Idiopathic ventricular fibrillation: inducibility and beneficial effects of class I antiarrhythmic agents

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ABSTRACT Ventricular fibrillation in patients without recognizable heart disease is uncommon and electrophysiologic data on such patients is limited. Over a 7 year period, five patients (three men and two women, ranging in age from 24 to 52 years) without demonstrable heart disease underwent electrophysiologic studies with pharmacologic drug testing because of single (four patients) or multiple (one patient) documented episodes of ventricular fibrillation. The arrhythmic event was unrelated to myocardial ischemia or infarction, metabolic or electrolyte disturbances, drug toxicity, preexcitation, or prolonged QT syndromes. In all three patients receiving no antiarrhythmic drugs and in two pretreated with amiodarone, a rapid poorly tolerated ventricular tachyarrhythmia requiring cardioversion was induced by programmed ventricular stimulation with up to two extrastimuli. In all instances, addition of either oral quinidine or oral disopyramide prevented the induction of sustained ventricular arrhythmias. All five patients were placed on antiarrhythmic drug regimens found effective during electrophysiologic studies and remained asymptomatic during follow-up periods ranging from 12 to 93 (mean 52) months. We conclude that in the patients with idiopathic ventricular fibrillation in our study: (1) programmed ventricular stimulation reliably replicated the spontaneous arrhythmia, (2) class I antiarrhythmic agents effectively prevented induction of the arrhythmia in the laboratory, and (3) in contrast to the severity of the presenting arrhythmia, a benign clinical course was observed during long-term therapy with class I antiarrhythmic agents.


In Western countries ventricular fibrillation usually occurs in patients with cardiac disease, most commonly in those with coronary artery disease, but also among those with cardiomyopathy, valvular or congenital heart disease, preexcitation, or prolonged QT syndromes.1 Occurrence of ventricular fibrillation in the absence of recognizable heart disease has been rarely reported in the literature. The first case of apparently idiopathic ventricular fibrillation was probably described by William Dock in 1929.2 Since then isolated cases have been reported, most of them before the advent of cardiac resuscitation techniques and diagnostic catheterization procedures.3-9 Electrophysiologic data on such patients is limited to only a few cases scattered among large series of patients with life-threatening ventricular tachyarrhythmias due to various causes.10-15 In those patients with idiopathic ventricular fibrillation, a low inducibility rate of ventricular tachyarrhythmia was found, and of those with inducible ventricular tachyarrhythmias, electrophysiologic drug testing data are available on only two patients.13,15

We here report a series of five consecutive patients with idiopathic ventricular fibrillation inducible in the electrophysiologic laboratory in whom class I antiarrhythmic agents were effective during both electrophysiologic study and long-term therapy.

Patients and methods

Study patients. During the last 7 years (February 1979 to January 1986) five consecutive patients without demonstrable heart disease were referred to our institution for electrophysiologic study because of documented ventricular fibrillation. Ventricular fibrillation occurred 4 days to 1 year before their referral for electrophysiologic study. In all cases, the diagnosis of ventricular fibrillation was made by paramedics or by physicians and defibrillation was required to abolish the arrhythmia. In all patients results of physical examination, 12-lead electrocardiography, chest x-ray, M mode and two-dimensional echocardiography, radionuclide ventriculography, left and right ventriculography, and coronary angiography were normal. None of the patients complained of chest pain or was taking any medications before cardiac arrest. None of the patients had evidence of acute
myocardial ischemia or infarction or metabolic or electrolyte disturbances.

**Electrophysiologic study.** Electrophysiologic studies were performed in patients in the postabsorptive nonsedated state after each gave informed consent. Initial electrophysiologic studies were performed in three patients while on no antiarhythmic medications and in two patients while on amiodarone. Discontinuation of amiodarone therapy before initial electrophysiologic study was refused by one patient (No. 3), and therapy could not be stopped in the other patient with recurrent ventricular fibrillation (No. 1) in whom this drug prevented recurrence of the arrhythmia.

**Technique.** During the initial study two quadripolar electrode catheters and one bipolar electrode catheter (USCI, No. 6F) were introduced in each patient through femoral veins and placed in the high right atrium, right ventricular apex, and at the His bundle area, respectively. During repeat electrophysiologic study of patients on long-term antiarrhythmic therapy only one electrode catheter placed in the right ventricle was used. Recordings were made with an eight-channel recorder (Mingograph 82). Intracardiac stimulation was carried out with a Medtronic stimulator, model 5325, with the use of stimuli of 3 mA intensity and approximately 2 msec in duration. Stimulation was performed from the distal and recordings from the proximal pairs of electrodes of the quadripolar catheters.

**Protocol of stimulation.** During the initial electrophysiologic study the following protocol of stimulation was used: (1) single and double atrial extrastimuli during right atrial pacing at a basic cycle length of 600 msec, (2) incremental atrial pacing until second-degree atioventricular block occurred, (3) bursts of rapid atrial pacing at the maximum atrial rate allowing 1:1 atioventricular conduction, (4) single and double right ventricular apical extrastimuli during sinus rhythm and during ventricular pacing at cycle lengths of 600, 500, and 400 msec, and when possible at 700 msec, and (5) bursts of rapid right ventricular apical pacing (5 to 10 beats) up to a rate of 250 beats/min. During subsequent electrophysiologic studies performed to evaluate oral antiarrhythmic regimens only ventricular stimulation was performed, according to the protocol described above. Ventricular stimulation was performed from the right ventricular apex in all patients and also from the right ventricular outflow tract in the last patient.

**Drug studies.** In two patients the effects of the intravenous administration of disopyramide (2 mg/kg body weight over a 5 min period) on induction of ventricular tachyarrhythmias were assessed at the completion of the initial electrophysiologic study. Programmed ventricular stimulation was repeated after oral administration of quinidine bisulfate (1500 to 2250 mg/day) in four patients and after disopyramide (600 mg/day) in one patient. In all patients serum blood levels of either quinidine or disopyramide were determined during electrophysiologic studies.

**Follow-up.** After discharge from the hospital the patients were followed at the outpatient clinic every 3 months during the first year and every 6 months afterward. Holter monitoring was performed every 6 months. In patients 1 to 5 both echocardiographic and radionuclide ventriculographic examinations were performed after 83, 60, 51, 12, and 8 months of follow-up, respectively.

**Results**

**Clinical data (table 1).** Three male and two female patients ranging in age from 24 to 52 years (mean 33) were studied. Three patients were of Sephardic origin while the remaining two were of Ashkenazic origin.

All patients were awake just before their cardiac arrests: two were lying, two were standing, and one was sitting. Two patients (Nos. 2 and 4) were under psychological stress during the minutes preceding the cardiac arrest, while the other three patients were engaged in normal activities. Four patients (Nos. 2 to 5) had had no cardiac symptoms (e.g. palpitations, dizziness, or syncope) in the past. One patient (No. 1) exhibited three episodes of syncope during the 24 hr preceding documentation of ventricular fibrillation.

None of the five patients had other medical problems in the past or a familial history of cardiac arrhythmias or sudden death.

The two patients who suffered cardiac arrest during a stressful situation (Nos. 2 and 4) were treated with a β-blocking agent (propranolol) by their referring physicians after their cardiac arrests. However, both of them discontinued this treatment a few days (patient 4) or 1 month (patient 2) after hospital discharge. In patient 1 who had recurrent ventricular fibrillation, therapy with amiodarone prevented recurrence of the arrhythmia, but 30 days into therapy, a presyncopal episode strongly suggesting a self-terminating ventricular tachyarrhythmia occurred and prompted electrophysiologic evaluation.

**Electrocardiographic data.** In all five patients standard electrocardiograms, including QT intervals, were normal. No patient had evidence of short PR interval or

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**TABLE 1**

**Clinical features**

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Sex/age (yr)</th>
<th>Prior symptoms</th>
<th>Documented arrhythmia</th>
<th>Activity at cardiac arrest</th>
<th>Stress situation</th>
<th>Time from arrhythmia to EPS</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M/28</td>
<td>Syncope (× 3)</td>
<td>VF</td>
<td>Lying</td>
<td>—</td>
<td>40 days</td>
</tr>
<tr>
<td>2</td>
<td>F/36</td>
<td>—</td>
<td>VF</td>
<td>Standing</td>
<td>Dispute</td>
<td>1 yr</td>
</tr>
<tr>
<td>3</td>
<td>M/52</td>
<td>—</td>
<td>VF</td>
<td>Standing</td>
<td>—</td>
<td>10 days</td>
</tr>
<tr>
<td>4</td>
<td>F/24</td>
<td>—</td>
<td>VF</td>
<td>Lying</td>
<td>Abortion</td>
<td>9 mo</td>
</tr>
<tr>
<td>5</td>
<td>M/25</td>
<td>—</td>
<td>VF</td>
<td>Sitting</td>
<td>—</td>
<td>4 days</td>
</tr>
</tbody>
</table>

**EPS** = electrophysiologic study; **VF** = ventricular fibrillation.

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VENTRICULAR PREEXCITATION. Spontaneous ventricular premature complexes were observed in one patient (No. 1); these complexes frequently occurred (up to ventricular trigeminy) and had a left bundle branch block morphology with a left axis deviation and a very short coupling interval (QR/QT = 0.8). In this patient spontaneous initiation of ventricular fibrillation was witnessed in 10 instances during the first 24 hr of hospitalization (figure 1). Ventricular fibrillation invariably followed rapid polymorphic ventricular tachycardia initiated by very early coupled premature ventricular complexes. Termination of ventricular fibrillation required cardioversion in eight instances; in two instances, however, ventricular fibrillation spontaneously terminated within about 15 sec and reorganization of ventricular activity was observed before termination of arrhythmia (figure 1).

Electrophysiologic findings (table 2). In all patients AH and HV intervals were normal and no antegrade preexcitation could be demonstrated by incremental right atrial pacing or atrial extrastimulation. Atrioventricular function was normal in all but two patients (Nos. 1 and 3) who were taking amiodarone at the time of study. In four of the five patients the effective refractory period of the right ventricular apex was normal, ranging from 220 to 250 msec at basic cycle lengths of 500 to 600 msec. In one patient (No. 3), the increase in the ventricular effective refractory period (280 msec) was most probably related to amiodarone therapy.

During initial electrophysiologic studies in all patients sustained rapid ventricular tachycardia-flutter (cycle length ranging from 140 to 250 msec) was induced by programmed right ventricular apical stimulation (figures 2 and 3). In all instances the QRS complexes at the onset of the induced ventricular tachycardia had a left bundle branch block morphology with a varying axis. The mode of induction was single (one patient) or double (four patients) ventricular extrastimuli during ventricular pacing. In all patients the induced tachycardias degenerated into ventricular fibrillation within a few seconds and required direct-current shock for termination.

Drug studies (table 3). Programmed ventricular stimulation performed after intravenous administration of disopyramide did not induce any ventricular arrhythmia in one patient (No. 2) (figure 4), but induced a presyncopal episode of self-terminating ventricular tachycardia-fibrillation in another patient (No. 6).

During oral therapy with disopyramide in patient 2, no more than one ventricular complex was induced by

**FIGURE 1.** Patient 1. Ventricular arrhythmias recorded on admission. Shown are electrocardiographic monitor strips. A, Normal sinus rhythm with ventricular trigeminy. Note the very short coupling interval of the ventricular premature complexes. B, Short run of rapid (260 beats/min) polymorphic ventricular tachycardia triggered by the early ventricular complexes. C, Ventricular fibrillation that required cardioversion. D and E, Self-terminating ventricular fibrillation. Note the reorganization of ventricular activity at the termination of fibrillation.
TABLE 2
Findings during initial electrophysiologic studies

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>AH (msec)</th>
<th>HV (msec)</th>
<th>APCLW (msec)</th>
<th>VERP (CL)</th>
<th>Characteristics of inducible arrhythmias</th>
<th>Drugs taken at the time of EPS</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Type</td>
<td>Pattern (CL)</td>
</tr>
<tr>
<td>1</td>
<td>100</td>
<td>50</td>
<td>545</td>
<td>220</td>
<td>VT→VF</td>
<td>Polymorphic</td>
</tr>
<tr>
<td>2</td>
<td>80</td>
<td>50</td>
<td>400</td>
<td>220</td>
<td>VT→VF</td>
<td>Polymorphic</td>
</tr>
<tr>
<td>3</td>
<td>100</td>
<td>50</td>
<td>570</td>
<td>280</td>
<td>VT→VF</td>
<td>Polymorphic</td>
</tr>
<tr>
<td>4</td>
<td>100</td>
<td>45</td>
<td>375</td>
<td>250</td>
<td>VT→VF</td>
<td>Polymorphic</td>
</tr>
<tr>
<td>5</td>
<td>90</td>
<td>40</td>
<td>310</td>
<td>220</td>
<td>VT→VF</td>
<td>Polymorphic</td>
</tr>
</tbody>
</table>

AM = amiodarone; APCLW = atrial paced cycle length-induced Wenckebach atrioventricular block; CL = cycle length (msec); DC = direct current; LBBB = left bundle branch block; S1S1 = basic ventricular cycle length; S1/S2 = single ventricular extrastimulus (S2) at a basic cycle length of S1S1; S1/S2/S3 = double ventricular extrastimuli (S2, S3) at a basic cycle length of S1S1; VERP = ventricular effective refractory period (msec); VT = ventricular tachycardia; VF = ventricular fibrillation; other abbreviations as in table 1.

aAt the onset of the induced ventricular tachycardia.

programmed ventricular stimulation (figure 4). Programmed stimulation from the right ventricular apex was performed after oral administration of quinidine in four patients (Nos. 1 and 3 to 5). No more than three ventricular complexes were induced in three patients (Nos. 1, 3, and 5). In patient 4 nonsustained ventricular tachycardias (no more than 14 complexes) were induced. In patient 5, programmed stimulation from the right ventricular outflow tract failed to induce any arrhythmia (figure 3).

![Figure 2](http://circ.ahajournals.org/)

**FIGURE 2.** Patient 2. Induction of rapid ventricular tachyarrhythmia by programmed ventricular stimulation during control electrophysiologic study. Shown are recordings from electrocardiographic leads I, II, III, and V1 and intracardiac electrogams from the atrioventricular junction (AVJ), high right atrium (HRA), and right ventricular apex (RVA). During right ventricular apical pacing at a fixed cycle length S1S1 of 600 msec (the last S1 beat only being shown), the introduction of two ventricular extrastimuli (S2, S3) results in the induction of a sustained irregular polymorphic ventricular tachycardia (cycle length 160 to 235 msec). Note the left bundle branch block morphology of the induced tachycardia.
Follow-up (table 4). All five patients were discharged on antiarrhythmic regimens that prevented induction of sustained ventricular tachyarrhythmias during electrophysiologic testing. These drug regimens included disopyramide (one patient) and quinidine alone (two patients) or associated with amiodarone (two patients). All patients remained asymptomatic and have had a normal life during follow-up period ranging from 12 to 93 (mean 52) months. One patient (No. 4) had a normal pregnancy and delivery while on quinidine therapy 1 year after her episode of ventricular fibrillation. In each patient repeated Holter monitoring did not show any significant arrhythmias. During the follow-up period, the cardiac examination, electrocardiogram, echocardiogram, and radionuclide ventriculogram remained normal in all patients.

Discussion

In the present report we describe five patients who experienced a single or multiple episodes of ventricular fibrillation. In no patient could organic heart disease be demonstrated by extensive invasive or non-invasive cardiac evaluation. During initial electrophysiologic studies performed in three patients on no drug and two on amiodarone, rapid poorly tolerated ventricular tachyarrhythmias degenerating into ventricular fibrillation were induced by programmed ventricular stimulation. In all cases addition of class I antiarrhyth-

Table 3
Electrophysiologic drug testing

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Serum level (mg/l)</th>
<th>Response</th>
<th>Mode of induction</th>
<th>Serum level (mg/l)</th>
<th>Response</th>
<th>Mode of induction</th>
<th>Quinidine</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Disopyramide (2 mg/kg iv)</td>
<td>Disopyramide (600 mg/day)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>2</td>
<td>No RVR</td>
<td>S1/S2/S3</td>
<td>4.7</td>
<td>1 RVR</td>
<td>S1/S2/S3</td>
<td>1500</td>
</tr>
<tr>
<td>2</td>
<td>3-6</td>
<td>No RVR</td>
<td>S1/S2/S3</td>
<td>4.7</td>
<td>1 RVR</td>
<td>S1/S2/S3</td>
<td>1500</td>
</tr>
<tr>
<td>3</td>
<td></td>
<td></td>
<td>S1/S2/S3</td>
<td>4.7</td>
<td>1 RVR</td>
<td>S1/S2/S3</td>
<td>1500</td>
</tr>
<tr>
<td>4</td>
<td></td>
<td></td>
<td>S1/S2/S3</td>
<td>4.7</td>
<td>1 RVR</td>
<td>S1/S2/S3</td>
<td>1500</td>
</tr>
<tr>
<td>5</td>
<td>2.6</td>
<td>NSVT-VF</td>
<td>S1/S2/S3</td>
<td>4.7</td>
<td>1 RVR</td>
<td>S1/S2/S3</td>
<td>2250</td>
</tr>
</tbody>
</table>

NSVT = nonsustained ventricular tachycardia; RVR = repetitive ventricular response; other abbreviations as in tables 1 and 2.
mic agents prevented this induction and no recurrent arrhythmia occurred during long-term therapy with antiarrhythmic regimens found effective during electrophysiologic study.

**Features of ventricular fibrillation.** Spontaneous initiation of ventricular fibrillation was witnessed in one of our patients (No. 1) on multiple occasions. In all instances ventricular fibrillation was initiated after a few complexes of rapid polymorphic ventricular tachycardia triggered by very early R-on-T ventricular premature complexes. The latter had a left bundle branch block morphology and a left axis deviation with a very short coupling interval. These electrocardiographic features are similar to those observed at the onset of tachyarrhythmia in reports of ventricular fibrillation or torsade de pointes occurring in patients with no obvious heart disease. Therefore, it is possible that some cases of idiopathic ventricular fibrillation represent a unique entity.

**Electrophysiologic features.** Effects of programmed cardiac stimulation in patients with idiopathic ventricular fibrillation have been reported in 19 patients. Sustained ventricular tachyarrhythmias were induced by programmed ventricular stimulation in seven pa-

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**TABLE 4**

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Drug therapy</th>
<th>Clinical course (follow-up in months)</th>
<th>Last follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>AM (400 mg/d) + Q (1500 mg/d)</td>
<td>Asymptomatic (93)</td>
<td>ECG: NL, Holter: Rare VPCs, Echo: NL, RVG: NL</td>
</tr>
<tr>
<td>2</td>
<td>D (600 mg/d)</td>
<td>Asymptomatic (70)</td>
<td>ECG: NL, Holter: NL, Echo: NL, RVG: NL</td>
</tr>
<tr>
<td>3</td>
<td>AM (200 mg/d) + Q (1500 mg/d)</td>
<td>Asymptomatic (61)</td>
<td>ECG: NL, Holter: NL, Echo: NL, RVG: NL</td>
</tr>
<tr>
<td>4</td>
<td>Q (1500 mg/d)</td>
<td>Asymptomatic (22)</td>
<td>ECG: NL, Holter: NL, Echo: NL, RVG: NL</td>
</tr>
<tr>
<td>5</td>
<td>Q (1500 mg/d)</td>
<td>Asymptomatic (12)</td>
<td>ECG: NL, Holter: NL, Echo: NL, RVG: NL</td>
</tr>
</tbody>
</table>

ECG = electrocardiogram; NL = normal; Q = quinidine bisulfate; RVG = radionuclide ventriculography; VPCs = ventricular premature complexes; other abbreviations as in tables 1 and 2.
patients\textsuperscript{10, 12, 14, 15} and rapid atrial pacing in one,\textsuperscript{13} while no ventricular arrhythmias could be induced in the remaining 11 patients.\textsuperscript{11, 13, 14} In our study, consistent results were observed. A sustained poorly tolerated ventricular tachyarrhythmia was induced by programmed ventricular stimulation with up to two extra-stimuli in all five of our patients. The reason for the differences in inducibility rates of ventricular tachyarrhythmia in our study and previous reports is not evident. A possible explanation in two of our patients could have been administration of amiodarone before the initial electrophysiologic study, which might have facilitated induction of sustained ventricular tachyarrhythmias.\textsuperscript{17}

Response to antiarrhythmic drugs. In all of our five patients, a class I antiarrhythmic agent (quinidine or disopyramide) prevented the induction of sustained ventricular tachyarrhythmias and no recurrent tachyarrhythmia was observed during long-term therapy with these agents. Similar beneficial effects of class I antiarrhythmic agents were occasionally observed when they were used empirically\textsuperscript{2, 4, 6, 12} or during electrophysiologic testing.\textsuperscript{13, 15} Whether the beneficial response to class I antiarrhythmic agents with respect to preventing induction of ventricular fibrillation was a unique characteristic of our patient group is unknown, since there is no study comparing the effects of these agents in preventing inducible ventricular fibrillation in patients with and without organic heart disease. However, Rae et al.\textsuperscript{18} found that an effective drug regimen including mostly class I antiarrhythmic agents was defined for 12 of 16 (75\%) patients with coronary heart disease and inducible ventricular fibrillation. Similarly, Schoenfeld et al.\textsuperscript{19} reported that antiarrhythmic agents similar to those tested by Rae et al. prevented induction of ventricular arrhythmias in 46 of 61 (75\%) patients with documented ventricular fibrillation, most of them with coronary heart disease. Therefore, it is possible that the beneficial response to class I antiarrhythmic agents in our patients was related to characteristics of the presenting arrhythmia rather than to some unique characteristics of the patient group.

Epidemiologic considerations. In contrast to the rarity of idiopathic sudden cardiac death in Western countries, recent reports have described a syndrome of sudden death among young apparently healthy Southeast Asian individuals who have no gross cardiac abnormalities.\textsuperscript{15, 20, 21} This syndrome almost always involves male patients and occurs during sleep. In the few instances in which an electrocardiographic recording was obtained at the time of sudden death, ventricular fibrillation was documented in all.\textsuperscript{15, 21, 22} In one report,\textsuperscript{22} initiation of ventricular fibrillation followed ventricular flutter triggered by very early premature ventricular complexes, a situation similar to that observed in one of our patients (No. 1). Interestingly, quinidine prevented induction of ventricular arrhythmias by programmed ventricular stimulation in one of the patients in a previous study.\textsuperscript{15}

The patients in our study differ in several important ways from the Asian patients. These include (1) ethnic differences, (2) a female/male incidence ratio of 2:3, and (3) occurrence of cardiac arrest unrelated to sleep in any of our patients. Therefore, our population group apparently represents a spectrum of patients with idiopathic ventricular fibrillation that is different from that observed in Southeast Asia.

Limitations of study. A limitation of the present study is the small number of patients evaluated. However, to the best of our knowledge, the present series is the largest reporting results of electrophysiologic studies and pharmacologic testing in patients with idiopathic ventricular fibrillation. Another limitation of the study is that, in the absence of endomyocardial biopsy, we do not know whether the arrhythmia in our patients was actually idiopathic in nature or due to occult morphologic cardiac abnormalities.\textsuperscript{12, 23} However, it is noteworthy that both the echocardiogram and radionuclide ventriculogram remained normal during the follow-up period, which exceeds 4 years in three patients, suggesting that the arrhythmia was not the early manifestation of a latent cardiomyopathy. In addition, in the few reports of patients with apparently idiopathic ventricular fibrillation in whom an endomyocardial biopsy sample or postmortem examination was obtained, normal findings were noted in most cases.\textsuperscript{12, 13, 15, 21, 22}

In conclusion, this study has reported a unique subset of patients with idiopathic ventricular fibrillation, a rarely encountered type of arrhythmia. In all patients, a sustained ventricular tachyarrhythmia could be induced during initial electrophysiologic study and induction was prevented by a class I antiarrhythmic agent. In contrast to the severity of the presenting arrhythmia, a benign clinical course was observed during long-term therapy including these agents. However, since very little is known about the natural history of ventricular fibrillation in patients without obvious heart disease, namely how likely such patients are to have recurrent cardiac arrests, some uncertainty remains about the significance to be attached to the long-term drug "responses" observed in our study.

We thank our colleagues of the Departments of Cardiology of Hillel-Jaffe Hospital (Hadera) and Rambam Medical Center (Haifa) for referring patients 2 and 4 of the study.
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