Evaluation of Warning Arrhythmias Before Paroxysmal Ventricular Tachycardia During Acute Myocardial Infarction in Man

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Summary

In order to determine the relationship of paroxysmal ventricular tachycardia (PVT) to any antecedent (premonitory) ventricular arrhythmias during the early phases of acute myocardial infarction, 24-hour Holter monitoring was begun on 52 male patients an average of 12.6 hours after the onset of prolonged chest pain that was documented as acute infarction. Twenty-four patients had PVT and 28 did not. We analyzed in detail the incidence of frequency of premature ventricular complexes (PVCs), prematurity and pairing during the 10 minutes immediately preceding PVT from a continuous 10-minute rhythm strip. There was no positive correlation between PVT and the number or complexity of PVCs in the 10 minutes immediately before ventricular tachycardia. These findings suggest that there is no consistent pattern or frequency of ventricular arrhythmia that could be identified as premonitory for PVT during the immediate pre-PVT period, even during the acute phase of myocardial infarction in man.

During acute myocardial infarction, ventricular tachycardia can degenerate into ventricular fibrillation. Paroxysmal ventricular tachycardia (PVT) has been reported to occur fairly frequently during the initial phases of acute myocardial infarction. Attempts to detect warning arrhythmias before ventricular fibrillation have generally not been successful; and several studies suggest that primary ventricular fibrillation is usually a sudden and unheralded catastrophe during the acute phase of myocardial infarction.

Other studies have reported that complex ventricular arrhythmias are noted frequently just before both ventricular tachycardia and ventricular fibrillation. Since PVT is a more common rhythm disturbance during acute myocardial infarction than ventricular fibrillation, and since ventricular tachycardia can degenerate into ventricular fibrillation, we examined the 10 minutes before episodes of PVT by Holter monitoring during the acute phase of myocardial infarction in man. We looked for a recurring pattern of ventricular arrhythmia that immediately precedes and might be predictive of PVT in these patients. We looked specifically at the incidence of three previously reported harbingers of PVT during acute myocardial infarction: the absolute frequency of premature ventricular complexes (PVCs), the in-
cidence of the R-on-T phenomenon as a measure of PVC prematurity and PVC pairing in the immediate pre-ventricular tachycardia period.

Materials and Methods

All patients admitted to the Little Rock Veterans Administration Hospital Coronary Care Unit over a 60-week period within 24 hours of the onset of prolonged chest pain suggestive of acute myocardial infarction underwent 24-hour Holter monitoring while in the unit. Fifty-two of these patients subsequently had diagnostic criteria of transmural infarction with the development of Q waves, typical ST-T-wave changes, and characteristic rise and fall of SGOT and LDH values. The mean period of time elapsing between the onset of prolonged chest pain and the beginning of the 24-hour tape recordings in all patients was 12.6 ± 6.4 hours (± sd). Thus, in all patients included in this study, the tape recordings were begun well within 24 hours of the onset of the prolonged episode of chest pain that subsequently led to acute myocardial injury. All patients were male, and the average age was 56 years, with a range of 47–75 years. Conventional medical management with antiarrhythmic drugs was not withheld from any patient during the study period.

The Avionics Model 650 Electrocardioscanner was used to analyze the tape recordings. This machine allowed playback of the tapes at 60 times real-time. We used an oscilloscopic display of a modified V1 lead, the RR interval and an R-wave-triggered sound system to detect and quantitate ventricular arrhythmias. The episodes of PVCs were detected by manually scanning all tapes and obtaining direct writeout of all PVC episodes detected. In the present study, PVC was defined as three or more consecutive beats of ventricular origin at a rate greater than 120 beats/min. The entire 10 minutes of ECG immediately preceding PVC was recorded as a continuous rhythm strip at a paper speed of 250 mm/sec. Each rhythm strip was examined, beat by beat, and the following information was obtained for each episode of PVC:

1) The number of PVCs present during the first, second through the fourth, and fifth through the tenth minutes preceding PVC.

2) The coupling intervals and prematurity indexes of all PVCs in the above periods. The prematurity index of PVCs was obtained by dividing the coupling interval of the PVCs by the QT interval of the preceding sinus beat. The R-on-T phenomenon was considered present when the prematurity index of a PVC was equal to or less than 1.0.

3) The incidence of pairing of PVCs during the 10 minutes preceding PVC.

Results

Of the 52 patients monitored, 24 (46%) had a total of 52 episodes of PVC documented during the monitored period. All episodes of PVC spontaneously reverted to sinus rhythm. The 52 episodes of PVC had the following characteristics: The average number of episodes per patient was two (range one to seven episodes). Twelve of the 24 patients with PVC had only one episode of PVC documented. The mean heart rate during episodes of PVC was 157 (range 125 beats/min to 214 beats/min). The 52 episodes of PVC had an average of five beats per episode (range three to 12 beats per episode). Sixteen of the 52 episodes lasted for three beats only. Twenty-five of the 52 episodes were initiated by ventricular fusion beats. Twenty-five of the 52 episodes were exactly regular throughout the episode. The other 27 episodes displayed very slight variation in rate during the initial two to three beats of each episode. Of the 12 patients having more than one episode of PVC each, three patients had PVC episodes with differing QRS configurations that lasted the entire duration of the episode. Only one patient had a single episode of PVC in which the QRS configuration of the initial beat of the run was distinctly dissimilar to the subsequent beats of the run of PVC. We saw no other instances of beat-to-beat QRS configuration change during a single episode of PVC.

There were no episodes of PVC documented in 28 of the 52 patients. The patients with PVC had significantly more ventricular arrhythmias than the patients without PVC: The median number of PVCs in the 24 patients with PVC was 5.0 per 1000 normal beats, compared with 0.7 PVCs per 1000 normal beats in the 28 patients without PVC (p < 0.005). Twenty-one of the 24 patients with the PVC had paired PVCs, but only seven of the 28 patients without PVC had paired PVCs (p < 0.005).

Sixteen of the 24 patients with PVC and nine of the 28 patients without PVC were receiving digoxin or antiarrhythmic drugs during Holter monitoring (p < 0.05). The breakdown of this therapy in the two groups of patients was as follows: Digoxin — four of 24 patients with PVC and four of 28 patients without PVC (NS). Lidocaine — 11 of 24 patients with PVC and seven of 28 patients without PVC (p < 0.05). Procainamide — two of 24 patients with PVC and four of 28 patients without PVC (NS). Lidocaine was used if a patient had significant ventricular ectopy. In the CCU at the Little Rock Veterans Administration Hospital, in a patient thought to have acute myocardial infarction, lidocaine therapy is initiated in any patient who has any of the following: > 5 PVCs/min, paired PVCs or runs of three or more consecutive PVCs. In the six of 52 patients receiving procainamide therapy during the period of observation, one patient had procainamide therapy before being admitted to the hospital, and this therapy was continued after admission. The other five patients had one oral dose of procainamide at the end of the observation period, since a decision had been made to start oral agents based on the need for lidocaine during the initial hours after admission. The indications for the use of digoxin in the eight of 52 patients receiving digoxin during the period of observation was as follows: runs of paroxysmal atrial ectopy in three patients and digitalization for incipient cardiac failure in four patients. The other patient who had been taking digitalis before his
present admission had this therapy continued after the current admission. In all cases, full digitalizing doses were not used during observation.

Anterior infarction occurred in 42% of the patients with PVT and in 44% without PVT. Similarly, inferior infarction was documented in 58% of patients with PVT and in 56% of those without PVT.

In the 52 rhythm strips with an episode of PVT analyzed, there were 809 PVCs before PVT. One patient (ER) had 544 PVCs (67% of total) before two episodes of PVT, and these PVCs usually occurred in a trigeminal pattern. In this patient, there was an average of 30–35 PVCs/min for the 3 minutes immediately preceding PVT on one strip; and for the entire 10 minutes preceding PVT on the second rhythm strip. The remaining 285 PVCs (33% of the total) were distributed among the remaining 50 rhythm strips with PVT. Dividing the 10-minute rhythm strips into the first, first through fourth, and fourth through tenth minutes before PVT revealed that in the 50 rhythm strips there was no ventricular arrhythmia at all during the first minute before PVT in 73% of the strips (fig. 1). Forty-four percent of the rhythm strips had no arrhythmia up to the fourth minute, and there was no ventricular arrhythmia at all during the entire 10 minutes in 19% of the rhythm strips. In the remaining rhythm strips with PVT in which ventricular arrhythmias were detected before PVT, 3% occurred within the first minute immediately before PVT, 10% occurred in the first 4 minutes, and 90% occurred between the fourth and tenth minutes before PVT (fig. 2).

Of the PVCs that occurred in the first minute before PVT, 93% were single and 7% were paired. Between the first and fourth minutes, 82% were single and 18% were paired. Between the fourth and tenth minutes, 85% were single and 15% were paired. Only 27% of the 50 episodes of PVT were preceded by paired PVCs; only 4% of the 50 episodes were preceded by more than 5 PVCs/min in the 10-minute period.

When we examined the prematurity index to identify PVCs that would be more likely to initiate serious ventricular arrhythmia, we found that the mean prematurity indexes of the PVCs occurring during the three time periods were 1.36, 1.40, and 1.39, respectively. The overall range of prematurity index for all PVCs was 0.63–2.40. When the R-on-T phenomenon was defined liberally as a prematurity index \( \leq 1.0 \), only 2% of the 285 PVCs in the 10 minutes preceding PVC qualified for the R on T phenomenon. Patient ER had a total of 544 PVCs before his two episodes of PVT, and all PVCs in this patient had a prematurity index of 0.90. In the remaining PVCs in the other 51 patients, the prematurity index of PVCs initiating PVT was less than 1.0 in only 14% of the 50 episodes of PVT. Conversely, 33% of the 265 PVCs that did not produce PVT had coupling intervals \( \leq 1.0 \).

When different combinations of the complex features of ventricular arrhythmia noted during the entire 10 minutes preceding PVT were analyzed, 27% of the episodes of PVT were preceded by paired PVCs (fig. 3); 17% contained PVCs demonstrating the R-on-T phenomenon; 4% had more than 5 PVCs/min; 6% had paired PVCs along with the R-on-T phenomenon and 4% had paired PVCs along with the R-on-T phenomenon and greater than 5 PVCs/min.

Seven of the 52 patients (13.5%) died. Patients with a diagnosis of acute myocardial infarction who did not experience prolonged chest pain within 24 hours of CCU admission or who died within 24 hours were excluded from study. Two patients with acute myocardial infarction died before 24-hour Holter monitoring was completed. One of these patients suffered massive anterior myocardial infarction complicated by acute mitral insufficiency and died in pump failure 12 hours after the Holter monitor started. The other patient had anterior myocardial infarction complicated by trifascicular block and complete atrioventricular block requiring temporary pacemaker placement. This patient also died in cardiogenic shock 8 hours after Holter monitoring was started. The incomplete tape recordings from these two patients were not analyzed in detail because all study criteria were not met. Two of the seven deaths that occurred during the third post-myocardial infarction week were sudden and were presumed to be related to arrhythmias. Two patients died in cardiogenic shock after an extension of their infarction or reinfarction during the third postinfarction week. Another patient died on the twelfth post-myocardial infarction day and had myocardial rupture at autopsy. The two other deaths were two patients who died after hospital discharge, one 4 and one 7 weeks after their acute myocardial infarction. Of the seven deaths, four were patients who had PVT documented during the first 24 hours of acute myocardial infarction.

**Discussion**

Conventional management of patients with acute myocardial infarction in the CCU usually involves lidocaine therapy in patients who develop "high-risk" PVCs, which include more than 5–6 PVCs/min, paired PVCs and PVCs that demonstrate the R-on-T phenomenon.
phenomenon. However, the results of this study show that even if the cardiac rhythm were observed closely for such “high-risk” PVCs during the 10 minutes before PVT, 19% of the episodes of PVT would have occurred anyway because there were no warning arrhythmias. Similarly, since no ventricular arrhythmias were noted during this study in the 1 minute before PVT in 73% of the episodes, close observation of the patient’s rhythm during this time would not have revealed treatable warning arrhythmias. We conclude that no consistent pattern of ventricular arrhythmias can be identified as premonitory for PVT during the 10-minute period immediately before PVT during the initial 24 hours of acute myocardial infarction in man. These results are in agreement with other studies evaluating the cardiac rhythm preceding primary ventricular fibrillation during acute myocardial infarction in man. Lawrie et al. found that of 12 patients with acute myocardial infarction who developed ventricular fibrillation, only two had premonitory ventricular arrhythmias that occurred early enough to allow suppressive anti-arrhythmic therapy. However, these investigators did not use Holter recordings. Wyman et al. used Holter recordings in selected patients with acute myocardial infarction and found that seven of 12 patients who developed ventricular fibrillation had no warning arrhythmias. However, Dhurandhar et al. noted significant warning ventricular arrhythmias before ventricular fibrillation during acute myocardial infarction in 12 of 20 episodes of ventricular fibrillation during myocardial infarction. Lie et al. noted this phenomenon in four of nine episodes of ventricular fibrillation. In the later study, the observations were made without concurrent use of antiarrhythmic therapy. We felt it inappropriate to withhold antiarrhythmic therapy for significant ventricular arrhythmias during acute myocardial infarction. However, whereas 46% of the episodes of PVT in our patients occurred while antiarrhythmic therapy was being administered, 54% of the episodes occurred in patients who did not receive antiarrhythmic therapy. In this latter group of patients, “high-risk” warning ventricular arrhythmia before PVT was absent before PVT in the preceding 10 minutes in 72% of the episodes of PVT that occurred in patients who did not take antiarrhythmic drugs. In the patients who took antiarrhythmic therapy, 70% of the episodes of PVT occurred without any “high-risk” warning arrhythmia. Thus, in this group of patients, regardless of whether or not concurrent antiarrhythmic therapy was administered, PVT was more likely to occur without premonitory “high-risk” ventricular arrhythmia.

The lack of correlation of the prematurity of PVCs initiating PVT during acute myocardial infarction to the onset of PVT has been previously described. This present study confirms this same lack of correlation between the R-on-T phenomenon and the propensity to develop PVT in these patients.

“High-risk” combinations of PVCs have previously been reported to be associated with an increased incidence of PVT. While this is certainly true in the overall setting of acute myocardial infarction when long periods of recorded cardiac rhythms are analyzed, the results of the present study suggest that in the immediate pre-PVT period (10 minutes), > 5 PVCs/min, paired PVCs and PVCs demonstrating the R-on-T phenomenon occur only infrequently. In fact, in this study only 4% of the episodes of PVT had such “high-risk” combination of PVCs in the 10 minutes preceding PVT (fig. 3).

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