

REVIEW ARTICLES

Exploring Postinfarction Reentrant Ventricular Tachycardia With Entrainment Mapping

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Ventricular tachycardia late after myocardial infarction is usually due to reentry in the infarct region. These reentry circuits can be large, complex and difficult to define, impeding study in the electrophysiology laboratory and making catheter ablation difficult. Pacing through the electrodes of the mapping catheter provides a new approach to mapping. When pacing stimuli capture the effects on the tachycardia depend on the location of the pacing site relative to the reentry circuit. The effects observed allow identification of various portions of the reentry circuit, without the need for locating the entire circuit. Isthmuses where

relatively small lesions produced by radiofrequency catheter ablation can interrupt reentry can often be identified. A classification that divides reentry circuits into one or more functional components helps to conceptualize the reentry circuit and predicts the likelihood that heating with radiofrequency current will terminate tachycardia. These methods are helping to define human reentry circuits.

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After surviving the acute phase of myocardial infarction, 2% to 5% of patients subsequently develop sustained monomorphic ventricular tachycardia due to reentry in the infarct region. Sustained ventricular tachycardia is often a difficult management problem. Patients are subject to recurrent attacks, and antiarrhythmic drug therapy fails to prevent episodes in >40% of patients (1). Implantable cardioverter-defibrillators terminate the arrhythmia either by antitachycardia pacing or shocks and are life-saving, but often fail to prevent symptoms. Ablative surgery is effective for selected patients but is associated with an operative mortality rate of 9% to >20% (2,3). Recently, the efficacy and safety of radiofrequency catheter ablation for supraventricular tachycardias have refocused interest in catheter ablation of ventricular tachycardia. Catheter ablation of ventricular tachycardia due to previous myocardial infarction is a formidable task because the reentry circuits can be large and difficult to define and ablate with catheter

techniques. Reentry circuits can be delineated by reconstructing the activation sequence based on electrogram recordings from a large number of sites (4-10). Although this is possible to some extent during intraoperative mapping, it is rarely possible with catheter techniques. Recently, pacing techniques have been applied during catheter mapping to determine whether a site is in the reentry circuit (11-15). During tachycardia, an electrical stimulus that has sufficient strength to capture produces a predictable effect depending on the pacing site location relative to the tachycardia circuit. In most cases the tachycardia can be reset or, with a train of several stimuli, entrained. These methods are improving our understanding of ventricular reentry circuits.

Ventricular Reentry Circuits

Intraoperative mapping studies have identified a variety of different reentry circuit configurations and shown that the reentry circuits can follow complex paths (4-10). These paths are defined by regions of inexcitable scar, functional block of excitation wavefronts or collision of wavefronts. Three different types of theoretic reentry circuits are shown in Figure 1 for illustration. In many cases, the circulating excitation wavefronts propagate through regions in the infarct that are relatively small masses of tissue, and their depolarization is not detectable on the standard electrocardiogram (ECG) recorded from the body surface. Electrograms recorded directly from these regions are low amplitude and often have fractionated potentials consistent with asynchronous activation of myofiber bundles and slow conduction (4-7,16-21). For these circuits,

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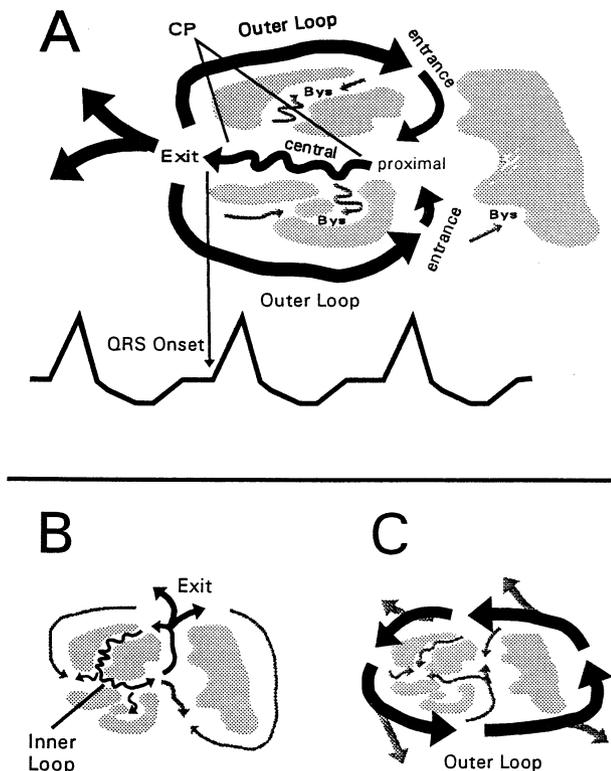


Figure 1. Three theoretic reentry circuits are shown originating from the same chronic infarct. In each panel, the reentry circuit is indicated by the **black arrows**. **Grey arrows** indicate excitation wavefronts from the circuit that depolarize tissue that is not in the circuit (bystanders). **Gray stippled areas** are inexcitable regions in the chronic infarct. **A**, Circuit that contains two loops and a common pathway (CP) through which conduction is slowed, creating a “figure eight” configuration. The common pathway is a relatively small mass of tissue in the chronic infarct, depolarization of which generates low amplitude signals that are not detectable in the standard body surface ECG. The common pathway has an exit and central and proximal regions. The QRS complex is inscribed after the excitation wavefront leaves the common pathway at the exit (arrow to the QRS onset at bottom) and begins propagating around the border of the scar through two outer loops. The excitation wavefronts then enter the infarct region through entrances to reach the proximal portion of the common pathway. Several regions that are in the chronic infarct but do not participate in the circuit are labeled as bystanders (Bys). **B**, Circuit that is contained entirely within the chronic infarct. This circuit consists of an exit and central, proximal and inner loop regions. **C**, Circuit that is a single outer loop. The reentry wavefront circulates around the margins of the chronic infarct. Excitation wavefronts collide in the paths within the chronic infarct; these paths behave as bystanders.

the onset of the QRS complex occurs when the circulating excitation wavefront emerges from the infarct region at some point along the border of the infarct, designated the *exit*, and propagates away to depolarize the larger mass of myocardium surrounding the infarct. To return to the exit region, the circulating excitation wavefront may propagate through relatively normal tissue along the border of the scar (designated an *outer loop*) (Fig. 1A) or through a path within the infarct region (designated an *inner loop*) (Fig. 1B). The wavefront then propagates through the region proximal to the exit. In some

circuits multiple loops are present. Circuits that have two loops sharing a common central region form a figure eight configuration (Fig. 1A) (22). In another type of reentry circuit, the infarct serves as an anatomic obstacle for a single loop along the border of the scar (Fig. 1C) (23,24). The entire circuit is functionally an outer loop.

In the majority of patients, some portion of the reentry circuit exists in the subendocardium, accessible to an endocardial electrode catheter. However, segments of the circuit may extend intramurally and to the subepicardium. In some cases the entire circuit is epicardial or intramural (7,8,10,21). An endocardial catheter may not effectively record signals from, or allow ablation of, segments of the reentry circuit that are deep to the endocardium. Thus, delineation of a complete circuit based on electrogram recordings is often not possible. In addition, some regions in the chronic infarct are excitable but are bystanders that do not participate in the reentry circuit (11,25-27). Analysis of electrograms from bystander regions may confound attempts to identify the reentry circuit.

Radiofrequency Ablation as a Mapping Tool

Radiofrequency catheter ablation can be used as a method to help determine whether a site is in a reentry circuit (11). As radiofrequency current is applied to an endocardial electrode and a large dispersive electrode patch on the body surface resistive heating occurs in the tissue beneath the endocardial electrode (28,29). The increase in temperature first impairs tissue function; further heating makes a permanent lesion. During ablation of accessory pathways in the Wolff-Parkinson-White syndrome, block in the accessory pathway is typically observed when the electrode temperature reaches 50°C, and permanent block usually occurs if the temperature reaches $\geq 60^\circ\text{C}$ (28,29). The high frequency current does not generally elicit propagating depolarizations; ectopic beats are not observed. Thus, block of conduction in an accessory pathway or termination of an arrhythmia during radiofrequency catheter ablation (Fig. 2) usually indicates that a portion of the reentry circuit has been heated sufficiently to impair function and, with uncommon exceptions, indicates that the ablation site is in the reentry circuit. This use of radiofrequency ablation is analogous to the use of cooling during intraoperative mapping (30,31). Interruption of ventricular tachycardia by cooling a site with a sterile ice cube or cryoprobe, or by pressure at the site, has been taken as evidence that the site is in the reentry circuit. Similar findings occur during intraoperative laser myocardial photocoagulation (21).

The size of radiofrequency ablation lesions produced with current electrode catheters is relatively small, generally <1 cm in diameter (29,32). Thus, interruption of tachycardia by ablation suggests that the ablation site is a relatively narrow portion of the reentry circuit. When ablation is performed in a broad path in the circuit, the diameter of the lesion may not be sufficient to interrupt reentry. Furthermore, interruption of reentry may not indicate permanent block of conduction. Radiofrequency current application may produce a permanent

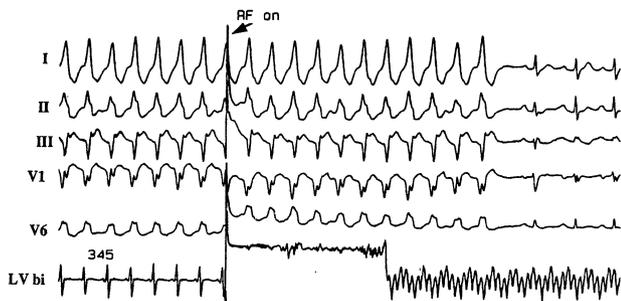


Figure 2. Termination of ventricular tachycardia by radiofrequency (RF) current application to a site in the reentry circuit. From the top are surface ECG leads I, II, III, V₁ and V₆ and an intracardiac recording from the mapping catheter (LV bi). Sustained monomorphic ventricular tachycardia with a rate of 174 beats/min (cycle length 345 ms) is present. At the time indicated by the arrow, application of radiofrequency current is begun, introducing electrical noise in the mapping catheter recording. Tachycardia terminates after 11 beats (~4 s), and sinus rhythm is restored.

lesion that is smaller than the diameter of the region that reached a sufficient temperature to block conduction. Thus, it is important to have other methods for determining whether a site is in the reentry circuit and for predicting whether sufficiently large lesions at that site will interrupt the reentry circuit.

We have used acute termination of ventricular tachycardia by radiofrequency catheter ablation to test methods for catheter mapping (11,15). The findings from catheter mapping and attempted ablation of 75 ventricular tachycardias in 37 patients with a previous myocardial infarction are summarized in Tables 1 and 2 and discussed in more detail later. For the purposes of this analysis, we included data from sites where 1) pacing from the mapping catheter entrained ventricular tachycardia; 2) endocardial electrograms were of sufficient quality to allow analysis for site classification (see later); and 3) radiofrequency current was then applied to the site during ventricular tachycardia to determine whether heating the site would terminate reentry. Sites where radiofrequency current applica-

Table 1. Clinical Characteristics of 37 Study Patients

| | |
|------------------------------|-----------|
| Men/women | 30/7 |
| Age (yr) | 64 ± 10 |
| LVEF (%) | 0.32 ± 11 |
| MI | |
| Ant/Lat | 13 |
| Inf | 20 |
| Ant+Inf | 4 |
| VT configurations | 4 ± 2 |
| VT cycle length (ms) | 406 ± 84 |
| Antiarrhythmic drug at study | |
| Amiodarone | 21 |
| Other | 6 |
| None | 10 |

Data presented are mean value ± SD or number of patients. Ant = anterior; Inf = inferior; Lat = lateral; LVEF = left ventricular ejection fraction; MI = myocardial infarction; VT = ventricular tachycardia.

Table 2. Mapping Sites and Interruption of Ventricular Tachycardia by Radiofrequency Current Application

| | No. of Sites Terminated | RF-Terminated VT |
|--------------------|-------------------------|------------------|
| Isthmus site | 85 | 29% |
| Exit | 35 | 37% |
| Central | 22 | 23% |
| Proximal | 28 | 25% |
| Inner loop | 46 | 9% |
| Adjacent bystander | 35 | 11% |
| Outer loop | 116 | 10% |
| Remote bystander | 116 | 3% |
| Total | 398 | 12% |

RF = radiofrequency current; VT = ventricular tachycardia.

tion changed the configuration of ventricular tachycardia or where termination was preceded by ventricular ectopic beats were excluded. Of 528 sites evaluated during 86 different monomorphic tachycardias, a total of 398 sites evaluated during 75 tachycardias met these criteria. Methods are described in greater detail in the Appendix.

Entrainment

Most of the stable, infarct-related reentry circuits causing monomorphic ventricular tachycardias that allow catheter mapping have an excitable gap (Fig. 3A). This is due to the fact that the time it takes the circulating excitation wave to propagate once around the circuit (i.e., the revolution time) exceeds the refractory period at each point in the circuit. If a catheter is placed at a site in the circuit, and a depolarizing stimulus applied after the site has recovered, but before arrival of the next circulating wavefront, the pacing stimulus will capture the underlying myocardium, producing wavefronts that travel in two directions in the circuit. The stimulated orthodromic wavefront travels in the same direction as the tachycardia wavefronts. The stimulated antidromic wavefront travels in the reverse direction and collides with a returning orthodromic wavefront. The stimulated orthodromic wavefront propagates through the circuit, resetting the tachycardia. If a train of several stimuli is applied at an appropriate rate, such that each stimulus falls during the excitable gap after the preceding stimulus, the reentry circuit is continuously reset. Continuous resetting of a reentry circuit is *entrainment* (33-37). Waldo (36) initially described entrainment in careful studies of atrial flutter and established criteria for identifying entrainment. Although Figure 3 (B and C) illustrates entrainment or resetting produced by pacing at a site in the reentry circuit, early studies focused on entrainment produced by pacing at sites outside the reentry circuit (Fig. 3, D and E). When the pacing site is outside the circuit, the stimulated wavefront propagates through the intervening myocardium to reach the reentry circuit. The stimulated wavefronts then enter the circuit and propagate in antidromic and orthodromic directions, resetting the tachycardia. Thus, entrainment can occur during pacing at sites that are either within or outside the

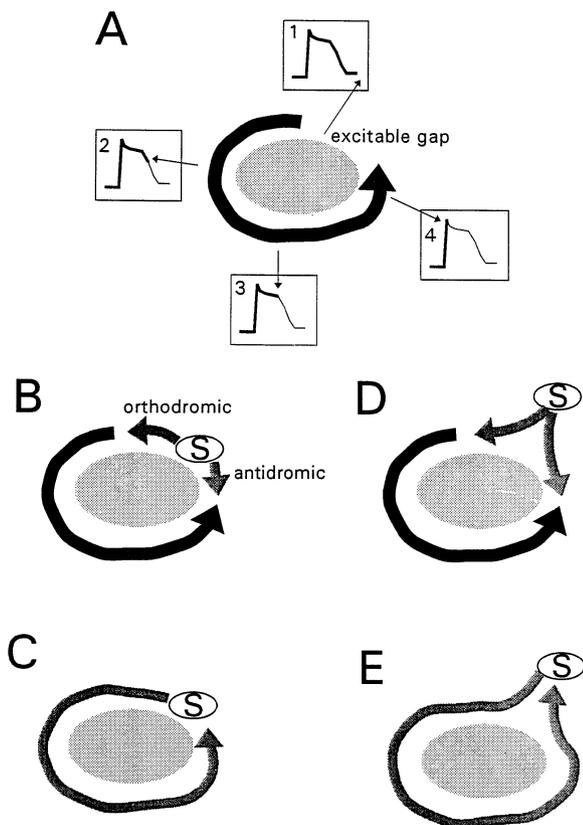


Figure 3. A simple theoretic reentry circuit consisting of a circulating wavefront (black arrow) around an obstacle (gray oval) is shown to illustrate an excitable gap (A), and the effects of a pacing stimulus at a site in the reentry circuit during the excitable gap (B and C) and at a site outside the reentry circuit (D and E). In A, the leading edge of the circulating wavefront is indicated by the large arrowheads. Representative action potentials that would be recorded at this point in time from around the circuit are shown in boxes 1 to 4. An action potential recorded from near the leading edge (box 4) reveals an action potential upstroke (bold line). Sites 2 and 3 are in later stages of repolarization. Site 1 is recorded from the region that has fully repolarized, preceding the excitation wavefront. Thus, at the point in time shown, site 1 is in an excitable gap between the head and tail of the arrow. B, Effects of a premature stimulus that depolarizes tissue at site 1 during the excitable gap. An antidromic wavefront is produced that collides with the circulating orthodromic wavefront. An orthodromic wavefront propagates through the circuit resetting the circuit. C, Stimulated orthodromic wavefront returns to the initial site of stimulation after making one revolution through the circuit. Thus, the time from the stimulus to the following depolarization (postpacing interval) equals the revolution time through the reentry circuit (tachycardia cycle length). D and E, Pacing at a site remote from the circuit. D, An appropriately timed stimulus produces excitation wavefront (gray arrow) that reaches the reentry circuit during the excitable gap and begins propagating in orthodromic and antidromic directions in the circuit. The antidromic wavefront collides with the returning orthodromic wavefront (black arrow) and is extinguished. The stimulated orthodromic wavefront continues through the circuit, resetting the tachycardia. E, Stimulated orthodromic wavefront returns to the initial site of stimulation after propagating to the circuit, then making one revolution through the circuit and returning back to the stimulation site. Thus, the time from the stimulus to the following depolarization (postpacing interval) exceeds the revolution time through the reentry circuit.

reentry circuit, and entrainment alone does not indicate the location of the pacing site relative to the reentry circuit. However, several other findings during entrainment that require analysis of the QRS configuration and recorded electrograms can be used to establish whether the pacing site is in the circuit (11-15).

Postpacing Interval After Entrainment

The postpacing interval measured after entrainment is an indication of the proximity of the pacing site to the reentry circuit (11). At sites in the reentry circuit, the stimulated orthodromic wavefront returns to the pacing site after one revolution through the reentry circuit (Fig. 3C). Thus, the interval from the last stimulus that captures to the next activation at the pacing site is equal to the revolution time through the circuit, which is the tachycardia cycle length. In contrast, pacing at a site remote from the reentry circuit can entrain tachycardia, but the interval from the last stimulus to the subsequent depolarization at the pacing site (*postpacing interval*) is equal to the conduction time from the pacing site to the circuit through the circuit and back to the pacing site (Fig. 3E). Thus, the postpacing interval exceeds the tachycardia cycle length. The longer the conduction time between the pacing site and reentry circuit, the greater the difference between the postpacing interval and the tachycardia cycle length. For postinfarction ventricular tachycardia, a postpacing interval within 30 ms of the tachycardia cycle length is associated with termination of tachycardia by radiofrequency catheter ablation, thereby indicating close proximity to the reentry circuit (11).

Analysis of the postpacing interval assumes that the conduction velocity through the reentry circuit and the reentry path do not change during pacing. If the conduction velocity slows or the length of the reentry path increases, the postpacing interval prolongs, and entrainment produced by pacing at such a reentry circuit site would then have a postpacing interval longer than the tachycardia cycle length. Because rapid pacing rates are likely to slow conduction velocity or alter the conduction path in the circuit (22,38,39), pacing rates only slightly faster than the tachycardia cycle length are used to entrain the tachycardia for measurement of the postpacing interval. The postpacing interval requires that local activation time be assessed from the electrogram recorded at the pacing site. In regions of slow, abnormal conduction, the recorded electrograms have a long duration and multiple rapid components that produce a fractionated appearance (4,17,20). It is not always possible to be certain of the precise activation time. In general, we assume that any component of an abnormal signal could indicate local activation and tend to err on the side of accepting a false positive result. Some recording systems do not allow signals to be recorded from the same electrodes that are used for pacing. Recording from sites adjacent to the pacing site, usually from the proximal electrodes on the same electrode catheter, introduces an error that is related to the conduction time between the pacing and recording sites (40).

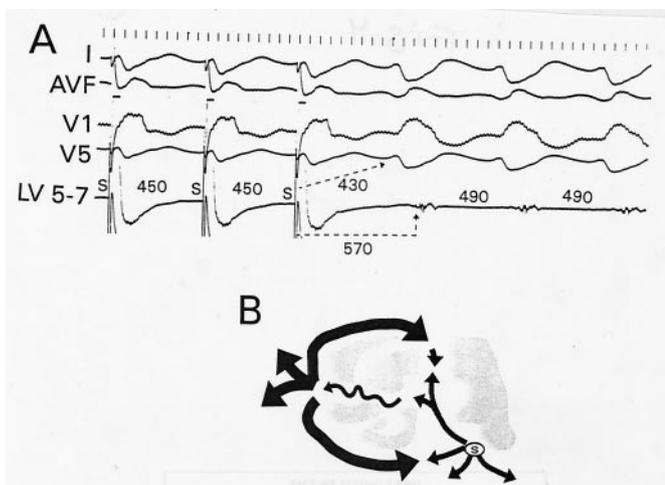


Figure 4. Pacing at a remote bystander is shown in data from a patient (A) and in a schematic of a reentry circuit (B). **A**, 50-ms time lines, surface ECG leads I, aVF, V₁ and V₅ and a bipolar recording from the distal electrode pair of the left ventricular mapping catheter at site 5-7 (LV 5-7). Sustained monomorphic ventricular tachycardia with a cycle length of 490 ms is present (last 2 beats). The last three stimuli (S) of a stimulus train at the mapping site are shown. Pacing at a cycle length of 450 ms entrains tachycardia with QRS fusion. In the left ventricular recording (LV 5-7), the postpacing interval is 570 ms and therefore exceeds the tachycardia cycle length by 80 ms, consistent with a bystander site. **B**, Pacing is at a bystander site in the infarct border region in the same reentry circuit as in Figure 1A. The stimulated wavefronts (gray arrows) propagate away from the border of the scar, altering the QRS configuration. Stimulated wavefronts also reach the reentry circuit and propagate through the circuit, resetting it. The postpacing interval is the conduction time from the pacing site to the circuit, through the circuit and back to the pacing site and exceeds the tachycardia cycle length.

With electrode catheters that have a 2.5-mm interelectrode distance, this error is relatively small (<30 ms) when conduction velocity is nearly normal, but increases markedly when conduction velocity is depressed to slower than 0.2 m/s, as may occur in infarct regions (4,40). In general, however, there is good agreement between the postpacing interval measured at the distal electrodes and that measured at the proximal electrodes of radiofrequency ablation catheters (40).

QRS Configuration During Entrainment

The configuration of the QRS complex during entrainment also provides useful information. During ventricular tachycardia, the QRS configuration is determined by the sequence of activation of the ventricular myocardium, most of which is remote from the infarct region. Pacing at a site remote from the infarct circuit (Fig. 4) produces wavefronts that alter the ventricular activation sequence. This changes the QRS configuration during pacing compared with that of ventricular tachycardia. During classic entrainment (Fig. 4), the stimulated wavefronts that propagate out from the pacing site collide with wavefronts that have traveled orthodromically through the reentry circuit and are now leaving the reentry circuit from its

exit. Thus, the QRS complex is due to fusion of wavefronts propagating away directly from the pacing site with those emerging from the tachycardia circuit. During pacing at a constant rate, the configuration of the fusion QRS complexes remains constant. If the pacing rate is increased, the stimulated wavefronts from the pacing site travel further, capturing more of the ventricle, and the QRS configuration more closely resembles that produced by pacing in the absence of tachycardia. Thus, the degree of fusion is progressive as the pacing rate increases, another feature of classic entrainment.

Entrainment with QRS fusion can also occur during pacing at some reentry circuit sites where the stimulated antidromic wavefronts propagate away from the infarct region (see later ["Outer loop sites"] and Fig. 5). However, at other sites in or near the reentry, circuit pacing entrains ventricular tachycardia without changing the QRS configuration (11-15). The tachycardia appears to be accelerated to the pacing rate, but the QRS configuration is identical to that of the tachycardia in the absence of pacing; QRS fusion is not detectable on the surface ECG (Fig. 6). This can occur when the stimulated wavefronts are confined in or near the circuit such that they reach the surrounding myocardium only by propagating through the circuit. During entrainment produced by pacing from such a site, collision of stimulated antidromic and orthodromic wavefronts is occurring near the pacing site, in or near the reentry circuit. The antidromic wavefront depolarizes only a small mass of tissue that is not detectable on the surface ECG. Thus, fusion between stimulated antidromic and orthodromic wavefronts is concealed. This has been called *entrainment with concealed fusion*, exact entrainment or a form of concealed entrainment (11-15). Concealed entrainment was initially used to describe the situation where pacing is performed at a site remote from the reentry circuit and the majority of the ventricle is captured by the stimulated wavefront that propagates away directly from the pacing site, rather than by the wavefront emerging from the tachycardia circuit (33). The QRS configuration is the same as that produced by pacing during sinus rhythm, and entrainment can be detected only if electrograms are recorded directly from the reentry circuit.

S-QRS Interval During Entrainment With Concealed Fusion

During entrainment with concealed fusion, the interval from the stimulus to the onset of the QRS complex indicates the conduction time from the pacing site to the reentry circuit exit. For pacing sites that are in the circuit, this provides a crude indication of the distance from the pacing site to the reentry circuit exit (Fig. 6 and 7). It also provides a means of determining whether the site is likely to be a bystander, adjacent to the reentry circuit (11,41). When the site is in the circuit, the electrogram to the QRS interval also indicates the conduction time from the site to the reentry circuit exit. Thus, the S-QRS interval during entrainment with concealed fusion approximates the electrogram to the QRS interval during

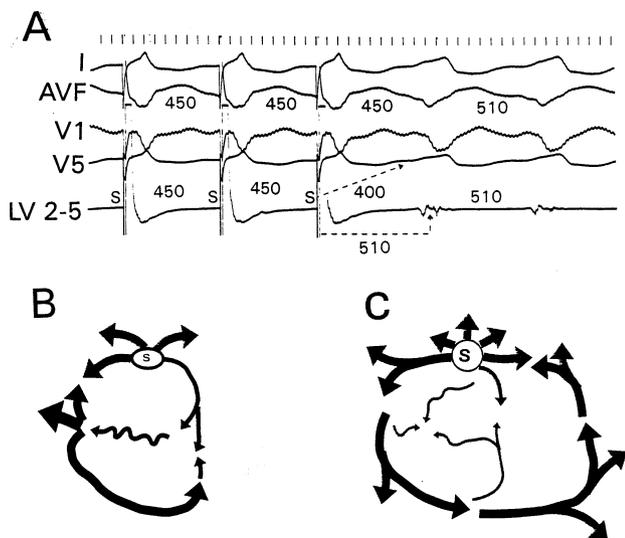


Figure 5. Pacing at an outer loop site is shown in data from a patient (A) and in schematics (B) of two reentry circuits shown in Figure 1, A and C. A, 50-ms time lines, surface ECG leads I, aVF, V₁ and V₅ and a bipolar recording from the distal electrode pair of the left ventricular mapping catheter at site 2-5. Sustained monomorphic ventricular tachycardia with a cycle length of 510 ms is present (last 2 beats of the panel). The last three stimuli (S) of a stimulus train at the mapping site are shown. Pacing at a cycle length of 450 ms advances all QRS complexes and electrograms to the pacing cycle length, consistent with entrainment. The QRS complexes immediately after each stimulus have a configuration different from that of the tachycardia due to fusion between antidromic wavefronts and orthodromic wavefronts that exit the reentry circuit. Tachycardia is entrained with QRS fusion. In the left ventricular recording (LV 2-5), electrical activity is present 510 ms after the last stimulus, as indicated by the **dashed arrow**. The postpacing interval therefore approximates the tachycardia cycle length, consistent with a reentry circuit site. B, Stimulation is performed at an outer loop site (S) in the reentry circuit shown in Figure 1A. Stimulated antidromic wavefronts (**gray arrows**) propagate away from the infarct border, altering the activation sequence distant from the infarct and thereby altering the QRS complexes. The stimulated orthodromic wavefront propagates through the circuit, resetting the reentry circuit. After the last stimulus, the pacing site is next depolarized by the orthodromic wavefront that has made one revolution through the circuit. The postpacing interval therefore approximates the tachycardia cycle length. C, Similar findings are shown for the reentry circuit that consists solely of an outer loop, as shown in Figure 1C.

tachycardia (Fig. 6 and 7). When the pacing site is adjacent to but not in the reentry circuit, the electrogram to the QRS interval does not indicate the conduction time from the pacing site to the reentry circuit exit. If pacing at a bystander site entrains tachycardia with concealed fusion, the S-QRS interval usually does not approximate the electrogram to the QRS interval (Fig. 8).

Types of Mapping Sites

Remote bystanders. Sites that are not in a reentry circuit are designated *bystanders*. Pacing at a bystander site can have any of several effects (11). The stimulated wavefronts may

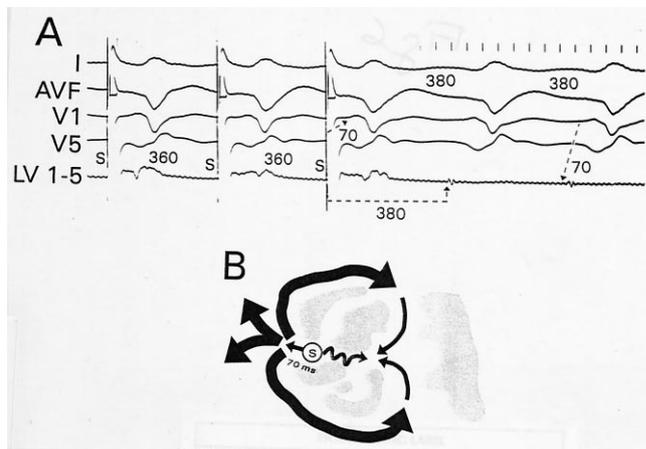


Figure 6. Pacing at an exit site is shown in data from a patient (A) and a schematic (B) of the theoretic reentry circuit shown in Figure 1A. A, 50-ms time lines, surface ECG leads I, aVF, V₁ and V₅ and an intracardiac bipolar recording from the mapping catheter at site 1-5. Sustained monomorphic ventricular tachycardia with a cycle length of 380 ms is present (last 2 beats of the panel). The last three stimuli (S) of a stimulus train at a cycle length of 360 ms are shown. Pacing accelerates the QRS complexes to the paced cycle length of 360 ms without altering their configuration compared with that of the QRS complexes during tachycardia (entrainment with concealed fusion). After the last stimulus, the next electrogram recorded at the pacing site occurs after 380 ms. The postpacing interval therefore matches the ventricular tachycardia cycle length, consistent with a reentry circuit site. Also, the S-QRS interval approximates the electrogram to the QRS interval during tachycardia, as indicated by the **dashed arrow** from the last QRS complex. The S-QRS interval is 70 ms, which is 19% of the tachycardia cycle length and is therefore consistent with an exit site. B, The stimulus site (S) is in the exit. Orthodromic excitation wavefronts are indicated by the **black arrows**. The stimulated antidromic wavefront is indicated by the **gray arrow**. The stimulated antidromic wavefront collides in the infarct with a returning orthodromic wavefront and is extinguished. The stimulated orthodromic wavefront exits the infarct from the same site as the tachycardia wavefronts, advancing the QRS complex without altering the QRS configuration (entrainment with concealed fusion). The next depolarization at the pacing site after the last stimulus occurs after the stimulated orthodromic wavefront has made one complete revolution through the reentry circuit. Thus, the postpacing interval approximates the tachycardia cycle length. The electrogram to the QRS interval during tachycardia reflects the conduction time from the mapping site to the point where the wavefront exits the circuit and begins to inscribe the QRS complex. This approximates the S-QRS interval during pacing.

collide with wavefronts from the tachycardia circuit without reaching the circuit; pacing then has no effect on tachycardia. Pacing at rates faster than the tachycardia for a sufficient length of time generally produces stimulated wavefronts that reach the circuit and entrain or terminate tachycardia (Fig. 4). If tachycardia is entrained, the postpacing interval exceeds the tachycardia cycle length. At most bystander sites, the stimulated wavefronts alter the sequence of ventricular activation, and thereby the QRS configuration. Such sites are designated *remote bystanders*. Depending on the location of the pacing site relative to the reentry circuit, there may be constant QRS fusion, or entrainment may occur only when the wavefronts

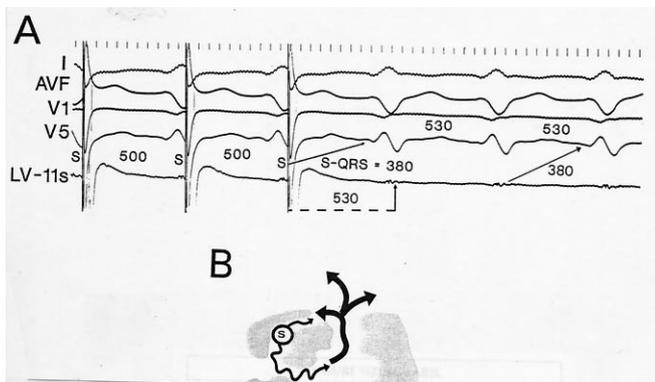


Figure 7. Pacing at an inner loop site is shown in data from a patient (A) and a schematic (B) of the reentry circuit shown in Figure 1B. A, 50-ms time lines, surface ECG leads I, aVF, V₁ and V₅ and a bipolar recording from the distal electrode pair of the left ventricular mapping catheter at site 11s. Sustained monomorphic ventricular tachycardia with a cycle length of 530 ms is present (last 2 beats of the panel). The last three stimuli (S) of a stimulus train at the mapping site are shown. Pacing at a cycle length of 500 ms entrains the tachycardia with concealed fusion as in Figure 3. In the left ventricular recording (LV-11s), electrical activity is present 530 ms after the last stimulus, as indicated by the **dashed arrow**. The postpacing interval therefore approximates the tachycardia cycle length, consistent with a reentry circuit site. Also, the S-QRS interval approximates the electrogram to the QRS interval. The S-QRS interval is 380 ms, which is 71% of the tachycardia cycle length and is therefore consistent with an inner loop site. B, Pacing is at an inner loop site in the same reentry circuit as in Figure 1B. The stimulated antidromic wavefront (**gray arrow**) collides with an orthodromic wavefront and is contained in the chronic infarct. The stimulated orthodromic wavefront resets the tachycardia with concealed fusion, similar to Figures 3 and 4 but with a longer S-QRS interval due to the long conduction time between the pacing site and the exit of the excitation wavefronts from the scar.

from the stimulus site have depolarized so much of the ventricles that the QRS configuration resembles the configuration produced by pacing in the absence of tachycardia (a form of concealed entrainment).

Radiofrequency current ablation terminates ventricular tachycardia at only 3% of sites classified as remote bystanders based on entrainment (Table 2). The reason that tachycardia terminates during ablation at some of these sites is not clear. In some cases, termination may be fortuitous. In other cases, heating may reach a portion of the reentry circuit that is further from the catheter than the tissue that was captured by pacing. Alternatively, radiofrequency current application may occasionally elicit sympathetic or parasympathetic changes that terminate tachycardia.

Outer loop sites. Outer loops are probably paths along the border of an infarct region. Pacing at these sites entrains tachycardia with QRS fusion, but the stimulated antidromic wavefronts are not confined in or near the circuit (Fig. 5). The stimulated wavefront can propagate directly away from the infarct region to depolarize tissue remote from the reentry circuit, altering the QRS configuration. Entrainment occurs with QRS fusion, but in contrast to the remote bystander sites, the postpacing interval matches the tachycardia cycle length. If

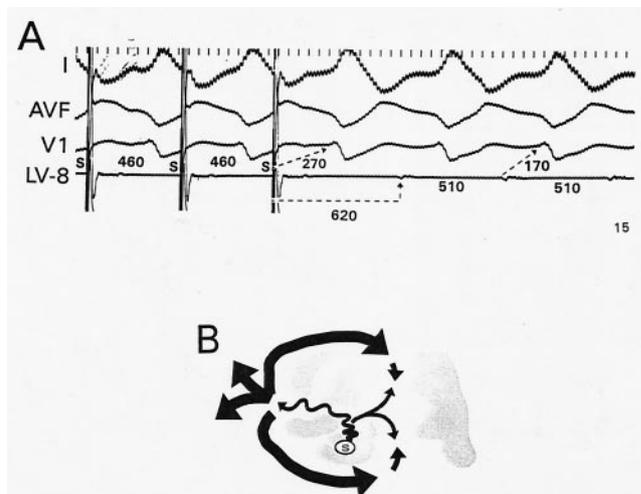


Figure 8. Pacing at an adjacent bystander is shown in data from a patient (A) and in a schematic (B) of the reentry circuit shown in Figure 1A. A, 50-ms time lines, surface ECG leads I, aVF and V₁ and a bipolar recording from the distal electrode pair of the left ventricular mapping catheter at site 8. Sustained monomorphic ventricular tachycardia with a cycle length of 510 ms is present (last 2 beats of the panel). The last three stimuli (S) of a stimulus train at the mapping site are shown. Pacing at a cycle length of 460 ms entrains tachycardia with concealed fusion. In the left ventricular recording (LV-8), the postpacing interval of 620 ms, indicated by the **dashed arrow**, exceeds the tachycardia cycle length by 110 ms, consistent with a bystander site. Also, the S-QRS interval of 270 ms exceeds the electrogram to the QRS interval of 170 ms. B, Pacing is at a “dead-end” bystander in the same reentry circuit as in Figure 1A. The stimulated wavefronts (**gray arrows**) reach the reentry circuit common pathway and then propagate in both directions in the circuit. The antidromic wavefronts collide with returning orthodromic wavefronts and are contained in the chronic infarct. The stimulated orthodromic wavefront entrains the tachycardia with concealed fusion. The postpacing interval is the conduction time from the pacing site to the circuit, through the circuit and back to the pacing site and exceeds the tachycardia cycle length (11).

these sites are located along the border of the infarct region, in continuity with the surrounding myocardium, they may be in a broad path of the reentry circuit. This is consistent with the observation that only 10% of radiofrequency current applications at 116 outer loop sites acutely terminated ventricular tachycardia (Table 2).

In some tachycardias a series of radiofrequency lesions across an outer loop region can eventually terminate tachycardia when narrower, “isthmus” regions of the circuit cannot be identified. Nakagawa et al. (42) reported on two patients who appeared to have an outer loop type of reentry circuit around a posttraumatic scar and a right ventricular outflow tract incision, respectively. Reentry was abolished by transecting the loop with a series of lesions extending from the scar to the anatomic boundary of the outer loop. However, interruption of a postinfarction reentry ventricular tachycardia by ablation in an outer loop region is often difficult.

Reentry circuit exit. Pacing at the reentry circuit exit produces entrainment with concealed fusion (Fig. 6). The stimulated antidromic wavefront collides with a returning

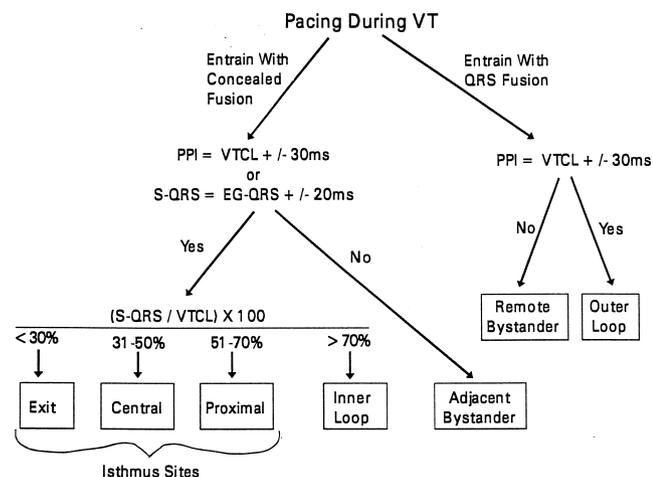


Figure 9. Flowchart of the mapping scheme. If pacing entrains tachycardia with concealed fusion, the postpacing interval (PPI) or S-QRS interval is assessed to determine whether the site is in the reentry circuit. If the site is not in the circuit, it is classified as an *adjacent bystander*. If the site is in the circuit, the S-QRS interval expressed as a percentage of the tachycardia cycle length is used to classify the site relative to the circuit exit. Sites with an S-QRS interval <70% of the ventricular tachycardia cycle length (VTCL) have the highest incidence of tachycardia termination and are referred to collectively as *isthmus sites*. If pacing entrains tachycardia with QRS fusion and the postpacing interval is consistent with a reentry circuit site, the site is classified as an *outer loop*. Sites where pacing entrains tachycardia with QRS fusion and the postpacing interval exceeds the tachycardia cycle length are *remote bystanders*. EQ-QRS = interval from electrogram recorded at the pacing site during ventricular tachycardia to QRS onset. See text for discussion.

orthodromic wavefront and is extinguished in the circuit. The stimulated orthodromic wavefront propagates away from the exit; thus, the QRS configuration during entrainment is the same as that during tachycardia. The postpacing interval matches the tachycardia cycle length. The S-QRS interval during entrainment matches the electrogram QRS interval during tachycardia. At exit sites, the conduction time from the pacing site to the point at which the QRS onset occurs, and thus the S-QRS interval, is relatively short; an arbitrary definition is $\leq 30\%$ of the tachycardia cycle length (e.g., ≤ 100 ms for a tachycardia that has a cycle length of 300 ms) (Fig. 7). Exit sites are desirable targets for ablation. Radiofrequency ablation terminated tachycardia at 37% of 35 exit sites (Table 2).

Central, proximal and inner loop sites. As the pacing site is moved further from the exit, to more proximal sites in the circuit, the S-QRS interval observed during entrainment with concealed fusion increases. Sites where pacing entrains tachycardia with concealed fusion with an S-QRS interval $>30\%$ of the tachycardia cycle length are designated as central, proximal or inner loop sites, depending on the length of the S-QRS interval (Fig. 7 and 9). Radiofrequency ablation is often effective at central and proximal sites (Table 2).

Sites designated inner loop sites have very long S-QRS intervals during entrainment with concealed fusion (Fig. 7).

Ablation at inner loop sites is much less effective; tachycardia terminates at only 9% of these sites. This portion of the reentry circuit may tend to be broad and less susceptible to focal radiofrequency lesions or may be in a loop proximal to the isthmus region. If other potential loops are present, ablation of one loop may not terminate reentry. It is also possible that some of these sites are actually outer loop sites. During pacing at rates slightly faster than the tachycardia, only minimal QRS fusion occurs at some outer loop sites. The S-QRS interval is relatively long, because the pacing site is proximal to most of the slow conduction in the circuit, and the site appears to be in an inner loop based on entrainment. Pacing at a faster rate may demonstrate greater fusion with a short interval between the stimulus and the QRS complex, consistent with rapid propagation of the stimulated excitation wavefronts away from the pacing site, exposing the site as an outer loop site.

Adjacent bystanders. Some bystander sites are sufficiently close to the reentry circuit that pacing entrains tachycardia with concealed fusion. The stimulated antidromic wavefronts are contained in or near the circuit such that they do not alter the ventricular activation sequence (Fig. 8), but the postpacing interval does not approximate the tachycardia cycle length, and the S-QRS interval during entrainment does not match the electrogram QRS interval during tachycardia. It is likely that these sites are in the infarct region, close to the circuit, and they are therefore designated adjacent bystanders. Radiofrequency catheter ablation interrupts tachycardia at 11% of adjacent bystanders (Table 2), suggesting that they are often close to the reentry circuit. In some cases, conduction slowing in the reentry circuit during entrainment may increase the postpacing interval and S-QRS interval such that a site in the circuit falsely appears to be an adjacent bystander.

Isthmus sites in the reentry circuit. Radiofrequency current terminated ventricular tachycardia at 29% of the 85 reentry circuit sites classified as exit, central or proximal on the basis of entrainment (Table 2). The high incidence of termination at these sites relative to other sites suggests that the exit, central and proximal sites are often narrow "isthmuses" in the reentry circuit where the circuit can be interrupted. This is further supported by analysis of the time from the beginning of radiofrequency current application to termination of tachycardia at sites where tachycardia terminated during ablation. The time to termination was shorter at exit, central and proximal sites (10 ± 11 s) than at all other types of sites (19 ± 16 s, $p = 0.005$). The average power during radiofrequency current application was similar at the "isthmus sites" (29 ± 8 W) and nonisthmus sites (28 ± 8 W, $p = 0.54$). Pacing entrained tachycardia from all sites consistent with adequate tissue contact. Thus, the shorter time to termination suggests that interruption of tachycardia occurred after heating a smaller mass of tissue, consistent with an isthmus, whereas termination at other sites may have required heating a greater mass of adjacent tissue. These observations are consistent with the findings of de Bakker et al. (5) and Downar et al. (6) who used extensive activation sequence mapping intraoperatively or in

explanted hearts to characterize reentry circuits. Those investigators identified "return pathways" of the reentry wavefront from which low amplitude, narrow electrograms were recorded, consistent with depolarization of a small mass of tissue. Similar isolated diastolic potentials recorded during catheter mapping of ventricular tachycardia often appear to originate from isthmuses (11,43,44).

Limitations of Entrainment for Mapping

Entrainment methods have many limitations. Entrainment can be applied only if ventricular tachycardia is sufficiently stable to allow pacing during that tachycardia. Rapid tachycardias that produce hemodynamic collapse cannot easily be evaluated. Whether faster tachycardias will have similar features is unknown.

The reentry circuit classification is consistent with available intraoperative mapping data. The terms isthmus, exit, central, proximal, inner loop, outer loop and bystander are useful to indicate constellations of findings during entrainment and provide a conceptual, intuitive framework for relating the mapping findings to reentry and to the likelihood that radiofrequency ablation will interrupt reentry. However, we do not have direct confirmation that the anatomy corresponds to our descriptions. The classifications of reentry circuit sites according to the S-QRS interval during entrainment with concealed fusion are arbitrary and unlikely to indicate precise anatomic distances between the pacing site and edge of the infarct.

Analysis of entrainment, the postpacing interval and the S-QRS interval assumes that pacing does not alter conduction velocities and conduction paths in the reentry circuit (11). Rapid pacing in circuits that are functionally determined can alter the reentry circuit, as shown in canine models of atrial flutter and ventricular tachycardia (22,29,38). Antiarrhythmic drugs, present in the majority of our patients (Table 1), can further decrease conduction velocity during pacing, increasing the postpacing and S-QRS intervals (11). Analysis of the postpacing interval and electrogram QRS interval assumes that the timing of local depolarization can be inferred from the electrogram, but this is subject to error in regions where the signal is fractionated (11,40,45,46).

Clinical Implications

A functional classification of mapping sites using entrainment allows a site by site interrogation of the ventricle without the need for recording from multiple sites simultaneously. These methods are applicable with present mapping technology and are aiding the investigation and ablation of human ventricular reentry circuits. It is likely that a variety of reentry circuit types exist. The susceptibility of these circuits to ablation is determined by the size and location of the reentry path and is likely to vary with circuit type. Types of tachycardias with characteristic locations that are susceptible to catheter ablation may be identified (47).

Appendix

Mapping and Ablation

Data from the first 18 patients have been previously reported (11,15). In the first 15 patients, sites were selected for application of radiofrequency current during ventricular tachycardia if the site had low amplitude and fractionated electrograms and if pacing from the site entrained tachycardia. The mapping site classification was not used to select sites for ablation. When the data in these initial 15 patients were analyzed, several characteristics that identified sites at which application of radiofrequency current interrupted reentry were identified (11). These included entrainment with concealed fusion, a postpacing interval that was within 30 ms of the ventricular tachycardia cycle length, the presence of isolated diastolic potentials during ventricular tachycardia and a stimulus to the QRS interval during entrainment with concealed fusion that is <70% of the tachycardia cycle length. In the next 23 patients, we specifically sought sites where one or more of these features were present. If such sites were absent, radiofrequency current was applied to sites with abnormal presystolic electrograms. All left ventricular programmed stimulation performed for entrainment used "unipolar" stimuli with the distal electrode of the mapping catheter as the cathode and an electrode catheter in the inferior vena cava as the anode to avoid confounding effects of anodal capture during bipolar stimulation (48). Stimuli had an amplitude of 5 to 10 mA and a pulse width of 2 ms, which was increased to 9 ms if consistent capture was not achieved. Radiofrequency current (250 or 500 kHz) was applied between the distal mapping electrode and a cutaneous adhesive electrode at 15 to 45 watts for 20 to 40 s during ventricular tachycardia. If tachycardia terminated, the application was continued for a total of 60 to 120 s or until an increase in impedance or boiling at the electrode tip was observed on transesophageal echocardiographic imaging (49).

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