Endocardial catheter mapping in patients in sinus rhythm: relationship to underlying heart disease and ventricular arrhythmias

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ABSTRACT Catheter mapping during sinus rhythm was performed in 132 patients with coronary artery disease and 26 patients with congestive noncoronary cardiomyopathy. Each of the patients had a clinical history of one of the following: no ventricular arrhythmia, nonsustained ventricular tachycardia, cardiac arrest, or sustained ventricular tachycardia. The characteristics of the endocardial electrogram and other measured indexes of slow endocardial conduction were compared between patients with different types of disease and in different arrhythmia groups to determine if differences existed. The cardiomyopathic group had a higher percent of normal endocardial electrograms than the coronary artery disease group, with no evidence of slow endocardial conduction. The sustained ventricular tachycardia group exhibited a greater percent of abnormal endocardial electrograms and more evidence of slow endocardial conduction, distinguishing this group from the three other arrhythmia groups. We conclude the following: (1) The underlying electrophysiologic substrate varies in patients with different ventricular arrhythmias. It is therefore inappropriate to analyze all patients with ventricular arrhythmias as a single group. (2) Patients with congestive noncoronary cardiomyopathy, regardless of the type of their arrhythmia, have a relatively normal endocardium. Those patients with serious ventricular arrhythmias should not be considered candidates for surgery directed at removing abnormal endocardium.


LEFT VENTRICULAR mapping in individuals in sinus rhythm has been performed in a variety of patient groups, including those with a normal left ventricle,1–7 conduction defects,8–14 coronary artery disease with or without ventricular tachycardia,12–22 and hypertrophic cardiomyopathy.23 These studies primarily described activation patterns only, although a few attempted to quantitatively and qualitatively characterize local electrograms. Two of these studies14,17 have evaluated the possibility that characteristics of electrograms could identify an electrophysiologic substrate that would differentiate patients with ventricular tachycardia from those without tachycardia in the presence of coronary artery disease. These studies have analyzed all patients with complex ventricular ectopy, nonsustained ventricular tachycardia, sustained ventricular tachycardia, or ventricular fibrillation as one group. This failure to distinguish between patients with different ventricular arrhythmias is a possible source of error not adequately addressed. Finally, no information exists describing the characteristics of local sinus rhythm endocardial electrograms in patients with congestive noncoronary cardiomyopathy with or without ventricular arrhythmia.

This study was performed to address these issues. Specifically, the characteristics of local endocardial electrograms obtained by catheter mapping during sinus rhythm were compared in patients with coronary artery disease and those with congestive noncoronary disease.
cardiomyopathy to see if differences existed. In addition, an analysis was performed with the use of the same characteristics to see if differences existed based on type (or absence) of ventricular arrhythmia in patients with either coronary artery disease or noncoronary cardiomyopathy.

Methods

Patient population. One hundred fifty-eight patients referred to the Hospital of the University of Pennsylvania underwent sinus rhythm endocardial catheter mapping for either clinical or research purposes after informed written consent was obtained. All protocols have been approved by the Committee on Studies Involving Human Beings. The group consisted of 128 men and 30 women with a mean age of 58 ± 10 years.

The patients were classified according to underlying heart disease based on results of history, physical examination, chest x-ray, echocardiography, radionuclide gated blood pool scan (132/158 84%) and/or coronary angiography, and left ventriculography (150/158, 95%). There were 132 patients with coronary artery disease and 26 patients with congestive noncoronary cardiomyopathy (referred to as those in the cardiomyopathy group). The patients were classified by type of spontaneous arrhythmia, hereafter referred to as clinical arrhythmia, based on history and results of 12-lead electrocardiography, 24 hr Holter monitoring, and in-hospital telemetric monitoring. One hundred and fifty-four (98%) of the patients also underwent electrophysiologic stimulation and were classified according to induced arrhythmia (see below). Four patients (three with coronary artery disease, one with cardiomyopathy) did not undergo electrophysiologic stimulation. The stimulation protocol used has been previously described. The technique includes application of up to three extrastimuli from multiple ventricular sites and at multiple cycle lengths. Of the 154 patients undergoing programmed electrical stimulation, 106 (69%) had an arrhythmia similar to that noted clinically while 48 (31%) (four or 8% no ventricular tachycardia; 12 or 25% nonsustained ventricular tachycardia; 28 or 59% cardiac arrest; four or 8% sustained ventricular tachycardia) were reclassified for the analysis of the induced arrhythmia. Of the 102 patients undergoing programmed electrical stimulation not presenting with a clinical cardiac arrest, only five (5%) had an arrhythmia induced that caused cardiovascular collapse. The distribution of patients by disease and type of arrhythmia is diagrammatically represented in figure 1.

Catheter mapping technique. Studies were performed in patients in the nonsedated, postabsorptive state by previously described methods. One quadripolar (5 mm interelectrode distance) catheter (No. 6F USCI) was inserted percutaneously into the femoral artery and advanced to the left ventricle under fluoroscopic guidance. One to two quadripolar catheters were inserted percutaneously and were advanced to the right ventricular apex and right ventricular outflow tract. Heparin was administered to each patient after insertion of the arterial catheter.

The left ventricular mapping scheme used has been previously described. Each of the 12 predesignated areas represented approximately 4 to 8 cm². The catheter sites were verified by multiple-plane fluoroscopy by at least two independent observers. Stability was ensured by recording from each site for a minimum of 5 to 30 sec. Electrograms were recorded with a 10 mm interelectrode distance and both a variable-gain (to achieve best definition) and a fixed-gain (1 mV/cm) amplification. All electrograms were filtered at 30 to 500 Hz and recorded on

FIGURE 1. Patient population grouped by type of heart disease (coronary artery disease [CAD] or cardiomyopathy [CM]). The number of patients in each spontaneous clinical arrhythmia group is given in boxes in the column labeled "clinical." The number at the end of each line depicts how the patients were distributed after electrophysiologic stimulation, accounting for the number of patients in each box in the "induced" column. It should be noted that four patients without clinical arrhythmia (three with CAD; one with CM) did not undergo electrophysiologic stimulation. No VT = no ventricular tachycardia; NSVT = nonsustained ventricular tachycardia; CA = cardiac arrest; VT = ventricular tachycardia.

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analog magnetic tape (Honeywell 5600) and a 16-channel Mingograf (Siemens Elema) at a paper speed of 200 mm/sec.

**Definitions.** The electrograms were classified as normal, abnormal, or fractionated based on criteria for amplitude, duration, and an amplitude/duration ratio previously reported. A normal electrogram had an amplitude of 3 mV or greater, a duration of 70 msec or less, and/or an amplitude/duration ratio of 0.046 or greater. A fractionated electrogram had an amplitude of 0.5 mV or less, a duration of 133 msec or greater, and/or an amplitude/duration ratio of 0.005 or less. In addition, a late electrogram was defined as any type of electrogram with a duration that extended beyond the end of the surface QRS.

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.

Total endocardial activation time was defined as the time from the earliest local activation to the latest local activation. The endocardial activation time, the duration of the longest electrogram recorded, and the presence of late electrograms were taken as indicators of slow endocardial conduction velocity.

Patients were classified by type of arrhythmia on clinical presentation and by the arrhythmia induced in the electrophysiological laboratory. Sustained ventricular tachycardia was defined as tachycardia lasting greater than 30 sec and not causing hemodynamic collapse. Cardiac arrest was defined as ventricular fibrillation or ventricular tachycardia producing hemodynamic collapse. Nonsustained ventricular tachycardia was defined as a ventricular tachycardia of at least three complexes (non–bundle branch reentry) lasting up to 30 sec and not requiring cardioversion. All other patients were classified as having no ventricular tachycardia.

**Data analysis.** Electrogram amplitudes, durations, and activation times were assessed at each site by two independent observers, with a minimum of four complexes being sampled. A maximum of 12 sites per patient was used for this analysis. Thus, a mean value was used when multiple samples were obtained from any given site. All patients included in this analysis had a minimum of 75% (i.e., nine out of 12 sites) of the endocardium sampled.

All results are expressed in terms of mean ± SD. The Student’s t test for unpaired data, the Mann-Whitney test, and R × C contingency table analysis were used as appropriate.

**Results**

**General data.** A mean of 17 ± 4 total sites, resulting in 11 ± 1 separate (maximum 12) sites, were obtained per patient. This was equally distributed among all patient subgroups.

Fifty-six (36%) of the patients were receiving antiarrhythmic agents at the time of mapping. An R × C contingency analysis revealed a significant disproportion of patients on antiarrhythmic agents in the various groups both with respect to type of heart disease (coronary artery disease group 54 [41%], cardiomyopathy group three [12%]; p < .01) and type of arrhythmia (no ventricular tachycardia group 0; nonsustained ventricular tachycardia group one [7%], cardiac arrest group 15 [39%], ventricular tachycardia group 40 [57%]; p < .01).

Although most patients (124 or 94%) in the coronary artery disease group had had a previous myocardial infarction, R × C contingency analysis revealed an uneven distribution among the groups classified by type of arrhythmia (no ventricular tachycardia group nine [100%], nonsustained ventricular tachycardia group 14 [93%], cardiac arrest group 32 [94%], ventricular tachycardia group 69 [99%]; p < .05).

The results of this analysis led to a repeat analysis eliminating data from those patients who were receiving antiarrhythmic agents or had no previous documented myocardial infarction, thereby correcting for this possible source of bias. The analysis controlling for antiarrhythmic agents and the absence of myocardial infarction gave numerically similar results. This latter analysis resulted in a smaller number of patients in some subgroups and occasionally changed the statistical power of observations. The results presented in the subsequent tables are for the entire population; all resultant changes from the later analysis are described in the tables or text.

Ejection fraction was similar in the coronary artery disease (34 ± 16%) and the cardiomyopathy (34 ± 13%) groups. The ejection fraction was significantly lower (p < .05) in the ventricular tachycardia group (30 ± 12%) than in the no ventricular tachycardia (40 ± 18%), nonsustained ventricular tachycardia (38 ± 17%), and cardiac arrest (37 ± 19%) groups when patients were classified by clinical arrhythmia. The ejection fraction was only significantly (p < .05) lower in the ventricular tachycardia group (30 ± 12%) than in the no ventricular tachycardia (43 ± 22%) and nonsustained ventricular tachycardia (38 ± 15%) groups when patients were classified by induced arrhythmia. These relationships remained unchanged when the type of heart disease was considered or when controlling for antiarrhythmic agents and no previous infarction.

When controlling for the presence of antiarrhythmic agents, no statistically significant differences in surface QRS duration existed between the coronary artery disease (122 ± 33 msec) and cardiomyopathy (120 ± 32 msec) groups, or among the no ventricular tachycardia (111 ± 22 msec), nonsustained ventricular tachycardia (116 ± 25 msec), cardiac arrest (108 ± 26 msec), or ventricular tachycardia (122 ± 29 msec) groups.

**Influence of underlying heart disease (table 1).** There were marked differences in the percent distribution of types of endocardial electrograms between the cardiomyopathy and coronary artery disease groups. The cardiomyopathy group exhibited almost all normal electrograms while fractionated electrograms were only recorded in three patients (all with ventricular
tachycardia). The indicators of endocardial conduction (endocardial activation time, duration of longest electrogram, and presence of late electrograms) showed significant slowing in the coronary artery disease group compared with the cardiomyopathy group. Late electrograms were not recorded in any of the patients with congestive noncoronary cardiomyopathy and ventricular tachycardia.

**Influence of coronary artery disease and arrhythmia**

Clinical arrhythmia (table 2). No differences existed with respect to percent distribution of types of electrograms among the no ventricular tachycardia, nonsustained ventricular tachycardia, or cardiac arrest groups, while the ventricular tachycardia group differed significantly from the nonsustained ventricular tachycardia group in the distribution of normal, abnormal, and fractionated electrograms. The ventricular tachycardia group, with respect to electrogram distribution, exhibited a greater percent of abnormal electrograms than the cardiac arrest group and a greater percent of fractionated electrograms than the no ventricular tachycardia group. The ventricular tachycardia group showed a significantly longer endocardial activation time than the cardiac arrest or nonsustained ventricular tachycardia group. No differences existed among the four groups with respect to percentage distribution of late electrograms.

Induced arrhythmia (table 3). The statistical relationships were altered when the patients in the coronary artery disease group were classified according to type of inducible arrhythmia. The cardiac arrest group was not distinguished from the ventricular tachycardia group (the percent of late sites was no different when controlling for the use of antiarrhythmic agents). The ventricular tachycardia group, compared with the no ventricular tachycardia and nonsustained ventricular tachycardia groups, showed a numerically greater difference with respect to the percent distribution of electrogram types and the cardiac arrest group differed from the no ventricular tachycardia group in the percent distribution of normal and abnormal electrograms in this analysis. The ventricular tachycardia group exhibited evidence of significantly slower endocardial conduction compared with the no ventricular and nonsustained ventricular tachycardia groups, while the cardiac arrest group appeared to have intermediate conduction characteristics. No significant differences existed among the groups with respect to percent distribution of late electrograms when controlling for the use of antiarrhythmic agents.

**Influence of cardiomyopathy and arrhythmia**

Clinical arrhythmia (table 4). The only significant difference among the cardiomyopathic patients when classified by type of clinical arrhythmia was a lesser percent distribution of normal electrograms and greater percent distribution of abnormal electrograms in patients in the ventricular tachycardia group compared with the other three groups. No significant differences in the indexes of endocardial conduction were present in these patients based on type of clinical arrhythmia.

Induced arrhythmia (table 5). No significant differences in the measured parameters were found among the cardiomyopathic patients with no ventricular tachycardia.

**TABLE 1**

<table>
<thead>
<tr>
<th>Influence of underlying heart disease</th>
<th>CAD</th>
<th>CM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients (n)</td>
<td>132</td>
<td>26</td>
</tr>
<tr>
<td>Normal sites (%)</td>
<td>50 ± 24</td>
<td>84 ± 18a</td>
</tr>
<tr>
<td>Abnormal sites (%)</td>
<td>43 ± 21</td>
<td>15 ± 18a</td>
</tr>
<tr>
<td>Fractionated sites (%)</td>
<td>7 ± 11</td>
<td>1 ± 3a</td>
</tr>
<tr>
<td>Endocardial activation time (msec)</td>
<td>66 ± 30</td>
<td>49 ± 14b</td>
</tr>
<tr>
<td>Duration of longest electrogram (msec)</td>
<td>118 ± 39</td>
<td>93 ± 25b</td>
</tr>
<tr>
<td>Late sites (%)</td>
<td>12 ± 14</td>
<td>2 ± 6a</td>
</tr>
</tbody>
</table>

CAD = coronary artery disease; CM = cardiomyopathy.

ap < .001; bp < .005.

**TABLE 2**

<table>
<thead>
<tr>
<th>Influence of coronary artery disease and clinical arrhythmia</th>
<th>No VT</th>
<th>NSVT</th>
<th>CA</th>
<th>VT</th>
</tr>
</thead>
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<tr>
<td>Patient (n)</td>
<td>9</td>
<td>15</td>
<td>38</td>
<td>70</td>
</tr>
<tr>
<td>Normal sites (%)</td>
<td>54 ± 25</td>
<td>63 ± 26</td>
<td>57 ± 27</td>
<td>42 ± 19a</td>
</tr>
<tr>
<td>Abnormal sites (%)</td>
<td>45 ± 24</td>
<td>35 ± 23</td>
<td>37 ± 23</td>
<td>48 ± 17b</td>
</tr>
<tr>
<td>Fractionated sites (%)</td>
<td>1 ± 3</td>
<td>2 ± 4</td>
<td>6 ± 12</td>
<td>10 ± 10c</td>
</tr>
<tr>
<td>Endocardial activation time (msec)</td>
<td>52 ± 11</td>
<td>54 ± 23</td>
<td>60 ± 28</td>
<td>73 ± 32</td>
</tr>
<tr>
<td>Duration of longest electrogram (msec)</td>
<td>86 ± 12d</td>
<td>102 ± 12</td>
<td>112 ± 34</td>
<td>129 ± 43p</td>
</tr>
<tr>
<td>Late sites (%)</td>
<td>15 ± 15</td>
<td>11 ± 11</td>
<td>8 ± 12</td>
<td>15 ± 16</td>
</tr>
</tbody>
</table>

No VT = no ventricular tachycardia; NSVT = nonsustained ventricular tachycardia; CA = cardiac arrest; VT = ventricular tachycardia.

ap < .005 VT vs NSVT; bp < .05 VT vs NSVT, CA; cp < .05 VT vs No VT, NSVT; dp < .05 VT vs NSVT, CA; ep < .05 No VT vs NSVT, CA.
PATHOPHYSIOLOGY AND NATURAL HISTORY—VENTRICULAR ARRHYTHMIA

TABLE 3
Influence of coronary artery disease and induced arrhythmia

<table>
<thead>
<tr>
<th></th>
<th>No VT</th>
<th>NSVT</th>
<th>CA</th>
<th>VT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient (n)</td>
<td>12</td>
<td>16</td>
<td>21</td>
<td>80</td>
</tr>
<tr>
<td>Normal sites (%)</td>
<td>77 ± 26</td>
<td>62 ± 19</td>
<td>50 ± 27(^c)</td>
<td>43 ± 19(^a)</td>
</tr>
<tr>
<td>Abnormal sites (%)</td>
<td>23 ± 26</td>
<td>37 ± 18</td>
<td>44 ± 26(^d)</td>
<td>47 ± 17(^a)</td>
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<tr>
<td>Fractionated sites (%)</td>
<td>0 ± 0</td>
<td>0 ± 0</td>
<td>6 ± 11</td>
<td>10 ± 11(^b)</td>
</tr>
<tr>
<td>Endocardial activation time (msec)</td>
<td>48 ± 12</td>
<td>53 ± 19</td>
<td>61 ± 30</td>
<td>73 ± 32(^a)</td>
</tr>
<tr>
<td>Duration of longest electrogram (msec)</td>
<td>97 ± 15</td>
<td>97 ± 17</td>
<td>108 ± 26</td>
<td>130 ± 43(^a)</td>
</tr>
<tr>
<td>Late sites (%)</td>
<td>5 ± 10</td>
<td>8 ± 11</td>
<td>7 ± 10</td>
<td>16 ± 15(^e)</td>
</tr>
</tbody>
</table>

Abbreviations are as in table 2.

\(^a\)p < .05 VT vs No VT, NSVT; \(^b\)p < .005 VT vs No VT, NSVT; \(^c\)p < .01 CA vs No VT; \(^d\)p < .05 CA vs No VT; \(^e\)p < .05 VT vs No VT, CA (p = NS when controlled for presence of drugs).

Sudden cardiac death remains a major cause of mortality facing the population. Although acute ischemia may initiate some of these lethal arrhythmias, the majority appear unrelated to acute ischemia. The combination of significant multivessel coronary disease, prior infarction, and left ventricular aneurysm is a sensitive index of significant tachyarrhythmias, but it is not specific. Recent studies in a limited number of patients undergoing operative procedures\(^{14, 17, 20}\) have attempted to more precisely identify a specific electrophysiologic substrate. All three studies have classified patients, irrespective of their clinical or induced ventricular arrhythmias, as a single group. The above results suggest this is inappropriate. Differences related to clinical prognosis and inducibility of arrhythmia with programmed electrical stimulation are recognized between patients with nonischemic cardiac arrest vs those with hemodynamically tolerated ventricular tachycardias. Little attention has been focused on identifying an electrophysiologic substrate that may separate these two groups. Patients with nonischemic cardiomyopathy and depressed ejection fractions are a high-risk population for sudden death,\(^{25}\) yet little is known about the electrophysiologic substrate underlying their demise.

This study has tried to address some of these problems by applying the technique of catheter mapping during sinus rhythm to patients with coronary artery disease and cardiomyopathy. The technique, although not without limitations, appears to provide information that distinguishes patients by type of heart disease and, to a lesser extent, type of arrhythmia.

Rationale of analysis. Reentry is generally believed to be the electrophysiologic mechanism of the arrhythmias under discussion in this patient population. Slow conduction, one requirement for reentry, is best represented (admittedly indirectly) by the measured param-

TABLE 4
Influence of cardiomyopathy and clinical arrhythmia

<table>
<thead>
<tr>
<th></th>
<th>No VT</th>
<th>NSVT</th>
<th>CA</th>
<th>VT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient (n)</td>
<td>2</td>
<td>10</td>
<td>9</td>
<td>5</td>
</tr>
<tr>
<td>Normal sites (%)</td>
<td>87 ± 7</td>
<td>90 ± 10</td>
<td>91 ± 11</td>
<td>60 ± 25(^a)</td>
</tr>
<tr>
<td>Abnormal sites (%)</td>
<td>13 ± 7</td>
<td>10 ± 10</td>
<td>9 ± 11</td>
<td>37 ± 27(^b)</td>
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<tr>
<td>Fractionated sites (%)</td>
<td>0 ± 0</td>
<td>0 ± 0</td>
<td>0 ± 0</td>
<td>3 ± 7</td>
</tr>
<tr>
<td>Endocardial activation sites (msec)</td>
<td>43 ± 11</td>
<td>50 ± 16</td>
<td>43 ± 14</td>
<td>49 ± 14</td>
</tr>
<tr>
<td>Duration of longest electrogram (msec)</td>
<td>92 ± 5</td>
<td>95 ± 23</td>
<td>83 ± 14</td>
<td>109 ± 43</td>
</tr>
<tr>
<td>Late sites (%)</td>
<td>4 ± 6</td>
<td>5 ± 8</td>
<td>0 ± 0</td>
<td>0 ± 0</td>
</tr>
</tbody>
</table>

Abbreviations are as in table 2.

\(^a\)p < .01 VT vs No VT, NSVT, CA; \(^b\)p < .05 VT vs No VT, NSVT, CA.

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TABLE 5
Influence of cardiomyopathy and induced arrhythmia

<table>
<thead>
<tr>
<th>Patients (n)</th>
<th>No VT</th>
<th>NSVT</th>
<th>CA</th>
<th>VT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal sites (%)</td>
<td>85 ± 9</td>
<td>88 ± 8</td>
<td>86 ± 10</td>
<td>60 ± 25</td>
</tr>
<tr>
<td>Abnormal sites (%)</td>
<td>5 ± 9</td>
<td>12 ± 9</td>
<td>14 ± 10</td>
<td>37 ± 27</td>
</tr>
<tr>
<td>Fractionated sites (%)</td>
<td>0 ± 0</td>
<td>0 ± 0</td>
<td>0 ± 0</td>
<td>3 ± 7</td>
</tr>
<tr>
<td>Endocardial activation time (msec)</td>
<td>90 ± 20</td>
<td>93 ± 18</td>
<td>85 ± 22</td>
<td>109 ± 43</td>
</tr>
<tr>
<td>Duration of longest electrogram (msec)</td>
<td>1 ± 3</td>
<td>6 ± 9</td>
<td>2 ± 4</td>
<td>0 ± 0</td>
</tr>
</tbody>
</table>

Abbreviations are as in table 2.

*P < .05 VT vs No VT, NSVT; *P < .01 VT vs No VT.

eters of QRS duration, endocardial activation time, duration of longest electrogram, and presence of late electrograms. The type of electrograms recorded (i.e., normal, abnormal, or fractionated) provide information about the presence of an underlying fixed electrophysiologic substrate. Clear differences were observed related to both type of heart disease and type of arrhythmia, thereby providing new insight into the specific electrophysiologic substrates causing arrhythmias in these patients.

The classification of patients by arrhythmias induced in the laboratory primarily affected those in the cardiac arrest group, since only 40% of the patients were still classified in this group after undergoing programmed stimulation. The 60% that were reclassified were equally divided between the no ventricular tachycardia and nonsustained ventricular tachycardia groups. The cardiac arrest group could then be differentiated from the no ventricular tachycardia group by the distribution of types of electrograms. It appears that in patients who present with nonischemic cardiac arrest, the varying responses to programmed electrical stimulation may be based on a specific electrophysiologic substrate.

**Influence of heart disease and arrhythmia.** The duration of the surface QRS did not distinguish either the coronary artery disease from the cardiomyopathy group or any of the arrhythmia groups from one another. In contrast, the total endocardial activation time and duration of the longest electrogram were both shorter in the cardiomyopathy group than the coronary artery disease group. The total endocardial activation time in patients with cardiomyopathy was only slightly longer than that reported in individuals with normal left ventricles, but was less than that reported by Vassallo et al. in patients with cardiomyopathy and left bundle branch block. This observation was consistent regardless of arrhythmia group. The patients with cardiomyopathy showed very few late sites, and no late sites were recorded in patients presenting with cardiac arrest or ventricular tachycardia. The majority of electrograms recorded from the endocardium were normal, with a slightly lower percent of normal electrograms in the cardiomyopathy–ventricular tachycardia group. Fractionated electrograms were only recorded in two patients, both of whom were in the cardiomyopathy–ventricular tachycardia group. These findings are significant in view of the recent report of Poll et al., who reported that patients with cardiomyopathy and inducible arrhythmias, either ventricular tachycardia or cardiac arrest, tended to show late potentials when signal-averaged techniques were used. Vassallo et al. reported that signal-averaged late potentials in patients with coronary artery disease and inducible arrhythmias were related to both the number and duration of late sites recorded on the endocardium. These data suggest that the endocardium is relatively unaffected in patients with cardiomyopathy and that the electrophysiologic substrate for arrhythmias is either intramural or epicardial. Further studies with intraoperative mapping will be necessary to clarify this issue.

In contrast, in the coronary artery disease group there were substantial differences among the patients with different types of arrhythmia with respect to the results of catheter mapping. The no ventricular tachycardia and nonsustained ventricular tachycardia groups were similar, both being distinguished from the ventricular tachycardia group by the fact that there was less evidence of slow endocardial conduction. Almost no fractionated electrograms were recorded in either the no ventricular tachycardia or nonsustained ventricular tachycardia group. The cardiac arrest group appeared to have slow conduction properties intermediate between those of the no ventricular tachycardia, nonsustained ventricular tachycardia, and ventricular tachycardia groups. There was a trend for the patients in whom cardiac arrest was induced to have a shorter endocardial activation time, shorter longest electro-
gram duration, and lower percent distribution of late and fractionated sites than those in whom ventricular tachycardia was induced. The cardiac arrest group was distinguished from the no ventricular tachycardia group with regard to the percent of normal and abnormal electrograms recorded. These data may suggest that an “intermediate substrate” exists in patients with cardiac arrest that is sufficiently abnormal to allow malignant tachyarhythmias to exist, yet well enough preserved to allow rapid tachycardia rates leading to cardiac arrest. This may only be a partial explanation because recent work from this laboratory failed to demonstrate a close correlation between electrogram durations and cycle length of ventricular tachycardia in patients with coronary artery disease and inducible arrhythmias.

Our data, unlike that reported previously, failed to show the percent distribution of late electrograms to be different in any of the four arrhythmia groups. It may be that late electrograms are simply a nonspecific marker of slow conduction related to previous infarction as opposed to an arrhythmogenic substrate. This is consistent with our previous report, which showed that late electrograms are not predictive of the site of origin of ventricular tachycardia.

Limitations. The technical limitations of interpreting data obtained by catheter mapping during sinus rhythm have been previously reported. Catheter mapping during sinus rhythm is a relatively specialized invasive technique that is not widely available. The information obtained in this study, however, may provide additional insight into risk factors for malignant ventricular arrhythmias.

A second limitation relates to the patient population. Fifty percent of the patients in this study had coronary artery disease and sustained ventricular tachycardia. The statistical power of the observations in other patient groups is therefore somewhat restricted.

Clinical implications. The technique of endocardial catheter mapping during sinus rhythm provides a method by which patients with coronary artery disease and ventricular tachycardia can be characterized and differentiated from those patients with no or nonsustained ventricular arrhythmia. Patients with coronary artery disease and inducible ventricular arrhythmias leading to cardiac arrest can be less reliably differentiated from those patients with no ventricular arrhythmia. Whether this technique can be applied to identify patients at risk for serious arrhythmias or is more useful than presently available noninvasive techniques for risk stratification was not addressed by this study. A prospective comparative trial would be indicated to resolve this question. The data suggest that patients with cardiac arrest or ventricular tachycardia and underlying coronary artery disease should not be considered as a single group for the purposes of data analysis.

Endocardial sinus rhythm catheter mapping appears less useful in patients with nonischemic cardiomyopathy. This suggests that the electrophysiologic substrate for serious ventricular tachyarhythmias in these patients is not on or adjacent to the endocardium. Further studies with intraoperative epicardial and intramura1 mappin are needed to elucidate the location and features of the substrate. The present results suggest that current surgical therapy for ventricular arrhythmias in patients with coronary artery disease (i.e., therapy directed toward the endocardium) is not appropriate for patients with nonischemic cardiomyopathy.

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