Surgical Endocardial Resection for the Treatment of Malignant Ventricular Tachycardia

ALDEN H. HARKEN, M.D., MARK E. JOSEPHSON, M.D., LEONARD N. HOROWITZ, M.D.

From the Divisions of Cardiovascular Surgery and Cardiology, University of Pennsylvania Medical School, Philadelphia, Pennsylvania

VENTRICULAR TACHYCARDIA is a frequent post-infarction and perioperative arrhythmia. Recurrent ventricular tachycardia is less common, but is both physiologically and psychologically incapacitating when it occurs. Despite early reports of success with coronary artery bypass grafting as a treatment for these arrhythmias, recent series report disappointing results of revascularization with or without concurrent left ventricular aneurysmectomy.[2,12,14] The purpose of this manuscript is to describe a new surgical technique—endocardial excision—as a treatment for recurrent ventricular tachycardia in 12 patients.

Methods

Twelve consecutive patients with recurrent sustained ventricular tachycardia refractory to medical therapy underwent surgery for control of their arrhythmias. Ten men and two women ranged in age from 44 to 68 years. All had coronary artery disease with documented myocardial infarction two weeks to 60 months prior to surgery. Hemodynamic and angiographic catheterization revealed significant coronary artery disease (mean of 1.75 vessels with >70% obstruction) and asynergy 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 4 to 40% and six patients had been considered inoperable by their referring institutions.

Preoperatively ventricular tachycardia recurred in each patient despite therapy with maximally tolerated doses of standard and available experimental agents. The drugs and doses included lidocaine (5 mg/kg intravenously), procainamide (750–2000 mg intravenously

or 6–12 g orally each day), quinidine (1.6–3 g each day), disopyramide (800–1600 mg orally each day), diphenylhydantoin (1 g intravenously or 400 mg per day), and aprindine (300 mg orally per day). Propranolol (0.1 mg/kg intravenously or 60–240 mg orally each day) was only employed in three patients; in the remaining nine patients propranolol was not tolerated due to left ventricular failure. Combinations of these agents had also failed to prevent the arrhythmias. Each patient had been hospitalized multiple times and required from 3 to 55 DC cardioversions and 1–20 defibrillations.

Prior to surgery 11 of the 12 patients underwent complete electrophysiologic study, including programmed ventricular stimulation and catheter endocardial mapping.[9,10] One patient underwent only programmed ventricular stimulation; 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.

Prior to discharge all patients underwent repeat electrophysiologic study to assess the inducibility of ventricular tachycardia.

Surgical Procedures

A median sternotomy was performed and the aorta and right atrium cannulated in the routine fashion. A Teflon®-coated stainless steel plunge electrode (0.005 inch in diameter) was inserted via 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 pa-

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FIG. 1. An operative photograph revealing a left ventricular apical ventriculotomy retracted open with clamps. Normothermic cardiopulmonary bypass is initiated and ventricular tachycardia induced. The earliest site of arrhythmia origin is located on the ventricular endocardium with a bipolar electrode.
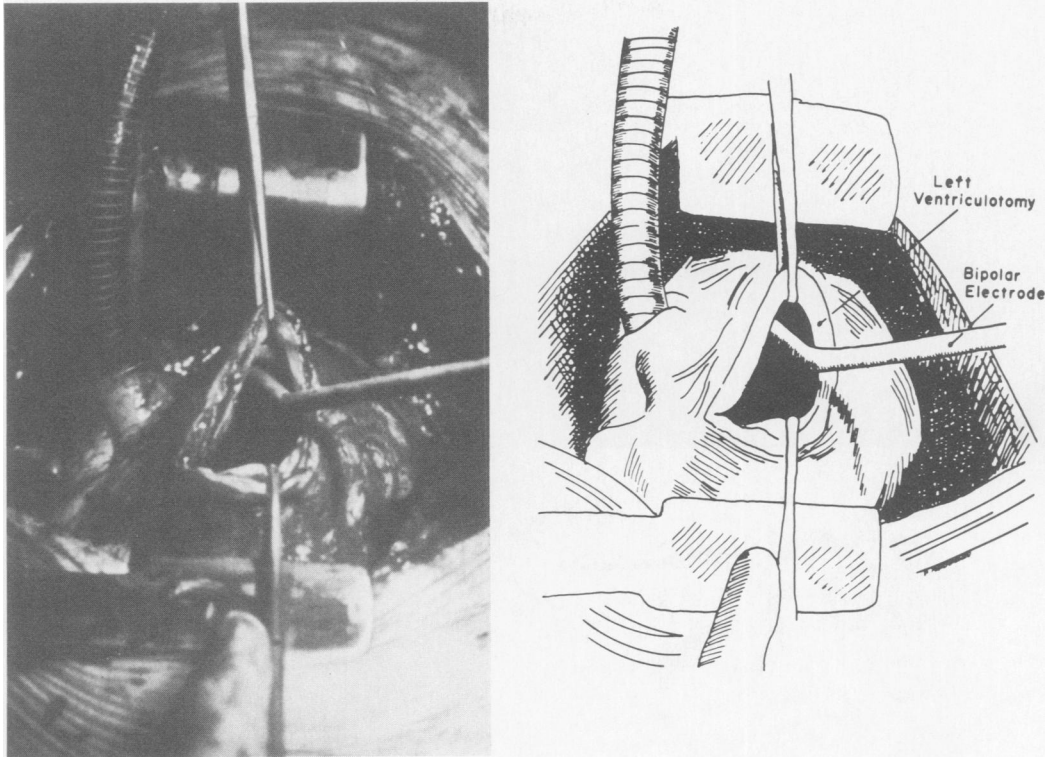
tients a temporary transvenous electrode catheter, placed preoperatively in the right ventricle, was used for pacing and/or recording a reference electrogram.

Epicardial mapping was then performed using standard techniques.[5,7,8] Bipolar electrograms were recorded at 50–66 predetermined epicardial sites with a hand-held probe or fingertip electrode with a 1 mm interelectrode distance. Three electrocardiographic leads (1, 2 and V_5 or V_{5R}) and the stable reference electrograms were recorded simultaneously with the exploring electrogram. Epicardial maps of both ventricles were obtained first 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. With the induction of ventricular tachycardia it was typically necessary to initiate normothermic cardiopulmonary bypass.

Following epicardial mapping an incision was made into the aneurysm. In four patients this was done during sustained ventricular tachycardia. A standard left ventricular aneurysmectomy was performed. In 9 cases the aneurysm involved 2–6 cm of the septum and this segment could not be resected in the conventional manner. Following 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[7,16] with a bipolar stick electrode (Fig. 1). 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.

In nine patients the arrhythmia origin involved the area of the aneurysm or akinetic segment adjacent to the intraventricular septum. In the remaining three cases, the tachycardia origin was located on the lateral edge of the aneurysm. In the cases in which a discrete aneurysm and septal origin were present, the edge of the aneurysm was grasped with clamps and the endocardium was undermined with scissors (Fig. 2). In this fashion an 8–25 cm² portion of endocardium extending 2–3 cm beyond the edge of the aneurysm, including area overlying scar tissue, was removed.

In the three patients in which the tachycardia originated from the lateral free wall of the left ventricle the endocardial excision was carried beyond the limits of the aneurysmectomy for 2–3 cm (Fig. 3). In each case the resection involved 25–40% of the circumference of the aneurysmectomy. In the remaining patient without a discrete aneurysm, the left ventricle was opened through a posterior akinetic area (Fig. 4). The incision is made sufficiently lateral so as not to necessitate inclusion of the posterior
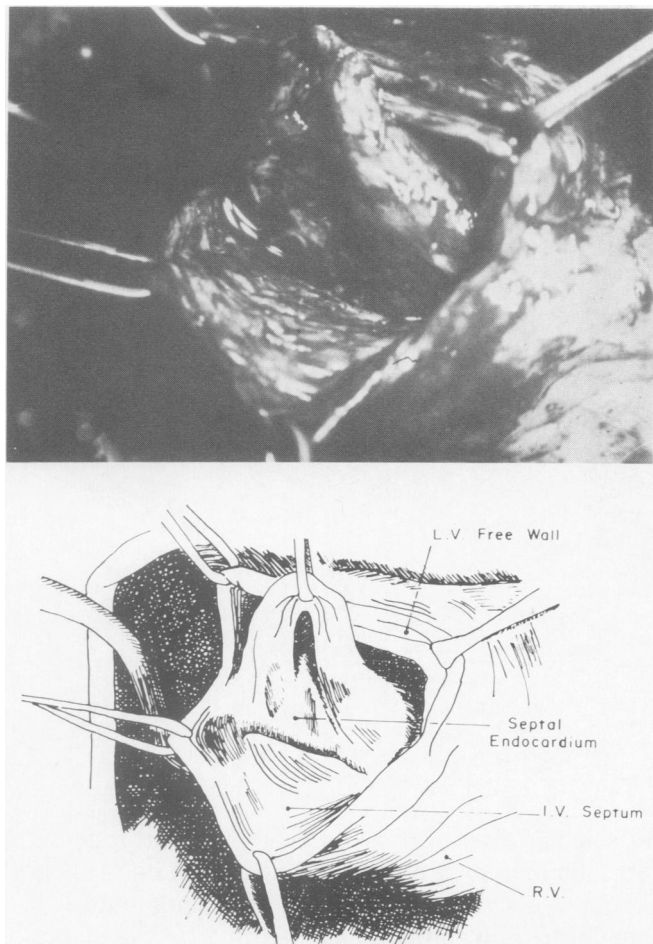
FIG. 2. Operative photograph revealing a left ventricular aneurysmectomy with edges held open. A large flap of endocardium is peeled off the septum in order to obliterate the arrhythmia origin.

descending vessels in the ventriculotomy closure. Care was taken to avoid the base of the posterior papillary muscle. Endocardial resection of the posterior third of the interventricular septum was accomplished through this incision.

Six patients underwent concomitant coronary revascularization with vein bypass grafts.

Results

Eleven of 12 patients survived operation. The single operative death occurred in a patient with a 4% ejection fraction and a recent (10 days) myocardial infarction.

In all patients ventricular tachycardia could be induced by programmed ventricular stimulation intraoperatively. Of particular importance was the fact that following 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.

Prior to discharge all patients underwent electrophysiologic study. 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. Despite the failure to initiate the tachycardia in these patients, ventricular premature depolarizations were observed in all patients prior to discharge in a 24 hour period of Holter monitoring. All patients have been free of symptomatic sustained ventricular tachycardia for a follow-up period of 8 to 18 months. Repeat Holter monitoring in all patients reveals persistence of asymptomatic PVC's. No patients have symptoms of congestive heart failure or a low output state.

Discussion

Ventricular aneurysmectomy with or without coronary vascularization is presently the most commonly employed surgical procedure for the ablation of drug resistent ventricular tachycardia. The efficacy of this form of therapy remains controversial and disappointing.[2,12,14] 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.[3]

Alternative methods of surgical abolition of ventricular tachycardia include cardiac sympathectomy,[3,13] cryosurgery,[4] electrocoagulation,[11] cardiac pacing,[1] and encircling ventriculotomy.[6] Results have been inconsistent.

Spielman and associates[15] have documented sites of epicardial ventricular tachycardia breakthrough that are 5 mm–6 cm distant from the endocardial site of tachycardia origin. These observations in dogs perhaps explain the variable and disappointing results with nondirected surgery or surgical excision directed by epicardial electrophysiological mapping.

The technique of endocardial excision directed by intraoperative endocardial mapping has proven effective in eliminating ventricular tachycardia in our 12 patients. We believe that surgery for cardiac arrhythmias is now on a firm electrophysiologic foundation. The techniques described in this study

FIG. 3. Operative photograph revealing a left ventricular aneurysmectomy. The tachycardia originated from the endocardial surface of the left ventricular free wall. The free wall endocardium is excised for 2–3 cm beyond the limits of the aneurysmectomy.
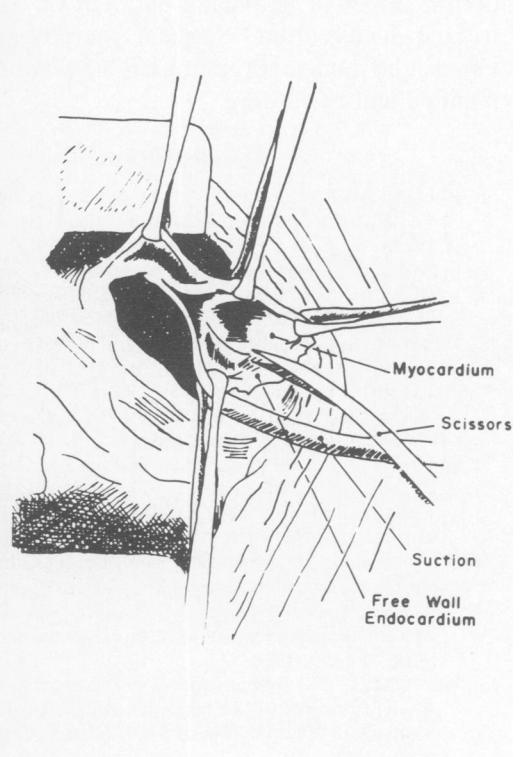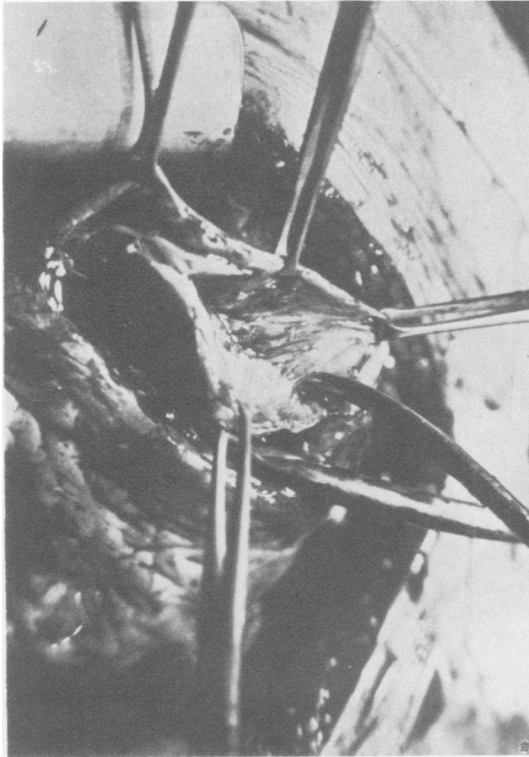

FIG. 4. Operative photograph revealing a posterior ventriculotomy in a patient with a posterior akinetic area, but no discrete aneurysm. The incision is made to avoid the posterior papillary muscle and sufficiently lateral so as not to necessitate inclusion of the posterior descending vessels in the ventriculotomy closure. Endocardial resection of the posterior third of the interventricular septum was accomplished through this incision.
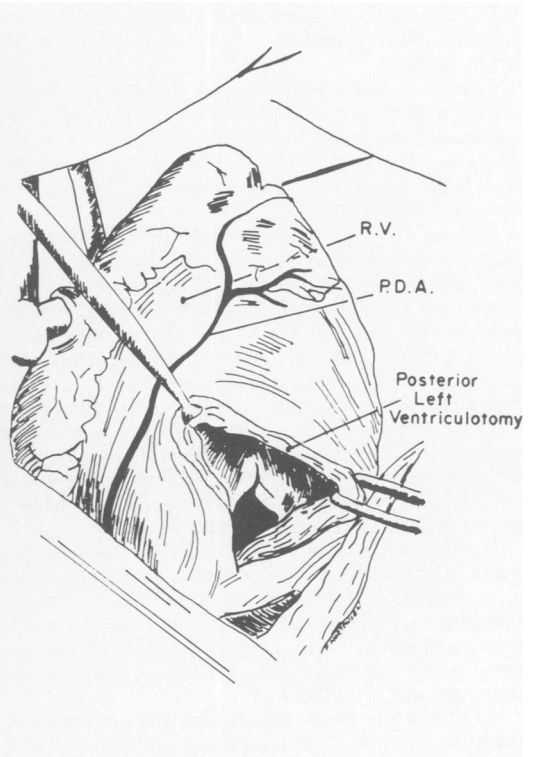
permit effective surgical treatment of recurrent ventricular tachycardia. Surgical therapy of refractory ventricular tachyarrhythmias is now rational, recommended and rewarding.

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DISCUSSION

DR. FRANK C. SPENCER (New York, New York): For at least four reasons, this report may be a major contribution with wide application.

First, in patients with coronary disease, death of arrhythmias is common. How common is simply unknown, because autopsy after sudden death cannot differentiate between an arrhythmia and an infarction. We simply do not know how many people die of something that might be prevented by a operation like this, as opposed to recurrent infarction.

Second, as Dr. Harken indicates, the current therapy for arrhythmias is unsatisfactory, including antiarrhythmic drugs, coronary bypass, or excision of aneurysm. One rather sobering and disappointing example is the data from the West Coast about successful cardiopulmonary resuscitation by laymen of someone who fibrillates on the street. On follow-up study one or two years later, a high mortality is found, probably from recurrence of the same problem, again illustrating the limitation of current forms of therapy.

Thirdly, the operative technique described by Dr. Harken provides a plausible explanation of the problem. He is mapping an electrically unstable area that is probably a mixture of scar and muscle, which would normally not be excised with a ventricular aneurysm and might not be treated by coronary bypass.

Finally, to speculate a bit, all of these patients had aneurysms, but death of arrhythmias, as mentioned earlier, may be common. With increasing use of Holter 24-hour monitoring, it is entirely conceivable that potentially malignant arrhythmias may be recognized and the patients operated upon simply for excision of the lethal myocardial scar. Hence this may not be limited surgically to the patient with aneurysm and arrhythmias.

As referred to in the manuscript, but not mentioned because of pressures of time, Cabrol in France and members of Dr. Sabiston's department at Duke have been studying a different technique, termed "encircling endocardial myotomy," again focusing upon the realization that simple excision of the area is inadequate, and one needs to get beyond the zone of excision to the endocardial scar. With all of these new developments, only long-term evaluation of results from several institutions will make a final decision about the value of this technique. At present, with 15 patients operated upon between two and 24 months, the results are most encouraging.

I have only two questions. It made me a little nervous watching the recurrent tachycardia with the intact heart. Did you have any emboli from dislodgment of clot? Second, do you need both forms of mapping? I could not determine how much the operation was mapping and how much was cutting. Do you have to map both inside and outside, or can you simply open the aneurysm, excise it, do your mapping on the inside, excise that, and simplify your procedure?

These are minor technical points.

DR. ALDEN H. HARKEN (Closing discussion): This paper is pure clinical investigation. We are obviously still trying to learn much about arrhythmias, and this remains a tiny clinical problem.

With better methods of resuscitation and CPR, we are seeing many more of these patients. However, we are still just learning about ventricular arrhythmias. We, therefore, have been performing the epicardial maps and endocardial maps intraoperatively, recognizing that in all probability, at least right now, the epicardial maps are not of value to us, but they are done primarily so that we can learn more about these kinds of arrhythmias.

In answer to your question of the emboli, opening a normothermic, beating heart certainly did worry me. I felt that if we had adequate decompression of the ventricle, I could open the ventricle so that there would be no override, there would be no expulsion of air or clot into the aorta; in fact, we have been fortunate in that area.

As you could see in one of those slides, there was a large mural thrombus there, but we have had no incidence of long-term neurologic deficit.

I believe that the problem of the magnitude of the problem is a real one. As I have stressed, this is pure clinical investigation. We are still trying to learn about the management of arrhythmias. Although these are, at least, to me, encouraging results, and we are now up to about 25 or 26 patients with similar results in the second 12 as with the first 12, I am loath to make any long-term predictions as to whether this is going to be anything of therapeutic value for many people.