic size was studied by solid angle analysis in the porcine heart. The pig was chosen because of similarities of gross coronary architecture (4) and collateral circulation (5) in this animal and man. Particular consideration was given to the spatial characteristics of the ischemic region—size, transmural shape and location, and distance from the recording electrode—and to how these factors influence the character of the electrogram and hence its proper interpretation. The model used in deriving the equations relating the recorded potential to the underlying ischemic potential gradient is presented first and is followed by the details of the experimental procedure used to test the theoretical findings.

Methods

MATHEMATICAL DERIVATIONS FROM THE SOLID ANGLE THEORY

The theoretical and mathematical formulations that follow are primarily based on a three-dimensional spherical model of the heart. In Figure 1, a small segment of the ventricular wall is diagramed illustrating the normal and the ischemic myocardial tissue with the boundary between them and their respective transmembrane poten-
transmembrane potentials of ischemia (broken curve) and normal (solid curve) tissue. Bottom Left: Electrocardiogram recorded by an electrode overlying the ischemic tissue. The TQ segment is located below the isoelectric line (broken) and the ST segment above. The total TQ-ST potential was measured from the beginning of the R wave to the plateau between the S and T waves. Top Right: Potential gradients existing at the boundary between normal and ischemic tissue during electrical diastole. Bottom Right: Potential gradients existing at the boundary between normal and ischemic tissue at mid-systole. Arrows indicate the direction of current flow (positive to negative) at the boundary.

In essence, the theory states that the potential, $V$, produced by a uniform potential gradient at any boundary in a conducting medium is equal to the product of the solid angle, $\Omega$, and the current density, $\Phi$. From the theoretical ischemic heart model illustrated in Figure 2, the potential $V$ recorded at either a precordial ($V_{pre}$) or an epicardial ($V_{epi}$) location can be expressed as:

$$V_{pre} = \Omega_{pre} \cdot \Phi = 2 \cdot \pi \cdot (\cos GE_B - \cos GE_a) \cdot \Phi,$$

$$V_{epi} = \Phi = 2 \cdot \pi \cdot (\cos GE_B - \cos GE_a) \cdot \Phi,$$

where $\Phi$ denotes the magnitude of the current density at that boundary, $\Omega_{pre}$ and $\Omega_{epi}$ are the solid angles constructed at the two locations, and $GP_a$, $GP_B$, $GE_a$, and $GE_B$ are defined as they are illustrated in Figure 2. The polarity of the recorded potential ($V_{pre}$ or $V_{epi}$) is positive or negative depending on whether an observer stationed at either location views first the positive or the negative side of the boundary separating the normal and the ischemic tissues.

We can now consider the specific application of the solid angle theory to electrode placement in either a precordial or an epicardial position. The current density as seen by each of these electrodes is diagramed in Figure 2. The charge boundary between the normal and the ischemic tissue during electrical systole is depicted by the negative and positive symbols, respectively. The ventricle has mean outer and inner radii of curvature equal to $R_o$ and $R_i$, respectively. Using this model, both ischemic size ($P$) and ischemic shape ($S$) can be characterized using angular notation. An ischemia with $S = 0$ radians is termed "transmural" with the extent of ischemic involvement evenly distributed throughout the thickness of the wall. Any ischemia bounded by an $S = 0$ radians is termed an "endocardial wedge" (example illustrated). Conversely, any ischemia with $S < 0$ radians is termed an "epicardial wedge" ischemia. An ischemia strictly localized to either the epicardial or the endocardial layer of the ventricular wall and not extending all the way through the wall at any point is appropriately termed an "epicardial" or an "endocardial" ischemia, respectively.
Since the solid angle $\Omega_{\text{pre}}$ (eqs. 1 and 2) is a function of angles $G_P$, and $G_B$ (eqs. 1 and 2), it is necessary to express these angles in terms of ischemic size, $P$, and ischemic shape, $S$. This requirement is easily accomplished with basic trigonometric identities (13) along with the ischemic heart model in Figure 2. For the actual computation of $\Omega$, we let $R_0 = 3.0$ cm and $R_i = 2.0$ cm with the epicardial electrode located $5.0$ cm above the epicardial electrode. For epicardial wedge, transmural, and endocardial wedge ischemias, we let $S = -0.523, 0.0,$ and $+0.523$ ($30^\circ$) radians, respectively.

**Experiments in the Intact Heart**

Experiments were performed in a total of 16 open- and closed-chest pigs weighing 30-45 kg. After induction of anesthesia with a small intravenous injection of thiopental, the pigs were anesthetized with an intravenous infusion of a warmed solution of alpha-chloralose (60 mg/kg). Supplementary doses of chloralose were given during the study to maintain a relatively uniform state of anesthesia. Respiration was maintained via a tracheostomy tube with a volume respirator (Harvard Apparatus Co.), regulated to maintain an arterial pH of 7.45 ± 0.05 throughout the experiment. The heart was exposed by a midsternal thoracotomy, and a pericardial cradle was created to support the exposed heart. The area of the left ventricular free wall supplied by branches of the left anterior descending coronary artery was selected for the study. Epicardial electrical potentials were measured by an atraumatic, firmly attaching electrode (14). The electrode was positioned over myocardium supplied by the particular arterial branches selected and fixed to the epicardial surface with a new tissue bonding agent, Eastman 910 (Eastman Chemical Division). This procedure permitted continuous monitoring of the epicardial electrograms with negligible base-line shift, which often is quite difficult to obtain with wick electrodes.

Silk ligatures (00) were placed around the selected artery at various distances from its origin, and reversible occlusion of any of these ligatures was achieved by tightening the silk snare with a polyethylene collar. Small areas of ischemia were produced by occluding the distal portion of either the first or the second major left ventricular branch of the anterior descending coronary artery. Large areas of ischemia were in turn produced with an occlusion of the anterior descending coronary artery just above the origin of the ventricular branch selected for the small ischemia, thus ensuring that the large area completely enclosed the small area. Recent studies have demonstrated fairly uniform myocardial distribution in the porcine heart with occlusion of a specific length of a coronary artery (15). With occlusion, an area of cyanotic discoloration was easily identifiable, and this area was drawn to scale on a previously sketched view of the porcine heart. With this method, the epicardial surface area involved in the ischemic process could be estimated by the area of cyanosis. The maximum TQ-ST segment deflection measured during each 2-minute occlusion could then be plotted against the estimated area of ischemic involvement.

A subset of experiments in five pigs was performed to assess the actual morphological changes in size, shape, and transmural distribution of the ischemic process within the ventricular myocardium with occlusion of various lengths of artery. In accordance with the method of Kloner et al. (16), 1 ml/kg of a filtered solution of 4% Thioflavin S in isotonic saline was injected intravenously 2 minutes after the coronary artery segment occlusion. This technique permits localization of an area of nonperfused myocardium, since with coronary artery occlusion this area remains motained. The pig was killed within 10 seconds after this injection, and the heart was rapidly extirpated and quickly frozen in a solution of isopentane and Dry Ice. It was then sliced in serial sections 5 mm or smaller from the apex to the base of the heart. The frozen sections were illuminated by a Leitz mercury vapor lamp through a Zeiss BG-12 excitation filter and photographed with Kodachrome II film using a yellow barrier filter at F/8 for 35 seconds.

Precordial electrograms were recorded by bringing the ligatures and collars outside the chest cavity, closing the incision, and recording from the left fifth intercostal space and the midclavicular line. A standard precordial electrode was used for recording the electrogram. The electrical signals were recorded with a Bioelectric amplifier (No. 8811A, Hewlett-Packard Co.) with a low- and a high-frequency response of 0.15 Hz and 300 Hz (-3db), respectively. This procedure allowed us to record accurately any base-line shifts of normally isoelectric segments of the electrogram during the course of ischemia; however, it did not permit us to analyze the component TQ and ST segment deflections but rather only the total TQ-ST deflection, the same parameter measured in the overwhelming majority of clinical and experimental ST segment studies. Separation of the total TQ-ST deflection into its individual TQ and ST segment deflections, however, has been accomplished by a number of investigators (8, 17) with the use of a direct-coupled amplifier. Total TQ-ST segment deflection in this study was measured from the base of the R wave to the flattest portion of the plateau between the S and T waves or between the R and T waves if no S wave was discernible (Fig. 1). All parameters were recorded on an eight-channel direct-writing ink recorder and on an FM magnetic tape recorder (Hewlett-Packard Co.).

**Results**

**Theoretical Findings**

Mathematical evaluation of a set of equations derived from the model (Fig. 2) and the solid angle theory (Eqs. 1 and 2) permitted the formulation of a series of theoretical predictions relating precordial and epicardial potentials to the size, shape, and time course of the underlying ischemic process.

**Application of Solid Angle Analysis.**—From Eqs. 1 and 2, $\Omega$ is seen to be a function only of the shape and the size of the ischemic involvement. However, $\Phi$ is a function of the potential gradient existing at the boundary between the two tissues. Since the potential gradient is believed to arise as a consequence of a decrease in the potassium (K+) gradient across the cells composing the ischemic zone (11), the longer the duration of the ischemic process, the greater the decrease in the K+ gradient and the larger the value of $\Phi$. A direct relationship between the transmembrane resting potential and...
the TQ segment deflection has been demonstrated (17).

Stating these points more concisely, we can make the following predictions. Prediction 1: $\Phi$ reflects the time course of the development of the ischemic process in its early, reversible stages. The longer the duration of the ischemic process, the greater $\Phi$ will be. Prediction 2: $\Omega$ reflects the size and the shape of the ischemic involvement. Prediction 3: Pharmacological or mechanical interventions that alter TQ and ST segment deflections may do so by altering $\Phi$, $\Omega$, or both.

The Precordial Electrode.—Figure 3 (left) provides the basis for certain predictions regarding the potential recorded from a precordial position. In this figure, $V_{pre}$ was calculated for a precordial location 5 cm above the epicardial surface. Since $V_{pre}$ is equal to the product of the solid angle $\Omega_{pre}$ and the charge density $\Phi$ (Eq. 1) and $\Phi$ is some increasing function of time (prediction 1), it was more convenient to plot $V_{pre}$ in terms of $2\pi \Phi$ than in millivolts. This procedure made the resultant curves independent of time. Prediction 4: In the presence of ischemia, the net TQ-ST potential recorded from a centrally located precordial electrode is generally positive, and hence the TQ segment is depressed and the ST segment elevated. The exceptions to this situation are an endocardial ischemia and a small endocardial wedge ischemia for which the net TQ-ST potential is negative. Prediction 5: With increasing size of ischemia, $P$, the potential recorded from the precordial electrode increases.

The Epicardial Electrode.—Figure 3 (right) provides the basis for additional predictions regarding potentials recorded from an epicardial position. In this figure, $V_{epi}$ was calculated for an epicardial location. One immediately notices the strong contrast between $V_{epi}$ and $V_{pre}$ as regards increasing ischemic size, $P$. More specifically the following further predictions can be made: Prediction 6: In the presence of ischemia, the net TQ-ST potential recorded from a centrally located epicardial electrode is positive. An exception to this situation is a purely endocardial ischemia for which the reverse is true. Prediction 7: The potential recorded from an epicardial location is approximately an order of magnitude greater than that recorded from a precordial location. Prediction 8: The magnitude and the sign of the potential recorded from either a precordial or an epicardial location is directly influenced by changes in the transmural shape of the ischemia. Prediction 9: With increasing size of ischemia, $P$, the potential recorded from the epicardial electrode decreases (the exception to this situation being the purely endocardial ischemia for which the potential increases to a peak as $l$ approaches 0.8 radians). This prediction is in
PRECORDIAL AND EPICARDIAL ELECTROGRAMS

TABLE 1
Maximum TQ-ST Segment Deflections (mV) Recorded from Precordial and Epicardial Locations during Occlusion of Small and Large Arterial Branches

<table>
<thead>
<tr>
<th></th>
<th>Small branch occlusion</th>
<th>Large branch occlusion</th>
<th>Large - Small</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vpre (5)</td>
<td>0.56 ± 0.023</td>
<td>0.78 ± 0.034</td>
<td>-0.22 ± 0.041</td>
<td>&lt; 0.015</td>
</tr>
<tr>
<td>Vepi (7)</td>
<td>7.94 ± 0.90</td>
<td>3.91 ± 0.54</td>
<td>-4.03 ± 0.48</td>
<td>&lt; 0.001</td>
</tr>
</tbody>
</table>

All values are means ± SE; the number of experiments is given in parentheses. P values were determined by a paired t-test (18).

In complete contrast to prediction 6 made for a precordial electrode for which one finds \( V_{pre} \) increasing as \( P \) increases.

EXPERIMENTAL FINDINGS

Cumulative data and paired t-test analysis (18) of occlusions are presented in Table 1. Comparisons of the potentials recorded from precordial and epicardial locations during small and large artery occlusions were carried out. Figures 4 and 5 show tracings from a representative experiment in the pig in which multiple alternate occlusions of large and small branches of the left anterior descending coronary artery were performed. The location of the ligatures is shown in the diagram along with the position of the epicardial electrodes. The area of ischemia could be readily identified by the rapid development of cyanotic discoloration of the involved myocardium.

In Figure 4, changes in the precordial electrogram at various times during the ischemia are shown. TQ-ST segment deflections from the initial zero base-line occurred as early as 10 seconds after occlusion and increased in magnitude progressively with the duration of ischemia. Also shown is a plot of the total TQ-ST segment deflection vs. time for ischemias involving large and small areas. The total TQ-ST deflection recorded from the large area of ischemic involvement (top tracings) at any point after the occlusion was significantly greater than the total deflection seen with the smaller area of involvement (bottom tracings).

In Figure 5, representative changes in the epicardial electrogram are shown. TQ-ST segment de-
Epicardial electrograms from large and small ischemias similar to those in Figure 4 with resultant plots of total TQ-ST segment deflection vs. time. Note that the total deflection recorded from the large ischemia (top tracings) at any point after the occlusion is less than the total deflection seen with the smaller (bottom tracings) ischemia.

Epicardial deflections from the initial zero baseline also occurred early and increased progressively with the duration of ischemia. Again a plot of the total TQ-ST deflection vs. time for large and small ischemic areas is shown. In this example, in contrast to the precordial electrograms of Figure 4, the total TQ-ST deflection recorded from the large area of ischemic involvement (top tracings) at any point after the occlusion was significantly less than the total deflection seen with the small area of involvement (bottom tracings).

As can also be seen from the analysis of cumulative data in Table 1, a significant difference existed between total TQ-ST segment deflections recorded from ischemias of different size. The larger ischemia consistently generated a greater TQ-ST segment deflection in the precordial electrogram. In distinct contrast, the larger ischemia generated a smaller deflection in the epicardial electrogram.

Evidence for the change in both size and shape of ischemia with occlusion at various lengths of arterial segments was obtained in five experiments in which Thioflavin S was injected. It became apparent that not only did occlusion of a longer length of artery increase the area and hence the size of ischemic involvement of the ventricular wall but it effectively and consistently altered the shape as well. We found a consistent pattern of transmural shape changes with occlusion of various lengths of artery. Occlusion of a distal portion of a small epicardial branch yielded an epicardial ischemia with a wedge shape but without an extension down to the endocardial surface. Occlusion of a larger artery increased the ischemic size still further, and the pattern of involvement resembled a transmural ischemia. Figure 6 shows ventricular segments of representative small and large myocardial ischemias from two experimental hearts. The area of ischemia in each section is detailed by the absence of fluorescence. The bottom photograph was obtained in a pig in which a short secondary left ventricular branch of the anterior descending coronary artery was occluded, thus depriving a relatively small area of the left myocardium of its blood supply. The area of nonperfusion is greatest in the outer layers of the ventricular wall tapering toward, but not extending to, the endocardial surface. In accordance with our terminology, this area is best termed an epicardial ischemia. In contrast, the top photograph was obtained in a pig in which the anterior descending coronary artery itself was occluded just distal to the origin of the first major left ventricular branch, thus depriving a relatively large area of the left ventricular myocardium of its blood supply. The area of nonperfusion is extensive, extending completely through the ventricular wall and involving the inner and outer layers to an equal degree. Despite the variety of occlusions produced in the porcine heart, neither an endocardial wedge nor a purely endocardial ischemia was observed.

Figure 7 shows the results from an experiment in Circulation Research, Vol. 37. October 1975
which a total of 15 occlusions involving various lengths of the left anterior descending coronary artery or its branches were made. The maximum TQ-ST segment deflection measured during each 2-minute occlusion was plotted against the estimated ischemic area. The figure also shows the best fit to this experimental data with the theoretical curve relating TQ-ST segment deflection to ischemic process. The theoretical curve was calculated for a transmural ischemia, and ischemic size, $P$, was converted into ischemic area. The experimental TQ-ST segment voltage varies as a function of ischemic area, not unlike that predicted from the solid angle analysis.

**Discussion**

Basic trigonometric and solid angle theorems were utilized to derive formally a series of equations relating the polarity and the magnitude of the TQ and ST segment deflections of the electrogram to the underlying ischemic process. On the basis of these equations, certain predictions were formulated which suggest that a strict interpretation of the total shift in TQ and ST segments recorded during ischemia can only be made after consideration of the duration, size, and transmural shape of the ischemic process. In addition, location of the recording electrode with respect to the ischemia was seen to bear critically on the interpretation.

Acute coronary artery occlusions experimentally induced in the porcine heart consistently yielded positive TQ-ST deflections when either the precordial or the epicardial electrode was situated over the ischemic segment of the left ventricle. This finding is in general agreement with predictions 4 and 6 for large and small ischemias, none of which could be described as purely endocardial. The existence of changes in the TQ segment which would correspond to any ST segment deflection was not demonstrable in these experiments because of the particular amplifier selected; however, such changes in the TQ segment have long been suspected from clinical observations (19, 20) and have been confirmed experimentally by Samson and Scher (8). With careful plotting of the TQ and ST segment deflections separately, it became evident to them that ST segment deflections usually can be seen earlier than those of the TQ segment.
and, although both TQ and ST segment shifts occur in concert, their mechanisms may be quite different. As suggested by Samson (9), the TQ segment is probably due to a decreased K⁺ gradient and the ST segment alteration appears to be related to an incomplete depolarization or an early repolarization of the ischemic tissue during electrical systole (7–10).

DURATION OF ISCHEMIA

From the experimental data presented in this paper the TQ-ST segment deflection rapidly increases in magnitude with the duration of the coronary artery occlusion (Figs. 4 and 5). This increase is probably brought about by progressive change in the magnitude of $\Phi$ which is due in turn to increasing K⁺ leakage out of the ischemic cell and a corresponding hyperkalemic condition in the ischemic tissue. Such a progressively hyperkalemic condition is also believed to be responsible for the biphasic alterations in ventricular activation time and R-wave voltage seen in the QRS complex following coronary artery occlusion (unpublished observation).

SIZE OF ISCHEMIA

The relationship of these recorded electrical potentials to the size and the shape of the ischemic process is considerably more complex. The potential recorded from a precordial position ($V_{\text{pre}}$) steadily increases in magnitude with increases in ischemic size, $P$. The particular transmural shape the ischemia assumes influences in turn both the rate at which the magnitude changes with $P$ and the polarity of the deflections (Fig. 3).

In distinct contrast, the potential recorded from an epicardial location ($V_{\text{epi}}$) steadily decreases in magnitude with increases in ischemic size, $P$. Ischemic shape in this case influences the rate of decline in magnitude with increases in $P$. Predictions 5, 7, and 8 along with the confirming experimental data in Table 1 and Figures 4, 5, and 7 serve to reemphasize the strikingly different nature of the TQ-ST segment potential changes recorded from either precordial or epicardial locations. If, on occasion, as has been our experience, the coronary artery is occluded high enough, no TQ-ST segment changes whatsoever will be recorded from a centrally located epicardial electrode despite obvious and extensive underlying myocardial injury; the boundary between normal and ischemic tissue is probably far removed from the electrode location and the corresponding solid angle it constructs is reduced to a near-zero value.

The data and curve in Figure 7 indicate that the relationship between experimental TQ-ST segment measurements and ischemic area can be closely approximated by the theoretical curve obtained from the solid angle analysis. The observed differences between the two can probably be attributed to either (1) deviations between the values of $R_o$ and $R_i$ used in the calculations and those found in the actual heart, (2) changes in ischemic shape which follow changes in ischemic size (see Fig. 6), or (3) differences in the rate at which the potential gradient $\Phi$ is established for a particular ischemic area.

ISCHEMIC SHAPE

From the theoretical curves presented in Figure 3 and corresponding prediction 8, it appears that the shape of the ischemic process may influence both the polarity and the magnitude of the TQ and ST segment deflections recorded from either precordial or epicardial locations. Currently it is difficult either to predict or to measure the shape of the ischemic process in the intact animal preparation. From the Thioflavin S injections, it became evident that the transmural shape of the ischemia changes with the size of ischemic involvement. In addition, as suggested by other studies in the dog, the shape of ischemic involvement may also be influenced by several hemodynamic factors such as the intraventricular pressure and the wall tension which may ultimately cause a redistribution of transmural heterogeneity of blood flow from endocardium to epicardium (21, 22).

Another factor which may influence the shape of ischemic involvement must also be considered. One would expect, purely on the basis of coronary anatomy, that an animal with extensive epicardial anastomoses, such as the dog (5), would be more prone to develop ischemias which, after our notation, would be termed endocardial or epicardial wedge and that other species, including the pig and man (5), with extensive endocardial anastomoses would be more prone to develop ischemias which are termed epicardial or epicardial wedge. Our studies with Thioflavin S indicate that the pig is indeed more likely to develop epicardial ischemia. An identical study by Kloner et al. (16) has shown a preference for endocardial ischemia in the dog. Which one of these three factors—size, intraventricular pressure, or coronary anastomotic anatomy—plays the predominant role in influencing ischemic shape awaits further studies, but it is reasonable to assume that changes in ischemic shape may have a significant effect on the TQ-ST segment deflections recorded from both precordial and epicardial locations as the theoretical data suggest.

Circulation Research, Vol. 37. October 1975
TQ-ST SEGMENT DEFLECTIONS AND PHARMACOLOGICAL INTERVENTIONS

Data from current investigations utilizing epicardial ST segment mapping techniques to evaluate the possible benefit of various interventions in limiting the extent of ischemic damage have generally been interpreted on the basis of three classical assumptions. First, the absence of a TQ-ST segment elevation indicates an absence of underlying ischemic injury. Second, an increase in the magnitude of the TQ-ST segment elevation indicates an increase in the extent of the underlying ischemic injury. Third, TQ-ST segment changes recorded from the epicardium are equivalent to those recorded from the precordium (23). However, in light of the theoretical and experimental evidence presented in the present paper one must interpret with caution the conclusions drawn from studies which have either made or accepted such assumptions. The possibility exists that administration of a given intervention will effect changes in the TQ-ST segment deflection in a variety of ways and directions (prediction 3) such that an ultimate decrease in the magnitude of the TQ-ST segment deflection will not necessarily indicate a decrease in the extent of the ischemic injury.

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