Sudden Death during Ambulatory Monitoring
Clinical and Electrocardiographic Correlations
Report of a Case

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SUMMARY A man with known coronary heart disease underwent treadmill exercise testing to determine his functional capacity. The test was negative for ischemia. Ventricular ectopic activity was noted at rest and in the recovery period. On the same day, while viewing a sporting event at home, the patient died suddenly. An ambulatory electrocardiographic recording documented ventricular fibrillation as the terminal mechanism. Ventricular ectopic activity and heart rate increased in the two hours prior to death, and ischemic ST-segment depression was noted at the time of the terminal arrhythmia. It is postulated that myocardial ischemia and catecholamine response lowered the threshold to ventricular fibrillation, thus facilitating the emergence of the fatal arrhythmia.

VENTRICULAR FIBRILLATION has been assumed to be the proximate cause of most cases of sudden, unexpected death which occur within minutes of collapse. Recent studies from mobile rescue teams have documented that ventricular fibrillation is indeed usually present at the time electrocardiographic monitoring is instituted in these victims. However, such records begin at least several minutes subsequent to the cardiovascular collapse, and documentation of the actual mechanism of the arrhythmic death in ambulatory subjects has not appeared in the literature. This case concerns a patient who died suddenly and unexpectedly at home while wearing an ambulatory electrocardiographic monitor.

Case Report

The patient, a 53-year-old retired systems analyst, sustained the first of three documented myocardial infarctions at age 36. Frequent premature ventricular contractions, first noted in 1972 after his third myocardial infarction, were treated with a variety of antiarrhythmic drugs. Procainamide had been discontinued after the onset of fever and arthralgias several weeks before death. When evaluated on the day of his death, the patient denied angina pectoris, dizziness, palpitation, or symptoms of congestive heart failure other than moderate exertional dyspnea. Physical examination of the cardiovascular system was unremarkable except for a presystolic gallop. A resting electrocardiogram showed evidence of an old inferior myocardial infarction and multifiform premature ventricular contractions (PVCs). A treadmill exercise test, performed to evaluate functional capacity, was carried out according to the Naughton protocol. This test was terminated at a heart rate of 125 beats/minute due to fatigue and dyspnea. The maximum workload was 6 METs, or multiples of resting oxygen consumption. No ischemic ST-segment abnormalities were observed. In the first three minutes of the recovery period, frequent multiform PVCs, some occurring in couplets, and a short period of slow idioventricular rhythm were noted. The patient remained asymptomatic throughout the entire recovery period and at five minutes after cessation of exercise, PVCs were again infrequent.

The patient returned home wearing an ambulatory electrocardiographic monitoring device. After dinner, while lying down, viewing an exciting televised basketball game, the patient made a choking sound and was found in an unresponsive state. An ambulance was summoned and the patient was taken to a nearby hospital where he was pronounced dead on arrival, approximately eight hours after the exercise test. No autopsy was performed.

Review of the first seven hours of ambulatory electrocardiographic data disclosed frequent multiform PVCs averaging approximately 5/minute, with occasional couplets (fig. 1). During the last 1½ hours of life, the PVC frequency increased to approximately 11/minute with couplets but no ventricular tachycardia. Heart rate, which had ranged from 80 to 96 beats/minute throughout most of the monitoring period, also increased in the final 1½ hours of life, reaching a maximum of 130 beats/minute during the last minutes (fig. 2). Review of the electrocardiogram revealed several periods, including the hour preceding death, of significant downsloping ST-segment depression, suggesting that ischemia was present. The terminal arrhythmic sequence was that of a couplet with two different morphologies, followed by a sinus beat, and another PVC with a coupling interval of approximately 0.36 seconds initiating rapid ventricular flutter which degenerated into ventricular fibrillation (fig. 3).

Discussion

The episode of sudden death described here occurred without warning in a high risk patient with known coronary heart disease and frequent PVCs. The role of the treadmill exercise test in the fatal sequence of events cannot be excluded, but the lack of chest pain or ischemic ST-segment abnormalities during the test suggests that it was not a significant factor.

Ambulatory monitoring identified certain electrocardiographic antecedents of the fatal arrhythmia. The heart rate, a major determinant of myocardial oxygen demands, increased immediately before death to a level even higher than that reached during the exercise test. This was accompanied by an increased frequency of PVCs during the last two hours of life. ST-segment depression, of fluctuating in-

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tensity throughout the monitoring period, was definitely ischemic at the time of death. This phenomenon of ischemic ST-segment abnormalities seen in ambulatory monitoring but not by treadmill exercise testing, has been noted by other authors, and underscores the importance of ambulatory monitoring in the detection of ischemia in circumstances other than those of physical exercise.

In addition, the excitement of viewing a championship sports event probably produced a catecholamine release — also known to be an important factor in the production of cardiac arrhythmias. An interesting feature of the fatal arrhythmia in this patient was that the PVC initiating it occurred after a long cycle length which followed a pair of coupled PVCs (fig. 3). This situation is known to result in an increased temporal dispersion of the refractory periods of myocardial cells.

We presume that the combination of ischemia and catecholamine release resulted in a lowered ventricular fibrillation threshold and provided the opportunity for a PVC — no different from thousands of others the patient had experienced on the same day — to initiate ventricular fibrillation and sudden death.

References

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