

# Sudden death after spontaneous ventricular fibrillation: case reports

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**SUMMARY.** *Patients resuscitated from ventricular fibrillation in the absence of myocardial infarction remain at high risk of sudden death, even if underlying risk factors are treated. Two such cases are reported here.*

## Introduction

VENTRICULAR fibrillation in coronary heart disease may follow acute myocardial infarction or may be spontaneous, producing unexpected sudden death. Because acute myocardial infarction often produces symptoms which result in medical help being called before ventricular fibrillation can supervene, most patients resuscitated from ventricular fibrillation come from the infarct group. Infarct patients who are resuscitated and survive to leave hospital have as good a long term prognosis as those who have not suffered this complication.<sup>1-3</sup> The spread of programmes to train the non-medical public to perform cardiopulmonary resuscitation with the back up of portable defibrillators carried by general practitioners and ambulances should result in more patients surviving an episode of spontaneous ventricular fibrillation.<sup>4,5</sup> This may not, however, greatly prolong their lives.

## Case reports

**Case 1.** Mrs A, aged 56 years, collapsed after walking round a 4 km 'fun run' course on 30 September 1986. She was carried into the medical tent where she was found to be in ventricular fibrillation. Defibrillation with a single 50 J shock converted her to fast atrial fibrillation and she was transferred to hospital. By the morning she was conscious and back in sinus rhythm. She gave no history of previous cardiac illness and investigation showed no evidence of infarction. Coronary angiography revealed occlusion of the right coronary artery and 70% narrowing of the left anterior descending artery. On 7 November 1986 triple vessel coronary artery bypass grafting was performed. There were no immediate post-operative complications, though there was an enzyme rise which suggested peri-operative infarction. On 11 November she was up and about, but the next day she suffered a further cardiac arrest from which she could not be resuscitated. A postmortem was not performed.

**Case 2.** Mr B, aged 68 years, suffered an acute myocardial infarct on 12 August 1986. He had been treated for mild hypertension with triamterene/hydrochlorothiazide, two tablets daily, since 27 September 1985 and was also taking glibenclamide 10 mg daily for diabetes mellitus. He was treated in hospital for 10 days, during which time he had brief episodes of atrial fibrillation and pericarditis. His drug regimen on discharge was unchanged. On 6 October 1986 he started on a course of thrice weekly medically supervised cardiac rehabilitation.<sup>6</sup> He was

making excellent progress on the course when, on 28 November 1986, he went into ventricular fibrillation towards the end of an exercise session. He received a 50 J shock and returned to sinus rhythm with multiple ventricular ectopics, but repeatedly went back into ventricular fibrillation. He reverted spontaneously from some of these episodes but most required DC conversion. He was transferred to hospital where he was treated with a lignocaine drip, and within two hours of the original arrest had stopped going into ventricular fibrillation. During that time he had received between 20 and 25 DC shocks. Investigation revealed no evidence of a further infarct, but showed a plasma potassium concentration of 2.3 mM. He was given intravenous and oral potassium supplements, which increased the level to 4.4 mM. He was discharged on 5 December 1986 on amiodarone 200 mg twice daily. He remained well until 19 December when he was found dead in his garden where he had been digging. A post-mortem was not performed.

## Discussion

The electrophysiology of sudden death after acute myocardial infarction is different to that in patients who have not infarcted. After infarction a very early ventricular ectopic (the R-on-T phenomenon) is the usual precipitating event.

In the absence of infarction, ventricular fibrillation is usually the outcome of prolonged and unusually rapid ventricular tachycardia occurring against a background of increasing ventricular ectopic activity.<sup>7-9</sup> If the patient is resuscitated, the underlying tendency to ventricular irritability is not altered, and he remains at high risk for sudden death, with a two year mortality of nearly 50%.<sup>10</sup> No antiarrhythmic drug has been shown to lower this risk,<sup>11</sup> though it is possible that the long term benefit of beta-blockers after myocardial infarction is due to this effect. Most people who die suddenly without infarction have severe coronary artery disease,<sup>12</sup> which is previously recognized in about 40% of them.<sup>13</sup> The chief predictors of sudden death in patients with overt coronary disease are extensive myocardial damage and cardiac failure<sup>13</sup> but those who have been resuscitated from previous non-infarct related cardiac arrest are at far higher risk than any other group. These patients would benefit from implanted defibrillators, whether or not they have correctable underlying contributory factors, such as coronary stenoses or electrolyte disturbance. As the number of people in this category increases, the cost of implanted defibrillators is likely to become a contentious issue.

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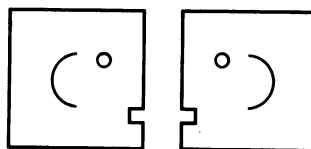
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