The potential gradient field created by epicardial defibrillation electrodes in dogs

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ABSTRACT Knowledge of the potential gradient field created by defibrillation electrodes is important for understanding and improvement of defibrillation. To obtain this knowledge by direct measurements, potentials were recorded from 60 epicardial, eight septal, and 36 right ventricular transmural electrodes in six open-chest dogs while 1 to 2 V shocks were given through defibrillation electrodes (1) on the right atrium and left ventricular apex (RA.V) and (2) on the right and left ventricles (RV.LV). The potential gradient field across the ventricles was calculated for these low voltages. Ventricular fibrillation was electrically induced, and ventricular activation patterns were recorded after delivering high-voltage shocks just below the defibrillation threshold. With the low-voltage shocks, the potential gradient field was very uneven, with the highest gradient near the epicardial defibrillation electrodes and the weakest gradient distant from the defibrillation electrodes for both RA.V and RV.LV combinations. The mean ratio of the highest to the lowest measured gradient over the entire ventricular epicardium was 19.4 ± 8.1 SD for the RA.V combination and 14.4 ± 3.4 for the RV.LV combination. For both defibrillation electrode combinations, the earliest sites of activation after unsuccessful shocks just below the defibrillation threshold were located in areas where the potential gradient was weak for the low-voltage shocks. We conclude that (1) there is a markedly uneven distribution of potential gradients for epicardial defibrillation electrodes with most of the voltage drop occurring near the electrodes, (2) the potential gradient field is significant because it determines where shocks fail to halt fibrillation, and (3) determination of the potential gradient field should lead to the development of improved electrode locations for defibrillation.


Knowledge of the shock field created in the heart by defibrillation electrodes is fundamental for understanding and improving defibrillation. It is thought that the cardiac potential gradient field created by the shock is an important factor in determining the cellular responses to defibrillation. Therefore measuring the potential gradient field created by shocks through the heart should help us understand the mechanism of defibrillation and improve its efficacy. Direct measurement of the potential gradient has been performed only with bipolar recording electrodes in a few sites in the heart. Thus the global distribution of the potential gradient and its relationship to postshock cardiac activation have not been defined. Consequently, the magnitude and distribution of the potential gradient created by different defibrillation electrode combinations and the relationship between the gradient field strength and the electrophysiologic responses of the myocardium to the defibrillation shocks remain unknown.

To determine the potential gradient created by different electrode combinations, we have constructed a computer-assisted mapping system that can directly record the extracellular cardiac potentials created by low-voltage shocks. The potential gradient field was calculated from these potentials and compared with the cardiac activation sequence immediately after defibrillation shocks given during electrically induced ventricular fibrillation for the same electrode combinations. The hypothesis was tested that for an unsuccessful shock just below the defibrillation threshold, the activation that leads to the resumption of fibrillation arises...
from a cardiac region where the potential gradient is weak.

Methods

Recording systems and electrodes. The study was done in two steps. The first step was to measure the potentials created by the different electrode combinations and to calculate the potential gradients. A 128-channel computer-assisted mapping system was used that can record cardiac potentials during low-voltage shocks. Unipolar recordings from 60 epicardial and 36 right ventricular anterior wall electrodes were recorded digitally with this system at a rate of 1000 samples/sec,6 with the reference electrode secured to the left leg. The second step was to map cardiac activation immediately after high-voltage shocks from bipolar recordings of 56 epicardial and four septal electrodes and to correlate these activation sequences with the potential gradient field determined for the low voltage shocks. Recordings during this step were made with a 64-channel mapping system that had been specially modified to record potentials soon after high-voltage shocks.5,7

Sock electrode array. The sock electrode array contained 60 button electrodes that were spaced evenly over the ventricular surface to record epicardial potentials. Upon completion of the potential measurements, four pairs of buttons on the apical part of the sock were disconnected so that these channels could be freed for septal recordings, leaving a sock with 56 recording electrodes for recording postshock activation.

Plunge electrode arrays. Two kinds of plunge electrodes were used. One kind, used to record right ventricular free wall potentials, was constructed from a 21-gauge needle. Each needle contained four bipolar electrodes with 0.5 mm between the poles of each bipolar and 1.5 mm between adjacent bipolar. Nine of these plunges were inserted perpendicularly into the right ventricular free wall. For unipolar recordings of the potential field, the more endocardial electrode of each bipolar pair was used.

A second kind of plunge, used to record from the interventricular septum, was 62 mm long and had two bipolar electrode pairs, 2.5 mm apart near the tip. The distance between the poles of each bipolar was 0.75 mm. The casing of the plunge was an 18-gauge intravenous catheter, with an anchor in the center that stabilized the electrode during the study and served as a marker for the electrode location during postmortem examination.9 Four of these plunges were used to make eight recordings of septal potentials. Two were used to make four-channel bipolar recordings of septal activations after defibrillation shocks.

Surgical preparation. Six mongrel dogs (mean body weight ± SD = 19.2 ± 2.9 kg, heart weight = 198 ± 33 g) were anesthetized with pentobarbital (30 to 35 mg/kg)10,11 and succinylcholine (1 mg/kg). Each was intubated with auffed endotracheal tube and ventilated with 30% to 60% oxygen through a Harvard respirator (Harvard Apparatus Co., Inc., S. Natick, MA). Ringer’s lactate was continuously infused and supplemented with potassium chloride, sodium bicarbonate, and calcium chloride when indicated. Via a separate intravenous line, pentobarbital was infused at a rate of approximately 0.05 mg/kg/min throughout the experiment to achieve adequate anesthesia. The dose of pentobarbital was adjusted according to the depth of anesthesia assessed by signs such as shivering, eyelid reflexes, and pedal reflexes.11 Succinylcholine at a bolus dose of 0.25 to 0.5 mg/kg was given no more than once per hour to decrease muscle contraction induced by the electric shocks.12 An arterial line was inserted into the femoral artery, and the systemic blood pressure was continuously displayed on an oscilloscope. Blood was withdrawn to determine the pH, PO2, PCO2, base excess, CO2 content, and the bicarbonate, sodium, potassium, and calcium concentrations. Normal metabolic status was maintained throughout the study by taking blood samples every 30 to 60 min and correcting any abnormal value.

The chest was opened through a median sternotomy, and the heart was suspended in a pericardial cradle. The sinus node was crushed by applying a hemostat at the junction of the superior vena cava and right atrium. For delivering defibrillation shocks, round mesh titanium patch electrodes, 4.5 cm2, were secured to the epicardium to form two different electrode combinations. One combination had the anode on the right atrium and the cathode on the left ventricular apex, to form the RA.V combination. The other had the anode on the right ventricular base and the cathode on the left ventricular base to form the RV.LV combination. A pair of pacing wires was inserted into the right ventricular outflow tract for ventricular pacing at a rate of 150 beats/min with 4 mA. A pair of sensing wires was inserted 1 cm below the pacing wires to sense ventricular activation. Shocks were given during paced rhythm at a predetermined interval after the last sensed depolarization to determine the combined impedance of the heart and defibrillation electrodes and to induce ventricular fibrillation by shocking during the vulnerable period.13

Recording potentials. Shocks of 1 to 2 V lasting 5 msec were given 300 msec after the last sensed R wave through the RA.V and RV.LV combinations, while the gain settings were automatically and individually adjusted for each channel of the 128-channel mapping system. The largest voltage that could be recorded without amplifier saturation was ±200 mV. For each electrode combination, the shock with the highest voltage that did not result in amplifier saturation was selected as the test voltage. The potentials recorded with this voltage were stored on tape for off-line analysis.14

Recording activations. After measurement of the potentials, 56 sock electrodes and four plunge septal electrodes were connected to the other mapping system to detect cardiac activation after the shock. The remaining recording electrodes were disconnected. Truncated exponential shocks of 5 msec duration were generated by a special device built by Intermedics, Inc. (Freeport, TX). To reduce amplifier saturation, the mapping system was disconnected during the shock.5,7 The time of onset of each shock recording varied with the shock strength and was 5 msec after the end of the shock for shocks less than 100 V and 20 to 25 msec for shocks of several hundred volts.

Before the defibrillation protocol was begun, shocks of 100 V were given during paced rhythm, 300 msec after the last sensed depolarization. The actual voltage and current delivered were displayed on an oscilloscope and the energy was calculated by their product multiplied by shock duration (0.005 sec). These shocks were used to optimize the amplifier gains and to determine the shock impedance. This impedance value was used to predict the voltage needed to deliver shocks of desired energy levels. To determine the pacing threshold and the area of epicardium directly depolarized by shocks of different strength,7 shocks of 1, 2, 6, 10, 15, 20, 25, 30, 40, 50, 60, 70, 80, 90, 100, 110, and 120 V were then given through the RA.V and RV.LV electrode combinations 300 msec after the last sensed R wave.

The protocol of Bourland et al.16 was followed for determination of the defibrillation threshold. Ventricular fibrillation was induced by giving shocks during the vulnerable period of ventricular paced rhythm15,16 via the RA.V or the RV.LV electrode combination. Defibrillation shocks were given 20 sec after the onset of fibrillation. If the shock was unsuccessful, defibrillation was achieved within 30 sec with a higher energy shock delivered through the RA.V combination. There was a 5 to 10 min interval between fibrillation episodes to avoid altering ventricular vulnerability and excitability18 or defibrillation thres-
old. The first shock was given with a predicted energy of 4 J for both combinations. Shock strength was then increased 20% during each successive episode of fibrillation until defibrillation was achieved. The energy level was then decreased 10% for each successive episode. The energy of the last successful shock was called the defibrillation threshold energy, and the highest energy that failed to defibrillate was called the subthreshold energy.

Data analysis. The dog was killed, and the locations of the epicardial recording electrodes were marked. The heart was then excised and weighed. The epicardial entrances of the right ventricular plunges were recorded. The septal plunge electrodes were removed, but the anchors were left in place to mark their location. After the electrode locations were recorded on a diagram of the ventricles, the heart was dissected to expose both sides of the interventricular septum. The locations of the anchors were recorded.

To analyze the potentials, the recording of one channel was displayed on a computer terminal (Tektronix 4014) and the time of the shock was determined. Based on known calibration signals, a computer program then calculated the potential recorded by each channel at the time of the shock. The difference between the maximal and minimal epicardial potential divided by the actual voltage applied to the defibrillation electrodes was calculated to find the fraction of the shock voltage dissipated across the ventricles.

The electrical gradient (mV/cm) on the epicardium was calculated from the spatial coordinates and voltages of the epicardial electrodes by the method given in the Appendix. The gradients obtained were divided by the actual voltage delivered across the defibrillation electrodes to obtain a normalized gradient per volt given. Isopotential and isogradient lines were drawn with the same method used for drawing the isochronal lines during cardiac activation. The mean gradients recorded by the electrodes on the anterior, basal, and left half of the epicardial surface were compared with that on the posterior, apical, and right half of the epicardial surface. Gradients for electrodes adjacent to the defibrillation electrodes were computed including the defibrillation electrode as one of the neighboring electrodes. Since the voltage drops across the electrode-tissue interface were not known, the potential at the defibrillation electrode was estimated from the potentials at the 60 epicardial recording electrodes by the method of Pilkington et al. with the addition of a least-square procedure because the set of equations was overdetermined.

The recordings from each channel of the 64-channel mapping system were displayed on a computer terminal (Tektronix 4014) for selection of the activation time. For shocks delivered 300 msec after the last sensed R wave, activations were analyzed immediately after the lowest voltage shock that captured the ventricles, after the shock that directly depolarized the whole epicardium, and after all shocks with strengths between these two values. For the unsuccessful defibrillation shock that was just below the defibrillation threshold, two activations before and five activations after the shock were analyzed. Isochronal maps were drawn for all activations selected. The early activation sites, defined as sites that activated earlier than the surrounding electrode sites, were determined for each isochronal map. The interval between the onset of the shock and the appearance of the first detected postshock cardiac activation was called the window width for that shock.

To compare the gradient field of the low-voltage shock with the origin of the postshock cardiac activation fronts after unsuccessful defibrillation shocks just below the defibrillation threshold, the epicardial gradient at the early site was identified in the gradient maps. If there was more than one early site, the mean of the gradients from all early sites was used. Nonpaired t tests, and correlation coefficients were used for statistical analysis of the results.

Results

Potential field created by different electrode combinations

Epicardial potential gradients. The potential field created by the RA.V combination was characterized by isopotential lines that were approximately circles perpendicular to and centered around the long axis of the heart (figure 1, A). The voltage drop was most marked at the apical portion of the ventricles near the ventricular defibrillation electrode. Thus the epicardial gradient in the basal half of the ventricles was smaller than that in the apical half (figure 1, B, table 1). The mean ratio of the highest to the lowest gradient measured was 19.4 ± 8.1. There were small nonsignificant differences between the gradients on the anterior and posterior halves of the epicardium and between gradients on the left and right halves of the epicardial surface (table 1). The mean percentage voltage drop across the ventricles was 17 ± 3% of the voltage applied to the RA.V electrodes.

Most isopotential lines for the potential field created by the RV.LV combination were semicircular, with the planes of the ellipses perpendicular to an imaginary line connecting the two defibrillation electrodes (figure 2, A). The isopotential lines were more closely spaced near the defibrillation electrodes. The highest gradients created by the RV.LV electrodes were at the base of the ventricles, near each epicardial defibrillation electrode (figure 2, B, table 1). The lowest gradients were along the anterior and posterior interventricular grooves and at the apex. The mean ratio of the highest to the lowest gradient measured was 14.4 ± 3.4. There were no differences between the gradients on the anterior and posterior epicardium and between gradients on the left and right half of the epicardial surface (table 1). The mean percentage voltage drop across the heart was 16 ± 3% of the voltage applied to the RV.LV electrodes.

Transmural and septal potential gradients. The mean epicardial gradient at the point of insertion of the right ventricular plunge electrodes and the mean transmural gradient along the plunge needles are shown in table 2. The transmural gradients were significantly less than the epicardial gradients. There was no correlation between the epicardial and transmural gradients at each plunge needle for the RV.LV electrode combinations. For RA.V combination, however, there was a significant but weak correlation (r = .64, p < .0001) between the epicardial and transmural gradients.

The gradient recorded by the septal plunges was 15
FIGURE 1. Example of the epicardial potential distribution created by the RA.V combination. This and all later figures are taken from the same animal. The maps are displayed as two complementary projections of the ventricles with the anterior left ventricular (LV) and right ventricular (RV) epicardium shown in the left diagram and the posterior left and right ventricular epicardium in the right diagram. The location of the apical defibrillation electrode is indicated by a minus sign (for cathode) within a cross-hatched circle. The plus sign within a circle indicates the location of the anodal defibrillation electrode in the right atrium. Numbers represent the locations of electrodes with satisfactory recordings and give the potential (A, mV) or epicardial gradient (B, mV/cm) for those locations. Closed circles indicate electrode sites where adequate recordings were not obtained. Asterisks indicate electrode sites for which no gradient was calculated because there were neighboring electrodes on only one side. The potentials and the gradients are normalized per volt of shock administered. The normalized isopotential lines are 25 mV/V apart, and the normalized isogradient lines are 25 mV/cm/V apart. A. When a 1 V shock was delivered, the voltage drop across the heart was 189 mV, 18.9% of the potential difference delivered to the electrodes. The isopotential lines were closer together at the apex than at the base of the heart, indicating a higher gradient at the apex as quantified in panel B. B. Gradient map of the same shock. The numbers represent the gradient (mV/cm per volt given). The higher gradient area was near the apex and the lower gradient area was at the base. There was a 102 mV/cm/V difference between the maximal and minimal gradients on the surface of the heart.

\[
\pm 26 \text{ mV/cm per volt given through the RA.V combination and } 59 \pm 78 \text{ mV/cm per volt given through the RV.LV combination.}
\]

Postshock cardiac activation. The potential gradient field created by the epicardial defibrillation electrodes predicted the site of several different electrophysiologic events induced by the shocks. Table 3 lists the shock strength required for these different electrophysiologic events. Examples of activations after shocks given during the TQ segment of paced rhythm are shown in figure 3 for the RA.V combination and in figure 4 for the RV.LV combination. At the pacing threshold, earliest activation occurred adjacent to the defibrillation electrodes where the potential gradient was high (figures 3, A, and 4, A). With stronger shocks, part of the epicardium was directly depolarized by the field of the shock (figures 3, B, and 4, B). An activation front arose at the border of the directly depolarized region and traversed the ventricles, depolarizing the remainder of the epicardium by conduction. As the shock energy increased, the percentage of the epicardial area that was directly depolarized by the shock field also increased (figures 3, C and D, and 4, C and D). With increasing shock strength, the increase in area directly depolarized corresponded roughly to the decrease in gradient across the epicardium (figures 1,
TABLE 2
Comparison of transmural and epicardial gradients (mean ± SD)

<table>
<thead>
<tr>
<th>Electrode configuration</th>
<th>Transmural gradient (mV/cm)(^a)</th>
<th>Epicardial gradient (mV/cm)(^a)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>RA.V</td>
<td>18 ± 22</td>
<td>35 ± 18</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>RV.LV</td>
<td>15 ± 25</td>
<td>34 ± 11</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

\(^a\)Per volt delivered across the defibrillation electrodes.

B, and 2, B). Thus the last regions to be directly depolarized as shock strength was increased (figures 3, D, and 4, D) were regions with low gradients.

During defibrillation, the first postshock activations after unsuccessful shocks just below the defibrillation threshold were located in regions of low gradient distant from the defibrillation electrodes (figures 5 and 6). In all dogs for both electrode combinations, earliest activation leading to the resumption of fibrillation after the high-voltage defibrillation shock was in a region of low potential gradient during the low-voltage shock (table 4). The mean window width between the onset of the shock and earliest recorded postshock activation was 69 ± 24 msec for the RA.V combination and 69 ± 25 msec for the RV.LV combination (p = 1.0). After a subthreshold defibrillation shock, the septum was never activated earlier than the ventricular epicardium for either electrode combination.

**Discussion**

Defibrillation is a life-saving procedure that is accomplished by administering a large electrical shock through electrodes on the thorax or directly on the heart. Although numerous studies of defibrillation have been performed, little is known about the distribution of the electric fields in the body produced by defibrillation shocks. Because this information is crucial for the improvement of defibrillation, participants at the Second Purdue Conference on Defibrillation concluded that development of a technique for measur-

**FIGURE 2.** Potential distribution created by the RV.LV combination of a 1.5 V shock. The potential and the gradients are normalized per volt of shock administered. The normalized isopotential lines are 25 mV/V apart, and the normalized isogradient lines are 25 mV/cm/V apart. A, The voltage drop between electrodes was 195 mV/V (19.5% of the voltage given). The normalized isopotential lines were closer together near the two defibrillation electrodes. B, Normalized isogradient map for the same shock. High gradient areas were located near the two defibrillation electrodes. There was a 75 mV/cm V difference between the maximal and minimal gradients on the surface of the heart.

**TABLE 3**
Shock strength required for different electrophysiologic events (mean ± SD)

<table>
<thead>
<tr>
<th>Electrophysiologic event</th>
<th>RA.V</th>
<th>RV.LV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pacing threshold (V)</td>
<td>3.5 ± 3.3</td>
<td>2.5 ± 0.5</td>
</tr>
<tr>
<td>100% depolarized (J)</td>
<td>0.063 ± 0.022</td>
<td>0.068 ± 0.036</td>
</tr>
<tr>
<td>Defibrillation threshold (J)</td>
<td>7.5 ± 4.2</td>
<td>14.1 ± 5.7(^a)</td>
</tr>
</tbody>
</table>

\(^a\)Includes only the four dogs that could be defibrillated with RV.LV combination.
Two studies have recorded potential gradients from a few bipolar electrodes in the body during transthoracic shocks. In one study, a long wire containing a single bipolar recording electrode was passed through the thorax of dogs. The wire was pulled through the thorax in small steps and recordings were made from the electrode at each step while 1 V shocks were given. The authors found that the potential gradient was highest in the subepicardial region of the left ventricular wall, where the gradients ranged from 50 to 140 mV/cm per volt of applied external potential difference. In the other study, recordings were made from five bipolar electrodes in the septum and right and left ventricular free walls of dogs while shocks of equal energy were given with defibrillation paddles of various sizes. Based on the measured potential gradients, the investigators concluded that a paddle diameter of 12 cm was best.

In this article we measured cardiac potentials created by low-voltage shocks administered via epicardial electrodes. From the potentials and the locations of the recording electrodes, we calculated the potential gradient over the ventricular epicardium and transmurally through the anterior right ventricular free wall. Although other variables such as the current density are also thought to determine whether or not a shock defibrillates the heart, Lepeschkin et al. presented evidence that the potential gradient is the more important variable. In addition, current density can be calculated from the potential gradient if cardiac impedance is known. Besides the principal role of the potential gradient field, ancillary factors undoubtedly also influence the effects of a defibrillation shock. For example,
The potential gradient field. The potential gradient field on the ventricular epicardium was extremely uneven for the defibrillation electrode combinations tested. The mean ratio of the highest to the lowest gradient measured was 19.4 for the RA.V combination and 14.4 for the RV.LV combination. Thus if a shock field is desired that has a uniform gradient throughout the ventricles, the defibrillation electrode combinations tested were far from ideal. The potential gradient fields created by transthoracic and transvenous shocks were not evaluated in this study although the same basic technique may be applicable. Investigation of transthoracic shocks may require closed-chest dogs with chronically implanted recording electrodes. Both transthoracic and transvenous shocks may have large transmural and septal gradients so that plunge electrodes will probably be required. The field generated by a transvenous catheter in the right side of the heart for the termination of ventricular tachycardia may be different from the RA.V combination because the catheter is surrounded by blood, which has a higher conductivity than cardiac tissue. Measurements are needed to determine whether this is the case.

Only a small percentage of the voltage applied to the defibrillation electrodes appeared across the major portion of the ventricles in which recording electrodes were located. For both epicardial defibrillation electrode combinations, only about 20% of the applied voltage was measured across the recording electrodes. This finding implies that the major portion of the applied voltage is being dissipated at the electrode-tissue interface and in the tissue immediately adjacent to the defibrillation electrodes. The percentage of the shock voltage appearing across the major portion of the ventricles may not be as small for high-voltage defibrillation as for the low-voltage shocks given in this study. We gave low-voltage shocks so that the potentials recorded from the heart would not exceed the range of our recording amplifiers (±200 mV). However, shock impedance of low-voltage shocks is greater than that
TABLE 4
Gradient at earliest activation site after subthreshold defibrillation shocks

<table>
<thead>
<tr>
<th>Dog No.</th>
<th>Gradient at the earliest site of activation after unsuccessful defibrillation shocks</th>
<th>Gradient at the earliest site of activation after successful defibrillation shocks</th>
</tr>
</thead>
<tbody>
<tr>
<td>RA.V combination</td>
<td>Minimum epicardial gradient during low-voltage shocks(^a)</td>
<td>Maximum epicardial gradient during low-voltage shocks(^a)</td>
</tr>
<tr>
<td>1</td>
<td>7</td>
<td>9</td>
</tr>
<tr>
<td>2</td>
<td>7</td>
<td>11</td>
</tr>
<tr>
<td>3</td>
<td>8</td>
<td>16</td>
</tr>
<tr>
<td>4</td>
<td>8</td>
<td>19</td>
</tr>
<tr>
<td>5</td>
<td>8</td>
<td>14</td>
</tr>
<tr>
<td>6</td>
<td>11</td>
<td>24</td>
</tr>
<tr>
<td>Mean</td>
<td>8 ± 2</td>
<td>15 ± 5</td>
</tr>
<tr>
<td>RV.LV combination</td>
<td>Minimum epicardial gradient during low-voltage shocks(^a)</td>
<td>Maximum epicardial gradient during low-voltage shocks(^a)</td>
</tr>
<tr>
<td>1</td>
<td>9</td>
<td>17</td>
</tr>
<tr>
<td>2</td>
<td>6</td>
<td>8</td>
</tr>
<tr>
<td>3</td>
<td>6</td>
<td>7</td>
</tr>
<tr>
<td>4</td>
<td>7</td>
<td>8</td>
</tr>
<tr>
<td>5</td>
<td>6</td>
<td>17</td>
</tr>
<tr>
<td>6</td>
<td>7</td>
<td>34</td>
</tr>
<tr>
<td>Mean</td>
<td>7 ± 1</td>
<td>15 ± 10</td>
</tr>
</tbody>
</table>

\(^a\)Gradients in mV/cm per volt given.

for high-voltage shocks.\(^{27}\) Thus cardiac potentials recorded during a 100 V shock may not be exactly 100 times that recorded for a 1 V shock. If the decrease in impedance with increase in shock voltage occurs primarily at the electrode-tissue interface or in the immediately adjacent tissue, then a larger fraction of the applied potential may appear across the ventricles with higher-voltage shocks. Thus the mapping method can be improved by recording the actual high-voltage field created during a defibrillation shock.

The mean septal gradient for the RV.LV combination was greater than the mean epicardial and transmural gradients. This is probably explained by the facts that the defibrillation electrodes were near the upper part of the septum and the gradient was principally across the septum for this electrode configuration. Although the epicardial gradient was a valid determinant of the region from which ventricular fibrillation resumed in the postshock period, more detailed studies of transmural and septal gradients will be necessary to understand the overall gradient field distribution of defibrillation electrode combinations.

**Relation of postshock activation and the potential gradient field.** The epicardial potential gradient field for low-voltage shocks delivered through epicardial defibrillation electrodes was strongly related to the site of capture at the pacing threshold, the region directly depolarized by shocks greater than the pacing threshold, and the site of earliest activation leading to the resumption of fibrillation after a defibrillation shock that was just below the defibrillation threshold. The strong relationship between the potential gradient field and the location of these electrophysiologic events provides evidence that (1) knowledge of the potential gradient field is important in the understanding of defibrillation and pacing and (2) the potential gradient field for low-voltage shocks is related to that for high-voltage shocks.

When a small shock was given that was of just sufficient strength to pace the ventricles, i.e., the stimulation threshold, activation originated adjacent to the epicardial defibrillation electrodes, a region of high voltage gradient. Activation fronts arising from this region then spread across the ventricles, depolarizing the remainder of the ventricles by conduction.\(^7\) As shock strength was increased above the pacing threshold, a larger region around the epicardial defibrillation electrodes was exposed to a potential gradient great enough to depolarize the cells. At the border of the region of cells that were directly depolarized by the field of the shock, activation fronts arose that depolarized...
of the ventricular muscle by conduction. Finally, a shock strength was reached that was strong enough throughout the heart to directly depolarize all of the myocardial fibers. The last portion of the ventricles to be directly depolarized as shock strength was increased was at the base of the ventricles for the RA.V combination and at the apex for the RV.LV combination. These are the regions of lowest potential gradient determined for the low-voltage shocks.

For shocks delivered during fibrillation with strength just below the defibrillation threshold, the region from which fibrillation resumed after the shock was also a function of the potential gradient field; the region was always a low-gradient region during the low-voltage shock. This finding confirms the hypothesis that for an unsuccessful shock just below the defibrillation threshold, the activation that leads to the resumption of fibrillation arises from a cardiac region where the potential gradient is weak. This finding is consonant with two different hypotheses of the mechanism of defibrillation: (1) the critical mass hypothesis and (2) the upper limit of vulnerability hypothesis. The critical mass hypothesis states that the defibrillation shock must annihilate all activation fronts within a critical mass of myocardium to halt fibrillation. According to this hypothesis, a shock just below the defibrillation threshold fails because it does not depolarize sufficient tissue to halt activation fronts within a critical mass. The regions not depolarized would be expected to be those of low-potential gradient, as was found in this study. For shocks that just failed to halt fibrillation, the long interval between the shock and earliest recorded activation after the shock (for example, the 71 msec interval in figure 5 and the 83 msec interval in figure 6) is difficult to explain by the critical mass hypothesis. On the basis of this and other findings, we believe that all activation fronts are halted during fibrillation with shock energies considerably less than that required for defibrillation.5

The upper limit of vulnerability hypothesis states that higher-energy shocks halt fibrillation and then reinitiate it by giving rise to activation fronts in myocardial regions that are stimulated by the shock during their vulnerable period.27,29 Thus defibrillation requires a potential gradient field throughout the ventricles that is both (1) above the level required to depolarize and (2) outside the range that induces fibrillation during the vulnerable period.27,29 According to this hypothesis, a shock with a markedly uneven field that is just below the defibrillation threshold fails because the weakest gradient in the region is slightly below the upper limit of the range of potential gradients that produce fibrillation during the vulnerable period. Thus activation leading to the resumption of fibrillation after the shock should arise from a region of low gradient, as was found in this study.

There is evidence that the defibrillation threshold is not a discrete value but is better represented by the probability of success at each energy level over a range of energies.30,31 There are several possible reasons for this observation. One is that the gradient field created by the defibrillation electrodes may change slightly from shock to shock, so that the field is sometimes weaker than is needed to defibrillate. Another possibility is that the state of tissue at the time of the shock changes slightly from shock to shock. A combination of these two factors may occur. Further investigation of these and other factors will be necessary to understand the mechanism of defibrillation.

Conclusions. These results indicate that it is possible to measure the potential distribution created by defibrillation electrodes and that the potential gradient field calculated from these potentials is important in determining the response of the myocardium to shocks delivered via the defibrillation electrodes. The results also indicate that the potential gradient field for epicardial defibrillation electrodes, as are used in the automatic implantable defibrillator,22 is markedly uneven. Such an uneven field is inefficient because it requires the field in most parts of the ventricles to be much

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**FIGURE 6.** Isochronal map of the first postshock activation of a 23.2 J unsuccessful defibrillation shock given via the RV.LV combination during fibrillation. The early sites of activation were located at the posterior and apical aspects of the ventricles (arrow) and appeared 83 msec after the shock. The base was the last region to be depolarized.
stronger than necessary for defibrillation to raise the field above the defibrillation threshold in the region where the field is weakest. Such an excessively high field in most regions of the heart not only increases the power requirement and battery drain of the defibrillator but also can damage the myocardium. By directly measuring the cardiac potential distribution created by many different defibrillation electrodes, it should be possible to determine a combination of defibrillation electrodes that creates a potential gradient field that is much more even than those investigated in this report.

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Appendix

The spatial coordinates of the epicardial electrodes were used to triangulate the heart surface and thus determine each electrode’s nearest neighbors. The gradient, \( \mathbf{E} \), was found by minimizing the function, \( F \), that incorporates the distance (d) between an observer electrode and its neighbors and the voltage (v) difference between the observer electrode and its neighbors. The function F is given in equation 1:

\[
F = \sum_{i=1}^{n} \left( \frac{v_i - v_o}{d_{i-o}} - \frac{E_x(x_i - x_o)}{d_{i-o}} + \frac{E_y(y_i - y_o)}{d_{i-o}} + \frac{E_z(z_i - z_o)}{d_{i-o}} \right)^{2}
\]

The symbols, x, y, and z represent the cartesian coordinates. The subscript o represents the observer, and the subscript i represents the i th neighbor. As each electrode in turn became an observer, the function F was minimized by partially differentiating F with respect to \( E_x \), \( E_y \), and \( E_z \) and setting each partial derivative equal to zero (equations 2 to 4).

\[
\frac{\partial F}{\partial E_x} = 0
\]

\[
\frac{\partial F}{\partial E_y} = 0
\]

\[
\frac{\partial F}{\partial E_z} = 0
\]

At this point in the procedure, three equations (equations 2 to 4) and three unknowns (\( E_x \), \( E_y \), and \( E_z \)) existed. \( E_x \), \( E_y \), and \( E_z \) were found by simultaneously solving equations 2 to 4. The magnitude of the gradient was obtained from equation 5:

\[
E_T = \sqrt{E_x^2 + E_y^2 + E_z^2}
\]

The gradients at the uppermost row of the electrodes were not calculated because of the absence of neighboring electrodes above them.

The epicardial gradient at the site of plunge insertion and the transmural potential gradient at the plunge were compared. The distance between the epicardial insertion of the right ventricular plunge and its three neighboring sock electrodes and the gradient of those neighbors were used to calculate the epicardial gradient at the site of plunge insertion (\( G_o \)):

\[
G_o = \frac{G_1}{d_{1-o}} + \frac{G_2}{d_{2-o}} + \frac{G_3}{d_{3-o}} \left( \frac{1}{d_{1-o}} + \frac{1}{d_{2-o}} + \frac{1}{d_{3-o}} \right)
\]

\( G_1 \), \( G_2 \), and \( G_3 \) represent the tangential epicardial gradient at the three neighboring sock electrodes. The transmural gradient was obtained by subtracting the endocardial potential from the epicardial potential and dividing by the distance between the two electrodes along the plunge needle (0.45 cm). The septal gradient was obtained by calculating the absolute difference between the potential recorded by the two electrodes on the plunge needle and then dividing by the distance between the two (0.25 cm).

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