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Value of Early Postoperative Epicardial Programmed Ventricular Stimulation Studies After Surgery for Ventricular Tachyarrhythmias

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The value of early postoperative epicardial programmed ventricular stimulation studies after electrophysiologically-directed surgery for ventricular tachyarrhythmia was assessed in 34 patients who underwent epicardial stimulation within 7 to 30 days (mean 9.8) of surgery and were followed up for at least 6 months. The antiarrhythmic operation performed was an endocardial ventriculotomy (full encircling or limited), an endocardial resection, a wall resection or a combination of these procedures. All these interventions were directed by intraoperative mapping during sinus rhythm. Temporary epicardial wire electrodes left at the time of surgery rather than endocardial catheter electrodes were used to perform the pacing. The stimulation protocol included the introduction of up to three ventricular extrastimuli and incremental burst ventricular pacing performed at twice diastolic threshold (9.2 ± 5.8 mA for the right ventricle and 6.0 ± 3.5 mA for the left ventricle). A study was considered positive when ventricular tachycardia, defined as 10 or more consecutive ventricular beats, was induced by any pacing modality.

Nineteen patients (Group I) had a negative study: after stimulation of both ventricles in 15 patients and of the left ventricle only in 4. Fifteen patients (Group II) had a positive study: after stimulation of the right ventricle in nine patients and of the left ventricle in six. The two groups were comparable with respect to preoperative clinical status, surgical procedures performed and postoperative ejection fraction. No arrhythmic events were observed in Group I during a mean follow-up period of 19.5 months (range 4 to 37), whereas seven arrhythmic events (47% incidence) occurred ($p = 0.0008$) in Group II during a mean follow-up period of 17.7 months (range 5 to 39). These arrhythmic events were sudden death (five patients) and sustained ventricular tachycardia (two patients).

It is concluded that temporary epicardially-placed electrodes can be used satisfactorily to perform programmed ventricular stimulation studies in the postoperative period, thereby avoiding the cardiac catheterizations otherwise necessary to perform these studies. In addition, the protocol used in this report of epicardial programmed ventricular stimulation early after surgery for ventricular tachyarrhythmia predicts a good outcome if the study is negative and identifies patients at a high risk for future arrhythmic events when positive.

In the past decade, programmed electrical stimulation of the heart has been used increasingly in the management of many arrhythmias, including ventricular tachycardia, where it has been of great aid in identifying a precise diagnosis and mechanism, in locating the tissue responsible for its generation and in assessing the efficacy of different therapeutic approaches (1–10). Recently, the use of intracardiac electrophysiologic investigation, including programmed ventricular stimulation, in assessing the results of surgical therapy for sustained ventricular tachyarrhythmia has also been emphasized (11–17). Cardiac catheterization, with all its attendant costs, inconvenience and morbidity (acceptably low), has been the only approach used thus far to perform programmed ventricular stimulation studies 10 to 30 days after the surgical procedure.

This paper reports our experience with the use of programmed ventricular stimulation employing temporary, Teflon-coated, stainless steel wire electrodes left on the ventricular epicardium at the time of surgery to assess the inducibility of ventricular tachycardia, predict the risk of future ventricular tachyarrhythmic events and establish the

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need for further antiarrhythmic therapy in patients undergoing surgical therapy for sustained, life-threatening ventricular tachyarrhythmias.

Methods

Study patients. In this study, we report on 34 of 51 consecutive patients who underwent electrophysiologically directed surgery at the University of Alabama in Birmingham between August 1978 and October 1982 for treatment of sustained, life-threatening ventricular arrhythmias in the setting of coronary artery disease. To be included in this study, patients had to have had postoperative programmed ventricular stimulation studies using temporarily placed epicardial wire electrodes and a minimum period of 6 months of follow-up. The latter criterion was based on previously reported observations in studies (12–15,17) using serial electrophysiologic testing of ventricular tachyarrhythmias indicating that most spontaneous arrhythmic events appear within the first 6 months to 1 year from the last positive electrophysiologic stimulation study.

Seventeen of the 51 patients were excluded from this analysis. Eight died during the initial hospitalization, three were tested using conventional electrode catheter methods during cardiac catheterization, five early in our series did not undergo postoperative electrophysiologic studies and one was discharged from the hospital receiving amiodarone therapy and had no postoperative electrophysiologic studies because of the questionable significance of ventricular stimulation studies in patients receiving this drug (18,19). Table 1 provides the relevant clinical data for the 34 patients included in this study.

Epicardial wire electrodes. In patients undergoing open heart surgery at the University of Alabama in Birmingham, it is customary to place a pair of temporary, Teflon-coated, epicardial, stainless steel wire electrodes on the atria and one or two electrodes on the right ventricle at the time of surgery before closing the chest (20,21). These wire electrodes (Davis and Geck 957-630-0 Flexon) are then brought out through the anterior chest wall for potential diagnostic and therapeutic use in the immediate postoperative period (20,21). In patients undergoing surgical treatment of life-threatening ventricular arrhythmias, this technique was modified to include placement of a pair of electrodes on both ventricles as well as on the right atrium. These wire electrodes were electrically isolated when not in use, as previously described (20,21).

Electrophysiologic study. Electrophysiologic study was performed with the patient in the nonsedated, postabsorptive state, either at the bedside or in the electrophysiology laboratory within 7 to 30 days (mean 9.8) after surgery. Informed consent was always obtained. Using a DR12 Electronics for Medicine switched beam oscilloscopic recorder, electrocardiographic leads I, II, III and V_1 were simultaneously recorded with bipolar electrograms from the right atrium and both ventricles. All data were recorded on photographic paper and simultaneously recorded on magnetic FM tape using a Honeywell 5600C tape recorder for later playback and analysis. A constant current programmable stimulator (Medtronic model 1349A) was used in all instances to deliver rectangular impluses of 2 ms duration at twice the diastolic threshold.

Stimulation protocol. The following was our standard protocol for the electrophysiologic studies. In a progressive fashion, up to three ventricular extrastimuli were introduced to scan diastole after delivering a basic drive train of eight paced beats (S_1) from each ventricle. At least two different basic drive cycle lengths (600 and 500 ms or 545 and 462 ms) were used for each pacing site. The ventricular extrastimuli were introduced in the following manner: When the effective refractory period of the first ventricular extrastimulus (S_2) was determined, the S_1S_2 interval was fixed at 25 ms longer than the effective refractory period of the S_2. Then, a second ventricular extrastimulus S_3 was introduced to scan diastole. When the effective refractory period of the S_3 was determined, the S_2S_3 interval was fixed at 25 ms longer than the effective refractory period of the S_3, the S_1S_2 interval remained at its previous fixed interval and the third premature ventricular beat (S_4) was introduced to scan diastole until its effective refractory period was reached. Also, bursts of 8 to 10 beats were introduced at each pacing site at incrementally faster rates (beginning at 150 beats/min and increasing the rate by increments of 10 beats/min) until 2:1 capture of the ventricles was produced or a ventricular tachyarrhythmia was induced. Burst pacing was always performed after introduction of two ventricular extrastimuli and before introduction of three ventricular extrastimuli.

A pacing study was considered positive when 10 or more consecutive ventricular beats were elicited. If ventricular tachycardia was precipitated and was not self-terminating, it was interrupted either by ventricular pacing or, if necessary, by direct current cardioversion. If ventricular tachycardia was associated with marked hypotension, direct current cardioversion was performed promptly. If ventricular fibrillation was precipitated, defibrillation was performed immediately. Patients with a positive study underwent serial electrophysiologic testing (1,2,4–8,10) using the previously described pacing protocol to assess the efficacy of different antiarrhythmic drugs in suppressing the inducibility of the ventricular tachyarrhythmia.

Follow-up and statistical analysis. Clinical follow-up was obtained in all patients by contacting the patient and the patient's physician. For the purposes of this study, either nonmonitored sudden death or electrocardiographically documented ventricular tachycardia lasting 10 or more beats was considered an arrhythmic event. Using the 2×2 chi-square test, the number of arrhythmic events in the follow-up period in patients with inducible ventricular tachyar-

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Table 1. Clinical Data for 34 Patients Included in Postoperative Programmed Ventricular Stimulation Study

Case		Age (yr) & Sex	CAD (no. of vessels)	Type of Surgery		Ejection Fraction		Antiarrhythmic Drug at Time of 1st Study	Follow-up (mo)
				CABG	Arrhythmia	(pre-op)	(post-op)		
Group I Patients									
1		47M	2	3	ER, LEEV	0.16	0.20	None	37, alive
2		64M	2	4	ER, LEEV	0.53	0.47	None	34, alive
3		48M	1	3	ER, LEEV, WR	0.43	0.50	None	33, alive
4		48M	1	1	WR	0.21	.	None	12, died AMI
5		65M	3	5	WR, ER, LEEV	0.37	. .	Quin for SVT	4, died CHF
6		66M	2	3	ER, LEEV, WR	0.18	0.22	None	29, alive
7		65M	3	2	WR, LEEV	. . .	0.34	None	26, alive
8		66M	2	1*	WR, ER	0.34	0 30	Quin for SVT	26, alive
9		66F	3	5	ER	. . .	.	None	23, alive
10		52M	1	0	ER, LEEV, WR	0.34	0.32	None	20, alive
11		36M	2	1*	LEEV	0.49	0.54	None	18, alive
12		71M	3	5	LEEV	0.23	0.34	None	17, alive
13		58M	2	3	WR, FEEV	0.44	0.39	None	16, alive
14		36M	3	2	ER	0.40	0.43	None	14, alive
15		51M	2	3	WR, LEEV	0 37	. .	None	13, alive
16		60M	2	3	WR	0.16	0.25	None	13, alive
17		52M	3, left main	6	WR, LEEV	0 18	0 20	None	10, alive
18		54M	1	3	WR, LEEV	0.25	0.26	None	8, alive
19		57M	1	1	WR, LEEV	0.15	0.36	None	8, alive
Group II Patients†									
20	IIa	45M	1	0	LEEV, WR	0.19	0.27	None	6, died CHF
21	IIa	60M	3	4	ER, LEEV	0.57	0.53	None	28, alive
22	IIa	62M	2	3	WR, LEEV	0.21	0.21	None	6, sudden death
23	IIa	66M	1	1	ER, LEEV	0.27	0 27	Quin for SVT	13, sudden death
24	IIa	44M	1	1	ER, LEEV, WR	0.29	0.16	None	29, alive, VT
25	IIa	76F	3	5	LEEV	0.20	0.26	None	8, sudden death
26	IIb	57M	2	3	WR, ER	0.20	. .	None	12, sudden death
27	IIb	52M	2	4	ER	0 40	. .	None	39, alive
28	IIb	64M	3	5	ER, LEEV, WR	0.31	0.30	None	34, alive
29	IIb	64M	3, left main	5	WR, ER, LEEV	0 26	0.32	None	20, alive
30	IIb	56M	3	4	ER	0.31	0.47	None	32, alive
31	IIb	51M	2	0	ER, LEEV, WR	0 21	0.35	None	5, sudden death
32	IIb	59M	3	4	LEEV	0 40	0.45	None	13, alive
33	IIb	70M	2	2	WR	0.28	0 44	None	11, alive
34	IIb	67M	1	2	WR	0.35	0.30	None	10, alive

*History of prior coronary artery bypass grafting. †Group IIa = patients 20 to 25; Group IIb = patients 26 to 34

AMI = acute myocardial infarction; CABG = coronary artery bypass grafting; CAD = coronary artery disease; CHF = congestive heart failure; ER = endocardial resection; FEEV = full encircling endocardial ventriculotomy; LEEV = limited encircling endocardial ventriculotomy; post-op = postoperative; pre-op = preoperative; Quin = quinidine; SVT = supraventricular tachycardia, VT = ventricular tachycardia; WR = wall resection.

rhythmia at the time of the first electrophysiologic study after surgery was compared with that in patients in whom no ventricular tachyarrhythmia was inducible. In the same two groups of patients, a Student's *t* test was used to compare the age, preoperative ejection fraction, postoperative ejection fraction, change in pre- and postoperative ejection fractions, nature of coronary artery disease, type of coronary artery bypass surgery and type of antiarrhythmic surgery. The Student's *t* test was also used in patients with a positive postoperative electrophysiologic study to compare these same variables in patients who had subsequent spontaneous arrhythmic events with those who had no subsequent arrhythmic events.

Results

Induced ventricular arrhythmias. The results of the postoperative programmed epicardial ventricular stimulation studies for all 34 patients are summarized in Figure 1. The initial study was negative in 19 patients (Group I, Cases 1 to 19) and positive in 15 (Group II, Cases 20 to 34). The arrhythmias induced by the programmed epicardial ventric-

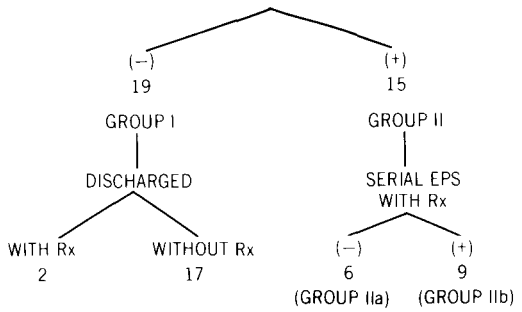

Figure 1. Results of the first postoperative electrophysiologic study (EPS) using programmed ventricular stimulation in 34 patients and subsequent management. Group IIa = patients discharged with inducibility of ventricular tachyarrhythmia suppressed by medication; Group IIb = patients discharged with inducibility of ventricular tachyarrhythmia *not* suppressed by medication. Rx = drug therapy.

ular stimulation in Group II included sustained ventricular tachycardia lasting more than 30 seconds in 10 patients, ventricular fibrillation in 1 patient and sustained but self-terminating ventricular tachycardia (spontaneous termination in less than 30 seconds) in 3 patients. Of the patients with sustained ventricular tachycardia, cardioversion was required to terminate the arrhythmia in five cases and rapid ventricular pacing was successful in terminating the arrhythmia in all other cases.

Right versus left ventricular stimulation. The mode of induction of the arrhythmia during the initial study is listed in Table 2. In Group I, 15 patients (79%) underwent stimulation from both ventricles and 4 from the left ventricle only. The latter occurred because of too high a right ventricular stimulus threshold (>30 mA) in two patients, failure to capture the right ventricle at all in one patient and the absence of epicardial electrodes on the right ventricle in one patient. In Group II, 10 of the 15 patients underwent stimulation from only one ventricle for the following reasons: a positive test from the first ventricular pacing site (7 patients), absence of epicardial electrodes on one ventricle (1 patient), excessive stimulus threshold (1 patient) and discomfort during left ventricular pacing due to phrenic nerve stimulation (1 patient). Left ventricular pacing was required to induce ventricular tachycardia in six patients, and in six patients, the introduction of three extrastimuli was required to elicit the arrhythmia.

Antiarrhythmic therapy (Fig. 1). Seventeen patients in Group I were discharged from the hospital while receiving no antiarrhythmic medication, and two patients were discharged while receiving the antiarrhythmic medication (quinidine) they were taking at the time of the study (for control of spontaneous postoperative atrial fibrillation). After serial drug testing of patients in Group II, the study became negative in six patients who were then discharged taking the appropriate antiarrhythmic medication (Group IIa). Nine continued to have a positive test despite antiarrhythmic therapy (Group IIb). Seven were discharged from the hospital while taking an antiarrhythmic medication that had failed to suppress the inducibility of ventricular tachycardia, one patient was discharged while receiving no antiarrhythmic medication and one patient was discharged while taking amiodarone.

Follow-up. Table 3 summarizes the long-term follow-up results in both groups of patients. In a mean follow-up period of 19.5 months (range 4 to 37), no arrhythmia events were observed in any of the patients in Group I. Although two deaths occurred, both were related to progressive low cardiac output 4 and 15 months after surgery, respectively. In contrast, in a mean follow-up period of 17.7 months (range 5 to 39), there were seven arrhythmia events (47% incidence) in patients in Group II. Two of the events were documented ventricular tachycardia and five were sudden death. The difference in the incidence of arrhythmia events between Groups I and II was highly significant statistically (probability [p] = 0.0008). Of the seven patients with arrhythmic events during the follow-up period, four had been discharged after the electrophysiologic study became negative while they were receiving antiarrhythmic therapy (Group IIa) and three had been discharged with the last electrophysiologic study having remained positive (Group IIb).

Table 2. Mode of Ventricular Tachycardia-Ventricular Fibrillation Induction

Pacing Site	1 ES	2 ES	Burst	3 ES	Total*
RV	0	6	0	3	9 (26%)
LV	0	3	0	3	6 (18%)
Total*	0	9 (26%)	0	6 (18%)	

*Number of patients (% of 34 patients).

Burst = incremental burst (10 beats) of ventricular pacing, ES = ventricular extrastimulus; LV = left ventricle; RV = right ventricle

Table 3. Long-Term Follow-Up

Group	Patients (no.)	Months (mean; range)	Patients With Arrhythmia Events (no.) SD	VT	SD + VT	%
I	19	19.5; 4 to 37	0	0	0*	0
II	15	17.7; 5 to 39	5	2	7*	47
IIa	6		3	1	4	
IIb	9		2	1	3	
All	34	16.9; 4 to 39	5	2	7	21

*Group I vs. Group II (chi-square), $p = 0.0008$.

Group I = patients with negative study; Group II = patients with positive study; Group IIa = ventricular response rendered negative by antiarrhythmic drug therapy before discharge from hospital; Group IIb = arrhythmia inducibility not suppressed by drugs; SD = sudden death; VT = sustained ventricular tachycardia.

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Group IIa. Further analysis of patients in Group IIa is of interest. One patient who died suddenly 6 months after surgery had a negative study while receiving phenytoin therapy. A serum phenytoin level obtained by the patient's physician 3 days before death was 6 mg/dl, whereas the level at the time of the negative study was 22.7 mg/dl. A second patient who died suddenly 8 months after surgery also had a negative study while receiving phenytoin. We learned subsequently that quinidine therapy, which had been administered postoperatively but had failed to suppress inducibility of ventricular tachycardia, had been added to this patient's regimen by her physician. Technically, this may have changed the patient's classification to Group IIb.

In two other patients in Group IIa, the protocol for the subsequent postoperative epicardial ventricular stimulation studies was incomplete by our present standards (10,22). In one patient, after the initial postoperative ventricular stimulation study had been positive from the right ventricular pacing site, the patient was retested on drug therapy only from the same right ventricular site. This study was negative. We were unable to perform left ventricular stimulation because pacing through the left ventricular epicardial wire electrodes produced phrenic nerve stimulation. This patient had spontaneous recurrence of ventricular tachycardia 3 months after hospital discharge. It should be noted that the vast majority of electrophysiology laboratories would have considered this study complete, because a site that was previously positive when paced had become negative (10). A subsequent repeat electrophysiologic study using endocardial pacing techniques demonstrated that while the patient was receiving drug therapy thought to have suppressed the inducibility of the ventricular tachycardia, the ventricular tachycardia was, in fact, only inducible with left ventricular stimulation. In the other patient in Group IIa, ventricular extrastimuli were introduced only after one basic drive cycle. This patient died suddenly at home 13 months after surgery.

Group IIb. Two sudden deaths occurred in this group 5 and 12 months after the last postoperative epicardial ventricular stimulation. In the other patient Group IIa, ventricminating (< 30 seconds) ventricular tachycardia while the patient was receiving quinidine therapy. It is important to note that at the time these patients were studied (December 1980), patients with pacing-induced self-terminating ventricular tachycardia lasting less than 30 seconds were not considered treatment failures. In fact, some investigators (17) still consider such a postoperative study negative. Furthermore, it was partly on the basis of these ventricular stimulation studies and long-term outcomes that we evolved our present definition of a positive electrophysiologic stimulation study.

Analysis of other data. Our primary aim has been to examine the technique of postoperative epicardial programmed ventricular stimulation studies rather than to examine the various factors that contributed to the results of the first postoperative electrophysiologic study. Nevertheless, it is of interest that there were no statistically significant differences between those patients with a positive or negative first postoperative electrophysiologic study in relation to the following factors: age, number of coronary arteries with 50% or greater occlusion, number of coronary arteries bypassed, type of antiarrhythmic surgery, preoperative ejection fraction, postoperative ejection fraction or change in pre- and postoperative ejection fraction.

However, when the postoperative ejection fraction is considered, two comparisons do demonstrate statistical significance. First, for the 28 patients whose postoperative ejection fraction was measured, the difference between the value in those who had no spontaneous arrhythmic event (22 patients, mean ejection fraction 0.37 ± 11) and those who did have such an event (6 patients, mean ejection fraction 0.26 ± 0.07) is statistically significant ($p < 0.05$). Second, when comparing the ejection fraction of those patients in Group II who had no subsequent spontaneous arrhythmic event (seven patients, mean ejection fraction 0.40 ± 0.10) with those patients in Group II who did have a subsequent spontaneous arrhythmic event (six patients, mean ejection fraction 0.26 ± 0.07), the mean ejection fraction is notably different ($p < 0.015$). Thus, patients with a positive first postoperative electrophysiologic study and a low ejection fraction seem to be at a particularly increased risk for subsequent arrhythmic events.

Removal of the temporary epicardial electrodes. In all patients, the epicardial electrodes were left in place until the day before discharge, when they were removed from the heart with a gentle tug and then pulled out through the anterior chest wall (20,21). No morbidity or mortality was associated with this maneuver.

Discussion

In this highly selected group of patients with coronary artery disease who had undergone surgical treatment for recurrent life-threatening ventricular tachyarrhythmias, the first postoperative ventricular stimulation study using epicardial ventricular wire electrodes temporarily placed at the time of surgery proved to be a good predictor of future ventricular arrhythmic events. The absence of arrhythmic events in patients whose first ventricular stimulation study was negative contrasts quite sharply with a 47% incidence of arrhythmic events in the patients whose first electrophysiologic study was positive. Thus, a positive study identifies a group of patients at a high risk for future arrhythmic events, particularly if the postoperative ejection fraction is low.

Importance of programmed ventricular stimulation protocol and interpretation of its results. In considering our findings, the type of ventricular stimulation protocol and

the interpretation of the results of the stimulation using that protocol have to be taken into consideration, particularly because there is a wide variation among institutions (1–6,8,10,17,22,23) in the definition of ventricular tachycardia, as well as in the stimulation protocols used to assess the interventions that control the arrhythmia. First, previously reported protocols have only used endocardial ventricular stimulation with catheter electrode techniques. Most of these protocols include pacing from two right ventricular sites. Not all protocols include pacing from the left ventricle, although recently, the need for programmed stimulation studies from the left ventricle as part of an electrophysiologic evaluation of ventricular tachycardia has been addressed (1,5,8,10,22,24–25). In addition, there remains a controversy regarding whether or not three ventricular premature beats should be included as part of the stimulation protocol, because most reported studies used catheter electrode techniques introducing only two premature ventricular beats. Then, there is the question of what constitutes clinically significant induced ventricular tachycardia. In patients after open heart surgical treatment of life-threatening ventricular arrhythmias, a standard definition has been only induced ventricular tachycardia lasting more than 30 seconds or associated with significant hypotension (17). Finally, most laboratories consider that if ventricular tachycardia is induced when pacing from a ventricular site, but is no longer induced when pacing from that same site after drug therapy, the electrophysiologic test has become negative.

The data from our study using temporary ventricular epicardial wire electrodes suggest that stimulation of both ventricles as well as the introduction of three extrastimuli are quite important in identifying all patients with inducible ventricular tachyarrhythmias (Table 2). Indeed, six patients (18% of our series) had a positive study with left ventricular stimulation. This number could be seven patients (21% of our series) because the study of one patient, originally positive with right ventricular stimulation became negative with right ventricular stimulation after drug therapy, but later was found to be positive with left ventricular stimulation. Six patients (18% of our series) required the introduction of three extrastimuli to induce sustained ventricular arrhythmias (three patients during stimulation of the right ventricle and three during stimulation of the left ventricle). This is consistent with the recent observations of Morady et al. (10) in patients who had not undergone open heart surgery and were studied using electrode catheter techniques.

Our observations also suggest that induced ventricular tachycardia lasting more than 10 beats but less than 30 seconds has important prognostic value. After learning about the sudden death of our two patients in Group IIb who were discharged from the hospital with inducible, sustained but self-terminating (< 30 seconds) ventricular tachycardia while receiving quinidine therapy, we modified our approach to strive for the suppression of any induced ventricular tachycardia of 10 or more beats. Finally, the occurrence of an arrhythmic event in one patient even though ventricular tachycardia was no longer inducible from the same pacing site after drug therapy supports the notion that pacing from at least a second ventricular site is necessary to demonstrate that an electrophysiologic test has truly become negative (10, 22).

Advantages and limitations of programmed epicardial ventricular stimulation studies. Our results clearly demonstrate that the use of temporarily placed Teflon-coated, stainless steel wire electrodes for postoperative programmed ventricular stimulation studies is safe and effective. It provides ready access to the ventricles for purposes of pacing and recording, thereby saving the time, expense and potential morbidity associated with catheter electrode techniques that would otherwise be necessary to perform the stimulation studies. The placement of the wire electrodes at the time of surgery adds minimally to the total duration of the operation, but this has been of no consequence.

The chief limitation of the use of the epicardial electrodes has been the relatively high stimulus strength required to capture the ventricles. In fact, in five patients, this led to an inability to capture the ventricles and, in one patient, to stimulation of the phrenic nerve. Epicardial placement of the electrodes in regions free of fat and use of lower impedance electrodes should assist in preventing this sort of problem. On the basis of the data in this and other studies (10,22) in the event of a failure to achieve pacing of either ventricular chamber using temporarily placed epicardial wire electrodes, we recommend performing pacing of that chamber or at least a second ventricular site with standard endocardial pacing techniques.

Another limitation is that, as currently conceived, our stimulation studies are only performed from one site in each ventricle. However, there is nothing to prevent additional pairs of electrodes from being placed on each ventricle or from performing unipolar ventricular pacing from two sites on each ventricle, the latter saving placement of extra electrodes.

Finally, we know of no study comparing the results of epicardial and endocardial programmed ventricular stimulation studies. However, there is no a priori reason to suspect that one technique is intrinsically better than the other. Similarly, one should consider whether the relatively high stimulus strengths used in this study adversely or inappropriately affect the results, but analysis of our study suggests that they do not.

Conclusions. This study demonstrates that pairs of epicardial, stainless steel wire electrodes temporarily placed in the right and left ventricles can be utilized safely to perform programmed ventricular stimulation studies postoperatively to assess the efficacy of surgery designed to treat life-threatening ventricular arrhythmias. Use of the epicardial ventricular electrodes should save the patient the inconvenience,

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expense, potential morbidity and even occasional mortality associated with catheterization procedures, as such. In addition, this study suggests a need for performing an aggressive electrophysiologic study, including stimulation of both ventricles, introduction of up to three ventricular extrastimuli and pacing at at least two basic drive cycle lengths at each pacing site. When this is performed after surgery for ventricular tachyarrhythmias in the setting of ischemic heart disease, epicardial ventricular stimulation studies offer a powerful tool for identifying a patient group at high risk for future arrhythmic events.

References

1. Fisher JD. Role of electrophysiological testing in the diagnosis and treatment of patients with known and suspected bradycardias and tachycardias. Prog Cardiovasc Dis 1981;24:25–90.
2 Wellens HJJ. Value and limitations of programmed electrical stimulation of the heart in the study and treatment of tachycardias. Circulation 1978;57:845–53.
3. Horowitz LN, Josephson ME, Farshidi A, Spielman SR, Michelson EL, Greenspan AM. Recurrent sustained ventricular tachycardia. 3. Role of the electrophysiologic study in selection of antiarrhythmic regimens Circulation 1978;58:986–97.
4 Mason JW, Winkle RA. Electrode-catheter arrhythmia induction in the selection and assessment of antiarrhythmic drug therapy for recurrent ventricular tachycardia. Circulation 1978;58.971–85.
5. Josephson ME, Seides SF. Clinical Cardiac Electrophysiology. Techniques and Interpretation. Philadelphia: Lea & Febiger, 1979:247–80
6. Mason JW, Winkle RA. Ability of induced ventricular tachycardia to predict long-term efficacy of antiarrhythmic drugs. N Engl J Med 1980;303:1073–7.
7. Wu D, Wyndham CR, Denes P, et al. Chronic electrophysiological studies in patients with recurrent paroxysmal tachycardia: a new method for developing successful oral antiarrhythmic therapy. In: Kulbertus HE, ed. Reentrant Arrhythmias. Baltimore: University Park Press, 1976:294–311
8. Horowitz LN, Josephson ME, Kastor JA. Intracardiac electrophysiologic studies as a method for the optimization of drug therapy in chronic ventricular arrhythmia. Prog Cardiovasc Dis 1980;23.81–98
9. Josephson ME, Horowitz LN, Spielman SR, Waxman HL, Greenspan AM Role of catheter mapping in the preoperative evaluation of ventricular tachycardia. Am J Cardiol 1982;49·207–20.
10. Morady F, Hess D, Scheinman MM. Electrophysiologic drug testing in patients with malignant ventricular arrhythmia: importance of stimulation of more than one ventricular site. Am J Cardiol 1982;50:1055–60.
11. Josephson ME, Harken AH, Horowitz LN. A new surgical technique for the treatment of recurrent ventricular tachycardia. Circulation 1979;60:1430–9.
12. Horowitz LN, Josephson ME, Kastor JA, Harken AH. Ventricular resection guided by epicardial and endocardial mapping for the treatment of recurrent ventricular tachycardia. N Engl J Med 1980;302:589–93.
13. Martin JL, Untereker WJ, Harken AH, Horowitz LN, Josephson ME. Aneurysmectomy and endocardial resection for ventricular tachycardia: favorable hemodynamic and antiarrhythmic results in patients with global left ventricular dysfunction. Am Heart J 1982;103:960–5.
14. Mason JW, Stinson EB, Winkle RA, et al. Surgery for ventricular tachycardia: efficacy of left ventricular aneurysm resection compared with operation guided by electrical activation mapping. Circulation 1982;65:1148–55.
15. Moran JM, Kehoe RF, Loeb JM, Lichtenthal PR, Sanders JH, Michaelis LL. Extended endocardial resection for the treatment of ventricular tachycardia and ventricular fibrillation. Ann Thorac Surg 1982;34:538–52
16. Kehoe RF, Moran JM, Loeb JM, Eheutlin T, Lichtenthal PR, Sanders JH. Extended endocardial resection for treatment of survivors of out-of-hospital ventricular fibrillation (abstr). Circulation 1982;66(suppl II):II–34
17. Josephson ME, Horowitz LN, Harken AH. Long-term results of endocardial resection for sustained ventricular tachycardia. Am Heart J 1982;104:51–7
18. Wellens HJJ, Bar FWHM, Vanagt EJDM, Brugada P. Medical treatment of ventricular tachycardia: considerations in the selection of patients for surgical treatment Am J Cardiol 1982;49:186–93.
19. Saksena S, Rothbart S, Kaufmann WE, Rathyen W. Chronic electrophysiologic, systemic and pharmacologic effect of amiodarone in recurrent ventricular tachycardia (abstr). Am J Cardiol 1982;49:1044.
20. Waldo AL, MacLean WAH, Cooper TB, Kouchoukos NT, Karp RB. Use of temporarily placed epicardial atrial wire electrodes for the diagnosis and treatment of cardiac arrhythmias following open-heart surgery. J Thorac Cardiovasc Surg 1978,76:500–5.
21. Waldo AL, MacLean WAH. Diagnosis and Treatment of Cardiac Arrhythmias Following Open Heart Surgery. Emphasis on the Use of Epicardial Wire Electrodes. New York: Futura, 1980:1–200.
22 Henthorn RW, Plumb VJ, Arciniegas JG, Zimmern SH, Waldo AL. Significance of the mode and site of ventricular tachycardia inducibility to assure adequate electrophysiologic study (abstr). J Am Coll Cardiol 1983;1·594.
23 Akhtar M. The clinical significance of the repetitive ventricular response (editorial) Circulation 1981;63:773–5.
24. Michelson EC, Spielman SR, Greenspan AM, Farshidi A, Horowitz LN, Josephson ME Electrophysiologic study of the left ventricle. Indications and safety. Chest 1979;75:592–60
25 Robertson JF, Cain ME, Horowitz LN, et al Anatomic and electrophysiologic correlates of ventricular tachycardia requiring left ventricular stimulation. Am J Cardiol 1981;48:263–8