Endocardial Excision: A New Surgical Technique for the Treatment of Recurrent Ventricular Tachycardia

Mark E. Josephson, M.D., Alden H. Harken, M.D., and Leonard N. Horowitz, M.D.

SUMMARY Twelve patients with medically refractory ventricular tachycardia secondary to ischemic heart disease underwent surgery for cure of their arrhythmia. Preoperatively, the tachycardia could be reproducibly initiated and terminated in each patient by programmed stimulation. In all instances, intraoperative mapping localized the tachycardia to the border of the aneurysm, a site not routinely resected during aneurysmectomy. In nine instances, the area of origin involved the septum. During bypass the tachycardia could still be induced after standard aneurysmectomy or ventriculotomy in 11 of 12 patients. On the basis of intraoperative mapping, resection of endocardium in the area of origin (25-40% of circumference of the aneurysmectomy) up to normal muscle was performed. In one patient without a discrete aneurysm, endocardial excision alone through a ventriculotomy was performed. There was one operative death due to cardiogenic shock (preoperative ejection fraction 5%) and one late death due to rupture of a mycotic aneurysm in the pulmonary artery. Before discharge, all patients underwent a repeat electrophysiologic study off antiarrhythmic agents and in none could ventricular tachycardia be initiated. Hemodynamic and angiographic catheterization showed improved hemodynamics and ejection fractions in all. The 10 survivors remained free of sustained ventricular tachycardia for 9–20 months, with one late nonarrhythmic death.

The surgical therapy of malignant ventricular arrhythmias has included ventricular aneurysmectomy and coronary artery bypass grafting. Despite early reports of success with coronary artery bypass grafting as a treatment for these arrhythmias, recent studies have shown that bypass surgery for treatment of ventricular arrhythmias not mediated by ischemia is less effective. Similarly, although aneurysmectomy with or without coronary bypass grafts has been reported to be a highly successful procedure, other studies are more pessimistic. In one study in which bypass grafting or aneurysmectomy or both were used, bypass grafting alone was least effective and associated with a higher mortality, suggesting that resection of myocardium is important. Thus, while aneurysmectomy and bypass grafting are widely used to treat malignant ventricular arrhythmias, the results have not been uniform, and some investigators question use of these procedures when the sole indication is arrhythmias. Several factors influence the results, including poor characterization of the arrhythmias relative to the type of arrhythmia (sustained vs nonsustained ventricular tachycardia, symptomatic vs asymptomatic, reentrant vs automatic, etc.) and the temporal relationship of the arrhythmia to ischemic events; and 2) varying surgical techniques. We describe a new surgical procedure, endocardial excision, that is an important adjunct to simple aneurysmectomy in treating reentrant sustained ventricular tachycardia associated with chronic ischemic heart disease.

Materials and Methods

Twelve consecutive patients with recurrent sustained ventricular tachycardia refractory to medical therapy underwent surgery for control of their arrhythmias (table 1). Ten men and two women, age 44-68 years, had coronary artery disease with documented myocardial infarction 2 weeks to 60 months before surgery. Hemodynamic and angiographic catheterization revealed significant coronary artery disease (mean of 1.75 vessels with > 70% obstruction) and asynery in all patients. Eight had anterior and/or apical aneurysms, two had anterolateral aneurysms, one had an inferior aneurysm and one had inferior akinesis without a discrete aneurysm. In nine patients, the aneurysm involved the septum. Ejection fractions ranged from 5–40%, and six patients had been deemed inoperable by surgeons at their referring institutions.

Ventricular tachycardia recurred preoperatively in each patient despite therapy with maximally tolerated doses of standard and available experimental agents that could be administered without hemodynamic or allergic contraindications. The drugs included lidocaine (5 mg/kg i.v.), procainamide (750–2000 mg i.v. or 6–12 g/day orally), quinidine (1.6–3 g/day orally), disopyramide (800–1600 mg/day orally), diphenhydantoin (1 g i.v. or 400 mg/day), and aprin-
Table 1. Patient Characteristics

<table>
<thead>
<tr>
<th>Pt</th>
<th>Sex/age (years)</th>
<th>Arrhythmias (n)</th>
<th>Infarction</th>
<th>Coronary angiogram (% occlusion)</th>
<th>Left ventricular angiogram</th>
<th>EF</th>
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<tbody>
<tr>
<td>1</td>
<td>M/44</td>
<td>VT (6)</td>
<td>AMI</td>
<td>100% LAD 100% RCA</td>
<td>Anteropapical aneurysm</td>
<td>20%</td>
</tr>
<tr>
<td>2</td>
<td>M/61</td>
<td>VT (80)</td>
<td>AMI</td>
<td>99% LAD</td>
<td>Anteropapical aneurysm</td>
<td>5%</td>
</tr>
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<td>3</td>
<td>M/59</td>
<td>VT (3)</td>
<td>IMI</td>
<td>90% RCA</td>
<td>Inferior aneurysm</td>
<td>25%</td>
</tr>
<tr>
<td>4</td>
<td>M/68</td>
<td>VT (6)</td>
<td>ALMI</td>
<td>100% LAD</td>
<td>Anterolateral aneurysm</td>
<td>20%</td>
</tr>
<tr>
<td>5</td>
<td>M/44</td>
<td>VT (20)</td>
<td>ASMI</td>
<td>100% RCA 80% LAD 75% LCX</td>
<td>Apical aneurysm</td>
<td>20%</td>
</tr>
<tr>
<td>6</td>
<td>M/66</td>
<td>VT (6)</td>
<td>ASMI</td>
<td>99% LAD 75% RCA</td>
<td>Anteropapical aneurysm, 1+ MR</td>
<td>30%</td>
</tr>
<tr>
<td>7</td>
<td>M/65</td>
<td>VT (3)</td>
<td>IMI</td>
<td>99% RCA 75% LAD 75% LCX</td>
<td>Inferior skinness</td>
<td>40%</td>
</tr>
<tr>
<td>8</td>
<td>M/61</td>
<td>VT (35)</td>
<td>ASMI</td>
<td>90% LAD 100% LCX 75% RCA</td>
<td>Anteropapical aneurysm, 2+ MR</td>
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<tr>
<td>9</td>
<td>F/49</td>
<td>VT (4)</td>
<td>ALMI</td>
<td>99% LAD</td>
<td>Anterolateral aneurysm, 1+ MR</td>
<td>25%</td>
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<tr>
<td>10</td>
<td>M/48</td>
<td>VT (25)</td>
<td>ASMI</td>
<td>100% LAD</td>
<td>Anteropapical aneurysm</td>
<td>25%</td>
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<tr>
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<td>ASMI</td>
<td>100% LAD</td>
<td>Anteropapical aneurysm</td>
<td>30%</td>
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<tr>
<td>12</td>
<td>M/62</td>
<td>VT (4)</td>
<td>AMI</td>
<td>100% LAD</td>
<td>Anteropapical aneurysm</td>
<td>35%</td>
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</table>

Abbreviations: ALMI = anterolateral myocardial infarction; AMI = anterior myocardial infarction; ASMI = anteroapical myocardial infarction; EF = ejection fraction; LAD = left anterior descending coronary artery; LCX = left circumflex coronary artery; MR = mitral regurgitation; RCA = right coronary artery; VF = ventricular fibrillation; VT = ventricular tachycardia.

dine (300 mg/day orally). Propranolol (0.1 mg/kg i.v. or 60–240 mg/day orally) was used in three patients; the remaining nine patients did not tolerate propranolol because of poor hemodynamic status. Combinations of these agents had also failed to prevent the arrhythmias. Each patient had been hospitalized several times and required multiple (three to 55) DC cardioversions or defibrillations (one to 20) if deterioration to ventricular fibrillation had occurred.

Before surgery, 11 of the 12 patients underwent complete electrophysiologic study, including programmed ventricular stimulation and catheter endocardial mapping, as previously described.\(^{13,14}\) One patient underwent only programmed ventricular stimulation because rapid hemodynamic deterioration made catheter endocardial mapping impossible. In all 12 patients, the ventricular tachycardia could be reproducibly initiated and terminated by programmed stimulation. In the patients in whom catheter mapping was accomplished preoperatively, the ventricular tachycardia, regardless of morphology, was shown to originate in the area of the aneurysm.

Before discharge, all patients underwent repeat electrophysiologic study to assess the inducibility of ventricular tachycardia. In each patient, stimulation of both the right and left ventricles was performed. Left ventriculography (both right and left anterior oblique projection) and hemodynamic measurements were performed before discharge to assess the effects of the surgical procedure on wall motion. One patient underwent complete hemodynamic and angiographic catheterization 1 month after discharge at the referring institution.

**Surgical Methods**

The heart was approached through a median sternotomy and prepared for cardiopulmonary bypass in the routine fashion. An arterial cannula was placed in the ascending aorta and a single venous cannula placed through a pursestring suture in the right atrium. A teflon-coated stainless steel plunge electrode (0.005 inch in diameter) was inserted by means of a 25-gauge needle in the right ventricular free wall for pacing. Similar intramural electrodes were placed in the right ventricle and in the normal appearing left ventricle and used to record reference electrograms. In
four patients, a temporary transvenous electrode catheter placed preoperatively in the right ventricle was used for pacing or recording a reference electrogram.

EPGcardial mapping was then performed using standard techniques. Bipolar electrograms were recorded at 50–66 predetermined epicardial sites with a hand-held probe or finger-tip electrode with a 1-mm interelectrode distance. Three electrocardiographic leads (1, 2, and V5 or V5R) and the stable reference electrograms were recorded simultaneously with the exploring electrogram. Epicardial maps of both ventricles were obtained during sinus rhythm. Ventricular tachycardia was then induced by programmed stimulation and the epicardial mapping procedure repeated. During ventricular tachycardia, particular attention was directed toward mapping along the edges of the aneurysm. During ventricular tachycardia, partial (30–50% of predicted flow) and occasionally complete cardiopulmonary bypass was initiated to maintain a mean central aortic pressure of 80–100 mm Hg.

After epicardial mapping, the heart was elevated and an incision made into the center of the aneurysm. In four patients, this was done during sustained ventricular tachycardia. The thin-walled aneurysm was cut to within 1 cm of the palpable border between normal myocardium and scar. In nine cases, the aneurysm involved 2–6 cm of the septum and this segment could not be resected in the conventional manner. Before closure of the aneurysmectomy, programmed stimulation was again undertaken. If the tachycardia was inducible, endocardial mapping of 36–50 preselected sites along the aneurysmal border and selected sites within the heart was performed. In one patient without a discrete aneurysm, a ventriculotomy was made in the scar, programmed ventricular stimulation repeated, and endocardial mapping of the induced ventricular tachycardia accomplished along the septal border of the akinetic segment.

All data were recorded on line on an Elema Mingograf and the earliest area of activation was determined by two of the investigators. Based on the data that showed the source of the arrhythmia to be within 2 cm of the border of the aneurysm (fig. 1), endocardial resection was then performed in the area determined to be the origin of the arrhythmia. In nine patients, the resection involved the area of the aneurysm or akinetic segment that involved the region of the interventricular septum. In the remaining three cases, the resection involved a lateral edge of the aneurysm. In patients with a discrete aneurysm, the edge of the aneurysm was grasped with clamps and the endocardium was undermined with scissors (figs. 2A and B). In this fashion an 8–25 cm² of endocardium extending 2–3 cm beyond the edge of the aneurysm, including the area overlying scar tissue, was removed (fig. 2C). In each case, the resection involved 25–40% of the circumference of the aneurysmectomy. In the remaining patient without a discrete aneurysm, endocardial resection of the posterior third of the interventricular septum was accomplished in an analogous manner.

In six patients, coronary artery bypass grafting to vessels with greater than 70% occlusion was performed by means of a totally decompressed, unrepaired ventriculotomy after endocardial resection. Total body and myocardial temperature was reduced to 30°C during the proximal and distal coronary anastomosis. After the grafts were completed, the ventricular resection was repaired in the standard fashion using large mattress sutures buttressed with felt pledgets. Patients were then rewarmed. Air was evacuated from the left ventricular suture line and a separate vent site in the aorta. The hearts were then electrically defibrillated. The electrodes were removed and the chest was closed.

**Results**

**Surgical Results**

Aneurysmectomy and endocardial excision were performed in six patients. In three, coronary artery bypass grafting to vessels not supplying the area from which ventricular tachycardia originated was performed; in three others, coronary artery bypass grafts

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**Figure 1.** A) Twelve-lead ECG during ventricular tachycardia (VT) (case 2). B) Epicardial and endocardial mapping during VT. Both panels are arranged top to bottom as ECG leads 1, 2, and V5, reference electrograms recorded in the right (RV) and left (LV) ventricles, mapping electrograms recorded at three sites and 10- and 100-msec time marks (T). On the left, the mapping sites are the site of epicardial breakthrough (EPI 1) (compare with fig. 1C), anterior left ventricle (EPI 2) and anterior right ventricle (EPI 3). On the right, the mapping sites are the site of earliest endocardial activity (ENDO 2) and the immediately adjacent sites (ENDO 1 and 3) (compare with fig. 1C). The vertical lines indicate the onset of the QRS complex and the activation times indicate time from the onset of the QRS. Note that the earliest epicardial activity occurred 30 msec after the onset of the QRS, while the earliest endocardial activity occurred 8 msec before the QRS. C) Epicardial and endocardial activation during VT. ECG lead 2 is shown above. The epicardial surface of the ventricles is shown in the anterior, left lateral and inferior views (clockwise from upper left). Epicardial breakthrough occurred 30 msec after the onset of the QRS on the inferopapical margin of the aneurysm (indicated by the 30). Twenty-millisecond isochrones indicate the pattern of ventricular activation. The location of the earliest endocardial electrogram is indicated by the solid circle on the anterolateral left ventricular wall. At the lower left, the epicardial surface of the LV is shown; the circle indicates the margin of the aneurysm. The activation times indicate time from the onset of the QRS. The asterisk indicates the location of epicardial breakthrough. The site of origin of the VT was 3 cm from the site of epicardial breakthrough.
were placed to vessels that supplied the area where the tachycardia originated.

Eleven of the 12 patients survived operation. Six patients required transient inotropic support and two patients required intraaortic balloon counterpulsation. These patients stabilized hemodynamically within 3 days and supportive measures were discontinued. No patient had sustained ventricular tachycardia despite infusions of catecholamines.

The single operative death occurred in patient 2, who had a 5% ejection fraction and a recent (10 days) myocardial infarction. Despite catecholamines, vasodilators and intraaortic balloon counterpulsation, the patient died of cardiogenic shock 24 hours after surgery. This patient had no ventricular tachy-arrhythmia after surgery, although 30 cardioversions had been required in the 24 hours before surgery.

There was one late death (patient 5) 6 weeks after surgery. This patient, who had no sustained ventricular tachycardia after surgery, developed pneumonia complicated by sepsis and an endo-vasculitis that led to rupture of his pulmonary artery.

In all surviving patients, repeat hemodynamic and angiographic catheterization revealed improved hemodynamics and left ventricular contractile function (fig. 3). In each patient, the left ventricular end-diastolic pressure decreased after surgery and ejection fraction increased. There were no untoward effects of endocardial resection on left ventricular function.

Electrophysiologic Results

In all patients, ventricular tachycardia could be induced intraoperatively by programmed ventricular stimulation. After ventriculotomy and/or resection of the aneurysm, the tachycardia either continued (in the four patients in whom this procedure was performed during a stable ventricular tachycardia) or was inducible in 11 of the 12 patients. Thus, in these patients, aneurysmectomy using standard techniques failed to remove the tissue responsible for the tachycardia. In each instance, epicardial or endocardial mapping located the site of origin at the border of the aneurysm (11 patients) or akinetic area (one patient). Further, in nine patients, the site of origin involved the septum or contiguous tissue, which are not amenable to excision. Endocardial resection was therefore performed to remove these areas.

All surviving patients underwent an electrophysiologic study before discharge. Eleven patients

![Figure 2. Excision of septal endocardium (case 10). (The septum is at the left of the opened aneurysm in both panels.) In all three patients, the aneurysm involved the septum to a variable degree. A) The subendocardium overlying the infarcted septum is grasped by a clamp and undermined by scissors. B) The subendocardium has been removed from the inferior edge of the septum to the mid-septum (5-6 cm) and 3-4 mm deep. C) The excised endocardium. The tissue is opalescent and semitransparent.](image-url)
Table 2. Results

<table>
<thead>
<tr>
<th>Pt</th>
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<th>Procedure</th>
<th>CAGB</th>
<th>Post-operative EPS*</th>
<th>Postoperative Holter</th>
<th>Follow-up</th>
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<td>VPDs (50-200/hr)</td>
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</tr>
<tr>
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<td>Yes</td>
<td>Septal</td>
<td>LCX, RCA</td>
<td>No VT</td>
<td>NP</td>
<td>No VT, died ruptured PA</td>
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<td>No VT</td>
<td>VPDs (0-78/hr), couplets</td>
<td>Asx, no VT</td>
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*Included single and double premature ventricular stimulation and rapid ventricular pacing at both right and left ventricles.
†No change from preoperative symptoms.

Abbreviations: An = aneurysmectomy; EPS = electrophysiologic study; Endo = endocardial; CAGB = coronary artery bypass graft; LCX = left circumflex coronary artery; LAD = left anterior descending coronary artery; PA = pulmonary artery; RCA = right coronary artery; VT = ventricular tachycardia; VPDs = ventricular premature depolarizations; SVPDs = supraventricular premature depolarizations; AIVR = accelerated idioventricular rhythm; Asx = asymptomatic (i.e., no symptoms of palpitations, cerebral dysfunction or congestive heart failure); CHF = congestive heart failure; NP = not performed.

were receiving no antiarrhythmic medication. Patient 10 was on 200 mg of quinidine sulfate every 6 hours for recurrent atrial fibrillation, but higher doses of this agent failed to prevent ventricular tachycardia preoperatively. In no instance could ventricular tachycardia be initiated by programmed stimulation, including rapid atrial or ventricular pacing and the introduction of single or double ventricular extrastimuli from both the right and left ventricles (fig. 4). Despite the failure to initiate the tachycardia in these patients, all patients had ventricular premature depolarizations during 24-hour Holter monitoring before discharge.

Follow-up

All patients except patient 1 were discharged on no antiarrhythmic medications despite the presence of ventricular ectopy. There has been one late death (8 months) due to rupture of a pseudoaneurysm. Gross postmortem examination of the heart revealed that the subendocardial resection was completely endotheialized and not involved in the pseudoaneurysm. The cause of the necrosis that resulted in the pseudoaneurysm was uncertain, but evidence of ischemic damage was present. This patient had no documented ventricular tachycardia before death.

The remaining nine patients have been free of symptomatic sustained ventricular tachycardia for 9–20 months. Repeat Holter monitoring in all patients has shown persistence of asymptomatic arrhythmias, including occasional multifocal ventricular premature depolarizations (six patients) and couplets (five patients). Exercise tests have been performed in seven patients, all of whom had improved exercise tolerance and had no exercise-induced ventricular tachycardia. No patients have symptoms of congestive heart failure or a low-output state.

Discussion

Ventricular aneurysmectomy with or without coronary vascularization is the most commonly used surgical procedure to abolish drug-resistant ventricular
FIGURE 3. Effects of subendocardial septal excision or septal ventricular contraction (case 10). A) Preoperative and B) postoperative left ventriculograms in the left anterior oblique view show the outline of the septum. Schematic diagrams of superimposed systolic (stippled) and diastolic figures are on the right. Septal subendocardial resection did not adversely influence septal motion.

FIGURE 4. Inability to initiate ventricular tachycardia 2 weeks postoperatively (case 3). Panels A and B are organized from top to bottom as ECG lead V1 and electrograms from the coronary sinus (CS) and right ventricular apex (RVA). Panel A was from the preoperative study, during which ventricular tachycardia could be reproducibly initiated by two ventricular extrastimuli (S2 and S3) during a paced ventricular cycle length (S,S,) of 600 msec. Panel B was obtained during the study 2 weeks postoperatively. At the same paced ventricular cycle length, double ventricular extrastimuli failed to initiate the tachycardia. No other stimulation technique was able to initiate ventricular tachycardia in this patient postoperatively.
tachycardia. The efficacy of this form of therapy remains controversial.\textsuperscript{1–12} A recent review of aneurysmectomy for recurrent ventricular tachycardia concluded that the results of this procedure were extremely variable, with a lower success rate for abolition of the arrhythmia and increased mortality in patients with severe myocardial dysfunction or recent infarction.\textsuperscript{19} Another recent review reported an overall success rate of 58%.\textsuperscript{6} Experience at the Hospital of the University of Pennsylvania before 1976 with simple aneurysmectomy with or without coronary artery bypass grafting for the treatment of recurrent sustained ventricular tachycardia in 15 patients revealed a 33% operative (30-day) mortality, including three patients with intractable ventricular tachycardia and fibrillation (unpublished observations). Moreover, only 60% (six of 10) of the remaining patients were free of symptomatic ventricular tachycardia, for an overall success rate of 40%.

The reported results vary for several reasons:

1) The arrhythmias for which surgery was undertaken varied from short (three to 10 complexes), non-sustained runs of asymptomatic ventricular tachycardia to sustained symptomatic ventricular tachycardia.

2) The mechanisms of the arrhythmias being treated differed, and their relationship to transient ischemia before surgery was not documented.

3) Surgical techniques vary from institution to institution.

4) In other studies, there have been no pre- or intraoperative evaluations of the arrhythmias to localize their origin to the tissue being resected or revascularized. Much of the variability of results may be explained by failure to resect the region responsible for arrhythmogenesis with simple aneurysmectomy, because this procedure does not include the border zone, which many investigators have suggested is the source of this arrhythmia.\textsuperscript{12, 19–22} Moreover, the septal portion of the aneurysm, which was shown to play a role in the tachycardia in nine patients, is not removed by standard procedures.

Role of Pre- and Intraoperative Mapping

In all but one of our patients, preoperative catheter endocardial mapping localized the origin of each tachycardia to the area of the aneurysm. This information provided a rational basis for aneurysmectomy. In all instances, the earliest epicardial breakthrough occurred after the onset of the QRS. Similar observations have been made by previous workers.\textsuperscript{16, 21–23} The limitations of epicardial mapping, however, have been recognized by experimental\textsuperscript{24} and human studies,\textsuperscript{18} which show that the epicardial breakthrough can occur several centimeters from the endocardial or intramural site of origin of the arrhythmia. Most significantly, ventricular tachycardia could be induced after simple aneurysmectomy in 11 of 12 patients. Endocardial mapping of each of the induced ventricular tachycardias after simple aneurysmectomy located the site of origin at the border of the aneurysm or infarct, regardless of the morphology or the results of epicardial mapping. In nine instances, this area involved the interventricular septum. Thus, the reason for the failure of aneurysmectomy alone or with bypass grafting to abolish ventricular tachycardia became apparent with demonstration that routine aneurysmectomy usually does not include the critical areas of diseased myocardium responsible for the arrhythmia.

Although several groups believe that the reentry pathway in myocardial ischemia is confined to a restricted area of nonspecific myocardium, we postulated that the subendocardial Purkinje fibers that survive myocardial infarction are a potential component of the reentrant circuit. We therefore developed a procedure of subendocardial resection in the area of the earliest activity as a method of removing the source of the arrhythmia. The extent of subendocardial resection varied from 25–40% of the circumference of the aneurysmectomy and was extended to normal-appearing tissue. We considered the potential for leaving an intramural focus intact by this procedure, but felt it unwise to include greater segments of transmural resection in the simple aneurysmectomy because this would make the final repair more difficult. Because the origin of arrhythmia appeared to involve the septum in nine patients, and because the septal resection is unfeasible, endocardial resection provided a reasonable alternative. Thus, preoperative and intraoperative mapping were crucial to locating the arrhythmia and hence represented the only method to guide resection of critical areas involved in the arrhythmia.

Relationship to Other Surgical Procedures

The refinement of the techniques of programmed stimulation and endocardial catheter mapping has provided a greater understanding of the mechanisms of ventricular tachycardia. Several authors have suggested that ventricular tachycardia associated with coronary artery disease and ventricular aneurysms is usually due to reentry and that the reentrant circuit is small and relatively protected and does not usually utilize the bundle branches.\textsuperscript{13, 25–27} The feasibility of surgically interrupting a reentrant circuit has been established in patients with the Wolff-Parkinson-White syndrome.\textsuperscript{27–29} Extension of this concept to ventricular tachycardia led to the development of simple ventriculotomy as a method of treating resistant ventricular tachycardia.\textsuperscript{20, 23} Ventriculotomy was originally applied to patients with delayed activation (postexcitation phenomenon) in early areas during the ventricular tachycardia. In these patients, transmural ventriculotomy 4–5 cm in length was performed at the site of epicardial breakthrough of the ventricular tachycardia. The procedure was highly successful in one group of patients, most of whom had right ventricular dysplasia.\textsuperscript{23} Similar results were obtained in a small number of patients by Spurrell et al.\textsuperscript{21, 22} Guiraudon, Fontaine and co-workers could not abolish ventricular tachycardia in patients in whom the arrhythmia could not be induced.\textsuperscript{19, 20, 23} As a result, these authors do not consider patients with noninducible ventricular tachycardia to be suitable candidates for simple ventriculotomy.
Fontaine et al. attempted to extrapolate these results of ventriculotomy in patients with dysplasia of the right ventricle to ventricular tachycardia in the setting of chronic ischemic heart disease and ventricular aneurysms. Ventriculotomy was successful in only one of 13 patients; in 11 patients, epicardial mapping could not adequately localize late potentials or the origin of the tachycardia. The reasons for the high incidence of uninterpretable data in Fontaine’s series in light of our studies and those reported by Wittig and Boineau and Gallagher et al. are unexplained.

Spurrell et al. described surgical sectioning of the left bundle branch as a method to control ventricular tachycardia in four patients in whom the mechanism of the ventricular tachycardia was interpreted as including the bundle branches. However, these patients also underwent resection of muscle or valve replacement or both, but the exact mechanism of ablation of the arrhythmia is unclear. Recent data suggest that the main bundle branches rarely form part of the reentrant circuit in recurrent ventricular tachycardia.

A new surgical treatment was recently reported by Guiraudon and colleagues in five patients with recurrent ventricular tachycardia. This technique, encircling endocardial ventriculotomy, involves a transmural ventriculotomy performed perpendicular to the wall and extended completely around the limits of endocardial fibrosis associated with prior infarction. Although the endocardial incision was made through a ventriculotomy in the central area of the aneurysm, myocardial resection was only carried out in two of the patients. The endocardial incision is repaired by a running suture, and the ventricle is closed in the standard fashion. The rationale for this procedure was the authors’ belief that the reentrant circuits responsible for inducible ventricular tachycardia are located along the border zone with participation of the ischemic subendocardial layers. All five patients remained free of symptomatic ventricular tachycardia.

Our results are similar to those of Guiraudon et al., although our technique differs significantly from theirs in that only the subendocardium in the area of the tachycardia is removed and no additional closure within the ventricles is required. The fact that subendocardial resection does not interfere with ventricular function and is, in fact, associated with improved contractile performance, was verified by repeat left ventriculography. Further follow-up, however, is required to establish which procedure — encircling endocardial ventriculotomy or subendocardial resection — is most widely applicable, technically feasible, and highly successful. With either procedure, a higher success rate is to be expected than with routine aneurysmectomy, with or without bypass grafting.

Limitations and Interpretation

Although it would have been desirable to use a “pure” procedure and perform endocardial resection only, the exigencies of surgery in patients with coronary artery disease prohibited achievement of that ideal. While endocardial resection and aneurysmectomy were performed as the sole procedures in six patients, coronary artery bypass grafts were placed in the remaining patients. However, in three patients, the coronary artery bypass graft supplied areas that were probably not responsible for the ventricular tachycardia.

Although aneurysmectomy was performed in 11 patients, we have evidence suggesting that this procedure alone would not be expected to abolish the arrhythmia. In all but one instance, ventricular tachycardia could be reinduced after simple aneurysmectomy. In fact, in four patients, ventriculotomy and myocardial resection was accomplished during a stable ventricular tachycardia. In one patient, endocardial resection was accomplished in the absence of myocardial resection with successful results. Therefore, the variable results of aneurysmectomy most likely occur due to failure to remove the source of the arrhythmia. Successful aneurysmectomy occurs when the critical areas are included in the suture line. Thus, although subendocardial resection was not undertaken as the sole procedure in most patients, there is some evidence to suggest that the endocardial resection, not the aneurysmectomy or coronary artery bypass grafts, is responsible for the successful abolition of ventricular tachycardia. Further data are required to assess the role of this procedure in the management of ventricular tachycardia in patients without coronary artery disease, e.g., cardiomyopathy or idiopathic ventricular tachycardia.

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