Complex ventricular arrhythmias in patients with Q wave versus non-Q wave myocardial infarction

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ABSTRACT We examined whether or not subsets of patients with complex ventricular arrhythmias after myocardial infarction are at high risk with respect to 1 year mortality after hospital discharge. Based on previous studies showing increased risk for those with non-Q wave infarcts, we hypothesized that complex PVCs (premature ventricular complexes) in this group might be associated with a poorer prognosis than complex PVCs in patients with Q wave infarcts. Seven hundred seventy-seven patients entering our study with acute infarction were followed prospectively for 1 year after undergoing a predischarge 24 hr ambulatory electrocardiographic examination. Patients were classified by electrocardiographic criteria into the following groups: Non-Q wave (n = 191), Q wave anterior (n = 261), and Q wave inferior infarction (n = 325). The following arrhythmias were classified as complex: multiform PVCs, couplets, and ventricular tachycardia. Sixty-two percent of patients with non-Q wave infarcts who did not survive 1 year had complex PVCs, compared with 32% of survivors (p < .01). No differences were seen in the Q wave subgroup. The survival for patients with Q wave and non-Q wave infarction without complex PVCs were nearly identical at 1 year (93% and 90%), whereas in patients with complex PVCs survival for those with Q wave and non-Q wave infarction was 92% and 76%, respectively (p < .001). Of those with non-Q wave infarction, only 4% of nonsurvivors were free of any PVCs, as compared with 28% of nonsurvivors in the Q wave group (p < .02). Stepwise linear discrimination analysis revealed that complex PVCs were independent of ejection fraction in those with non-Q wave infarction, but were closely associated with ejection fraction in those with Q wave infarction. Thus, the presence of complex PVCs at the time of hospital discharge is an important predictor of 1 year mortality in the presence of non-Q wave, but not Q wave infarction.


RISK STRATIFICATION during the course of acute myocardial infarction may identify patients who are subsequently at high risk for recurrent infarction or death after recovery. Recognition of high-risk patients may permit specific interventions to be directed toward the factors responsible. Although the presence of complex ventricular arrhythmias recorded in the late hospital phase of acute infarction have been a strong predictor of subsequent mortality,1-12 attempts to improve survival by pharmacologic suppression have resulted in little success.13-15

It seems possible that if subsets of patients with complex arrhythmias who are at particularly high risk can be identified, treatment could be selectively aimed at such groups. Although non-Q wave and Q wave infarctions as classified by electrocardiography cannot always be differentiated anatomically, it is clear that they differ clinically and prognostically.16, 17 Data suggest that patients with non-Q wave infarction, despite an initially favorable prognosis, have a long-term mortality similar to or greater than that of patients with Q wave infarction.18, 19 Recently we have suggested that a non-Q wave infarction may be an unstable event and that variables such as infarct extension during the course of the acute infarction may be markers for late mortality.20, 21 Accordingly, the present study was undertaken to test the hypothesis that complex PVCs (premature ventricular complexes) occurring in the late hospital phase of acute infarction signify a poorer
prognosis for patients with non-Q wave infarction compared with the prognosis for those with Q wave infarction.

Methods

Patients. The study group consisted of 1,783 patients with acute infarction who were admitted to the hospital within 24 hr after onset of symptoms. Of the 1,658 patients eventually discharged from the hospital, 1,406 had been followed for 1 year at the time of the study. Of this group, 777 patients underwent predischARGE 24 hr ambulatory electrocardiographic (ECG) monitoring, and only this group was analyzed in this study for 1 year outcome. Ambulatory ECG monitoring was not required as part of our protocol and its performance was at the discretion of the attending physician. This could have led to some bias toward ECG monitoring in more gravely ill patients. However, statistical analysis comparing this group with the group of patients who did not undergo predischARGE ECG monitoring showed no differences in arrhythmias in the intensive care unit, discharge ejection fraction, or 1 year survival. However, there were higher peak creatine kinase (CK) levels, a longer stay in the intensive care unit, and more evidence of heart failure while in the hospital in the patients electrocardiographically monitored.

Q wave infarction was diagnosed by development of pathologic Q waves (as described below) and at least one of the following: (1) chest pain considered characteristic of myocardial ischemia, and (2) elevation of total creatine kinase. Q waves acceptable for diagnosis were as follows: (1) anterior = Q ≥ 0.04 sec in any precordial lead, (2) inferior = Q in leads III and aVF ≥ 0.04 sec or >25% of R wave in depth, and (3) lateral = Q in leads I and aVL ≥ 0.04 sec or >25% of R wave. Posterior infarctions were those resulting in an initial R wave in lead V1 or V2 of 0.04 sec with an R/S greater than 1. Infarctions resulting in both anterior and lateral Q waves were classified as anterior, and those resulting in inferior and posterior or inferior and lateral Q waves were classified as inferior. Non-Q wave infarction was diagnosed by (1) the presence of characteristic chest pain, (2) elevation of CK-MB, and (3) the following electrocardiographic criteria: (a) persistent new T wave inversion and/or ST segment changes, and (b) absence of new pathologic Q waves.

Two-channel (leads V1 and V5) 24 hr ambulatory electrocardiograms were analyzed by computer with manual overread by a technician, with final review by a physician. Complex PVCs were identified as one or more of the following: multiform PVCs, couplets, and/or ventricular tachycardia (3 or more beats with heart rate >100 beats/min).

Data concerning these patients were available from a data base maintained by the Specialized Center of Research (SCOR) on Ischemic Heart Disease at the University of California San Diego Medical Center. These patients were recruited during the period from 1979 to 1983 from the University of California San Diego Medical Center, the San Diego Veterans Administration Medical Center, the United States Naval Regional Medical Center in San Diego, and the Vancouver General Hospital, Vancouver, British Columbia.

Clinical and laboratory variables. The methods of data acquisition and definitions of variables have been reported in detail previously.22,23 Data from the history and physical examination and laboratory findings, chest x-rays, and electrocardiograms were assessed. The data base was examined univariately for variables related to complex PVCs in both the Q wave and the non-Q wave groups. Multivariate analyses were done on variables for which univariate screening showed an association with mortality at a significance level of p ≤ .05. Historical variables selected included age, sex, previous infarction, congestive heart failure, typical angina pectoris, chronic obstructive pulmonary disease, hypertension, bundle branch block, and new or changing angina within 1 month before hospital admission. The following clinical parameters were assessed throughout the period in the intensive care unit: maximal heart rate, minimal systolic blood pressure, maximal respiratory rate, the presence of S3 gallop, systolic murmur suggesting mitral regurgitation, apex impulse to the left of the midclavicular line, the presence and extent of rales, peripheral or sacral edema, and mental confusion. From chest radiographs, the maximal degree of pulmonary venous congestion was graded (0 to 4) as previously described.24

Laboratory findings included maximal levels of CK, maximum leukocyte count, and maximum creatinine and blood urea nitrogen. From the ECG tracings, the maximal measured QRS duration, infarct location, maximal PR interval, arrhythmias, and conduction disturbances were noted. At discharge, the administration of digitalis, diuretics, procainamide, quinidine, and β-blocking agents was recorded. The cardiothoracic ratio and degree of pulmonary congestion were measured on the discharge chest radiograph. In a subgroup of 496 patients, a discharge left ventricular ejection fraction was measured by radionuclide techniques or cardiac catheterization.

Follow-up. All patients were followed by telephone at 3, 6, and 12 months after hospital discharge. Information regarding death was obtained from death certificates, hospital records, or telephone interviews with the personal physician or family members. Only cardiac deaths were included in the analyses. A committee reviewed all available data to determine cause of death. Sudden death was defined as death presumed to occur within 24 hr after onset of symptoms. Sixty percent of these occurred within the first 1 hr. There was greater than 98% follow-up at 1 year.

Statistical methods. Univariate statistical analyses were performed by the chi-square test for discrete variables or t tests for continuous variables to assess differences in characteristics between patients with and without complex PVCs, as well as to study the influence of these variables on prognosis. To evaluate the independent importance of prognostic variables, the stepwise linear discrimination model (PLM) available in the Biomedical Computer Programs package of statistical programs was used.25 We have previously shown that this approach provides similar results to logistic regression analysis.26 Survival curves for the different populations were compared with the Mantel-Cox statistic as calculated by the survival function program (PIL) provided in the same software package.

Results

The occurrence of noncomplex and complex PVCs in patients with Q wave and non-Q wave infarctions in relation to selected clinical variables is presented in Table 1. Among the 791 patients studied (616 men and 175 women, mean age 61 years, range 29 to 89 yr), the location of infarction was classified as Q wave in 75% (inferior 41%, anterior 34%) and as non-Q wave in 25%. Complex PVCs were present on predischARGE 24 hr ambulatory monitoring in 41% of patients with non-Q wave infarction and 36% of those with Q wave infarction (p = NS). In patients without complex PVCs, 1 year mortality after hospital discharge was similar in the non-Q wave and Q wave groups (9% vs
7%, p = NS). Among patients with complex PVCs, the 1 year mortality in the non-Q wave group was 27% vs 9% in the Q wave group (p < .01).

Sixty-five variables were examined for association with complex PVCs in the Q wave and non-Q wave categories. Prior history of angina pectoris, myocardial infarction, and heart failure were not associated with complex PVCs in either group. Peak CK levels were higher in patients with Q wave infarctions, but were not significantly higher in those with complex PVCs in either group (table 1). There was no association between early arrhythmias in the coronary care unit and subsequent complex PVCs in either the Q wave or non-Q wave group. In the Q wave group, complex PVCs were associated with a significantly higher incidence of postinfarction left ventricular failure than that in the non-Q wave group, as evidenced by the occurrence of an S3 gallop (56% vs 45%, p < .01), basilaris rales (73% vs 54%, p < .001), rales above scapulae (11% vs 2%, p < .05), and increased cardiothoracic ratio (0.49 ± 0.05 vs 0.47 ± 0.05, p < .01). In the non-Q wave group, there was no significant association between complex PVCs and heart failure (table 1).

Complex PVCs were present in 39% of the patients with non-Q wave infarctions who survived 1 year (n = 126) (figure 1), while in those patients who did not survive one year (n = 26), complex PVCs occurred in 69% (p < .01). No such differences between survivors and nonsurvivors with respect to complex PVCs were seen in the anterior or inferior Q wave infarct groups. The cumulative survival curves beginning at discharge for patients with Q wave and non-Q wave infarctions with and without complex PVCs are illustrated in figure 2. In patients without complex PVCs (figure 2, A), the survival curves for patients with Q wave infarctions and the curves for those with non-Q wave infarctions were not significantly different, with a 1 year survival of 93% for the Q wave. (inferior = 95%, anterior = 92%) and of 91% for the non-Q wave group. However, when survival was compared among patients with complex PVCs, signifi-

**TABLE 1**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Non–Q wave infarction (n = 191)</th>
<th>Q wave infarction (n = 586)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Noncomplex PVCs</td>
<td>Complex PVCs</td>
</tr>
<tr>
<td>Total patients</td>
<td>113</td>
<td>78</td>
</tr>
<tr>
<td>Mean age (years)</td>
<td>62 ± 13</td>
<td>67 ± 10</td>
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<tr>
<td>Males (%)</td>
<td>76</td>
<td>80</td>
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<tr>
<td>Hx heart failure (%)</td>
<td>17</td>
<td>18</td>
</tr>
<tr>
<td>Hx angina (%)</td>
<td>57</td>
<td>64</td>
</tr>
<tr>
<td>Hx infarction (%)</td>
<td>33</td>
<td>36</td>
</tr>
<tr>
<td>Deaths — 1 year (%)</td>
<td>10</td>
<td>21</td>
</tr>
<tr>
<td>Survivors — 1 year (%)</td>
<td>90</td>
<td>73</td>
</tr>
<tr>
<td>Hospitalization</td>
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<tr>
<td>Mean peak CK</td>
<td>639 ± 725</td>
<td>589 ± 408</td>
</tr>
<tr>
<td>CTR</td>
<td>0.48 ± 0.05</td>
<td>0.49 ± 0.04</td>
</tr>
<tr>
<td>Mean ejection fraction</td>
<td>0.54 ± 0.13</td>
<td>0.50 ± 0.15</td>
</tr>
<tr>
<td>S1 (%)</td>
<td>30</td>
<td>37</td>
</tr>
<tr>
<td>Basilaris rales</td>
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<td>62</td>
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<td>RAS</td>
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<td>19</td>
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<tr>
<td>Diuretic</td>
<td>34</td>
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</tbody>
</table>

Mean values are ± 1 SD. Probability values based on Chi-square or t test.
CCU = coronary care unit; CTR = cardiothoracic ratio; Hx = history; RAS = rales above scapulae; V-tach = ventricular tachycardia; V-fib = ventricular fibrillation.
significant differences were seen (figure 2, B). The survival at 1 year for the non-Q wave group was 73%, as compared with 90% for the Q wave anterior group (p < .05) and 94% for the Q wave inferior group (p < .001). Approximately 50% of deaths in each group were classified as "sudden," irrespective of whether or not complex PVCs were present.

Types of ventricular arrhythmias detected on 24 hr monitoring. Figure 3 shows the breakdown of arrhythmias detected by 24 hr ambulatory ECG monitoring as related to survival in Q wave and non-Q wave infarction groups. Of all the types of arrhythmias present, the greatest differences in survivors vs nonsurvivors were in the categories of multiform PVCs and couples. These were present with greater frequency in nonsurvivors with non-Q wave infarctions than in survivors (33% vs 16% for multiform and 24% vs 7% for couples, p < .01), but their incidence did not differ in survivors and nonsurvivors of Q wave infarctions. In fact, there was no category of arrhythmia alone that was able to discriminate between survivors and nonsurvivors of Q wave infarction, although the small numbers of patients having ventricular tachycardia or R on T PVCs preclude accurate statistical analysis for those groups. Of all nonsurvivors of non-Q wave infarction, only one patient (4%) was totally free of any ventricular arrhythmias, as compared with 31 (25%) of the survivors (p < .01). Among nonsurvivors of Q wave infarction, 30% had no complex ventricular arrhythmias.

Relationship of left ventricular ejection fraction to complex PVCs. Table 1 reveals an association between the presence of complex PVCs and a decreased ejection fraction in the Q wave (ejection fractions without and with complex PVCs: 49 ± 13% vs 43 ± 14%, respectively, mean ± SD, p < .01), but not in the non-Q wave group (54 ± 12% vs 50 ± 14%, p = NS). This lack of association between complex PVCs and ejection fraction in patients with non-Q wave infarction remains when the relationship is examined in terms of mortality. We have recently found that an ejection fraction of 45% best discriminates between survival and death in our population database and thus used this dichotomized ejection fraction for comparison. The 1 year survival of non-Q wave infarction in all patients with complex PVCs compared with that in patients with complex PVCs and an ejection fraction less than 45% is presented in figure 4. The inclusion of lowered

FIGURE 1. Relationship between complex PVCs on discharge Holter recordings and 1 year survival.

FIGURE 2. Cumulative survival in patients with Q wave vs those with non-Q wave infarction with and without complex PVCs. A, In patients without complex PVCs followed for 1 year after discharge there was an 8% mortality in the Q wave (inferior = 5%, anterior = 10%) and a 10% mortality for non-Q wave group. B, In patients with complex PVCs, the cumulative survival at 1 year in the non-Q wave group was 76% as compared with 90% in the Q wave anterior (p < .05) and 93% in the Q wave inferior group (p < .01).

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FIGURE 3. Arrhythmias present at discharge (Holter) as related to 1 year survival in Q wave and non-Q wave infarction groups. Multifocal PVCs and couplets were present with greater frequency in nonsurvivors with non–Q wave infarction than in survivors (33% and 24% vs 16% and 7%, respectively, p<.01), but did not differ in survivors and nonsurvivors of Q wave infarctions. Only 4% of nonsurvivors of non-Q wave infarction were free of any ventricular arrhythmias as compared with 25% of survivors (p<.01).

ejection fraction in the analysis adds only 5% to predicted mortality over 1 year. The inset of figure 4 separates patients with and without complex PVCs into groups with normal and low ejection fractions. In the Q wave group, patients with complex PVCs were equally divided into the groups of those with ejection fractions less than and greater than 45%. Among those with non–Q wave infarctions and complex PVCs, twice as many patients had ejection fractions greater than 45%.

Multivariate analysis of predictors of mortality. Stepwise linear discriminant analysis was performed to evaluate independent contributions of ventricular arrhythmias and other variables to postinfarction mortality. No independent contribution to mortality could be attributed to ejection fraction in the non–Q wave group. In this group, the combination of complex PVCs, age, and pulmonary congestion predicted 74% of subsequent deaths. As expected, in contrast to the case in the non–Q wave group, there was no independent influence of complex PVCs on mortality. In the group of patients with Q wave infarction and PVCs the combination of ejection fraction, radiographic pulmonary congestion, and ages above the scapulae predicted 88% of subsequent deaths.

Complex PVCs and antiarrhythmic therapy at discharge. As can be seen in table 1, antiarrhythmic therapy at discharge was not related to type or location of infarction, and such therapy was given to a similar percentage of patients with and without complex PVCs on their discharge 24 hr electrocardiogram. The small numbers of patients in each group discharged on antiarrhythmic medication preclude accurate statistical assessment of the effect of this variable on survival. However, among those discharged on antiarrhythmic therapy, there was a trend toward increased 1 year survival in patients with non–Q wave infarction compared with that in patients with Q wave infarction. Thus, of the 17 patients with non–Q wave infarction discharged on therapy, 11 were dead after 1 year, compared with 21 of 24 patients with Q wave infarction similarly followed and on therapy (p = .08).

Discussion

The present study emphasizes the importance of complex PVCs occurring in the late hospital phase of acute myocardial infarction in predicting late cardiac death after non–Q wave myocardial infarction. In patients with Q wave infarction, complex PVCs alone were not associated with increased late mortality. In agreement with Moss12 and Vismara et al,11 we found no relationship between PVCs in the coronary care unit and the occurrence of similar arrhythmias on ambulatory recordings 1 to 3 weeks later in either group.

One of the first studies to show an association between ventricular arrhythmias after recovery from myocardial infarction and late cardiac death was the coronary drug project,12 which reported that the presence of one or more PVCs on a 12-lead electrocardiogram was associated with an increased risk of sudden death over the ensuing 2 to 5 years. More recently,
ambulatory ECG monitoring of 1 to 24 hr has been used. Ruberman et al. 3 examined the 1 hr recordings of 1739 men with recent myocardial infarction. Complex PVCs were associated with a threefold increase in risk of cardiac death at 24 months. Moss et al., 29 in analyzing survivors of acute myocardial infarction, found that arrhythmias of Lown grade 2 or greater identified patients at high risk of cardiac death or new infarction. Subsequent analysis revealed complex PVCs to be an independent contributor to cardiac death. 1

The criteria proposed by Lown and Wolf 30 for grading the severity of ventricular arrhythmias remain in common use, although objections have been raised to their validity. 31 Our index of complex arrhythmias included repetitive ventricular arrhythmias (couplets and ventricular tachycardia) and multiform beats. The Multicenter Investigation of the Limitation of Infarct Size (MILIS) found that both repetitive PVCs and multiform beats provide strong indexes of sudden cardiac death. 32 In a recent study to determine the relationship of Holter monitoring to programmed electrical stimulation, Gradman et al. 33 found that 100% of patients with induced sustained ventricular tachycardia, and 95% of those with nonsustained induced tachycardia, had multiform PVCs on their preceding 24 hr ambulatory electrocardiograms.

The results of the present study strongly suggest that complex PVCs in the recovery phase of acute infarction indicate high-risk status only in patients with non-Q wave infarction. The 1 year survival for these patients was 73%, compared with 90% for the Q wave group (p < .01). Few other studies have examined this relationship. Vismara et al. 11 followed 64 patients who had a 10 hr ambulatory ECG recording before hospital discharge after acute infarction. Of the 12 patients who subsequently died, eight of whom had complex PVCs, there was no difference in the incidence of Q wave and non-Q wave infarctions. However, the small number of patients in that study severely limited the possibility of distinguishing between the two infarct categories. Davis et al. 34 followed 940 patients with myocardial infarction for 12 to 60 months and found that the combination of anterior infarction, left ventricular dysfunction, and PVCs identified a high-risk subset with a 6 month mortality of 15%. Patients with non-Q wave infarctions were not analyzed separately, and the above combination would be expected to yield a high proportion of patients with a poor ejection fraction. Kotler et al. 4 followed 162 patients less than 65 years of age and in New York Heart Association class I or II for 30 to 54 months. Complex PVCs were found to be an independent risk factor, and there were no differences between patients with Q wave and non-Q wave infarctions.

Again the discrepancy from the present study may be explained by the smaller number of patients, a younger age group with possibly less severe coronary heart disease, and a longer follow-up period. Kotler et al. 4 did find that in the patients with non-Q wave infarction and no complex PVCs, no deaths occurred during follow-up. This supports the view that, in patients with non-Q wave infarction, freedom from complex PVCs carries a favorable prognosis, as indicated by our study. Of all nonsurvivors of non-Q wave infarction, only one (4%) was totally free of all ventricular arrhythmias, as compared with 25% of survivors (p < .01). In patients with Q wave infarctions, freedom from PVCs was no more common in survivors than nonsurvivors.

Relationship of complex PVCs to left ventricular function. The relationship between ventricular arrhythmias, left ventricular function, and prognosis after myocardial infarction has been controversial. While several studies show an independent effect of ventricular arrhythmias on mortality after adjustments are made for ventricular dysfunction, 1, 2 others have concluded that ventricular arrhythmias are so strongly associated with left ventricular dysfunction that this association accounts for the relationship between arrhythmias and subsequent death. 35 Much of the above controversy stems from the variety of methods used in previous studies. Arrhythmias were often detected on resting 12-lead electrocardiograms, 1 to 4 hr ambulatory or resting recordings, or recordings made later than 1 month after discharge; left ventricular dysfunction was often inferred from the clinical findings only. Recently, Bigger et al. 10 examined the relationship between ventricular arrhythmias, left ventricular dysfunction, and mortality after infarction in 760 patients in whom predischARGE 24 hr ECG recordings as well as radionuclide ventriculograms were obtained. They concluded that ventricular arrhythmias and ventricular dysfunction were independently related to high risk of death. MILIS presented results in 386 patients followed 2 years, and reported a strong univariate association between repetitive and multiform PVCs and mortality, as well as an independent association with left ventricular dysfunction. 32

The present study indicates that complex PVCs predict mortality only in the subgroup of patients with non-Q wave infarction, whereas mortality in the Q wave infarction group was closely related to poor ventricular function and did not relate to the presence or absence of complex PVCs. Shultze et al. 3 found that
90% of 81 patients with complex PVCs had ejection fractions of 0.40 or less. In the present study, only one-third of the patients in the non-Q wave infarction group with complex PVCs had ejection fractions of less than 45%, while in the Q wave group complex PVCs were equally distributed among those with fractions of greater or less than 45%.

**Implications for treatment.** Non-Q wave infarction is generally associated with a relatively small amount of necrosis and a low initial mortality when compared with Q wave infarctions. 21 Despite the initial favorable prognosis, prospective follow-up in our patients has confirmed previous reports 18, 19 that the 1 to 2 year mortality for patients with non-Q wave infarctions is similar to and possibly greater than that for patients with Q wave infarctions. 21 The present study indicates that this late equalization of mortality may largely be due to the death of patients with non-Q wave infarcts who have complex PVCs. Although the mechanisms of late death in patients with non-Q wave infarctions is not known, Rigo et al. 16 in a long-term study of 49 patients with non-Q wave and 111 patients with Q wave infarcts, suggested the possibility that patients with non-Q wave infarcts may have a higher incidence of fatal arrhythmias after discharge than patients with Q wave infarcts, based on a higher incidence of recurrent ischemia in the non-Q wave group. However, it is possible that the increased incidence of complex ventricular arrhythmias is causally unrelated to the increased late mortality in such patients with non-Q wave infarction, and that an unstable ischemic state with recurrent myocardial infarction is the cause. Nevertheless, it is also possible that death during recurrent ischemia or infarction occurs primarily in those with ongoing arrhythmias, or that primary arrhythmic death without concurrent ischemia occurs in some of these patients.

The best current evidence favors an independent association between ventricular arrhythmias after myocardial infarction and subsequent mortality, and therefore it seems reasonable to recommend antiarrhythmic therapy in an effort to reduce this risk. To date, randomized, double-blind studies have not shown antiarrhythmic drugs to be of great benefit, as reviewed elsewhere. 14, 37 However, most of the studies have focused on patients who have recovered from myocardial infarction, without attempts at categorization of arrhythmias or type of infarction. The present study demonstrates that patients with non-Q wave infarcts who have complex PVCs on a predischarge ambulatory recording are at high risk for cardiac death in the subsequent year, irrespective of the ejection fraction. This group may provide one logical choice of patients in whom to test the efficacy of antiarrhythmic therapy in the setting of a randomized clinical trial.

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