PATHOPHYSIOLOGY AND NATURAL HISTORY
ARRHYTHMIA

Entrainment of ventricular tachycardia: explanation for surface electrocardiographic phenomena by analysis of electrogams recorded within the tachycardia circuit

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ABSTRACT Transient entrainment was demonstrated during 59 pacing events in 18 episodes of sustained uniform ventricular tachycardia (VT) while recording electrogams from the site of origin of tachycardia (LE-SOO). During entrainment, the morphology of the initial component of the LE-SOO was identical to the morphology observed during the tachycardia in 13 VTs (group I), but in five VTs (group II), the initial component changed at a “critical” paced cycle length. The presence of the proposed surface electrocardiographic criteria for entrainment — fixed fusion and a first postspacing complex without fusion but occurring at the paced cycle length — were integrally dependent on the morphologic changes in the local presystolic electrogram. Fixed fusion of the surface electrocardiogram at one or more paced cycle lengths was detected during entrainment at 35 of 59 paced cycle lengths in 12 of 18 tachycardias, 10 of which were group I and two of which were group II VTs. Fixed fusion demonstrated by analysis of the LE-SOO was observed at one or more pacing cycle lengths in 17 of 18 VTs. In five tachycardias in which surface electrocardiographic fusion was not observed, fixed fusion was evident on analysis of the left ventricular LE-SOO during right ventricular pacing. The first postspacing interval, as measured at the surface electrocardiogram, was consistently equal to the paced cycle length in only one of 18 tachycardias and was greater than the VT cycle length in eight of 17 tachycardias. A pathway with a long conduction time was demonstrated during entrainment. However, in those 12 VTs in patients in whom pacing was performed at more than one cycle length and there was preservation of the LE-SOO morphology, the conduction time between the stimulus and presystolic electrogam remained constant. Thus, no evidence for “atrioventricular nodal-like” decremental conduction was observed over a wide range of pacing cycle lengths. We conclude that: (1) two of the previously proposed criteria for diagnosis of entrainment (fixed fusion on the surface electrocardiogram and a first postspacing interval equal to the paced cycle length) are overly restrictive criteria for definition of “entrainment” of VT, (2) analysis of endocardial recordings from the site of origin of tachycardia during attempted entrainment of VT is useful for documenting the presence of entrainment, and (3) such analysis provides a basis for the understanding of surface electrocardiographic phenomenon associated with entrainment.


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IN MACROSCOPIC models of reentry, such as circus-movement tachycardias incorporating an accessory atrioventricular pathway, the mechanism of transient entrainment has been shown to result from: (1) the premature penetration into the antegrade limb of a reentrant circuit of each stimulated impulse, and (2) collision between the stimulated wavefront and the prior tachycardia (or stimulated) wavefront retrogradely within the circuit. The first entrained beat results in retrograde collision between the stimulated and tachycardia impulse, while in all subsequent beats,
the collision occurs between the presently stimulated wavefront and that stimulated previously. This continuous resetting of the tachycardia circuit by a stimulated wavefront results in activation of those elements of the tachycardia circuit between the entrance and exit at the paced cycle length.1–3

Based on observations during overdrive pacing of supraventricular arrhythmias, Waldo et al.1–3 and Brugada and Wellens4 developed criteria for determining the presence of entrainment based on the surface electrocardiogram. They included: (1) acceleration of the tachycardia to the paced cycle length, (2) fixed fusion of the surface electrocardiogram during pacing at a constant cycle length, and (3) progressive fusion during pacing at shorter cycle lengths. Subsequently, an additional criterion was proposed: lack of fusion at the first nonpaced ventricular complex but fusion at the paced cycle length.5–7

These criteria have also been used for the recognition of entrainment of ventricular tachycardia.5–10 Meeting these criteria was believed to be evidence not only of entrainment but also of a reentrant mechanism of the arrhythmia. However, it has been noted that in many instances fusion was either absent or unrecognized and that the first nonpaced complex occurred at an interval greater than the paced cycle length, yet the basic phenomenon of continuous resetting was present during known reentrant rhythms. The ability to demonstrate surface electrocardiographic fusion and thus fulfill the proposed diagnostic criteria for entrainment depends on enough of the ventricular myocardium being depolarized by both the stimulated and tachycardia wavefronts so that the presence of both wavefronts will be recognized and produce a fusion QRS complex. Since fusion between the stimulated wavefront and that emanating from the tachycardia circuit (the orthodromic wavefront) may be restricted to a small area (after depolarization of a majority of the myocardium by the stimulated impulse), the basic resetting phenomenon of entrainment may not be recognized on the surface electrocardiogram. If, during attempted entrainment, electrograms from the site of “origin” of the tachycardia are recorded and these electrograms occur at the frequency of the paced cycle length with resumption of the identical tachycardia after cessation of pacing, then strong evidence for the presence of entrainment exists regardless of whether or not the surface electrocardiographic criteria are present.

The purpose of this study was to analyze the response of ventricular tachycardia to overdrive pacing while simultaneously recording the endocardial site of tachycardia origin by: (1) evaluating the relationship between the electrocardiographic and endocardial correlates of fusion, (2) evaluating the mechanisms for apparent electrocardiographic fusion, and (3) providing an explanation for why the proposed criterion requiring that a first postpacing interval be equal to the paced cycle length is often absent during entrainment of ventricular tachycardia.

Methods

Patients and tachycardias. Eighteen ventricular tachycardias were evaluated in 16 patients who comprised the study population. All patients had coronary artery disease and a myocardial infarction that occurred in the remote past. The following inclusion criteria were fulfilled by the 18 ventricular tachycardias studied: (1) sustained ventricular tachycardia was reproducibly initiated by programmed stimulation and had a uniform morphology, (2) ventricular tachycardia cycle length was stable (±20 msec change over 20 consecutive beats), (3) ventricular tachycardias were hemodynamically well tolerated, (4) complete endocardial activation mapping of all ventricular tachycardias had been performed with a catheter positioned at the site of origin of tachycardia during the pacing protocol, and (5) entrainment, as defined below, had occurred during each pacing sequence.

For the study of 13 tachycardias, fluoroscopically guided endocardial activation mapping and pacing were performed in the electrophysiologic laboratory (catheter mapping), and for that of the remaining five, intraoperative endocardial mapping was performed as previously described.11–12 Right ventricular pacing was performed at multiple cycle lengths during 10 ventricular tachycardias. Entrainment criteria were evaluated during 59 pacing trials of the 18 ventricular tachycardias. If pacing to entrain the ventricular tachycardia was performed at only one cycle length, this was considered the longest paced cycle length.

Stimulation and recording. Bipolar stimulation and recording was performed from a right ventricular site in all instances by use of the distal electrode pair of a No. 6F quadripolar catheter with an interelectrode distance of 5 mm. The distal electrode was the cathode. To initiate ventricular tachycardia, one to three ventricular extrastimuli and/or rapid pacing was performed as previously described. The pacing stimuli during ventricular tachycardia were delivered in the following fashion: (1) each train of stimuli was delivered at a fixed paced cycle length, (2) each train lasted for at least 20 beats, (3) at each paced cycle length, an incremental number of beats was delivered to ensure that the ventricular tachycardia was not terminated and then reinitiated, (4) pacing was performed at a cycle length shorter than the tachycardia cycle length by at least 20 msec, and (5) if more than one train was delivered, the pacing cycle length was sequentially reduced in 10 to 30 msec decrements. The first impulse of the train was synchronized to the local electrogram at the pacing site during the ventricular tachycardia with a coupling interval equal to the paced cycle length of the stimulus train. A total of 59 trains (3.2 trains per ventricular tachycardia) was delivered, ranging from one to 11 trains per ventricular tachycardia (table 1). At least three surface electrocardiographic leads were recorded, usually I, aVF, and V5 (or alternatively leads I, II, III, and V4R). The local electrogram at the pacing site was recorded from the proximal bipolar pair of a quadripolar catheter (inter-electrode distance of 5 mm). Local electrograms from the site of origin of the ventricular tachycardia were recorded from the distal electrode and a proximal electrode with an interelectrode distance of 10 mm during endocardial mapping in the electrophysiologic laboratory (13 tachycardias). When recordings...
were made intraoperatively (five tachycardias), the bipolar pair of a custom-made ring (2 mm interelectrode distance) or a bipolar pair of a plaque electrode (1 mm interelectrode distance) were used. Local electrograms were filtered between 30 to 40 and 500 Hz. Data obtained during the procedures were displayed on a multichannel oscilloscope (Electronics-For-Medicine VR-16), recorded with an ink-jet recorder (Seimens Elema Mingograf) at a paper speed of 100, 200, or 250 mm/sec, and simultaneously recorded on analog magnetic tape (Honeywell 5600).

**Definitions**

Local electrogram at the site of origin of tachycardia (LE-SOO). The site of earliest presystolic endocardial activity recorded in diastole during activation mapping of ventricular tachycardia was termed the LE-SOO. The presystolic endocardial activity always demonstrated a fixed relationship to the onset of the surface electrocardiogram and by definition always preceded electrical systole as manifest on the surface electrocardiogram.

**Criteria for entrainment.** (1) Demonstration of pacing-induced continuous resetting of the ventricular tachycardia by the presence of a less than fully compensatory pause after cessation of pacing. (2) A transient increase in the rate of all ventricular endocardial recordings, including the LE-SOO to the paced cycle length, with immediate return of the QRS of tachycardia and endocardial recordings to their original morphology, cycle length, and temporal relation to one another on cessation of pacing. (3) No evidence for termination or reinitiation of the ventricular tachycardia. In no instance did incremental pacing during normal sinus rhythm initiate tachycardia. Furthermore, during ventricular tachycardia, the beat-to-beat effect of overdrive pacing (first with three, then with four, and incrementally up to 18 extrastimuli) was evaluated to ensure that no termination had occurred as a result of pacing.

**Fusion**

**Fixed fusion in the surface electrocardiogram.** Fixed fusion in the surface electrocardiogram was said to be present during entrainment if: (1) the surface electrocardiogram was of a constant morphology, representing a hybridization of the QRS morphology of the ventricular tachycardia and that observed during right ventricular apical pacing while the patient was in normal sinus rhythm, or (2) the onset of the surface electrocardiogram preceded the stimulus artifact of each paced beat by a fixed coupling interval.

**Fusion in the LE-SOO.** Fusion observed with the LE-SOO was present when a portion of the ventricular myocardium was activated by the wavefront from the pacing site and the initial component of the LE-SOO was unchanged during pacing and therefore activated by the tachycardia (first entrained interval) or antegradely stimulated wavefront within the circuit (second and all subsequent entrained intervals). Usually, the stimulus artifact occurred after the onset of the LE-SOO, analogous to criteria 2 above.

The first postpacing interval. The interval between the last paced complex and the first nonpaced complex measured at either the surface QRS onset, the pacing site, or the LE-SOO was considered the first postpacing interval.

**Results**

**Morphologic characteristics of the electrogram from site of origin of tachycardia.** The morphologic characteristics of the LE-SOO were evaluated during entrainment. The morphology of the initial component of the local electrogram remained unchanged during entrainment at all paced cycle lengths attempted in 13 (72%) ventricular tachycardias (table 1 and figure 1). These ventricular tachycardias constitute group I. In five ventricular tachycardias, designated group II, a distinct change in the initial component of the LE-SOO was observed during entrainment at one or more paced cycle lengths (figure 2). In four of the five group II ventricular tachycardias, the change in the LE-SOO was rate dependent and was observed only at shorter paced lengths. As shown in table 2, there was no significant difference between group I and group II tachycardias with respect to tachycardia cycle length, the longest or shortest paced cycle length used to entrain the tachycardia, or the longest or shortest paced cycle length used as a percent of the ventricular tachycardia cycle length. However, the LE-SOO was significantly more premature relative to the onset of the QRS in group I than in group II ventricular tachycardias.

**Surface electrocardiographic fusion.** Fixed fusion of ventricular activation as manifest in the surface electrocardiogram was observed during entrainment in 12 of the 18 ventricular tachycardias at one or more paced cycle lengths. Eleven of the 18 ventricular tachycardias were entrained at more than one cycle length. A total of 59 episodes of entrainment were analyzed for fixed fusion of the surface electrocardiogram. Fixed surface electrocardiographic fusion was recognizable in only 35 of 59 (59%) episodes of entrainment. Fixed fusion was more frequently observed during right ventricular pacing in group I than in group II ventricular tachycardia (27/30 or 90% vs 10/29 or 34%, respectively; p < .05).

Fixed fusion was observed in 10 of 13 group I ventricular tachycardias at all paced cycle lengths and two of five group II ventricular tachycardias, but only at long paced cycle lengths. At the shortest pacing cycle length used, all 10 group I ventricular tachycardias that exhibited fixed fusion at long pacing cycle lengths continued to do so while none of the group II ventricular tachycardias exhibited fusion. Surface electrocardiographic fusion was never observed when a change in the initial component of the LE-SOO occurred (figure 3).

In six ventricular tachycardias, three group I and three group II, fusion was not observed at any paced cycle length. Two of the six ventricular tachycardias (one group I and one group II) in which electrocardiographic fusion could not be demonstrated had a left bundle branch block morphology with a leftward and superior axis. The QRS morphology observed during right ventricular pacing in normal sinus rhythm was not significantly different from the tachycardia morphology to allow for recognition of fusion on the surface electrocardiogram. Four of the six tachycardias in which surface electrocardiographic fusion could not be...
TABLE I
Characteristics of tachycardia during entrainment

<table>
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<tr>
<th>VT No.</th>
<th>VT cycle length</th>
<th>No. of paced cycle lengths attempted</th>
<th>Longest paced cycle length</th>
<th>Shortest paced cycle length</th>
<th>Surface ECG fusion</th>
<th>Change in the initial component of the LE-SOO</th>
<th>Stimulus to LE-SOO</th>
<th>First post-pacing interval measured at the surface ECG</th>
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</table>

VT = ventricular tachycardia; ECG = electrocardiogram.

demonstrated during entrainment had a “fully paced” morphology at all paced cycle lengths. At the shortest paced cycle lengths attempted, all group II tachycardias manifested a fully paced QRS morphology.

Fusion of the LE-SOO. Simultaneous activation of ventricles by the stimulated and tachycardia wavefronts was manifest at the level of the LE-SOO during 42 of 59 (71%) pacing events that entrained the 18 ventricular tachycardias (figure 4). Fusion at the level of the LE-SOO was always present when surface electrocardiographic fusion was observed. All 13 group I tachycardias demonstrated fusion of the local electrogram (including the two that demonstrated surface electrocardiographic fusion). However, at the shortest paced cycle length that entrained the tachycardia, no group II tachycardia demonstrated intracardiac or surface electrocardiographic fusion.

Intracardiac fusion was rate dependent. At the shortest paced cycle length attempted, all group II tachycardias demonstrated a change in the initial component of the LE-SOO (figure 2). Despite “complete” capture of the surface QRS (i.e., a fully paced morphology) and change in the LE-SOO, the tachycardia was not terminated.

Relationship of fusion to LE-SOO morphology. The 59 pacing events were evaluated to determine the relationship between surface electrocardiographic fusion and the presence or absence of change in the LE-SOO. Electrocardiographic fusion was only manifest during
35 of 59 (59%) pacing events. As depicted in figure 3, the positive predictive value of a preserved LE-SOO morphology (initial components) to correctly identify those tachycardias during which surface electrocardiographic fusion would be observed was 83%. The ability of a change in the LE-SOO morphology to accurately predict the absence of fusion was 100%.

First postpacing interval. Considering all paced cycle lengths used to entrain the tachycardias, the first postpacing interval was different from the pacing cycle length by at least 20 msec in 18 of 18 ventricular tachycardias (100%) when this interval was measured at the pacing site and different in 17 of 18 VT (95%) when the interval was measured at the onset of the QRS complex. In the absence of a morphologic change in the LE-SOO during entrainment, the first postpacing interval as measured at this presystolic electrogram was always equal to the paced cycle length. When entrainment occurred with concomitant change in the LE-SOO (group II VT at short paced cycle lengths), the first postpacing interval was never equal to the pacing cycle length regardless of the site at which it was measured.

However, regardless of whether there was a change in the LE-SOO morphology during entrainment, the first postpacing QRS complex always exhibited an LE-SOO that was morphologically identical to that observed during ventricular tachycardia and preceded the QRS by an interval identical to that observed during the tachycardia before entrainment.

Conduction time to the LE-SOO during entrainment. In six group I ventricular tachycardias, pacing was performed at multiple cycle lengths. The conduction time from the stimulus artifact to the onset of the LE-SOO was evaluated during entrainment to evaluate cycle length–dependent changes in conduction within the tachycardia circuit. At the longest paced cycle length
attempted, the mean conduction time from stimulus artifact to the LE-SOO was 244 ± 71 msec and it was 243 ± 72 msec at the shortest pacing cycle length attempted (p = NS). Thus, there was no evidence of a cycle length–dependent change in conduction time within the reentrant circuit in any of these group I ventricular tachycardias; i.e., the stimulus to LE-SOO remained constant (figure 5).

In the group II ventricular tachycardias, the conduction time from the stimulus to the onset of the LE-SOO at the longest paced cycle length attempted and the shortest paced cycle length at which there was preservation of the initial components of the LE-SOO was 394 ± 180 and 403 ± 185 msec, respectively (p = NS). However, at the shortest paced cycle length attempted, all five tachycardias exhibited a significant change in the initial component of the LE-SOO morphology compared with its morphology during ventricular tachycardia. Conduction times from the pacing site to the LE-SOO at the longest pacing cycle length were significantly different from those of the shortest pacing cycle length (394 ± 180 vs 93 ± 139 msec, respectively; p < .001).

**Discussion**

Entrainment is the continuous resetting of a tachycardia circuit. Therefore, during constant-rate pacing, entrainment of ventricular tachycardia will result in the activation of all ventricular tissue responsible for maintaining the tachycardia at the paced cycle length, with the resumption of the intrinsic tachycardia morphology and rate after cessation of pacing. Unfortunately, it is virtually impossible to document the acceleration of all tissue responsible for maintaining the reentrant circuit

### TABLE 2

**Influence of cycle length on entrainment**

<table>
<thead>
<tr>
<th></th>
<th>VT cycle length (msec)</th>
<th>Paced cycle length (msec)</th>
<th>Shortest paced cycle length (msec)</th>
<th>Paced cycle length/VT cycle length</th>
<th>LE-SOO to QRS onset during VT (msec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group I</td>
<td>13</td>
<td>352 ± 65&lt;sup&gt;a&lt;/sup&gt;</td>
<td>298 ± 63&lt;sup&gt;a&lt;/sup&gt;</td>
<td>270 ± 72&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.842 ± 0.098&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Group II</td>
<td>5</td>
<td>375 ± 42&lt;sup&gt;a&lt;/sup&gt;</td>
<td>321 ± 38&lt;sup&gt;a&lt;/sup&gt;</td>
<td>278 ± 28&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.825 ± 0.087&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
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</table>

Values are mean ± SD.

For group I vs group II: ^p = NS; ³p < .002.

VT = ventricular tachycardia.
to the paced cycle length. Therefore, a number of surface electrocardiographic criteria have been proposed for establishing the presence of entrainment: (1) constant ventricular fusion at a constant pacing rate,2-4 (2) progressive fusion or different degrees of fusion at different pacing rates,2-4 (3) an interval from the last paced complex to the first nonpaced complex that is less than the ventricular tachycardia cycle length,7 and (4) a first postpacing ventricular complex displaying no fusion but occurring at the paced cycle length.4

Ventricular tachycardia in patients with remote myocardial infarctions is believed to be due to reentry. The reentrant circuit or a portion of the circuit is usually located on the endocardial surface. Endocardial catheter mapping is a well-accepted method of localizing the electrograms within the reentrant circuit.11-13 The presystolic electrical activity seen during tachycardia is assumed to represent recording within the reentrant circuit. In each of the 18 ventricular tachycardias evaluated, presystolic electrical activity was recorded from the reentrant circuit before, during, and after right ventricular pacing. Thus, we were able to document not only the transient acceleration of the majority of ventricular myocardium (reflected in the surface electrocardiogram) to the paced cycle length, but also the acceleration of tissues directly responsible for maintaining the tachycardia (the LE-SOO).

**Morphology of the LE-SOO.** The morphologic characteristics of the LE-SOO were analyzed during ventricular tachycardia to evaluate and explain the proposed criteria for entrainment. The tachycardias were divided into two groups based on the morphology of the LE-SOO during entrainment. The morphology of the initial component of the local electrogram at the site of origin did not change during entrainment in any group I ventricular tachycardia and remained unchanged at long paced cycle lengths in four of five group II tachycardias. However, at short paced cycle lengths, while all group I ventricular tachycardias maintained the morphology of the initial component of the electrogram recorded at the site of origin, all group II ventricular tachycardias exhibited a distinct change in the morphology of this electrogram.

The explanation for this morphologic change may be extrapolated from observations made during transient entrainment of macroreentrant circuits. During ativoventricular reciprocating tachycardia utilizing a left-sided accessory pathway, Waldo et al.6 demonstrated that pacing from the high right atrium could result in entrainment of the tachycardia with preservation of the coronary sinus atrial electrogram morphology. It was demonstrated that this was due to collision of the stimulated wavefront with the activation wavefront of the tachycardia at a point within the atrium after the coronary sinus atrial tissue (which is part of the reentrant circuit) had been depolarized by the activation wavefront of the tachycardia. However, Waldo et al. also demonstrated that at a critical paced cycle length the atrial electrogram recorded by the coronary sinus catheter could be captured by the stimulated wavefront with a resultant change in the atrial electrogram morphology.

Similarly, in all group I tachycardias and most group II tachycardias during entrainment at long paced cycle lengths, the LE-SOO was captured by the normally propagating wavefront of the tachycardia. However, with all group II tachycardias at a shorter paced cycle length, the local electrogram at the site of origin was now captured by the stimulated wavefront of activation (figure 6). We have termed this phenomenon of change in the morphology of the initial component of the electrogram recorded within the reentrant circuit during entrainment “retrograde capture.”

**Requirements for fusion during entrainment.** The presence of retrograde capture during entrainment provides one possible explanation for the absence of surface electrocardiographic fusion, the so-called “hallmark of

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FIGURE 3. A chi-square demonstrating the relationship between a change in the presystolic left ventricular electrogram during entrainment and the presence or absence of surface electrocardiographic fusion during the 59 pacing events. Of particular note is the absence of surface electrocardiographic fusion being correctly predicted (100%) by a change in the morphology of the initial component of the presystolic LE-SOO. Conversely, however, the absence of a change in morphology of the presystolic electrogram recorded at the site of tachycardia origin did not always predict the presence of surface electrocardiographic fusion.

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<tr>
<th>Surface Electrocardiographic Fusion</th>
<th>LE-SOO Morphology During Entrainment</th>
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<td>CHANGE</td>
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Sensitivity 100%
Specificity 71%
(+Predictive Value 83%
(-Predictive Value 100%
entainment.” In order for fusion to be observed on the surface electrocardiogram, there must be collision of the stimulated wavefront and the tachycardia wavefront within the ventricular myocardium at a point outside the reentrant circuit. If the retrogradely stimulated wavefront penetrates into the reentrant circuit and collides with the tachycardia wavefront (or previously stimulated antegrade wavefront) before the point at which the tachycardia wavefront would be exiting to the mass of the myocardium, then no fusion will be evident on the surface electrocardiogram and the surface electrocardiogram will appear entirely paced (figure 7). This is exactly what was observed in all group II ventricular tachycardias at a short paced cycle length. The phenomenon of retrograde capture also explains the rate dependency of fusion. At long paced cycle lengths, the retrogradely stimulated wavefront did not reach the exit of the reentrant circuit before activation of a portion of the ventricular myocardium by the tachycardia wavefront and thus fusion could be observed. However, at shorter paced cycle lengths the retrogradely stimulated wavefront penetrated further into the circuit and to a point proximal to where the tachycardia wavefront exited the circuit, as evidenced by capture of a presystolic electrogram. Therefore, the surface electrocardiogram appeared fully paced. Thus, fusion of the stimulated and tachycardia wavefronts occurred only within and not outside the reentrant circuit.

Fusion restricted to and evidenced only by recordings from the reentrant circuit, as seen in figure 4, may be called “concealed fusion” and could explain a mechanism of “concealed entrainment (entrainment without surface electrocardiographic fusion). Concealed entrainment could also be applied to ventricular tachycardia in which the LE-SOO is captured retrogradely by each stimulated impulse but the tachycardia resumed on cessation of pacing. If the entire circuit could be recorded, an area of fusion (i.e., two wavefronts propagating in opposite directions in one circuit) would be seen.

No fusion was observed on the surface electrocardiograms for five ventricular tachycardias in the absence of retrograde capture. One group I and one group II tachycardia had a QRS morphology identical to that observed with right ventricular apical pacing (a left bundle branch block pattern with a leftward and superior axis). The morphology of these two tachycardias was similar to that observed during right ventricular pacing (during normal sinus rhythm) to allow for identification of fusion. As noted by others, it is usually easier to appreciate surface electrocardiographic fusion when pacing from the ventricle ipsilateral to the tachycardia bundle branch morphology. The remaining three tachycardias appeared “fully paced” during entrainment from the right ventricular apex. It is possible that an insufficient mass of myocardium was depolarized by the tachycardia wavefront to allow for its observation on the surface electrocardiogram or that a less presystolic electrogram recorded from the reentrant circuit would have shown retrograde capture.

FIGURE 4. Surface electrocardiographic leads I, aVF, and V1 and intracardiac recordings from the right ventricular apex (RVA) and from the left ventricular site of tachycardia origin (LV-SO). The initial component of the LE-SOO is identical during ventricular tachycardia and entrainment. Thus, fusion (the simultaneous activation of the ventricle by two wavefronts) is demonstrated by activation of a majority of the ventricular myocardium from the right ventricular apical pacing site (as manifest in the surface electrocardiogram) and by activation of a wavefront originating within the tachycardia circuit. Note that the first postpacing interval is identical to the paced cycle length when measured at the left ventricular site of origin of tachycardia. However, the first postspacing interval as measured at the surface electrocardiogram or at the right ventricular apex is much longer than the paced cycle length.
The first postpacing interval. In preliminary studies, we have previously suggested that fulfillment of the criteria of a first postpacing interval equal to the paced cycle length is not necessary for entrainment of ventricular tachycardia. In the present prospective series we have shown that this criteria has an extremely low sensitivity, seven of 18 (39%), when its occurrence at any paced cycle length is considered. Moreover, in six of seven ventricular tachycardias in which the first postpacing interval as measured at the surface electrocardiogram equaled the paced cycle length, this phenomenon was rate dependent and occurred only at long paced cycle lengths. Thus, a first postpacing interval equal to the paced cycle length was consistently observed only in one of 18 (5%) ventricular tachycardias at all paced cycle lengths producing entrainment.

When fusion is observed during entrainment of ventricular tachycardia, the initial portion of the QRS complex frequently represents activation of the ventricles from the pacing site. Although the terminal portion of the QRS complex reflects activation via the tachycardia circuit, the actual point at which the tachycardia wavefront exits the reentrant circuit and activates a sufficient mass of myocardium to become manifest on the surface electrocardiogram is obscured. If the point within the fused QRS complex that first reflects activation from the tachycardia circuit could be determined, then the time from this point in the last paced complex to the onset of the first nonpaced complex should equal the paced cycle length used to entrain the tachycardia. The degree to which the first postpacing interval as measured on the surface electrocardiogram exceeds the paced cycle length is primarily a reflection of the time during which the ventricles are depolarized by the pacing wavefront before the activation of a significant portion of the myocardium by the tachycardia wavefront.

In the absence of retrograde capture, the first postpacing interval as measured at the LE-SOO is always equal to the paced cycle length. Thus, the absence of a first postpacing interval equal to the paced cycle length as measured on the surface electrocardiogram is not the result of decremental conduction within the circuit.

Pathway with slow conduction. The presence of a pathway with a slow conduction time has been postulated to be a necessary component for maintaining a reentrant circuit. Okumura et al. provided evidence for a pathway with a slow conduction time within the ventricular tachycardia circuit by comparing the conduction time between the stimulus to a presystolic electrogram during entrainment and during pacing in normal sinus rhythm. The much longer conduction time noted during the tachycardia strongly suggests that a pathway with a long conduction time was present. When retrograde capture during entrainment of ventricular tachycardia occurred, it was possible to demonstrate two conduction times from the right ventricle to a site within the reentrant circuit. The conduction time from the stimulus to presystolic electrical activity during entrainment of ventricular tachycardia was compared in the presence and absence of retrograde capture. In group II tachycardia, conduction time in the absence of change in the initial component of the presystolic electrogram was 394 ± 180 msec. With retrograde capture this conduction time was markedly decreased to 92 ± 140 msec (p < .001). Since there is a large discrepancy in the time between the activation of two spatially fixed points within the ventricle, it seems likely that at least one pathway with a long conduction time was present and located between the entrance and exit of the reentrant circuit. This conduction time from stimulus to presystolic electrogram was relatively fixed in response to varying paced cycle lengths. This long conduction time does not prove slow conduction since

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it may also be a reflection of a longer pathway of activation in the antegrade direction. The fixed conduction time of a stimulus to a presystolic electrogram at multiple paced cycle lengths suggests that the tissue through which the impulse with a long conduction time traverses during tachycardia (or entrainment) lacks decremental properties. Direct measurements of conduction velocity are necessary to distinguish slow conduction velocity from relatively normal velocity with a longer path length.

It can be argued that the presystolic electrical activity recorded during ventricular tachycardia merely represents areas of prolonged and/or slow conduction unrelated to the tachycardia circuit — the so-called dead-end pathways. These two hypotheses are diagrammatically represented in figure 8. Our findings that the LE-SOO returns to the same morphology and identical timing relative to the onset of the surface electrocardiogram of the first postspacing ventricular complex and all subsequent beats favors (as depicted in figure 8) a close relationship between the local electrogram and the tachycardia circuit. This local electrogram at the site of origin has to be part of the tachycardia circuit of a “side branch” connected immediately with the circuit and “protected” from the rest of the myocardium.

We believe that overdrive pacing of ventricular tachycardia can be used as a means of distinguishing late sites or dead-end pathways that are unrelated to the tachycardia circuit from early sites that reflect activation within the reentrant circuit by analyzing the morphology and timing of presystolic electrical activity relative to the onset of the surface electrocardiogram on the first postspacing beat after entrainment. During ablative therapy, it is extremely important to confirm the location of an origin of tachycardia, as determined by activation mapping and observation of the resetting or entrainment phenomenon.

**Conclusion.** Entrainment of ventricular tachycardia is the transient increase in the frequency of activation of
FIGURE 7. A tachycardia circuit during entrainment. The bold arrows represent the stimulated wavefront of activation, while the unshaded arrows represent the tachycardia impulse (or previously stimulated impulse). The shaded area within the reentrant circuit reflects the region between the exit and the entrance of the reentrant circuit. If retrograde collision between the stimulated and tachycardia wavefront occurs within the shaded region, then the wavefront from the tachycardia can potentially exit the reentrant circuit to activate a portion of the mass of the myocardium. Under this circumstance, fusion may be evident on the surface electrocardiogram. However, as shown by the figure on the right, if retrograde collision occurs at a point outside the shaded region, the tachycardia wavefront will never be able to exit from the reentrant circuit and thus the surface electrocardiogram during entrainment will always appear fully paced. See text for discussion.

the tissue responsible for maintaining the ventricular tachycardia circuit to the paced cycle length with the resumption of the identical tachycardia after cessation of pacing. Although surface electrocardiographic criteria such as fixed fusion and a first postpacing interval equal to the pacing cycle length have allowed for identification of entrainment, the absence of these criteria does not rule out the presence of entrainment. Presystolic endocardial activity, which presumably is a direct recording from the reentrant circuit (or tissue integrally related to the circuit), has provided an understanding of the surface electrocardiographic phenomena observed during entrainment.

For fusion to be observed on the surface electrocardiogram, the tachycardia and stimulated wavefronts must collide within the reentrant circuit after the tachycardia wavefront has exited from the circuit. If this collision occurs before the exit of the reentrant circuit (as manifest by a change in morphology of the initial component of the presystolic electrogram, i.e., retrograde capture), then the surface electrocardiogram will appear “fully paced.”

The first postpacing interval as measured on the surface electrocardiogram is generally not equal to the paced cycle length. When fusion is seen during entrainment, the initial portion of the QRS complex usually reflects activation of the ventricles by the stimulated

FIGURE 8. The mechanism by which entrainment of ventricular tachycardia will help differentiate early presystolic electrical activity from late diastolic activity that may be unrelated to the tachycardia circuit. The ladder diagrams are similar to those in figure 7. The electrograms in the top panel were recorded from a site unrelated to the tachycardia circuit but because of delayed activation may appear to show presystolic activity. However, because it is unrelated to the tachycardia circuit, activation may occur in a different manner during pacing and during the tachycardia. Therefore, there is no reason for this diastolic electrogram to precede the first nonpaced complex by the same interval as during the tachycardia. On the contrary, if the electrogram recorded is part of the tachycardia circuit it should always precede the first nonpaced complex by the same interval as during the tachycardia. See text for discussion.
wavefront. Therefore, the degree to which the first postspacing interval exceeds the paced cycle length should reflect the period of time during which the ventricles are depolarized by the stimulated wavefront before the activation of any portion of the ventricle by the tachycardia wavefront. Alternatively, decremental conduction within the reentrant circuit could explain a first postspacing interval that exceeds the pacing cycle length.

There was no evidence for decremental conduction within the reentrant circuit, as evidenced by: (1) a constant stimulus to presystolic electrogram interval over a wide range of pacing cycle lengths, and (2) a first postspacing interval as measured at the LE-SOO equal to the paced cycle length (in the absence of retrograde capture). Of note, however, was a dramatic decrease in the interval between the stimulus and the presystolic electrogram when there was change in the initial components of the presystolic electrogram. This would seem to imply that at least two activation pathways exist between the stimulus site and the presystolic electrogram. One pathway has a long conduction time and the other a relatively shorter conduction time. Although a pathway with a long conduction time appears to be present within the reentrant circuit, we found no evidence for atrioventricular node–like decremental properties within this pathway.

References

Entrainment of ventricular tachycardia: explanation for surface electrocardiographic phenomena by analysis of electrograms recorded within the tachycardia circuit.

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