Two monitored cases of sudden death outside hospital¹

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SUMMARY Two patients known to have coronary artery disease died suddenly outdoors while active. Neither had symptoms or signs of acute myocardial infarction. They were being monitored by continuous tape recording of the electrocardiogram at the time of death. In one patient the cause of death was cardiac arrest, preceded by bigeminy and multiform ventricular ectopic beats, in the other ventricular fibrillation preceded by atrial fibrillation, multiform ventricular ectopic beats, and ST depression. These observations are added to the limited reported cases in which the mechanism leading to sudden death outside the hospital is recorded.

A large proportion of the total mortality from acute coronary events occurs outside hospital (World Health Organisation, 1976). Continuous monitoring of patients with acute myocardial infarction in coronary care units has now provided information about life-threatening and lethal arrhythmias (Lown, 1966; Mogensen, 1970; DeSanctis *et al.*, 1972). However, few cases have been documented with arrhythmias preceding and leading to sudden death outside hospital. Monitored cases are described by Bleifer *et al.* (1974) and Gradman *et al.* (1977). Baum *et al.* (1974) and Liberthson *et al.* (1974) reported arrhythmias monitored a few minutes after the acute attack.

The increased use of portable tape recorders has increased the chance of monitoring the electrocardiogram of patients who die suddenly while active in their usual occupation. Two such cases form the substance of this report, and can be added to the few cases thus far recorded.

Case reports

CASE 1

An 81-year-old man, who had had angina pectoris for many years, was referred to the clinic by his general practitioner for evaluation of his cardiac condition. Thirteen years earlier the typical pattern of an inferior infarction was found in an electrocardiogram taken for routine purposes. In recent weeks he had increasing anginal symptoms and some

dyspnoea. At physical examination there were no abnormalities. The blood pressure was 160/80 mmHg, the heart rate was regular at 84 beats/minute. The electrocardiogram again showed the old inferior infarction, left ventricular hypertrophy, and ischaemic ST changes. No signs of fresh infarction were present. The serum levels of creatinine phosphokinase, α -hydroxybutyric dehydrogenase, and glutamic oxaloacetic transaminase were normal. In addition, serum levels of sodium at 143 mmol/l and potassium at 3.8 mmol/l were normal.

After attachment of a tape recorder in order to monitor the electrocardiogram during his usual activities, the patient was sent home. About two hours later the patient collapsed while walking outdoors. The ambulance service tried cardiac massage and artificial respiration but the patient remained unconscious and died about half an hour later.

Strips of the continuous electrocardiogram (Fig. 1) show at the outset periods with bigeminy and multiform ventricular ectopic beats. A few minutes before the syncope the ventricular rhythm slowed down without recognisable P waves but with normal ventricular complexes. This was followed by a cardiac arrest of about 2 minutes' duration. During the next half hour the rate varied from 40 to 80 beats per minute, while the ventricular rhythm showed bizarre complexes alternating with arrest. The last strip shows the moment the patient died in asystole. No necropsy was done.

CASE 2

This 63-year-old man was already under treatment by his cardiologist for 15 years because of angina

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Fig. 1 Case 1. Sudden death caused by arrest. This figure shows a series of strips taken from a continuously recorded electrocardiogram, with a portable tape recorder. At 10.15 h a period of bigeminy and multiform ventricular beats; at 11.13 h again a period of bigeminy; at 12.32 h decrease of the heart rate followed by an arrest of about 2 minutes. At 13.08 h final arrest.

pectoris and hypertension. Twelve years ago he had had a slight myocardial infarction. Six months previously he suddenly collapsed. On the electrocardiogram then taken atrial fibrillation was present. The patient was treated with anticoagulants, digoxin, and isosorbide dinitrate. In recent days symptoms of dizziness and syncope returned and he was referred to us. The electrocardiogram again showed atrial fibrillation and an old anterior myocardial infarction but no signs of ischaemia or fresh infarction. No laboratory tests were done. Since the patient felt well he was sent home with a tape recorder for monitoring his electrocardiogram. In the afternoon without warning he died suddenly while washing his car. The ambulant electrocardiogram (Fig. 2) showed atrial fibrillation with some multiform ventricular ectopic beats. A few minutes before death the ventricular rate increased, ST depression appeared, and after a ventricular ectopic beat ventricular flutter started. It finally changed

into ventricular fibrillation just before death. No necropsy was done.

Fig. 2 Case 2. Sudden death caused by ventricular fibrillation. This figure shows a series of strips taken from a continuous electrocardiogram recorded with a portable tape recorder. At 14.35 h and 15.16 h atrial fibrillation with multiform ventricular ectopic beats; at 15.20 h increased heart rate, ST depression, and a ventricular ectopic beat followed by ventricular flutter; 15.22 h alternating ventricular flutter and ventricular fibrillation; at 15.31 h rough and at 15.36 h fine ventricular fibrillation.

Discussion

Two cases of primary sudden death are presented. History and physical findings were negative, while biochemical and electrocardiographic signs of an acute myocardial infarction were absent, though both patients had had angina pectoris for many years and had had myocardial infarcts years ago. This finding is in agreement with the results of Baum *et al.* (1974) who reported that in many (50.5%) resuscitated cases of primary out-of-hospital ventricular fibrillation no acute myocardial infarction could be shown. Nevertheless, 94 per cent of their patients who were subsequently studied by coronary arteriograms showed severe coronary

artery disease. In the first reported case the apparent cause of death is cardiac arrest and not ventricular fibrillation. Premonitory symptoms of dizziness and syncope were missing and the first clinical attack proved lethal. On the other hand the arrest was preceded by warning arrhythmias like bigeminy and multiform ventricular ectopics, which could have been harbingers of imminent ventricular fibrillation. In fact asystole took place. Liberthson *et al.* (1974) reported that in 28 per cent of cases of prehospital sudden coronary death monitored by fire rescue squads, terminal rhythms other than ventricular fibrillation, such as bradycardia and asystole, were found.

The second case is a typical example of primary ventricular fibrillation preceded by warning arrhythmias in a patient with known coronary atherosclerotic heart disease. The finding of atrial fibrillation probably reassured the physician in the interpretation of the syncope of his patient. This experience indicates that ambulatory electrocardiography may improve diagnostic accuracy of the physician in such patients.

Surprisingly, few cases of sudden death with rhythm monitoring outside hospital are reported. Gradman *et al.* (1977) reported a case of primary ventricular fibrillation quite similar to case 2. Warning arrhythmias, tachycardia, and ST depression preceded the acute attack. Bleifer *et al.* (1974) described a case of ventricular fibrillation in a patient with a symptomless acute myocardial infarction.

Hinkle (1978) saw a case of primary ventricular fibrillation also preceded by warning arrhythmias.

The now widespread and still increasing use of ambulant monitoring and the reported predictive value of arrhythmias (Oliver *et al.*, 1974; Ryan *et al.*, 1975; Van Durme, 1975; Moss *et al.*, 1975) should enhance the chance of finding more such cases. It is hoped that pooling of this type of information will increase the knowledge about lethal arrhythmias and its prodromal warnings in order to improve our management of secondary prevention.

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