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Stimulation as a Key to Tachycardia Localization and Ablation*

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"Identification and Catheter Ablation of a Zone of Slow Conduction in the Reentrant Circuit of Ventricular Tachycardia in Humans" by Morady and colleagues from the University of Michigan and Fontaine from Paris, appearing in this issue of the Journal (1), represents a major break from previous mapping techniques. In their article the authors declare the primacy of stimulation mapping over activation mapping for specific types of ventricular tachycardia. Their report joins a growing number of reports that emphasize the importance of the results of stimulation as a means of tachycardia localization (2–4). The reader of such reports may well wonder about the origin of these changes in viewpoint and about the universality of the findings. Some of these issues are addressed in this editorial.

Background

Three distinctly different mapping techniques are used clinically for the localization of tachycardias. Although these techniques are applicable to supraventricular as well as ventricular arrhythmias, the latter will be used as the model throughout the present discussion.

Activation mapping. Recordings are made from multiple sites during tachycardia. The earliest depolarization occurring before the inscription of the QRS complex is considered the "site of origin" (5). In most instances, successively later depolarizations occur as the distance from the site of origin increases. Successive depolarizations can be recorded throughout the QRS complex and beyond. For rapid tachycardias there may be no "gap" between the final depolarizations associated with one QRS complex and the earliest depolarizations related to the next QRS complex. The question then arises as to whether there is a continuum between the late potentials recorded under these conditions and the early depolarization; in other words, has an entire reentrant circuit been recorded or do the late depolarizations merely derive from myocardium at a distance from the actual circuit? Spacial relations can sometimes answer this question, but in other instances the dilemma is difficult to resolve. With slow tachycardias there may be relatively long periods of electrical silence during which no depolarizations can be recorded (6). Because it is assumed that most tachycardias are due to reentry, the implication is that the period of electrical silence represents slow conduction through diseased tissue resulting in depolarizations too small to be recorded using conventional clinical techniques (1,6–8). It is this area of the circuit that Morady and colleagues have addressed.

Therapy based on activation mapping has involved surgical or other ablation of the site of earliest recordable depolarization (the "site of origin" as previously described). This has been quite successful in the operating room (9–11). Activation mapping has not proved as successful when used with catheter ablation techniques (12–14). Maps that would be considered almost ideal in the operating room with well circumscribed sites of origin have been ablated only to have the tachycardia return. The larger amount of tissue removed or destroyed by surgical techniques probably accounts for the greater success of surgery. More detailed explanations will appear in subsequent sections.

Sinus rhythm or passive mapping. Electrograms are recorded from multiple sites during sinus rhythm (7,15,16). Delayed, low amplitude or fractionated potentials are sought that may indicate areas of slow conduction that could be part of a reentrant circuit. Such abnormal potentials have also been sought using surface signal-averaging techniques and appear to correlate with the presence of reentrant circuits (17). With operative or catheter mapping, sinus mapping has proved somewhat problematic because abnormal potentials may be recorded in many different sites in some patients and can be entirely absent in others.

Pace mapping and stimulation mapping. The concept is that stimulation at the "site of origin" of a tachycardia should reproduce the QRS configuration precisely (pace map match) in each of the 12 leads of a surface electrocardiogram (ECG) (18). Using conventional pacing amplitudes, approx-

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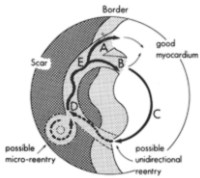

Figure 1. Ventricular tachycardia (schematic). A large area of the ventricle is represented by the **circle**. To the **left** is dense scar represented by the **cross-hatched area** and to the **right** is normal "good" myocardium. The **stippled area** represents a border zone. A, B, C, D and E represent sites of recording and stimulation. See text for details.

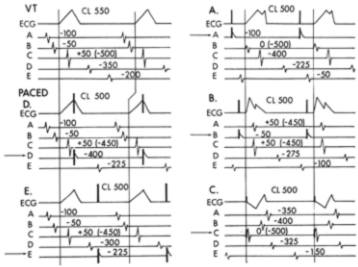

Figure 2. Ladder diagram of ventricular tachycardia (VT) and pacing at sites A, B, C, D and E as described in the text. CL = cycle length in milliseconds; ECG = electrocardiogram.

imate pace map matches can be obtained, often over a wide and imprecise area (19), but 12 lead pace map matches have proved difficult to obtain in most patients. Morady and colleagues (1) have extended the pace map technique through the use of high amplitude stimulation in scarred areas with high stimulation thresholds.

A variation on the pace map technique involves stimulation in an attempt to differentiate early "site of origin" potentials from late potentials that may be irrelevant (20,21). During tachycardia, extrastimuli that are able to enter the circuit and reset the tachycardia should result in early depolarization at the "true" site of origin whereas the timing of irrelevant late depolarizations will be widely scattered. Similar effects will be observed on tachycardia recovery beats after cessation of pacing that failed to interrupt the tachycardia. Again, Morady and colleagues have extended this concept by looking at the sequence of depolarization during pacing in the zone of slow conduction.

Illustrations, Explanations and Questions

Although some of the following illustrations are complex, they may help clarify a variety of issues and concepts.

Figure 1: schematic representation of a large area of a ventricle in a patient with ventricular tachycardia. The hatched area to the left represents dense and inexcitable scar. Normal "good" myocardium is to the far right. The stippled area represents a border zone consisting of a mixture of slowly conducting diseased myocardial fibers interspersed with scar tissue.

This schematic diagram is arranged (with some poetic license) to account for the phenomena observed by Morady and colleagues (1). Area A represents the earliest activation or "site of origin" that would be mapped conventionally. Area B represents a nearby slightly later area of reentry into the good myocardium separated from A by an island of less excitable tissue, somewhat in the form of a river delta. The wave front propagates quickly (smoothly curved arrow) through the good myocardium represented by area C. Two alternative mechanisms are then shown and are represented by dashed lines. After propagating through the myocardium, the wave front could reenter the border and scarred zones, traveling slowly (wavy lines) to areas D and E and finally reentering the good myocardium at areas A and B. An alternative would be an area of microreentry within the scarred area shown at the lower left of the illustration. Note that the slowly conducting tissue from areas D and E forms a channel bounded by inexcitable scar. For the events described by Morady and colleagues, the macroreentrant circuit would have to be unidirectional, without the possibility of retrograde conduction through the scarred area into the good myocardium.

Figure 2: recording and pacing at sites A, B, C, D and E from Figure 1. In Figure 2, conditions depicted in Figure 1 are represented in ladder diagram format. A representative ECG is shown together with recordings from sites A to E. Timings (in milliseconds) are shown relative to the QRS onset, which is identified by the ECG, and by the narrow vertical lines connecting the panels. These sequences are consistent with the observations of Morady and colleagues (1) with unidirectional conduction within the slowly conducting zone. In discussing the details of Figure 2 it is assumed that the macroreentrant scenario from Figure 1 exists. Results would be similar if the microreentrant scenario were used except that pacing at sites A, B and C would not produce the same sequence at sites D and E. The panel at the upper left represents sustained ventricular tachycardia (VT); subsequent panels represent the effects of pacing at sites D, E, A, B and C and are represented in that order. The three

illustrations on the left (panels VT, D and E) all have identical QRS complexes and initial sequences of depolarization. This is in contrast to panels A, B and C to the right, even though panel A represents pacing at the "site of origin" as conventionally determined.

Panel VT. Ventricular tachycardia exists at a cycle length of 550 ms. The "earliest" depolarization is at site A, with a timing of −100 ms with respect to the QRS complex. Area B follows at −50 ms. According to the conventions of Figure 1, the depolarization at area B represents an alternative exit into the good myocardium rather than conduction from site A to site B. Depolarization of the good myocardium proceeds, with site C depolarizing 50 ms after the QRS inscription, equivalent to −500 ms with respect to the subsequent QRS complex. Finally, areas D and E in the slowly conducting zone are depolarized at −350 and −200 ms respectively. Sites D and E could be considered as the truly early sites of depolarization if they could be recorded in each instance. However, electrograms are often difficult to record from areas of dense scar using clinical techniques. Therefore, recordings from areas D and E are shown here but are not critical to the argument or (as shown by Morady and colleagues) to the clinical results.

Panel D: pacing at site D. The pacer stimulus is large, representing the high threshold at this site. The stimulated wave front propagates to site E, requiring 175 ms in contrast to the 150 ms shown in Panel VT, as a result of relative refractoriness in the slowly conducting zone. Compared with panel VT, an additional 25 ms are also required to conduct from site E to site A. The stimulated wave front, having traveled down the channel from D to E, then emerges at sites A and B in the same sequence as for spontaneous ventricular tachycardia, producing an identical ECG and A,B,C sequence.

Panel E: pacing at site E. Again, a high stimulus amplitude is required. The wave front propagates from site E to A,B,C in the same fashion as spontaneous ventricular tachycardia, again producing an identical ECG. As with panel D, there is some slowing of conduction in the circuit. The timing at site D in panel E assumes only anterograde macroreentry in the circuit. The actual timing at site D is irrelevant. Note that the stimulus to QRS interval is markedly prolonged in panels D and E, reflecting stimulation in the slowly conducting zone.

Panel A: pacing at site A. In this example, pacing at A, the conventional site of origin, does not exactly reproduce the ECG configuration during ventricular tachycardia. This is because during ventricular tachycardia the ECG configuration is a fusion due to depolarization of the good myocardium by way of pathways to sites A and B (Fig. 1). Thus, stimulation at site A does not produce the same A,B,C sequence as observed during spontaneous tachycardia. Note that in panels A and B the pacemaker stimulus amplitude is of moderate amplitude, reflecting the intermediate characteristics of the border zone.

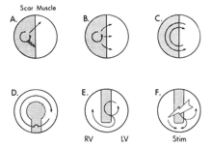

Figure 3. Ventricular tachycardia scenarios. See text for details. LV = left ventricle; RV = right ventricle; Stim = stimulation amplitude.

Panel B: pacing at site B. Virtually equivalent to pacing at site A, the QRS depolarization and the sequence of A,B,C differ from those of spontaneous ventricular tachycardia.

Panel C: pacing at site C. Only a low amplitude stimulus is required to obtain capture at this site in the good myocardium. However, the QRS configuration and sequence of depolarization are entirely different from those observed during spontaneous ventricular tachycardia.

Figure 2: implications. Relatively small lesions at site D or E would interrupt the tachycardia circuit or "pathway" to good myocardium, blocking it with dense scar. A similarly small lesion at site A *or* site B would leave intact the alternative route to the good myocardium. Thus "successful" catheter ablation of site A, the conventional "site of origin," would leave the alternative pathway B intact. Subsequent tachycardias might have a slightly different QRS complex, leading to speculation as to whether the ablation had caused a "new" tachycardia. If sites A and B are relatively close together, a *surgical* resection might be expected to remove both sites A and B, which would be effective in preventing recurrent tachycardia. Because conduction in good myocardium is syncytial, ablation of any type at area C or elsewhere in the good myocardium would not have any significant effect on the arrhythmia.

Nonuniversality of ventricular tachycardia mechanisms and treatments (Fig. 3). The successes achieved with identification and ablation of the zone of slow conduction should not lead to unbridled optimism or enthusiasm. The patients described by Morady and colleagues (1) were highly selected: they had incessant ventricular tachycardia that was relatively slow, well tolerated and of a single configuration. Many patients have rapid, poorly tolerated ventricular tachycardia even in the presence of antiarrhythmic drugs. Patients may have ventricular tachycardia with several configurations and apparent sites of origin (11). The figures in this editorial emphasize reentrant circuits having anatomically defined pathways. Experimental data (8) make it clear

that many reentrant circuits are related to functional arcs of block that can vary in physical position and that may be associated with wave fronts producing a figure eight pattern rather than a conventional circle. Unidirectional conduction within a circuit does not always prevail (11), and it remains uncertain how often high amplitude stimulation in the zone of slow conduction will be able to produce a "pace map match." Some of these issues are addressed in Figure 3.

Figure 3: schematic circuit diagrams. In these figures, the stippled area represents scarred myocardium with a zone of slow conduction assumed to exist within. The clear area represents good myocardium and is labeled "muscle."

Figure 3, panel A. There is a microreentrant circuit within the scar. Several possible pathways to the good myocardium exist, the fastest of them represented by the straight arrow slanting upward and producing a conventional "site of origin." Pacing along the shaft of this straight arrow in the scarred area would reproduce all the conditions outlined by Morady et al. and detailed in Figures 1 and 2. In this instance, however, an alternative pathway from the reentrant circuit to the good myocardium exists that is quite different from the nearby pathways depicted in Figures 1 and 2. Here, the alternative pathway conducts very slowly (indicated by the wavy line) and would reach the good muscle at a site quite remote from the conventional site of origin. Thus, ablation of the more rapid pathway not only would fail to affect the microreentrant circuit but also would permit expression of the slow pathway, resulting in a very different site of origin and tachycardia configuration. Successful ablation of the tachycardia would depend on precise localization of the microreentrant circuit or separate identification and ablation of each pathway.

Figure 3, panel B. Again, a microreentrant circuit exists within the zone of slow conduction, this time with several pathways to the good myocardium having roughly equivalent conduction characteristics. Both a pace map match and a conventional site of origin would be difficult to achieve. As with panel A, precise localization of the microreentrant circuit would be required to achieve successful ablation at a single site. It is not certain whether pacing at such a site would result in a perfect pace map match, given the multiple alternative exit pathways into the good myocardium.

Figure 3, panel C. A macroreentrant circuit exists capable of conduction in either direction. This results in two different sites of origin and two different configurations. Operating room maps make it clear that such scenarios are not rare (11). In this situation, pacing in the zone of slow conduction could result in both anterograde and retrograde conduction in the circuit, resulting in fusion complexes that would differ from either of the spontaneous tachycardia configurations.

Figure 3, panel D. Macroreentry exists throughout a large area of myocardium, with slow conduction through a narrow isthmus of slowly conducting tissue. These circuits could behave much as those described by Morady and colleagues. Some of these circuits are certainly capable of conduction in either direction, resulting in limitations such as those described for panel C. It is our unpublished clinical impression that several alternative pathways may exist through the isthmus in some patients, resulting in limitations somewhat similar to those described for panel B.

Figure 3, panels E and F. In panel E a "mini" macroreentrant circuit is shown involving a scar largely surrounded by good muscle. Such a circuit may exist in a papillary muscle or in the septum, which may be scarred on the left side while good myocardium remains on the right. Using the septal scenario in panel E, the circuit depolarizes the myocardium to the right (the left ventricle) with subsequent conduction from the left to the right ventricle. In panel F the stimulus is of sufficient amplitude to capture both the circuit and the right ventricle. This results in failure to achieve a pace map match. Our unpublished data indicate that increasing stimulation amplitudes at a site on the scarred septum can result in a whole sequence of QRS configurations, representing differing degrees of fusion.

Ventricular Tachycardia Mapping: The State of the Art

The attractiveness of catheter ablation of ventricular tachycardia has focused attention on the need for better mapping techniques. As part of this process, increasing interest has been directed to the zone of slow conduction. The finding that pacing within the zone requires higher than usual pacing stimulus amplitudes, but can result in virtually perfect pace map matches, represents a significant contribution. From an interpretative viewpoint, what then is the state of the art?

Sinus mapping. This can be useful in some instances but is nonspecific.

Activation mapping. This is helpful in most instances but may be nonspecific. As illustrated earlier in this editorial, the conventional concept of a "site of origin" is tenuous. Potentials may be too low in amplitude to permit mapping in the slow conduction zone (1,6–8). Ablation of the "site of origin" may fail to elminate the target tachycardia (12–14). Disappearance of an apparent early potential without termination of the tachycardia is evidence that the apparent site of origin is *not* in fact a critical part of the circuit. However, termination of the tachycardia after disappearance of an early potential is not foolproof evidence that the early potential was a critical part of the circuit.

Pacing and extrasystole-enhanced activation mapping (20,21). If the tachycardia is reset by spontaneous extrasystoles, programmed stimulation or pacing, it may be possible to draw several inferences. If the reset tachycardia resumes with the presumed early potential in its original position, it is indeed likely that this represents an early rather than a very

late potential. However, as illustrated in Figures 1 and 3, demonstration of an early potential does not necessarily indicate that a critical portion of the reentrant circuit has been identified.

Pace mapping. The report by Morady and colleagues (1) suggests that a perfect 12 lead pace map match combined with a prolonged stimulus to QRS interval identifies a narrow conducting pathway surrounded by inexcitable tissue that is used in the production of the spontaneous tachycardia QRS complex and represents a suitable site for an ablation attempt. As shown in Figures 1 and 3, such sites are not necessarily critical portions of the tachycardia circuit. Even when such sites are part of the circuit, bidirectional conduction during pacing may prevent a pace map match.

Pacing during tachycardia may increase the likelihood of achieving a pace map match. Pacing during sinus rhythm may promote bidirectional conduction and failure to achieve a match. Pacing during established tachycardia may produce a match: anterograde conduction would proceed in the direction producing a match; retrograde conduction would not cause changes in the QRS complex if the retrograde wave front collided with the anterograde wave front in the zone of slow conduction. The pace mapping approach in the zone of slow conduction involves the concept of entrainment (22), which can occur with or without fusion depending on circuit characteristics and site of stimulation (23).

The ideal map. Many, perhaps the majority, of patients with ventricular tachycardia do not meet the most stringent criteria for each of the mapping techniques. The most accurate maps combine as many positive components as possible, without producing any evidence that the site in question is irrelevant. Because of the many clinical substrates and tachycardia configurations that prevail, it would be premature to move beyond such general guidelines.

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References

1. Morady F, Frank R, Kou WH, et al. Identification and catheter ablation of a zone of slow conduction in the reentrant circuit of ventricular tachycardia in humans. J Am Coll Cardiol 1988;11:775–82.
2. Stevenson WG, Weiss J, Wiener I, Wohlgelernter D, Yeatman L. Localization of slow conduction in a ventricular tachycardia circuit: implications for catheter ablation. Am Heart J 1987;114:1253–8.
3. Stevenson WG, Rivitz SM, Wiener I, et al. Do fractionated electrograms indicate slow conduction? Evidence from pacing mapping (abstr). Circulation 1987;76(suppl IV):IV-83.
4. Kuck KH, Kentsch M, Geiger M. Pacemapping in patients with ventricular tachycardia: different characteristics during pacing at the "site of origin" (abstr). Circulation 1987;76:IV-413.
5. Josephson ME, Horowitz LN, Farshidi A, Spear JF, Kastor JA, Moore EN. Recurrent sustained ventricular tachycardia. 2. Endocardial mapping. Circulation 1978;57:440–7.
6. Harris L, Downar E, Mickleborough L, Shaikh N, Parson I. Activation sequence of ventricular tachycardia: endocardial and epicardial mapping studies in the human ventricle. J Am Coll Cardiol 1987;10:1001–7.
7. El-Sherif N, Scherlag BJ, Lazzara R, Hope RR. Re-entrant ventricular arrhythmias in the late myocardial infarction period. 1. Conduction characteristics in the infarction zone. Circulation 1977;55:686–702.
8. Mehra R, Zeiler RH, Gough WB, El-Sherif N. Reentrant ventricular arrhythmias in the late myocardial infarction period. 9. Electrophysiologic anatomic correlation of reentrant circuits. Circulation 1983;67:11–24.
9. Mason JW, Stinson EB, Winkle RA. Surgery for ventricular tachycardia: efficacy of left ventricular aneurysm resection compared with operation guided by electrical activation mapping. Circulation 1982;65:1148–55.
10. Josephson ME, Harken AH, Horowitz LN. Long-term results of endocardial resection for sustained ventricular tachycardia in coronary disease patients. Am Heart J 1982;104:51–7.
11. Waspe LE, Brodman R, Kim SG, et al. Activation mapping in patients with coronary artery disease with multiple ventricular tachycardia configurations: occurrence and therapeutic implications of widely separate apparent sites of origin. J Am Coll Cardiol 1985;5:1075–86.
12. Morady F, Scheinman MM, DiCarlo LA. Catheter ablation of ventricular tachycardia with intracardiac shocks: results in thirty-three patients. Circulation 1987;75:1037–49.
13. Benditt D, Camm AJ, El-Sherif N, et al. Catheter ablation for control of ventricular tachycardia: a report of the Percutaneous Cardiac Mapping and Ablation Registry. PACE 1986;9:1391–5.
14. Fisher JD, Brodman R, Waspe LE, Kim SG. Nonsurgical electrical ablation (fulguration) of tachycardias. Circulation 1987;75 (suppl III):III-194–9.
15. Wiener I, Mindich B, Pitchon R. Fragmented endocardial electrical activity in patients with ventricular tachycardia: a new guide to surgical therapy. Am Heart J 1984;107:86–90.
16. Cassidy DM, Vassallo JA, Miller JM, et al. Endocardial catheter mapping in patients in sinus rhythm: relationship to underlying heart disease and ventricular arrhythmias. Circulation 1986;73:645–52.
17. Breithardt G, Borggrefe M. Recent advances in the identification of patients at risk of ventricular tachyarrhythmias: role of ventricular late potentials. Circulation 1987;75:1091–6.
18. Curry PVL, O'Keefe DB, Pitcher D, Sowton E, Deverall PB, Yates AK. Localization of ventricular tachycardia by a new technique—pace-mapping (abstr). Circulation 1979;60(Suppl II):II-25.
19. Josephson ME, Waxman HL, Cain ME, Gardner MJ, Buxton AE. Ventricular activation during ventricular endocardial pacing. II. Role of pace-mapping to localize origin of ventricular tachycardia. Am J Cardiol 1982;50:11–22.
20. El-Sherif N, Gough WB, Restivo M. Reentrant ventricular arrhythmias in the late myocardial infarction period. 14. Mechanisms of resetting, entrainment, acceleration, or termination of reentrant tachycardia by programmed electrical stimulation. PACE 1987;10:341–71.
21. Almendral JM, Stamato NJ, Rosenthal ME, Marchlinski FE, Miller JM, Josephson ME. Resetting response patterns during sustained ventricular tachycardia: relationship to the excitable gap. Circulation 1986;74:722–30.
22. Waldo AL, Henthorn RW, Plumb VJ, MacLean WAH. Demonstration of the mechanisms of transient entrainment and interruption of ventricular tachycardia with rapid atrial pacing. J Am Coll Cardiol 1974;3:422–30.
23. Okumura K, Henthorn RW, Epstein AE, Plumb VJ, Waldo AL. Further observations on transient entrainment: importance of pacing site and properties of the components of the reentry circuit. Circulation 1985;72:1293–307.