Implantable Defibrillator Event Rates in Patients With Unexplained Syncope and Inducible Sustained Ventricular Tachyarrhythmias

A Comparison With Patients Known To Have Sustained Ventricular Tachycardia

Neil P. Andrews, BMBS, MRCP, Richard I. Fogel, MD, FACC, Gemma Pelargonio, MD, Joseph J. Evans, MD, FACC, Eric N. Prystowsky, MD, FACC

Indianapolis, Indiana

OBJECTIVES

To assess the clinical significance of inducible ventricular tachyarrhythmias among patients with unexplained syncope.

BACKGROUND

Induction of sustained ventricular arrhythmias at electrophysiology study in patients with unexplained syncope and structural heart disease is usually assigned diagnostic significance. However, the true frequency of subsequent spontaneous ventricular tachyarrhythmias in the absence of antiarrhythmic medications is unknown.

METHODS

In a retrospective case-control study, the incidence of implantable cardiac defibrillator (ICD) therapies for sustained ventricular arrhythmias among patients with unexplained syncope or near syncope (syncope group, n = 22) was compared with that of a control group of patients (n = 32) with clinically documented sustained ventricular tachycardia (VT). Sustained ventricular arrhythmias were inducible in both groups and neither group received antiarrhythmic medications. All ICDs had stored electrograms or RR intervals. Clinical variables were similar between groups except that congestive cardiac failure was more common in the syncope group.

RESULTS

Kaplan-Meier analysis of the time to first appropriate ICD therapy for syncope and control groups produced overlapping curves (p = 0.9), with 57 ± 11% and 50 ± 9%, respectively, receiving ICD therapy by one year. In both groups, the induced arrhythmia was significantly faster than spontaneous arrhythmias, but the cycle lengths of induced and spontaneous arrhythmias were positively correlated (R = 0.6, p < 0.0001). During follow-up, three cardiac transplantations and seven deaths occurred in the syncope group, and two transplantations and five deaths occurred in the control group (36-month survival without transplant 52 ± 11% and 83 ± 7%, respectively, p = 0.03).

CONCLUSIONS

In patients with unexplained syncope, structural heart disease and inducible sustained ventricular arrhythmias, spontaneous sustained ventricular arrhythmias occur commonly and at a similar rate to patients with documented sustained VT. Thus, electrophysiologic testing in unexplained syncope can identify those at risk of potentially life-threatening tachyarrhythmias, and aggressive treatment of these patients is warranted. (J Am Coll Cardiol 1999;34:2023–30) © 1999 by the American College of Cardiology

The development of syncope in patients with structural heart disease is associated with an increased risk of sudden death (1). Understandably, many clinical investigators believe that in a significant proportion of these patients, syncope represents the first presentation of a potentially life-threatening ventricular tachyarrhythmia that fortuitously and spontaneously resolved. Programmed ventricular stimulation is a useful technique for reliably reproducing previously documented sustained ventricular tachycardia in patients with coronary artery disease and to a lesser extent in those with cardiomyopathy (2,3). Although electrophysiologic testing has proved less sensitive in detecting bradycardiacarrhythmias, because of its potential value in detecting tachyarrhythmias, it has become established in the investigation of unexplained syncope in patients with heart disease (4–7). Indeed, induction of clinically significant ventricular tachyarrhythmias in patients with unexplained syncope and structural heart disease is now a class 1 indication for implantation of an implantable cardiac defibrillator (ICD) (8).
Electrophysiologic testing in unselected patients with unexplained syncope predominantly yields negative results and among such low risk patients, the potential for false positive results is greatest (8–12). Including induction of ventricular flutter or fibrillation with three extrastimuli may also increase false positive results (13–15), although induction of such arrhythmias by two or less extrastimuli may be a more clinically relevant end point (16). Thus, reported results of electrophysiologic testing in syncope patients vary widely according to patient selection, the pacing protocol used and the definition used for clinically relevant inducible tachyarrhythmias. Furthermore, even when electrophysiologic study (EPS) guided drug therapy is used, patients with inducible ventricular arrhythmias have a worse prognosis than their noninducible control subjects (4,12,17–21). Thus, using conventional techniques and therapies, a valid assessment of the true value of electrophysiologic testing in patients with unexplained syncope has been fraught with difficulties.

The development of ICDs with facility for recording electrogoms or RR intervals when combined with appropriate patient selection and a refined programmed stimulation protocol allows, for the first time, a valid assessment of the significance of sustained ventricular tachycardia (VT) initiated at electrophysiologic testing in patients with unexplained syncope. In this study, we examined the incidence during follow up of appropriate ICD therapies (defibrillations) among patients with syncope, structural heart disease and inducible sustained ventricular tachyarrhythmias. As a control group, we used patients with documented sustained VT and inducible sustained VT who were treated with ICD implantation.

METHODS

Study patients. In patients who receive an ICD, our standard policy is to avoid routine administration of antiarrhythmic drug therapy unless needed to control frequent arrhythmias. This policy allowed us to perform a retrospective case-control study comparing the incidence of appropriate ICD therapies, in the absence of antiarrhythmic drug therapy, between patients with unexplained syncope (syncope group) with those who had documented spontaneous sustained VT (control group).

Cases and controls were identified from our register of ICD implants. All patients who had a device with capability of storing electrograms or RR intervals implanted before January 1, 1996 were evaluated for inclusion in the study. Of these 175 patients, 30 potential cases with a history of unexplained syncope and 93 potential control subjects with a history of documented sustained VT were identified. Six syncope and 47 control patients were excluded because they were receiving antiarrhythmic drug therapy at the time of their initial clinical event, diagnostic EPS or during follow-up prior to their first appropriate ICD therapy. A further two cases and 14 control subjects were excluded because of noninducibility at initial EPS. Thus, the study groups consisted of 22 syncope patients and 32 control subjects.

Our policy for the use of EPS in the clinical investigation of unexplained syncope has restricted the use of EPS to those patients with structural heart disease who had first been clinically assessed by an electrophysiologist (E.N.P., J.J.E., R.I.F.). In addition, all patients underwent at least 48 hours of telemetry monitoring prior to EPS. Other cardiac workup included cardiac catheterization in 91% of the syncope group and 100% of control subjects, nuclear stress tests in those who did not undergo cardiac catheterization and in all patients whose catheterization predated the EPS by > six months. LV ejection fraction was calculated from radionuclide angiography (41% of syncope patients and 38% of control subjects), echocardiography or from cardiac catheterization data.

Electrophysiology study. Studies were performed in the postabsorptive state, after withdrawal of all antiarrhythmic drug therapy for ≥5 half-lives (3). Patients were sedated with 1 to 4 mg of intravenous midazolam. Two to three multipolar electrode catheters were percutaneously inserted under local anesthesia through the femoral vein and advanced under fluoroscopic guidance to the high right atrium, His bundle area and right ventricle. Spontaneous intervals were obtained as well as corrected sinus node recovery times (syncope only), atrioventricular node and His-Purkinje conduction and refractoriness, and evaluation of inducible supraventricular tachycardia and VT (10). The programmed ventricular stimulation protocol utilized up to three extrastimuli at two paced cycle lengths starting at the RV apex, then testing the RV outflow tract if no sustained ventricular arrhythmias were induced. Attempts to reproduce the sustained ventricular arrhythmia were always made unless defibrillation had already been required. The Multi-center Unsustained Tachycardia Trial (MUSTT) definitions of ventricular tachyarrhythmias, and the clinical relevance of their induction, were adopted (16). In brief, induction of sustained (>30 s or requiring termination because of loss of consciousness) monomorphic VT by ≤3 ventricular extrastimuli and induction of sustained ventricular flutter or fibrillation by ≤2 ventricular extrastimuli were considered clinically relevant tachyarrhythmias. Ventricular flutter was defined as a rapid monomorphic VT with a cycle...
length of \( \leq 220 \) ms and no identifiable isoelectric interval between QRS complexes.

**Follow-up.** July 31, 1996 was prospectively determined as the last day of data collection for the principal analysis. A repeat analysis was performed in December 1998; by this time, one patient in the syncope group and four in the control group were unavailable for follow-up. Stored ECGs and RR intervals from ICDs were interpreted by one investigator (E.N.P.) who was blinded to the grouping of the patient. Published guidelines on the interpretation of stored data were adopted (22–24). Specifically, tachyarrhythmias that were preceded by syncope or near syncope, whose onset was associated with an abrupt change in configuration, whose RR intervals varied by \( \leq 30 \) ms once initiated or that had a mean RR interval \( \geq 240 \) ms were defined as appropriate. In indeterminate cases, data such as the cycle length of tachyarrhythmia (CLs) of previously documented atrial and ventricular arrhythmias, patient interviews, Holter or treadmill recordings or lead integrity testing were sought. A conservative approach was adopted: where reasonable doubt existed, the episode was categorized as inappropriate.

Cardiac transplantation and nonsudden deaths were recorded and analyzed separately but did not count as arrhythmia events. Sudden death was defined as death within 1 h of onset of cardiac symptoms. All patients in the study groups were included in the Kaplan-Meier analysis of ICD events and those who died, underwent transplantation or completed follow-up without receiving an appropriate ICD therapy were left censored. Electrocardiographically documented episodes of sustained VT that occurred at rates below the programmed cutoff for the device were counted as events. Histories of syncope or near syncope unrelated to ICD episodes were actively sought from patient charts and interviews.

**Statistical methods.** Baseline clinical and electrophysiologic characteristics were compared by Fisher exact test or by unpaired \( t \) tests. Kaplan-Meier curves were compared by the log rank method. The baseline characteristics listed in Table 1, the number of extrastimuli required for induction and the cycle lengths of the induced rhythm were all analyzed for prediction of events among the syncope group by Mantel Cox survival comparisons (this identified only two characteristics with \( p \) values \( \leq 0.1 \), thus no multivariate analysis was performed). Data comparing VT cycle lengths in cases and control subjects were “disconnected” and thus a one-way analysis of variance (ANOVA) was used. Comparisons of CL data within each group were made by one-way repeated measures ANOVA (control group) and paired \( t \) test (syncope group). Correlations were tested by Pearson’s method. \( p \) values \( < 0.05 \) were assigned significance. Two-tailed \( p \) values were exclusively used. Mean values \(( \pm \text{SEM})\) were used unless otherwise indicated.

**RESULTS**

**Patient characteristics.** There were 22 patients in the syncope group and 32 in the control group. Both groups had similar ages (69 ± 2 and 66 ± 1 years, respectively) and men predominated (82% and 78% respectively, Table 1). In the syncope group 17 had syncope and 5 had near syncope. Those with near syncope were all sufficiently concerned
about their symptoms to seek medical attention within 48 h of the episode. Twenty-two of the control subjects (69%) had syncope or near syncope at the time of their spontaneous sustained VT. The vast majority of both groups had underlying coronary artery disease (Table 1). The exceptions were two cases of idiopathic dilated cardiomyopathy in each group, one case of hypertensive cardiomyopathy in the syncope group, one case of valvular heart disease and one with hemodynamically unstable and drug-resistant idiopathic VT in the control group. Although the extent of coronary disease and left ventricular ejection fraction was similar in both groups, a history of congestive cardiac failure was more common in the syncope group (Table 1).

Prior to ICD implantation, EPS guided serial drug testing was more commonly performed in control subjects than cases (Table 2). The median number of failures to respond to drug therapy was 1 (range 1 to 3) for both groups. ICDs with stored electrograms (as opposed to stored RR intervals) were more frequent in the control group, whereas transvenous systems were more common among the syncope group (Table 2).

### Electrophysiology testing

The syncope and control groups had identical His to ventricular conduction time (HV) intervals and minimum CLs that maintained 1:1 AV conduction (Table 2). The corrected sinus node recovery time for the syncope group was normal in all but one patient (866 ms) who subsequently had sustained VT during follow-up (CL, 219 ms) associated with syncope. There were no significant differences in the number or coupling intervals of extrastimuli required for arrhythmia induction between the groups (Table 2).

The CLs of induced monomorphic tachycardias were significantly shorter in the syncope group than in control subjects (Table 2). In both groups, the VT induced at EPS was shorter (11 ± 2%) than spontaneous episodes (Figs. 1A and 1B). Despite these differences in average VT CLs, the CLs of induced monomorphic tachycardias correlated positively with those of subsequent spontaneous monomorphic tachycardias (R = 0.6, p < 0.0001 for both groups combined). This correlation was particularly evident in the syncope group (Fig. 2).

### Follow-up

Complete follow-up data were available for the first 18 months after ICD implantation and 90% of patients.

### Table 2. Electrophysiologic and ICD Characteristics

<table>
<thead>
<tr>
<th></th>
<th>Syncope (n = 22)</th>
<th>Control (n = 32)</th>
<th>p-Value</th>
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</thead>
<tbody>
<tr>
<td>Sinus and AV node function</td>
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<td></td>
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<tr>
<td>Sinus CL</td>
<td>817 ± 30</td>
<td>819 ± 27</td>
<td>0.96</td>
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<tr>
<td>cSNRT</td>
<td>365 ± 59</td>
<td></td>
<td></td>
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<tr>
<td>AH</td>
<td>73 ± 4</td>
<td>81 ± 4</td>
<td>0.19</td>
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<tr>
<td>HV</td>
<td>59 ± 4</td>
<td>59 ± 3</td>
<td>0.90</td>
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<tr>
<td>AVN 1:1</td>
<td>385 ± 16</td>
<td>401 ± 13</td>
<td>0.45</td>
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<tr>
<td>PVS induction characteristics</td>
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<tr>
<td>paced CL (S1–S1)</td>
<td>477 ± 17</td>
<td>469 ± 15</td>
<td>0.71</td>
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<tr>
<td>S1–S2 coupling interval</td>
<td>264 ± 3</td>
<td>279 ± 12</td>
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<tr>
<td>S2–S3 coupling interval</td>
<td>216 ± 5</td>
<td>228 ± 5</td>
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<td>S3–S4 coupling interval</td>
<td>205 ± 6</td>
<td>212 ± 5</td>
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<td>induction with S2, n (%)</td>
<td>0 (0)</td>
<td>1 (3)</td>
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<td>induction with S3, n (%)</td>
<td>12 (55)</td>
<td>12 (38)</td>
<td>0.27</td>
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<tr>
<td>induction with S4, n (%)</td>
<td>10 (45)</td>
<td>19 (59)</td>
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<td>Serial EP guided drug testing*</td>
<td>3 (14)</td>
<td>13 (41)</td>
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<td>ICD</td>
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<tr>
<td>Travenous system</td>
<td>22 (100)</td>
<td>23 (72)</td>
<td>0.007</td>
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<td>Noncommitted shocks</td>
<td>22 (100)</td>
<td>32 (100)</td>
<td>1.00</td>
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<tr>
<td>Stored electrograms†</td>
<td>11 (50)</td>
<td>26 (81)</td>
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<td>Arrhythmias (CL)‡</td>
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<tr>
<td>Presenting MVT-S</td>
<td>N/A</td>
<td>315 ± 10</td>
<td></td>
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<tr>
<td>Induced monomorphic arrhythmia</td>
<td>256 ± 7</td>
<td>282 ± 7</td>
<td>0.047§</td>
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<tr>
<td>MVT-S, n (%)</td>
<td>19 (86)</td>
<td>30 (94)</td>
<td>0.39</td>
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<td>ICD arrhythmia</td>
<td>287 ± 18</td>
<td>298 ± 10</td>
<td>0.47§</td>
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Values are in milliseconds (mean ± SEM) unless otherwise stated. cSNRT = corrected sinus node recovery time; AVN 1:1 = minimum paced cycle length that maintained 1:1 AV conduction; PVS = programmed ventricular stimulation; S1–S2 = first extrastimulus following train of 8 paced beats; S2–S3 = second extrastimulus; S3–S4 = third extrastimulus; MVT-S = monomorphic sustained ventricular tachycardia.

*All patients who underwent electrophysiology guided drug therapy failed to respond to at least one drug.
†Devices without stored electrograms had stored RR intervals.
‡Includes only monomorphic tachycardias. Proportions were compared by Fisher exact test and numerical data by t tests, except where indicated (§one-way ANOVA).
were followed up for 34 months. Median length of follow-up (including nonsurvivors) was 34 (range, 2 to 60) and 46 (7 to 82) months in syncope and control groups, respectively. Kaplan-Meier analysis of the time to defibrillator therapy for syncope and control groups produced overlapping curves ($p = 0.9$) (Fig. 3). The proportion of syncope patients and control subjects receiving an appropriate defibrillator therapy (Fig. 4) by 12 months was 57 ± 11% and 50 ± 9%, respectively, and by 24 months, these proportions were 63 ± 11% and 63 ± 9%, respectively. Of the 14 patients in the syncope group with an ICD VT, 8 had syncope or near syncope and an additional patient received therapy while asleep.

Two patients in the syncope group had syncope that was not associated with an ICD event. One of these was clinically diagnosed as having hypoglycemia and the other was diagnosed as having neurocardiogenic syncope. The latter patient also had syncope that immediately preceded an appropriate ICD defibrillation. Three patients in the syncope group had ventricular flutter or fibrillation induced by two extrastimuli. Two of the patients subsequently received appropriate ICD defibrillations. In each case, syncope preceded defibrillation and the CLs recorded were 195 and 219 ms, respectively. Among the syncope group, the only baseline clinical or electrophysiology characteristics that were at all useful in predicting subsequent occurrence of an appropriate ICD therapy were NYHA class III or IV and presence of bifascicular block. Both were only marginally

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**Figure 1.** A, Comparison of CLs of monomorphic tachycardias induced at EPS and recorded by the ICD during follow-up for the syncope group (squares). B, Comparison of CLs of monomorphic tachycardias recorded at clinical presentation, induced at EPS and recorded by ICD during follow-up for control patients (circles). Monomorphic tachycardia includes sustained ventricular flutter and sustained monomorphic VT. Solid bars represent means ± SEM.

**Figure 2.** Plots of relation between cycle length of monomorphic tachycardias induced at EPS and subsequent spontaneous episodes recorded by the ICD in syncope group (A, squares) and control group (B, circles). Monomorphic tachycardia includes sustained ventricular flutter and sustained monomorphic VT.

**Figure 3.** Kaplan-Meier "survival" estimates for time to first appropriate ICD event in cases with unexplained syncope and control subjects with documented sustained VT. The number of patients remaining in the study at six monthly intervals are indicated below the abscissa.
significant for a subgroup analysis (p = 0.04 and p = 0.03, respectively).

Syncope and control survival curves remained overlapping when cases with only a history of near syncope were excluded (p = 0.9 for difference between curves), when patients with ventricular flutter or fibrillation induced at EPS were excluded (p = 0.8) or when those who had previously failed serial drug testing had been excluded (p = 0.8). Indeed, among the control subjects (syncope group data could not be analyzed as only three patients underwent serial drug testing), failure of antiarrhythmic drug therapy to suppress induced VT did not predict subsequent occurrence of an appropriate ICD therapy (p = 0.4).

During follow-up, three cardiac transplantations and seven deaths (five cardiac, no sudden deaths) occurred in the syncope group compared with two transplants and five deaths (all cardiac, one sudden) among the control subjects (36-month survival without transplant, 52 ± 11% and 83 ± 7%, respectively, p = 0.03). Excluding transplantations, the 36-month actuarial survival rate was 60 ± 12% in the syncope group and 89 ± 6% in the control group (p = 0.06).

DISCUSSION

This study provides three lines of evidence to support the diagnostic value of induced sustained VT at EPS in patients

Figure 4. Surface ECGs and intracardiac electrogram (RV, right ventricular outflow tract) recorded at induction of monomorphic VT (CL, 225 ms) during baseline EPS in a patient with unexplained syncope (A). Stored ICD intracardiac electrogram recorded between proximal and distal shocking electrodes (B) in the same patient two and a half months later. This spontaneous tachycardia (rate 265 beats/min, CL 226 ms) resulted in syncope and was terminated by a 34-J shock.
with unexplained syncope: 1) the incidence of VT during follow-up in the syncope patients was identical to control patients with previously documented spontaneous VT; 2) nearly 40% of syncope patients had spontaneous recurrence of syncope as a result of VT that was recorded and terminated by the ICD; and 3) the rate of the induced VT positively correlated with that subsequently recorded by the ICD.

Although this study was not designed to compare mortality between the two groups, it is noteworthy that the actuarial incidence of nonsudden death and cardiac transplantation at three years was significantly higher in the syncope (48%) than control group (17%). This occurred despite effective ICD therapy that prevented all but one sudden death. The poorer prognosis in the syncope group was consistent with a significantly higher incidence of congestive heart failure at baseline.

The absence of antiarrhythmic drug therapy in all study patients at the time of their baseline spontaneous event, EPS and follow-up provided a unique opportunity to examine the relationship between VT induced at EPS and subsequent spontaneous episodes recorded by the ICD. This showed that the induced VT rates were on average significantly faster than spontaneous episodes, but the rates were positively correlated. Although a correlation of QRS configuration cannot be performed between the induced and subsequent spontaneous VT (ICD), the comparative VT rates suggest that the VT at EPS was not merely a marker for a group prone to sustained VT, but actually identified the cause of the syncope. Actuarial analysis of the incidence of appropriate ICD therapies in this study suggests that approximately 70% of patients with unexplained syncope and inducible VT will eventually have spontaneous sustained VT.

Comparison with previous studies. Previous studies using similar pacing protocols to this present study have reported induction of sustained ventricular arrhythmias in 21% to 50% of patients with unexplained syncope and structural heart disease (11,12,25,26). Patients with negative results from electrophysiologic testing have a very low incidence of sudden death (1% per year) (4,12). Patients with positive test results have a poorer prognosis, which is due to both an increased risk of nonsudden cardiac death and sudden death that occurred despite antiarrhythmic therapy (17–21). The reported high mortality rate in these studies likely reflects the severity of underlying heart disease; indeed, a multivariate analysis suggested that in this population, ejection fraction was of primary importance in predicting survival (20). These reports are consistent with our data in that we found a high overall mortality even though there were no sudden deaths in our group of patients with unexplained syncope treated by ICD implantation.

The incidence of appropriate shocks reported in this study was higher than previously reported for unselected recipients of ICDs who frequently received antiarrhythmic drugs (27). However, in patients with documented VT who received ICD therapy without antiarrhythmic drugs, the actuarial incidence of VT at 12 months was 50% (24), a comparable incidence to this present study. This suggests that our control group was a representative sample of patients with documented VT. In a recent study of patients with unexplained syncope, there was a 22% one-year probability of an ICD shock (28). However, a significant proportion of patients were treated with antiarrhythmic drug therapy that may have prevented recurrent VT. Another recent study reported a 66% one-year probability of an appropriate ICD therapy in a small subgroup with unexplained syncope and inducible monomorphic VT (29).

Study limitations. The vast majority of patients included in this study had coronary artery disease with left ventricular dysfunction. Since programmed ventricular stimulation is less sensitive and less specific in patients with nonischemic cardiomyopathies (3), the results of this study cannot necessarily be extrapolated to such patients. Similarly, our study used a maximum of three extrastimuli and excluded patients with ventricular flutter or fibrillation induced by more than two extrastimuli. Results from this study should not be applied to the use of more aggressive pacing protocols.

Despite restricting the study to patients with ICDs that had stored electrograms or RR intervals, we cannot assume that all apparently appropriate ICD events were due to ventricular tachyarrhythmias. As the interpreting electrophysiologist was blinded to group allocation, any such errors should have affected cases as much as control subjects. Including only ICDs with event storage capabilities and excluding patients receiving antiarrhythmic drugs improved the ability of the study to estimate the true incidence of spontaneous tachyarrhythmias, but resulted in a relatively small sample size. Finally, the successful cardioversion of suspected VT should not be equated with prevention of sudden death.

Clinical implications. In patients with coronary artery disease, impaired left ventricular function and unexplained syncope, induction of sustained VT at EPS reliably identifies those at risk of potentially life-threatening ventricular tachyarrhythmias. In these patients, aggressive treatment directed toward the prevention of such arrhythmias is warranted. Since they may also have an increased risk of nonarrhythmic death, clinical management should include aggressive treatment of their underlying cardiac disease.

Reprint requests and correspondence: Dr. Eric N. Prystowsky, The Care Group, LLC, 8333 Naab Rd, Indianapolis, Indiana 46260.

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