ELECTROPHYSIOLOGIC STUDIES

Anterior Left Ventricular Aneurysm: Factors Associated With the Development of Sustained Ventricular Tachycardia

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Fifty patients with anterolateral left ventricular aneurysm secondary to prior myocardial infarction underwent aneurysmectomy, at which time endocardial sinus rhythm mapping was performed. Forty patients had a history of recurrent sustained monomorphic ventricular tachycardia, and 10 had an aneurysm but no history of spontaneous sustained tachycardia. A comparison of the clinical, angiographic and sinus rhythm endocardial electrographic characteristics of these two groups revealed that the patients without spontaneous ventricular tachycardia had more severe coronary artery disease (2.6 ± 0.5 versus 1.9 ± 0.8 coronary arteries having >70% stenosis; p < 0.03), underwent surgery earlier after infarction (3 ± 2 versus 46 ± 53 months; p < 0.003) and had less extensive wall motion abnormalities on contrast ventriculography (10 of 8 versus 13 of 35 patients assessed had an abnormally contracting ventriculographic segmental length >60%; p < 0.04).

During intraoperative programmed electrical stimulation, all 40 patients with and 4 of 10 without a history of spontaneous ventricular tachycardia had inducible tachycardia. The patients with inducible tachycardia had a larger area of endocardium from which abnormal electrograms (duration >70 ms or amplitude <0.7 mV) were recorded (62 ± 17 versus 45 ± 20% of electrograms; p < 0.03) as well as fractionated (duration >90 ms, amplitude <0.3 mV) electrograms (20 ± 14 versus 9 ± 7% of electrograms; p < 0.04) than did patients without inducible tachycardia, but there were no angiographic differences between groups.

These data suggest that 1) differences between groups of patients with versus without either inducible or spontaneous ventricular tachycardia are more quantitative than qualitative; and 2) the pathophysiologic substrate for ventricular tachycardia may develop relatively early after infarction, but other factors determine the development of spontaneous episodes of tachycardia. In the absence of features that clearly identify those patients with an aneurysm who are at high risk for future episodes of spontaneous ventricular tachycardia, it may be reasonable to consider performing pre- or intraoperative stimulation or blind subendocardial resection in such patients at the time of elective aneurysmectomy.

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The development of a left ventricular aneurysm occurs in as many as 35% of patients after acute anterior wall myocardial infarction (1,2). Such aneurysms are associated with a high incidence of serious ventricular arrhythmias, especially uniform sustained ventricular tachycardia, but the factors that determine which patient with an aneurysm will develop these potentially lethal arrhythmias are unknown. In the present study, we performed intraoperative endocardial mapping and programmed electrical stimulation in a group of patients with postinfarction anterior left ventricular aneurysm at the time of surgery for ventricular tachycardia or aneurysm resection, or both. We sought to elucidate differences in clinical, angiographic, hemodynamic and endocardial electrographic characteristics between subgroups of patients that might identify factors associated with the development of sustained ventricular tachycardia.
Methods

Study patients. The study group consisted of 50 patients with an anterolateral left ventricular aneurysm (defined as an area of dyskinetic wall motion on right anterior oblique cineventriculography or gated nuclear scanning) after acute anterior wall myocardial infarction. All patients in this study underwent elective left ventricular aneurysmectomy, with or without coronary artery bypass grafting. There were 41 men and 9 women with a mean age of 59 ± 9 years. All 50 patients underwent preoperative hemodynamic and angiographic cardiac catheterization including selective coronary cineangiography, and all but 3 underwent left ventriculography (right anterior oblique projection). All patients had angiographic evidence of prior complete occlusion of the left anterior descending coronary artery, usually before the origin of any branch vessels, and no other totally occluded coronary arteries. Ten patients had an aneurysm and no history of spontaneous sustained ventricular tachycardia (lasting >30 s or requiring cardioversion), whereas 40 patients had a left ventricular aneurysm and a history of spontaneous sustained ventricular tachycardia of uniform configuration occurring from 1 to 150 months (mean ± SD 33 ± 44) after myocardial infarction. The surgical indication was medically refractory angina in the 10 patients with no history of ventricular infarction. All patients in this study had medically refractory sustained ventricular tachycardia, whereas 40 patients had a left ventricular aneurysm and a history of spontaneous sustained ventricular tachycardia.

Preoperative programmed electrical stimulation protocol. All patients with spontaneous ventricular tachycardia underwent preoperative programmed electrical stimulation using a protocol of one to three ventricular extrastimuli delivered from two right ventricular sites at paced cycle lengths of 600 and 400 ms. Five of the patients without spontaneous tachycardia underwent preoperative hemodynamic and angiographic cardiac catheterization including selective coronary cineangiography, and all but 3 underwent left ventriculography (right anterior oblique projection). All patients had angiographic evidence of prior complete occlusion of the left anterior descending coronary artery, usually before the origin of any branch vessels, and no other totally occluded coronary arteries. Ten patients had an aneurysm and no history of spontaneous sustained ventricular tachycardia (lasting >30 s or requiring cardioversion), whereas 40 patients had a left ventricular aneurysm and a history of spontaneous sustained ventricular tachycardia of uniform configuration occurring from 1 to 150 months (mean ± SD 33 ± 44) after myocardial infarction. The surgical indication was medically refractory angina in the 10 patients with no history of ventricular tachycardia in the remaining 40 patients it was medically refractory sustained ventricular tachycardia.

Intraoperative programmed electrical stimulation. In all patients, consisted of introduction of a maximum of three programmed ventricular extrastimuli delivered at two to four times diastolic threshold (1 ms pulse width) from the right ventricular endocardial plunge electrode at drive cycle lengths of 500 and 400 ms. When induced, sustained ventricular tachycardia was mapped on the endocardium in the same manner as during sinus rhythm, and was followed by subendocardial resection (4) of the area or areas with earliest electrical activity in the latter half of diastole, including a 1 to 2 cm surrounding margin.

Data Analysis

Ventriculographic assessment. Aneurysm size and contractile segment function were analyzed as follows. Tracings of end-diastolic and end-systolic frames of the right anterior oblique ventriculogram were made independently by two observers (W.G.K. and J.M.M.) and digitized using a microcomputer system (Hewlett-Packard 9826). Each tracing was marked at points of demarcation between normal and abnormal contractile segments as being either dyskinetic or akinetic. A customized computer program allowed the assessment of the relative size of the abnormally contracting segment as a function of the entire end-diastolic perimeter using the method of Feild et al. (5). The overall ejection fraction was calculated, as well as an idealized ejection fraction based on the systolic motion of normally contracting segments. The excess ejection fraction was derived according to the equation (6): Excess ejection fraction = 0.67 (1 – % Abnormally contracting segment/100)3. Tracings from the two observers were analyzed independently, and the mean results for each patient were determined.

With these methods, cineventriculograms in 43 of the 50 patients could be assessed. No assessment could be made in seven patients (five with and two without spontaneous tachycardia) because of technically poor studies precluding the assessment of basal segment wall motion (two patients), no ventriculogram obtained (three patients), and excessive ventricular ectopic beats (one patient) or atrial fibrillation during the ventriculogram (one patient).

Electrographic assessment. Intraoperative endocardial electrograms obtained during sinus rhythm were evaluated as follows. Electrographic amplitude (mV) and duration (ms) from each endocardial site were measured for three consecutive beats, and the mean results for each site were determined. Electrograms were arbitrarily characterized as normal (duration <70 ms, amplitude >0.7 mV) or abnormal (all others). Abnormal electrograms were further classified as fractionated (duration >90 ms, amplitude <0.3 mV, with
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Figure 1. Examples of intraoperative electrographic recordings.
Shown are (top to bottom) surface electrocardiograph c leads 1, 2, 3
and V,R; an intracardiac reference electrogram from the right
ventricle (RV) and two electrograms recorded from the left ventric-
endocardium. A time reference is also shown as 1 mV calibration
signals to the right of the intracardiac electrogram. The vertical
dotted line denotes the end of the surface QRS complex for deter-
mination of late electrograms. The right ventricular electrogram is
an example of a normal electrogram. Below it are two abnormal
electrograms: the first is fractionated as well as late extending
beyond the end of the QRS complex) and the second is a split
electrogram.

Figure 1. Examples of intraoperative electrographic recordings.

A dotted line denotes the end of the surface QRS complex for detection of late electrograms. The right ventricular electrogram is an example of a normal electrogram. Below it are two abnormal electrograms: the first is fractionated as well as late extending beyond the end of the QRS complex) and the second is a split electrogram.

Results

Presence or Absence of Spontaneous Sustained Ventricular Tachycardia

Clinical characteristics. All 70 patients who underwent intraoperative electrographic recordings were regrouped according to the presence (44 patients) or absence (26 patients) of spontaneous ventricular tachycardia. The only clinical characteristic that distinguished the two groups was time from acute infarction to surgery (mean 68 ± 8 months for patients with spontaneous ventricular tachycardia versus 57 ± 8 months for patients without ventricular tachycardia; p < 0.001). There were no significant differences in angina or congestive heart failure between these two groups.

Angiographic/hemodynamic characteristics (Table 1B). Patients who had a history of episodes of spontaneous ventricular tachycardia were older than the 40 patients without such a history (mean age 68 ± 8 versus 57 ± 8 years; p < 0.001). The 10 patients without spontaneous ventricular tachycardia had a larger area of abnormal wall motion on ventriculography than did those without spontaneous tachycardia (13 of the 35 patients with spontaneous ventricular tachycardia had an abnormally contracting segment length >60%, compared with 0 of the 8 patients without spontaneous tachycardia (p < 0.04). No other angiographic or hemodynamic differences were observed between these groups.

Electrographic analysis (Table 2A). There were no significant differences in the prevalence of any type of endocardial electrogram or in the duration of the longest electrogram between patients with and without spontaneous ventricular tachycardia.

Presence or Absence of Inducible Sustained Ventricular Tachycardia

Clinical characteristics. At the time of surgery, sustained ventricular tachycardia of uniform configuration could be induced in all 40 patients who had a history of spontaneous tachycardia and in 4 of the 10 patients without any history of episodes of spontaneous tachycardia. Eleven distinct tachycardia configurations with a mean cycle length of 46 ± 71 ms (range 230 to 460) were induced in these latter four patients (six were initiated with double extrastimuli, four with triple extrastimuli and one with the ventricular drive at a paced cycle length of 400 ms. When patients were regrouped according to the presence (44 patients) or absence (6 patients) of inducible tachycardia, the only clinical characteristic that distinguished the two groups was time from acute infarction to surgery (3 ± 2 months for patients without inducible tachycardia versus 42 ± 53 months for patients with tachycardia; p < 0.003). There was no significant difference in age or the presence of angina or congestive heart failure between these two groups.

Angiographic/hemodynamic characteristics (Table 1B). There were no significant differences in angiographic or hemodynamic characteristics between patients with and without inducible tachycardia. Specifically, relative aneurysm size was not significantly different between the two groups.

Electrographic characteristics (Table 2B). Compared with patients without inducible ventricular tachycardia, patients with inducible tachycardia at the time of surgery had a significantly larger area of endocardium from which abnormal electrograms were recorded (62 ± 17 versus 45 ± 20% of electrograms for patients without inducible ventricular tachycardia; p < 0.03). The same was true of fractionated electrograms (20 ± 14% versus 9 ± 7%; p < 0.04). The distributions of late and split electrograms and the duration of the longest endocardial electrogram were also recorded for each patient.
of the longest electrogram did not differ between these groups. Examples of electrographic distributions for patients with no spontaneous or inducible ventricular tachycardia, no spontaneous tachycardia but inducible tachycardia and those with spontaneous tachycardia are shown in Figure 2.

Among the five patients without spontaneous ventricular tachycardia who underwent preoperative programmed stimulation, one had no arrhythmia induced and two had a maximum of six beats of nonsustained induced ventricular tachycardia; none of these patients had inducible tachycardia intraoperatively. Of the remaining two patients, one had rapid ventricular tachycardia (cycle length 220 ms), that quickly degenerated to ventricular fibrillation requiring cardioversion and the other had three configurations of uniform sustained ventricular tachycardia initiated preoperatively. Both of these latter patients had inducible sustained uniform ventricular tachycardia intraoperatively.

**Discussion**

Patients with postinfarction anterior left ventricular aneurysm are at increased risk for developing life-threatening ventricular arrhythmias. This study demonstrates that patients who develop spontaneous uniform sustained ventricular tachycardia have a larger aneurysm on ventriculography than do those without spontaneous ventricular tachycardia, and that patients with inducible (though not necessarily spontaneous) ventricular tachycardia have significantly more widespread abnormalities of their sinus rhythm electrograms than do patients without inducible ventricular tachycardia.

**Presence versus absence of spontaneous ventricular tachycardia.** Patients without spontaneous ventricular tachycardia differed from those with spontaneous ventricular tachycardia in that they 1) underwent surgery earlier after infarction, 2) had more severe coronary disease, and 3) had less extensive wall motion abnormalities (percent abnormally contracting segment). The first two differences are likely related in that the major surgical indication for patients with spontaneous ventricular tachycardia was the tachycardia (which generally does not develop until several months or years after infarction), whereas the need for revascularization prompted surgery in patients without spontaneous ventricular tachycardia.

The difference in the extent of ventriculographic wall motion abnormalities (based on a cut-off point of abnormally contracting segment of 60%) was quite sharp between patients with and without spontaneous ventricular tachycardia. The meaning of this difference is not entirely clear. It is reasonable to assume that a larger area of abnormal wall

### Table 1. Angiographic and Hemodynamic Characteristics of 50 Patients

<table>
<thead>
<tr>
<th></th>
<th>No. of Diseased Coronary Vessels</th>
<th>% ACS &gt;40</th>
<th>Ejection Fraction (%)</th>
<th>LVEDP (mm Hg)</th>
<th>PC Wedge Pressure (mm Hg)</th>
<th>Cardiac Index (liters/min per m²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spontaneous VT</td>
<td>Absent (n=10)</td>
<td>2.6 ± 0.5</td>
<td>54 ± 3</td>
<td>28 ± 8</td>
<td>19 ± 8</td>
<td>2.9 ± 0.2</td>
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<td></td>
<td>Present (n=40)</td>
<td>1.9 ± 0.8</td>
<td>56 ± 7</td>
<td>25 ± 9</td>
<td>18 ± 8</td>
<td>2.5 ± 0.6</td>
</tr>
<tr>
<td>p Value</td>
<td>0.026</td>
<td>NS</td>
<td>0.04</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Inducible VT</td>
<td>Absent (n=6)</td>
<td>2.5 ± 0.6</td>
<td>54 ± 4</td>
<td>23 ± 7</td>
<td>21 ± 9</td>
<td>2.8 ± 0</td>
</tr>
<tr>
<td></td>
<td>Present (n=44)</td>
<td>1.9 ± 0.8</td>
<td>56 ± 7</td>
<td>26 ± 9</td>
<td>18 ± 8</td>
<td>2.6 ± 0.6</td>
</tr>
<tr>
<td>p Value</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
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*Defined as >70% luminal narrowing (maximum of three vessels); (see text for definition of excess ejection fraction. ACS = abnormally contracting segment (see text); Cont Seg = contractile segment; LVEDP = left ventricular end-diastolic pressure; PC Wedge = pulmonary capillary wedge; VT = ventricular tachycardia.

### Table 2. Electrocardiographic Characteristics of 50 Patients

<table>
<thead>
<tr>
<th></th>
<th>No. of Sites</th>
<th>% Abnormal</th>
<th>% Frontalized</th>
<th>% Late</th>
<th>% Split</th>
<th>Duration of Longest Electrogram (ms)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spontaneous VT</td>
<td>Absent (n=10)</td>
<td>50 ± 11</td>
<td>55 ± 22</td>
<td>12 ± 8</td>
<td>16 ± 20</td>
<td>3 ± 6</td>
</tr>
<tr>
<td></td>
<td>Present (n=40)</td>
<td>46 ± 10</td>
<td>61 ± 19</td>
<td>20 ± 15</td>
<td>10 ± 11</td>
<td>4 ± 5</td>
</tr>
<tr>
<td>p Value</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Inducible VT</td>
<td>Absent (n=6)</td>
<td>52 ± 6</td>
<td>45 ± 20</td>
<td>9 ± 7</td>
<td>10 ± 7</td>
<td>2 ± 3</td>
</tr>
<tr>
<td></td>
<td>Present (n=44)</td>
<td>46 ± 10</td>
<td>62 ± 17</td>
<td>20 ± 14</td>
<td>12 ± 14</td>
<td>4 ± 5</td>
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<tr>
<td>p Value</td>
<td>NS</td>
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Abbreviations as in Table 1.
motion implies a larger "border zone" between scarred areas and more normal myocardium in which arrhythmogenic areas could exist. The gross assessment of percent abnormally contracting segment, however, includes akinetic as well as dyskinetic areas, and cannot distinguish scar from severely ischemic but viable muscle that contracts abnormally. Additionally, as there was no difference in contractile segment ejection fraction between the groups, it may be that, in patients without spontaneous tachycardia (who underwent surgery earlier after infarction), the ventricle had had less time to dilate than was possible in patients operated on later after infarction. Thus, the usefulness of the difference in apparent aneurysm size is uncertain.

Although the proportions of abnormal and fractionated electrograms were greater in patients with spontaneous ventricular tachycardia, there were no statistically significant differences in electrographic characteristics between patients with and without spontaneous tachycardia. These differences in electrographic proportions were more marked when patients were segregated according to the presence or absence of inducible tachycardia, suggesting that the group of patients without spontaneous tachycardia was a mixture of patients with and without a clinically silent "substrate" for ventricular tachycardia.

Presence versus absence of inducible ventricular tachycardia. The only variables that distinguished patients according to the presence or absence of inducible ventricular tachycardia were 1) time from infarction to surgery, and 2) differences in extent of electrographic abnormalities. The difference in time from infarction to surgery has already been discussed. The differences in electrographic characteristics are of considerable interest for several reasons.

First, although patients with inducible ventricular tachycardia had a greater proportion of abnormal as well as fractionated (but not late or split) electrograms than did those without inducible tachycardia, the differences were quantitative rather than qualitative. All patients in this series had abnormal electrograms, and all but one had fractionated activity. Thus, it was not simply whether these abnormalities were present, but how widespread they were, that distinguished the two groups of patients. Second, because patients with inducible tachycardia had a greater proportion of abnormal and fractionated electrograms, one could hypothesize a causal relation between these electrograms and inducible ventricular tachycardia. It is reasonable to assume that the greater the derangement in the electrophysiologic milieu, the more likely it is for the substrate for ventricular tachycardia to exist. Third, the fact that the four patients without
spontaneous ventricular tachycardia who also had inducible tachycardia displayed electrographic characteristics indistinguishable from those with spontaneous tachycardia suggests that, if observed long enough, these patients might eventually develop spontaneous tachycardia. The factors that determine the timing of the first episode of spontaneous tachycardia are not clear, but these data suggest that the substrate for ventricular tachycardia is present in at least some patients well before the mean time of onset of spontaneous tachycardia (33 months in this series).

Comparison with previous studies. Previous studies in human patients have suggested an association between the presence of "fragmented" (3,7) or late (8) endocardial electrograms and ventricular tachycardia. Although our data are in general agreement with, they are not directly comparable with these previous studies for several reasons:

1) Study group. Most prior series (7,9,10) were composed of patients with nonsustained ventricular tachycardia or ventricular fibrillation, as well as sustained uniform ventricular tachycardia. There is some evidence (11) that nonsustained ventricular tachycardia and ventricular fibrillation may not be associated with the same anatomic or electrophysiologic substrate as sustained ventricular tachycardia. Additionally, prior series have been composed of patients with an anterior or inferior aneurysm. The present study considered a relatively homogeneous group of patients with an anterior left ventricular aneurysm, and evaluated one type of arrhythmia (sustained ventricular tachycardia of uniform configuration).

2) Sampling techniques. In previous studies, the sampling of endocardial electrograms was concentrated in the areas of greatest abnormality (that is, near aneurysm borders) rather than from the entire endocardial surface. The sampling methods used in the present study reduce this bias and give a more accurate overall impression of the extent of electrophysiologic abnormalities.

3) Classification of various electrographic types. This is not uniform in previously reported series, which defined "fragmented" or "fractionated" electrograms as being ≥50 ms in duration/< 1 mV in amplitude (10) or "polyphasic and of low amplitude" without quantitating amplitude or duration (9). In our study, a more strict definition was applied, which required a greater degree of abnormality to be present. It is not possible to determine which, if any, definition is "correct." Definitions of "late" electrograms are similarly varied. In contrast to prior studies (10), in our investigation no predilection of fractionated electrograms for septal sites was observed (Fig. 2), nor was there any association between the duration of the longest electrogram and the presence of ventricular tachycardia. This may be due to more complete sampling of endocardial electrograms in our series.

We are not aware of any prior series in which all patients undergoing aneurysmectomy had extensive endocardial mapping or programmed electrical stimulation as in the current study. Other investigators (7) have suggested that endocardial mapping characteristics during sinus rhythm could be used to guide surgical therapy in cases of sustained ventricular tachycardia. Our data do not bear directly on this question. However, it would seem that this approach offers little advantage because the electrographic differences observed between patient groups were quantitative rather than qualitative (all patients in this series had abnormal electrograms, and all but one patient had fractionated electrograms). Prior work from our laboratory (3,12) also suggests that no particular type of electrogram specifies arrhythmogenic areas.

The signal-averaged ECG may provide an additional tool to help determine which patients merit additional investigation. Unfortunately, signal-averaged ECGs were obtained in too few patients without spontaneous ventricular tachycardia in this study to allow meaningful comparisons. Likewise, although there seemed to be a close correspondence between results of preoperative and intraoperative stimulation in the patients without spontaneous tachycardia, the small number of patients studied precludes drawing any conclusions.

Limitations. There are several potential limitations inherent in the current study. A discussion of the most significant of these follows.

1) Arbitrary definitions of type of electrogram. As noted earlier, there are no "correct" criteria for fractionated, split and late electrograms. Rather, these degrees of abnormality are operationally defined, thus introducing a bias into electrogram interpretation. We attempted to use criteria that can be objectively applied and that we believe allow adequate separation of these types of electrograms.

2) Errors in measurement. Accurate determination of electrogram duration is difficult, especially in low amplitude electrograms in which electrical activity gradually returns to the baseline value. We attempted to mitigate this problem by recording at a rapid paper speed and measuring the same electrogram in each of several cycles, with excellent reproducibility.

3) Assessment of wall motion. This was made using single plane ventriculography, and thus we could not evaluate lateral wall and especially basal septal contractility. Additionally, the assessment of abnormal wall motion was not contingent on whether the segment was infarcted or potentially viable but severely ischemic (13).

4) The presence of small numbers of patients. Especially in those without spontaneous ventricular tachycardia, this limitation renders statistical comparisons difficult in that type II errors (the probability of not detecting a true statistical difference because of small sample size) were quite large (in the 50 to 70% range). Thus, a larger group of patients might have allowed a clearer distinction between
groups, especially in the proportion of electrograms that showed a trend toward significance.

5) The presence of sicker patients. Our group of patients without spontaneous ventricular tachycardia was clearly biased toward "sicker" patients who had more severe coronary disease and underwent surgery earlier after infarction than did the patients with spontaneous tachycardia. It is unclear whether this bias is relevant, except possibly in the size of the ventriculographic percent abnormally contracting segment, as noted previously.

6) Predictive value of induced tachycardia. One of the most difficult issues concerns how well the presence of inducible uniform ventricular tachycardia correlates with the likelihood that spontaneous ventricular tachycardia will develop in patients without any prior history of tachycardia episodes. The limited reported data suggest that, in general, inducible uniform ventricular tachycardia strongly implies the presence of a substrate for ventricular tachycardia because some "induced only" tachycardias have subsequently been shown to recur spontaneously (14,15). It is also possible that the intraoperative inducible ventricular tachycardia in these patients was an artifact of the circumstances of the intraoperative setting (ventriculotomy, temperature) in that additional ventricular tachycardia configurations are often induced during surgery for ventricular tachycardia that have not been observed prior to this setting.

Conclusions. A left ventricular aneurysm is a frequent consequence of anterior wall myocardial infarction and is occasionally associated with the development of spontaneous ventricular tachycardia. We demonstrated that a significant number of patients (4 of 10 in this series) with anterior left ventricular aneurysm and no history of spontaneous ventricular tachycardia have inducible tachycardia in the intraoperative setting. Patients without spontaneous ventricular tachycardia differed from those with spontaneous tachycardia only on the basis of time from infarction to surgery, severity of coronary artery disease, and extent of wall motion abnormalities. The major differences between patients with and without inducible ventricular tachycardia were in electrographic characteristics. Patients with inducible ventricular tachycardia (regardless of the presence of absence of spontaneous tachycardia) had more extensive areas of abnormal and fractionated endocardial electrical activity, although these abnormalities were present to some extent in nearly all patients.

This study suggests that because the pathophysiologic substrate for sustained ventricular tachycardia appears to develop relatively soon after infarction but yet may not become manifest until years later, some other time-dependent factors must be important in determining the onset of spontaneous episodes of tachycardia. These factors might include changes in frequency of tachycardia-initiating events over time, such as ventricular premature depolarizations, as well as changes in autonomic tone and the presence of ischemia or antiarrhythmic drugs.

Implications. These data may apply to patients with a postinfarction left ventricular aneurysm having no history of spontaneous ventricular tachycardia who are undergoing open heart surgery (often with aneurysmectomy). Because there are no preoperative angiographic or hemodynamic features that identify which of these patients may have inducible ventricular tachycardia (substrate), it might be reasonable to consider performing prophylactic "blind" subendocardial resection on these patients at the time of aneurysmectomy (16). Another possible approach would be use of or intraoperative electrophysiologic stimulation to select patients who should undergo subendocardial resection with or without endocardial mapping. The present study does not directly address these questions, however, and further work is needed in this area.

We thank the physicians in the Divisions of Cardiac Anesthesia and Cardiothoracic Surgery and the members of the Division of Cardiothoracic Surgery nursing staff for their assistance and patience in performing these studies. We are also grateful to Nancy Murphy and Carol Davis for excellent secretarial assistance in the preparation of the manuscript.

References


