ABSTRACT  We studied 37 patients undergoing endocardial resection for medically refractory ventricular tachycardia (VT). Each was studied before and after surgery by programmed ventricular stimulation and signal-averaged electrocardiography. Low-amplitude late potentials were identified preoperatively in 76% of patients. In the 24 patients without postoperative VT the effect of surgery was to shorten the filtered QRS duration (137 ± 27 to 121 ± 26 msec; p = .003), increase the voltage in the last 40 msec of the filtered QRS (16.5 ± 16.1 to 39.0 ± 29.4 μV; p = .003), and decrease the incidence of late potentials (71% to 33%; p = .03). The filtered QRS complex was unchanged in 13 patients whose VT persisted after surgery. No preoperative variable predicted which patients would not have inducible VT after surgery. However, loss of a late potential after surgery in nine of 10 patients was associated with absence of inducible VT (p < .02). Loss of a late potential was not necessary for surgical success. Eight of 18 patients with a persistent late potential did not have inducible VT. The signal-averaged electrocardiogram predicted a successful outcome after endocardial resection if the late potential was no longer present.


THERE IS substantial evidence that ventricular tachycardia (VT), particularly when associated with coronary artery disease, involves reentrant mechanisms.1–4 Rentry requires unidirectional block and conduction delay sufficient to give the previously refractory tissue time to recover excitability. Several workers have directly recorded delayed ventricular activation in humans and animals with VT, which is believed to be the substrate of the arrhythmia.4–8 Signal-averaging techniques have been used to detect low-level activity at the end of the QRS complex from the body surface in patients with VT.9–13 By endocardial and epicardial mapping techniques, it has been demonstrated that these signals, called "late potentials," correspond to areas of slow or delayed activation in the ventricular myocardium.9, 14, 15 Late potentials appear to be sensitive and specific for identifying patients with recurrent sustained VT.10, 12, 13

Endocardial resection performed in association with left ventricular aneurysmectomy and guided by activation mapping during VT has been successful in controlling medically refractory VT associated with coronary artery disease.16, 17 The methods for evaluating the effectiveness of endocardial resection include Holter monitoring and invasive catheter electrophysiologic testing. This study was undertaken to analyze changes in body surface late potentials after surgery that is known to successfully control VT and to ascertain whether this noninvasive marker could predict postoperative control of VT by endocardial resection.

Methods

**Patients.** We studied 37 consecutive patients (26 men, 11 women) with inducible VT who underwent endocardial resection for control of their arrhythmias. Each patient underwent a left ventricular aneurysmectomy and endocardial resection for control of recurrent sustained VT refractory to conventional and experimental antiarrhythmic agents. These patients had previously been tested on 2.4 ± 1 (mean ± SD) antiarrhythmic agents. Patients with bundle branch block before or after operation were not included because of the inability of signal averaging to detect late potentials in the presence of bundle branch block.15

Thirty-six of the 37 patients had had a previous myocardial infarction (25 anterior, 11 inferior) and one had had dilated cardiomyopathy with a left ventricular aneurysm (table 1). Thir-
ty-two of 36 patients with myocardial infarction had left ventricular aneurysms documented by cardiac catheterization.

Electrophysiologic testing. All patients underwent electrophysiologic studies before and after surgery according to previously described techniques,\textsuperscript{3,17} including introduction of one

### TABLE 1

<table>
<thead>
<tr>
<th>Location of MI</th>
<th>Preop.</th>
<th>Postop.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>QRS duration (msec)</td>
<td>Voltage last (μV)</td>
</tr>
<tr>
<td>A</td>
<td>127</td>
<td>1.9</td>
</tr>
<tr>
<td>I</td>
<td>183</td>
<td>3.0</td>
</tr>
<tr>
<td>I</td>
<td>166</td>
<td>14.5</td>
</tr>
<tr>
<td>I</td>
<td>124</td>
<td>5.9</td>
</tr>
<tr>
<td>I</td>
<td>133</td>
<td>4.0</td>
</tr>
<tr>
<td>A</td>
<td>150</td>
<td>1.7</td>
</tr>
<tr>
<td>A</td>
<td>145</td>
<td>20.2</td>
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<tr>
<td>A</td>
<td>121</td>
<td>21.5</td>
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<tr>
<td>A</td>
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<tr>
<td>A</td>
<td>148</td>
<td>28.9</td>
</tr>
</tbody>
</table>

### Results

Preoperatively, 30 patients (81%) had a filtered QRS duration $>120$ msec with a mean duration of $141 \pm 26$ msec (figure 2). Late potentials were present
before surgery in 28 patients (76%), with a mean root mean square voltage for all patients in the last 40 msec of the filtered QRS of 15.9 ± 15.8 μV (figure 2).

Control of VT with surgery was not related to site of origin of VT or location of endocardial resection. After surgery 24 patients (65%) were free of inducible VT. Seventeen of these had preoperative late potentials. Nine of the 17 patients lost the late potential after operation (figure 3). However, persistence of a late potential after surgery did not preclude surgical success (figure 4). Overall, for patients without VT after operation the QRS duration decreased by 17 ± 25 msec (from 138 ± 27 to 121 ± 26 msec; p = .003; figure 5), voltage in the last 40 msec increased by 22.6 ± 34.9 μV (from 16.5 ± 16.1 to 39.0 ± 29.4 μV; p = .003; figure 5), and the incidence of late potentials decreased from 71% to 33% (p = .03). In the 13 patients whose VT persisted after surgery the filtered QRS complex was generally unchanged (figure 6).

After surgery the patients who continued to have inducible VT had a longer QRS duration (146 ± 33 vs 121 ± 26 msec; p < .005), a lower terminal QRS voltage (16.4 ± 18.8 vs 39.0 ± 29.4 μV; p < .01), and a higher incidence of late potentials (85% vs 33%; p < .01) than those patients without inducible tachycardia.

No feature of the filtered QRS complex before operation predicted the absence of inducible VT after operation. Noninducibility was predicted by loss of a late potential or an increase in the late QRS voltage after operation. The VT was eliminated in nine of 10 patients who lost their late potentials after operation (sensitivity 0.53, specificity 0.91; p < .02). Similarly, 17 of 20 patients whose terminal QRS voltage increased by 5 μV or greater had no inducible VT after operation (sensitivity 0.71, specificity 0.77; p < .01). Ten of 11
patients with a large voltage increase (20 μV or greater) had no inducible VT after operation (sensitivity 0.42, specificity 0.92; p < .05). Neither an increase nor a decrease in QRS duration after endocardial resection predicted surgical outcome. Change in late QRS voltage or loss of the late potential was independent of site of origin of VT or location of endocardial resection.

Persistence of a late potential after surgery did not preclude surgical success. Eight of the 18 patients whose late potential persisted after surgery had no inducible VT.

Data from the patients tested before surgery while on antiarrhythmic drugs were analyzed separately to determine whether the presence of antiarrhythmic agents influenced the results. In these patients the QRS duration decreased by 22 ± 27 msec (from 145 ± 28 to 122 ± 29 msec; p = .007). For the 26 patients tested before surgery while on no drugs, the mean QRS duration decreased by 5 ± 16 msec (from 139 ± 25 to 134 ± 32 msec; p = .16; figure 7). Except for the change in QRS duration after surgery, there was no significant difference between the patients on and off drugs in the voltage in the last 40 msec (figure 8), voltage change after operation, success in controlling VT, incidence of late potentials, or ability of various parameters to predict surgical outcome.

Discussion

Fragmented and markedly delayed electrograms have been recorded from infarcted myocardium.\textsuperscript{3, 4, 5} Studies in animals\textsuperscript{6, 7} and humans\textsuperscript{1, 3, 4} suggest that these areas of fragmented and delayed electrograms participate in reentrant arrhythmias. Endocardial recording in animals and humans demonstrates that the onset and maintenance of VT are dependent on delayed ventricular activation.\textsuperscript{1, 3, 6} Fragmented activity may span diastole with ventricular premature stimulation.
and thus initiate sustained VT. Late potentials at the end of the QRS complex have been detected from the body surface by several workers.9-11 Endocardial and epicardial mapping studies suggest that these late potentials correspond to areas of slow activation.14, 15 The duration of directly recorded fragmented electrograms correlates with the filtered QRS duration.14, 15 There is evidence that late potentials are the body surface representation of the slowly conducting areas of myocardium that are the substrate for reentry. In a previous study12 late potentials were shown to be sensitive and specific for identifying patients with VT.

We have analyzed the effect of endocardial resection on filtered QRS duration and voltage in the last 40 msec of the filtered QRS. In general, endocardial resection results in a shortened QRS duration and an increased voltage in the terminal portion of the QRS. This effect is more pronounced in those patients free of inducible VT after operation. No preoperative parameter was identified to predict successful control of VT by endocardial resection.

Postoperative loss of the late potential or an increase in the terminal QRS voltage was associated with successful endocardial resection. The greater the voltage increase, the more specific the test for loss of VT. There is, however, a compromise in sensitivity. Persistence of late potentials after surgery does not necessarily predict surgical failure. Unfortunately, in those patients without preoperative late potentials no prediction could be made regarding postoperative control of VT. These results agree with those of Breithardt et al.,19 although the signal-processing techniques used were different. Isolated examples of loss of late potentials after surgery have been reported.10, 11

In contrast to the effects of surgery, in our experience antiarrhythmic drugs do not change the incidence of late potentials despite effective control of VT.19, 20 Procainamide, quinidine, and amiodarone increase the duration of the filtered QRS complex and procainamide decreases the late QRS voltage. No significant changes in the signal-averaged electrocardiogram have been observed with disopyramide, phenytoin, and mexiletine.19, 20 The shorter QRS duration after operation we noted in patients who received antiarrhythmic agents before operation may have been due in part to a withdrawal of drug effect.

The mechanism of VT control after endocardial resection appears to be surgical removal of at least part of the slowly conducting tissue involved in reentry. In those patients without a late potential after operation, it appears that areas of markedly delayed conduction have been removed and in 90% of cases, the VT is eliminated. In those patients whose late potentials persist but whose VT is controlled, sufficient substrate may have been removed to interrupt the reentrant circuit or produce exit block. However, some areas of slow conduction remain so that they are detected on the body surface. It is also possible that the area responsible for the late potential was unrelated to the tachycardia and was specifically not removed.

**FIGURE 7.** Analysis of filtered QRS duration before and after surgery in those patients tested before surgery while on (DRUGS) or off (NO DRUGS) antiarrhythmic drugs and in the group as a whole (ALL). The p values are for comparison between preoperative and postoperative values. Values shown are mean ± SD. NS = not significant.

**FIGURE 8.** Analysis of voltage in the last 40 msec of the filtered QRS complex in those patients tested before surgery while on or off antiarrhythmic agents and in the group as a whole. Same format as in figure 7.
Patients with recurrent sustained VT but without late potentials may have insufficient conduction delay to extend beyond the normal QRS. The area of delayed activation may occur within the QRS and may be masked.\textsuperscript{15} There is evidence that slow conduction in the ventricular myocardium is rate dependent.\textsuperscript{1, 2} Therefore recording during sinus rhythm may not be the most sensitive way to detect areas of slow conduction. The sensitivity of the test may be improved by performing signal averaging with atrial pacing at faster rates or after premature depolarizations.

The lower sensitivity (76\%) of late potentials for detecting patients with VT seen in this study compared with that in previous studies may be due to several factors. This study contains a higher proportion of patients with anterior infarction than previously reported.\textsuperscript{12} These patients have a higher mean voltage in the last 40 msec of the filtered QRS complex\textsuperscript{12} and anterior infarction may be associated with lower incidence of late potentials. Moreover, there was a selection bias in the study, since we examined patients with only medically refractory VT who require surgical therapy.

The results of this study have shown loss of a late potential to be an accurate predictor of surgical success in controlling VT. A prospective study is required to compare this noninvasive method with catheter electrophysiologic testing after surgery to predict clinical outcome. Further experience with this technique may obviate the need for electrophysiologic study after surgery in those patients who lose their late potentials.

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