Nonsustained Ventricular Tachycardia in Ambulatory Patients: Characteristics and Association with Sudden Cardiac Death

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Thirty-seven patients with nonsustained ventricular tachycardia ($\geq$ triplets) were identified retrospectively from a population of 518 consecutive patients referred for 24-hour Holter monitoring and studied to determine the prognostic significance of this finding. Ten of these 37 patients suffered sudden cardiac death during a mean follow-up of 19 $\pm$ 5 months. Nine of 19 patients with a diagnosis of congestive cardiomyopathy or history of congestive heart failure died suddenly compared to only one of the other 18 patients. No other patient data were predictive of sudden death. Remarkably, no characteristic of the ventricular tachycardia including beats per episode, episodes per day, rate, prematurity index (RR'/QT) of the initiating beat, or the occurrence of associated arrhythmias was important prognostically. Thus, patients with congestive cardiomyopathy or congestive heart failure and nonsustained paroxysmal ventricular tachycardia are at a high risk for sudden death and are ideal candidates for prophylactic interventions.

SUDDEN CARDIAC DEATH is a leading cause of mortality in the United States (1-3), and ventricular fibrillation is believed to be the commonest arrhythmia associated with sudden cardiac death (4-8). Some investigators (4, 6) have emphasized the need to identify those patients at high risk for ventricular fibrillation and who are therefore candidates for prophylactic therapy. A grading system of ventricular ectopy has been proposed in an effort to identify such a population (6, 9).

Isolated ventricular premature depolarizations are associated with coronary artery disease and sudden cardiac death (10-12), but they are too widely prevalent to be specific indicators of a particularly high-risk population (3, 6, 13). Frequent ventricular premature depolarizations, multiform ventricular depolarizations, and ventricular couplets have all been associated with increased risk of sudden death (2, 3, 5, 6, 11-22). However, at least in the setting of acute infarction, the prevalence of these warning arrhythmias may not be related to the incidence of subsequent ventricular fibrillation (23, 24). Closely coupled ventricular premature depolarizations (that is, "R-on-T") have also been correlated with risk of sudden death in some studies (3, 4, 7, 25, 26), while other studies have questioned the significance of this association. Certainly early coupled ventricular premature depolarizations are not required for the initiation of ventricular fibrillation (14, 18, 27).

Ventricular tachycardia ($\geq$ triplets) is a common arrhythmia in the setting of acute ischemia (5, 6, 28, 29) and at least in that context has a well-recognized potential to deteriorate into ventricular fibrillation (30, 31). In the ambulatory setting, however, much less is known

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about its prognostic significance. Nonsustained ventricular tachycardia may be a common rhythm disturbance in ambulatory patients, including those who are active and asymptomatic (11, 13, 18, 28). Although there may be an increased risk of sudden death associated with runs of ventricular premature depolarizations (2, 14), some reports have suggested that this risk has been overestimated (11, 29, 32). In addition, comparatively little is known about the electrocardiographic characteristics of ventricular tachycardia in the ambulatory setting, and in particular whether any specific characteristics are associated with the subsequent development of ventricular fibrillation.

Our study has examined the characteristics and prognostic significance of paroxysmal nonsustained ventricular tachycardia recorded by long-term ambulatory monitoring techniques in 518 consecutive patients monitored for various clinical indications, with a mean follow-up of 19 months. Each episode of ventricular tachycardia was analyzed for rate, duration, prematurity index, and associated ventricular ectopy. Patient records were reviewed for diagnoses, clinical course, and incidence of sudden death. Thirty-seven patients were identified with nonhemodynamically significant paroxysmal, nonsustained ventricular tachycardia. None of these patients had symptoms of syncope, near syncope, chest pain, or dyspnea in association with episodes. We report on the outcome of this population and the characteristics of the observed ventricular tachycardia.

Materials and Methods

Twenty-four-hour long-term electrocardiographic tapes recorded by the Clinical Electrocardiographic Laboratory at the Hospital of the University of Pennsylvania on 518 consecutive patients over a 2-year period were retrospectively reviewed for the presence of nonsustained paroxysmal ventricular tachycardia. Primary cardiac diagnoses included ischemic heart disease in 183 patients (35%); valvular heart disease in 82 patients (16%); and other cardiovascular diagnoses in 29 patients (6%), including cardiomyopathy and hypertensive cardiovascular disease. Two hundred twenty-four patients (43%) had no known structural heart disease. Long-term electrocardiographic recordings were ordered by the patient's personal physicians for a variety of clinical indications. Major indications for monitoring included palpitations in 223 patients (43%); chest pain in 129 patients (25%); syncope in 125 patients (24%); dizziness in 122 patients (23%); and dyspnea in 62 patients (12%); many patients had more than one presenting complaint. Monitoring was primarily done with simultaneous two-channel recordings of precordial leads V1 and V5. Tapes were read by a trained analyst using a computerized scanner and were interpreted by an independent research service (Anthropometrics, Incorporated, Haddonfield, New Jersey, using the Avionics 660A computerized scanner and Avionics 445 recorder; Avionics Biomedical Division, Del Mar Engineering Laboratories, Irvine, California), whose average error rate for analyst-computer-determined arrhythmia frequency compared to an independent real-time analysis was about 7%. Samples of designated ventricular premature depolarizations on all recordings by both systems were printed out and further verified by a cardiologist. Every episode of ventricular tachycardia was printed out in real-time for verification and analysis. Accompanying each recording was a patient history log that included symptoms, activity, and medications taken during the 24-hour period.

For the purposes of this study, nonsustained paroxysmal ventricular tachycardia was defined as the occurrence of three or more consecutive ventricular premature depolarizations that were self-terminating without specific therapeutic intervention and were without associated hemodynamic sequelae, including syncope or presyncope, acute weakness, diaphoresis, or dyspnea—and thus would not necessarily require treatment for a clinical indication. The awareness of palpitations was not in itself considered significant if ventricular tachycardia was otherwise well tolerated. Recordings done within 7 days of an acute myocardial infarction were excluded from the study. Patients with non-self-terminating ($\geq$ 1 minute) ventricular tachycardia were also excluded.

Each electrocardiographic tape was reviewed manually to determine each of the following: the number of episodes of ventricular tachycardia per 24 hours, the number of ventricular premature depolarizations per episode, the rate of the ventricular tachycardia, the prematurity index of the initiating beat of each episode (RR^1/QT), the number of hours with greater than 10 ventricular premature depolarizations, and the presence of multiform ventricular premature depolarizations, ventricular couplets, ventricular premature depolarizations with "R-on-T" (defined as having a prematurity index < 1), and supraventricular arrhythmias. Corresponding entries in the patient's history log were noted. In all cases, charts were reviewed and data on the patient's age, sex, cardiac diagnoses, and history of congestive heart failure tabulated. Follow-up data were obtained by reviewing the patient's records, contacting the primary physician, and, when necessary, contacting the patient or the patient's family directly.

For the purposes of this study, congestive heart failure was defined as a history of appropriate symptoms requiring digoxin or diuretic therapy, or both, a diagnosis of congestive heart failure made by the patient's physician, or of cardiomegaly confirmed by chest roentgenogram or echocardiogram. In patients with valvular heart disease, other evidence in addition to pulmonary congestion (if mitral stenosis) or cardiomegaly (if mitral or aortic regurgitation) was required to diagnose congestive heart failure. Coronary artery disease was defined by a history of documented myocardial infarction, typical angina pectoris, or a positive coronary angiogram, or a combination of these. Sudden cardiac death was defined as cardiac death within 6 hours of the onset of symptoms, similar to the definition of Vismara and colleagues (5). Because of the retrospective nature of the study, we did not feel that we could reliably determine the time of onset of symptoms any more precisely than within 6 hours. Data on drug therapy were not subjected to analysis because it was impossible to document patient compliance and to be sure of medication regimens throughout the follow-up period.

STATISTICAL ANALYSIS

Patients were divided into two groups, with Group I consisting of those patients who died suddenly and Group II those patients who did not. Groups I and II were compared to each other and to the total group by the following statistical methods (33). Ages were compared using a Student's *t*-test for difference in means. The Z test for difference in proportions was used in comparing historical symptoms and grades of associated ectopy on the recordings. The Wilcoxon rank sum test for same distribution and shift in location was used to analyze the properties of the ventricular tachycardia. This test is based on median values and therefore reduces the impact of a long tail on the distribution curve. With the size of the population in this study, assuming a type-I error of 0.05 and a type-II error of 0.10, two populations would have to be distributed with peaks of their distribution curves at 0.25 and 0.75 around 0.50 in order to be detected. Lesser differences could be missed in the current analysis.

Results

Thirty-seven patients with nonsustained nonhemodynamically significant ventricular tachycardia were identified from the 518 patients reviewed. Follow-up data were

obtained in 100% of patients, with a range of follow-up in survivors of 12 to 30 months (mean, 19 months ± 5 [SD]). Ten of the 37 patients suffered sudden death in the follow-up period; seven of the sudden deaths occurred within 3 months of the detection of the ventricular tachycardia. These 10 patients comprised Group I. The remaining 27 patients who did not die suddenly comprised Group II. Two of these patients died in the follow-up period, one with a cerebrovascular accident and one with severe congestive heart failure secondary to cyanotic congenital heart disease.

CARDIAC DIAGNOSIS

The patient's cardiac diagnoses are summarized in Table 1. Although there was no significant difference in the prevalence of coronary artery disease or valvular heart disease between the two groups, there was a statistically significant greater prevalence, at the $P < 0.05$ level, of congestive cardiomyopathy in Group I than in Group II. Nine patients in Group I but only four in Group II had congestive cardiomyopathy.

PATIENT PROFILES

Table 2 outlines the profile of the patients. The mean age of the population was 56.4 years, with a range from 15 to 79 years. There was no difference in the mean ages between the two groups. There were 21 males and 16 females, again with no difference between the groups.

A history of congestive heart failure was present in 51% of the study population. There was a highly significantly greater incidence of a history of congestive heart failure in Group I than in Group II. Whereas nine of 10 patients in Group I had a history of congestive heart failure, only 10 of 27 patients in Group II had a similar history ($P < 0.005$). Thus of the 37 patients in the study, 19 had a history of congestive heart failure and 18 did not. Nine of these 19 patients with nonsustained ventricular tachycardia on a Holter recording and a history of congestive heart failure died suddenly in the follow-up period, whereas only one of 18 patients with nonsustained ventricular tachycardia but no history of congestive heart failure died suddenly ($P < 0.005$).

Remarkably, none of the 37 patients with nonsustained ventricular tachycardia had symptoms of syncope, near syncope, chest pain, or dyspnea in association with their arrhythmias, and therefore none had to be excluded from this analysis. Thus, there was no difference in the incidence of symptoms reported in association with the occurrence of nonsustained ventricular tachycardia in the two groups. Only three of 37 patients, one in Group I and two in Group II, reported palpitations at a time when ventricular tachycardia was recorded on the tape.

Table 1. Cardiac Diagnoses

	Group I ($N = 10$)	Group II ($N = 27$)	P Value
Coronary artery disease	2	8	NS*
Valvular heart disease	3	10	NS
Congestive cardiomyopathy	5	4	<0.05
Other disorders	0	5	NS

* NS = not significant.

Table 2. Patient Profiles

	Group I ($N = 10$)	Group II ($N = 27$)	P Value
Age			
Mean, *yrs*	59.4	55.3	NS*
Range, *yrs*	15–79	25–76	
Sex			
Males, *no.*	6	15	NS
Females, *no.*	4	12	
History			
Patients with congestive heart failure, *no.*	9	10	<0.005
Patients with palpitations, *no.*	7	18	NS

* NS = not significant ($P > 0.05$).

CHARACTERISTICS OF THE VENTRICULAR TACHYCARDIA

One hundred three episodes of nonsustained ventricular tachycardia were recorded in the 37 patients (mean 2.8 episodes per patient, range 1 to 11.) Sixty-six episodes (64%) were three beats in duration, 16 episodes (16%) had four beats, nine episodes (9%) had five beats, and 12 episodes (11%) had more than five beats. The characteristics of the ventricular tachycardia in the two groups are outlined in Table 3. The number of episodes of ventricular tachycardia per 24-hour recording was not significantly different between the two groups (mean 2.4 episodes per patient in Group I and 2.9 episodes per patient in Group II.) Thirteen patients had one episode of nonsustained ventricular tachycardia, 12 had two episodes, six had three episodes, and six had more than three episodes.

There was no difference in the duration of each episode of ventricular tachycardia in patients in the two groups. The mean duration of each episode in patients in Group I was 3.8 beats compared with 4.1 beats per episode in patients in Group II. The mean number of beats of ventricular tachycardia per 24 hours was 9.0 in Group I and 12.0 in Group II (not significant [NS]). Figure 1 details the rates of the ventricular tachycardia in the patients in the two groups. The mean rate in Group I was 160 ± 36 (SD) beats per minute and was not significantly different from the mean rate in Group II, which was 146 ± 29 (SD) beats per minute. The range of rates was nearly identical in the two groups.

ONSET OF THE VENTRICULAR TACHYCARDIA

The prematurity index (RR^1/QT) for the initiating beat of each episode of nonsustained ventricular tachycardia is shown in Figure 2. The mean prematurity index per episode of ventricular tachycardia was 1.35 in Group I (range 0.56 to 3.93) and 1.37 in Group II (range 0.63 to 2.31). This difference was not significant. The mean prematurity index per patient in Group I was 1.39 and in Group II was 1.44 (NS). Only 19 of 103 episodes were initated by a beat with a prematurity index < 1, again with no difference between Groups I and II. Only seven

Table 3. Characteristics of the Ventricular Tachycardia

	Group I (N = 10)	Group II (N = 27)	P Value
Episodes of ventricular tachycardia/patient, *mean no. (range)*	2.4 (1–5)	2.9 (1–11)	NS*
Beats/episode of ventricular tachycardia, *mean no. (range)*	3.8 (3–6)	4.1 (3–26)	NS
Beats of ventricular tachycardia/24 h, *mean no. (range)*	9.0 (5–16)	12.0 (3–41)	NS

* NS = not significant.

of these 37 patients had any episodes of nonsustained ventricular tachycardia with a prematurity index < 1; three of these were in Group I and four in Group II (NS).

CHARACTERISTICS OF ASSOCIATED VENTRICULAR PREMATURE DEPOLARIZATIONS

Associated ventricular premature depolarizations were seen on the recordings of all 37 patients. The mean number of hours with ventricular premature depolarizations present was 19.2 (range 3 to 24) in Group I and 12.4 hours (range 1 to 24) in Group II (NS). The mean number of hours with greater than 10 ventricular premature depolarizations per hour was 13.6 (range 5 to 24) in Group I and 13.4 (range 0 to 24) in Group II (NS). Table 4 details the number of patients with different grades of associated ectopy. There was no difference between Groups I and II in the number of patients with > 10 ventricular premature depolarizations per hour, multiform ventricular premature depolarizations (six of 10 versus 15 of 27), ventricular couplets (eight of 10 versus 21 of 27), or ventricular premature depolarizations with prematurity index < 1 ("R-on-T", two of 10 versus six of 27). Associated supraventricular tachyarrhythmias (atrial fibrillation, atrial flutter, or paroxysmal atrial tachycardia) were commonly seen in both groups (five of 10 in Group I and 14 of 27 in Group II [P = NS]).

Discussion

The present study shows that the occurrence of nonsustained episodes of ventricular tachycardia (≥ triplets) detected by 24-hour long-term electrocardiographic recordings is associated with considerable risk of sudden death (10 of 37 patients) during a mean follow-up period of 19 months (that is, mortality 17% per year). This risk was most marked (nine of 19 patients, mortality 29% per year) for patients with a diagnosis of congestive cardiomyopathy or history of congestive heart failure. Sudden death during the follow-up period occurred, however, in only one of 18 patients for those patients with ventricular tachycardia detected on Holter monitoring but without known heart failure. Other demographic or patient data were not helpful in identifying patients at highest risk for sudden death.

Characteristics of nonsustained ventricular tachycardia were also analyzed in detail. Remarkably no specific characteristic including the number of episodes of ventricular tachycardia per day, number of beats per episode, rate of ventricular tachycardia, prematurity index of the initiating beat, or occurrence of associated arrhythmias identified those patients who subsequently suffered sudden death.

PREMATURITY INDEX

We recorded 103 episodes of nonsustained ventricular tachycardia in these 37 ambulatory patients. The mean prematurity index was 1.37. Only 19 of the 103 episodes (18%) were initiated by a ventricular premature depolarization with a prematurity index < 1. The prematurity index per episode and per patient was no different in those patients who subsequently died suddenly compared with those who did not, suggesting that the prematurity index was not an important prognostic variable.

The significance of isolated ventricular premature depolarizations with close coupling intervals has been debated in the literature (3, 7, 15, 16, 18, 21, 23-27, 29, 31, 32, 34). Although some studies have supported the concept that closely coupled ventricular premature depolarizations have a bad prognostic implication (3, 7, 21, 31), other studies have questioned this association (15, 16, 27). De Soyza and colleagues (16) documented ventricular tachycardia in 27 of 52 patients with acute myocardial infarction. They noted that the prematurity index of the ventricular premature depolarizations that initiated ventricular tachycardia was no different from the prematurity index of the other ventricular premature depolarizations in the same patient that did not initiate ventricular tachycardia, and also was no different from the prematurity index of ventricular premature depolarizations in the group of patients who did not develop ventricular tachycardia at all.

The finding that most episodes of nonsustained ventricular tachycardia in our selected population were initiated by relatively late-coupled ventricular premature depolarizations is consistent with the series reported by Anderson and associates (29), who identified 66 ambulatory patients with ventricular tachycardia from a population of

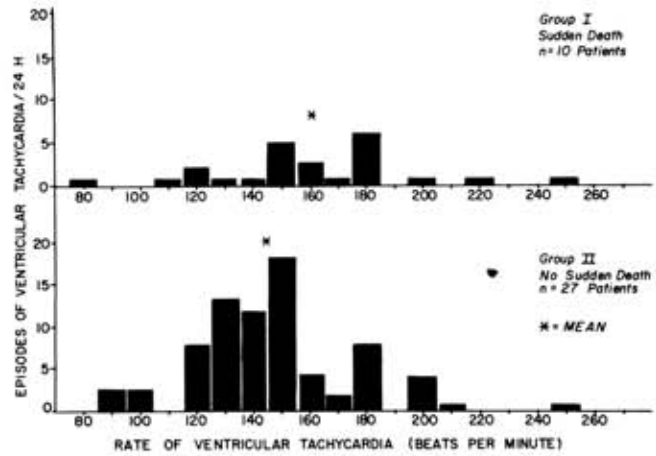

Figure 1. Bar graphs comparing the rates for all episodes of nonsustained ventricular tachycardia in Group I (sudden death) and Group II (no sudden death) patients.

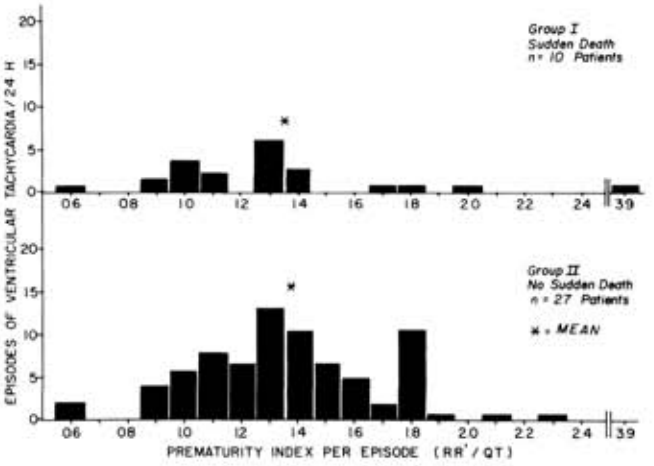

Figure 2. Bar graphs comparing the prematurity index *(RR'/QT)* of the initiating beat for all episodes of nonsustained ventricular tachycardia in Group I (sudden death) and Group II (no sudden death) patients.

915 patients after myocardial infarction. Only two of the 66 patients had ventricular tachycardia with a prematurity index for the initiating beat of < 1. The mean prematurity index for the whole group was 1.49 ± 0.37. Similarly, Boudoulas and co-workers (35) observed that episodes of nonsustained ventricular tachycardia were initiated by early-coupled ventricular premature depolarizations in only three of 45 ambulatory patients in whom this arrhythmia was detected by 24-hour ambulatory monitoring. Winkle and colleagues (32) reported on 94 episodes of ventricular tachycardia in 23 ambulatory patients. The mean prematurity index per episode in their series was 1.31 ± 0.28. Similar data are available on ventricular tachycardia occurring in the setting of an acute myocardial infarction. Roberts and associates (34) noted that 86% of 92 episodes of ventricular tachycardia in 38 patients were initiated by late-coupled ventricular premature depolarizations. Both episodes of ventricular fibrillation recorded in their study were also initiated by late-coupled ventricular premature depolarizations. In the previously cited study by De Soyza and co-workers (16), the mean prematurity index of ventricular premature depolarizations initiating ventricular tachycardia was 1.52 ± 0.28. In only 12% of the episodes of ventricular tachycardia was the prematurity index < 1.

These studies, in combination with our series, suggest not only that late-coupled ventricular premature depolarizations are capable of initiating ventricular tachycardia, but also that in most instances of ventricular tachycardia recorded in the ambulatory setting or in the setting of acute infarction, the initiating ventricular premature depolarization is a relatively late-coupled beat. Because we do not know the overall prevalence of early- and late-coupled ventricular premature depolarizations in our population, we cannot comment on the relative likelihood of early- or late-coupled beats inducing an episode of ventricular tachycardia. Finally, the nature of the beats initiating fatal arrhythmias is also unclear because patients are rarely monitored at the time of sudden death. Studies in the setting of acute infarction suggest that ventricular fibrillation may be commonly initiated by ventricular premature depolarizations with a prematurity index < 1 (23, 24, 31). Almost nothing is known about the initiation of ventricular fibrillation in the ambulatory setting, although Boudoulas and associates (35) did describe three cases with ventricular fibrillation recorded by ambulatory monitoring. In each case ventricular fibrillation was initiated by a late-coupled ventricular premature depolarization.

CHARACTERISTICS OF THE VENTRICULAR TACHYCARDIA AND THE ASSOCIATED ECTOPY

The rate of the nonsustained ventricular tachycardia varied widely in our population from 80 to 250 beats per minute and did not correlate with the subsequent incidence of sudden death. Eighty-nine percent of all the episodes were five beats or less in duration. Neither the duration of the episodes nor the number of episodes occurring in 24 hours was different comparing the sudden death and nonsudden death groups. Associated complex ventricular ectopy, including ventricular premature depolarizations greater than 10 per hour, multiform ventricular premature depolarizations, and ventricular couplets, was very frequent. However, the prevalance of these associated complexes was not significantly different comparing Groups I and II. Therefore, none of the characteristics that we examined of either the ventricular tachycardia or of the associated ectopy discriminated the group of patients who subsequently died suddenly.

Remarkably, the occurrence of even one to five episodes per day of nonsustained ventricular tachycardia was associated with an increased risk for sudden death in patients with congestive cardiomyopathy or history of congestive heart failure. Thus Holter monitoring for less than 24 hours might not be sufficient to detect all patients at such increased risk. We do not have data, however, to comment on the degree of risk associated with episodes occurring less frequently than once per day.

PROGNOSTIC SIGNIFICANCE OF VENTRICULAR TACHYCARDIA

In this selected group of patients with three or more

Table 4. Associated Arrhythmias in Patients with Ventricular Tachycardia

	Group I ($N = 10$)	Group II ($N = 27$)	*P* Value
Patients with >10 ventricular premature depolarizations/h, *no.*	10	24	NS*
Patients with multiform ventricular premature depolarizations, *no.*	6	15	NS
Patients with ventricular couplets, *no.*	8	21	NS
Patients with ventricular premature depolarizations with prematurity index <1, *no.*	2	6	NS
Patients with supraventricular tachyarrhythmias, *no.*	5	14	NS

* NS = not significant.

beats of ventricular tachycardia detected by long-term electrocardiographic recordings, the incidence of subsequent sudden death in the mean 19-month follow-up period was very high at 27%. Seven of the 10 sudden deaths occurred within the first 3 months of the follow-up period. Importantly, since these patients were originally identified from a population of patients monitored for a specific clinical indication, these findings cannot be extrapolated to ventricular tachycardia found in asymptomatic patients detected by monitoring done for routine screening purposes.

Other studies have also found that nonsustained ventricular tachycardia occurring in ambulatory patients with underlying cardiac disease has bad prognostic implications. The Coronary Drug Research Project (12) found by multivariate analysis that runs of ventricular premature depolarizations were associated with an increased risk of death that was not simply a reflection of the number of ventricular premature depolarizations. Although they did not specify in this report how many of these deaths were sudden, in a follow-up report (3) they noted that runs of ventricular premature depolarizations or early beats were together related to an increased risk of sudden death. In a study of patients' postmyocardial infarction, Anderson and co-workers (29) found that at 48 months, increases in the overall mortality of the group of patients with ventricular tachycardia compared to the group without ventricular tachycardia were approaching statistical significance. The ratio of sudden deaths to overall mortality was, however, unchanged. The authors suggested that the mortality may be related to the greater severity of the underlying heart disease.

A history of congestive heart failure was very highly correlated with the risk of sudden death in our population. Ninety percent of the patients with sudden death had a history of congestive heart failure compared to 37% of the patients in the nonsudden death group ($P < 0.005$). Forty-seven percent of the patients with ventricular tachycardia and a history of congestive heart failure died suddenly, while only 6% of those with ventricular tachycardia but without a history of heart failure suffered sudden death ($P < 0.005$). This incidence of sudden death is much greater than would be expected from congestive heart failure alone (36, 37), indicating that both variables, the ventricular tachycardia and the congestive heart failure, were important. Thus, the yearly mortality from all causes for a mixed population of men and women with congestive heart failure in the Framingham study was about 17%, compared to the 29% yearly mortality from sudden death in our series. Other studies have suggested the prognostic importance of the combination of ventricular ectopy and abnormal left ventricular function. Schulze and colleagues (22) followed 81 patients after myocardial infarction; eight of these patients suffered sudden death. They noted a greater incidence of complex ventricular ectopy in patients with poorly functioning ventricles and an increased incidence of sudden death in those patients with complex ventricular ectopy. Patients with poorly functioning ventricles but with complex ventricular premature depolarizations did not have as high a risk of sudden death. Rodstein and associates (21) followed 712 patients who had one or more extrasystoles on a routine ECG. They noted a greatly increased mortality ratio (number of deaths/number of predicted deaths) in those patients with cardiac abnormalities in addition to extrasystoles. Ruberman and co-workers (3) found in their study of 1739 men followed for up to 4 years after myocardial infarction that in those patients with either simple or complex ventricular premature depolarizations, the additional presence of congestive heart failure substantially increased the risk of sudden death.

Our findings must be interpreted with caution. Patient selection was by design retrospective; this was done so that follow-up could be obtained on a population adequate for analysis. Patients were selected from among those referred to a university-hospital clinical Holter monitor service and were therefore not necessarily representative of all patients with nonsustained episodes of ventricular tachycardia. In addition, patient therapy or compliance with therapy at the time of initial Holter recordings could not be confirmed. Moreover, patient therapy subsequent to the initial Holter monitor was not designed prospectively according to a controlled protocol. Instead, patients were managed individually by their referring physicians with variable efficacy and compliance. In this retrospective study, it was not possible to determine with certainty how the results of Holter monitoring affected subsequent treatment. Data were not adequate to comment on whether any prescribed therapy (including digoxin) was associated either positively or negatively with the occurrence of sudden death in follow-up. According to available information, many patients had no change in their regimens prescribed on the basis of Holter findings. Of far greater interest would be the effect of prescribed therapy evaluated in a controlled prospective study in which therapy was carefully monitored. Myerburg and co-workers (38) have recently demonstrated the importance of consistently maintaining therapeutic drug levels in reducing the incidence of recurrent sudden death in survivors of prehospital cardiac arrest.

Another inherent limitation was the definition of sudden death, defined as within 6 hours of the onset of symptoms for the purpose of this study. Undoubtedly in a population of patients with known congestive cardiomyopathy or history of congestive heart failure, "sudden death" might also result from a cerebrovascular or coronary artery embolus or from a massive pulmonary embolus. Autopsy studies were not done routinely; so these data were, unfortunately, not available. And although some patients were on chronic anticoagulation therapy, confirmation of the degree of compliance was not uniformly available, and therefore these data were not subjected to further analysis.

Nevertheless, this study presents strong evidence that the combination of even infrequent paroxysmal and nonsustained ventricular tachycardia plus congestive heart failure identifies a population of patients at especially high risk of sudden cardiac death. This would be an ideal population in which to evaluate prospectively the potential benefit of therapy designed to prevent sudden ar-

rhythmic death. Characteristics of ventricular tachycardia do not appear to be independent risk factors for sudden cardiac death.

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