Surgical ablation of ventricular tachycardia with sequential map-guided subendocardial resection: electrophysiologic assessment and long-term follow-up

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ABSTRACT A new operative technique of sequential map-guided subendocardial resection (SER) was used in 45 consecutive patients for the treatment of sustained ventricular tachycardia due to coronary artery disease. This technique is characterized by map-guided SER or cryothermic ablation during normothermic cardiopulmonary bypass, followed by repeated sequences of programmed stimulation to assess adequacy of resection. The patients' mean age was 59 ± 10 years and the mean left ventricular ejection fraction was 34 ± 12%. Twenty-five (56%) patients had a history of myocardial infarction within the previous 2 months. After ventriculotomy, 34 patients (76%) had inducible monomorphic ventricular tachycardia. These patients underwent repeated sequences of ventricular tachycardia induction and mapping during normothermic bypass followed by successive SER or cryothermic ablation until sustained monomorphic ventricular tachycardia was no longer inducible. Twenty-seven patients had a total of 60 discrete, mappable tachycardias induced and seven patients had 10 discrete tachycardias that were too fast to accurately map. In the remaining 11 patients, no ventricular tachycardia was inducible after ventriculotomy and SER, which included all visually identifiable scar, was performed. The mean cardiopulmonary bypass time was 102 ± 27 min. Forty-one of 45 patients (91%) survived to hospital discharge, and 35 of 41 patients (85%) had no inducible ventricular tachycardia at postoperative electrophysiologic evaluation performed in the absence of all antiarrhythmic drugs. The remaining six patients had no inducible ventricular tachycardia with drug therapy. All four operative nonsurvivors had refractory cardiac collapse preoperatively. Over 19 ± 12 months of follow-up, there were four sudden cardiac deaths and no nonfatal recurrences of ventricular tachycardia. There were seven additional cardiac deaths. Actuarial cardiac survival was 0.57, and freedom from arrhythmic events was 0.76 at 42 months. Thus, in the absence of cardiogenic shock, the technique of sequential map-guided SER achieves: (1) a high operative survival with acceptable perfusion times, (2) excellent long-term arrhythmia control, and (3) survival comparable to that in patients with similar left ventricular function and no history of ventricular tachyarrhythmia.


LEFT VENTRICULAR aneurysmectomy and subendocardial resection has been demonstrated as an effective means of controlling life-threatening ventricular tachycardia associated with coronary artery disease and left ventricular aneurysm.1–7 The rationale for this approach includes disruption of the site of microreentry at the aneurysm border zone,8–10 as well as alteration of left ventricular wall stress with possible resultant changes in local conduction.11,12 However, a considerable divergence of opinion exists regarding the optimal surgical technique for achieving these goals. Initial nondirected approaches to aneurysmectomy1–3, 5, 13 have been superceded by approaches employing electrophysiologic map-directed subendocardial resection.4, 6, 7, 14 Analysis of data from patients in whom this technique was unsuccessful identified multiple morphologies of ventricular tachycardia and disparate sites of origin of ventricular tachycardia as factors associated with a poor result.15 To address these potential shortcomings, we developed a new technique of
sequential map-guided endocardial ablation. This approach is characterized by: (1) map-guided local subendocardial resection, (2) reinduction of ventricular tachycardia during normothermic bypass, and (3) repetitive sequences of map-guided subendocardial resection and/or cryothermic ablation followed by programmed stimulation until monomorphic ventricular tachycardia is no longer inducible. The results of our surgical series and analysis of factors associated with the success of this approach are presented in this report.

Method

Study patients. Between September 1983 and September 1986, 45 consecutive patients underwent electrophysiologic evaluation, left ventricular aneurysmectomy, and map-guided subendocardial resection at the University of Virginia; the indication was refractory recurrent sustained ventricular tachycardia. All patients had documented coronary artery disease and all had suffered one or more episodes of documented ventricular tachycardia or cardiac arrest not associated with an acute (within 48 hr) myocardial infarction. Patients underwent contrast ventriculography and selective coronary angiography in multiple projections by standard techniques. A coronary artery stenosis was considered significant if there was 50% or more vessel narrowing by visual estimate in any projection. Recommendations for surgical treatment of ventricular tachycardia were based on the presence of an apparent discrete left ventricular aneurysm and the patient’s clinical course. Patients were defined as having preoperative cardiogenic shock if they demonstrated a systolic blood pressure less than 80 mm Hg and showed clinical signs of peripheral hypoperfusion and/or had a measured cardiac index 1.8 liters/min or less. Refractory cardiac collapse was defined as two or more episodes of ventricular tachycardia or fibrillation requiring electrical conversion without an intervening period of hemodynamic stability until the time of surgery.

Electrophysiologic study. Initial electrophysiologic study was performed in the fasting state at least 24 to 48 hr after discontinuation of all antiarrhythmic drugs whenever possible. Three or four quadrupolar electrode catheters were inserted percutaneously into femoral and/or antecubital veins and then advanced under fluoroscopic guidance to positions in the high right atrium, across the tricuspid valve adjacent to the bundle of His, in the right ventricular apex, and when indicated, in the coronary sinus. Intracardiac recordings were filtered between 30 and 500 Hz and displayed on a multichannel oscilloscope. The signals were stored on FM magnetic tape and reproduced for analysis on a high-speed ink-jet recorder at paper speeds of 100 to 200 mm/sec. Programmed cardiac stimulation was performed with a constant-current stimulator that delivered rectangular pulses of 2 msec duration at an output of 2 mA.

The ventricular stimulation protocol used has been described previously. Briefly, it includes single, double, and triple extrastimuli delivered after an 8 beat ventricular drive at paced cycle lengths of 600 (or 500) and 400 msec with the catheter positioned in the right ventricular apex. If no sustained ventricular arrhythmia is induced, the catheter is repositioned to the right ventricular outflow tract and the protocol is repeated. Eleven patients did not undergo preoperative electrophysiologic testing because of the presence of significant disease in the left main coronary artery (four patients) or because of frequent or incessant episodes of hemodynamically unstable ventricular tachycardia that precluded systematic preoperative electrophysiologic evaluation (seven patients).

Serial electrophysiologic testing was performed with a single right ventricular catheter and the protocol outlined above to assess the patients’ responses to drug trials or to surgery. Nine patients underwent preoperative arrhythmia induction and catheter mapping by standard techniques. However, 25 of 34 patients were unable to tolerate prolonged episodes of tachycardia because of hemodynamic instability or anginal symptoms and did not undergo complete preoperative mapping. Postoperative studies were performed 7 to 10 days after surgery. Ventricular tachycardia was defined as sustained if it lasted 30 sec or more or required countershock due to hemodynamic deterioration. At the postoperative electrophysiologic study, control of ventricular tachycardia was defined as failure to induce 15 beats or more of “clinical” ventricular tachycardia. Postoperatively, sustained ventricular tachycardia was considered to be “nonclinical” if only ventricular fibrillation or rapid polymorphic tachycardia was induced with triple ventricular extrastimuli in patients who had previously manifested only monomorphic ventricular tachycardia clinically or during electrophysiologic testing.

Surgical techniques. All patients were anesthetized with nitrous oxide, narcotics, and muscle relaxants. Normothermic cardiopulmonary bypass was initiated with the perfusate temperature maintained at 38° to 40°C. Three pairs of stainless steel electrodes were sutured to the right ventricular free wall and the lateral and posterior left ventricular free walls for recording reference electrograms and programmed ventricular stimulation. Stimulation was performed in an attempt to initiate a sustained ventricular tachycardia. If ventricular tachycardia was not inducible, isoproterenol (2 to 6 μg/min) was infused and programmed stimulation was repeated. If nonmappable rapid monomorphic ventricular tachycardia, polymorphic ventricular tachycardia, or ventricular fibrillation was induced, procainamide (500 to 1000 mg) or lidocaine (75 to 150 mg) was administered in an attempt to organize the rhythm. After induction of ventricular tachycardia, the aneurysm was incised and opened.

Endocardial mapping was performed starting at the border zone of the aneurysm with a hand-held bipolar electrode (2 mm interelectrode distance). A sequential clockwise mapping technique at the aneurysm border as well as 1 cm from the margin as described by Horowitz et al.7 was used. The site of origin of tachycardia was defined as the earliest site of endocardial activation in the latter half of diastole that preceded the onset of the surface QRS complex. Right ventricular endocardial mapping was not performed. After the initial endocardial map was completed, a map-guided subendocardial resection was performed during normothermic bypass (figure 1). After completion of the resection programmed stimulation was repeated. If the same tachycardia or a new morphologic ventricular tachycardia was induced, endocardial mapping was repeated and subendocardial resection at the site of origin of ventricular tachycardia was performed. Repetitive sequences of programmed stimulation, endocardial mapping, and subendocardial resection and/or cryothermic ablation were performed until stable monomorphic ventricular tachycardia was no longer inducible. When required, cryothermy (Ron Wallach, Inc., Fairfield, CT) was applied to the desired location for 2 min with a 1.5 cm² probe cooled to −60°C. Cryothermic ablation was indicated if further resection of viable myocardium would result in unacceptable disruption of left ventricular structures (e.g., deep septum or atrioventricular groove). If the site of origin of tachycardia was repeatedly mapped to the region of a papillary muscle, it was resected and the mitral valve was replaced. The induction of ventricular fibrillation or polymorphic ventricular tachycardia after subendocardial resection was not considered to be clinically significant.18

After ventriculotomy, some patients had rapid monomorphic...
ventricular tachycardia that could not be reliably mapped, even after infusion of procainamide. Because of the short cycle lengths of these tachycardias relative to the QRS duration, the onset of the QRS on the surface electrocardiogram could not be consistently identified since the endocardial electrograms often spanned diastole. In these cases, resection of visually identifiable scar was performed and programmed stimulation was repeated (figure 1). If a nonmappable tachycardia was again induced, repetitive sequences of subendocardial resection and/or cryoablation of border zone tissue were performed until stable monomorphic ventricular tachycardia was not inducible. Finally, in patients in whom no ventricular tachycardia could be induced after ventriculotomy, a single radical resection of all visually identifiable scar, including a margin of border zone tissue, was performed and no further programmed stimulation was done after the initial attempt.

After completion of subendocardial resection, the ventriculotomy was closed, and if no further procedures were required, the patient was weaned from normothermic bypass. If revascularization or valve replacement was required, the patient was cooled after completion of the subendocardial resections, the aorta was cross-clamped, crystalloid cardioplegia was administered, and the procedure was completed by standard techniques.

Postoperative evaluation. Patients were monitored continuously postoperatively until their follow-up electrophysiologic study. Patients in whom clinical ventricular tachycardia was not induced by programmed stimulation were considered to be surgical successes and were discharged on no antiarrhythmic medications. If an arrhythmia similar to one observed before surgery could be initiated, patients underwent serial electropharmacologic testing. These patients were considered to have had surgical modification of their tachycardias if (1) drugs that had been ineffective preoperatively were now effective, or (2) clinically incessant or frequently recurrent and unstable tachycardias were now quiescent off all drugs, but required medication to suppress arrhythmia induction with programmed stimulation.

Patients were seen for follow-up 6 to 8 weeks after hospital discharge and were evaluated for functional status by use of the New York Heart Association (NYHA) classification system. Further follow-up data were obtained during routine clinic visits at 6 to 12 months intervals or by telephone contact with the patients and/or their referring physicians. In patients who died during follow-up, attempts were made to define the cause of death through medical records or death certificates. An arrhythmic death was defined as death that was sudden (i.e., occurring within 1 hr of onset of symptoms and not preceded by primary circulatory collapse) or death preceded by a documented sustained wide-complex tachycardia unassociated with an acute myocardial infarction. Arrhythmic deaths and those associated with acute myocardial infarction or progressive congestive heart failure were classified as cardiac deaths.

Statistical analysis. Individual data were compiled and stored in a computerized data bank. Continuous data are presented as mean values ± SD. To determine differences between group means, a one-way analysis of variance with Duncan's multiple-range test was used. Discrete variables were analyzed with use of contingency tables with appropriate chi square of Fisher's exact statistics. Follow-up event rates were calculated by a Kaplan-Meier life table analysis.

Results

Preoperative data. The study population consisted of 45 patients with clinical characteristics as outlined in table 1. All patients had a history of prior myocardial infarction and 25 patients (56%) had suffered a myocardial infarction within 2 months of surgery. Amiodarone was discontinued 5.4 ± 3.4 days before surgery in the five patients who had received this drug over a long term. A complete preoperative electrophysiologic evaluation was performed in 35 stable patients, with induction of 1.4 ± 0.6 morphologic ventricular tachycardias per patient (25 patients with one ventricular tachycardia, seven patients with two, and three patients with three discrete ventricular tachycardia morphologies). The mean cycle length of ventricular tachycardia was 308 ± 76 msec. Of the 48 tachycardias identified in these patients, 17 had a left bundle branch block morphology, 22 had a right bundle branch block morphology, and nine had an indeterminate morphology.

Operative data. Twenty-seven of the 45 patients (60%) had one or more mappable ventricular tachycardias induced during normothermic cardiopulmo-
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**TABLE 2**
Clinical, electrophysiologic, and operative data from 27 patients with inducible, mappable ventricular tachycardia intraoperatively after ventriculotomy.

Patient No.: identification number of the patient
Age (yr): age in years
Sex: male (M) or female (F)
Recent MI (≥8 weeks): yes (yes/no) indicating whether a recent myocardial infarction occurred
LVEF (%): left ventricular ejection fraction in percentage
Aneurysm location: anterior or inferior
Baseline VT CL: baseline ventricular tachycardia cycle length
Intraop VT CL: intraoperative ventricular tachycardia cycle length
Site: description of the site of the ventricular tachycardia
SER/cryo sequences: site of energy delivery for radiofrequency or cryoablation
CPB time (min): duration of cardiopulmonary bypass in minutes
Periop complications: list of perioperative complications
Inducible VT postop: yes/no indicating whether ventricular tachycardia was inducible postoperatively
TABLE 2  
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ap = apical; ant pap = anterior papillary muscle; CL = cycle length; CPB = cardiopulmonary bypass; cryo = cryothermic ablation; inf = inferior; intraop = intraoperative; LVEF = left ventricular ejection fraction; MI = myocardial infarction; MR = mitral regurgitation; periop = perioperative; postop = postoperative; post pap = posterior papillary muscle; sept = interventricular septum; SER = subendocardial ablation; sup = superior; VSD = ventricular septal defect; VT = ventricular tachycardia.

**Slowing of intraoperative VT compared with baseline VT secondary to treatment with antiarrhythmic drugs at the time of surgery.

†Two or more morphologic tachycardias mapped to a similar site, ablated with a single SER.

nary bypass after left ventriculotomy (table 2 and figure 2). In these 27 patients, a total of 61 discrete morphologic ventricular tachycardias from 47 left ventricular sites were induced (2.3 ± 0.9 ventricular tachycardias/patient). Twenty-one patients had two or more discrete ventricular tachycardias arising from either the same (five patients) or disparate left ventricular sites (16 patients). There was a total of 3.4 ± 2.4 serial resections and/or cryothermic ablations per patient (range 1 to 10). Twelve patients in this group (44%) required two or more resections and/or cryolesions at a single site (all septal) for one morphologic ventricular tachycardia.

Seven of 45 patients (16%) had 10 discrete morphologic ventricular tachycardias induced after ventriculotomy that were too rapid to allow for adequate map-guided resection. Therefore, visually directed surgical resections or cryolesions were performed, with repeat arrhythmia induction attempted after each ablation. A mean of 2.7 ± 2.4 ablations per patient were performed (range 1 to 6) until no further monomorphic ventricular tachycardia could be induced. Because of the rapid nature of the tachycardias in these seven patients, both before and during the operation, data available from a preoperative mapping study to help guide the resection were not available. After serial ablations were completed, 30 of the 34 patients with inducible ventricular tachycardia after ventriculotomy had either no inducible tachyarrhythmia or only ventricular fibrillation induced (figure 3). Four patients continued to have monomorphic sustained ventricular tachycardia, but because further ablations would potentially cause unacceptable disruption of the ventricular architecture, such as creating a ventricular septal defect or damaging the mitral valve anulus, the procedure was terminated.

Eleven of 45 patients (24%) had no inducible sustained monomorphic ventricular tachycardia after ventriculotomy. In five of those patients, ventricular tachycardia had been induced during the operation, but after the aneurysm had been incised the arrhythmia could not be reinitiated. In these 11 patients, a single extended resection of all visible scar with resection of the body of the aneurysm was performed.

Left ventricular aneurysmectomy with subendocardial resection alone was performed in 17 patients. This procedure was combined with coronary artery bypass grafting in 21 patients, with mitral valve replacement in three patients, and with the combination of both in an additional four patients. Total cardiopulmonary bypass time was 102 ± 27 min. Nonfatal postoperative complications included a ventricular septal defect in
FIGURE 2. Intraoperative endocardial map. Tracings show surface electrocardiographic leads I, aVF, reference epicardial electrograms from the right ventricle (RV epi), and left ventricle (LV epi), and endocardial electrograms from the aneurysm border zone (left) and 1 cm deeper into the border zone (right) in a clockwise fashion (12 o’clock = anterior). Presystolic fractionated activity was mapped to sites 10 to 12 and 10-1 to 12-1. Electrogram tracings were retouched.

one patient, and both mitral incompetence and a small ventricular septal defect in another patient.

Surgical mortality. There were four deaths that occurred in the immediate postoperative period or later in the hospitalization directly as a result of complications from surgery. Six patients underwent emergency surgery because of refractory cardiac collapse or cardiogenic shock secondary to a recent cardiac arrest. All four perioperative deaths occurred in this subgroup of patients. One patient developed ventricular fibrillation from which he could not be resuscitated and two patients had irreversible cardiogenic shock within 24 hr of surgery. The fourth patient had a postoperative course complicated by refractory congestive heart failure and sepsis, and died of cardiogenic shock in the late perioperative period. Thus, the operative mortality in this select high-risk subgroup of patients was 67%.

Among the 39 patients who underwent surgery on an elective basis, there were no operative deaths. For the overall series, the surgical survival was 91%. Factors associated with operative mortality (by univariate analysis) included emergency surgery (p = .0001), a
FIGURE 3. Serial electrocardiograms and cardiac electrogars from patient 7 (table 2). A. Recordings from surface electrocardiographic leads I, aVF, and V1, and intracardiac recordings from the right ventricular apex (RVA) during induction of sustained ventricular tachycardia preoperatively. B. Intraoperative recordings during sustained ventricular tachycardia from electrocardiographic lead I, the epicardial electrogram from the right ventricular free wall (RV epi), and the endocardial electrogram from the mid septum (Mid Sept) showing late diastolic activity preceding the surface QRS onset by 53 msec. C. Intraoperative recordings from electrocardiographic lead I and the right ventricular epicardial electrogram during programmed ventricular stimulation. After completion of all resections, only ventricular fibrillation is inducible. D. Recordings from electrocardiographic leads I, aVF, and V1, and intracardiac electrogram from the RVA at the postoperative electrophysiologic study. No ventricular tachycardia is induced despite programmed ventricular stimulation with triple extrastimuli.
greater number of arrhythmic episodes preoperatively (12.5 ± 14.2 vs 4.3 ± 5.4, p = .018), and a higher left ventricular end-diastolic pressure at preoperative cardiac catheterization (31 ± 3 vs 21 ± 8 mm Hg, p = .01). No differences were seen between groups with regard to type of operation, total cardiopulmonary bypass time, or history of recent myocardial infarction.

**Postoperative evaluation.** All 41 operative survivors underwent postoperative electrophysiologic evaluation 6 to 14 days after surgery off of all antiarrhythmic drugs (figure 4). Thirty-five patients (85%) did not have inducible sustained monomorphic ventricular tachycardia. Thirty-four of these patients were discharged on no antiarrhythmic medications; one patient was discharged on 200 mg/day amiodarone for recurrent paroxysmal atrial fibrillation. Three of 41 patients (7%) had an arrhythmia induced at postoperative study that was similar to that observed preoperatively, but showed evidence of ventricular tachycardia modulation as a result of surgery. In two of these patients suppression of arrhythmia was demonstrated (by electrophysiologic criteria) with antiarrhythmic drugs that had been ineffective preoperatively, and they were discharged on procainamide (one patient) and amiodarone (one patient). The third patient had had incessant, medically refractory ventricular tachycardia 2 weeks after acute myocardial infarction. He had no spontaneous recurrences postoperatively, but received long-term amiodarone therapy since a monomorphic ventricular tachycardia was induced during postoperative electrophysiologic evaluation. The remaining three patients had no apparent changes in responses to programmed stimulation after surgery. These patients were treated with amiodarone over the long term, and had no tachycardia induced by programmed stimulation during amiodarone therapy after an oral loading period. Therefore, at the time of hospital discharge, all 41 patients who survived the perioperative period (100%) had successful suppression of the monomorphic sustained ventricular tachycardias induced by programmed stimulation either with surgery alone or surgery in combination with antiarrhythmic medications.

Patients in whom ventricular tachycardia was induced postoperatively were similar to those in whom it was not induced with regard to all clinical and electrophysiologic variables examined, including preoperative ventricular tachycardia cycle length, prior amiodarone use, history of recent myocardial infarction, site of aneurysm resection, number of morphologically distinct tachycardias induced intraoperatively, and number of resections and/or cryolesions performed (table 3). There was no difference in the likelihood of inducing ventricular tachycardia postoperatively between patients treated with sequential subendocardial resection (four of 30 patients) and those treated with a single extended subendocardial resection (two of 11, p = NS).

**Follow-up.** Patients were followed for a mean of 19 ± 12 months (median 16, range 2 to 42), during which time there were 14 deaths. Four patients died of progressive congestive heart failure, three patients died secondary to acute myocardial infarction, and two patients died of noncardiac causes. One patient died as a result of sudden complete heart block with resultant cardiogenic shock. During electrophysiologic evaluation, this patient had had a QRS duration of 60 msec and an HV interval of 45 msec. Four patients died suddenly 6, 10, 22, and 34 months after surgery, respectively. Three of these patients had documented multivessel coronary artery disease, one of whom also had class IV congestive heart failure. None of these patients had had ventricular tachycardia induced at postoperative electrophysiologic evaluation and none

**TABLE 3**

<table>
<thead>
<tr>
<th>Clinical and operative data of patients with and without inducible VT at postoperative electrophysiologic testing</th>
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<tbody>
<tr>
<td>VT not inducible (n = 35)</td>
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<tr>
<td>-----------------------------</td>
</tr>
<tr>
<td>Clinical VT CL (msec)</td>
</tr>
<tr>
<td>Preop amiodarone use (n)</td>
</tr>
<tr>
<td>Recent MI (&lt;2 months)</td>
</tr>
<tr>
<td>Anterior aneurysm location</td>
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<tr>
<td>No. of VTs intraop</td>
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<tr>
<td>VT not inducible intraop</td>
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<tr>
<td>No. of SERs and cryo/patient</td>
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CL = cycle length; cryos = cryothermic ablations; intraop = intraoperative; MI = myocardial infarction; preop = preoperative; SER = subendocardial resection; VT = ventricular tachycardia.
had been treated over the long term with antiarrhythmic medication.

Over the course of follow-up, there were no recurrences of nonfatal sustained ventricular tachycardia. This yielded an actuarial probability of freedom from arrhythmic events in operative survivors of 0.76, and an actuarial probability of arrhythmia-free cardiac survival of 0.57 at 42 months (figure 5). In follow-up, there was one nonfatal myocardial infarction, and six long-term survivors had new or worsening congestive heart failure. The mean NYHA functional class at the time of postoperative follow-up (6 to 8 weeks after hospital discharge) was 1.5 ± 0.8 (class I, 26 patients; II, nine patients; III, six patients).

Discussion

Although the concept of electrophysiologically guided surgical therapy for ventricular tachycardia is supported by experimental studies,9,10 practical application of this approach has been limited by several potential factors. These include the presence of multiple morphologic ventricular tachycardias from disparate sites of origin, difficulties in identification and precise localization of the site and depth of origin of ventricular tachycardia, particularly with septal sites,19,20 and possible inability to induce ventricular tachycardia intraoperatively. The technique of sequential map-guided subendocardial resection described above was developed by modification of the approach originally described by Harken et al.21 in response to these difficulties. By permitting immediate evaluation of efficacy of resection or cryoablation, it simplifies and shortens intraoperative mapping, and results in an excellent surgical outcome.

Our results show that evaluation of surgical efficacy can be reliably assessed intraoperatively by the inability to induce monomorphic ventricular tachycardia. By the standard approach of map-guided subendocardial resection, an intraoperative assessment of the adequacy of resection is not available since resection is performed during cold cardioplegia while the aorta is cross-clamped. This is a significant limitation of that approach since, in our series, ventricular tachycardia was remapped to the same endocardial site after initial map-guided resection in 42% of the patients. This tissue often appeared visually normal after removal of the scarred subendocardium, and was only identified as an incompletely resected site by subsequent initiation of ventricular tachycardia and repeat endocardial mapping.

Since the sequential map-guided resection technique is performed during normothermic bypass, mapping of multiple morphologic ventricular tachycardias from disparate sites is facilitated by allowing sequential ventricular tachycardia induction and mapping after each subendocardial resection. This approach therefore offers a significant advantage over the previous subendocardial resection technique, since in the latter case all sites of ventricular tachycardia must be mapped before the initial resection, an approach that is not always technically feasible. Disparate sites of ventricular tachycardia were observed in 59% of our patients, a prevalence similar to that reported by others.15 In some patients, certain morphologies of monomorphic ventricular tachycardia were induced intraoperatively that had not been observed clinically, especially in those individuals with recent myocardial infarction. Although the significance of these rhythms is uncer-
tain, there is evidence to suggest that they may recur postoperatively if inadequately treated. The theoretical advantages of the technique of sequential subendocardial resection are further supported by the excellent outcome by both electrophysiologic and clinical criteria, and by an acceptable surgical mortality in this high-risk patient population.

**Previous studies.** Standard map-directed subendocardial resection has a reported success rate of 65% to 80%, as defined by the inability to induce monomorphic sustained ventricular tachycardia at postoperative electrophysiologic study in the absence of antiarrhythmic drugs. Nondirected radical endocardial resection has yielded approximately equivalent results, and is particularly useful when the clinical ventricular tachycardia cannot be initiated after ventriculotomy. However, problems still exist regarding the possibility of unnecessary resection of viable myocardium, resulting in hemodynamic compromise. In addition, identification of tissue planes, especially in the period soon after myocardial infarction, may be difficult and may result in resection of too much or too little tissue. With the use of sequential subendocardial resection, extension of the resection into the intramural interventricular septal myocardium was frequently necessary to completely ablate the ventricular tachycardia induced intraoperatively. These tachycardias localized to the septum may be similar to the ventricular tachycardias described in patients with coronary disease that are thought to originate from the right ventricle. Our results suggest that a right ventriculotomy, as recommended by Kraitch et al., may not be necessary in these patients since the site of origin may be within the interventricular septum and approachable through the left ventriculotomy alone. The results of postoperative electrophysiologic testing and clinical follow-up in the present study suggest that the present technique is an improvement over approaches that use a single focal map-directed resection. These results are comparable to those reported by authors using more radical resection techniques, but the sequential approach may result in less resection of viable myocardial tissue.

The overall operative mortality of the present study is slightly lower than that in most other series, but examination of individual patient outcomes highlights the importance of patient selection in interpretation of these data and in comparison of surgical mortality among different centers. The 67% and 0% operative mortalities for patients undergoing emergency and elective procedures, respectively, suggest that changes in the characteristics of population to whom the operation is offered can lead to dramatic differences in overall surgical outcome. This observation suggests that withholding surgery as a final option for patients in whom all other modalities have failed may not be optimal, and that surgical therapy should be considered as an early option in selected stable patients. Also, the observation that two of six patients with ongoing cardiac arrests survived surgery and had a benign postoperative course implies that even an admittedly high-risk surgical intervention should be considered in patients who face almost certain mortality with medical therapy alone.

**Limitations.** The relatively high prevalence of patients in whom tachycardia could not be induced intraoperatively after ventriculotomy (24%) is higher than that reported by some authors, but similar to the experience reported from other large series. Our patient population may be different from some reported series, with a higher prevalence of patients with recent myocardial infarctions and/or shorter cycle lengths of the tachycardias observed clinically and at electrophysiologic study. Although attempts were made to perform preoperative catheter mapping, this was not practical in the majority of cases due to hemodynamic instability during tachycardia, and therefore these limited data were not included in the analysis.

A theoretical limitation of our approach is that it might require prolonged periods of normothermic bypass. However, the actual mean bypass times were well within the range reported by others. Although extended periods of normothermic cardiopulmonary bypass could conceivably lead to impairment of left ventricular function, no ischemic arrest was induced during normothermia and hence coronary perfusion during normothermia was always maintained. Additionally, no significant impairment in functional class was observed in the majority of our patients during postoperative follow-up. Since there was no randomization or comparison with patients who did not undergo this surgical intervention, however, comparisons with other surgical series and nonsurgical forms of therapy must be approached with caution.

**Clinical implications.** Our results suggest that the sequential map-guided subendocardial resection technique compares favorably with previously used surgical approaches for recurrent ventricular tachycardia. The advantages of our approach include facilitation of mapping of ventricular tachycardia of disparate sites and that originating from deep within the interventricular septum. In addition, it provides immediate assessment of the efficacy of resection. This technique has an acceptably low operative morbidity and mortality, particularly in hemodynamically stable patients. The long-
term outcome of these patients compares favorably with that of patients treated with long-term antiarrhythmic drug therapy who have comparable impairment of left ventricular function, and avoids the significant toxicity associated with long-term antiarrhythmic drug therapy. Although surgical therapy for the treatment of arrhythmias has traditionally been reserved for patients in whom all other forms of therapy have failed, the above results suggest that it should be offered to selected patients early in their therapeutic course, before the initiation of potentially toxic drugs.

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