Ventricular Tachycardia During Follow-up in Patients Resuscitated From Ventricular Fibrillation: Experience From Stored Electrograms of Implantable Cardioverter-Defibrillators

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Objective. The purpose of this study was to use the electrogram storage capabilities of the implantable cardioverter-defibrillator (ICD) to categorize any arrhythmic event during follow-up in a group of patients who had survived an episode of ventricular fibrillation (VF) and to possibly identify clinical predictors of future arrhythmic events.

Background. Little is known about the electrophysiologic characteristics of ventricular arrhythmias recurring during follow-up in survivors of VF as the sole documented arrhythmia at the time of resuscitation.

Methods. Forty patients (58 ± 10 years; 73% men; left ventricular ejection fraction 42 ± 18%; 70% with coronary artery disease) who had survived an episode of VF and subsequently received an ICD capable of intracardiac electrogram recording and storage were followed for 23 ± 11 months. In all patients, the arrhythmogenic substrate was investigated by means of programmed electrical stimulation (PES).

Results. Among the 40 patients, 41 episodes of ventricular arrhythmias were documented in 13 patients (33%): 36 episodes of ventricular tachycardias (VT) were recorded in 11 patients (28%) and 5 episodes of VF were recorded in the remaining 2 patients (5%). Age, gender, cardiac disease and left ventricular ejection fraction failed to distinguish between patients with clinical recurrences and patients without. The sensitivity, specificity and positive accuracy of PES were 29%, 63% and 46%, respectively, for prediction of ventricular arrhythmia recurrence; 45%, 70% and 36%, respectively, for prediction of VT; and 50%, 98% and 50%, respectively, for prediction of VF during follow-up.

Conclusions. In survivors of VF receiving ICD therapy, VT is the most common ventricular arrhythmia recorded on device-incorporated electrograms during follow-up. This finding, associated with the relatively well-preserved ventricular function, may account for the ability of these patients to survive at time of the index arrhythmia; the use of antitachycardia pacing as a modality to treat arrhythmia recurrences may contribute to reduce the incidence of shock during follow-up in these patients.

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At the time of an emergency intervention, victims of out-of-hospital cardiovascular collapse present with ventricular fibrillation (VF) in approximately 40% of cases, with asystole and electromechanical dissociation becoming more common the longer the intervention is delayed (1). In situations in which the time between the clinical event and the first cardiac electrogram could be kept under 4 min, the incidence of VF was found to be 95%; on the other hand, VT was observed during that time span in only approximately 1% of patients, suggesting that this arrhythmia does not generally result in cardiovascular collapse (2).

Other observations support this conclusion. A clinical history of sustained ventricular tachycardia (VT) has been documented in only 1% of cardiac arrest survivors (3). Prior to cardiac arrest, antiarrhythmic drugs were prescribed in only 9.5% of victims, mostly to suppress ventricular ectopy (3). In a selected group of patients with coronary artery disease (CAD) receiving external direct-current shock therapy within 30 s of collapse, VF and VT were the documented rhythms in approximately 90% and 10% of cases, respectively (4).

Presently, little is known about the natural history and electrophysiologic characteristics of ventricular arrhythmias recurring during follow-up in survivors of VF as the sole documented arrhythmia at the time of resuscitation. This information has mainly been prevented by 1) the tendency to report under the same category patients with different arrhythmias, such as VT, VF or syncope associated with an inducible ventricular arrhythmia during programmed electrical stimulation (PES); and 2) the systematic use of antiarrhythmic drug therapy after the index event (5–16).

Implantable cardioverter-defibrillator (ICD) therapy (17–21) has enabled us to monitor the occurrence of malignant ventricular arrhythmias during follow-up in the absence of
pharmacologic interactions. The ability to retrieve intracardiac electrograms recorded immediately before, during and immediately after intervention of the device offers new insights into the clinical history of patients receiving ICD therapy (22–24). Understanding the arrhythmic profile in patients surviving VF would be important to better stratify their risk of sudden death; it would also help to optimize the programming of intervention modalities in patients receiving ICD therapy.

The purposes of this study were 1) to use the electrogram storage capabilities of the ICD to document and categorize any arrhythmic event during follow-up in a group of patients who had survived an episode of VF as the sole documented arrhythmia at the time of resuscitation and 2) to possibly identify predictors of future arrhythmic events.

Methods

Patients. Patients were included in this study if 1) they had cardiac arrest secondary to documented VF that was not related to an acute myocardial infarction; 2) VF was the only arrhythmia documented at the time of cardiac arrest; and 3) they had an ICD that was capable of intracardiac electrogram recording and storage. All patients with a prior history of VT were excluded from this study.

Between November 1991 and January 1995, 40 consecutive patients who met the inclusion criteria were enrolled. Mean patient age was 58 ± 10 years; 29 patients (73%) were men. The underlying heart disease was CAD in 28 patients (70%), dilated cardiomyopathy in 5 and mitral valve prolapse and hypertrophic cardiomyopathy in 1 patient each. One patient had experienced VF 27 days after repair of an atrial septal defect. In four patients, no structural heart disease (idiopathic VF) could be identified by means of physical examination, 12-lead electrocardiogram (ECG), echocardiography, coronary angiography, right and left ventriculography and nuclear magnetic resonance imaging. Mean left ventricular ejection fraction (LVEF) was 42 ± 18%. Patient characteristics are shown in Table 1.

Baseline electrophysiologic study. All patients underwent PES prior to ICD implantation. Up to three extrastimuli (0.5 ms pulse width, twice diastolic threshold) were given at two right ventricular sites (apex and outflow tract) during sinus rhythm and basic drive pacing (8 beats) at cycle lengths of 640, 510 and 440 ms. Isoproterenol administration, burst ventricular pacing and left ventricular stimulation were not used in this protocol for arrhythmia induction.

### Table 1. Patient Demographics

<table>
<thead>
<tr>
<th></th>
<th>CAD (28 patients)</th>
<th>Idiopathic VF (4 patients)</th>
<th>Others (8 patients)</th>
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<tbody>
<tr>
<td>Age (yrs)</td>
<td>60 ± 8</td>
<td>54 ± 17</td>
<td>51 ± 10</td>
</tr>
<tr>
<td>Gender (male)</td>
<td>23 (82%)</td>
<td>2 (50%)</td>
<td>4 (50%)</td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>38 ± 16</td>
<td>67 ± 9</td>
<td>42 ± 17</td>
</tr>
<tr>
<td>Prior MI</td>
<td>24 (86%)</td>
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CAD = coronary artery disease; LVEF = left ventricular ejection fraction; MI = myocardial infarction; VF = ventricular fibrillation. Idiopathic VF identifies patients in whom the index arrhythmia occurred in the context of no structural heart disease.

Implantable cardioverter-defibrillator. A tiered therapy ICD was implanted in all patients via a nonthoracotomy approach. The devices used in this study (Ventak P2 1625, Ventak PRX II/III 1715/1720, Cardiac Pacemakers Inc. [CPI], St. Paul, Minnesota) are provided with arrhythmic event storing capability by means of intracardiac electrogram recording. Rate detection is performed using a bipolar electrode configuration between the distal coil and the tip electrode of the lead system (Endotak, CPI) advanced to the right ventricular apex. The defibrillation field is generated between the coil in the right ventricular apex and a second coil located in the superior vena cava.

In addition to intracardiac electrogram recording, the devices provide antibiotic pacing and two to three arrhythmia detection zones. The Ventak P2 is able to discharge 34-joule shocks in the two therapy zones, whereas the Ventak PRX provides antitachycardia pacing modalities in two to three zones. According to the characteristics of the target arrhythmia, only one detection or therapy zone was activated at the time of hospital discharge. In the present study, the cut-off rate was set at 180 beats/min or at least 20 beats/min more than the patient’s maximal sinus rate, as assessed during Holter monitoring and stress testing. All ICDs were programmed in an uncommitted mode, thus allowing a charge to be diverted in the event of spontaneous termination of the arrhythmia.

Stored intracardiac electrograms. In the Ventak–Endotak system, the two coils used for shock discharge are connected in a bipolar configuration to record intracardiac electrograms; this configuration allows good detection of atrial activation potentials and of differences in polarity and morphology between QRS complexes generated during VT and those generated during sinus rhythm or regular narrow-QRS supraventricular tachycardia (25).

The cumulative storing capacity of the devices is 2.5 min for a maximum of five sensed arrhythmic events. For each event, intracardiac electrograms can be retrieved from the 10 s preceding arrhythmia detection, the 10 s preceding device intervention and the 10 s following the intervention.

Follow-up. Defibrillators were interrogated after all clinically apparent device interventions and on routine follow-up every 2 to 3 months. In cases of device intervention, all intracardiac electrograms relating to the intervention were
analyzed. Symptoms before, during and after the intervention were assessed and classified as syncope in cases of unconsciousness and presyncope in cases of mild to severe dizziness preceding device intervention.

Arrhythmia classification. Before the analysis of stored intracardiac electrograms, a current 12-lead ECG was recorded from the patient and compared with that recorded at the time of cardiac arrest and with those available during follow-up. The intervention-related intracardiac electrogram was retrieved from the device and the following criteria for arrhythmia diagnosis were applied.

1. Ventricular fibrillation: An irregular tachycardia with regard to QRS complex polarity, amplitude, morphology and sequence, and a mean heart rate >250 beats/min.
2. Ventricular tachycardia:
   a. Monomorphic: A regular tachycardia with QRS complexes exhibiting one of the following characteristics: dissociation from clearly detectable P complexes (ventriculoatrial dissociation); a significantly different morphology and/or duration (>40 ms) compared with that observed during sinus rhythm.
   b. Polymorphic: An irregular tachycardia with regard to QRS complex polarity, amplitude, morphology and sequence, and a mean heart rate ≤250 beats/min.
3. Atrial flutter: A regular tachycardia exhibiting no differences in electrogram QRS morphology and polarity compared with sinus rhythm, and associated with clearly detectable P complexes with a fixed or variable, though regular, degree of atrioventricular (AV) conduction.
4. Atrial fibrillation: An irregular tachycardia exhibiting a variability of more than 60 ms between consecutive QRS complexes that was associated with no differences in QRS morphology compared with sinus rhythm and polarity, and with no detectable P complexes.
5. Sinus tachycardia: A regular tachycardia exhibiting P and QRS complexes identical to those recorded during sinus rhythm, with a mean rate exceeding the programmed cut-off rate.

In the present study, any stored episode of VF triggered by one to three consecutive ventricular extrabats was regarded as “primary” VF.

Device interventions delivered in the presence of P and QRS complexes identical to those recorded during sinus rhythm and with a mean rate lower than the programmed cut-off rate were regarded as failures in the sense–shock circuit and led to revision of the defibrillator system. All arrhythmic episodes that did not meet the criteria for VF or VT were regarded as nonventricular arrhythmias.

Antiarrhythmic drugs. At the time of hospital discharge following the index event, antiarrhythmic drugs were not used. If permanent or paroxysmal supraventricular arrhythmias were associated with high ventricular rate, antiarrhythmic drugs of class IV (verapamil) or AV nodal radiofrequency catheter ablation were used to prevent inappropriate discharges by the device.

Statistics. Data are presented as median and mean values ± SD, where appropriate. Comparisons were made using SPSS for Windows (version 6.1.2., SPSS). Differences of clinical baseline variables between patient groups with and without an arrhythmia recurrence were analyzed with Wilcoxon rank-sum test for continuous but not normally distributed variables (age, LVEF) and Fisher exact test for categoric variables (gender, CAD or not). Logistic regression analysis (SPSS for Windows) was applied to identify independent predictors of the presence or absence of ventricular arrhythmia detection during follow-up among these clinical baseline variables. Cycle lengths of arrhythmia recurrences were analyzed using the Wilcoxon rank-sum test. Values of p < 0.05 were considered significant.

Results

Patients were followed for 23 ± 11 months (median 22, range 1 to 45 months). Six patients died after 8 ± 8 months (median 7, range 1 to 20 months); death occurred secondary to heart failure in five patients (in one patient this was concomitant with an acute myocardial infarction, and in another patient it was perioperative due to heart failure); one patient died of a lung carcinoma.

Arrhythmia detection. During follow-up, 23 patients (57%) experienced 101 arrhythmic events (range per patient: 1 to 14 events) that led to an ICD intervention. Stored intracardiac electrograms from all episodes could be retrieved and analyzed. In all cases, the underlying arrhythmias could be diagnosed according to the criteria mentioned above. Forty-one episodes of ventricular arrhythmias were documented in 13 patients (33%); 60 episodes of nonventricular arrhythmias were documented in 13 patients (33%); and 3 patients experienced both ventricular and nonventricular arrhythmia episodes. Seventeen patients had no ICD interventions during follow-up. The distribution and cause of ICD therapies delivered during follow-up are summarized in Table 2 according to the underlying substrate.

Ventricular fibrillation. Five episodes of VF were observed in two patients (5%); these episodes accounted for 12% of all ventricular arrhythmic events. In all cases, the arrhythmia was initiated by one or two premature ventricular beats (primary VF). One patient had no structural heart disease; the other had a surgically corrected atrial septal defect. In both patients, VF was the only arrhythmia documented during follow-up. In all cases the ventricular arrhythmia was properly detected by the device and successfully terminated with the first shock.

Ventricular tachycardia. Eleven patients (28%) experienced a total of 36 episodes of monomorphic VT. In 9 patients (23%), only VT was observed (Fig. 1), which accounted for 32 of the 36 episodes (i.e., for 78% of all ventricular arrhythmic events). Mean tachycardia cycle length was 282 ± 43 ms (median 295 ms, range 180 to 330 ms; Fig. 2). Of the 11
patients, 7 had CAD, 3 had dilated cardiomyopathy and 1 had no structural heart disease. In the two other patients, four episodes of VT (i.e., 10% of all ventricular arrhythmic events) degenerated into VF. In all cases the ventricular arrhythmia was properly detected by the device and successfully terminated with the first shock.

Nonventricular arrhythmias (Table 2). Of the 60 nonventricular arrhythmic events, atrial fibrillation was responsible for 43 episodes (73%) in 9 patients and atrial flutter for 1 episode (2%) in another patient; of the former arrhythmia, 13 therapies were recorded in 1 patient during 45 min. Among six patients with recurrent discharge, control of ventricular rate was achieved by verapamil administration in four patients and by AV nodal radiofrequency catheter ablation and pacemaker implantation in two patients. Four discharges (7%) were delivered in one patient during sinus tachycardia, exceeding the programmed cut-off rate. Twelve therapies (20%) were delivered in four patients, due to failure of the sense–shock circuit in one and to lead displacement or fracture in three patients; two patients in the latter group had no structural heart disease. Inappropriate ICD therapy did not induce ventricular arrhythmia in any patient.

Clinical presentation of arrhythmic events. The two patients with primary VF experienced syncope during all five episodes. During the 36 VT episodes in 11 patients, syncope was experienced by 1 patient (VT cycle length was 240 ms). Thirteen episodes of VT (mean cycle length 252 ± 46 ms, median 250 ms, range 180 to 330 ms), including the 4 that degenerated into VF, were associated with presyncope. Neither syncope nor presyncope was experienced by any patient during the remaining 22 episodes of VT, which at 302 ± 29 ms (median 300 ms, range 230 to 330 ms) had a significantly longer mean cycle length than the VT episodes associated with presyncope (p < 0.005). The nonventricular events were not associated with syncope or presyncope. Occurrence of ventricular arrhythmias was homogeneously distributed during follow-up.

Clinical predictors of arrhythmia detection. Age, gender, presence of CAD and LVEF failed to distinguish between patients who experienced clinical recurrences and those who did not. The two patients with primary VF during follow-up were 30 and 57 years old and had LVEFs of 79% and 60%, respectively. Patients with VT had a mean age of 59 ± 13 years and a mean LVEF of 42 ± 16%. Patients with ischemic heart disease and dilated cardiomyopathy did not develop primary VF during follow-up.

Predictive value of programmed ventricular stimulation. During baseline PES, a monomorphic VT could be induced in 14 patients (35%, group 1) and VF in 7 patients (18%, group

Figure 1. Intracardiac electrograms retrieved during follow-up from one patient developing monomorphic VT. Shown are the stored intracardiac recordings of the 10 s preceding arrhythmia detection (top panel), the 10 s preceding therapy delivery (middle panel) and the 10 s following therapy delivery (lower panel). (Top panel) During one episode of atrial fibrillation, a ventricular extrabeat induces a regular VT with a heart rate of 225 beats/min. (Middle panel) Ongoing VT. (Lower panel) After shock delivery, atrial fibrillation is observed.

Figure 2. Distribution of VT cycle lengths recorded by ICD-stored electrograms during follow-up.

![Histogram of VT cycle lengths](image-url)
Table 3. Clinical Outcome of Patients Relative to Response at Programmed Electrical Stimulation

<table>
<thead>
<tr>
<th>VT at PES (14 patients)</th>
<th>VF at PES (7 patients)</th>
<th>No VA at PES (19 patients)</th>
</tr>
</thead>
<tbody>
<tr>
<td>VT during follow-up</td>
<td>5 (36)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>VF during follow-up</td>
<td>0 (0)</td>
<td>1 (14)</td>
</tr>
</tbody>
</table>

PES = programmed electrical stimulation; VA = ventricular arrhythmia; VF = ventricular fibrillation; VT = ventricular tachycardia.

2): no ventricular arrhythmias were inducible in the remaining 19 patients (47%, group 3). The clinical outcome of the general population and of patients with CAD relative to the response to PES is reported in Tables 3 and 4, respectively. During follow-up (Table 3), VT and primary VF occurred in five and zero patients, respectively, in group I, in zero and one patient, respectively, in group II, and in six and one patients, respectively, in group III. The sensitivity, specificity and positive and negative accuracy of PES were 29%, 63%, 46% and 44%, respectively, for prediction of ventricular arrhythmia recurrence, 45%, 70%, 36% and 77%, respectively, for prediction of ventricular tachycardia recurrence, 45%, 70%, 36% and 77%, respectively, for prediction of VF and 50%, 98%, 50% and 98%, respectively, for prediction of VT during follow-up.

In the five patients in whom clinical recurrence of VT was predicted during PES, the tachycardia cycle length during the electrophysiologic study did not match the one recorded by the intracardiac electrogram during follow-up.

The one patient in whom VF was predicted during PES had no structural heart disease.

**Underlying substrate and arrhythmia recurrence (Table 2).** Seven of 28 patients (25%) with CAD developed ventricular arrhythmia recurrence during follow-up; in all cases the documented arrhythmia was monomorphic VT (Table 4). Of the four patients with no underlying heart disease, two (50%) had recurrences. In one patient, the documented arrhythmia was a monomorphic VT; in the other patient, four episodes of primary VF were recorded. Among the 8 patients with other substrates, 15 ventricular arrhythmic events were observed in 4 patients (50%) during follow-up: monomorphic VT accounted for 14 events and primary VF for 1 event.

**Discussion**

**Major findings of the study.** This study shows that in 40 patients treated with an ICD who had survived an episode of cardiovascular collapse secondary to VF and in whom VF had been the only ventricular arrhythmia ever documented until that event, VT rather than VF was the prevailing recurrent arrhythmia. During a follow-up of approximately 2 years, primary VF was observed in only 5% of patients, whereas VT was observed in 28%. Although episodes of primary VF were always associated with syncope, this hemodynamic consequence accompanied only 1 of 36 episodes of VT. Roughly one-third of VT episodes, including four that degenerated into VF, were accompanied by presyncopal attacks, whereas the remainder of VT episodes did not cause hemodynamic impairment. The VTs of the latter category had a significantly longer cycle length (corresponding to a mean heart rate of 199 beats/min; number of episodes, 21) than the VTs resulting in either syncope or presyncope (mean heart rate 239 beats/min; number of episodes, 13).

The role of VT as the most frequent arrhythmia responsible for ambulatory sudden collapse and death has been previously outlined in heterogeneous groups of patients undergoing 24-h Holter monitoring at the time of the terminal event (26). However, in contrast to the present study, most patients (approximately 95%) had not experienced ventricular arrhythmias causing cardiac arrest prior to the monitored terminal event. Therefore, the findings of this study contribute to further define the clinical profile of survivors of VF as the sole documented arrhythmia at time of resuscitation.

Recent reports have pointed to different pathophysiologic and electrophysiologic characteristics between patients presenting with VF and those presenting with regular VT. The former tend to have a higher ejection fraction (27) and a lower incidence of late potentials on signal-averaged electrograms (28), and are less likely to have regular VT induced during PES (7,13,27–30). Furthermore, induced VTs during PES tend to be faster and more often polymorphic in patients with documented VF compared with patients in whom only VT has been documented (13,29,30). The characteristics of the population in this study confirm these observations, as suggested by an LVEF above 40% and a 35% incidence of induced monomorphic VT.

**Characteristics of recurrent ventricular arrhythmias.** During a follow-up of approximately 2 years, 33% of patients experienced 1 or more episodes of ventricular arrhythmia. Notably, 85% of these patients had a VT and only 15% a primary VF; in the former group, 11% of VTs degenerated into VF before ICD treatment was initiated. These data extend and complete previous observations. In a study performed with ICDs during 14 months of follow-up, Raftt et al. (30) studied 55 patients with VF as the sole documented arrhythmia at time of resuscitation who were treated with ICDs not provided with electrogram storage capability; they found 18% and 10% incidences of recurrent VT and VF, respectively. Referral modalities, a longer follow-up and the ability to recognize VT degenerating into VF likely account for the different incidence and distribution of ventricular arrhythmias in the present study.

Symptoms during ventricular arrhythmias varied depending on the cycle length of the underlying event. Syncope or...
Clinical predictors of arrhythmia detection. No single clinical parameter was found to predict the risk of arrhythmia recurrence; however, it is of note that the two patients with primary VF during follow-up were both young, had a normal LVEF and did not have ischemic heart disease or dilated cardiomyopathy as the underlying substrate.

Role of PES. Programmed ventricular stimulation proved to be a poor predictor of both recurrence and type of ventricular arrhythmias. A ventricular arrhythmia was inducible in 53% of patients; this finding is in agreement with previous reports (7,13,27–29). Only 29% of patients who had a ventricular arrhythmia inducible in the electrophysiology laboratory also presented with an arrhythmia during follow-up. In the five patients in whom a VT recurrence was predicted by PES, the cycle length of the tachycardia in the two conditions did not match. In addition, the one patient in whom VF was predicted during PES had no structural heart disease. On the other hand, 47% of patients who did not have any arrhythmia inducible in the electrophysiology laboratory developed a ventricular arrhythmia during follow-up.

Underlying substrate and arrhythmia recurrence. Recurrence of ventricular arrhythmic events was observed in 25% of patients with CAD, 50% of patients without structural heart disease and 50% of patients with underlying substrates other than CAD. In the former group of patients, VT was the only documented arrhythmia. Although a prior myocardial infarction was documented in 86% of cases, it is of note that patients in this study exhibited a ventricular substrate poorly amenable to sustained and stable reentry. Suggestive of this were 1) the clinical documentation of VF (index event), 2) the low VT inducibility rate (35%) at PES, and 3) the fast rate of the VT episodes recorded by stored electrograms during follow-up.

A similar mechanism may explain the occurrence of monomorphic VT during follow-up in other patients of this study, such as one of four patients without organic heart disease and three of eight patients with underlying substrates other than CAD. Primary VF occurred in one patient without organic heart disease and in one patient with an underlying substrate other than CAD.

Inappropriate discharges. In this study, the incidence of inappropriate discharges was high. Supraventricular tachycardias or system failure caused one or more ICD discharges in 33% of patients, a figure in agreement with a recent review on the limitations and late complications associated with ICD therapy (32). The absence of concomitant antiarrhythmic drug therapy at the time of hospital discharge certainly played a role in the reported incidence of inappropriate discharges in this series. Notably, 77% of patients with inappropriate discharges were otherwise free of any ventricular arrhythmias during follow-up. It is likely that, in the absence of telemetry, a significant number of discharges caused by supraventricular tachycardias or system failure would be interpreted as appropriate; this may account for the somewhat lower recurrence rate of ventricular arrhythmias in this study compared with previously published data (21).

Clinical implications. The present data show that primary VF is not a common arrhythmic event in survivors of cardiac arrest in whom VF is the sole documented arrhythmia at the time of resuscitation. These patients represent a selected group among those experiencing sudden death in the general population. Although several factors may account for their ability to survive such an event, it is likely that the modality of tachycardia onset and perpetuation, as documented in this study during recurrent ventricular arrhythmias, plays a role (32).

We found that clinical recurrences in these patients are characterized mostly by episodes of regular, though fast, monomorphic VT, which, if sustained, would likely lead to hemodynamic instability (32). On the basis of these findings, one may speculate that at least some of these patients could survive an episode of cardiac arrest because of the stability of the initial arrhythmia associated with a relatively well-preserved ventricular function. Should this be the case, the arrhythmia recorded at the time of resuscitation would represent a degeneration of the initial arrhythmia. On the other hand, patients with a similar or even better ejection fraction but with primary VF, as well as those with a poor ventricular function in the presence of a fast regular VT, would be less likely to survive the arrhythmic episode.

Limitations. Although ICD allows analysis of the clinical outcome free of influences on the electrophysiologic substrate by antiarrhythmic agents, follow-up in patients receiving ICD does not reflect the true natural history. Exacerbation of ventricular arrhythmias during the postoperative period after implantation (33), as well as the induction of new arrhythmias by pacemaker interventions or inappropriate shocks (34), have been reported and may influence the occurrence of arrhythmic events during follow-up.

Classification of ventricular arrhythmias in the short time between the onset of detection and redetection prevents a distinction between sustained and nonsustained events. It also does not allow us to establish the duration of the monomorphic VT in the event that it eventually degenerates into VF.

Conclusions. In patients who have survived VF as the sole documented arrhythmia at time of resuscitation and are currently receiving ICD therapy, VT is by far the most common ventricular arrhythmia recorded on device-incorporated electrograms during follow-up. Though regular, VT is generally fast and not predictable by electrophysiologic investigation. This finding, associated with the relatively well-preserved ventricular function exhibited by these patients, may account for their ability to survive at the time of the index arrhythmia. Preliminary data obtained from similar patient populations (35) suggest that the use of antitachycardia pacing (36) as a modality to treat arrhythmia recurrences may contribute to reduce the incidence of shocks during follow-up. Confirmation in larger series of the observations made in the present study is recommended to guide the programming modality in survivors of VF.
References


