

Surgery for Ventricular Tachycardia

Gerald M Lawrie, MD

HOUSTON METHODIST DEBAKEY HEART & VASCULAR CENTER, HOUSTON METHODIST HOSPITAL, HOUSTON, TEXAS

ABSTRACT: The treatment of drug-refractory chronic ventricular tachycardia (VT) has undergone a revolution over the last 50 years. We now have automatic implantable cardioverter defibrillator therapy with pace-terminating capabilities, and catheter ablation of VT has refined mapping and improved methods of lesion generation. Between 1980 and 1993, Houston Methodist Hospital became a leader in the diagnosis and surgical ablation of VT and other arrhythmias. This is a brief account of that period and some of the experiences and lessons that have led to significant advances used today.

INTRODUCTION

Fifty years ago, patients with recurrent sustained ventricular tachycardia (VT) or resuscitated VT and ventricular fibrillation (VF) had a bleak prognosis. Treatment was mostly limited to drugs, some of which had serious side effects including proarrhythmic exacerbations that were sometimes fatal. Some patients had VT resistant to multiple drugs or developed severe side effects during their treatment, requiring cessation of the only drug capable of treating their VT. They often required prolonged hospital admissions on high doses of intravenous medications.

The majority of these patients had advanced coronary artery disease with a history of one or more myocardial infarctions. In this period, the late 1960s and early 70s before the introduction of coronary angioplasty or thrombolysis, patients who had an acute myocardial infarction (MI) often developed chronic scarring in the ventricular myocardium. A minority of patients would develop large left ventricular (LV) aneurysms. Recurrent MIs were common, as were depressed ejection fractions.

THE BEGINNING OF SURGICAL THERAPY FOR VT

By 1970, following the widespread adoption of coronary artery bypass surgery (CAB), isolated revascularization had been used in a number of patients with chronic recurrent VT. Unfortunately, isolated CAB was found to be of little benefit.^{1,2} This was because chronic recurrent VT in ischemic heart disease is most commonly due to reentrant circuits based on subendocardial scars, not active ischemia.

LEFT VENTRICULAR ANEURYSMECTOMY

In 1953, Wasserman and Yules reported a death from drug-refractory VT of a patient with a long history of postinfarction LV aneurysm (LVA) and postulated that the LVA was the source of

the VT.³ The first successful surgical treatment of a postinfarction LVA was performed in 1955 by Likoff and Bailey to relieve congestive heart failure (CHF), not VT. They excised the LVA without cardiopulmonary bypass.⁴ The first reported surgical cure of VT by LVA resection was reported by Couch in 1958.⁵ In the next decade, there were sporadic reports of LVA resections for VT with variable results.⁶

At this point, the experience at Houston Methodist (then known as The Methodist Hospital) was confined to patients with LVAs treated with LVA aneurysmectomy performed blindly without electrophysiology (EP) guidance as one part of a procedure designed to alleviate CHF; this was often performed with concomitant procedures such as CAB and valve surgery for mitral regurgitation. Cure of VT was considered a bonus. For patients with monomorphic VT and localized apical LVAs with well-preserved basal LV function, the drug-free cure rates averaged about 50% and the mortality rate was about 10%. These results of conventional LVA resection were disappointing, not only because they failed to cure the VT but also because recurrences of drug-refractory VT postoperatively in the presence of an already compromised LV was sometimes fatal.

INTRODUCTION OF MAP-DIRECTED VT SURGERY

The introduction of preoperative clinical endocardial EP mapping in the 1970s led to recognition that the approximate position of the fixed pathologic substrate from which drug-refractory monomorphic VT usually arose could be identified by preoperative endocardial mapping. This was first established in patients with scarred areas from healed myocardial infarctions that are located mainly subendocardially.

Once pre- and intraoperative mapping data became available in patients with LVAs, the sites of VT origin were commonly

found not where the dense LVA scar ended but within the transition zone between scar and viable muscle. With growing experience of preoperative catheter EP mapping, it became apparent that the low cure rate of VT from conventional LVA resection was because the VT originated not from the fibrous bulge of the LVA resected by traditional LVA surgery but from the transition zone where fibrous tissue was intermingled with viable myocardium. It was this mixture of scar and viable muscle that provided the heterogeneity of electrical conduction (anisotropism) that was ideal for forming the reentrant circuits reported by Wellens in 1972.⁷

However, scarring could occur anywhere in the ventricles, and the VT could occur without the presence of a large scar such as an LVA. Intraoperative visual inspection was unreliable because only a minority of scars had the essential complex electrical qualities to initiate and sustain VT. Furthermore, as more patients were identified with nonischemic VT, it became apparent that the intraoperative appearance of the ventricles could be normal to gross inspection.⁸

Once it was demonstrated that the VT approximate site of origin could be localized, interest in surgical ablation of these sites rapidly followed. In 1975, Wittig and Boineau reported on a surgical cure of three patients with intractable VT who underwent intraoperative epicardial and endocardial mapping.⁹ In 1975, Gallagher et al. reported a successful cure of VT during LVA resection using epicardial map guidance.¹⁰ In 1975, Spurrell et al. reported on two map-directed cases of non-aneurysm-related VT cured by surgical ablation.¹¹ Three years later, Josephson et al. reported results of preoperative endocardial mapping in 17 patients.¹² Good correlation was found between preoperative endocardial mapping findings and subsequent intraoperative surgical findings.

In 1978, Guiradon introduced a new technique for postinfarction VT called “encircling endocardial ventriculotomy” that was designed to create a line of block to prevent re-entry from the scarred areas of the LV (Figure 1 C).¹³ EP mapping was not considered necessary pre- or intraoperatively, and all five patients were cured of VT. He subsequently modified this by using rows of cryolesions instead of incisions. Although innovative, this procedure was used very little in the United States.

MAP-DIRECTED VT SURGERY LAUNCHES

In 1979, Josephson et al. reported a new surgical technique called “subendocardial resection” in which dissection was performed along a plane between the scarred muscle and the deeper normal muscle (Figure 1 A). This technique removed all the junctional area of mixed scar and viable muscle extending from the dense scar of the LVA into

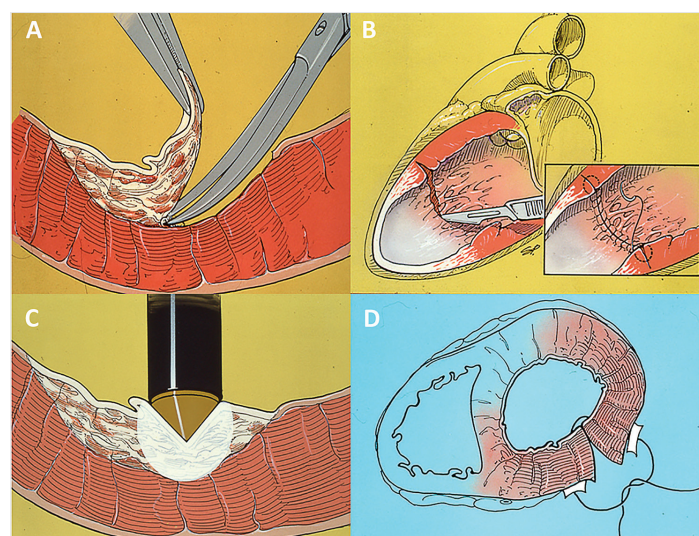


Figure 1.

Surgical techniques. (A) Endocardial resection. (B) Cryotherapy at -70°C . (C) Encircling endocardial incision. (D) Excision of arrhythmogenic tumors or myocardium.

normal myocardium.¹⁴ In this landmark study, which included 12 patients with a history of MI as well as preoperative programmed electrical stimulation (PES), all but one tolerated endocardial mapping of their VT. All the VTs were located in the general area of the LVAs. All underwent intraoperative biventricular epicardial mapping during sinus rhythm, and all experienced VT induced by PES while supported on cardiopulmonary bypass (CPB). The LVA was entered while on CPB. Endocardial mapping was performed between 36 and 50 sites along the border and elsewhere. When a VT site of origin was identified within 2 cm of the LVA border, resection of the scarred thickened endocardium was performed back 2 to 3 cm onto normal muscle. These observations were expanded to 100 patients followed for 28 ± 19 months.¹⁵ Late survival was 91%. Cure of VT with surgery alone was 66% (60/91 patients). Factors associated with surgical failure were VT sites > 5 cm apart (64% vs 30% failure), multiple morphologies (47% vs 25 % failure), VT sites of origin (inferior wall 41% vs 12%), no discrete LVA, and VT with right bundle branch block morphology (20% vs 7% failure). This landmark paper energized interest worldwide in map-directed surgery for ischemic scar-related VT.

While ischemic heart disease has accounted for the large majority of VT ablation surgeries, a significant minority of patients with nonischemic VT have also undergone map-directed surgery.⁸ These patients had a variety of etiologies for their VT and in many cases had some important EP differences from the ischemic patients. We found pre- and intraoperative EP mapping guidance essential when treating these patients.

ELECTROPHYSIOLOGIC MAP-DIRECTED SURGERY AT HOUSTON METHODIST HOSPITAL

In August 1980, Dr. Christopher Wyndham became chief of the Cardiac Electrophysiology Unit at Houston Methodist and Baylor College of Medicine. He soon established a large and productive program with significant academic output. The hospital could now undertake a range of surgical procedures performed with guidance from EP studies performed pre-, intra- and postoperatively. In addition to VT, we began to treat a full range of supraventricular arrhythmias.

DEVELOPMENT OF COMPUTERIZED AUTOMATED EP MULTIPPOINT MAPPING

In 1980, there was still debate about the value of activation mapping in surgical VT ablation.¹⁶ However, the results of non-map-directed surgery were generally poor and unpredictable. We concluded that the most sophisticated level of epicardial and endocardial mapping data would be essential to better understand the mechanisms of cardiac arrhythmias. Accurate substrate localization, both in terms of site and extent of ablation needed, would facilitate improvement of surgical techniques to meet the goal of 100% drug-free cures and lower surgical mortality rates.

At first, we had only hand-held bipolar probes and mapped point to point. The first report of a sock-mounted electrode array in humans was by Gallagher et al. in 1982.¹⁷ However, the sock was inelastic, and good contact of all electrodes was difficult to obtain. In 1983, de Bakker et al. reported the first global ventricular endocardial mapping with an electrode array mounted on an inflatable balloon.¹⁸ We immediately began to develop a computerized pre- and intraoperative multichannel mapping system capable of simultaneous data acquisition from the epicardium and endocardium using sock and balloon arrays. The ultimate goal was to study myocardial activation in three dimensions. One of the electrical engineers involved was Matt Pruka, who subsequently led the successful commercialization of this work into a fully automated EP lab system (Pruka Engineering).

In the mid-1980s, the first prototype system became available, consisting of an epicardial sock and an endocardial balloon.¹⁹ Printed circuits were fabricated on long strips of Kapton tape. Each of these individual printed circuits had copper wire runs going to 20 individual electrodes, and adjacent pairs of electrodes were matched to form bipolar electrodes. There were 120 electrodes on the sock and 80 on the balloon. These electrode signals were analyzed by a custom computerized system.^{19,20} Color isochronal map generation was automated and rapid (Figure 2).¹⁹

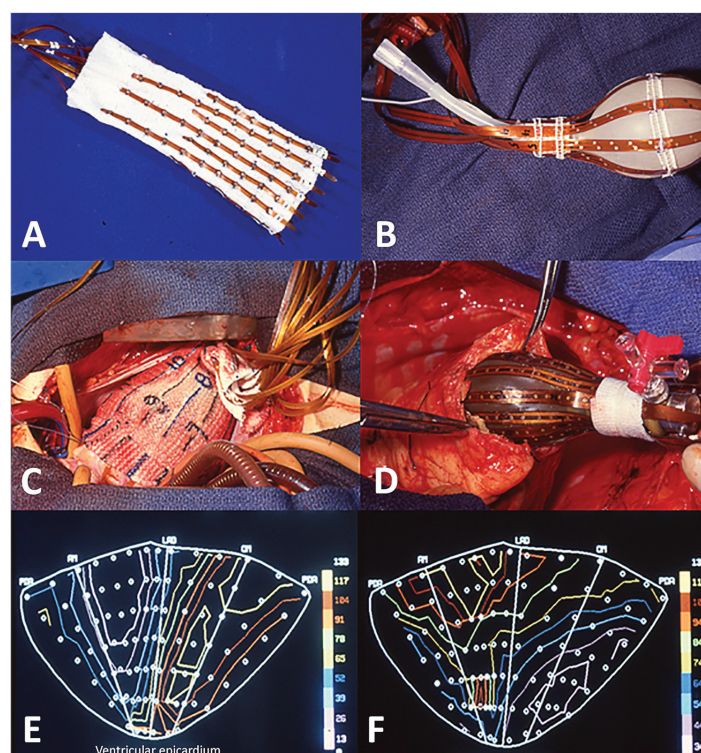


Figure 2.

(A) Epicardial mapping sock with Kapton strips holding electrodes. (B) Endocardial mapping balloon with Kapton strip electrodes. (C) Sock positioned on left ventricular (LV) epicardium, apex is to right. (D) Balloon being inserted in the beating heart through a small apical ventriculotomy into the LV, where it will be inflated. (E) Automated epicardial activation map in sinus rhythm. (F) Map in ventricular tachycardia showing inferoapical earliest activation below posteromedial papillary muscle.

This technology greatly improved the quality and completeness of the mapping studies and reduced the time needed to perform them, which was important since many VTs were rapid and poorly tolerated, causing unstable angina, hypotension, or acute heart failure. Intraoperative mapping often required cardiopulmonary bypass support, and the VT could be nonsustained, converting to sinus rhythm. Some general anesthetic agents proved to have excellent antiarrhythmic qualities. The VT could deteriorate after induction directly into VF before point-to-point mapping could be completed. Intraoperative endocardial mapping data was often difficult to obtain because of difficulty in inducing sustained VT.

At Houston Methodist, the full mapping system was used in the last 57 of the 128 patients who received surgical VT ablations. The ability to obtain accurate mapping from a single beat of VT would prove to be transformational: When hand-held point-to-point mapping was used, only 75% of patients had any useful mapping data to guide the surgery, whereas with the new automated system, 95% had useful data. This led to lower

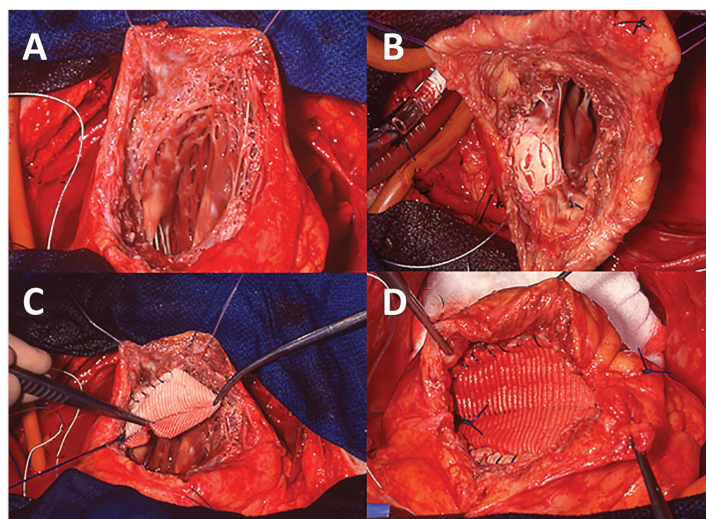


Figure 3.

Same patient as shown in Figure 2. (A) Anteroapical and inferior left ventricular aneurysm (LVA) opened and extensive endocardial resection performed. (B) Base of posteromedial papillary muscle cryoablated with 20 mm lesion. (C) Oval Dacron patch is being sewn to junction of LVA with normal tissue, deep within the fibrous LVA to remodel LV cavity. (D) Patch closure is completed. Scar of aneurysm will be closed over patch.

surgical mortality by shortening the amount of myocardial stress induced by recurrent induction of rapid VT and marked reduction in perioperative VT recurrence.^{8,16,21-23}

SURGICAL TECHNIQUES

The most common surgical techniques used initially were extended LV aneurysm resection combined with subendocardial resection.^{14,15,24} Initially, cryotherapy was rapidly introduced as a supplement for ablation of septal foci to avoid the perforation sometimes seen from endocardial resection on the septum (Figure 3), but it soon became our main method of ablation at all sites (Figures 1-4).

Because cryotherapy destroys only myocardial cells, the coronary arteries and connective tissue framework of the myocardium are preserved; therefore, it can be used on the interventricular septum, the mitral and tricuspid annuli, and the papillary muscles with no risk of provoking mitral regurgitation. Cryotherapy produces a razor-sharp line of scar after the lesion heals, with no residual anisotropism. When patients had no LVA, the most common ablative technique used was cryotherapy.⁸

Cryotherapy has only two disadvantages: (1) Each nitrous oxide cryolesion requires 2 minutes to create (we used a 20-mm probe tip whenever possible for VT ablation with cryotherapy), and (2) the spreading ball of frozen muscle eventually reaches a size where the insulating properties of the ice prevent further enlargement.

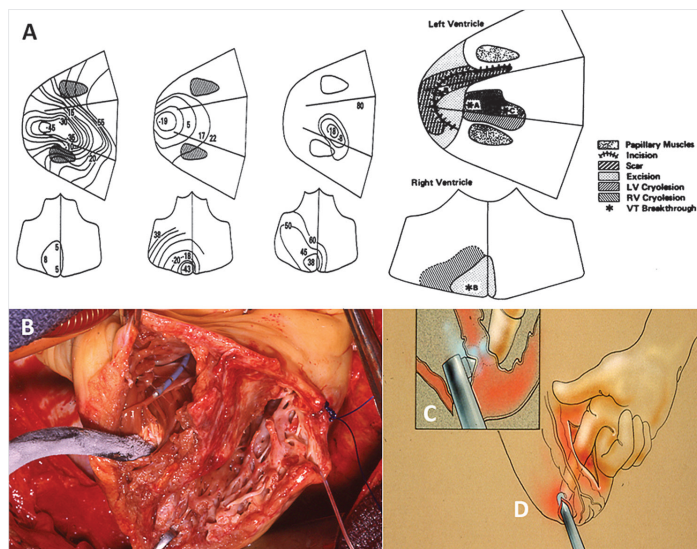


Figure 4.

(A) Patient with 3 ventricular tachycardia breakthroughs mapped pre- and intraoperatively, 2 on the left ventricular side of septum and 1 on the right ventricular side. Locations of breakthroughs and procedures performed are shown in diagram. (B) Biventricular cryotherapy was applied to the septum, C and D, as well as apical left ventricular aneurysm resection.

Thus, where depth of penetration was critical, two maneuvers had to be used. First, the tissue and probe were firmly pressed against each other. Second, this was insufficient at times, especially on the septum, and cryotherapy had to be applied to both ventricular endocardial surfaces (Figure 4).²⁵ When planning cryoablation, we generally assumed a potential failure to map the full extent of the area supporting the reentrant area break-out on the mapping; therefore, we would cryoablate at least an additional 1- to 2-cm area around the mapped site of earliest activation. Using an “extended” ablation, we would start ablating areas between 8 cm² and 25 cm² and expand the ablation area to up to 40 cm². This raised our cure rate to > 95% while lowering surgical mortality.²¹ For patients who did not require a ventriculotomy as part of their surgery, we developed transannular cryoablation used in conjunction with transannular insertion of the balloon endocardial mapping array figure (Figure 5).²⁶

THE AUTOMATIC IMPLANTABLE CARIOVERTER DEFIBRILLATOR

The treatment of those who survived VT/VF arrests was revolutionized by the development of the automatic implantable defibrillator, which was first implanted by Mirowski et al. on February 4, 1980.²⁷ Soon the automatic implantable cardioverter defibrillator (AICD) was developed to prevent deaths from shock-induced asystole. Later, PES was incorporated to terminate arrhythmias.²⁸ This continues to be the most effective therapy to prevent death from these arrhythmias.

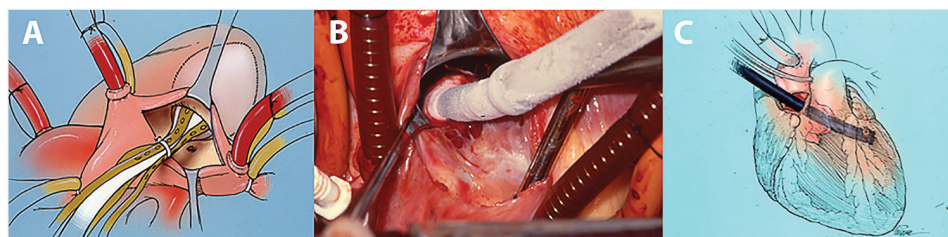


Figure 5.

Transannular balloon mapping and cryoablation of a high right ventricular septal nonischemic lesion. (A) Balloon inserted through tricuspid valve on the beating heart. (B) Transannular cryotherapy was performed on the cold arrested heart. No ventriculotomy was performed. (C) In several other cases, transaortic cryotherapy was performed.

In 1980, we joined the first preclinical trial of the first Intech AICD device (Intech Systems Group), which at the time required a sternotomy or thoracotomy incision to implant two large epicardial patches and an epicardial pacing wire. Because of its large size, the generator was implanted in the upper abdomen under the rectus abdominis muscle. We developed a left subcostal limited access approach for implantation that reduced the magnitude of the surgery in these critically ill patients.²⁹ We also gained valuable insight studying the impact of lead geometry, site, and polarity on lead thresholds.^{30,31} In 1983, we received the Governors' Award at the American College of Cardiology 32nd Annual Scientific Session, where our scientific exhibit on surgical treatment of cardiac arrhythmias received the First Place Award for Excellence (Figure 6).

SURGERY RESULTS FOR VENTRICULAR TACHYCARDIA

Between 1980 and 1991, we performed 128 map-directed VT ablations. Ischemic VT accounted for 114 of these patients while 14 were treated for nonischemic VT. Perioperative mortality was 8.1% for those with ischemic VT overall. The mean preoperative ejection fraction (EF) was 38.3% for survivors versus 24.9% for those who died (4 of which had EF < 20%). For patients with an LVEF > 31%, perioperative mortality was 1%. There was no mortality in the 14 patients with nonischemic

VT. The overall recurrence of VT was 12%, 4% in the later experience between 1986 and 1991.¹⁶ These results compared favorably with a review by Onufer and Cain that reported a 14% mortality rate and 28% reinducibility rate of VT in 1,236 patients from 24 prior studies.³²

During this same period, we began implanting an increasing number of AICDs. Our total experience with the epicardial AICD system reached 256 patients with a perioperative mortality of 3.9%. Improved results were obtained with use of the limited access left subcostal thoracotomy.²⁹



Figure 6.

Governors' Award from American College of Cardiology (1983).

SURGERY FOR SUPRAVENTRICULAR TACHYCARDIA

By 1985, we had performed 580 intracardiac EP studies, and 90 patients had undergone map-directed arrhythmia surgeries for a wide range of diagnoses with excellent results.³³ By the end of our EP surgery program, we had performed map-directed ablation of 123 patients with Kent bundles, mostly off-pump via an epicardial approach. We also performed smaller numbers of AV-nodal ablations, dual AV-nodal pathways, ablation of atrial flutter and atrial focal tachycardias, and 45 cut-and-sew Maze procedures. There were no deaths, and the long-term cure rate overall was 98%.^{33,34}

THE END OF MAP-DIRECTED VT SURGERY

By the early 1990s, it was apparent that referrals for direct map-guided VT surgery were declining rapidly. Perioperative mortality was high in the early 1980s, and this bad reputation had lingered. In the early days of VT surgery, many patients with EFs between 15% and 30% in critical condition with no other options were operated on as a last resort. Many would now have received an AICD and medication. The early point-to-point mapping was slow and laborious, and mapping of VT morphologies was often incomplete. Advanced coronary artery disease was usually present.

Use of thrombolytic therapy and early balloon angioplasty that reduced infarct size was now the standard management of acute myocardial infarction. LVAs at this point were rarely seen. In a recent study,³⁵ the current incidence of LVA development after acute MI was estimated at 0.14%, and VT/VF developed in 18.11% of these patients. The incidence of LVA resection declined from 14.5% in 2006 to 9.8% in 2014. While this constitutes a relatively small number of patients, LVA is a progressive condition that may produce congestive heart failure. When combined with VT, the situation may lead to referral for heart transplantation.

THE STICH TRIAL, LEFT VENTRICULAR ANEURYSMS, AND VT

The STICH trial (The Surgical Treatment for Ischemic Heart Failure) evaluated patients with an EF $\leq$ 35% and “anterior akinesia or dyskinesia amenable to surgical ventricular reconstruction” to determine if resecting dysfunctional, scarred myocardium designed to remodel the LV wall and produce a smaller LV volume would improve survival compared with CAB alone.³⁶ Although STICH did not study the effect of resecting true, discrete, fibrous LVAs (and LVA is never mentioned in STICH publications), the negative finding that remodeling of diffusely hypokinetic LVs is ineffective has led many cardiologists to believe that resection of true LV aneurysms is futile. On the contrary, this is an effective therapy to relieve CHF in patients in whom the regional EF of the basal portion of the LV is preserved; in the context of this review, it is also an opportunity to ablate VT when present. Ideally, these patients will have well-preserved residual basal LV function and in systole will show a clear line of demarcation between the contractile muscle and the aneurysmal balloon-like expansion. Modern techniques of LVA resection use patch closure of the residual LV to optimize myocardial fiber orientation and LV volume, and results have improved significantly.³⁶

CONCLUSION

The era of map-directed VT surgery contributed to our current understanding of VT in several ways. It established the re-entrant nature of most VTs. It enabled human studies of the structure and function of scars that cause VT. It accelerated the development of multipoint computerized mapping systems. It led to recognition of the multiple sites of VT origins in some patients and the phenomenon of multiple morphologies arising from a single substrate in others. It enabled cure of VT from tumors and a variety of other nonischemic conditions.

Map-directed VT surgery should still be considered an option for patients with mappable VT who have had unsuccessful catheter ablation and who continue to experience clinically troublesome episodes. It should also be considered if a patient needs open heart surgery for another indication. Patients with true LVAs, CHF, and VT may benefit from re-evaluation for direct surgery and LVA resection, perhaps avoiding heart transplantation.

KEY POINTS

- The introduction of computerized pre- and intraoperative mapping revolutionized the results of map-directed surgery for ventricular tachycardia (VT).
- Resection of discrete left ventricular aneurysms (LVAs) combined with map-directed VT ablation still may be useful in some patients with congestive heart failure and VT. The STICH trial did not study the results of LVA resection.
- Drug-free cure rates of > 90% can be achieved with these techniques in properly selected patients.

Corresponding Author:

gmlawrie@att.net

Conflict of Interest Disclosure:

The author has completed and submitted the *Methodist DeBakey Cardiovascular Journal* Conflict of Interest Statement and none were reported.

Keywords:

ventricular tachycardia, arrhythmia ablation, left ventricular aneurysm

REFERENCES

1. Lehrman KL, Tilkian AG, Hultgren HN, Fowles RE. Effect of coronary arterial bypass surgery on exercise-induced ventricular arrhythmias. Long-term follow-up of a prospective randomized study. *Am J Cardiol*. 1979 Nov;44(6):1056-61. doi: 10.1016/0002-9149(79)90169-3.
2. Weiner DA, Levine SR, Klein MD, Ryan TJ. Ventricular arrhythmias during exercise testing: mechanism, response to coronary bypass surgery and prognostic significance. *Am J Cardiol*. 1984 Jun 1;53(11):1553-7. doi: 10.1016/0002-9149(84)90578-2.
3. Wasserman E, Yules J. Cardiac aneurysm with ventricular tachycardia: case report and brief review of the literature. *Ann Intern Med*. 1953 Oct;39(4):948-56. doi: 10.7326/0003-4819-39-4-948.
4. Likoff W, Bailey CP. Ventriculoplasty: excision of myocardial aneurysm; report of a successful case. *J Am Med Assoc*. 1955 Jul 16;158(11):915-20. doi: 10.1001/jama.1955.02960110021006.
5. Couch OA. Cardiac aneurysm with ventricular tachycardia and subsequent excision of aneurysm; case report. *Circulation*. 1959 Aug;20(2):251-3. doi: 10.1161/01.cir.20.2.251.
6. Sami M, Chaitman BR, Bourassa MG, Charpin D, Chabot M. Long term follow-up of aneurysmectomy for recurrent ventricular tachycardia or fibrillation. *Am Heart J*. 1978 Sep;96(3):303-8. doi: 10.1016/0002-8703(78)90040-6.
7. Wellens HJ, Schuilenburg RM, Durrer D. Electrical stimulation of the heart in patients with ventricular tachycardia. *Circulation*. 1972 Aug;46(2):216-26. doi: 10.1161/01.cir.46.2.216.
8. Lawrie GM, Pacifico A, Kaushik R. Results of direct surgical ablation of ventricular tachycardia not due to ischemic heart disease. *Ann Surg*. 1989 Jun;209(6):716-27. doi: 10.1097/00006558-198906000-00009.
9. Wittig JH, Boineau JP. Surgical treatment of ventricular arrhythmias using epicardial, transmural, and endocardial mapping. *Ann Thorac Surg*. 1975 Aug;20(2):117-26. doi: 10.1016/s0003-4975(10)63864-7.
10. Gallagher JJ, Oldham HN, Wallace AG, Peter RH, Kasell J. Ventricular aneurysm with ventricular tachycardia. Report of a case with epicardial mapping and successful resection. *Am J Cardiol*. 1975 May;35(5):696-700. doi: 10.1016/0002-9149(75)90059-4.

11. Spurrell RA, Yates AK, Thorburn CW, Sowton GE, Deuchar DC. Surgical treatment of ventricular tachycardia after epicardial mapping studies. *Br Heart J*. 1975 Feb;37(2):115-26. doi:10.1136/hrt.37.2.115.
12. Josephson ME, Horowitz LN, Farshidi A, Spear JF, Kastor JA, Moore EN. Recurrent sustained ventricular tachycardia. 2. Endocardial mapping. *Circulation*. 1978 Mar;57(3):440-7. doi:10.1161/01.cir.57.3.440.
13. Guiraudon G, Fontaine G, Frank R, Escande G, Etievent P, Cabrol C. Encircling endocardial ventriculotomy: a new surgical treatment for life-threatening ventricular tachycardias resistant to medical treatment following myocardial infarction. *Ann Thorac Surg*. 1978 Nov;26(5):438-44. doi:10.1016/s0003-4975(10)62923-2.
14. Josephson ME, Harken AH, Horowitz LN. Endocardial excision: a new surgical technique for the treatment of recurrent ventricular tachycardia. *Circulation*. 1979 Dec;60(7):1430-9.
15. Miller JM, Kienzie MG, Harken AH, Josephson ME. Subendocardial resection for ventricular tachycardia: predictors of surgical success. *Circulation*. 1984 Oct;70(4):624-31. doi:10.1161/01.cir.70.4.624.
16. Lawrie GM, Pacifico A. Surgery for ventricular tachycardia. In: Zipes DP, Jaliffe J, editors. *Cardiac electrophysiology from cell to bedside*, 2nd edition. Philadelphia, PA: W. B. Saunders Company; 1995. p. 1547-1552.
17. Gallagher JJ, Kasell JH, Cox JL, Smith WM, Ideker RE, Smith WM. Techniques of intraoperative electrophysiologic mapping. *Am J Cardiol*. 1982 Jan;49(1):221-40. doi:10.1016/0002-9149(82)90296-x.
18. de Bakker JM, Janse MJ, Van Capelle FJ, Durrer D. Endocardial mapping by simultaneous recording of endocardial electrograms during cardiac surgery for ventricular aneurysm. *J Am Coll Cardiol*. 1983 Nov;2(5):947-53. doi:10.1016/s0735-1097(83)80244-7.
19. Pieper CF, Lawrie GM, Parsons D, Lacy J, Roberts R, Pacifico A. Design and implementation of a new computerized system for intraoperative cardiac mapping. *J Appl Physiol* (1985). 1991 Oct;71(4):1529-39. doi:10.1152/jappl.1991.71.4.1529.
20. Pieper CF, Lawrie GM, Roberts R, Pacifico A. Bandwidth-induced errors in parameters used for automated activation time determination during computerized intraoperative cardiac mapping: theoretical limits. *Pacing Clin Electrophysiol*. 1991 Feb;14(2 Pt 1):214-26. doi:10.1111/j.1540-8159.1991.tb05092.x.
21. Krafchek J, Lawrie GM, Roberts R, Magro S, Wyndham CR. Surgical ablation of ventricular tachycardia: improved results with a map-directed regional approach. *Circulation*. 1986 Jun;73(6):1239-47. doi:10.1161/01.cir.73.6.1239.
22. Lawrie GM, Pacifico A, Kaushik R, Nahas C, Earle N. Factors predictive of results of direct ablative operations for drug-refractory ventricular tachycardia. Analysis of 80 patients. *J Thorac Cardiovasc Surg*. 1991 Jan;101(1):44-55.
23. Lawrie GM, Pacifico A. Surgery for ventricular tachycardia. *J Cardiovasc Electrophysiol*. 1991;2:65-76.
24. Lawrie GM, Pacifico A. Combined resection and cryoablation for ischemic ventricular tachycardia. Cox JL (Ed.), *Cardiac arrhythmia surgery. State of the art reviews*, Hanley & Belfus, Philadelphia (1990), pp. 255-266.
25. Krafchek J, Lawrie GM, Wyndham CR. Cryoablation of arrhythmias from the interventricular septum: initial experience with a new biventricular approach. *J Thorac Cardiovasc Surg*. 1986 Mar;91(3):419-27.
26. Lawrie GM, Pacifico A, Kaushik RR. Transannular cryoablation of ventricular tachycardia. Surgical technique and results. *J Thorac Cardiovasc Surg*. 1989 Dec;98(6):1030-5; discussion 1035-6.
27. Mirowski M, Reid PR, Mower MM, et al. Termination of malignant ventricular arrhythmias with an implanted automatic defibrillator in human beings. *N Engl J Med*. 1980 Aug 7;303(6):322-4. doi:10.1056/NEJM198008073030607.
28. Mirowski M, Mower MM, Reid PR. Management of sustained ventricular tachycardia. *J Am Coll Cardiol*. 1985 Jul;6(1):213-4.
29. Lawrie GM, Griffin JC, Wyndham CR. Epicardial implantation of the automatic implantable defibrillator by left subcostal thoracotomy. *Pacing Clin Electrophysiol*. 1984 Nov;7(6 Pt 2):1370-4. doi:10.1111/j.1540-8159.1984.tb05710.x.
30. Lawrie GM, Kaushik RR, Pacifico A. Right mini-thoracotomy: an adjunct to left subcostal automatic implantable cardioverter defibrillator implantation. *Ann Thorac Surg*. 1989 May;47(5):780-1. doi:10.1016/0003-4975(89)90147-1.
31. O'Neill PG, Boahene KA, Lawrie GM, Harvill L, Pacifico A. The automatic implantable cardioverter-defibrillator: effect of patch polarity on defibrillation threshold. *J Am Coll Cardiol*. 1991 Mar 1;17(3):707-11. doi:10.1016/s0735-1097(10)80187-1.
32. Onufer JR, Cain ME. Impact of mapping and ablation of ventricular tachycardia on management strategies for the 1990s. *J Cardiovasc Electrophysiol*. 1991;2:77-91.
33. Lawrie GM, Wyndham CR, Krafchek J, Luck JC, Roberts R, DeBakey ME. Progress in the surgical treatment of cardiac arrhythmias. Initial experience of 90 patients. *JAMA*. 1985 Sep 20;254(11):1464-8.
34. Lawrie GM, Pacifico A. The role of surgery in the treatment of tachyarrhythmias. *Int J Cardiol*. 1989 Nov;25(2):151-8. doi:10.1016/0167-5273(89)90101-0.
35. Bhardwaj B, Kumar V, Patel N, et al. Implications of left ventricular aneurysm in patients with acute myocardial infarction: an analysis from national inpatient sample database. *J Am Coll Cardiol*. 2018 Mar;71(11):146(A).
36. Ruzza A, Czer LSC, Arabia F, et al. Left Ventricular Reconstruction for Postinfarction Left Ventricular Aneurysm: Review of Surgical Techniques. *Tex Heart Inst J*. 2017 Oct 1;44(5):326-35. doi:10.14503/THIJ-16-6068.