The value of catheter mapping during sinus rhythm to localize site of origin of ventricular tachycardia

Dennis M. Cassidy, M.D., Joseph A. Vassallo, M.D., Alfred E. Buxton, M.D., John U. Doherty, M.D., Francis E. Marchlinski, M.D., and Mark E. Josephson, M.D.

ABSTRACT We assessed the value of endocardial catheter mapping in 52 patients in sinus rhythm and with 102 morphologically distinct ventricular tachycardias. The local bipolar electrograms from various regions of the left ventricle were assessed and quantitatively classified with respect to the characteristics of amplitude and duration. With the use of this assessment we found that electrograms from the site of origin were of significantly lower amplitude and longer duration; however, because such an overlap occurred with electrograms that were not from sites of origin, this does not serve as a useful clinical marker. Various types of electrograms, including normal, abnormal, fractionated, abnormal late, fractionated late, and longest, were evaluated with respect to sensitivity, specificity, and positive predictive value. None of these types possessed the ability to reliably localize the site of origin of ventricular tachycardia. We therefore conclude that endocardial catheter mapping during sinus rhythm is not useful as a guide in localized surgical therapy of ventricular tachycardia. Surgery guided only by the results of mapping during sinus rhythm would result in a more extensive excision than that directed by maps obtained during ventricular tachycardia and in some cases would result in the exclusion of the area considered to be the site of origin of the tachycardia.

Circulation 69, No. 6, 1103-1110, 1984.

THE MANAGEMENT of ventricular tachycardia has been improved with the application of new surgical techniques.1 Paramount to the success of surgery is the ability to localize the site of origin of ventricular tachycardia by endocardial mapping procedures.2,3 Mapping during ventricular tachycardia has been accomplished both before surgery in the catheterization laboratory and during surgery. It is not, however, possible to map all morphologically distinct tachycardias in all patients. This is related either to an inability to induce ventricular tachycardia or an inducible ventricular tachycardia that is not tolerated hemodynamically. This limitation could be overcome if endocardial maps obtained during sinus rhythm could supply information that would be of predictive value in localizing the site of origin of ventricular tachycardia. Although such an approach has been suggested,4,5 no data has been available to test the usefulness of such techniques. Our study was therefore undertaken to critically evaluate the role of endocardial mapping during sinus rhythm to localize the site of origin of ventricular tachycardia. The specific aims were (1) to quantitatively assess the amplitude and duration of local left ventricular endocardial electrograms in a population of patients with coronary artery disease and ventricular tachycardias, and (2) to determine if these or any other qualitative electrographic property possessed characteristics that could localize the site of origin of ventricular tachycardia.

Methods

Patient population. Fifty-two patients referred to the hospital of the University of Pennsylvania for electrophysiologic evaluation of ventricular tachycardia underwent sinus rhythm endocardial catheter mapping during the course of their studies.
There were 45 men and seven women with a mean age of 58 years (range 34 to 75).

All patients had coronary artery disease and previous histories of myocardial infarction. The electrocardiographic pattern of infarction was anterior in 38, inferior in eight, and mixed in six. In all the patients coronary artery disease was assessed by coronary angiography and left ventriculography. All patients had left ventricular aneurysms and reduced left ventricular function as determined by the finding of depressed ejection fraction (mean 29%, range 12% to 45%). All patients had histories and documentation of recurrent sustained ventricular tachycardia.

Electrophysiologic study. Studies were performed in patients in nonsonedated, postabsorptive state after informed written consent had been obtained. Thirty-three patients were not on any antiarrhythmic therapy, while the remaining 19 were on one antiarrhythmic drug each. The patients who were receiving drugs underwent mapping both while in sinus rhythm and during ventricular tachycardia while on the same antiarrhythmic drug. Whenever possible the two maps were obtained with the catheter at the same site; this was accomplished by stopping and starting the ventricular tachycardia. In other patients the map during sinus rhythm was obtained first, followed by the map during ventricular tachycardia. If more than one morphologically distinct ventricular tachycardia was induced it was also mapped.

One quadrupolar catheter (No. 6F USCI) was inserted percutaneously into the femoral artery and advanced to the left ventricle of each patient under fluoroscopic guidance. One to two quadrupolar catheters were inserted percutaneously and were advanced to the right ventricular apex and right ventricular outflow tract. Catheters had a 5 mm interelectrode distance. Heparin, 5000 units as a bolus, followed by 1000 units/hr, was administered after insertion of the arterial catheter in each of the patients.

The left ventricular mapping scheme used is illustrated in figure 1. The 12 sites represent segmental areas of the heart of approximately 5 to 10 cm². Eight to 32 electrograms were recorded in each patient. The catheter sites were independently verified by multiple-plane fluoroscopy in the presence of two experienced physicians familiar with this mapping scheme. The catheter was repositioned to another site to confirm reproducibility of recordings and only minimal morphologic differences between local electrograms were noted. Stability was ensured by recording from each site for a minimum of 5 to 30 sec. Electrograms were recorded with the use of a 10 mm interelectrode distance. The intracardiac electrograms were recorded at variable gain to achieve the best electrographic definition and were accompanied by a 1 mV calibration signal. A 10 mm bipolar fixed-gain signal was also recorded at a 1 cm/mV amplification at each site. Electrograms were filtered at 30 to 500 Hz. Intracardiac recordings were simultaneously displayed on a multichannel oscilloscope (Electronics for Medicine VR16) and were stored on analog magnetic tape (Honeywell 5600) and recorded on a 16-channel Mingograf (Siemens Ælema) at a paper speed of 200 mm/sec.

Definitions. Electrographic amplitude (in mV) was defined as the peak-to-peak deflection measured in the 10 mm variable-gain bipolar electrogram.

Electrographic duration (in msec) was defined as the time from the earliest electrical activity that deviated from a stable baseline to the onset of the amplification signal decay artifact (an artifact caused by electronic decay of an amplified filtered signal) measured in the 10 mm fixed-gain bipolar electrogram (figure 2). The amplification and duration measurements were combined to give an amplitude/duration ratio to allow equal emphasis to be placed on each of these values.

Local activation time at a site was defined as the time from the onset of the surface QRS to the time at which the largest rapid deflection of the local electrogram crossed the baseline. This measurement was obtained from the 10 mm variable-gain bipolar electrogram.

Electrograms were defined as normal based on the measurements obtained from 112 electrograms in patients with normal left ventricles. The normal values represent those from 95% of all electrograms obtained in these patients. Normal amplitude was defined as one of 3 mV or more and normal duration was defined as one of 70 msec or less. The normal amplitude/duration ratio was defined as 0.046 or greater. In cases in which only one of the amplitude or duration measurements were abnormal, the amplitude/duration ratio was used to define normality.

Electrograms failing to satisfy the above criteria for normality were defined as abnormal. Basic statistical methods were then used to define the mean and SD values for amplitude, duration, and amplitude/duration ratio for the electrograms of this population.

The electrograms that were the most abnormal were defined as fractionated and were characterized by low amplitude, high frequency, multicomponent signals of prolonged duration. This group was quantitatively defined as consisting of those abnormal electrograms that were greater than 1 SD from the mean values of the abnormal electrograms as a group. Again, if only one parameter of amplitude or duration met the criteria for fractionation, then the amplitude/duration ratio was used to define fractionation.

Late electrograms were defined as those in which a component was recorded after the inscription of the latest surface QRS activity. This was a timing characteristic and hence it could be applied to any of the three types of electrograms described above.
patients had only one morphology, 17 had two morphologies, 13 had three morphologies, one had four morphologies, and one patient had five ventricular tachycardia morphologies. The total number of electrograms available for analysis was 546 (10.5 per patient). Of these electrograms, 102 were from sites of origin while the remaining 444 were from non-sites of origin. The number of electrograms that met the normal criteria was 234 (42%); the remaining 312 (58%) electrograms were considered abnormal.

Abnormal electrograms — quantative analysis. The mean abnormal electrographic amplitude was 1.4 ± 0.9 mV and the mean abnormal electrogram duration was 93 ± 40 msec. The mean abnormal electrographic amplitude/duration ratio was 0.017 ± 0.012.

The generation of these data allowed a quantitative definition of fractionated electrograms. The quantitative criteria used to define fractionated electrograms based on values 1 SD from the mean became an amplitude of 0.5 mV or less, a duration of 133 msec more, and/or an amplitude/duration ratio 0.05 less.

Classification of electrograms. A distribution of each type of electrogram is seen in table 2. Of the 312 abnormal electrograms, 47 were fractionated by the described criteria. This represents 9% of all electrograms. Eighty of the abnormal electrograms were classified as late, representing 15% of all electrograms and 26 (5%) were considered both fractionated and late. Examples of the various types of electrograms are shown in figure 2.

Analysis of sites of origin. Results of the quantitative comparative analysis of site of origin and non-site of origin is shown in table 3. The sites of origin had lower amplitudes (2.0 ± 2 vs 3.6 ± 2.8 mV; p < .001), longer durations (88 ± 33 vs 75 ± 27 msec; p <

TABLE 1

<table>
<thead>
<tr>
<th>Definition of sensitivity, specificity, and positive predictive value</th>
</tr>
</thead>
<tbody>
<tr>
<td>True positive = Electrographic type at site of origin</td>
</tr>
<tr>
<td>False positive = Electrogram type, not site of origin</td>
</tr>
<tr>
<td>True negative = Nonelectrogram type, not site of origin</td>
</tr>
<tr>
<td>False negative = Nonelectrogram type at site of origin</td>
</tr>
</tbody>
</table>

Sensitivity = True positive / (True positive + False negative)
Specificity = True negative / (True negative + False positive)
Positive predictive value = True positive / (True positive + False positive)

aDefined by one of the following: normal, abnormal, fractionated, abnormal late, fractionated late, or longest.

bDefined by all electrograms not exhibiting particular characteristic being analyzed.
of normal electrograms was 6%, and for abnormal electrograms it was 29%. Fractionated electrograms only predicted site of origin 21% of the time. The characteristic of lateness added little to the predictive value; abnormal late electrograms had a 33% predictive value while fractionated late electrograms positively predicted the origin of tachycardia 31% of the time. Similarly, the longest electrogram had a positive predictive value of only 31%. Therefore, no electrographic type manifested a good positive predictive value.

**Single ventricular tachycardia morphology analysis.** A separate analysis was performed on data from the 20 patients who had only one distinct ventricular tachycardia morphology. These results are summarized in table 5. The distribution and value of each of the types of electrograms were similar to those in the entire population. Abnormal electrograms were fairly sensitive (80%) but were not specific (65%) and were of poor positive predictive value (21%) for the site of origin of ventricular tachycardia. The only difference noted in this analysis was the lack of normal electrograms at the site of origin of ventricular tachycardia. This most likely reflects the fewer number of morphologically distinct ventricular tachycardias analyzed (20 vs 82).

An analog map from one patient is shown in figure 3. This map shows the dispersion of various types of electrograms throughout the entire endocardial surface, regardless of site of origin.

**Discussion**

Mapping during ventricular tachycardia has been established to be both safe and useful; however, it may be associated with several potential problems. The time required to map during ventricular tachycardia can result in unfavorable hemodynamics. Some ventricular tachycardias lead to rapid hemodynamic collapse and patients with these tachycardias are not suitable candidates for endocardial mapping. Occasionally tachycardias are totally noninducible, are of different morphologies than spontaneously documented ventricular tachycardias, or have rapidly changing morphologies that make them unsuitable for activation mapping. Finally, operative mapping leads to increased cardiopulmonary bypass time and its inherent problems. It would therefore be beneficial to be able to predict the site of origin of ventricular tachycardia from mapping during sinus rhythm.

It has been demonstrated with both epicardial and endocardial mapping during sinus rhythm that patients with recurrent sustained ventricular tachycardia have a
variety of abnormal electrographic findings that are not present in patients without this arrhythmia. This may well define an electrophysiologic substrate for ventricular tachycardia. However, the mere presence of such electrograms does not allow the conclusion that mapping during sinus rhythm can be used to guide surgical therapy of ventricular tachycardia. This hypothesis can only be proved if such electrograms are shown to be sensitive, specific and, most importantly, to possess positive predictive value for the site of origin of ventricular tachycardia. The goal of this study was to evaluate the hypothesis that endocardial mapping during sinus rhythm in patients is useful in guiding localized surgical therapy of ventricular tachycardia.

**Classification of electrograms.** The literature dealing with mapping techniques is confusing with regard to electrographic descriptions. The confusion has arisen because of a lack of a standardization with regard to amplifier and filter settings and interelectrode distance of recording apparatus and most importantly because of a lack of quantitative electrographic description of characteristics of amplitude and duration. This last problem can be partially attributed to the fact that there has been no quantitative description of bipolar electrographic characteristics of normal left ventricles.

In a prior study we defined quantitative standards for normal bipolar electrograms based on the findings in 10 subjects with normal left ventricles from whom 112 bipolar electrograms were obtained for analysis. The description of abnormal electrograms is the result of comparison with this data base.

Of the electrograms recorded in patients in sinus rhythm, the ones that are fractionated are the most abnormal. Only recently have microelectrode techniques been used in attempts to define fractionated activity. The qualitative description that is in general agreement with results of clinical mapping is that of an electrogram with high frequency, low amplitude, multicomponent signals of prolonged duration. The quantitative descriptions in the literature, however, have varied from none to amplitudes less than 1 mV and durations greater than 50 msec, without any details about how these values were obtained. Our definition of fractionation encompasses all previous definitions and is in fact more rigid. We believed that the significance of this type of electrogram would be best judged by analyzing the most abnormal (more than 1 SD from the mean) of the abnormal electrograms. To make sure these criteria were not too rigid abnormal electrograms were also analyzed as a group.

Electrograms having late activation or exhibiting

---

**TABLE 4**

Sensitivity, specificity, and positive predictive value of each electrogram type (entire population)

<table>
<thead>
<tr>
<th>Electrogram type</th>
<th>Sensitivity (for site of origin)</th>
<th>Specificity (for site other than site of origin)</th>
<th>Positive predictive value (for site of origin of electrogram type)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>14/102 (14%)</td>
<td>224/444 (50%)</td>
<td>14/234 (6%)</td>
</tr>
<tr>
<td>Abnormal</td>
<td>88/102 (86%)</td>
<td>220/444 (50%)</td>
<td>88/312 (29%)</td>
</tr>
<tr>
<td>Fractionated</td>
<td>10/102 (10%)</td>
<td>407/444 (92%)</td>
<td>10/47 (21%)</td>
</tr>
<tr>
<td>Abnormal late</td>
<td>26/102 (26%)</td>
<td>390/444 (88%)</td>
<td>26/80 (33%)</td>
</tr>
<tr>
<td>Fractionated late</td>
<td>8/102 (8%)</td>
<td>426/444 (96%)</td>
<td>8/26 (31%)</td>
</tr>
<tr>
<td>Longest</td>
<td>11/102 (16%)</td>
<td>408/444 (92%)</td>
<td>16/52 (31%)</td>
</tr>
</tbody>
</table>

---

**TABLE 5**

Analysis of electrographic distribution, sensitivity, specificity, and positive predictive value in patients with one morphologically distinct ventricular tachycardia

<table>
<thead>
<tr>
<th>Electrogram type</th>
<th>Number (%)</th>
<th>Sensitivity (for site of origin)</th>
<th>Specificity (for site other than site of origin)</th>
<th>Positive predictive value (for site of origin of electrogram type)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>97 (49%)</td>
<td>0/20 (0%)</td>
<td>80/177 (45%)</td>
<td>0/97 (0%)</td>
</tr>
<tr>
<td>Abnormal</td>
<td>78 (51%)</td>
<td>16/20 (80%)</td>
<td>115/177 (65%)</td>
<td>16/78 (21%)</td>
</tr>
<tr>
<td>Fractionated</td>
<td>22 (11%)</td>
<td>4/20 (20%)</td>
<td>159/177 (90%)</td>
<td>4/22 (18%)</td>
</tr>
<tr>
<td>Abnormal late</td>
<td>13 (7%)</td>
<td>4/20 (20%)</td>
<td>168/177 (95%)</td>
<td>4/13 (31%)</td>
</tr>
<tr>
<td>Fractionated late</td>
<td>12 (6%)</td>
<td>2/20 (10%)</td>
<td>167/177 (94%)</td>
<td>2/12 (17%)</td>
</tr>
<tr>
<td>Longest</td>
<td>20 (10%)</td>
<td>2/20 (10%)</td>
<td>159/177 (90%)</td>
<td>2/20 (10%)</td>
</tr>
</tbody>
</table>
late potentials are believed by some investigators to be significant.5,16–18 Previous work from our laboratory has demonstrated that normal endocardial bipolar electrograms do not extend beyond the first 50% of the surface QRS complex.6 We therefore elected to define as late any electrogram that extended beyond the surface QRS complex. Again the intent was to best determine the significance of this finding.

The last type of electrogram analyzed was the longest electrogram recorded by endocardial mapping. Long electrograms may represent slow conduction, which is one of the requirements for reentry and reentry is presumed to be the mechanism of sustained ventricular tachycardia. It is conceivable that the site of origin exists near this area, but an obvious limitation exists in the use of this electrographic characteristic in patients who have tachycardias of more than one morphologic type originating from nonadjacent sites.

Another electrogram reported to be a useful marker for ventricular tachycardia is the “split” or “double potential” electrogram.3,5,17,18 This electrographic finding was observed so rarely during endocardial catheter mapping that it was not suitable for use in the analysis. This may be a function of the interelectrode distance used in this investigation or of the number of sampling sites available.

Analysis of sites of origin. The electrograms from the sites of origin were characterized by lower amplitude and longer duration. However, considerable overlap existed with the other electrograms so that this finding alone would not be helpful in distinguishing between recordings from sites of origin and those from other sites.

The electrograms from the sites of origin were generally abnormal (86%). This is in accord with what previous investigators have characterized the left ventricular electrograms in this patient population.4,9

Two findings were striking in the analysis of the site of origin. First, a completely normal electrogram was obtained from the site of origin 14% of the time (14 patients, 16 morphologically distinct ventricular tachycardias). This finding is not surprising since ventricular tachycardia can arise from normal left ventricles. Electrograms recorded from these normal left ventricles show no difference between the site of origin vs non-site of origin.9 The second important finding was that the electrograms of the left ventricular endocardium that were not from sites of origin were abnormal 50% of the time. These two facts have to be considered if the application of localized directed surgery is being considered.

Predictive value. Mapping in patients during sinus rhythm, if it is to be clinically useful, must be able to aid in the localization of the site of origin of ventricular tachycardia. Although abnormal electrograms were sensitive (86%) they lacked specificity (50%). The various other types of abnormal electrograms had higher specificity but very low sensitivity. None of the types of electrograms demonstrated a positive predictive value of greater than 33% and an attempt to combine the various types would still fail to give a positive predictive value of greater than 40%.

There are a number of possible explanations for the fact that the mapping, during sinus rhythm, of endocardial electrograms does not predict site of origin of ventricular tachycardia. Our patient population had depressed left ventricular function (mean ejection fraction of <30%) with diffuse myocardial disease,

which typifies patients with recurrent sustained ventricular tachycardia. The electrograms recorded, regardless of type, may simply reflect this diffuse myocardial disease. The size and depth of the actual anatomical circuit has not been ascertained with the currently available techniques. Recordings obtained during endocardial catheter mapping may reflect activity outside this circuit, thereby rendering such information less useful. Finally, conduction velocity and activation patterns during sinus rhythm at the recorded site of origin of ventricular tachycardia may not be linear, so that the assumption that such electrograms are a marker of the slow conduction necessary for reentry may not be valid.

Limitations. A major limitation of this study is related to the number of points sampled. Although a maximum of 12 sites were used for this analysis, these values were derived from up to 32 samples in some patients. This, however, may still not be enough. It is conceivable that if a much larger sample could be obtained the results would be different. However, a limited number of points can be sampled with endocardial catheter mapping during sinus rhythm, and it is this method that must be used clinically. Also, preliminary data obtained with intraoperative mapping under direct visualization support our findings.

A second limitation related to the technique of catheter mapping involves precise localization of the catheter position, which can be difficult. Each location was confirmed in at least three fluoroscopic positions in the presence of two independent physicians experienced with the endocardial mapping scheme. When possible the catheter was maintained in the same position for recording of the sinus rhythm and ventricular tachycardia electrograms; this was accomplished by starting and stopping the tachycardia. Otherwise the catheter was repositioned each time to the area from which a recording had previously been made. An earlier study from this laboratory showed a good correlation between results of catheter and operative mapping, suggesting reasonable reproducibility of this technique. Finally, when more than one electrogram was obtained from an area the average value was used in the analysis. We therefore believe that adherence to these guidelines has minimized the chance of an error that could result from catheter mispositioning.

It might be argued that the criteria selected for some of the types of electrogram were too rigid, but if significant differences are to be found, they should be found with the use of the most abnormal electrograms. It should be noted that if intermediate criteria had been chosen some gain in sensitivity would have resulted, but at the expense of specificity and with no significant improvement in positive predictive value.

This analysis was performed on a selected population. All patients had coronary artery disease and left ventricular aneurysms. The application of these results to other patient populations with ventricular tachycardia may not be justified.

We conclude that surgical therapy of ventricular tachycardia based on results of endocardial catheter mapping during sinus rhythm would lead to excision of a larger area of endocardium than that based on results of mapping during ventricular tachycardia. In addition, not all sites of origin would be excised if only those showing abnormality on the map during sinus rhythm were selectively removed.

References
Cassidy et al.

WM: Techniques of intraoperative electrophysiologic mapping. Am J Cardiol 49: 221, 1982


The value of catheter mapping during sinus rhythm to localize site of origin of ventricular tachycardia.

D M Cassidy, J A Vassallo, A E Buxton, J U Doherty, F E Marchlinski and M E Josephson

Circulation. 1984;69:1103-1110
doi: 10.1161/01.CIR.69.6.1103

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/69/6/1103