Location of Diastolic Potentials in Reentrant Circuits Causing Sustained Ventricular Tachycardia in the Infarcted Canine Heart

Relationship to Predicted Critical Ablation Sites

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Background—The complete reentrant circuit for ablation of reentrant ventricular tachycardia (VT) in humans can rarely be localized by mapping. As a result, surrogate markers, such as diastolic electrical activity, subsequently confirmed by entrainment, have been used. However, ablation at those sites has had variable efficacy. The reasons for this variability are not clear.

Methods and Results—We correlated activation maps of reentrant circuits in the epicardial border zone of 4-day old infarcted dog hearts with the corresponding ECGs for 45 VTs to determine the regions of the reentrant circuits activated during diastole. In VTs with a figure-8 reentrant pattern, the center point of the central common pathway, the part of the circuit critical for the maintenance of reentry, was activated in early diastole in 32 of 35 VTs (91.4%), in late diastole in 1 (2.9%), and in systole in 2 (5.7%). Regions outside the circuit were rarely activated in diastole. In 10 VTs, the reentrant circuit was characterized by a single reentrant loop. In these circuits, no one region was predicted to be critical for maintenance of reentry, and a segment of the circuits was activated during diastole. However, regions peripheral to the circuit were also activated during diastole.

Conclusions—The pattern of reentrant activation determines the specificity of diastolic activity for locating critical sites for ablation of VT. (Circulation. 1998;98:2598-2607.)

Key Words: ablation ■ myocardial infarction ■ mapping ■ reentry ■ tachycardia

Ablation of reentrant sustained ventricular tachycardias (VTs) associated with ischemic heart disease has not been uniformly successful because localization of the complete reentrant circuit cannot always be accomplished by mapping.¹,² As a result, surrogate markers, such as fractionated electrograms and continuous electrical activity³ or the timing of activation with respect to the ECG, have been used as guidelines for locating the circuits, subject to confirmation by entrainment at these sites.²,³,⁴ For example, sites activated at various times during diastole, either early, mid, or late, have been designated as indicating activity in the circuit.¹,⁴ In addition, even when the circuit is located successfully, termination of reentry may require ablation of a critical region of the circuit, whereas ablation of other regions in the circuit might not be effective. This may account for the variable efficacy of ablating sites with diastolic activity in stopping VT even when entrainment has suggested that ablation should be successful.¹,³ The reason for this variability is not completely understood but was investigated in this study in a canine infarct model of VT.⁶

Methods

Experimental Model

Reentrant circuits were mapped in the epicardial border zone (EBZ) of 29 healing canine infarcts,² during sustained monomorphic VT (>30 seconds' duration), 4 days after surgical occlusion of the left anterior descending coronary artery (LAD). Tachycardias were induced by standard programmed stimulation protocols. All our methods, mapping electrode arrays with 196 or 312 bipolar electrodes and recording instrumentation for these studies, have been described in previous publications.⁷-⁹

Data Processing

Our methods for determining local activation times and drawing isochronal maps have been described in detail previously.²,⁹ The time of activation of each region of the reentrant circuits was then correlated with the surface ECG (leads I and II) to determine which part of the circuits were activated during electrical systole (during the QRS complex) and which parts were activated during electrical diastole (the remaining cardiac cycle that is not during the QRS). Although it is often difficult to determine the onset and end of the QRS with precision during VT, when there was an isoelectric segment between the end of the T wave and the beginning of the
subsequent QRS, the onset of the QRS was taken as the earliest reproducible deviation from the isoelectric baseline in either lead I or II. The end of the QRS was taken as the time of return of the last wave of the complex to baseline in both leads (example in Figure 7). In cases in which there was no isoelectric segment between the end of the T wave and the subsequent QRS, the onset of the QRS was indicated by a negative deflection superimposed on the T wave (example in Figure 1). In cases in which no isoelectric segment was evident between the QRS complex and the T wave, the end of the QRS was selected at the inflection point that separates the convex QRS wave returning to baseline from the concave T wave departing from baseline (example in Figure 2). In cases of rapid tachycardias, the onset of the QRS could be determined on the first tachycardia beat occurring after premature stimulation when it had the same morphology as during the sustained tachycardia (example in Figure 3). The diastolic period was further subdivided by designating the midpoint of the diastolic interval as middiastole, the time between the end of the QRS complex and this midpoint as early diastole, and the time between the midpoint and the onset of the subsequent QRS as late diastole. The time resolution of these measurements on the ECG was 4 ms. In addition, the exit route from the EBZ to the rest of the ventricles was determined as previously described. This is the region of the electrode array margin activated within 10 ms before the onset of the QRS of the surface ECG during tachycardia.

Results

Reentrant Circuits With a Figure-8 Activation Pattern

Because ablation of the central common pathway (CCP) in figure-8 reentrant circuits is predicted to be the vulnerable region for termination of tachycardia because it is the only region that is part of both reentrant circuits, we determined whether its time of activation in 35 tachycardias (mean cycle length ± SD, 216 ± 39 ms) had a consistent relationship to the ECG.

Figure 1A shows the pattern of activation of 1 of the figure-8 reentrant circuits during a VT with a cycle length of 233 ms. The reentrant wave front at the beginning of the time...
window is moving from the left lateral margin (LL) toward the LAD margin of the electrode array (isochrones 0 to 60 ms). The sequence of isochrones shows that it progresses in this direction between 2 lines of functional conduction block, indicated by the thick black lines (not present during sinus rhythm or ventricular pacing), that are parallel to each other and to the long axis of the myocardial fiber bundles. At the ends of these lines of block, the wave front splits into 2, 1 moving to the left around the end of the left line of block and then back toward the LL margin to complete 1 loop of the figure-8 circuit, the other moving to the right around the end of the right line of block and then back to the LL margin to complete the other loop of the circuit. These 2 wave fronts coalesce at the LL margin. The region of the circuit between the 2 lines of functional block that is common to both reentrant wave fronts is the CCP, and the regions of the circuit lateral to the lines of block are designated the outer pathways. In this example, the CCP extends from isochrone 0 (the same as isochrone 230) to isochrone 70, a time of 70 ms, ≈30% of the activation time for the complete circuit. The exit route to the ventricles for the reentrant impulse is shown by the asterisks in Figure 1A. The time interval from the exit from the CCP (70 ms) to the exit point to the ventricles (120 ms) was 50 ms.

Figure 1B shows the relationship of the time of activation of different regions of the circuit to the ECG. Because the entire reentrant circuit is in the EBZ, electrograms occur both during electrical systole and diastole. Systole corresponds to isochrones 120 to 210 (90 ms) on the map, in the outer pathways of the figure-8 circuit. Electrograms a through d, which were recorded from this region, occur during the QRS complex (Figure 1B). Diastole corresponds to isochrones 210 to 230 ms and 0 to 120 ms (140-ms duration). Electrograms e through m (Figure 1B) are activated during diastole. During
70 ms of the 140-ms diastolic interval, there is activation of the CCP (isochrones 0 to 70); electrograms f through k, which were recorded in the CCP, were activated during diastole. Therefore, in this example, activation of the CCP occurs during early (electrodes f through i) and late (electrodes j and k) diastole. Regions outside the CCP are also activated during diastole: isochrones 210 to 230, which include electrode e, and isochrones 70 to 120, which include electrodes l and m.

Figure 2 shows another relationship between activation of a figure-8 reentrant circuit and the ECG during a tachycardia with a cycle length of 216 ms. The curved arrows in Figure 2A indicate 2 wave fronts moving clockwise and counterclockwise around 2 lines of functional block (thick black lines). The exit route to the ventricles is at the LAD margin (asterisks), activated at 210 ms. In this example, it is more difficult to designate the exact time of activation of the CCP because of the transverse orientation of the left line of block. In the strictest definition, the wave front passes between the 2 lines of block between isochrones 120 to 150, which is the CCP. Activation of this region requires only 30 ms of the 216-ms cycle length of the tachycardia. In Figure 2B, in which electrograms from the circuit around the right line of block are displayed along with the ECG, it can be seen that the time of activation between electrograms h and i, which designate approximately the beginning and end of the CCP, occurs during only a small portion of the early diastolic interval. Regions of the circuit outside the CCP, between isochrones 100 to 120 (electrogram g) and 150 to 220 (electrograms j and k), are also activated during early and late diastole. Systole corresponds to isochrones 0 to 100 (electrograms a through f in Figure 2B). Therefore, in this example, the very short CCP is activated during only a very small part of early diastole, with activation outside the CCP occurring during most of diastole.
Figure 3A shows the activation map of another circuit during a VT with a cycle of 148 ms. Two reentrant wave fronts, shown by the curved arrows, are moving clockwise and counterclockwise around 2 parallel lines of functional block (thick black lines). Another short and mostly transverse line of block is located above the right line of block but is not an integral part of the circuit. As a consequence of the short cycle length, the duration of the QRS of 108 ms (systole) was 73% of the cycle length, and diastole (40 ms) was only 27% of the cycle length (diastole was 61% and 54% of the cycle lengths for the tachycardias shown in Figures 1 and 2, respectively). A large part of this reentrant circuit, extending from isochrones 110 to 150 and 0 to 70 (108 ms), was activated during systole, and only a small part of the circuit, extending from isochrones 70 to 110 (40 ms), was activated during diastole. Activation of the CCP between the 2 lines of block begins at isochrone 20 and extends to isochrone 50. Therefore, electrodes c, d, and e are in the CCP and occur during systole. Electrograms f and g, at the exit from the CCP, are activated at the end of systole. Electrogram h occurs during diastole but is not within the CCP.

To compare the location of the time of activation of the CCP in the tachycardia cycle from all tachycardias with different cycle lengths, we normalized the beginning and the end of the activation of the CCP (and consequently the time of activation of the CCP) with respect to the cycle length of the tachycardia (Figure 4). For each tachycardia (labeled 1 to 35), the thin horizontal line represents activation of the entire CCP with respect to the onset of the QRS (dashed vertical line at the left) and the entire cycle length (ie, to the onset of the subsequent QRS, represented by the dashed vertical line at the right). The solid circle is the midpoint of the CCP. The location of the early diastolic interval for each tachycardia is indicated by the horizontal shaded bar. Therefore, the time between the dashed vertical line at the left and the beginning of each shaded bar indicates QRS (systole) duration. The time between the end of the shaded bar and the dashed vertical line at the right indicates the late diastolic interval. For example, activation of the CCP for tachycardias 2 through 5 begins before the onset of early diastole (end of systole) and ends before the onset of late diastole. Conversely, activation of the CCP of tachycardias 27 through 29 begins after the onset of early diastole and extends into late diastole.

The total time for activation of the complete CCP occurred over >1 ECG segment (systole and early and late diastole) in 29 of the 35 tachycardias (Figure 4). In 8 of the tachycardias, activation of the CCP occurred during systole and early diastole (2–5, 7, 9, 22, 30); in 17 tachycardias, it occurred during early and late diastole (10, 13–15, 17–19, 21, 24–29, 31, 34, 35); and in 4 tachycardias, it occurred during systole and early and late diastole (12, 20, 32, 33). In 1 tachycardia, the CCP was activated entirely during systole (1), and in 5 it was activated entirely during early diastole (6, 8, 11, 16, 23). Despite this variability, the midpoint of the CCP (black dot in Figure 4) was located in early diastole (shaded area in Figure 4) in 32 of the 35 tachycardias (91.4%), whereas in 1 tachycardia (2.9%, tachycardia 28 in Figure 4), it was located in late diastole and in 2 tachycardias (5.7%, tachycardias 1 and 30 in Figure 4), it was located in systole. Figure 4 also shows the time elapsed from the end of activation of the CCP to the exit point to the ventricle, which occurs at the onset of the QRS (1.00 in Figure 4). Significant variability occurred, ranging from experiments in which the exit to the ventricles occurred long after activation of the CCP (experiments 2 through 9 in Figure 4) to experiments in which the exit to the ventricles occurred almost immediately after activation of the CCP (experiments 32 and 35 in Figure 4).

Significant variability also occurred in the time for activation of the entire CCP for all 35 tachycardias, which ranged from 20 to 140 ms (mean±SD, 57±24 ms). The time for activation of the CCP was directly proportional to the tachycardia cycle length (Figure 5A; \( r^2=0.53 \)). However, the percentage of the tachycardia cycle length during which the reentrant impulse activated the CCP was not related to the tachycardia cycle length (Figure 5B; range, 9% to 44%; mean, 26±8%), because there was an increase in activation time in other regions of the circuit as well as the CCP as the cycle length increased.

During the time interval of activation of the CCP, the electrical activity on the EBZ was restricted to the CCP in 27
of the 35 tachycardias (77%); no other region of the EBZ was being activated at the same time as the CCP (Figures 1 and 2). In the other 8 tachycardias (23%), activity at sites not located in the CCP was occurring at the same time as the activation of the CCP. The time of overlap ranged from 7 to 20 ms (mean±SD, 13±5 ms) (Figure 3). Those sites were unrelated to the maintenance of the reentrant circuit responsible for the tachycardia.

**Reentrant Circuits With a Single Reentrant Loop Activation Pattern**

In 10 VTs, the reentrant circuit on the EBZ was characterized by a single reentrant loop (mean cycle length±SD, 184±23 ms), although this does not eliminate the possibility that another reentrant loop occurred outside the mapped region that involved normal myocardium. An example of 1 of these circuits with a cycle length of 190 ms is shown in Figure 6. The sequence of isochrones from 10 to 190 ms is around a central fulcrum of functional block (thick black line), as shown by the curved arrows (Figure 6A). There are 2 exit routes to the ventricles from the EBZ at the LAD and the LL margins of the electrode array (asterisks in Figure 6A). Additional short lines of block are also located at the bottom right of the map. Figure 6B shows the ECG and electrograms recorded from the circuit (circled sites on the activation map).

**Figure 5.** A, Relationship between time for activation of CCP and cycle length of tachycardia (CL). Each of 35 tachycardias is represented by a black dot. Data were fitted by a linear regression model; r is correlation coefficient. Slope of regression line is significantly different from zero. B, Relationship between percent of tachycardia cycle length occupied by CCP activation and tachycardia CL. Slope of regression line is not significantly different from zero (Sigma Stat, Jandel Scientific Software).
The region of the circuit activated during isochrones 120 to 190 and 190 to 10 (electrograms g through i and a) was activated in systole, and the region between isochrones 10 and 120 (electrograms b through f) was activated during diastole, with the region between isochrones 10 and 65 activated during early diastole. Figure 6C shows electrograms recorded at the boxed sites in Figure 6A, indicating that large regions of the EBZ located at a distance from the central region of block are also activated during the systolic and diastolic intervals (both early and late diastole) because of the centrifugal spread of the wave front away from the central region. Therefore, in circuits caused by single reentrant wave fronts rotating around a central region of block, it might not be possible to locate a specific region of the circuit on the basis of the timing of electrograms with relation to the ECG, and regions distant from the circuit can be activated throughout the diastolic interval.

In 8 of the 10 reentrant circuits, in addition to the central fulcrum of block around which activation rotated, other lines of block were present in close proximity to the circuit. The effect of the additional lines of block was often to confine the electrical activity to smaller regions (close to the central line of block) during a specific time in the cardiac cycle than would be expected if only the central line were present. In Figure 7A, the reentrant wave front (arrows) rotates around a central region of block, the vertical thick black line, surrounded by circled electrodes. In addition to this central line of block, another line of block having an inverted U shape is present above it. As a result, activity in the EBZ between isochrones 60 and 200 (~60% of the 230-ms tachycardia cycle) is confined to the region between this line and the line of block forming the central fulcrum; activation cannot spread centrifugally away from the circuit as in Figure 6A. The additional line
of block resulted in the creation of a bystander pathway\(^1\) (at the top of the inverted U) from isochrones 110 to 150 (open arrow). In Figure 7A, isochrones 0 to 80 correspond to systole (see ECG and electrograms a through f in Figure 7B), isochrones 80 to 160 to early diastole (electrograms g through i), and isochrones 160 to 230 to late diastole (electrograms j and k). Electrogram l was recorded in the bystander pathway, which was located in an area that was activated every other reentrant beat (2:1 conduction). Therefore, the time between both lines of block (the time of confined electrical activity in the EBZ) corresponds to early and part of late diastole on the ECG. In the other 6 tachycardias with a single reentrant loop and additional lines of block, the areas of confined epicardial electrical activity occurred during early diastole in 3 tachycardias and during late diastole in the other 3.

During figure-8 reentrant circuits (Figures 1 to 3), the 2 reentrant wave fronts arrive at the entrance to the CCP at approximately the same time, and both complete a reentrant excursion. This requires that each wave front has a similar revolution time around each of the 2 lines of block. However, 1 of the wave fronts may arrive at the entrance much earlier than the other to complete its circuit. We have classified these circuits as having a single reentrant loop. An example is shown in Figure 8A. The completed circuit is shown by the black curved arrows; the reentrant wave front rotates in a clockwise direction around the vertical line of block, surrounded by circled electrodes a through i, in 170 ms (cycle length of the tachycardia). There is also a longer horizontal line of block around which another wave front rotates in the counterclockwise direction (open arrows). However, at 160 to 170 ms, when the clockwise circuit is completed, the counterclockwise circuit is turning around the left end of the line of block and then collides with the clockwise wave front but does not complete its circuit. The counterclockwise pathway, there-
fore, is a bystander pathway. There is a narrow region between the 2 lines of block at the right, between isochrones 10 and 40, which might be a vulnerable site for ablation, although a lesion here might also serve to enlarge the circuit. Electrodes j, f, and g are located in this region, with only site g activated in (early) diastole, whereas sites j and f are activated at the end of systole (Figure 8B). Isochrones 10 to 30 also extend away from this narrow region between the lines of block, toward the apex (at the left) and LL margins. Other recording sites within these isochrones (k and l), which are not in the narrow region, are also activated at the end of systole and beginning of diastole (Figure 8B). Sites h, i, j, and k are activated in mid and late diastole, are also not within the possible vulnerable region (Figure 8B). Therefore, diastolic activity does not seem to be suggestive of an effective ablation site.

Discussion

A major challenge for the ablation of reentrant VTs has been to locate the reentrant circuit, and more specifically, a region of the circuit that is crucial for maintenance of circulating activity. This is particularly problematic in the infarcted ventricles, because circuits may have different sizes and shapes, be caused by different mechanisms (anatomic or functional), and involve different regions in different hearts (reviewed in Reference 11). Current electrical mapping techniques used clinically are often inadequate to map complete ventricular reentrant circuits.\(^1\),\(^3\),\(^4\) Therefore, surrogate markers of reentrant circuit location have been sought. One such marker has been the timing of local electrical activity with respect to the ECG during tachycardia. Whereas during normal sinus rhythm, activation of the ventricles occurs mainly during the QRS complex, during reentrant excitation, activation of the circuit has to occur throughout the cardiac cycle, including diastole, leading to the suggestion that regions of continuous\(^2\) or isolated\(^3\),\(^12\) diastolic activity represent all or part of the reentrant circuit. Thus, these regions became targets for ablation of VT with variable success. Sites of earliest activation (at least 50 ms preystolic\(^1\)) have also been postulated to represent exit routes from the circuit to the ventricles and were assumed to be close to regions necessary for the maintenance of reentry.\(^3\),\(^5\) However, the success rate of ablation at these exit sites has been modest. Subsequently, stimulation at sites of diastolic activity (concealed entrainment) have proved to be very useful for identifying which of those sites are truly integral parts of the reentrant circuit.\(^2\),\(^3\),\(^5\),\(^12\)

Sites that are activated during diastole but do not meet the criteria determined to indicate localization in the circuit by stimulation techniques have been designated as bystander pathways.\(^3\) Conversely, the application of radiofrequency energy at sites that meet the criteria for concealed entrainment is not always effective in the elimination of the tachycardia.\(^2\),\(^3\),\(^12\)

Despite the differences between the experimental model of VT used in our study and clinical tachycardia, examination of our data can provide a conceptual framework for understanding the different relationships between circuit activation and the ECG that may be a cause of the variable efficacy of ablation of diastolic sites. An important concept that can be derived from our experimental results is that the pattern of reentrant activity is an important determinant of the relationship between reentrant circuit activation and the ECG. In figure-8 reentrant circuits, there is a specific region of the circuit that must be ablated to terminate tachycardia. This vulnerable region is the CCP, which is activated by both reentrant wave fronts.\(^9\) Our results show that at least part of this region is almost always activated during diastole (34 of 35 circuits), although segments of this region can also be activated during systole. More specifically, we consistently found that the midpoint of the CCP was activated during early diastole, a time during which, in most cases, no other region of the EBZ was activated. Activity during late diastole most often occurred between exiting the CCP and the exit route to the ventricles. The specificity of early diastolic activity coinciding with CCP activation occurred because the lines of block that form the boundaries of the CCP prevent the reentrant wave front from exiting to the ventricles and, therefore, prevent the onset of the QRS until the impulse leaves this region. This is expected whether the lines of block are functional, as in anisotropic reentry,\(^7\) or anatomic, as proposed in the model of Stevenson et al.\(^1\) However, our results show that variability of activation patterns, even when the circuit is figure-8, can influence the ease at which such diastolic activity might be located. Whereas the “textbook” figure-8 pattern involves rotation of 2 reentrant wave fronts around long parallel lines of block, with a long CCP between the 2 (Figure 1), Figure 2 shows that sometimes when the lines of block have different directions, the vulnerable CCP may be quite small and contribute to only a small segment of diastolic activity. This may explain some clinical observations in which critical sites in the circuit have been difficult to identify from stimulation in regions of diastolic activity.\(^2\),\(^3\) The vulnerable region of the circuit might also be activated entirely during systole, when tachycardia cycle length and the diastolic interval are short (Figure 3). In addition, the variable distance between the location of the exit route and the vulnerable region of the circuit in this model is also noteworthy and may provide a reason for the variable efficacy of ablation at exit routes in the termination of VT.\(^1\) In figure-8 reentry (Figures 1 to 3), regions not in the CCP were also activated during early and late diastole, showing why diastolic activity alone is not always an accurate predictor of CCP location.\(^3\)

Sometimes only a single reentrant circuit could be mapped, although we cannot rule out the presence of an additional circuit outside the mapped region. Nevertheless, analysis of these circuits provides possible explanations for the lack of specificity of diastolic activity, and pacing at sites of diastolic activity, for pointing out crucial regions for ablation during some VTs. When there is only a central fulcrum of block (Figure 6), there can be centrifugal spread of activity away from the reentrant circuit. When this occurs, large regions that are not crucial for reentry are activated simultaneously with the circuit (considered to be that part of the reentrant wave front closest to the central region of block). Indeed, in general, even when the location of the reentrant circuit is clearly seen from high-resolution maps as in Figure 6, a localized vulnerable
region for effective ablation that is critical for the maintenance of reentry is not obvious. Also, stimulation at sites of diastolic activity even within the reentrant circuit might not cause exact entrainment in this pattern of reentry. Single-loop reentry, however, can be modified by lines of block that are not part of the circuit and that can prevent the reentrant wave front from spreading outward to all regions of the EBZ (Figure 7). Activation in part of the circuit may be confined to narrow regions by the additional lines of block, which might be vulnerable to an ablation lesion (Figure 7). The region delineated by the additional line of block in Figure 7 formed a bystander pathway, which, although activated early in diastole, was not activated every beat and therefore could not be a crucial part of the reentrant circuit. Similarly, regions of diastolic activity that are not activated 1:1 with each QRS complex during tachycardia have been described during clinical studies, as have bystander pathways with more consistent activation patterns. In addition, pacing at sites in bystander pathways such as sites j, k, and l in Figure 8, which are activated during early diastole, was not activated at every beat and therefore could not be a crucial part of the reentrant circuit. Similarly, regions of diastolic activity that are not activated 1:1 with each QRS complex during tachycardia have been described during clinical studies, as have bystander pathways with more consistent activation patterns. In addition, pacing at sites in bystander pathways such as sites j, k, and l in Figure 8, which are activated during early diastole, might result in concealed entrainment but not be effective sites for ablation.

Limitations
Our results must be considered in light of the limitations of the relationship of this animal model to human VT, including the epicardial location of the reentrant circuits and the role of anisotropy in causing functional reentry. An additional limitation is the probability of inaccuracies in defining the onset and termination of the QRS complex. Had more than 2 ECG leads, including precordial leads, been recorded, an earlier QRS onset and a longer QRS duration might have been evident and no clear diastolic interval been located in more of the tachycardias with short cycle lengths. Even when multiple leads are used, however, precise determination of the beginning and end of the QRS may not be possible.

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References
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