The Response to Procardiamide During Electrophysiologic Study for Sustained Ventricular Tachyarrhythmias Predicts the Response to Other Medications

HARVEY L. WAXMAN, M.D., ALFRED E. BUXTON, M.D., LAURA M. SADOWSKI, B.A., AND MARK E. JOSEPHSON, M.D.

SUMMARY We evaluated 126 patients with inducible sustained ventricular tachyarrhythmias to assess whether the response to procainamide during electrophysiologic study could predict responses to other conventional antiarrhythmic agents and combinations of agents. Thirty of 42 patients in whom ventricular tachycardia was not inducible after the administration of procainamide and 69 of 84 patients in whom ventricular tachycardia was inducible after procainamide underwent serial electrophysiologic studies. Forty-three of 67 antiarrhythmic regimens (64%) tested in the patients in whom ventricular tachycardia could not be induced after procainamide prevented induction of ventricular tachycardia, compared with 10 of 145 regimens (7%) tested in the patients in whom ventricular tachycardia could be induced after procainamide. Sixty of the 69 patients in whom ventricular tachycardia remained inducible after procainamide had ventricular tachycardia induced on all other conventional antiarrhythmic regimens tested. By comparison, of the 30 patients in whom ventricular tachycardia became noninducible after procainamide, 25 had no ventricular tachycardia inducible on at least one other antiarrhythmic regimen tested. Thus, the response to procainamide accurately predicted the response to other conventional antiarrhythmic agents during electrophysiologic study.

PROGRAMMED ventricular stimulation can be used to guide antiarrhythmic drug therapy in patients with sustained ventricular tachyarrhythmias. However, repeated testing of multiple antiarrhythmic agents may be required to design effective therapy. These repeated studies are expensive, time consuming and difficult for patients.

Procardiamide is a frequently used antiarrhythmic drug for treating ventricular arrhythmias. High doses are often required to control sustained ventricular tachyarrhythmias. Previous studies in our laboratory have demonstrated that procainamide is the most successful of standard agents for preventing inducible ventricular tachycardia. The present study was therefore undertaken to determine whether the response to procainamide could predict the response to other conventional antiarrhythmic agents using electrophysiologic studies in patients with inducible sustained ventricular tachyarrhythmias.

Methods

Patients

The patient population consisted of 107 men and 19 women, ages 16–77 years. Cardiac diagnoses included atherosclerotic heart disease in 107 patients, 102 of whom had prior myocardial infarction, 57 with left ventricular aneurysm. Other diagnoses included cardiomyopathy in six patients, tetralogy of Fallot in one patient, mitral valve prolapse in three patients, arhythmogenic right ventricular dysplasia in one patient, sarcoidosis with left ventricular aneurysm in one

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From the Clinical Electrophysiology Laboratory, Hospital of the University of Pennsylvania, and the Cardiovascular Section, Department of Medicine, University of Pennsylvania School of Medicine, Philadelphia, Pennsylvania.

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Address for correspondence: Harvey L. Waxman, M.D., 656 Ravdin Building, Hospital of the University of Pennsylvania, 3400 Spruce Street, Philadelphia, Pennsylvania 19104.

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and hypertensive cardiovascular disease in one. Six patients had no evidence of organic heart disease (primary electrical disease). All patients had sustained ventricular tachyarrhythmias (requiring pacing for termination or cardioversion due to degeneration to ventricular fibrillation and/or hemodynamic collapse) induced during the control electrophysiologic study while not taking antiarrhythmic medications.

Electrophysiologic Study

All patients gave written informed consent. They were studied in a nonasated, postabsorptive state. Patients were initially studied after all antiarrhythmic medications were discontinued for 48–72 hours (control study). During the control study, catheter electrodes were inserted percutaneously using local anesthesia and positioned using fluoroscopic guidance in the high right atrium, at the atrioventricular (AV) junction for recording of the His bundle electrogram, in multiple right ventricular and occasionally multiple left ventricular sites. During subsequent electrophysiologic studies used to test various antiarrhythmic regimens, usually only one catheter electrode positioned at one or more right ventricular sites was used. Number 6F quadrupolar catheter electrodes (USCI) were used in all instances except for recording of the His bundle electrogram, in which case a #7F tripolar catheter electrode was used. Stimulation was performed from the distal pair and recording from the proximal pair of electrodes on the quadripolar catheters. Stimulation was performed with a custom designed programmable stimulator (Bloom Associates, Inc.) adjusted to deliver rectangular impulses 1 msec in duration at approximately twice diastolic threshold using a constant current source. Recordings were made using a 16-channel physiologic recorder (VR-16, Electronics for Medicine) and real-time records obtained with a jet ink recorder (Siemens Elema minigraph) at a paper speed of 100–250 mm/sec. Data were stored on a 14-channel magnetic tape recorder (Honeywell 5600C) and retrieved on photographic paper at a speed of 100–150 mm/sec for illustrative purposes.

Definitions

S₃ and V₁ refer to the stimulus during the drive cycle and the ventricular response to that stimulus respectively. S₂ and V₂ refer to the premature stimulus delivered after eight spontaneous or paced beats and the ventricular response to that premature stimulus respectively. S₄ and V₄ and S₃ and V₃ refer to the second and third premature stimuli and ventricular responses, respectively. The ventricular effective refractory period is defined as the longest S₃ S₄ which fails to produce a propagated ventricular response.

Stimulation Protocol

Ventricular stimulation consisted of one or two, and in two-thirds of the patients, three premature ventricular stimuli at two ventricular paced cycle lengths (usually 600 and 400 msec) delivered at two right ventricular and, in a few patients, left ventricular sites. Stimulation was begun in late diastole after eight paced or spontaneous beats and the S₃ was moved earlier in diastole by 10-msec decrements until it failed to capture (ventricular effective refractory period). The S₂ was then moved approximately 50 msec beyond the effective refractory period and the S₁ was added at twice this interval (ventricular effective refractory period plus 50 msec). The S₁ was moved earlier in diastole by 10-msec decrements until it failed to capture. The S₄ was then moved earlier in diastole by 10-msec decrements until the S₄ again captured. The S₁, S₂, and, in some cases, S₃ were delivered in this manner until the S₄ failed to capture. If premature stimulation failed to induce sustained ventricular arrhythmias, then rapid ventricular pacing at cycle lengths of 350–250 msec for 5–60 seconds, as tolerated by the patient, was performed. If this stimulation protocol was unsuccessful at two right ventricular sites (most often the right ventricular apex and the right ventricular outflow tract), then left ventricular stimulation was performed using a similar stimulation protocol. Stimulation comparable to that used at baseline study with respect to site of stimulation and stimulation protocol was used in each patient during all follow-up studies.

Administration of Antiarrhythmic Agents

Procainamide was administered orally (10 cases) at a dose of 4–12 g/day and intravenously (93 cases) at a dose of 1–2.5 g at 50 mg/min followed by a 4–10-mg/min continuous infusion. The efficacy of procainamide was evaluated after both intravenous and oral administration using similar dosing regimens in 23 patients. Other agents tested included lidocaine, 3–5 mg/kg over 10 minutes, followed by a continuous infusion of 4 mg/min; quinidine 300–600 mg orally every 6 hours; disopyramide 100–300 mg orally every 6 hours; and phenytoin 300–400 mg/day orally (table 1). All orally administered drugs were given for at least five half-lives to achieve steady-state levels up to maximally tolerated dosages before restudy. Combinations of agents were also used, the most common of which were procainamide and phenytoin and quinidine and phenytoin. Propranolol alone or in combination was only used as an antiarrhythmic agent in seven cases, mainly because of the severely impaired hemodynamic status of our patients. The number of drug trials with each antiarrhythmic agent or combination of agents is listed in table 1.

Statistical Analysis of Data

The t test for unpaired data and chi-square analysis were used when appropriate. All numerical values are expressed as mean ± SD unless otherwise specified.

Results

Response to Procainamide

In 42 patients (33%), ventricular tachycardia became noninducible and in 84 patients (67%), the tachycardia remained inducible after the administration of procainamide (table 2). Serum procainamide levels were available in 44 patients in whom ventricular
tachycardia remained inducible and in 19 patients in whom ventricular tachycardia was not inducible on procainamide. The mean serum level in those patients in whom ventricular tachycardia remained inducible was 13.8 ± 6 mg/l and in those in whom ventricular tachycardia was not inducible, 12.2 ± 5 mg/l. This difference was not significant.

A somewhat higher percentage of ventricular tachyarrhythmias that remained inducible after procainamide were induced with one ventricular premature depolarization during the control electrophysiologic study compared with those noninducible after procainamide, 32% vs 19%. Nevertheless, there was no significant difference between the two groups in the mode of induction of ventricular arrhythmia during the control study (table 2).

Eighty of the 84 patients in whom ventricular tachycardia was inducible after procainamide and 36 of the 42 in whom tachycardia was not inducible after procainamide had ventricular tachycardia induced with a uniform morphology during the control electrophysiologic study. The mean cycle length of ventricular tachycardia in these two groups of patients was not significantly different, 293 ± 68 vs 276 ± 64 msec (table 2). The remaining four patients in whom ventricular tachyarrhythmias were inducible and the remaining six patients in whom tachyarrhythmias were not inducible after procainamide had rapid polymorphic ventricular tachycardia induced during the control study that often had a torsade de pointes morphology and degenerated into ventricular fibrillation. In these 10 patients, an accurate ventricular tachycardia cycle length could not be determined during the control electrophysiologic study.

A significantly larger percentage of patients with inducible ventricular tachycardia during procainamide therapy had prior myocardial infarction or left ventricular aneurysm (89% and 56%, respectively) compared with those with no arrhythmia inducible on procainamide (67% and 27%, respectively). The absence of apparent organic heart disease was more prevalent in the group with no inducible ventricular tachycardia on procainamide (table 3).

### Serial Electrophysiologic Studies

In 69 of 84 patients in whom ventricular tachycardia

### Table 1. Antiarrhythmic Regimens

<table>
<thead>
<tr>
<th>Medications</th>
<th>Route</th>
<th>Dose</th>
<th>No. of trials</th>
</tr>
</thead>
<tbody>
<tr>
<td>Procainamide</td>
<td>Oral</td>
<td>4–14 g/day</td>
<td>33</td>
</tr>
<tr>
<td></td>
<td>Intravenous</td>
<td>1–2.5 g</td>
<td>116</td>
</tr>
<tr>
<td>Quinidine</td>
<td>Oral</td>
<td>300–600 mg q6h</td>
<td>64</td>
</tr>
<tr>
<td>Disopyramide</td>
<td>Oral</td>
<td>100–300 mg q6h</td>
<td>46</td>
</tr>
<tr>
<td>Lidocaine</td>
<td>Intravenous</td>
<td>3–5 mg/kg over 10 min</td>
<td>38</td>
</tr>
<tr>
<td></td>
<td></td>
<td>followed by 4-mg/min infusion</td>
<td></td>
</tr>
<tr>
<td>Phenytoin</td>
<td>Oral</td>
<td>300–500 mg/day</td>
<td>32</td>
</tr>
<tr>
<td>Propranolol</td>
<td>Oral</td>
<td>40–120 mg q6h</td>
<td>6</td>
</tr>
<tr>
<td>Phenytoin and</td>
<td></td>
<td>Dosages as above</td>
<td>17</td>
</tr>
<tr>
<td>Procainamide</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Phenytoin and</td>
<td></td>
<td>Dosages as above</td>
<td>7</td>
</tr>
<tr>
<td>Quinidine</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Quinidine and</td>
<td></td>
<td>Dosages as above</td>
<td>1</td>
</tr>
<tr>
<td>Propranolol</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lidocaine and</td>
<td></td>
<td>Dosages as above</td>
<td>1</td>
</tr>
<tr>
<td>Disopyramide</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Table 2. Response to Procainamide

<table>
<thead>
<tr>
<th>Procainamide level (µg/ml)</th>
<th>Inducible (84)</th>
<th>Noninducible (42)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1VPD</td>
<td>13.8±6 (n = 44)</td>
<td>12.2±5* (n = 19)</td>
</tr>
<tr>
<td>2VPD</td>
<td>27 (32%)</td>
<td>8 (19%)*</td>
</tr>
<tr>
<td>3VPD</td>
<td>39 (46%)</td>
<td>24 (57%)*</td>
</tr>
<tr>
<td>4VPD</td>
<td>15 (18%)</td>
<td>8 (19%)*</td>
</tr>
<tr>
<td>VDP</td>
<td>2 (3%)</td>
<td>0*</td>
</tr>
<tr>
<td>RVP</td>
<td>2 (5%)*</td>
<td></td>
</tr>
<tr>
<td>Control arrhythmia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>VT</td>
<td>80 pts (CL-293±68 msec)</td>
<td>36 pts* (CL 276±64 msec)</td>
</tr>
<tr>
<td>TDF/VF</td>
<td>4 pts</td>
<td>6 pts</td>
</tr>
<tr>
<td>Serial studies</td>
<td></td>
<td></td>
</tr>
<tr>
<td>69 pts</td>
<td>2.1±1 study/pt</td>
<td></td>
</tr>
<tr>
<td>30 pts</td>
<td>2.2±1 study pt*</td>
<td></td>
</tr>
</tbody>
</table>

*NS.

Abbreviations: VDP = ventricular premature depolarization; RVP = rapid ventricular pacing; VT = ventricular tachycardia; TDF/VF = torsades de pointes/ventricular fibrillation.
remained inducible after procainamide and in 30 of the 42 patients in whom ventricular tachycardia was not inducible after procainamide, serial electrophysiologic studies on other antiarrhythmic regimens were performed. These patients underwent one to five serial electrophysiologic studies. The mean number of serial electrophysiologic studies (i.e., studies in addition to procainamide) performed in the two groups of patients were similar, 2.1 ± 1 vs 2.2 ± 1. The remaining patients in each group were treated with either experimental drugs or surgery because of clinical failure on conventional antiarrhythmic regimens.

One hundred forty-five drug trials with conventional agents or combinations of agents were performed in the 69 patients in whom ventricular tachycardia remained inducible after procainamide (fig. 1). None of the 22 trials with lidocaine, two of 42 trials with quinidine (5%), four of 32 trials with disopyramide (13%), and four of 49 trials with phenytoin, propranolol, or combinations of agents (8%) prevented induction of ventricular tachycardia. Thus, only 10 of 145 drug trials (7%) in these patients prevented ventricular tachycardia induction. An example of serial electrophysiologic studies in a patient in whom ventricular tachycardia remained inducible after procainamide is shown in figure 2.

Sixty-seven drug trials with conventional antiarrhythmic agents were performed in the 30 patients in whom ventricular tachycardia was not inducible after the administration of procainamide. Nine of 16 trials with lidocaine (56%), 16 of 22 with quinidine (73%), 10 of 14 with disopyramide (71%), and eight of 15 with phenytoin, propranolol, or combinations of agents (53%) prevented induction of ventricular tachycardia (fig. 3). In all, 43 of the 67 trials (64%) resulted in noninducibility of ventricular tachycardia. An example of serial electrophysiologic studies in a patient who responded to procainamide is shown in figure 4.

### Table 3. Underlying Heart Disease

<table>
<thead>
<tr>
<th></th>
<th>Inducible on procainamide (n = 84)</th>
<th>Noninducible on procainamide (n = 42)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>ASHD and MI</td>
<td>74 (88%)</td>
<td>28 (67%)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>LV An</td>
<td>46 (55%)</td>
<td>11 (26%)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Other</td>
<td>8 (10%)</td>
<td>11 (26%)</td>
<td>&lt;0.05</td>
</tr>
</tbody>
</table>

Percentages in parentheses refer to percent of patients with inducible (84 patients) or noninducible (42 patients) ventricular tachycardia on procainamide.

Abbreviations: ASHD = atherosclerotic heart disease; MI = myocardial infarction; LV An = left ventricular aneurysm.

Antiarrhythmic Medication Levels

Drug levels of quinidine, disopyramide and phenytoin were available at the time of electrophysiologic study in approximately half of the patients. There were no significant differences in the quinidine and phenytoin levels between patients in whom ventricular tachycardia was inducible compared with those in whom it was not inducible on each drug. The mean level of disopyramide was higher during study in patients in whom ventricular tachycardia was not inducible compared with those in whom it was inducible, 5.0 mg/l vs 3.0 mg/l (p < 0.01) (table 4). There were not enough lidocaine levels to allow accurate analysis.

There were no significant differences in the levels of disopyramide or phenytoin between patients in whom ventricular tachycardia was inducible or noninducible on procainamide. The quinidine levels in the patients in whom ventricular tachycardia remained inducible while taking procainamide were somewhat higher than the levels in the patients in whom ventricular tachycardia was not inducible while taking procainamide (table 5).

### Patient Response

In 60 of the 69 patients in whom ventricular tachycardia remained inducible after procainamide, ventricular tachycardia remained inducible on all other conventional antiarrhythmic regimens tested (fig. 5). By comparison, of the 30 patients in whom ventricular tachycardia became noninducible after procainamide, at least one other antiarrhythmic regimen prevented induction of ventricular tachycardia in 25. Thus, the predictive value of inducibility of ventricular tachycardia on procainamide for inducibility of ventricular tachycardia on other conventional antiarrhythmic agents was 87%, while the predictive value of noninducibility on procainamide for noninducibility of ventricular tachycardia on other conventional antiarrhythmic agents was 83%.

The percentage of patients with atherosclerotic heart disease and prior myocardial infarction was similar in patients in whom ventricular tachycardia was inducible during all antiarrhythmic regimens tested and those in whom it responded to one or more regimens. However, the percentage of patients with left ventricular aneurysm was lower and the prevalence of other cardiac diagnosis (including atherosclerotic heart dis-
ease without prior myocardial infarction and primary electrical disease) was higher among the patients who responded to one or more antiarrhythmic regimens (table 6).

Analysis similar to that performed with procar-
ainide using quinidine and disopyramide revealed that these medications also were predictive of response during programmed ventricular stimulation. Thirty-nine of 50 patients (78%) in whom ventricular tachycardia was inducible during quinidine therapy had ventricular tachycardia inducible on all other regimens tested, whereas 18 of 23 patients (78%) with no ventricular tachycardia inducible during quinidine therapy had no ventricular tachycardia inducible on at least one other regimen tested (p < 0.001). Twenty-eight of 35 patients (80%) in whom ventricular tachycardia was inducible during disopyramide therapy had ventricular tachycardia inducible on all other regimens tested, whereas 12 of 16 patients (75%) with no ventricular tachycardia inducible during disopyramide therapy had no ventricular tachycardia inducible on at least one other regimen (p < 0.001).

Discussion

Electrophysiologic testing can be used to guide therapy of patients with sustained ventricular tachyar-rhythms using conventional antiarrhythmic agents or combinations of agents. Long-term follow-up has con-

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**FIGURE 2.** Serial electrophysiologic studies in a patient in whom ventricular tachycardia remained inducible after procainamide. During the control electrophysiologic study, sustained ventricular tachycardia with a cycle length of 280 msec is induced with two premature ventricular stimuli delivered at the right ventricular apex (RVA). After the administration of procainamide, 1 g intravenously to achieve a serum level of 18.3 µg/ml, ventricular tachycardia is induced with a single premature ventricular beat. The ventricular tachycardia is slowed and now has a cycle length of 340 msec. The ventricular tachycardia induced also has a morphology different from that induced during the control study. After the administration of quinidine, 400 mg orally every 6 hours for 48 hours to achieve a serum level of 3.3 µg/ml, ventricular tachycardia is again induced with a single premature ventricular beat. The ventricular tachycardia is now markedly slowed and has a cycle length of 430 msec. After the administration of disopyramide 200 mg orally every 6 hours to achieve a serum level of 5.6 µg/ml, ventricular tachycardia is again induced with a single premature ventricular beat delivered at the RVA. The ventricular tachycardia is slow and has a cycle length of 440 msec. Thus, in this patient with inducible ventricular tachycardia during the control studies and on procainamide, ventricular tachycardia was also inducible on quinidine and disopyramide.

**FIGURE 3.** Results of serial electrophysiologic testing in the 30 patients in whom ventricular tachycardia was not inducible after the administration of procainamide. "Other" refers to phenytoin, propranolol and combinations of antiarrhythmic agents. Forty-three of the 67 drug trials performed in this group of patients prevented the induction of ventricular tachycardia.
confirmed the predictive value of acute drug studies using provocative testing in this group of patients. This testing is most useful in patients with paroxysmal arrhythmias in whom empiric drug therapy often requires prolonged observation with multiple recurrences before an adequate regimen can be devised. However, electrophysiologic testing is expensive, time consuming, and difficult for patients and their families. Furthermore, in our experience, only approximately one-third of patients with recurrent sustained ventricular tachycardia so tested will respond to conventional agents or combinations. The remainder require experimental agents such as amiodarone or surgery for control of arrhythmias.

Procainamide is an antiarrhythmic agent widely used for the control of ventricular arrhythmias. It is effective in controlling ventricular arrhythmias associated with acute ischemia as well as for recurrent sustained ventricular tachycardia unassociated with acute ischemia. Procainamide has been effective in preventing induction during invasive electrophysiologic study in 12.5–45% of cases. These variable response rates may be attributed to patient populations, drug dosages and criteria for success. We observed a 33% response rate. These relatively good results achieved with procainamide are probably attributable to the high dosages used and high levels achieved. Good results with large doses of procainamide, 3–12 g/day, has been reported from our laboratory in a small group of patients with inducible sustained ventricular tachycardia. The mean serum level of procainamide in that study was 13.6 ± 8.6 μg/ml, similar to the levels achieved in the present study.

Most studies using provocative testing to devise a regimen for control of patients with sustained ventricu-

![Table 4](http://circ.ahajournals.org/)

**Table 4. Drug Levels (μg/ml)**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Inducible n</th>
<th>Noninducible n</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Quinidine</td>
<td>4.2 ± 1.3</td>
<td>3.5 ± 1.7</td>
<td>NS</td>
</tr>
<tr>
<td>Disopyramide</td>
<td>3.0 ± 1.0</td>
<td>5.0 ± 1.9</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Phenytoin</td>
<td>13.3 ± 4.2</td>
<td>12.0 ± 5.2</td>
<td>NS</td>
</tr>
</tbody>
</table>

![Table 5](http://circ.ahajournals.org/)

**Table 5. Drug Levels (μg/ml)**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Inducible on procainamide n</th>
<th>Noninducible on procainamide n</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Quinidine</td>
<td>4.2 ± 1.4</td>
<td>3.4 ± 1.3</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Disopyramide</td>
<td>3.2 ± 1.1</td>
<td>4.5 ± 2.2</td>
<td>NS</td>
</tr>
<tr>
<td>Phenytoin</td>
<td>12.1 ± 4.3</td>
<td>13.7 ± 4.8</td>
<td>NS</td>
</tr>
</tbody>
</table>
lar tachyarrhythmias have involved relatively few patients.1-4, 6-10 Furthermore, they were usually aborted after a single effective antiarrhythmic regimen was attained. Although several studies have reported patients who failed to respond to procainamide who responded to other regimens, the dosages of procainamide or serum levels achieved were either low or not reported.6-10 Early experience in our laboratory with relatively few patients suggested that the effect of specific drugs was random and unpredictable.2

The rationale for testing multiple antiarrhythmic regimens in spite of response to one regimen is based upon the large number of side effects associated with each individual antiarrhythmic agent. If side effects develop during treatment with one agent, it is useful to have one or more alternative therapeutic regimens available without the need for rehospitalization and repeat electrophysiologic evaluation.

The purpose of the present investigation was to determine whether the response to procainamide could predict the response to other conventional antiarrhythmic agents or combinations of agents as assessed by intracardiac electrophysiologic study and thereby obviate the need for repeated testing of other conventional agents. We observed in 69 patients in whom ventricular tachycardia was inducible during procainamide therapy that only 10 of 145 drug trials on other antiarrhythmic regimens prevented induction of ventricular tachycardia. By comparison, in 30 patients in whom ventricular tachycardia was not inducible on procainamide, 43 of 67 drug trials resulted in noninducibility of ventricular tachycardia. Sixty of the 69 patients with inducible ventricular tachycardia on procainamide had inducible ventricular tachycardia on all other regimens tested, whereas 25 of the 30 patients with no ventricular tachycardia inducible on procainamide had no ventricular tachycardia inducible on at least one other regimen tested.

Although the number of patients and drug trials were smaller, similar trends were observed using disopyramide or quinidine as the predictive agent. The predictive value of inducibility of ventricular tachycardia during quinidine and disopyramide therapy on all other agents was 73% and 80%, respectively. The predictive value of noninducibility of ventricular tachycardia on quinidine and disopyramide for noninducibility of ventricular tachycardia on at least one other regimen was 77% and 81%, respectively.

Thus, the response to procainamide yielded the best predictive value and was derived using the most data. Response to procainamide predicted the response to other regimens tested, with a predictive value of 87% for inducibility and 83% for noninducibility of ventricular tachycardia.

**Limitations**

One to five antiarrhythmic regimens were tested in our patients in addition to procainamide. The mean number of other regimens tested was 2.2 ± 1. Testing larger numbers of regimens could have resulted in a higher response rate in patients in whom ventricular tachycardia remained inducible on procainamide.

In conclusion, the response to procainamide predicts the response to other conventional antiarrhythmic agents during electrophysiologic study. Therefore, patients who fail to respond to procainamide should be evaluated for response to experimental agents or surgery without necessarily undergoing prolonged testing with other conventional regimens. There are a few patients who fail to respond to procainamide who respond to other conventional agents. Therefore, in patients who are poor surgical candidates or who are poor candidates for experimental agents, repeated electrophysiologic testing may be worthwhile.

**References**


The response to procainamide during electrophysiologic study for sustained ventricular
tachyarrhythmias predicts the response to other medications.
H L Waxman, A E Buxton, L M Sadowski and M E Josephson

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