Subendocardial resection for ventricular tachycardia: predictors of surgical success

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ABSTRACT We retrospectively evaluated the first 100 patients who underwent mapping-guided subendocardial resection (SER) at our hospital for drug-refractory sustained ventricular tachycardia caused by coronary artery disease. There were 91 survivors of surgery with 200 morphologically distinct types of ventricular tachycardia. Eighty-three patients (91%) were cured of ventricular tachycardia by SER alone (60 patients or 66%) or by SER in combination with antiarrhythmic drug therapy (23 patients or 25%) (mean follow-up, 28 ± 19 months). There were four late sudden deaths and four patients continued to have rare episodes of spontaneous ventricular tachycardia after surgery despite receiving antiarrhythmic drugs. Factors associated with failure of SER alone to cure ventricular tachycardia were presence of disparate sites of ventricular tachycardia origin (>5 cm between mapped sites of origin; 64% vs 30% failure rate) and presence of multiple morphologically distinct spontaneous tachycardias (47% vs 25% failure rate). A log-linear model of multivariate analysis identified disparate sites of origin of ventricular tachycardia and the absence of a discrete left ventricular aneurysm as the only independent variables associated with failure of surgery alone. Inferior wall site of origin (41% vs 12% failure) and right bundle branch block morphology of ventricular tachycardia (20% vs 7% failure) were also significantly associated with failure of surgery to cure ventricular tachycardia. Mapping-guided SER is a highly effective mode of treatment for drug-refractory ventricular tachycardia, despite the existence of subgroups of patients with higher-than-average surgical failure rates.


RECURRENT sustained ventricular tachycardia remains a major cause of cardiovascular morbidity and mortality. Antiarrhythmic drug and pacemaker therapy are successful in controlling ventricular tachycardia in a substantial number of patients, but some 40% continue to have recurrent tachycardia despite these interventions. The advent of new techniques has allowed surgery to take on an increasing role in the therapy of ventricular tachyarrhythmias in recent years. Currently, the most frequently used operation is subendocardial resection, in which a 1 to 2 mm thick sheet of endocardium, which contains the site of origin of ventricular tachycardia, is removed. This procedure may be either guided by electrophysiologic mapping, or unguided (removal of all abnormal-appearing endocardium). The present study reports the operative results in 100 patients undergoing subendocardial resection for recurrent ventricular tachycardia and an analysis of factors that predict surgical success or failure in curing patients of their tachycardias.

Materials and methods

Patient population. The study group included the first 100 consecutive patients with prior myocardial infarction and recurrent sustained ventricular tachycardia who underwent subendocardial resection for control of ventricular tachycardia at the Hospital of the University of Pennsylvania between October 1977 and February 1983. There were 85 men and 15 women, with ages ranging from 32 to 78 years (mean 55). Seventy-nine patients had a history of anterior myocardial infarction and 21 had inferior infarction. These and other patient characteristics are listed in table 1. All patients underwent preoperative hemodynamic and electrophysiologic catheterization; during electrophysiologic study (EPS), ventricular tachycardia was induced and endocardial catheter mapping of as many morphologically distinct ventricular tachycardias as possible was performed with the localization schema shown in figure 1 and techniques previously described. All patients in this series had been on numerous antiarrhythmic drug regimens that had failed...
TABLE 1
Patient characteristics (n = 100)

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior MI (n)</td>
<td>79</td>
</tr>
<tr>
<td>Inferior MI (n)</td>
<td>21</td>
</tr>
<tr>
<td>Ejection fraction</td>
<td>28 ± 9</td>
</tr>
<tr>
<td>HV interval (msec)</td>
<td>58 ± 14</td>
</tr>
<tr>
<td>Left ventricular end-diastolic pressure (mm Hg)</td>
<td>17 ± 8</td>
</tr>
<tr>
<td>Cardiac index (l/min/m²)</td>
<td>2.8 ± 0.7</td>
</tr>
<tr>
<td>No. of antiarrhythmics failed</td>
<td>4.7 ± 1.7</td>
</tr>
</tbody>
</table>

**Plus or minus** values are mean ± SD.

MI = myocardial infarction.

*Eighty-five men; 15 women. Mean age 56 ± 10 yr.

(i.e., persistent ventricular tachycardia inducibility during EPS, spontaneous ventricular tachycardia recurrence, or intolerable drug side effects) and/or pacemaker therapy. The surgical procedure consisted of the initiation and electrophysiologic mapping of as many morphologically distinct ventricular tachycardias as possible, epicardially and/or endocardially, during normothermic cardiopulmonary bypass. Once an endocardial site of origin (see definitions below) had been identified for each tachycardiac morpholgy, cold cardioplegia was instituted and subendocardial resection was carried out that included the site of origin and a 1 to 1 1/2 cm margin on all sides and 1 to 2 mm thick. Additional surgical procedures in some patients included coronary artery bypass grafting, aneurysmectomy, and cryoablation of sites of origin not easily accessible to subendocardial resection (table 2). Patients underwent repeated angiographic and electrophysiologic catheterization approximately 10 days after surgery; EPS at that time included rapid ventricular pacing and introduction of single and double (and in 51 patients, triple) ventricular premature depolarizations during at least two paced cycle lengths from two right ventricular and at least one left ventricular site. If a sustained ventricular tachycardia (see definitions) was observed during this study, serial electrophysiologic studies of antiarrhythmic drug effects were performed to assess the ability of drugs to suppress the inducibility of ventricular tachycardia.

In 87 of the 100 patients endocardial catheter maps of 143 distinct types of ventricular tachycardia were obtained and in 96 patients intraoperative maps of 209 ventricular tachycardias were obtained (some mapped both by catheter and in the intraparative period). Four patients did not undergo intraoperative mapping (one because of intraoperative aortic rupture, three because of persistent polymorphic ventricular tachycardia or ventricular fibrillation). Catheter maps were used to guide surgery in these patients as well as in others in whom a previously observed ventricular tachycardia was not observed during surgery (a total of 14 ventricular tachycardias in 11 patients). Six ventricular tachycardias in six patients were never mapped by catheter or during surgery.

There were nine operative deaths. The causes of death were refractory congestive failure (n = 6), perioperative myocardial infarction (n = 2), and unexplained electromechanical dissociation in a patient initially doing well after surgery. These patients had a total of 21 distinct types of ventricular tachycardia, none of which occurred spontaneously before death; however, none of these patients survived to undergo postoperative EPS, and thus no definitive statement can be made concerning the effect of surgery on their arrhythmias. Data on these patients and their ventricular tachycardias were thus excluded from further analysis, leaving 91 patients with 200 ventricular tachycardias. One hundred twenty-nine of the ventricular tachycardias occurred spontaneously before surgery and 71 were observed only after electrophysiologic induction (39 at EPS, 32 intraoperatively).

**Definitions.** Sustained ventricular tachycardia was that lasting more than 30 sec or resulting in loss of consciousness and requiring cardioversion.

Spontaneous and induced ventricular tachycardias were defined as follows. Spontaneous ventricular tachycardia was defined as a spontaneously occurring preoperative ventricular tachycardia and induced ventricular tachycardia was that which was never observed to occur spontaneously but that could be repeatedly induced and sustained in either the electrophysiology laboratory or operating room. All patients had at least one spontaneously occurring ventricular tachycardia.

Ventricular tachycardia morphologies were considered distinct if they displayed contralateral bundle branch block morphology, or if their frontal plane QRS axes were more than 90 degrees divergent.

Site of origin of tachycardia was defined as the endocardial site of earliest electrical activity for a given ventricular tachycardia.

![FIGURE 1. Endocardial catheter mapping schema.](http://circ.ahajournals.org/)

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dia, identified by the earliest local electrogram that was recorded between the midpoint of electrical diastole and the onset of the QRS complex. Sites of origin were grouped for statistical purposes into septal (sites 2, 3, and 4 in figure 1), inferior wall (sites 1, 5, 6, and medial aspects of 7 and 8), and anterior sites (9, 10, 11, 12 and lateral aspects of 7 and 8); papillary muscles were further designated as anterior (9, 7 through 9) and inferior (5, 5 through 7).

Disparate sites of origin of ventricular tachycardia were sites of origin of at least two morphologically distinct ventricular tachycardias that were two sites or more distant from one another (figure 1); this was a distance of 5 cm or more.

Operative death was that occurring within 30 days of the date of surgery.

Spontaneous recurrence of ventricular tachycardia was defined as the postoperative observation of sustained ventricular tachycardia of uniform morphology that occurred spontaneously (not induced by programmed electrical stimulation).

Postoperative tachycardia inducibility was the ability to repeatedly induce ventricular tachycardia of uniform morphology that lasted more than 10 sec during EPS by the above protocol of programmed electrical stimulation.

Surgery alone was considered a failure when there was postoperative recurrence of ventricular tachycardia spontaneously, as a result of programmed stimulation, or as unexplained sudden cardiac death.

The following parameters were analyzed for their predictive capacity: (1) Patient characteristics considered included age, sex, history of cardiac arrest, location of previous myocardial infarction, time elapsed from most recent myocardial infarction, presence of ventricular aneurysm, number of antiarrhythmic drug therapies tried before surgery, prior use of amiodarone, angiographic ejection fraction (EF), HV interval (in msec), left ventricular end-diastolic pressure (LVEDP, in mm Hg), number of morphologies of spontaneous ventricular tachycardia (single or multiple), total number of distinct ventricular tachycardia morphologies (spontaneous or induced), and whether ventricular tachycardias arose from disparate sites during mapping. (2) Characteristics of ventricular tachycardia considered included cycle length (msec), type of bundle branch block morphology (right or left), frontal plane QRS axis (inferior [0 to +179 degrees]; superior [-1 to 180 degrees]), ability to map an early site during ventricular tachycardia (catheter or intraoperative), clinical vs induced ventricular tachycardia, and site of origin, according to regions defined above.

Statistical analyses were performed as follows: All variables were first analyzed singly (univariate analysis) with regard to their correlation with surgical success (defined as above). The unpaired Student t test and Fisher exact test were used when appropriate, with a p < .05 level of significance. Variables that had univariate predictive capacity were then subjected to a multivariate analysis with a log-linear model^2 in order to obtain an overall scheme that would predict surgical outcome.

Results

Patients (figure 2). The overall results of surgery are shown in table 3. There were 91 survivors of surgery; of these, 60 were complete clinical successes (i.e., no ventricular tachycardia, on no medication); 23 additional patients were partial successes (no ventricular tachycardia, but on medication) for an overall success rate of 91% among survivors of surgery. Eighty-nine of the 91 operative survivors underwent postoperative EPS as detailed above (figure 2). Ventricular tachycardia was not inducible in 64 (72%); three of these patients had late spontaneous recurrences of ventricular tachycardia (>6 months after surgery), but have since been free of ventricular tachycardia on antiarrhythmic medications. There were three sudden deaths in this group that were presumed for purposes of this study to be due to recurrent ventricular tachycardia. (Although

![FIGURE 2](image-url) Results of surgery for ventricular tachycardia among survivors of surgery.
TABLE 3
Results of surgery (n = 100)

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Count</th>
</tr>
</thead>
<tbody>
<tr>
<td>Operative deaths</td>
<td>9</td>
</tr>
<tr>
<td>Complete clinical success (no VT, no medications)</td>
<td>60</td>
</tr>
<tr>
<td>Partial clinical success (no VT on medications)</td>
<td>23</td>
</tr>
<tr>
<td>Cardiac arrest</td>
<td>4</td>
</tr>
<tr>
<td>Spontaneous VT despite medications</td>
<td>4</td>
</tr>
</tbody>
</table>

VT = ventricular tachycardia.

these three patients were presumed to have had lethal recurrences of ventricular tachycardia, one patient died suddenly during exertional chest pain, one had a long QT interval [0.7 sec] as a result of the use of quinidine for atrial arrhythmias, and one patient who had more than 15 episodes/year of ventricular tachycardia before surgery died after 29 arrhythmia-free months.) The two patients not undergoing postoperative EPS have been free of spontaneous ventricular tachycardia for a minimum of 3 years each. Twenty-five patients (28%) had inducible ventricular tachycardia after surgery; ventricular tachycardia was of a sustained, previously seen (spontaneous or induced) morphology in 18, a nonsustained, previously seen morphology in two, and a "new" (never seen before or during surgery) morphology in five patients. Ventricular tachycardia was rendered noninducible with antiarrhythmic medication in 10 of these patients; there have been no spontaneous recurrences in this group, which includes three patients who have stopped taking medication. One patient died suddenly 11 months after surgery. The remaining 15 patients still had inducible ventricular tachycardia while on antiarrhythmic medications at the time of hospital discharge; of these, four continue to have rare spontaneous episodes of ventricular tachycardia.

Fourteen patients had recurrences of spontaneous ventricular tachycardia at some time after surgery; in nine, ventricular tachycardia recurred before hospital discharge and all of these patients had inducible ventricular tachycardia after surgery. Three had no inducible ventricular tachycardia while on drugs. Seven patients had documented recurrences of ventricular tachycardia after hospital discharge (four patients from the "inducible" group and three from the "noninducible" group). Overall, four patients (3%) continue to have drug-refractory ventricular tachycardia spontaneously. Long-term follow-up of these patients (free of ventricular tachycardia while on antiarrhythmic drugs) was 28 ± 19 months.

There have been 24 late deaths. Causes of death were congestive heart failure (n = 10), recurrent myocardial infarction (n = 5), sudden death presumed to be secondary to ventricular tachycardia (n = 4), and miscellaneous causes (n = 5). Recurrence of ventricular tachycardia was not implicated in any of the non-sudden late deaths.

Ventricular tachycardias (figure 3). The 91 surgical survivors had 200 morphologically distinct ventricular tachycardias; 129 occurred spontaneously, and 71 were induced only (EPS or intraoperatively). One hundred ten of the spontaneous ventricular tachycardias
and 61 of those induced were eliminated by surgery alone (85% each). An additional 10 spontaneous and three induced ventricular tachycardias were rendered noninducible in patients on antiarrhythmic medication (total of 120 spontaneous and 64 induced ventricular tachycardias, 93% and 90%, respectively). Four ventricular tachycardias (2%; two spontaneous, two induced) recurred spontaneously despite antiarrhythmic drug therapy.

The sites of stimulation from which ventricular tachycardia was induced after surgery are shown in figure 4. Ventricular tachycardia was first observed during stimulation at the right ventricular apex in 18 patients (72%), a second right ventricular site was required in another three patients (12%), and in four patients (16%) left ventricular stimulation was necessary to induce ventricular tachycardia.

Ventricular tachycardia was induced with single or double extrastimuli from the right ventricle (apex or outflow tract) in 16 patients (64%). Five patients required triple extrastimuli, and one quadruple extrastimuli from a right ventricular site, for induction of ventricular tachycardia after surgery. In all patients with ventricular tachycardia induced only by left ventricular stimulation double or triple extrastimuli were required.

Patient characteristics (table 4). Variables that were significantly associated with failure of surgery alone to cure a patient of ventricular tachycardia were presence of disparate sites of ventricular tachycardia origin (64% failure of surgery alone as opposed to 30% for patients with concordant sites of ventricular tachycardia origin; p = .03) and presence of multiple morphologically distinct spontaneous ventricular tachycardias (47% failure of surgery alone as opposed to 25% for patients with a single clinical ventricular tachycardia; p = .04). None of the other variables were significantly associated with failure of surgery alone.

In the multivariate analysis (Appendix), the strongest predictor of failure of surgery alone to cure a patient of all ventricular tachycardias was disparate sites of ventricular tachycardia origin (coefficient 0.973; a secondary independent variable was the absence of a discrete left ventricular aneurysm [coefficient (−)0.896].

Characteristics of ventricular tachycardia (table 5). The variable that was most significantly associated with the failure of surgery alone to eradicate a specific ventricular tachycardia morphology was the site of origin of ventricular tachycardia. The greatest difference was between inferior wall and noninferior wall site of origin (41% vs 12% rate of failure of surgery alone fail-

### TABLE 4
Predictors of surgical success — patient characteristics (n = 91)

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Present</th>
<th>Absent</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Disparate sites of VT origin</td>
<td>7/11 (64)%</td>
<td>24/80 (30)%</td>
<td>.03</td>
</tr>
<tr>
<td>Multiple VT morphologies</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Spontaneous</td>
<td>17/36 (47)%</td>
<td>14/55 (24)%</td>
<td>.04</td>
</tr>
<tr>
<td>Spontaneous and induced</td>
<td>28/73 (38)%</td>
<td>3/18 (17)%</td>
<td>NS</td>
</tr>
<tr>
<td>History of cardiac arrest</td>
<td>10/23 (43)%</td>
<td>21/68 (31)%</td>
<td>NS</td>
</tr>
<tr>
<td>Location of MI</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anterior</td>
<td>21/71 (30)%</td>
<td>10/20 (50)%</td>
<td>NS</td>
</tr>
<tr>
<td>Inferior</td>
<td>10/20 (50)%</td>
<td>21/71 (30)%</td>
<td>NS</td>
</tr>
<tr>
<td>Left ventricular aneurysm</td>
<td>25/82 (30)%</td>
<td>6/9 (67)%</td>
<td>NS</td>
</tr>
<tr>
<td>Prior treatment with amiodarone</td>
<td>12/24 (50)%</td>
<td>19/67 (18)%</td>
<td>NS</td>
</tr>
</tbody>
</table>

VT = ventricular tachycardia; MI = myocardial infarction.

^Numbers in parentheses are percentages.

### TABLE 5
Predictors of surgical success — characteristics of ventricular tachycardia (VT) (n = 194)

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>VT's with characteristic</th>
<th>VT's without characteristic</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Site of VT origin</td>
<td>16/149 (11)%</td>
<td>13/45 (27)%</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Septum</td>
<td>4/23 (17)%</td>
<td>25/171 (15)%</td>
<td>NS</td>
</tr>
<tr>
<td>Anterior</td>
<td>9/22 (41)%</td>
<td>20/172 (12)%</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Inferior</td>
<td>5/11 (45)%</td>
<td>24/183 (13)%</td>
<td>.02</td>
</tr>
<tr>
<td>Nonpapillary muscle</td>
<td>6/17 (32)%</td>
<td>23/177 (15)%</td>
<td>&lt;.05</td>
</tr>
<tr>
<td>Papillary muscles (both)</td>
<td>24/121 (20)%</td>
<td>5/73 (7)%</td>
<td>&lt;.05</td>
</tr>
<tr>
<td>VT morphology</td>
<td>12/72 (16)%</td>
<td>17/122 (14)%</td>
<td>NS</td>
</tr>
</tbody>
</table>

RBBB = right bundle branch block.

^Numbers in parentheses denote percent.
ure; p < .001). Secondary were inferior wall not involving the papillary muscle (45% vs 13% failure rate for surgery alone from other sites; p < .02), nonseptal vs septal site of origin (29% vs 11%; p < .01), and site of origin in either papillary muscle (32% vs 15%; p < .05). Additionally, a bundle branch block type of morphology predicted outcome in that 20% of right bundle branch block ventricular tachycardias vs 7% of left bundle branch block ventricular tachycardias were not cured by surgery alone (p < .05).

Fourteen ventricular tachycardias (in 11 patients) were mapped by catheter but not during surgery. One of these patients (with two types of ventricular tachycardia) died before the postoperative EPS. In the remaining patients, two tachycardias in two patients were not cured by surgery alone, so that catheter mapping data successfully guided surgical treatment for 10 of 12 ventricular tachycardias, as assessed by postoperative EPS.

Ventricular tachycardia that was induced after surgery, while of the same morphology as preoperative or intraoperative tachycardia, was generally more difficult to induce than it had been before surgery. Additionally, half of these postoperatively inducible tachycardias responded to antiarrhythmic medications, whereas none had been responsive to drug therapy before surgery. There were five new ventricular tachycardias seen postoperatively (of morphologies never observed before), the significance of which is unclear, but each patient in whom they occurred had been on amiodarone before surgery. All of these new ventricular tachycardias were rendered noninducible in patients on drugs and none of these have recurred spontaneously in those on antiarrhythmic regimens.

Discussion

Subendocardial resection is of proven value in the treatment of drug-refractory ventricular tachycardia.8 This is underscored by the fact that, despite a postoperative ventricular tachycardia inducibility rate of 28% in the 91 patients in this series, only four continued to have rare recurrent episodes of spontaneous ventricular tachycardia unresponsive to antiarrhythmics and only an additional four had cardiac arrests (three were in patients who had had no prior recurrence of ventricular tachycardia). The disparity between these figures may in part be due to the in-depth postoperative EPS, which includes left ventricular stimulation from at least one site as well as the standard right ventricular stimulation protocol. Five of the patients in this series who had inducible ventricular tachycardia after surgery would not have had them if stimulation had ceased after no ventricular tachycardia was induced from the right ventricle by a standard stimulation protocol (i.e., up to three ventricular premature depolarizations at several paced cycle lengths from two right ventricular sites; figure 4). In that these ventricular tachycardias were almost always morphologically similar to preoperative tachycardias, we still regarded them as cases of failure of surgery alone.

Two general patient characteristics were highly associated with the inability of surgery to cure patients of all types of ventricular tachycardias: the presence of disparate sites of ventricular tachycardia origin and the presence of spontaneously occurring multiple morphologically distinct types of ventricular tachycardia. There may be a common reason behind these findings — that is, patients with larger areas of ventricular muscle damage as a result of prior infarction may have a greater mass of tissue within which the proper electrophysiologic substrate for reentry may exist. These data suggest that the use of a wider resection margin may be necessary in these subgroups of patients with multiple morphologically distinct types of ventricular tachycardia and/or disparate sites of origin of tachycardia.

In the multivariate analysis, the presence of disparate sites of origin of ventricular tachycardia was the strongest predictor of failure of surgery alone. The presence of multiple morphologically distinct types of ventricular tachycardia was not identified as an independent predictor, although obviously patients had to have at least two types of ventricular tachycardia in order to have disparate sites of origin, and thus these factors are not entirely independent. The multivariate analysis also identified the absence of a discrete left ventricular aneurysm as a secondary predictor of failure of surgery alone. This reflects the higher rate of surgical failure for tachycardias in the inferior wall since all of the patients who lacked discrete aneurysms had inferior infarctions.

Factors that predicted the failure of subendocardial resection to cure a given ventricular tachycardia in this series were mainly related to site of origin of the tachycardia, as determined by mapping (catheter or intraoperative). The region from which ventricular tachycardia was least likely to be cured, the inferior free wall (especially the area not involving the papillary muscle), presents more technical difficulties for the surgeon than do other areas. The endocardium of this region is highly trabeculated, resulting in greater difficulty in peeling a homogeneous layer of tissue. This is in distinct contrast to the septum and, to a lesser extent,
to the anterior free wall, which are usually smoother. The proximity of the mitral valve apparatus (papillary muscle, chordae tendineae, mitral valve leaflets, and annulus) also render the inferior free wall a difficult area in which to perform endocardial resection, and perhaps these factors and their relationship to the ability to obtain the proper amount of resection specimen explain the failure rate of surgery for ventricular tachycardias arising from this region. These tachycardias appear superficially to have no peculiar characteristics to distinguish them from those arising from other areas (no difference in rate, mode of induction, etc.). However, there may be some difference in the substrate from which they arise; in our studies the inferior wall infarctions tended not to encompass as large an area as did anterior infarctions, and often (30%) there was no aneurysm at all. Some patients with inferior wall infarctions had minimal wall thinning and little endocardial scar. It is noteworthy that these ventricular tachycardias were no more difficult to map than those from other areas, and thus localization of the area to be resected was not a problem.

Ventricular tachycardia arising in a papillary muscle was also less frequently cured by surgery alone than that originating in other areas. We did not replace mitral valves because of origin of ventricular tachycardia in a papillary muscle, although one mitral valve was replaced for other indications. We have recently used cryothermal ablation (at $-60^\circ$ C for 3 min) of the base of the involved papillary muscle and obtained better results than those with subendocardial resection alone.

There was also a higher rate of surgical failure in patients with ventricular tachycardias of a right bundle branch block morphology. This is also likely to be related to the location of site of origin, in that virtually all left bundle branch block ventricular tachycardias arose in the septum (the region with the lowest failure rate), whereas only 60% of right bundle branch block ventricular tachycardias arose in the septum, and that all tachycardias arising in the free walls (with their higher failure rates) were of right bundle branch block morphology. Thus, of the ventricular tachycardia-related factors analyzed, only site of origin and the closely related bundle branch block morphology had a bearing on surgical outcome.

Finally, it is of note that ventricular tachycardias that were induced only (EPS or intraoperatively) but never documented spontaneously recurred equally as often as clinical ventricular tachycardias after surgery (inducibility or spontaneous VT recurrence; figure 3). These induced-only tachycardias are as clinically significant as the "clinical" ventricular tachycardia, and cannot be dismissed simply as artifacts of stimulation.

Implications. The results of this study underscore the importance of preoperative endocardial ventricular tachycardia catheter mapping in that no other currently available technique could reveal sites of origin of ventricular tachycardia in the inferior free wall or two tachycardias having disparate sites of origin. Additionally, preoperative endocardial catheter mapping of tachycardia was instrumental in guiding subendocardial resection in treatment for 14 ventricular tachycardias in 11 patients in whom these tachycardias were not observed during surgery.

In cases in which unfavorable preoperative predictors exist, it may be that greater thought should be given to alternative forms of therapy (drug combinations, pacemaker, automatic internal defibrillator) and that subendocardial resection in these cases might be viewed less favorably. This is not to say that surgery should be avoided in these patients. We believe that a modification in surgical approach (i.e., larger endocardial resection, adjunctive local cryothermal ablation, or encircling endocardial cryoablation) may make these patients as likely to be cured by subendocardial resection as patients having only favorable predictors. Selected use of encircling endocardial ventriculotomy may also be indicated in some cases. Despite these predictors of surgical failure, subendocardial resection remains a very successful technique in the management of recurrent ventricular tachycardia overall, with an end result of 3% of patients having postoperative cardiac arrest, 3% having documented spontaneous recurrence of ventricular tachycardia despite drugs, and a 5 year actuarial survival of 62%.

We thank Mrs. Carol Dresden for advice on statistical analysis and Maria Coscia and Nancy Walker for excellent secretarial assistance.

References

THERAPY AND PREVENTION—VENTRICULAR TACHYCARDIA


Appendix: Details of the multivariate analysis

A log-linear multivariate regression analysis was performed on the following patient-related variables: presence of disparate sites of origin of ventricular tachycardia, presence of multiple morphologically distinct types of ventricular tachycardia (spontaneous, or spontaneous + induced), presence of left ventricular aneurysm, prior treatment with amiodarone, and history of cardiac arrest. The equation derived follows the format

$$\text{Prob}(x) = \frac{e^{\text{logit}_1}}{1 + e^{\text{logit}_u}}$$

where $x$ is failure of surgery alone and $\text{logit}_u$ is $b + C_1 Y_1 + C_2 Y_2 + \ldots + C_n Y_n$ where $b$ is a derived constant, $C_1$ is the coefficient for variable 1, $C_2$ is the coefficient for variable 2, and so on, and $Y$ is (+1) if the characteristic is present and $Y$ is (−1) if the characteristic is absent.

In this analysis, the constant ($b$) = 0.770; $C_1$ (coefficient for presence of disparate sites of ventricular tachycardia origin) = 0.973; and $C_2$ (coefficient for presence of a discrete aneurysm) = (−0.896). For example, in a patient with disparate sites of ventricular tachycardia origin and an aneurysm

$$\text{Prob} \text{ of failure of surgery alone} = \frac{e^{0.770} + (0.973)(1) + (-0.896)(1)}{1 + e^{0.770} + (0.973)(1) + (-0.896)(1)} = e^{0.847}$$

or a 70% probability of failure of surgery alone for a patient with these characteristics.
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