We examined the harbingers of 68 episodes of paroxysmal ventricular tachycardia in 42 patients with documented acute myocardial infarction. Late ventricular premature contractions initiated 46 bouts of paroxysmal ventricular tachycardia, while 17 were engendered by early ventricular premature contractions and five by atrial premature contractions. Paroxysmal ventricular tachycardia related to early ventricular premature contractions tended to last longer and failed to respond to therapy with lidocaine more often than paroxysmal ventricular tachycardia begun by late ventricular or atrial premature contractions. Ventricular fibrillation occurred in six cases of paroxysmal ventricular tachycardia due to early ventricular premature contractions but was absent in paroxysmal ventricular tachycardia related to late ventricular or atrial premature contractions. The “malignant” potential of a given ventricular premature contraction cannot be assessed from its degree of prematurity alone.

In 1940, Wiggers and Wegria demonstrated a “vulnerable period” in the canine ventricle during which single electrical shocks elicited repetitive ventricular firing. Later, Smirk and Palmer made similar observations in man and described the “R on T” phenomenon, i.e., the production of ventricular arrhythmias by ventricular premature contractions whose R wave interrupted the T wave of an antecedent normal beat. The concept of vulnerability was then widely accepted as an important determinant of the malignant potential of ventricular premature contractions. Newer experimental and clinical evidence suggests (1) that this arrhythmogenic tendency is not restricted to ventricular premature contractions exhibiting the “R on T” pattern, and (2) that serious ventricular arrhythmias may be initiated by ventricular premature contractions and even by atrial premature contractions occurring later in diastole. We analyzed 68 episodes of paroxysmal ventricular tachycardia in 42 consecutive patients with acute myocardial infarction, hoping to characterize the precursors of this arrhythmia with respect to their degree of prematurity, frequency, contour, and site of origin.

Materials and Methods

During an 18-month interval, using previously described methods and electromagnetic tape recordings, we monitored 424 consecutive patients admitted to the coronary care unit for a minimum of 72 hours for proved or suspected acute myocardial infarction. Monitoring was begun in all patients within 24 hours of the onset of symptoms.

Paroxysmal ventricular tachycardia was defined as six or more successive beats of ventricular origin at a rate greater than 100 beats per minute. We analyzed all ectopic beats preceding paroxysmal ventricular tachycardia for their frequency, site of origin or contour, occurrence in pairs or salvos (defined as two to four successive ventricular premature contractions), and degree of prematurity. The “prematurity index” of ventricular premature contractions was determined by dividing their coupling interval by the Q-T interval of the antecedent supraventricular beat. We designated “late” ventricular premature contractions as those with a prematurity index greater than one and “early” ventricular premature contractions as those with a prematurity index less than one. Those patients with more than an occasional ventricular premature contraction received lidocaine in a 100-mg bolus, followed by an infusion of 4 mg of lidocaine per minute.

Results

Constant monitoring revealed 68 instances of paroxysmal ventricular tachycardia in 42 patients with acute myocardial infarction confirmed by a typical history, appropriate enzymatic changes, and either new Q waves or typical ST-T changes of nontransmural infarction. There were 31 men and 11 women (age range, 29 to 82 years; mean age, 59 years). There were 22 anterior, 18 inferior, and two nontransmural infarcts.

Ventricular premature contractions with a frequency of greater than six per minute preceded 45 episodes (66 percent) of paroxysmal ventricular tachycardia, while multiform ventricular premature contractions were seen in 11 episodes (16 percent).
Salvoes or bursts of ventricular premature contractions initiated paroxysmal ventricular tachycardia on seven occasions (10 percent) without prior warning, while ventricular premature contractions of right and left ventricular origin occurred with nearly equal frequency 24 (35 percent) and 39 (57 percent) respectively. Atrial premature contractions or atrial arrhythmias introduced five bouts of paroxysmal ventricular tachycardia.

The degree of prematurity of the ventricular premature contractions responsible for paroxysmal ventricular tachycardia was of special interest in this series. Only 17 episodes of paroxysmal ventricular tachycardia were begun by early ventricular premature contractions (prematurity index < 1.0) (Fig 1), while 46 episodes were related to late ventricular premature contractions (prematurity index > 1.0) (Fig 2), and five owed their origin to atrial premature contractions (Fig 3). The mean duration of paroxysmal ventricular tachycardia begun by early ventricular premature contractions was 22 beats, while paroxysmal ventricular tachycardia related to late ventricular and atrial premature contractions was more transient (eight and six beats, respectively). Administration of lidocaine terminated only four (24 percent) of the 17 episodes of paroxysmal ventricular tachycardia due to early ventricular premature contractions, while therapy with this drug was much more effective in paroxysmal ventricular tachycardia due to late ventricular and atrial premature contractions; such therapy abolished 41 (89 percent) of the 46 episodes of paroxysmal ventricular tachycardia due to late ventricular premature contractions and all five instances of paroxysmal ventricular tachycardia due to atrial premature contractions. Interestingly, six (35 percent) of the 17 bouts of paroxysmal ventricular tachycardia due to early ventricular premature contractions deteriorated into ventricular fibrillation despite therapy with lidocaine, while ventricular fibrillation failed to occur in paroxysmal ventricular tachycardia due to late ventricular or atrial premature contractions.

**DISCUSSION**

Our data concerning the frequency of ventricular premature contractions preceding paroxysmal ventricular tachycardia and their contour, site of origin,
FIGURE 3. Strips A and B were obtained several hours apart during first day of hospitalization of 48-year-old man with acute anteroseptal infarction. In each strip, fourth beat is atrial premature contraction that introduces short episode of paroxysmal ventricular tachycardia. This arrhythmia was easily terminated by therapy with lidocaine, and course of patient's illness while he was hospitalized was benign.

and occurrence in pairs or salvos are consistent with previous reports. \(^6\) \(^9\) Initiation of paroxysmal ventricular tachycardia by atrial premature contractions has also been described by others in clinical and experimental models associated with coronary occlusion, \(^11\) electrolyte imbalance, \(^15\) atrioventricular nodal bypass tracts, \(^13\) and prolonged Q-T intervals. \(^14\) Basically, these situations involve nonuniform ventricular depolarization owing to the prematurity of the atrial premature contraction or to defects in ventricular conduction.

The observation that most episodes of paroxysmal ventricular tachycardia in our patients were begun by late ventricular premature contractions, rather than early ones, supports recent experimental and clinical evidence that "malignant" ventricular premature contractions need not exhibit the "R on T" phenomenon. \(^5\) \(^9\) It is becoming increasingly evident that in patients with acute myocardial infarction, the malignant potential of a ventricular premature contraction is related more to the underlying changes in activation of the ischemic myocardium than to its degree of prematurity. \(^5\) Ischemia increases the duration of the vulnerable period by causing dispersion of excitation owing to local areas of delay in conduction. These produce reentrant ventricular premature contractions that lead to further fragmentation of activation, reentry, and sustained paroxysmal ventricular tachycardia.

The relationship between delay and fragmentation of potentials recorded from ischemic myocardium, prolonged ventricular activation time, and ventricular arrhythmias has been clearly established. \(^4\) \(^5\) \(^8\) \(^9\) \(^15\)

We must agree with those who have stated that the likelihood of a given episode of ventricular premature contractions to engender paroxysmal ventricular tachycardia cannot be determined from its degree of prematurity alone. \(^4\) \(^5\) \(^9\)

Finally, we are intrigued by the major differences in the behavior of paroxysmal ventricular tachycardia that are apparently related to its onset. In our series, paroxysmal ventricular tachycardia began by early ventricular premature contractions lasted longer and failed to respond to therapy with lidocaine more often than paroxysmal ventricular tachycardia initiated by late ventricular premature contractions. Further, ventricular fibrillation occurred only in paroxysmal ventricular tachycardia related to early ventricular premature contractions. Since, to our knowledge, these observations have not been previously reported, more investigation is necessary to confirm or refute them.

REFERENCES

1 Wiggers CJ, Wegria R: Ventricular fibrillation due to single, localized induction and condenser shocks applied during the vulnerable phase of ventricular systole. Am J Physiol 128:500-505, 1940
Turkey

The rather incongruous name, turkey, now used for the birds, both wild and domesticated, is closely connected with the history of domestication of the fowls. When the Spaniards conquered the Aztecs in Mexico, they found that the Indians had domesticated an interesting bird; the males strutted around in pompous fashion and made peculiar gobbling noises. The Spaniards introduced these birds into Europe, where the people promptly confused them with guinea fowls. The guinea fowl, a native of Africa, was known as a turkey in some areas because some of the domesticated stock had been exported from Turkey. Out of this confusion, the American fowls were also called turkeys. Our ancestors hunted the birds so enthusiastically that they were virtually exterminated in the New England states before 1850. The idea was conceived of increasing the population of wild turkeys by rearing the birds in captivity and releasing them at appropriate times. Wild turkeys were successfully reared in captivity; the difficulty arises at graduation. Most of them refuse to become wild. The birds often proceed to join the first flock of tame turkeys they contact.