Ectopic Ventricular Prematurity and Its Relationship to Ventricular Tachycardia in Acute Myocardial Infarction in Man

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SUMMARY

In order to determine the role of the coupling interval of a premature ventricular contraction (PVC) in the development of paroxysmal ventricular tachycardia (PVT) during the early phase of acute myocardial infarction in man, 52 male patients with documented acute myocardial infarction had 24-hour Holter monitoring commenced within 24 hours of the onset of prolonged chest pain. Review of the tape recordings revealed that 27 patients had PVT documented, while 25 patients did not. Analysis of the data on the two groups showed that the frequency of PVCs, coupled PVCs, and accelerated idioventricular rhythm (AIVR) were found to be associated with a significantly increased incidence of ventricular tachycardia.

The mean coupling interval of the PVCs initiating episodes of ventricular tachycardia was not significantly different from either the mean coupling interval of the isolated PVCs in the patients with PVT or the mean coupling interval of the PVCs in the patients without PVT. This suggests that the coupling interval of a ventricular ectopic is a poor predictor of ventricular tachycardia in the early phases of acute myocardial infarction.

Additional Indexing Words:
Premature ventricular contraction Prematurity index Accelerated idioventricular rhythm Coupling interval Paroxysmal ventricular tachycardia Holter monitoring

Since Wiggers et al. demonstrated that electrical stimulation of the mammalian ventricle in late systole could lead to ventricular fibrillation, further confirmation of the increased incidence of ventricular fibrillation with the R on T phenomenon has become available in the form of several reports on animal experiments. However, a recent report by Williams et al. has also suggested that ventricular ectopics occurring relatively late in diastole in dog hearts can also consistently initiate ventricular fibrillation. The frequency with which ventricular ectopics with long coupling intervals initiated paroxysmal ventricular tachycardia (PVT) was noted during analysis at the Little Rock Veterans Administration Hospital of 24-hour tape recorded rhythms of patients during the initial 24 hours of an acute myocardial infarction. This finding stimulated the present investigation of the role of the coupling interval of a premature ventricular contraction (PVC) in the production of ventricular tachycardia in acute myocardial infarction in man. Other well known precursors of ventricular tachyarrhythmia were also analyzed in this same group of patients.

Materials and Methods

All patients admitted to the coronary care unit over a 60 week period commencing in September 1972, within 24 hours of the onset of prolonged chest pain suggestive of acute myocardial infarction, had a 24-hour tape recording made of their rhythm using the Holter monitoring system. Of the patients whose rhythms were so recorded, only 52 patients subsequently developed a diagnostic Q wave with or without evolutionary changes of ST segments and T waves along with characteristic rise and fall of SGOT and LDH values to confirm a diagnosis of myocardial infarction. These 52 patients formed the basis of the present study. The average age of these patients was 52 years, with a range of 24 to 73 years. All 52 patients were males. The medical management of the patients was not changed during the tape recordings. Two patients who initially qualified for the study had to be excluded subsequently because one required temporary pacing during the period of observation, and the other died in cardiogenic shock within five and one half hours of the onset of recording. A modified V5 lead was used during all tape recordings.

The tape recordings were analyzed using the Avionics
electrocardioscanner and the Instruments for Cardiac Research rhythm analyzer, and the following data were obtained: (1) the number of episodes of PVT, defined as three or more successive beats of ventricular origin at a rate greater than 100 beats/min, (2) the number of episodes of accelerated idioventricular rhythm (AIVR), defined as three or more successive beats of ventricular origin at a rate between 60 and 100 beats/min, (3) the total number of PVCs per 1000 normal beats, (4) the number of episodes of coupled PVCs, (5) the coupling intervals and the prematurity indices of all PVCs initiating PVT and the coupling intervals and prematurity indices of the other PVCs not initiating PVT, (6) the QRS morphology of the isolated PVCs and the PVCs initiating AIVR, and the QRS morphology during the episodes of PVC and AIVR, (7) the time of appearance of PVT and AIVR in relation to the time of commencement of the tape recording, (8) the heart rate immediately preceding episodes of PVT and AIVR, and (9) the mean heart rates over the first 30 minutes during each four hour period. A mean heart rate was then obtained for these six 30 minute periods.

The prematurity index of a PVC was determined by dividing the coupling interval of the PVC by the QT interval of the preceding sinus beat. In patients with more than 200 PVCs per 24-hour recording, the measurements were made on 20% of the PVCs selecting every fifth PVC for measurement.

**Results**

Out of the 52 patients, 27 patients (52%) had a total of 131 episodes of PVT documented during the tape recording. The remaining 25 patients had no PVT documented.

While 59% of the 27 patients with PVT had AIVR documented during the 24-hour recording, only 8% of the 25 patients without PVT had AIVR documented ($P < 0.005$). Thirteen out of the 16 patients who had both AIVR and PVT had at least one instance where the QRS morphology appeared to be similar during both forms of arrhythmia. The QRS morphology during PVT and AIVR was different during all the episodes in three out of the 16 patients who had both forms of arrhythmia documented.

The median number of PVCs in the 27 patients with PVT was 5.0 per 1000 normal beats. This was significantly greater than the median number of PVCs in the 25 patients without PVT, 0.7 PVCs per 1000 normal beats ($P < 0.005$).

While 89% of the 27 patients with PVT had coupled PVCs, only 16% of the 25 patients without PVT had coupled PVCs ($P < 0.005$).

The mean coupling interval of the PVCs initiating all 131 episodes of PVT was $0.59 \pm 0.14$ sec (mean $\pm$ sd) (table 1). This was not significantly different from the mean coupling interval of the PVCs not initiating PVT in the 27 patients with PVT ($0.55 \pm 0.09$ sec) or the mean coupling interval of the PVCs in the 25 patients without PVT ($0.55 \pm 0.14$ sec).

Similarly, the mean prematurity index of the 131 PVCs initiating PVT ($1.52 \pm 0.28$) was not significantly different from the mean prematurity index of the other PVCs not initiating PVT in the 27 patients with PVT ($1.42 \pm 0.21$) or the mean prematurity index of the PVCs in the 25 patients without PVT ($1.44 \pm 0.28$ (table 1).

While only 12% of the 131 PVCs initiating PVT had a prematurity index of less than one, 16% of the isolated PVCs in the patients with PVT had a prematurity index less than one (table 1). Thirteen percent of the PVCs in the patients without PVT also had a prematurity index less than one.

The QRS configuration of all isolated PVCs not initiating PVT was similar in 4% of the 27 patients with PVT and in 20% of the 25 patients without PVT. The episodes of PVT had similar QRS configuration in 63% of the 27 patients with PVT and different QRS configuration in the remaining 27% of these patients. The PVC initiating PVT had a QRS configuration different from that during the arrhythmia in only 3% of all episodes of PVT observed in 27 patients.

Of all the episodes of PVT observed, 50% of these occurred within the first 6 hours of starting each recording. Twenty-nine percent of the episodes occurred during the second 6 hours, 13% during the third 6 hours and 8% during the last 6 hours of the 24-hour recording. The isolated PVCs in patients with and without PVT were predominantly noted during the initial 12 hours of the recording. However, an accurate breakdown of the times of occurrence of each of these isolated PVCs could not be obtained.

**Table 1**

*Characteristics of PVCs in Patients With and Without Paroxysmal Ventricular Tachycardia*

<table>
<thead>
<tr>
<th></th>
<th>Mean coupling interval (sec)</th>
<th>Prematurity index</th>
<th>Percentage of PVCs with prematurity index &lt; 1</th>
</tr>
</thead>
<tbody>
<tr>
<td>PVCs initiating PVT</td>
<td>$0.59 \pm 0.14$ (sd)</td>
<td>$1.52 \pm 0.28$ (sd)</td>
<td>12</td>
</tr>
<tr>
<td>Other PVCs in patients with PVT</td>
<td>$0.55 \pm 0.09$ (sd)</td>
<td>$1.42 \pm 0.21$ (sd)</td>
<td>16</td>
</tr>
<tr>
<td>PVCs in patients without PVT</td>
<td>$0.55 \pm 0.14$ (sd)</td>
<td>$1.44 \pm 0.28$ (sd)</td>
<td>13</td>
</tr>
</tbody>
</table>

Abbreviations: PVC = premature ventricular contraction; PVT = paroxysmal ventricular tachycardia; sd = standard deviation.
VENTRICULAR TACHYCARDIA IN ACUTE MI

The heart rates immediately preceding each episode of PVT are shown in figure 1. Of all the episodes of PVT observed, 15% of these episodes occurred at sinus rates below 60 beats/min. Sixty-seven percent of the episodes of PVT occurred at sinus rates between 60 and 100 beats/min. The remaining 18% of the episodes of PVT occurred at preceding sinus rates in excess of 100 beats/min.

The mean heart rate immediately preceding all episodes of PVT (in the 27 patients with PVT) varied between 60 and 105 beats/min with an over-all mean preceding heart rate for this group of 80 ± 13 beats/min. The mean heart rate of the 25 patients without PVT was essentially similar, ranging between 53 and 105 beats/min with an over-all mean heart rate of 76 ± 16 beats/min.

Inferior myocardial infarction was present in 55% of the patients with PVT. Forty-five percent of these same patients had anterior myocardial infarction. Fifty-six percent of the patients without PVT had inferior myocardial infarction, while 44% of these same patients had anterior myocardial infarction.

During the period of recording, lidocaine was given to 40% of the patients with PVT and 24% of the patients without PVT, procaine amide was given to 7% of the patients with PVT and 4% of the patients without PVT, and digoxin was given to 11% of the patients with PVT and 4% of the patients without PVT. None of the patients in either group received quinidine, propranolol, or diphenylhydantoin during the recordings.

The average age of the 27 patients with documented PVT was 55 years with a range of 42 to 73 years. The average age of the 25 patients without documented PVT was 53 years with a range of 24 to 63 years.

The in-hospital mortality rate for the 52 patients on the study was 9.6%. Two of these were sudden deaths during the third post myocardial infarction week. Two more patients died in cardiogenic shock following extension or re-infarction, again during the third post myocardial infarction week. No autopsies were obtained on these four patients. The other patient who died in the hospital had autopsy proven cardiac rupture which occurred on the twelfth post myocardial infarction day. Two more late deaths occurred among the 47 survivors making the total mortality for this group 13.5%. Four out of the seven deaths were patients who had PVT during the tape recordings made during the coronary care unit stay.

Discussion

While the results of this study vary considerably from previous concepts as to the role of the coupling interval of PVCs and AIVR in the genesis of PVT in acute myocardial infarction, our results confirmed previous reports6,7 where the frequency of PVCs and coupled PVCs were associated with an increased incidence of ventricular tachyarrhythmia.

In the present study, PVCs with short coupling intervals were infrequently associated with PVT, only 12% of the PVCs initiating PVT having a prematurity index of less than one. Similar findings have been reported by Bleifer and others8 in a study of ambulatory patients with known ischemic heart disease. These investigators have reported that the R on T phenomenon by itself did not produce ventricular tachycardia in their patients. Kleiger et al.9 also recently reported that only one episode of PVT was initiated by an early PVC out of 19 separate episodes of PVT noted in patients in the late recovery phase of acute myocardial infarction. This latter study was carried out two weeks to 18 months post myocardial infarction on 141 patients. In the study reported by Moss and others10 on 33 patients whose arrhythmias were recorded during the pre-hospital phase of acute myocardial infarction, PVCs with short coupling intervals were significantly more prevalent in the 12 patients having serious ventricular arrhythmias than the 21 patients who did not have subsequent serious ventricular arrhythmias. However, in four out of the five patients with ventricular fibrillation or ventricular tachycardia in the same study, “the tachyarrhythmia immediately followed a long R to R cycle length produced by the compensatory pause of a preceding VPB.” While it cannot be disputed that PVCs with short coupling intervals can lead to ventricular tachyarrhythmia, our study further confirms the fact

![Figure 1](http://circ.ahajournals.org/) The distribution of heart rates preceding episodes of paroxysmal ventricular tachycardia.

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that PVCs with long coupling intervals frequently precipitate serious ventricular tachyarrhythmia. A probable explanation for the predisposition of PVCs with long coupling intervals to produce ventricular tachyarrhythmia has been offered by Williams et al.4 who studied this problem with the aid of bipolar subepicardial and subendocardial leads in the dog heart. Occlusion of the left anterior descending artery caused marked delay and block of locally recorded electrical activity only in the ischemic epicardium. This delay of local activation in turn caused total ventricular activation time to increase by as much as 320 msec (increased dispersion of ventricular activation).

At this time, PVCs delivered even late in diastole caused ventricular tachycardia and ventricular fibrillation. Those PVCs showing the greatest dispersion of ventricular activation were found to precipitate ventricular fibrillation.

The effect of varying heart rates on the incidence of ventricular tachyarrhythmia in acute myocardial infarction is yet unclear. The results of this study show that while the great majority of the episodes of PVT occurred during sinus rates ranging from 60 to 100 beats/min, 15% of the episodes occurred at rates below 60 beats/min, while 18% of them occurred at rates in excess of 100 beats/min.

The results of our study also demonstrate that AIVR and PVT frequently co-exist in the same patient in the presence of acute myocardial infarction. Such co-existence of these two forms of arrhythmia have been reported to be uncommon.11 These conclusions emphasize the need for close observation of patients demonstrating AIVR during the course of an acute myocardial infarction for more serious forms of ventricular arrhythmia.

In the present study, the location of the infarct did not appear to be a significant factor in the predisposition to ventricular tachycardia. The incidence of anterior infarction was almost identical in the patients with and without PVT. There was also no significant difference in the average age of the patients with and without PVT in the present study.

A review of the drug therapy administered to the patients during the recordings reveals that there was no significant difference in the number of patients receiving digoxin, procaine amide, and lidocaine in the two groups of patients with and without PVT. This suggests that drug regimen used during the recordings did not appear to be a factor in the production of the PVT observed in these patients.

The reported incidence of PVT in acute myocardial infarction varies considerably in the literature. These figures are affected by the definition of PVT used and the method used to compile the data. Lown et al.12 reported a 28% incidence of PVT in 300 consecutive cases of acute myocardial infarction. Mounsey13 reported a 5% incidence of PVT in 103 consecutive acute myocardial infarction patients. In our small group of patients, a 52% incidence of PVT was observed during the initial stages of acute myocardial infarction. This increased incidence is related to two factors: (a) the more accurate method of data collection used, that is, continuous recording of rhythm on electromagnetic tape as opposed to the relatively inaccurate conventional coronary care unit monitoring,14 and (b) the selection of patients in the initial stages of myocardial infarction.

In conclusion, the present study suggests that while the coupling interval of a PVC has relatively poor predictive value in the development of PVT, (1) the frequency of PVCs, (2) coupled PVCs, and (3) AIVR were found to be associated with a significantly increased incidence of PVT in acute myocardial infarction.

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