Intraoperative endocardial mapping during sinus rhythm: relationship to site of origin of ventricular tachycardia

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ABSTRACT Mapping-guided endocardial resection has proved to be an effective therapy for recurrent sustained ventricular tachycardia. However, some patients cannot be mapped during ventricular tachycardia, so that guidance from findings during normal sinus rhythm would be highly desirable. We examined the frequency, timing, and duration of several abnormal types of electrograms recorded endocardially during sinus rhythm and related these findings to activation mapping during sustained ventricular tachycardia. Thirteen patients with extensive myocardial infarction complicated by recurrent sustained ventricular tachycardia were studied intraoperatively during sinus rhythm and induced ventricular tachycardia with a standardized mapping scheme involving the entire endocardial surface. Fractionated electrograms (multicomponent with amplitude <1 mV and duration >50 msec) were recorded in all patients. This type of electrogram could be recorded at up to 36% of mapped sites. Split electrograms (two components separated by isoelectric period) were also frequently seen but involved only a mean of 5.8% of mapped sites. Late electrograms (inscribed entirely after the QRS complex) were only recorded in four of 13 patients at a mean of 5% of mapped sites. The location of these electrograms was related to an arbitrary 8 cm² zone around the earliest site of endocardial activation recorded during ventricular tachycardia. The longest fractionated electrogram was closely related to nine of 22 morphologies of induced ventricular tachycardia, split electrograms were related to seven of 16 morphologies, and late electrograms to two of four morphologies. We have concluded that extremely abnormal electrograms recorded endocardially during sinus rhythm are widespread in patients with extensive myocardial infarction complicated by ventricular tachycardia. These electrograms may be associated with, but are not specific for, sites of origin of ventricular tachycardia. Surgical procedures based on sinus rhythm mapping of these electrogram types would likely result in more extensive surgical excision than those guided by endocardial activation during ventricular tachycardia.


PREOPERATIVE and intraoperative mapping of endocardial activation of ventricular tachycardia has been used to guide subendocardial resection in patients with refractory ventricular tachycardia.1–3 In some patients, however, tachycardias cannot be mapped as a result of such factors as cycle length of tachycardia, hemodynamic embarrassment, changing morphology of tachycardia, or failure to induce tachycardia. Fractionated and other abnormal electrograms recorded from the endocardium have been noted in patients with ventricular tachycardia4–7 and have been suggested as markers for the substrate of reentry. If these abnormal electrograms were reliably associated with the site of earliest endocardial activation during ventricular tachycardia, mapping during sinus rhythm could be used to guide surgical therapy. In the present study we evaluated the characteristics of abnormal endocardial electrograms measured in sinus rhythm during surgery for refractory ventricular tachycardia, and related the findings to the earliest site of endocardial activation during induced ventricular tachycardia.

Methods

The clinical characteristics of the study population are listed in table 1. There were eight men and five women with a mean...
TABLE 1
Clinical characteristics of patients (n = 13; eight men and five women)

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean age</td>
<td>52 years (range 37–61)</td>
</tr>
<tr>
<td>Ischemic heart disease</td>
<td>13</td>
</tr>
<tr>
<td>Vessels &gt;70% occluded</td>
<td>1.8 ± 0.8</td>
</tr>
<tr>
<td>Aneurysm</td>
<td>13</td>
</tr>
<tr>
<td>Mean ejection fraction</td>
<td>0.29 ± 0.06</td>
</tr>
<tr>
<td>Surgery</td>
<td></td>
</tr>
<tr>
<td>Endocardial resection</td>
<td>13</td>
</tr>
<tr>
<td>Aneurysmectomy</td>
<td>13</td>
</tr>
<tr>
<td>CABG</td>
<td>10</td>
</tr>
</tbody>
</table>

CABG = coronary artery bypass graft.

Age of 52 years. All patients had ischemic heart disease with a history of distant myocardial infarction. A mean of 1.8 coronary vessels had at least a 70% obstructive lesion. Each patient had angiographic evidence of anterior left ventricular aneurysm. The mean ejection fraction was 29%. Seven patients had intraventricular conduction delay on their electrocardiograms. No patient had a bundle branch block pattern. The indication for surgery was medically refractory, sustained ventricular tachycardia in all patients. Each underwent subendocardial resection and aneurysmectomy and 10 underwent coronary bypass grafting.

Endocardial mapping was performed, under normothermic conditions and while the patient was on cardiopulmonary bypass, through a ventriculotomy using a fingertip bipolar electrode with an interelectrode distance of 1.5 mm. Signals were filtered at 40 to 500 Hz. Electrograms from each site were recorded simultaneously with surface leads I, F, and V_{5R} or V_{6}, as well as with right and left ventricular reference electrograms. A 1 mV calibration signal was recorded at each site during sinus rhythm and all recordings were obtained at a paper speed of 200 mm/sec on an ink-jet recorder (Elma Mingograph). The moment of activation at each site was taken as the point at which the largest rapid deflection crossed the baseline, or at sites at which no rapid deflection was present as the earliest deviation from the baseline at that site. After the endocardial sinus map was obtained, induction of tachycardia was carried out with use of programmed ventricular extrastimuli delivered through a right ventricular endocardial electrode catheter positioned before surgery at the right ventricular apex. Mapping of the morphology of each induced ventricular tachycardia was done at the same endocardial site as was used for the sinus rhythm recording. The earliest sites of endocardial activation during ventricular tachycardia was defined as the earliest electrical activation occurring before the surface QRS in the last half of electrical diastole.

Electrograms were recorded during sinus rhythm and during ventricular tachycardia by the scheme shown in figure 1A and figure 1, B and C. Electrograms were recorded sequentially at each of 12 ‘clock’ sites, starting at the palpable border of the aneurysm. This pattern of recording was repeated at each of up to five rows spaced approximately 1 cm apart, which are represented in figure 1A as a series of planes cutting through the left ventricle from apex to base.

The electrograms recorded in patients in sinus rhythm were classified into three types and are illustrated in figure 2. Fractionated electrograms were arbitrarily defined as high-frequency, multicomponent signals with an amplitude of less than 1 mV and a duration greater than or equal to 50 msec. Fractionated electrograms outlasting the end of the QRS complex were designated as delayed. Split potentials were defined as two-component electrograms with isoelectric periods between the components of at least 30 msec. Late potentials were defined as electrograms inscribed entirely after the end of the QRS complex recorded on the body surface, either alone or as part of a split electrogram.

Results

Electrogram distribution. Thirty-five to 60 sites were measured during sinus rhythm in the 13 patients, with a mean of 48 sites each. All 13 patients had fractionated electrograms recorded on the endocardial surface at a mean of 10 sites per patient (or 21% of all measured sites). Split potentials were detected in 10 patients. In the 10 patients in whom split potentials were present, a mean of 2.7 sites (range 1 to 5) with split potentials were recorded. This represented 5.8% of the total number of mapped sites in the patients in whom split electrograms were present. Late potentials were recorded in only four patients at a mean of 2.3 sites, or 5%, of the total number of mapped sites (table 2).

Characteristics of fractionated electrograms. The characteristics of recorded fractionated electrograms are listed in table 3. The mean width for all fractionated electrograms was 86 msec. The mean width of the longest fractionated electrogram for patients in sinus rhythm was 129 msec. This was significantly longer than the mean width for all other fractionated electrograms measured. In 12 of 13 patients, fractionated electrograms starting within the QRS complex outlasted the end of the QRS. In these patients, a mean of 3.3 sites (1 to 6) had such delayed electrograms. In nine patients the latest fractionated electrogram was also the longest, and in four the latest and longest electrograms were recorded at disparate sites. The total endocardial activation time measured from the earliest onset to the end of electrical activity recorded on the endocardium was a mean of 171 msec and exceeded the surface QRS by a mean of 59 msec.

Spatial distribution of fractionated electrograms. Fractionated electrograms were widely distributed in most patients. They were found to coincide with areas of healed myocardial infarction, and in this group of patients were found predominantly on the ventricular septum (figure 3). Expressed as the number of clock sites in which the fractionated electrogram could be recorded, these electrograms could be found to cover two to nine segments, and in the majority of patients seven to nine clock segments were involved (figure 4). The greatest density of fractionated electrograms was found in the first two sequential rows recorded from the aneurysm edge (figures 1 and 4); however, up to five sequential rows contained fractionated electrograms in individual patients.
Spatial distribution of split and late potentials. Split and late potentials were fewer in number and less widely distributed than fractionated electrograms. They were almost uniformly observed in close proximity to fractionated electrograms in general, and specifically were often closely associated with the longest fractionated electrogram recorded during sinus rhythm. Six of nine late potentials were part of a split electrogram.

Relationship of abnormal sinus rhythm electrograms to site of tachycardia origin. The sites of origin of induced ventricular tachycardia, defined as the sites of earliest endocardial activation during ventricular tachycardia relative to the onset of the surface QRS, were compared with the locations of the longest fractionated electrograms, as well as the locations of any observed split or late potentials. Any of these type of electrograms falling within an area defined by one radial segment or clock site on either side of the site of origin of ventricular tachycardia and 2 cm toward the apex or the base (approximate area 8 cm²) were considered to be close to the site of origin of ventricular tachycardia. Those outside this arbitrary boundary were called dis-
FIGURE 1. B and C. B. Activation mapping during one of two morphologically distinct ventricular tachycardias. Surface electrocardiographic leads and electrograms are arranged as in figure IA. Presystolic electrical activity is designated by the dark arrows. The asterisk denotes the earliest site of activation during ventricular tachycardia of this morphology. The open arrow represents an electrogram that exactly straddles electrical diastole. The longest fractionated electrogram illustrated in figure IA was considered close to the site of origin of ventricular tachycardia in B, C., Endocardial activation mapping during the second of two morphologically distinct morphologies of ventricular tachycardia. The data is arranged in the same manner as before. The earliest sites of endocardial activation during ventricular tachycardia are designated by the black arrows. The asterisk denotes the earliest site. As before, the open arrow designates electrograms that straddle electrical diastole. The earliest site of endocardial activation during ventricular tachycardia of this morphology was considered distant from the most abnormal findings during sinus rhythm (see text).

FIGURE 2. Electrogram types and definitions. In each example is shown surface leads I, II, and V5R, a right ventricular reference electrogram, and recordings from the left ventricular endocardial probe. Ten millisecond time lines and 1 mV calibration signals are also displayed.
TABLE 2

<table>
<thead>
<tr>
<th>Electrogram frequency</th>
<th>Fractionated</th>
<th>Split</th>
<th>Late</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients (n)</td>
<td>13</td>
<td>10</td>
<td>4</td>
</tr>
<tr>
<td>Mean sites per patient (range)</td>
<td>10 (2–17)</td>
<td>2.7 (1–5)</td>
<td>2.3 (1–4)</td>
</tr>
<tr>
<td>% Total mapped sites (range)</td>
<td>21 (4–36)</td>
<td>5.8 (2–10)</td>
<td>5.0 (2–8)</td>
</tr>
</tbody>
</table>

sites of origin of two types of ventricular tachycardia and distant from those of two others. The longest fractionated electrogram recorded during sinus rhythm was close to at least one type of ventricular tachycardia in seven patients, and far from all sites of origin of ventricular tachycardia in six patients. Of the four patients in whom the longest and latest fractionated electrograms were at disparate sites, the site of latest activity in sinus rhythm was close to the site of a ventricular tachycardia of another morphology in two. Examples of maps obtained during sinus rhythm and during ventricular tachycardia are shown in figures 1 and 5.

The mean width of fractionated electrograms within an area defined as one radial segment on either side of

TABLE 3

<table>
<thead>
<tr>
<th>Mean characteristics of fractionated electrograms</th>
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<tbody>
<tr>
<td>Electrogram width (msec)</td>
<td>86 ± 29</td>
</tr>
<tr>
<td>Longest electrogram (msec)</td>
<td>129 ± 31</td>
</tr>
<tr>
<td>Exceed surface QRS</td>
<td>12/13 patients</td>
</tr>
<tr>
<td>Total endocardial activation (msec)</td>
<td>171 ± 30</td>
</tr>
<tr>
<td>Surface QRS width (msec)</td>
<td>112 ± 13</td>
</tr>
</tbody>
</table>

the earliest site of endocardial activation during each ventricular tachycardia did not differ from the mean width of those recorded from the remainder of the left ventricle (figure 6).

Discussion

Fractionated and other type of abnormal electrograms have been recorded from epicardial, intramyocardial, and endocardial sites of previous myocardial infarction in a variety of animal preparations as well as in human beings. These electrograms share the characteristics of high-frequency components with diminished amplitude, prolonged duration, or both. Fractionated electrograms have been of special interest because of reports of the association of these electrograms with the initiation, maintenance, and termina-

FIGURE 3. Detailed endocardial septal map during sinus rhythm. Local electrograms were recorded from multiple septal sites in a grid pattern as illustrated in the drawing and arbitrarily numbered 1 to 9. Electrograms recorded in this way are positioned as recorded on a grid represented by the dotted lines. Also shown are 1 mV calibration signals for each row, or for individual electrograms when change of gain occurred. Multiple bizarre low-amplitude multicomponent electrograms were recorded from nearly the entire left ventricular septum. Such findings are characteristic of patients with extensive anteroseptal infarction complicated by ventricular tachycardia. The dark arrows indicate His-Purkinje spikes and underline the survival of His-Purkinje tissue despite extensive infarction.
fraction of ventricular tachycardias arising after myocardial infarction. These electrograms have been felt to represent slow and disorganized conduction through ischemically damaged tissue, which may form the substrate for reentrant ventricular arrhythmias. This interpretation has not been uniformly accepted, however, and some have maintained that fractionated electrical activity may represent characteristics unrelated to reentrant activity, such as artifact resulting from catheter movement or other phenomena.

Ischemically damaged myocardium has been shown in a variety of preparations to be composed of fibrous tissue intermingled with partially damaged and completely normal surviving myocardial cells. This anatomic heterogeneity is also reflected in electrophysiologic heterogeneity. Gardner et al. have demonstrated that the origin of fractionated electrograms is surviving myocardial cells that have been uncoupled and electrically isolated by intervening fibrous tissue. This results in a slow and desynchronized depolarization of normal cells (nonuniform anisotropy) that inscribes a high-frequency multicomponent electrogram of low amplitude and long duration when recorded extracellularly.

Fractionated electrograms recorded during sinus rhythm have been reported in patients with coronary artery disease after myocardial infarction. Furthermore, patients with ventricular tachycardia have more frequent and longer fractionated electrograms. In our patients, fractionated electrograms were widely distributed and frequently found on the endocardial surface. Up to 36% of sites in these uniformly and completely mapped ventricles showed fractionated electrical activity. The distribution and degree of involvement cannot be directly compared with that in other studies because of differences in mapping detail and technique. The frequency of finding fractionated and other abnormal electrograms is consistent with the extensive myocardial damage that is associated with chronic recurrent ventricular tachycardia. Split electrograms have been recorded during sinus rhythm in patients with ventricular arrhythmias with or without myocardial infarction. These electrograms are believed to possibly represent desynchronized activation of tissue by one or more wavefronts. Like others, we have recorded these electrograms in fewer patients and at fewer sites than fractionated electrograms.

These fractionated and other abnormal electrograms are associated with a prolonged endocardial activation time, as has been noted in both animal and human preparations. In these studies total endocardial activation was markedly prolonged, with one or more long fractionated electrograms extending beyond the end of the surface QRS in all but one patient.

If these prolonged and delayed electrograms represent slow conduction manifest during sinus rhythm, in theory the longest or latest electrogram during sinus rhythm might be a likely site for reentry to occur. The appearance of locally recorded electrograms has been

| Table 4: Relationship of electrogram to ventricular tachycardia |
|------------------|----------|--------|--------|
|                  | Longest | Split  | Late   |
| Close            | 9       | 7      | 2      |
| Far              | 13      | 9      | 2      |
| Total VTs        | 22      | 16     | 4      |

VTs = ventricular tachycardias.
suggested as being useful in determining the boundaries of viable and nonviable myocardial tissue during performance of aneurysmectomy and coronary bypass. In addition, it has been suggested that findings during sinus rhythm could be helpful in directing surgical therapy for ventricular tachycardia arising in the setting of coronary disease or right ventricular dysplasia. This would be highly desirable, since activation mapping during ventricular tachycardia cannot always be performed due to characteristics such as tachycardia cycle length and failure of induction of tachycardia. The results of detailed mapping in our patients would suggest that neither the type, duration, nor timing of electrograms recorded during sinus rhythm is

FIGURE 5. The relationship of abnormal electrograms recorded during sinus rhythm to site of origin of ventricular tachycardia. A. The fractionated electrograms recorded during sinus rhythm in one patient in whom 42 sites were mapped is shown, along with a 1 mV calibration signal and time lines at the bottom. This patient had an extensive anteroseptal infarction. The asterisk denotes the longest fractionated electrogram recorded during sinus rhythm. B and C. Endocardial activation mapping during two morphologically distinct ventricular tachycardias. Surface leads I, II, and VSR, right ventricular and left ventricular references, and the earliest site of endocardial activation and its adjoining site are shown for both panels. The longest fractionated electrogram recorded during sinus rhythm, shown in A (asterisk), was associated with the ventricular tachycardia shown in B. The local electrogram associated with the ventricular tachycardia in C, while abnormal, was far from being the most abnormal.
completely reliable in localizing all sites of earliest endocardial activation during ventricular tachycardia. Too many extremely abnormal electrograms can be found during sinus rhythm on the endocardial surface to permit a localized endocardial resection. Resection of all sites of fractionated, split, and delayed electrograms would require resections of up to one-third of the endocardial surface of the left ventricle. Reports have appeared of removal of all visually abnormal endocardium in patients with ventricular tachycardia, resulting in ablation of the arrhythmia. In some cases this has required removal of papillary muscle and mitral valve replacement. It is still unclear whether resection of all tissue that appears abnormal results in an undue hemodynamic burden in these patients with already-compromised left ventricular function.

The poor correlation between abnormalities noted in sinus rhythm and the earliest site of endocardial activation during ventricular tachycardia is not totally surprising. To assume that the most abnormal electrograms represent slow and abnormal conduction is justifiable. However, one cannot assume linearity of conduction through areas under the bipolar electrode. Furthermore, slow conduction is not the only determinant of reentry. In theory, the area in which there is the longest conduction and shortest refractoriness would be the most likely site for reentry. However, refractoriness also appears to have a heterogeneous distribution in infarcted tissue and ischemically damaged myocardium may, in fact, be more refractory than normal tissue. The appropriate combination of conduction and refractoriness may not occur at a site at which conduction is longest.

Influences of the autonomic nervous system are only beginning to be understood with respect to ventricular tachycardia and may also be a determinant not reflected by the duration of the local electrogram. It is also possible that failure to show a relationship between abnormalities in sinus rhythm and ventricular tachycardia is related to the mapping procedure itself. Mapping done during sinus rhythm and ventricular tachycardia are not simultaneous and are open to errors of reproducibility of site selection. In this study, however, we used a detailed and uniform grid of the left ventricular endocardial surface and thereby minimized any discrepancy between sites of origin of ventricular tachycardia and sinus rhythm abnormalities arising as a result of failure to return to the same mapping site. Errors in localizing the earliest site of endocardial activation could also add to observed inaccuracy, but again they have been minimized by the standard detailed mapping scheme. Techniques such as high-density simultaneous recording of endocardial and transmural electrograms during sinus rhythm and ventricular tachycardia may be required to further clarify the relationship between conduction in sinus rhythm and the occurrence of local reentry. The response of local electrograms to perturbations such as atrial and ventricular pacing and extrastimuli may provide more information about potential sites of reentry than do sinus rhythm recordings. This possibility requires future study.

Abnormal electrograms recorded during normal sinus rhythm are widespread in patients with coronary artery disease, distant myocardial infarction, and ventricular tachycardia. The most common of these abnormal electrograms are fractionated. Split potentials can be recorded in most patients but are fewer in number than fractionated electrograms. Many of the fractionated electrograms recorded during sinus rhythm extend beyond the inscription of the surface QRS and result in a markedly prolonged endocardial activation time. Discrete electrograms inscribed after the surface QRS are infrequently seen, however. These electrograms may be associated with, but are not specific for, sites of origin of ventricular tachycardia. Therefore, surgery procedures based on sinus rhythm mapping of these electrograms would likely result in more extensive surgical excision than those guided by endocardial activation during ventricular tachycardia.

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References
DIAGNOSTIC METHODS-VENTRICULAR TACHYCARDIA


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