Nonsustained ventricular tachycardia in patients with coronary artery disease: role of electrophysiologic study

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ABSTRACT  Sixty-two consecutive patients with chronic coronary artery disease referred for evaluation of nonsustained ventricular tachycardia (VT) underwent electrophysiologic studies. Sustained VT was induced by one to three ventricular extrastimuli in 28 patients (45%). Therapy was guided by the results of electrophysiologic testing in 44 patients: 19 patients without inducible sustained VT received no antiarrhythmic therapy, and 25 patients with inducible sustained or symptomatic nonsustained VT received therapy guided by the results of electrophysiologic studies. The results of electrophysiologic studies were ignored by physicians for a second group of 18 patients: four had inducible sustained VT but no antiarrhythmic therapy, and 14 had inducible sustained or nonsustained VT and received antiarrhythmic therapy not guided by results of electrophysiologic testing. After a mean follow-up period of 28 months, 11 patients had died suddenly. Seven of the 11 patients who died suddenly had inducible sustained VT. Three of 44 patients in the group receiving therapy guided by electrophysiologic studies died suddenly versus eight of 18 in the group receiving therapy not guided by electrophysiologic studies (p = .001). Only one of 19 patients without inducible sustained VT who were not treated experienced sudden death. Two of four patients with inducible sustained VT who did not receive antiarrhythmic therapy died suddenly. Multivariate analysis of the relationship of induced arrhythmias, left ventricular ejection fraction, site of myocardial infarction, history of syncope, or type of antiarrhythmic therapy to outcome revealed a greater than twofold increased risk for sudden cardiac death in patients whose therapy was not guided by results of electrophysiologic study. We conclude that patients with coronary artery disease and nonsustained VT who are asymptomatic and are without inducible sustained VT have a relatively low probability of sudden death and do not require antiarrhythmic therapy. Patients with inducible sustained VT are at a significantly increased risk for sudden cardiac death, and in this group therapy that prevents induction of sustained VT is associated with a lower rate of sudden cardiac death than empiric therapy.


INDICATIONS for treatment of nonsustained ventricular tachycardia (VT) in patients with chronic coronary artery disease are still debated. Likewise, the optimum method to choose antiarrhythmic therapy for this entity has not been defined. Although the detection of nonsustained VT by ambulatory electrocardiographic monitoring after a recent (within 1 month) myocardial infarction is associated with an increased risk for sudden cardiac death, such patients are equally likely to die nonarrhythmic cardiac deaths.1-4 Thus, spontaneous nonsustained VT is not a specific predictor of sudden cardiac death, and it does not necessarily indicate the potential for development of sustained ventricular tachyarrhythmias. Similar observations have been made in ambulatory patients with chronic coronary disease but without recent infarctions.5 We have previously reported that in approximately 40% of patients with chronic coronary artery disease and nonsustained VT, programmed stimulation will induce sustained
VT. We and others have suggested that the patients with inducible sustained VT are at increased risk for sudden cardiac death. The purpose of the present study was to analyze outcome retrospectively in a large group of patients with chronic coronary artery disease who presented with nonsustained VT to determine which patients are most likely to benefit from prophylactic antiarrhythmic therapy, and to evaluate whether antiarrhythmic therapy guided by electrophysiologic testing results in improved survival compared with that of patients receiving antiarrhythmic therapy guided by other means.

**Patient population.** We studied 62 patients with coronary artery disease who presented with nonsustained VT. No patient had suffered a cardiac arrest remote from an acute myocardial infarction. The mean age of these patients was 60 ± 10 years. Coronary artery disease was demonstrated at cardiac catheterization in 53 of the 62 patients. All but three patients had experienced a previous myocardial infarction that occurred a mean of 43 months before electrophysiologic study (range, 2 months to 15 years). Of the 59 patients with prior infarction, two had non-Q wave infarctions, 27 had anterior infarctions, and 30 had inferior infarctions. The left ventricular ejection fraction was 41 ± 15% (mean ± SD); 33 patients had ejection fractions of 0.40 or less. Sixteen patients had left ventricular aneurysms demonstrated at cardiac catheterization and/or radionuclide angiography.

Nonsustained VT was detected on “routine” Holter recordings in 33 asymptomatic patients (53%). Nonsustained VT was discovered during evaluation of palpitations in 11 patients (18%) and syncope in 18 patients (29%). Nonsustained VT occurred in each case in the absence of antiarrhythmic drugs. The arrhythmia was not discovered during exercise testing in any case.

**Methods**

**Definitions.** Sustained VT was tachycardia lasting longer than 30 sec or in the electrophysiology laboratory requiring termination in less than 30 sec because of hemodynamic decompensation.

Nonsustained VT was that 3 beats to 30 sec in duration at rates of 120 beats/min or more.

Inducible VT was that reproducibly initiated by programmed electrical stimulation. To be considered inducible the tachycardia had to have morphologic characteristics similar to those of the spontaneous nonsustained VT. Because of difficulty obtaining multiple standard electrocardiographic leads of nonsustained VT in most cases, we considered VT to be inducible if the spontaneous and induced VT were both uniform or both polymorphic.

Sudden cardiac death was that witnessed to be instantaneous or unexpected death occurring during sleep.

**Protocol for electrophysiologic study.** All studies were performed in patients in the nonfasted, postabsorptive state after all antiarrhythmic drugs had been discontinued at least 48 hr. Incremental atrial pacing at a rate of 100 beats/min to the rate that resulted in atrioventricular block was performed, followed by programmed atrial stimulation with single atrial depolarizations at one or more drive cycle lengths at the high right atrium. Right ventricular stimulation was performed at the apex and outflow tract with one to three extrastimuli at two or more drive cycle lengths (most often 600 and 400 msec) and incremental pacing to a minimal cycle length of 250 msec. Bipolar stimuli were delivered with a pulse width of 1 msec at twice diastolic threshold. Single extrastimuli scanned diastole in 10 msec decrements until sustained VT replicating the spontaneous arrhythmia or ventricular refractoriness was reached. Double extrastimuli were then delivered with the S2 placed 40 msec beyond the effective refractory period of the ventricle and the S2-S3 interval initially equal to the S1-S2 interval. Each stimulus was decremented by 10 msec intervals. If double extrastimuli at both the right ventricular apex and outflow tract failed to induce sustained ventricular tachycardia, triple extrastimuli were delivered to all patients. The protocol with triple extrastimuli began with S2 placed 40 msec beyond the effective refractory period and the S2-S3 and S2-S4 intervals equal to the S1-S2 interval. Each extrastimulus was decremented by 10 msec.

Electrocardiographic leads I, aVf, and V1, together with intracardiac electrograms from the atrium, atioventricular junction, and one or more right ventricular sites, were recorded simultaneously on a multichannel oscilloscope (Electronics for Medicine VR16). Intracardiac electrograms were filtered at 30 to 500 Hz. Data were recorded simultaneously onto magnetic tape (Honeywell Model 5600) and an ink-jet recorder (Siemens-Elema Mingograf) at paper speeds of 100 mm/sec.

The end point for programmed stimulation in the baseline state was reproducible initiation of sustained VT resembling the spontaneous tachycardia. If sustained VT was induced patients underwent serial drug testing as described previously. The aim of programmed stimulation during testing of type I drugs was initiation of 10 or fewer complexes of VT for patients with sustained VT induced in the baseline study, and no VT for those with only nonsustained VT induced at the baseline study. The stimulation protocol was the same for the control and drug tests.

Based on the results of previous studies that suggested that patients with inducible sustained VT who presented with only nonsustained VT were at increased risk for sudden cardiac death, we recommended that patients receive antiarrhythmic therapy only if sustained VT was induced in the baseline state, or, if careful assessment revealed that nonsustained VT was likely to be responsible for syncope or palpitations and the induced nonsustained VT correlated with the patient’s symptoms. Assignment of antiarrhythmic therapy was to be based on the results of electrophysiologic testing. Patients with sustained ventricular tachycardia that remained inducible on type I drugs were to be treated with amiodarone. Twenty-seven of 28 patients with inducible sustained VT underwent testing with a type I antiarrhythmic agent. In 24 patients these agents were tested intravenously, and in all but two patients the drug that the patient was receiving at hospital discharge was tested after oral administration. In 44 patients therapy was guided by the results of electrophysiologic tests; in 18 the primary care physicians did not follow these recommendations. Four patients with inducible sustained VT did not receive long-term antiarrhythmic therapy. Fourteen patients received antiarrhythmic therapy that was not guided by the results of electrophysiologic study: doses lower than those recommended on the basis of the electrophysiologic test were given (one patient), agents proven ineffective in preventing induction of sustained VT were administered (nine patients), or asymptomatic patients without inducible sustained ventricular tachycardia received antiarrhythmic therapy (four

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patients). Among these 14 patients at baseline electrophysiologic study, six had inducible sustained VT, four had only nonsustained VT induced, and four had no inducible VT.

Patients were followed at the Hospital of the University of Pennsylvania or by telephone contact at least every 6 months. At each contact the patient's current medications and history of any arrhythmic events were reviewed.

**Data analysis.** Continuous data are described as the mean ± SD or median as appropriate. Statistical analysis of data was performed with Fisher's exact test or two-tailed t-test as appropriate. Multivariate analysis of survival data was then performed with the Cox proportional-hazards model. Variables analyzed included follow-up in months, status at end of follow-up (alive, sudden cardiac death, nonsudden death), ejection fraction (dichotomized to values ≤40% versus those >40%), results of baseline electrophysiologic tests (sustained VT induced, no sustained VT induced), patient age, location of myocardial infarction, and history of syncope. Univariate analysis of the relationship between each factor and sudden cardiac death was initially performed. Following this the significant variables (p < .10) were fitted to a model that was then used to construct risk ratios for sudden cardiac death for the significant univariate predictors. Based on the Cox model, Kaplan-Meier estimates of survival functions were constructed.

**Results**

**Characteristics of spontaneous nonsustained VT.** Most patients had infrequent episodes of spontaneous tachycardia: 28 had three or less per day documented (of these, 16 had less than one per day). Thirty-four had more than three episodes per day (of these only five patients had >10 per day). The maximum duration of spontaneous episodes of nonsustained VT documented was fairly short: the median number of complexes was nine (range three to 99). Fifty-five patients had uniform nonsustained VT and seven had polymorphic VT. The mean cycle length of nonsustained VT was 359 ± 79 msec.

**Results of programmed stimulation.** Nineteen patients had no inducible VT, in 15 patients only nonsustained VT was induced (figure 1), and 28 patients sustained VT was induced. The morphology of tachycardia was uniform in 24 of the 28 patients with inducible sustained VT. Sustained VT was induced in two patients by single ventricular extrastimuli, in 10 patients by double ventricular extrastimuli, and in 16 patients by triple ventricular extrastimuli (figure 2). The mean cycle length of sustained VT induced by single and double extrastimuli (306 ± 77.3 msec) was slightly longer than that induced by triple extrastimuli (266 ± 57 msec; p = NS). There was a trend toward lower
mean left ventricular ejection fraction in patients with inducible sustained VT (37%) compared with that in those in whom only nonsustained VT was induced (42%) or no VT was induced (49%), but these differences did not reach statistical significance. Ten of 28 patients with inducible sustained VT had left ventricular aneurysms versus six of 34 without left ventricular aneurysms (p = NS).

Twenty-seven of the 28 patients with inducible sustained VT underwent a total of 51 electrophysiologic studies on antiarrhythmic medication: intravenous procaainamide, 23; oral procaainamide, four; oral quinidine, seven; disopyramide, five; lidocaine, one; procaainamide plus lidocaine, one; procaainamide plus phenytoin, one; phenytoin, one; disopyramide plus phenytoin, one; ethmozine, one; propranolol, three; and amiodarone, three. Twelve patients were drug responders (programmed stimulation induced 10 or fewer beats of VT). Five of the 12 responders had ejection fractions greater than .40 versus two of 15 nonresponders (p = NS). Eleven of 15 patients in whom only nonsustained VT was induced underwent serial drug testing. Seven of these 11 responded to drugs; i.e., VT was no longer inducible.

Treatment modality as assigned. Based on the intention to treat principle as outlined in the Methods section, 44 patients received therapy guided by the results of electrophysiologic studies while 18 received nonelectrophysiologically guided therapy. The groups were similar to each other with respect to a number of clinical and arrhythmic variables (table 1). Serial drug testing was performed in 27 patients in the electrophysiologically guided group and 12 patients in the nonelectrophysiologically guided group. Overall, 15 patients in the electrophysiologically guided group versus three patients in the nonelectrophysiologically guided group responded to type I agents (p = NS). In the electro-

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physiologically guided group, inducible sustained VT became noninducible in 11 of 18 patients (10 in response to type I agents, one on amiodarone). In the nonelectrophysiologically guided group, one of 10 patients with inducible sustained VT became noninducible in response to a type I agent (p = .01 vs the electrophysiologically guided group). Discharge medications in the electrophysiologically guided group included procainamide in four, quinidine in three, disopyramide in four, disopyramide plus phenytoin in one, β-blockers in three, and amiodarone in seven. Electrophysiologically guided surgery (map-guided subendocardial resection) plus coronary artery bypass grafting was performed in two patients (both had noninducible VT after surgery) and coronary artery bypass grafting alone was performed in two patients. Twenty-two patients in the electrophysiologically guided group were discharged off all antiarrhythmic medication.

Medications in the nonelectrophysiologically guided group included procainamide in two, quinidine in six, phenytoin in one, tocainide in one, verapamil in one, and amiodarone in two. Coronary artery bypass surgery was performed in one patient and four patients received no antiarrhythmic therapy.

Results of follow-up. No patient was lost to follow-up. After a mean follow-up of 28 months, 18 deaths had occurred. Eleven were sudden and seven occurred nonsuddenly. The interval from infarction to electrophysiologic study (and treatment) in the 11 patients experiencing sudden death ranged from 2 to 72 months (mean 46.7 months). Four of the patients who died suddenly had left ventricular aneurysms. Three of the patients suffering sudden death had a history of syncope. Seven of the sudden deaths occurred in patients with inducible sustained VT. In all cases, the inducible sustained VT had a uniform morphology. In four of seven patients sustained VT was induced by triple extrastimuli. The mean cycle length of induced sustained VT was 268 ± 73 msec in the seven patients who experienced sudden death vs 259 ± 48 msec in the 21 patients without subsequent sudden death (p = NS). Three of the 15 patients in whom only nonsustained VT was induced died suddenly compared with one of the 19 patients without inducible VT (p = NS).

The mean left ventricular ejection fraction in the 11 patients dying suddenly was 37% vs 45% in the patients without sudden death (p = NS). However, sudden death occurred in one of 29 patients with ejection fractions greater than 0.40 vs 10 of 33 patients with ejection fractions of 0.40 (p < .01).

Three of the sudden deaths occurred in the electrophysiologically guided group, while eight occurred in the nonelectrophysiologically guided group (p = .001; figure 3). Two patients suffered syncope without documented arrhythmias during follow-up (11 and 26 months after study). Neither was receiving antiarrhythmic medication at the time, and one had inducible sustained VT and one had nonsustained VT only. No patient receiving amiodarone died suddenly.

Multivariate analysis of outcome by the Cox proportional-hazards model revealed that the data fit the model with a chi-square value = 17.92 (p = .0005), resulting in a proportional reduction in error due to the model of 37.5%. Three variables achieved moderate statistical significance (p < .10) with respect to relationship to sudden cardiac death. Patients in the nonelectrophysiologically guided group who did not receive therapy had a 2.5-fold increased risk for sudden cardiac death (p = .0087), those in the nonelectrophysiologically guided group who received therapy had a 1.6-fold increased risk (p = .0268), and patients with ejection fractions of 0.40 or less had a 2-fold increased

FIGURE 3. Relationship between treatment modality assigned, inducibility of sustained VT, and subsequent sudden cardiac death. EPS = electrophysiologic study; VT-S = sustained VT; SCD = sudden cardiac death.
risk (p = .0577). Kaplan-Meier estimates of survival based on this model revealed high survival rates for the patients in the electrophysiologically guided group regardless of ejection fraction and for those in the nonelectrophysiologically guided group with ejection fractions greater than 0.40. However, both the treated and untreated patients receiving nonelectrophysiologically guided therapy who had ejection fractions of 0.40 or less had significantly lower projected survival (figure 4).

Discussion

This study extends previous observations by us and others regarding the role of electrophysiologic studies in the management of patients with nonsustained VT. Earlier smaller studies suggested that the presence of inducible VT in patients with left ventricular dysfunction due to chronic coronary artery disease identified a subset of patients at increased risk for sudden cardiac death.7,8 Our earlier studies suggested that sustained VT was induced only rarely in patients with cardiomyopathy and that programmed stimulation was not likely to be a useful predictor of sudden death in this patient population.11 Based on experience with patients who have spontaneous episodes of sustained VT in association with chronic coronary artery disease, we suspected that reproducible induction of sustained VT indicated a substrate placing these patients at risk for spontaneous episodes of sustained VT. Therefore, we decided that patients who presented with nonsustained VT in whom sustained VT could be reproducibly initiated should receive antiarrhythmic therapy, while those patients without inducible sustained VT should not require specific antiarrhythmic therapy to prevent sudden cardiac death.

Physicians caring for almost one-third of the present group (29%) did not follow recommendations based on the results of electrophysiologic studies. This then formed the basis for a retrospective analysis of outcome based on whether antiarrhythmic therapy was administered according to the results of electrophysiologic studies or other means (e.g., ambulatory electrocardiographic monitoring). Therapy was not randomized in the present study. However, there was no

**FIGURE 4.** Kaplan-Meier estimates of survival related to ejection fractions and antiarrhythmic therapy assigned in patients with nonsustained VT. EPS = patients whose therapy was guided by electrophysiologic studies; non-EPS = patients whose therapy was not guided by the results of electrophysiologic study; + Rx = patients receiving antiarrhythmic therapy; No Rx = patients not given antiarrhythmic therapy in each group.
significant difference in the degree of left ventricular dysfunction, prevalence of aneurysms, extent of coronary artery disease, prevalence of inducible sustained ventricular tachycardia, history of syncope, or rate and duration of spontaneous ventricular tachycardia in the group receiving therapy guided by results of electrophysiologic studies and these variables in the group that did not. In a further effort to search for sources of potential bias, we analyzed outcome with regard to patients with a history of syncope. Similar proportions of patients in the electrophysiologically and nonelectrophysiologically guided groups had a history of syncope, and syncope did not predict subsequent sudden cardiac death.

The optimum protocol for programmed stimulation in this patient population has been debated. Some studies have used only up to two extrastimuli, presumably in hopes of avoiding induction of “nonclinical” arrhythmias. However, in this study, over half (four of seven) of the patients with inducible sustained VT who died suddenly required triple extrastimuli for induction. These patients would have been “missed” by less “aggressive” stimulation protocols. Therefore, we believe that it is appropriate to employ three extrastimuli in this patient population, as long as one makes an attempt to distinguish nonspecific (i.e., most polymorphic) from presumably clinically relevant (and therefore treatable) induced tachycardias.

The results of this study support those of earlier studies demonstrating that asymptomatic patients with nonsustained VT in whom sustained VT cannot be induced are at relatively low risk for sudden cardiac death and therefore only a minority are likely to benefit from long-term antiarrhythmic therapy. Four sudden deaths occurred in patients without inducible sustained VT. Three of these patients were receiving empiric antiarrhythmic therapy, thus raising the possibility that these drugs exerted a proarrhythmic effect. Seven of the 11 patients experiencing sudden death had inducible sustained VT. Five of these seven patients were in the nonelectrophysiologically guided therapy group. Of note, two of the four patients in the nonelectrophysiologically guided group who had inducible sustained VT and did not receive therapy died suddenly, and three of six patients in this group with inducible sustained VT who were receiving nonelectrophysiologically guided (empiric) therapy died suddenly. In contrast, only two of 18 patients with inducible sustained VT in the electrophysiologically guided group receiving therapy died suddenly. Perhaps more striking are the data from the estimated survival curves. These estimates of survival, based on a model constructed from the data, suggest that electrophysiologically guided therapy is associated with similar survival rates in patients with ejection fractions greater and lower than 0.40. In contrast, patients in the nonelectrophysiologically guided group with ejection fractions of less than 0.40 had significantly lower estimated survival than the patients in the nonelectrophysiologically guided group with ejection fractions greater than 0.40 and in the electrophysiologically guided therapy group with ejection fractions of 0.40 or less. The actual rate of sudden death was low in patients with ejection fractions greater than 0.40, suggesting that patients with ejection fractions of 0.40 or less are more likely to benefit from an electrophysiologic approach to therapy. The greater survival in patients with ejection fractions greater than 0.40 was probably not only due to a greater likelihood of response to therapy since there was no significant difference in drug response rates in patients with ejection fractions of 0.40 or less and those with ejection fractions greater than 0.40.

We conclude that patients with coronary artery disease and nonsustained VT who do not have inducible sustained VT have a relatively low risk of sudden death and many probably do not require antiarrhythmic therapy. Patients with inducible sustained VT have significantly increased risk for sudden cardiac death, and in this group, therapy that suppresses inducible sustained VT is associated with a lower rate of sudden cardiac death than therapy that does not prevent induction of sustained VT. Patients whose inducible sustained VT is not suppressed by antiarrhythmic therapy are at increased risk of sudden cardiac death.

**Limitations of study.** Interpretation of the results of programmed stimulation in patients with nonsustained VT is limited by the inability to record multiple simultaneous electrocardiographic leads during episodes of spontaneous tachycardia in most patients. Thus, the ability to demonstrate concordance between spontaneous and induced arrhythmia is limited. The foundations of clinical electrophysiology are based on the assumption that arrhythmias induced in the laboratory replicate a patient’s spontaneous arrhythmias. We are now attempting to extend the boundaries of clinical electrophysiology by using the results of programmed stimulation to predict a risk for occurrence of arrhythmias in the future. Others have ignored morphologic characteristics of spontaneous nonsustained VT, and argued that in patients with nonsustained VT all that programmed stimulation need demonstrate is the ability to induce sustained VT without regard for its morphology. However, it seems likely that this would result in a less accurate prediction of subsequent clini-
cal events. We have attempted to circumvent these difficulties in part by considering tachycardias inducible only if both the spontaneous and induced arrhythmia had uniform morphology or both were polymorphic. Polymorphic tachycardias may appear uniform if only a single electrocardiographic lead is recorded. Thus, we attempted to obtain multiple electrocardiographic leads of the spontaneous tachycardia. However, this was possible in only a minority of the patients. In spite of this limitation, it would appear that programmed stimulation has clinical utility when applied in this manner to patients with nonsustained VT and chronic coronary artery disease.

The patients with inducible sustained VT in the electrophysiologically guided group were more likely to have induction prevented by antiarrhythmic therapy than the patients in the nonelectrophysiologic group. Thus, the differences in survival may be due in part to the ability of electrophysiologic testing to identify a group at high risk of sudden cardiac death, rather than an ability to modify outcome. We believe that the lower risk of sudden death in the patients who responded to antiarrhythmic drugs was due to the fact that they were discharged on a regimen that prevented induction of sustained ventricular tachycardia, rather than an intrinsically lower risk of sudden death. Recent data suggest that patients with spontaneous sustained VT in whom the VT becomes noninducible in response to drugs have significant risk of recurrence if drugs are stopped.14

The other major limitation of this study is, as described above, the fact that therapy was not administered in a randomized, controlled fashion. However, as previously noted, the analysis has sought to limit potential sources of bias, and our findings do suggest that therapy guided by the results of electrophysiologic testing may result in significantly improved outcome compared with empiric therapy.

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