Ventricular Refractory Period Extension Caused by Defibrillation Shocks

Robert J. Sweeney, PhD, Robert M. Gill, BS, Mitchell I. Steinberg, PhD, and Philip R. Reid, MD

In pentobarbital-anesthetized dogs, transcardiac shocks of up to 30 J or pacing stimuli were delivered to myocardial tissue at different times in the electrical cycle. When delivered midway or later into electrical systole, shocks, but not pacing stimuli, greatly extended the refractory period as determined by left ventricular pacing. There was a positive correlation between both the shock energy and timing and the amount of delay. A 30-J shock given 10 msec before the end of the refractory period extended the refractory period by 63±15 msec (p<0.001), whereas the same shock given 40 msec earlier produced only 25±10 msec (p<0.001) of extension. By comparison, a 5-J shock given at those times produced 36±18 (p<0.005) and 10±8 msec (p<0.01) of extension, respectively. When delivered early into electrical systole, both a pacing stimulus and a shock had no substantial effect on the tissue refractory period. Because the tissue that is late in electrical systole would otherwise be the first to repolarize if no shock were given, the selective refractory period extension may create a period after the shock during which no tissue is repolarized to a level sufficient for wavefront propagation. Thus, the energy- and time-dependent refractory period extension may help explain the mechanism by which ventricular defibrillation occurs during transcardiac shocks. (Circulation 1990;82:965-972)

According to the early concepts of the basic mechanism of electrical defibrillation, a shock was believed to directly depolarize all the cells in the heart.1-2 Accordingly, after a shock, no repolarized tissue remained to support wavefront propagation. In the mid-1970s, defibrillation was recognized as a more complex process that could be successful even though depolarization wavefronts were not terminated in every part of the myocardium.3-5 In keeping with this concept, the same shock was believed to produce different intensities throughout the heart, and if the shock intensity was sufficiently large in a "critical mass" of tissue, then the defibrillation might still be successful.

The view that the termination of fibrillatory wavefronts in a local region depended on the local shock intensity led to the development of better defibrillation methods.6-9 but this view contributed little to the basic understanding of the mechanism involved. Recently, investigators10-15 have reexamined the early concept that depolarization wavefronts are terminated because the shock directly depolarizes the tissue. One finding16 is that in relatively refractory tissue at local shock intensities less than about 5 V/cm new depolarization wavefronts are created, whereas at higher intensities, they are not. Thus, in addition to the local intensity of the shock, the success at terminating a wavefront may also depend on the relation of the shock to the timing of the tissue in its electrical cycle.

From our present understanding, a fibrillatory depolarization wavefront is believed to activate tissues at different times as it moves rapidly along a fibrillatory pathway, and the tissues along these pathways are believed to be at different positions within their electrical cycles. Unlike a pacing pulse, a transcardiac shock is experienced by all the tissues along the pathway at the same time. Thus, different tissues along the pathway experience a shock at different times within their electrical cycles. We believed that a better insight into the basic mechanism by which wavefronts are terminated might be found by examining the electrophysiological effect of a shock delivered at different times during the tissue's electrical cycle. The specific objectives of this study were to characterize the effect of a shock on the ventricular refractory period during pacing at various rates and
to characterize the effect on refractory period when a transcardiac shock of various energy was delivered at different times during the electrical cycle.

Methods

The study was divided into three sections, each with a separate group of animals. In group 1 (average weight, 19.3 kg; n=4), we measured the influence of 1-10-J shocks on the ventricular tissue refractory period at selected pacing rates. In group 2 (average weight, 15.3 kg; n=4) and group 3 (average weight, 19.4 kg; n=7), we measured the influences of 1-30-J shocks and shock timing on the tissue refractory period while pacing at a constant rate. The experimental preparation was similar for all groups. These experiments conformed to the guiding principles of the American Physiological Society.

Experimental Preparation

Healthy, adult mongrel dogs were anesthetized with sodium pentobarbital (30 mg/kg i.v.) and ventilated through a cuffed endotracheal tube with room air by a Harvard respirator (Harvard Apparatus, South Natick, Mass.). Blood gases were monitored (1304, IL, Instrumentation Laboratory, Lexington, Mass.), and tidal volume and respiratory rate were adjusted as needed. Fluid and electrolyte adjustments were made through a femoral vein cannula, and arterial pressure was recorded from a femoral artery cannula. Body temperature was maintained with a water-circulating pad. An electrocardiogram (lead II) was obtained with platinum needle electrodes.

Truncated exponential transcardiac shocks were delivered with a 12-cm² titanium spring electrode (C-10, CPI, Cardiac Pacemaker Inc., St. Paul, Minn.) as the anode and positioned in the right atrium through the left jugular vein. Through a left thoracotomy a 12-cm² patch electrode (A-67, CPI) was used as cathode and positioned around the left ventricular apex. Both electrodes were connected to a modified external cardioverter-defibrillator (ECD energy accuracy approximately 7%, CPI). Modifications to the defibrillator allowed for a continuously adjustable leading-edge voltage and remote triggering.

A bipolar, platinum pacing electrode (0.4 cm spacing between poles, 0.45 cm deep) was sutured to the midanterior left ventricle and used for introduction of 13 training pacing pulses (S₁) and the premature pacing pulse (S₂). Pacing stimulation was performed with an adjustable stimulator with an isolated current source (Caltronics, Inc., Indianapolis, Ind.). The pacing pulses were rectangular pulses 2 msec in duration set to either two or four times the diastolic threshold current, which was approximately 0.2 mA.

The pacing electrode was surrounded by eight insulated, stainless steel, unipolar plunge electrodes that were implanted 0.4–0.5 cm deep. The recording electrodes formed two rings, 0.5 and 2.0 cm away from the center of the pacing electrode. A stainless steel, unipolar reference electrode (5.5 cm×4 cm) was positioned subcutaneously below the thoracotomy incision. Electrograms from the eight recording electrodes were amplified (×200) and filtered between 100 Hz and 10 kHz. Because of the high-pass filtering, the amplifiers recovered 10 to 20 msec after the shock. Lead II electrocardiogram, blood pressure, and all electrograms were recorded on paper (ES-1000, Gould Inc, Cleveland, Ohio) and on FM tape.

The plunge electrodes were used to record local electrograms to determine whether a propagated response occurred because of the S₂ stimulus. The timing of the S₂ stimulus was changed to determine the refractory period as shown in Figure 1. The recordings from the four electrodes closest to the pacing site are shown. Figure 1 shows the last S₁ stimulus artifact and its response, the shock, and the S₂ stimulus artifact. In Figure 1A, the S₂ stimulus did not elicit a response that propagated to the ring of electrodes. In Figure 1B, the S₂ stimulus was given 5 msec later, and it elicited a propagated response that indicated the tissue at the pacing site was no longer refractory.

Group I

In group 1, the influence of various energy shocks on ventricular refractory period was examined at different pacing rates. As shown in Figure 2A, the refractory period was measured with (Shock) and without (Control) a transcardiac shock delivered simultaneously with the last S₁ (LS₁) pacing stimulus.

Before each experimental run, the pacing threshold was remeasured at a ventricular pacing rate of 200 beats/min to verify its stability. The first half of each run was a control, consisting of refractory period measurements at pacing rates of 171, 200, 240, and 300 beats/min in random order by the method described below. The second half of each run consisted of measuring the refractory periods at these same pacing rates (again, in random order) but with a transcardiac shock delivered simultaneously with the LS₁. The shock energy was constant for all shocks within a run, and the runs were repeated with increasing shock energies of 1, 2, 5, and 10 J with a 15-minute interval between runs.

Because a transcardiac shock was delivered with each LS₁ (except in the controls), a method to measure the refractory period while minimizing the number of shocks was developed. Thirteen S₁ training pulses were delivered and followed by an S₂ pulse. Initially, the S₂ pulse was delivered 130 msec after the LS₁, and the electrograms from each of the eight recording electrodes were examined to determine whether the premature beat elicited a propagated response. If no response was elicited, the S₂ was moved in 5-msec steps until a propagated response was elicited. There was a 60-second delay between series of training pulses. For 10-J shocks, a delay of 120 seconds was used. The measure of refractoriness was defined as the longest LS₁-S₂ interval that did not elicit a propagated response. If ventricular fibrillation was induced in the animal, a rescue shock (10 or 15 J) was given, and an additional 4 minutes was allowed for recovery.
**A LS1 to S2 is 216 ms**

![Filtered unipolar electrograms from recording electrodes surrounding the measurement site. Panel A: A pacing artifact and evoked response are visible for the last S1 (LS1) and the previous S1 pacing stimulus. The tissue is paced with a basic cycle length of 300 msec and has a refractory period of 156 msec. At the arrow, a 15-J transcardiac shock is introduced 126 msec after the LS1 (30 msec before the end of the refractory period). The S2 stimulus is introduced 216 msec after the LS1. The S2 pacing artifact is visible in electrogamms 3 and 4, but no response is evoked. Panel B: S2 stimulus is introduced 5 msec later (221 msec) and an evoked response indicates that the tissue at the pacing site is no longer refractory.](image)

**Group 2**

In group 2, the influence of the shock on the refractory period was examined as a function of the tissue's relative position within its electrical cycle at the instant the shock was administered. As shown in Figure 2B, either a shock (Shock) or a pacing stimulus (Control) was delivered at various times relative to the LS1 stimulus. The pacing rate was kept as close to 200 beats/min as possible for all group 2 measurements.

In the first half of each group 2 run, a control pacing stimulus was introduced from the pacing electrode at the following times with respect to the LS1 in the order shown: 0, 25, 50, 75, 100, 125, -25, -50, -75, -100, and -125 msec. One measurement of refractory period after the LS1 was obtained for a control stimulus at each of these times. A negative timing number indicates that the control stimulus was delivered before the LS1 pacing pulse, and a positive timing number indicates that it was delivered after the LS1.

When a control stimulus or shock was introduced before the LS1, it might have depolarized the tissue at the pacing site before the LS1 stimulus could do so. This made the term "refractory period" somewhat ambiguous. The phrase “refractory period after the LS1” was used to indicate that the measurement always started at the LS1 stimulus and ended at the S2 stimulus that had failed to evoke a response regardless of when the control or shock stimuli were introduced.

**FIGURE 2.** Panel A: Stimulus sequence for group I. Control: a series of S1 training stimuli is followed by an S2 stimulus then repeated after a 60-second delay. S1 is adjusted to obtain the refractory period (RP) after the last S1 (LS1). Shock: a series similar to control except a transcardiac shock is delivered simultaneously with the LS1. Panel B: Stimulus sequence for groups 2 and 3. Transcardiac shock (Shock) or control pacing stimulus (Control) is delivered at various times relative to the LS1.
In the second half of each group 2 run, the measurements were repeated with the transcardiac shocks instead of the control pacing stimuli. Group 2 runs were performed with shock energies of 1, 5, and 10 J in ascending order. Fifteen minutes was allowed for the animal to recover between runs. In group 2, the refractory periods were measured as in group 1 except that the S1-S2 did not always start at 130 msec but rather at a value that was closer to the expected refractory period. This modification allowed for fewer shocks to the animal.

**Group 3**

In group 3, the influence of shock strength and timing was examined. Shocks of 1–30 J were delivered 10–70 msec before the end of the tissue refractory period. The pacing rate was kept as close as possible to 200 beats/min for all group 3 measurements. The method to measure refractory period during the run was the same as in group 2.

To provide a reference time for delivery of the shocks or the control stimuli, an additional measurement of the refractory period was made at the beginning of each group 3 run. This reference refractory period was measured with a series of 13 training beats and a 2-second delay between series, and the S1 was adjusted to within 1 msec. During that run, the shocks or control stimuli were then timed to occur at fixed times before the end of that reference refractory period.

Each group 3 run used a single timing of 10, 30, 50, or 70 msec before the end of the reference refractory period. The runs were made in random order. Within each run, the measurements were made in random order with either the control stimulus or a 1-, 5-, 10-, 15-, 20-, or 30-J transcardiac shock. Fifteen minutes was allowed for recovery of the animal between group 3 runs.

**Statistical Analysis**

All hypothesis testing was accomplished by two-sided paired t tests for a significant difference from 0 at the 0.05 level. Data are recorded as mean±SD.

**Results**

**Group 1**

The results of group 1 are shown in Figure 3. In Figure 3, the refractory periods were plotted against the pacing rate. The relation between ventricular tissue refractory period and pacing rate was essentially unchanged by the shocks because they only produced a small, parallel, downward shift of the curve from control. When averaged across all animals, pacing rates, and shock energies, the paired comparisons of refractory periods with and without shocks delivered simultaneously with the LS showed that the shocks produced only a small (4%), but significant (p<0.001), reduction in the refractory periods. For each shock energy, the reductions were averaged across all animals and pacing rates.

**Group 2**

Representative results for the group 2 animals are shown in Figure 4, which contains data for 10-J shocks at a pacing rate of 234±23 beats/min. The average pacing rate for group 2 was 230±25 beats/min, and the average refractory period was 130±5 msec. The refractory period after the LS2 was plotted against the time (relative to the LS2) that the 10-J transcardiac shock was introduced. The curves for the pacing stimulus (Control) and the shock are nearly identical at times less than 75 msec. Similar results were obtained for the other shock energies. Typically, measurements were not possible at -125 msec because shock stimuli consistently produced ventricular fibrillation and because pacing stimuli had an inconsistent effect on the tissue so that the refractory period could not be measured. For shocks introduced 75 msec or more after the LS1, there was an increasing disparity between the curves. This
difference was significant only for shock energies of 5 and 10 J delivered later than 75 msec after the LS1.

**Group 3**

The results of the group 3 animals are shown in Figure 5. The average pacing rate for group 3 was 202±5 beats/min. The reference refractory periods obtained at the beginning of each run were subtracted from the refractory periods measured when either shocks or the control pacing stimuli were introduced. In this way, the change in the refractory period was obtained and plotted against the time of the shocks or control stimuli relative to the end of the reference refractory period.

Figure 5 shows several characteristics. First, the refractory periods either remained the same or increased when shocks were introduced, but they did not substantially change when the pacing stimuli were introduced. Second, the increase in refractory period (refractory period extension) due to the shocks depended strongly on when they were delivered. For shocks delivered 70 msec before the end of the reference refractory period, no substantial increase in the refractory period was observed even for the 30-J shocks. As the shocks were delivered closer to the end of the refractory period, the increased refractory periods became significant (about 45% for the 30-J shock). Because only the timing of the shock was altered, this influence must have been related to the timing of the tissue within its electrical cycle at the moment the shock was delivered.

With constant timing relative to the end of the refractory period, larger shocks were observed to produce a progressively larger increase in the refractory period. The increase varied monotonically with shock energy, but it appeared to approach a maximum for shock intensities in the range of 15–30 J.

In groups 2 and 3, the shocks sometimes produced fibrillation when they were delivered close to the end of the refractory period. For higher-energy shocks, this was rarely a problem. However, for lower-energy shocks, and particularly for the 1-J shocks, incidents of fibrillation occurred with a greater frequency that precluded some measurements at these energy levels.

**Discussion**

We can summarize the above findings as follows: 1) The shocks directly depolarized tissue that was nonrefractory when the shock was delivered, 2) the shocks had little effect on the relation between refractory period and pacing rate, 3) the shocks had little effect on tissue electrical state if that tissue was early in its electrical systole when the shock was delivered, 4) tissue near the end of electrical systole when the shock was delivered experienced a substantial refractory period extension that depended on the shock energy and the timing of the shock.

**Shocks Given Simultaneously With the Last S1**

Because the shocks occurred simultaneously with the LS1 pacing stimulus in group 1, the tissue at the pacing electrode was accommodated to the basic pacing rate and at the beginning of its electrical systole when the shock was delivered. The refractory period measured with shocks included was only 4% less than that measured without shocks both overall and for each individual pacing rate. Interestingly, these small reductions of the refractory period were about the same at all the shock energies tested. The reason for this reduction is unknown. Based on this information, we conclude that the refractory period adjustment to basic pacing rate is essentially preserved across the shock events.

**Shocks Given to Nonrefractory Tissue**

At times before the LS1, the tissue at the pacing site was expected to be repolarized because sufficient time had passed after the previous S1 stimulus. As typified by the sloping line on the left of Figure 4, one finding from group 2 was that the time interval from the LS1 to the minimum S2 was decreased when either a pacing stimulus or a transcardiac shock was given at times before the LS1. This decrease can be explained for the control pacing stimulus given before the LS1. If the control stimulus depolarized the tissue at the pacing site before the LS1, had the opportunity to do so, then the tissue refractory period would begin before the LS1 (at the time of the pacing stimulus) and end concomitantly earlier. Thus, as observed, the interval measured from the LS1 to the S2 would become smaller.

Transcardiac shocks had the same influence as the control pacing stimuli when either were delivered before the LS1. This indicated that the shocks, like the control pacing stimuli, acted to directly depolarize the tissue at the pacing site. This was not surprising because the intensity of the transcardiac shocks would be expected to depolarize the entire myocardium. It was surprising that the shocks had little other effect on the tissue's subsequent refractory

![Graph showing change in tissue refractory period](http://circ.ahajournals.org/DownloadedFrom)
period. Similar to the findings from group 1, the refractory periods were slightly reduced when the transcardiac shocks were introduced. Thus, we conclude that transcardiac shocks given to repolarized tissue act to depolarize that tissue without altering the subsequent refractory period.

**Shocks Given Early in the Refractory Period**

As typified by the flat portion in the center of Figure 4, another finding in the group 2 animals was that the refractory period after the last $S_2$ remained unchanged if either transcardiac shocks or the control pacing stimuli were given after the last $S_2$ by up to 50–75 msec. Under these conditions, the tissue at the pacing site was already depolarized by the $S_2$, and early in its electrical systole when the shocks or control pacing stimuli were delivered. Because the tissue had already been depolarized by the $S_2$, a control pacing stimulus given early in the refractory period was expected and observed to have little influence on the tissue refractory period.

It was surprising to find that the refractory period after the last $S_2$ also remained essentially unchanged when transcardiac shocks of 1, 5, and 10 J were given instead of the control pacing stimuli. This observation suggests that shocks given to tissue that was early in its refractory period had little influence on the time required for that tissue to become nonrefractory. Based on these observations, we conclude that shocks given to tissue that is early in its refractory period do not substantially affect the duration of that refractory period.

**Shocks Given Late in Refractory Period**

The most interesting finding from the group 2 animals was that the refractory period of the tissue was extended when shocks were given later than 50–75 msec after the $S_2$, as typified by the right-hand portion of Figure 4. The results in group 3 animals confirmed this finding in greater detail. Because this refractory period extension did not occur for the control pacing stimulus delivered with the same timing, it represented a difference due to the transcardiac shock. The refractory period extension was greater for higher-intensity (energy) transcardiac shocks and for shocks given later in the refractory period. Based on these observations, we conclude that shocks selectively extend the refractory period of the tissue depending on the shock’s intensity and also the timing relative to the end of the tissue refractory period. The effect of a shock on tissue may also be characterized by the local potential gradient created by the shock.7 Because transcardiac shocks produce different local potential gradients throughout the heart6 and because our data (e.g., Figure 5) show a relation between refractory period extension and shock energy, the locally measured increase in refractory period may vary according to the location on the heart. In this study, it was the shock energy to the entire heart, not the local potential gradient at the measurement site, that was controlled.

**Comparison of Shocks With Pacing Stimuli**

From these observations, it appears that a defibrillation shock acts on tissue very much like a pacing stimulus when the shock is delivered to tissue in electrical diastole and to tissue that is early in its electrical systole. This parallel is reasonable because both the pacing stimulus and the shock can produce a local current density in the tissue at the pacing electrode. A fundamental difference is that a pacing stimulus affects tissue near the pacing electrode, whereas the shock affects tissue throughout the myocardium.

The concept that a shock can modify the refractory period has been previously proposed with regard to the initiation of arrhythmias.15,16 Prolongation of action potentials due to strong stimulation has also been reported with transmembrane recordings17 and dyes that are sensitive to transmembrane voltage.18 An inhibition phenomenon with pacing stimulation has also been previously described.19–21 In pacing inhibition, a strong pacing stimulus is given well before the tissue’s absolute refractory period. Although the strong pacing stimulus does not elicit a propagated response, it increases the tissue refractory period so that a test ($S_2$) stimulus given after the absolute refractory period failed to elicit a propagated response that would otherwise occur. Although pacing inhibition has not been widely investigated, it also exhibits an intensity- and time-dependent nature20 in keeping with the observations made for shocks in the present study. Namely, both higher-intensity pulses and greater-delayed pulses produce a greater inhibition.

The refractory period extension attributed to the shock may be due to the cellular excitability phenomenon previously described.22–24 The “graded response” to stimulation involves both a graded local response that is seen at the stimulus site and a graded decremental conduction response away from that site. Because a defibrillation shock is experienced by all tissue at once, the conduction aspect of the graded response does not help to explain these observations. However, a graded local response may occur in all tissue simultaneously because of the shock. If the time required for the action potential to reach a particular state of repolarization is considered, the graded local response23 to a cellular stimulus is qualitatively similar to increased refractory periods due to shocks. The intensity dependence and timing dependence of the response are also qualitatively similar.

**Implications for the Mechanism of Defibrillation**

All these measurements were made during ventricular pacing, so their direct applicability in the case of a fibrillating heart is uncertain. In addition to low myocardial blood flow, a fibrillation event is characterized by a high cellular activation rate on the order of 400–600 depolarizations/min. This rate is much greater than those used in this study. However, if the
phenomena observed during ventricular pacing apply as well during fibrillation, a deeper insight is gained of both the process of defibrillation and the basic mechanism by which depolarization wavefronts may be terminated by the shock. The implications discussed below derive by extending the findings of this study to the case of fibrillation.

First, if during fibrillation, the tissue accommodation to pacing rate is preserved across the shock (i.e., group I results), then even after a successful shock, the tissue is accommodated to the fast stimulation rates associated with fibrillation. Second, if the refractory period of the tissue that is early (or midway) into its electrical systole when the shock occurs is not significantly altered by the shock, then that tissue should repolarize in the same approximate order that it would have if no shock had been introduced. Because the tissue was depolarized by the previous depolarization wave, it must repolarize along approximately the same pathway taken before the shock.

Considered together, these two implications indicate that even after a successful defibrillation there may be a brief period of time (e.g., within a time period of one refractory period) during which the accommodation to the fast activation rate and the cellular timing along fibrillatory pathways still exist. The myocardium in this condition may be expected to have a greater susceptibility to the reinitiation of fibrillation than it would have at other times. As the period of time without depolarization increases, this susceptibility would diminish because 1) the tissue along the fibrillatory pathways would repolarize, thus erasing the pathways, and 2) the tissue would begin to accommodate to a slower rate.

If depolarization activity was introduced immediately after a successful defibrillation or if the shock failed to terminate some depolarization activity, an interplay between reinitiation and recovery may result. In the tissue nearest the earliest activity, the depolarization may spread into the fibrillatory pathways, thus tending to reinitiate fibrillation. However, depolarization activity requires time to spread over the myocardium so that the more distant tissue would have more time to recover. When defibrillation shocks are near the defibrillation threshold, the interplay of these factors may produce the transient slowing and organization after unsuccessful shocks and the fast activations that sometimes follow successful shocks.

A third implication results from predicting the influence of a lower intensity shock on fibrillating tissue. As demonstrated in groups 2 and 3, a 1-J shock was sufficient to depolarize the repolarized tissue at the pacing site but was not sufficient to cause a measurable extension in the refractory period. This suggests that a low-intensity shock directly depolarizes the repolarized tissue just in front of an advancing depolarization wavefront and, thus, terminates that wavefront. However, it also suggests that the same shock creates a new depolarization wavefront at the boundary between tissue that is still refractory to the shock and the tissue that is not refractory to the shock. The chief reason for the creation of the new wavefront is that tissue is predicted to repolarize after the low-intensity shock with the same timing that it would if the shock had not been given. Just after the low-intensity shock, the following condition may arise at this boundary: On the repolarized side of this boundary, the tissue is directly depolarized by the low-intensity shock and, thus, at the beginning of its electrical systole. On the other side, the tissue continues to become repolarized because its refractory period is not extended by the low-intensity shock. Then, the tissue that has just repolarized on one side of this boundary is adjacent to the tissue that has just been depolarized on the other side. This proximity of newly depolarized tissue to newly repolarized tissue may lead to a new depolarization wavefront that essentially replaces the one that was terminated.

The most important implication is derived by applying the same reasoning for a high-intensity shock. In addition to directly depolarizing tissue that is repolarized, it is predicted that the high-intensity shock will also extend the refractory period of the tissue that is late in electrical systole when the shock is delivered. With a low-intensity shock, this tissue would have been the first to become repolarized after the shock and would also be the first to be depolarized by the new depolarization wavefront. Because the higher-intensity shock selectively extends the refractory period of that tissue, it may establish a zone of tissue in which the repolarization is delayed. This zone is always immediately in front of the newly depolarized tissue. The inability of the depolarization to propagate across these zones may be the mechanism responsible for terminating fibrillation. The spatial extent of these zones and the duration of increased refractoriness would both increase with increasing shock intensity. Such a mechanism of refractory period extension may account for the relatively high-shock intensities required for defibrillation relative to the intensities required to directly depolarize the tissue.

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