Surgery for Ventricular Tachycardia: Efficacy of Left Ventricular Aneurysm Resection Compared with Operation Guided by Electrical Activation Mapping


SUMMARY Sixty-five patients underwent surgery for recurrent ventricular tachyarrhythmias. The 32 patients in group 1 underwent simple left ventricular aneurysm resection. The 33 patients in group 2 underwent myocardial resection or incision guided by intraoperative mapping of the electrical activation sequence. The clinical, hemodynamic and angiographic characteristics of the two groups were similar. Although actuarial survival in the two groups was similar through 24 months, late attrition in group 1 patients has left only 21 ± 13% (± SEM) alive by life-table analysis at 94 months. Arrhythmia recurrence has been greater in group 1 than in group 2. In group 1, 50 ± 9% at 1 month and 56 ± 9% of patients at risk at 3 months had recurrences by actuarial analysis. In group 2, only 13 ± 6% at 1 month, 17 ± 7% at 3 months and 29 ± 9% at 24 months relapsed. Death was caused by ventricular tachyarrhythmias in 12 of the 17 patients (71%) who died in group 1, but only three of 12 (25%) who died in group 2.

We conclude that surgery of the left ventricle, guided and modified by intraoperative mapping of the electrical activation sequence, frequently eliminates ventricular tachyarrhythmias and may be more effective than blind resection of left ventricular aneurysm.

RECURRENT ventricular tachycardia (VT) is usually a complication of coronary artery disease with prior myocardial infarction. In 1959, Couch reported that removal of aneurysmal left ventricular scar eliminated a patient's paroxysmal VT. Subsequent reports emphasized the antiarrhythmic efficacy of aneurysm resection alone or in combination with coronary artery bypass grafting. However, other series have been distinguished by early and late failure to eliminate ventricular arrhythmias. In recent studies, arrhythmia was controlled by surgical therapy directed to specific myocardial sites by mapping of the electrical activation sequence. This directed approach might be superior to blind left ventricular aneurysm resection. We compared the antiarrhythmic efficacies of routine left ventricular aneurysm resection and of surgical therapy guided by electrical activation mapping in patients with prior myocardial infarction.

Materials and Methods

Patient Selection

Thirty-two patients with ventricular arrhythmias underwent routine, or blind, left ventricular aneurysm resection (group 1). Thirty-three patients underwent intraoperative electrical activation sequence mapping during VT; the site of myocardial resection or incision was based on the results of mapping (group 2). Patients in group 1 underwent surgery before July 1, 1979; patients in group 2 underwent surgery after that date. Patients were included if they fulfilled four selection criteria: (1) the primary indication for surgery was VT or ventricular fibrillation (VF) that had occurred at least twice; (2) coronary artery disease, prior myocardial infarction and segmental left ventricular contraction abnormalities were present; (3) acute myocardial infarction had not occurred within 3 weeks of surgery; and (4) myocardial resection or incision was performed at operation.

Additional inclusion criteria were used for group 2 only: recurrent VT had been unresponsive to therapeutic concentrations of at least four antiarrhythmic agents and selection of the site of left ventricular myocardial resection or incision or both was based on intraoperative electrical mapping.

Tachyarrhythmias were documented at least twice by electrocardiographic recordings in all 65 patients. Prior myocardial infarction was diagnosed on the basis of evolutionary electrocardiographic and serum enzyme changes or by pathologic Q waves consistent with transmural myocardial infarction. Coronary artery disease and segmental left ventricular contraction abnormalities were documented in each case by coronary and left ventricular angiography.

Thirty of the 33 group 1 patients were included in a previous report of the results of conventional surgical therapy of recurrent ventricular tachyarrhythmias in 56 patients. Three additional patients who fulfilled the specified criteria for group 1 underwent surgery after completion of that report. Twenty-six patients in the original report were excluded from the present study: Seven underwent coronary artery bypass grafting alone; nine had suffered acute myocardial infarc-
tion within 3 weeks of surgery; two had had only a single documented episode of sustained ventricular tachyarrhythmia before surgery; two were not operated on primarily for tachyarrhythmias (one for congestive heart failure and one for unstable angina pectoris), though VT had occurred in both; and six did not fulfill our criteria for unequivocally documented ventricular tachyarrhythmias, because electrocardiographic recordings showing wide-complex tachyarrhythmias had not been obtained by or were not available to us from referral sources at the time of this analysis.

Operative Techniques

Blind Left Ventricular Aneurysm Resection

Left ventricular aneurysms were resected during mildly hypothermic cardiopulmonary bypass using standard techniques. Myocardial preservation during periods of cross-clamping was provided by topical hypothermia (4°C saline lavage) alone or in combination with a single 500-ml bolus of hyperkalemic, hypothermic cardioplegia solution infused into the aortic root. All patients in group 2 and two patients in group 1 received cardioplegia. Concomitant coronary artery bypass grafting, if performed, was carried out during the single cross-clamp interval.

Surgery Guided by Mapping

After cannulation for cardiopulmonary bypass, quadripolar electrode plagues with 2-mm interpolar distances were sutured to normal-appearing epicardial sites on the right and left ventricles. Reference electrograms were recorded with two of the electrodes and pacing was done with the other pair. Normothermic cardiopulmonary bypass was initiated and VT was induced by right or left ventricular extrastimulation. Ventricular tachycardia was induced at operation in all 33 group 2 patients. Epicardial mapping of 49 sites encompassing the entire ventricular epicardial surfaces was performed with a finger-held bipolar probe. The left ventricle was then opened through aneurysm or scar and complete endocardial mapping of 24–72 sites was carried out during VT. After endocardial mapping, myocardial resection or incision was performed. If this did not eliminate established and inducible VT, additional endocardial mapping and myocardial resection or incision were repeated until the patient's typical VT could no longer be induced. The aorta was then clamped and systemic and local cooling begun. Concomitant mitral valve replacement or coronary artery bypass grafting was performed before closure of the ventricle. In one patient, a postinfarction ventricular septal defect was repaired before endocardial mapping.

One or more of four operative procedures were based on findings from activation sequence mapping. Myocardial resections were performed if one or two separate sites of early (pre-QRS) electrical activity were found during VT of one or more morphologies. These myocardial resections, containing the sites of earliest electrical activity, consisted of endocardial and subendocardial tissue 2–6-mm thick and of cross-sectional area of 1–15 cm². A complete encircling endoventriculotomy was performed when mapping failed to identify one or two discrete sites of early activity during ventricular tachycardia. The third operative technique, partially encircling endoventriculotomy, when deemed technically feasible, was carried out around the portion of the endocardial perimeter of aneurysm not removed by subendocardial resection. The practice of routinely attempting partially encircling endoventriculotomy after myocardial resection was abandoned after the first 10 patients because of frequent technical infeasibility and our impression that it compromised left ventricular function. The fourth technique, simple transmural ventriculotomy, was performed when earliest electrical activity during tachycardia was near the center of the aneurysmal scar (in two subjects) or at another free-wall epicardial site (in one patient).

All patients in both groups who survived surgery left the operating room receiving an i.v. lidocaine drip, which was discontinued after 36–48 hours in stable patients.

Quality of Mapping

A scoring system was used to assess the quality of a patient's electrical activation sequence mapping data. The maximal score for data of high quality was 4 and the lowest score was 0. Points were deducted from 4 to arrive at the mapping score as follows: Two points were deducted if mapping was incomplete as a result of the inability to obtain a full endocardial mapping study (unless epicardial mapping had identified an early free-wall site, incision through which terminated and rendered VT uninducible). One point was subtracted if the VT rate or surface lead QRS morphologies were such that the onset and end of the QRS complex could not be clearly distinguished and required an assumption to be made regarding QRS onset to permit analysis. One point was deducted if more than two VT morphologies were induced intraoperatively, at least one of which had not been seen preoperatively, and if these morphologies resulted in more than two sites of earliest electrical activity at least 2 cm apart during VT. New morphologies of ventricular tachycardia were identified if the major QRS vector in one or more leads changed by more than 90° with the heart in its normal position in the chest, and if the time between right and left ventricular reference electrograms changed by more than 15 msec.

Follow-up

Follow-up was retrospective in group 1 and prospective in group 2. Survival, arrhythmia recurrence, antiarrhythmic drug therapy and cause of death in nonsurvivors were determined. Ambulatory 24-hour ECG monitoring was performed in all patients who survived beyond 1974. Twenty-five group 2 patients underwent VT induction studies before discharge from the hospital. No group 1 patients were so studied.

The duration of follow-up was different for the two groups. Survival from operation until completion of follow-up was 0–94 months in group 1 and 0–24
months in group 2. Total follow-up was 689 patient-months in group 1 and 374 patient-months in group 2. In group 1, the mean follow-up was 21.5 ± 24.1 months for all patients and 42.4 ± 27.4 months for the 24 patients who survived hospitalization. In group 2, the mean follow-up was 11.0 ± 7.8 months for all patients and 14.4 ± 5.8 months for the 25 who survived hospitalization.

Postoperative arrhythmia recurrence was recorded if sustained VT or VF occurred as a primary event (that is, not a result of preceding hemodynamic collapse). Using our original criteria, episodes of VT lasting six beats or longer were considered arrhythmia recurrences. However, all patients with short runs of VT postoperatively also had sustained VT or VF lasting 30 seconds or longer or requiring immediate cardioversion.

**Statistical Analysis**

Life-table analysis was performed by the technique of Cutler and Ederer. Standard errors for the cumulative proportions in the life-table analyses were determined by the method of Greenwood. Using the standard errors, actuarial differences in the two groups were statistically analyzed using the formula

\[ Z = \frac{P_x - P'_x}{\sqrt{SE(P_x)^2 + SE(P'_x)^2}} \]

where \( P_x \) is the cumulative proportion surviving or without arrhythmia recurrence at the \( x \) follow-up interval, \( SE(P_x) \) is the standard error of \( P_x \), and \( P'_x \) is the comparison proportion. Other statistical comparisons were performed using Fisher’s exact 2 × 2 contingency test or the \( t \) test for unpaired samples.

**Results**

**Clinical Data**

Clinical characteristics of the 65 patients in group 1 and 2 are shown in tables 1 and 2. The groups are similar in age, gender, severity of congestive heart failure, incidence of recent myocardial infarction, and presence of angina pectoris. There is a similar incidence of one-, two- and three-vessel coronary artery disease. Both groups had predominantly anterior left ventricular contraction abnormalities (75% in group 1 and 61% in group 2). There was a greater incidence of posterior left ventricular dyskinesis or aneurysm formation in the map-directed surgical group (33% vs 12.5%). The angiographic performance of the remainder of the left ventricle, that is, of those segments not involved in the infarcted, noncontractile segments, was evaluated subjectively. There was a similar distribution in the two groups of normal and impaired performance of uninfarcted left ventricular segments, and the hemodynamic variables were also similar.

There were important differences between groups in arrhythmia characteristics (table 2). In group 1, 28 patients had recurrent VT and four VF. All patients in group 2 (as defined by selection criteria for map-directed surgical intervention) had recurrent VT and had not responded, because of arrhythmia recurrence.

**Table 1. Clinical, Angiographic and Hemodynamic Characteristics**

<table>
<thead>
<tr>
<th></th>
<th>Group 1</th>
<th>Group 2</th>
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<tbody>
<tr>
<td>n</td>
<td>32</td>
<td>33*</td>
</tr>
<tr>
<td>Age (years)</td>
<td>57 ± 10</td>
<td>60 ± 9</td>
</tr>
<tr>
<td>Sex (male:female)</td>
<td>3.6:1</td>
<td>5.6:1</td>
</tr>
<tr>
<td>CHF class†</td>
<td></td>
<td></td>
</tr>
<tr>
<td>I</td>
<td>16%</td>
<td>3%</td>
</tr>
<tr>
<td>II</td>
<td>41%</td>
<td>48%</td>
</tr>
<tr>
<td>III</td>
<td>25%</td>
<td>36%</td>
</tr>
<tr>
<td>IV</td>
<td>19%</td>
<td>12%</td>
</tr>
<tr>
<td>MI within 6 weeks</td>
<td>19%</td>
<td>18%</td>
</tr>
<tr>
<td>Angina present</td>
<td>17%</td>
<td>30%</td>
</tr>
<tr>
<td>Coronary disease‡</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3-vessel</td>
<td>47%</td>
<td>43%</td>
</tr>
<tr>
<td>2-vessel</td>
<td>22%</td>
<td>24%</td>
</tr>
<tr>
<td>1-vessel</td>
<td>31%</td>
<td>33%</td>
</tr>
<tr>
<td>Performance of uninfarcted LV segments‡</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>37%</td>
<td>36%</td>
</tr>
<tr>
<td>Mild or moderate hypokines</td>
<td>41%</td>
<td>39%</td>
</tr>
<tr>
<td>Severe hypokines</td>
<td>22%</td>
<td>24%</td>
</tr>
<tr>
<td>Hemodynamic (mean ± SD)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVEDP (mm Hg)</td>
<td>20 ± 10</td>
<td>18 ± 9</td>
</tr>
<tr>
<td>CI (l/min/m²)</td>
<td>2.7 ± 0.8</td>
<td>2.7 ± 0.5</td>
</tr>
</tbody>
</table>

*There were no statistically significant differences in any of the listed variables between the two groups.
†Stenosis > 70%.
‡Judged subjectively by visual inspection of the angiograms.
Abbreviations: CHF = congestive heart failure; MI = myocardial infarction; LV = left ventricular; EDP = end-diastolic pressure; CI = cardiac index.

or intolerable side effects, to adequate antiarrhythmic drug therapy, which included at least four clinical antiarrhythmic drug trials in which plasma concentrations or dosages were adequate. Only nine of the group 1 patients (28%) had been treated with and did

**Table 2. Preoperative Arrhythmia Characteristics**

<table>
<thead>
<tr>
<th></th>
<th>Group 1</th>
<th>Group 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>VT*</td>
<td>28 (87.5%)</td>
<td>33 (100%)</td>
</tr>
<tr>
<td>VF†</td>
<td>4 (12.5%)</td>
<td>0</td>
</tr>
<tr>
<td>Unresponsive to adequate drug therapy‡</td>
<td>9 (28%)</td>
<td>33 (100%)</td>
</tr>
<tr>
<td>Unresponsive to inadequate drug therapy‡</td>
<td>7 (22%)</td>
<td>0</td>
</tr>
<tr>
<td>Responsive to drug therapy‡</td>
<td>16 (50%)</td>
<td>0</td>
</tr>
<tr>
<td>Time from last episode of VT to surgery (days)‡</td>
<td>14.47 ± 22.44</td>
<td>3.03 ± 5.24</td>
</tr>
</tbody>
</table>

*Some patients with recurrent VT had episodes of degeneration of VT to VF.
†Sustained VT never documented.
‡p < 0.005; other comparisons not significantly different.
§Induced arrhythmias excluded.
Abbreviations: VT = ventricular tachycardia; VF = ventricular fibrillation.
not respond to adequate plasma concentrations or dosages of two or more antiarrhythmic agents. Seven patients in group 1 (22%) may have failed to respond to antiarrhythmic therapy because the plasma concentration or dosage was low. Sixteen group 1 patients (50%) appeared to have responded (nonrecurrence of VT or VF for at least 3 weeks) to the most recent antiarrhythmic drug regimen before left ventricular aneurysm resection. The time from the most recent tachyarrhythmia to surgery was significantly different in the two groups (14 ± 22 days in group 1 and 3 ± 5 days in group 2, p < 0.005). Thus, medical therapy of group 1 patients was less extensive than that of group 2 patients. The ventricular tachyarrhythmias of the map-directed surgical patients may have been more refractory than those of group 1 patients, but this possibility cannot be unequivocally confirmed.

Surgical Procedures

Group 1 patients, by definition, underwent simple left ventricular aneurysm or scar resection without intraoperative mapping. Twenty-one (66%) had concomitant coronary artery bypass grafting.

Specific surgical procedures were performed on group 2 patients, based on findings from activation sequence mapping (fig. 1). Twenty-eight of the 33 patients underwent resection of myocardial tissue (in addition to the usual trimming of the edges of the ventriculotomy done to facilitate closure of the ventricle). In 23, myocardial resection alone was performed; in four, myocardial resection was combined with endoventriculotomy, which encircled the remaining border of the aneurysm not involved by the resection in three of the patients and which was placed through a region of early electrical activity in one patient. The remaining patient who underwent myocardial resection received a free-wall, transmural ventriculotomy at the site of a subsidiary early focus found after closure of the original ventriculotomy. In the five remaining patients, incisions were performed without resection of myocardial tissue. The incision was an encircling endoventriculotomy in three and in the ventriculotomy used to enter the left ventricle in two. In these latter two cases, early electrical activity during VT was recorded from epicardial tissue near the center of the left ventricular aneurysm, and in both cases the ventriculotomy that passed through these early sites rendered VT no longer inducible.

Concomitant coronary artery bypass grafting was performed in 16 of the group 2 patients (48%). Mitral valve replacement was required in three of them, in one case because of preoperative mitral valve incompetence and in the other two because of necessary inclusion of portions of papillary muscle in the region of myocardial resection.

Survival

Seventeen patients in group 1 (53%) and 12 patients in group 2 (36%) died during follow-up. The 17 deaths in group 1 were caused by ventricular tachyarrhythmias in 12 patients, left ventricular failure in four, and cancer in one. The 12 deaths in group 2 resulted from VT or VF in three patients, left ventricular failure in eight, and exsanguination upon pulmonary catheter removal in one.

Although there were more deaths in group 1, actuarial analysis of survival (fig. 2) did not demonstrate a significant difference in survival rates between the two groups through the 24 months of follow-up available for group 2 patients. At 24 months, the survival rate in group 2 was 63 ± 8% (± SEM). In group 1, survival was 61 ± 8% at 36 months and 21 ± 13% at 94 months.

Arrhythmia Recurrence

Spontaneous VT or VF recurred postoperatively in 22 group 1 patients (69%), but in only eight group 2 patients (24%) (table 3). In both groups, VT was the dominant recurring arrhythmia after surgery. Unmonitored sudden deaths were considered to be due to tachyarrhythmia recurrences. Actuarial analysis of these incidence rates (fig. 3) revealed a highly significant difference in arrhythmia recurrences in the two groups through the 24 months available for comparison. At 1 month, 50 ± 9% of group 1 patients, but only 13 ± 6% of group 2 patients, had had a recurrence of VT or VF by actuarial analysis. At 3 months, the figures were 56 ± 9% and 17 ± 7%, respectively. At 24 months, 71 ± 9% of patients in group 2 remained free of arrhythmia recurrence. At 94 months, all patients at risk in group 1 had had a recurrence of VT or VF. If the two patients who had no spontaneous recurrence of VT, but did have inducible VT, are considered to have had arrhythmia recurrences within 1 month, the actuarial figures for group 2 are only modestly changed to nonrecurrence rates of 80 ± 7%, 76 ± 8% and 64 ± 9% at 1, 3 and 24 months, respectively, and the significant differences between the two groups persist.

Postoperative antiarrhythmic drug therapy also differed between the two groups (table 3). In group 1,
Quality of Mapping Related to Arrhythmia Recurrence in Group 2

The mean quality of mapping score was 3.42 ± 1.00 (± sd) for group 2 patients, 2.75 ± 1.39 for the eight patients with postoperative recurrence of VT or VF and 3.64 ± 0.76 for the 25 without VT or VF. The mean score for the former patients was significantly lower (p = 0.026). Three of the eight patients with postoperative VT or VF had perfect mapping scores (4 points) and three had low mapping scores (2 points in two and 0 in one). From among all group 2 patients, seven had poor mapping scores (2 points or lower). Four of the seven patients nevertheless had a good operative result with regard to arrhythmia recurrence. Five of the 25 patients with a mapping score 3 points or more had a recurrence of VT or VF.

Findings Associated with Postoperative Death Due to Congestive Heart Failure

Twelve patients, four from group 1 and eight from group 2, died from congestive heart failure within 49 months after operation. These patients had poorer left ventricular function than the other 55 patients. All but two were in New York Heart Association classes III and IV preoperatively. None of them had good angiographic performance of left ventricular segments not involved by aneurysm or scar; four of them had poor and six fair function of these segments. The mean left ventricular end-diastolic pressure was 20 ± 11 mm Hg and cardiac index was 2.3 ± 0.64 ml/min/m² for this group of patients. Although their left ventricular performance was poorer, their operative procedures were not different from those in the rest of the patients. All four group 1 patients who died postoperatively of

Table 3. Postoperative Arrhythmia Characteristics

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<tbody>
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<td>VT</td>
<td>16 (50%)</td>
<td>6 (18%)</td>
</tr>
<tr>
<td>VF</td>
<td>4 (12.5%)</td>
<td>1 (3%)</td>
</tr>
<tr>
<td>Unmonitored sudden death</td>
<td>2 (6%)</td>
<td>1 (3%)</td>
</tr>
<tr>
<td>VT inducible only</td>
<td>*0</td>
<td>2 (6%)</td>
</tr>
<tr>
<td>Antiarrhythmic therapy administered</td>
<td>28 (87.5%)</td>
<td>9 (27%)</td>
</tr>
</tbody>
</table>

*VT induction studies were not performed in group 1 patients.

Abbreviations: VT = ventricular tachycardia; VF = ventricular fibrillation.
congestive heart failure underwent routine left ventricu- 
lar aneurysm resection and coronary artery bypass 
graffing (to one vessel in one, two vessels in two, and 
three vessels in one). Seven of the eight group 2 pa-
tients underwent myocardial resection and one simple 
ventriculotomy. Four of these eight had concomitant 
coronary artery bypass grafting and one of them un-
derwent mitral valve replacement.

Discussion

Our results support two conclusions. First, routine 
left ventricular aneurysm resection usually does not 
prevent recurrence of VT or VF. Second, specific 
myocardial resection or incision based on mapping of 
the electrical activation sequence may be a more effec-
tive surgical means of eliminating recurrent ven-
ctricular tachyarrhythmias. A similar conclusion was 
previously expressed by Harken and associates.19

The antiarrhythmic efficacy of blind left ven-
tricular aneurysm resection reported here was worse 
than that of a previous study from Stanford Univer-
sity Medical Center.13 The data were derived from 
only some of the patients included in the 
previous report as well as from patients undergoing 
blind aneurysm resection since that report. Several 
patients in the previous report were excluded from this 
study.

Through 24 months of follow-up, survival was not 
different in the two surgical groups despite the im-
provement in arrhythmia in group 2. The causes of 
death, however, were different. In group 1, 71% of 
deaths were due to ventricular tachyarrhythmias and 
24% were due to congestive heart failure, compared 
with 24% and 67%, respectively, in group 2. The 
higher incidence of deaths related to heart failure in 
group 2 may be a result of poorer preoperative hemody-
namic status, though this was not detected in our 
clinical assessment (table 1), or a result of myocardial 
injury by surgery. A similar incidence of mortality 
related to heart failure might have been seen in group 
1 patients if so many of them had not died of arrhyth-
mias early after operation. Nevertheless, the eight 
deaths from heart failure in group 2 should arouse 
awareness of the possibility that the new operative 
techniques may worsen left ventricular pumping per-
formance. Before these techniques can be considered 
truly superior to previous methods, a lower overall 
mortality rate must be documented.

This study is not a randomized, prospective com-
parison of the two surgical techniques. Thus, con-
clusions regarding the comparison of surgical results 
must be drawn cautiously. Unknown influences, 
rather than the surgical procedures per se, might ac-
count for the differences in outcome in the two groups. 
Subtle changes in intraoperative and postoperative 
care may have provided a better opportunity for 
success in group 2. Use of postoperative elec-
trophysiologic study may have improved outcome in 
group 2 (though it did not significantly alter the in-
cidence of arrhythmia recurrence). The duration of 
follow-up is different for the two groups; with more 
protracted follow-up in group 2, the gap in incidence 
of arrhythmia recurrence between the two groups may 
close. Unrecognized bias in patient selection could 
have predetermined a better result in group 2. How-
ever, the clinical characteristics of the two groups were 
strikingly similar. The severities of coronary artery 
disease and of congestive heart failure were nearly 
identical. Also, the lower incidence of documented 
medical intractability of ventricular tachyarrhythmi 
as the longer mean time between the last episode of 
tachyarrhythmia and surgery in group 1 patients may 
have biased arrhythmia manageability in their favor.

The unsatisfactory antiarrhythmic efficacy of rou-
tine left ventricular aneurysm resection should be 
emphasized. A rate of relapse of ventricular tachy-
arrhythmias of 50 ± 9% during the postoperative 
hospitalization is high, especially compared with the 
13 ± 6% rate of early recurrence in the map-directed 
group. Simple left ventricular aneurysm resection may 
not be an adequately effective primary surgical 
therapy for chronic recurrent ventricular tachy-
arrhythmias. New approaches, such as map-directed 
surgery, experimental antiarrhythmic agents, or im-
plantation of electronic devices such as the automatic 
anti-tachycardia pacemaker26 or the internal defibrilla-
tor,27 may be preferable.

Studies in at least one animal model of nonacute 
myocardial infarction have shown a focal origin of 
ventricular tachycardia in the tissue bordering the 
perimeter of postinfarction scar.28 Although no animal 
arrhythmias so far reported are truly comparable to 
chronic recurrent VT due to myocardial infarction in 
humans, findings in this model suggest that recurrent 
VT often arises from myocardial tissue left intact dur-
ing routine left ventricular aneurysm resection. How-
ever, removal of a left ventricular aneurysm 
without physical excision of the border tissue does 
eliminate recurrent ventricular tachyarrhythmias in 
a minority of patients. There are several explana-
tions for the occasional efficacy of simple aneurysm 
resection. Suture repair of the ventriculotomy might 
cause enough damage to the border tissue to eliminate 
tachycardias arising there. Or, tissue responsible for 
tachycardia might reside partially or fully within the 
resected aneurysmal scar itself. This appeared to be 
the case in two patients who underwent mapping. 
There is histologic evidence that postinfarction scar 
tissue contains viable myocytes;29 these cells might 
develop automaticity or localized reentry, or provide a 
portion of a reentry circuit involving border or normal 
tissues as well. Simple aneurysm resection may im-
prove hemodynamics considerably, which could also 
account in part for its occasional antiarrhythmic 
efficacy.

The frequent success of surgical procedures guided 
by activation sequence mapping has been ascribed to 
accurate removal of all or most tissue directly respon-
sible for generating the arrhythmia. A proportion of 
the failures of the map-directed method may be ex-
plained by inadequate mapping. In our series, map-
ing was less definitive in group 2 patients who had 
postoperative recurrences of VT or VF. Inability to 
obtain endocardial mapping data, indistinctness of
QRS onset and multiplicity of VT morphologies contributed equally to poor quality of mapping in these patients. However, three of the patients in group 2 with arrhythmia recurrences had had apparently excellent intraoperative activation sequence mapping studies. There are several possible explanations for surgical failure in patients with adequate mapping studies. The simplest explanation, an inadequate surgical resection or incision, is impossible to disprove; however, some of the ineffective resections in our series were as extensive as practical. Another explanation for failure of the focus mapping technique is that many, perhaps even most, chronic recurrent VTs may not be focal in origin, but due to reentry circuits traversing long distances, perhaps 20 cm or more. Reentry circuits of this sort could result in essentially continuous excitation of myocardium with no individual site being the true origin of the arrhythmia. A focal resection would be effective only if it removed a portion of the circuit that could not be substituted for by adjacent tissue with similar electrophysiologic properties. Failure may also be explained by emergence of previously concealed subsidiary foci or reentry circuits.

Novel surgical therapies of ventricular tachyarrhythmias, some of them not yet fully described in the literature, are being assessed at several centers. Map-directed surgery is not universally applicable. Some recurrent VTs, which may be due to abnormal automaticity, cannot be induced; other tachycardias are inducible but cannot be mapped. The encircling endoventriculotomy22 does not depend upon intraoperative mapping. However, this method cannot be used safely in all patients and may significantly compromise left ventricular function in some patients. Development and selection of the proper surgical interventions against ventricular tachyarrhythmias is greatly hampered by our ignorance of the mechanisms responsible for these arrhythmias. Nevertheless, simple left ventricular aneurysm resection, with or without concomitant coronary bypass grafting, may be less effective in preventing recurrent ventricular tachyarrhythmias than surgery based upon activation sequence mapping.

Addendum

Since the submission of this manuscript, 15 more patients have undergone map-directed surgery for a primary indication of recurrent VT due to myocardial infarction. Mean follow-up is 8.7 ± 7.5 months for the entire group of 48 patients, and 11.8 ± 6.5 months for those surviving to discharge. Two of 15 recent patients have died but none have had arrhythmia recurrences. Actuarial survival through 1, 3 and 24 months is 81 ± 6%, 76 ± 7% and 67 ± 8%, respectively. Actuarial nonrecurrence of ventricular tachyarrhythmias is 91 ± 4%, 88 ± 3% and 76 ± 8%, respectively.

References

25. Greenwood M: The errors of sampling of survivorship tables (appendix I). In Reports on Public Health and Medical Sub-
CRYOABLATION TO THE AV CONDUCTION SYSTEM/Ohkawa et al. 1155

Anatomic Effects of Cryoablation of the Atrioventricular Conduction System

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SUMMARY Because of the value of cryoablation of the atrioventricular (AV) conduction system in the treatment of refractory cardiac rhythm disorders, the anatomic effects of cryoablation on the cardiac conduction system must be defined. In this report we summarize studies done on four patients who had intractable recurrent supraventricular tachyarrhythmias or refractory atrial flutter-fibrillation. They were treated by cryoablation of the AV conduction system and died 8–360 days postoperatively. Serial sections of the AV conduction system were studied. Cryoablation produced lesions that completely destroyed most of the AV node in three cases, the penetrating portion of the His bundle in all four cases, and the branching portion of the His bundle in two cases. The right bundle branch was not involved markedly in any case. The lesions were discrete and sharply delimited; the patient who died 8 days postoperatively had hemorrhage, necrosis and slight inflammatory infiltrate; patients who survived for 49–360 days showed collagen deposition. The AV nodal artery and its branches showed slight to marked intimal thickening in three cases. Small, partly organized thrombi were present just behind the tricuspid valve in two patients. We conclude that cryoablation of the AV conduction system produced discrete cardiac lesions that did not markedly damage the tricuspid valve or aorta.

A CRYOSURGICAL TECHNIQUE for ablating the atrioventricular (AV) node–His bundle has been used in patients with disabling supraventricular tachyarrhythmias unresponsive to medical management.1–3 This is an effective technique, and thus will probably become widely used. Therefore, the anatomic effects of the cryoablation procedure on the cardiac conduction system must be defined. We describe the findings in four patients who died 8–360 days after cryoablation.

Materials and Methods

Patient Population

Four deceased patients (two men and two women) with supraventricular tachyarrhythmias or atrial flutter-fibrillation with or without associated ventricular tachyarrhythmias were studied. All of them showed resistance to medical therapy and were referred to the Duke University Medical Center for surgical treatment. A brief report of the anatomic findings in one of these patients (case 4, table 1) was reported previously.4

Operative Procedures

All patients had an electrophysiologic assessment to determine the mechanism of their arrhythmias. The results of these studies have been reported.5–4 The cryosurgical technique used in this study has been described in detail elsewhere.1,3 After identification of the His bundle by observation of the electrograms generated by mapping the endocardial tissue with a handheld bipolar probe, the cryoprobe was applied three times to overlapping His bundle areas for 3 minutes each at a temperature of −50 to −60°C. After rewarming, the area was probed again and the electrograms were observed for complete AV block. All patients had implanted pacemakers.

Case 1 was treated only by cryosurgery and case 2 had concurrent coronary artery bypass grafting, encircling endocardial ventriculotomy and aneurysmectomy. Case 3 had concurrent division of the posteroseptal pathway in the crux region. Case 4 had ablation of a focus of ventricular tachycardia in the left ventricle and ablation of the His bundle region, which was contiguous to an accessory pathway.

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