ABSTRACT—The objective of our study was to characterise 52 hospital survivors of pre-hospital ventricular fibrillation and record their initial management in hospital. A retrospective review was undertaken of ambulance report forms, hospital notes, and electrocardiograms, in one teaching hospital and three district general hospitals in South Wales, of 53 patients discharged from hospital between February 1987 and April 1992 after resuscitation from pre-hospital ventricular fibrillation by ambulance personnel. Twenty patients showed evidence of acute myocardial infarction (group 1), eight patients had a diagnosis of possible acute myocardial infarction (group 2), and 25 patients had no evidence of acute myocardial infarction (group 3). Nineteen patients in group 1 experienced chest pain before collapse compared with only six patients in group 3 (p < 0.001). Five patients in group 1 had a previous history of ischaemic heart disease compared with 17 patients in group 3 (p < 0.01). A greater proportion of patients in group 3 were taking diuretic medication (15 of 25 vs 4 of 20; p < 0.01) but there was no difference in potassium levels on admission to hospital. Cardiologists were involved in the management of a minority of patients (21 of 53); only eight patients underwent cardiac catheterisation; and only three were referred for electrophysiological studies. Patients in group 3 were more likely to be discharged taking empiric antiarrhythmic drugs (13 of 25) than patients in group 1 (2 of 20) (p < 0.01). Not enough use is made of noninvasive and invasive investigations in the management of survivors of pre-hospital ventricular fibrillation. We propose guidelines for the appropriate management of such patients by clinicians involved in their care, allowing more logical treatment to be aimed at reducing the likelihood of sudden cardiac death.

The majority of patients resuscitated after cardiac arrest from out-of-hospital ventricular fibrillation by ambulance paramedics do not, subsequently, show evidence of acute Q-wave myocardial infarction [1,2], yet these patients are at high risk of further cardiac arrest and sudden death [3,4]. To reduce this risk it would be logical to institute appropriate coronary revascularisation [5], drug therapy guided by electrophysiology studies [6], and implantation of automatic defibrillators [7].

We wished to characterise our series of survivors of pre-hospital cardiac arrest and to assess the management during their initial hospitalisations.

Method

We reviewed ambulance report forms, hospital notes, and discharge summaries of patients resuscitated from out-of-hospital ventricular fibrillation between February 1987 and April 1992 by South Glamorgan ambulance service personnel. South Glamorgan is an urban and semi-rural county with a population of 396,000 contained in 187 square miles. Patients were admitted to one of four hospitals—three district general hospitals (two with coronary care units run by general physicians and one with an intensive care unit run by anaesthetists) and one teaching hospital offering invasive cardiology and cardiac surgical services [8]. Cardiologists managed patients admitted to the coronary care unit in the teaching hospital, and were readily available for advice in one district general hospital. The remaining hospitals had no designated, trained cardiologist. Facilities for echocardiography and 24-hour electrocardiogram monitoring were available in all hospitals, exercise stress-testing was performed in three of the hospitals, and cardiac catheter facilities were based in the teaching hospital. Signal-averaged electrocardiography was not routinely performed during the period of study. The region did not possess an electrophysiology service.

Patients were categorised into three groups:

Group 1: definite acute myocardial infarction, as judged by sequential electrocardiogram changes, including the development of new Q-waves.

Group 2: possible acute myocardial infarction, as judged by new ST/T-wave abnormalities persisting for the duration of hospital admission, without Q-wave development but with elevation of serum creatine kinase levels.

Group 3: no evidence of acute myocardial infarction, as judged by transient ST/T-wave changes without Q-wave development.
A rise in the serum creatine kinase levels was felt to be an unreliable sign of acute myocardial infarction [9], though its absence indicated the absence of myocardial infarction.

Continuous variables are expressed as mean ± 95% confidence intervals, and are compared between groups using the unpaired t-test, while the remaining characteristics are analysed using the chi-squared test. P values of less than 0.05 are judged to be significant.

Results (Tables 1–5)

During the study, 56 patients were discharged from hospital having been resuscitated from out of hospital ventricular fibrillation. Relevant hospital notes were available for 53 patients and these are reported here.

Group 1
Twenty patients (37.7% of total) were discharged with a definite diagnosis of Q-wave myocardial infarction; seven anterior and 15 inferior myocardial infarctions. There were 13 men and seven women with mean age 62.5 years (95% confidence interval 57.4–67.6 years). Five of them had a previous history of ischaemic heart disease and all but one had chest pain before their cardiac arrest. Seven had recurrent cardiac arrests in hospital (six had ventricular fibrillation, one showed electromechanical dissociation).

Eleven patients received intravenous thrombolytic therapy. Cardiologists were involved in the management of nine patients, with a minority of patients undergoing non-invasive investigations and three having cardiac catheterisation (one undergoing coronary

| Table 3. Initial hospital course and management |
|--------------------------------------------|
| Group 1 | Group 2 | Group 3 |
|----------------|---------|---------|
| In hospital VF | 7 | 2 | 8 |
| Thrombolytic | 11 * | 3 | 3 |
| Lignocaine | 12 | 5 | 14 |
| Other antihrrythmics | 5 | 2 | 13 |

* P < 0.01 vs Group 3.
VF = ventricular fibrillation.

| Table 4. In-hospital management and investigation |
|---------------------------------------------|
| Group 1 | Group 2 | Group 3 |
|----------------|---------|---------|
| Cardiologist review | 9 | 4 | 8 |
| 24-hr tape | 5 | 1 | 10 |
| Echo | 5 | 1 | 4 |
| Exercise test | 2 | 1 | 5 |
| Catheter | 3 | 1 | 4 |
| EPS | 1 | 0 | 2 |

EPS = Electrophysiological study.
Echo = Echocardiogram.

| Table 5. Drugs on discharge |
|----------------------------|
| Group 1 | Group 2 | Group 3 |
|----------------|---------|---------|
| Diuretic | 6 | 5 | 11 |
| Digoxin | 1 | 2 | 3 |
| Betablocker | 9 | 2 | 5 |
| Aspirin | 17 | 3 | 15 |
| Antiarrhythmics | 2 * | 1 | 13 |
| ACE inhibitors | 2 | 2 | 4 |
| Warfarin | 1 | 2 | 3 |
| Calcium antagonist | 4 | 0 | 4 |
| Nitrate | 3 | 1 | 3 |

* P < 0.01 vs Group 3.
artery surgery). One patient was referred for electrophysiological studies.

On discharge from hospital 17 patients were taking aspirin, nine a betablocker, and two were receiving antiarrhythmic therapy.

**Group 2**

The eight patients in group 2 were significantly older ($p < 0.05$) than patients in the other two groups: mean age 72.4 years (95% confidence interval 64.2–80.5). Three patients had a past history of ischaemic heart disease. A further three, who had chest pain before the cardiac arrest, received thrombolytic therapy. Cardiologists were involved in the management of half of the patients. Only one patient had cardiac catheterisation during the hospitalisation and then declined surgery. Five patients were discharged taking diuretics, and one took an antiarrhythmic drug.

**Group 3**

There was no evidence of myocardial infarction in 25 (47.1% of total) patients. There were 20 men and five women with mean age 63.34 years (95% confidence interval 59.4–67.4 years). These patients were significantly more likely to have a past history of ischaemic heart disease ($p < 0.01$) and less likely to have suffered chest pain prior to collapse than patients in group 1 ($p < 0.001$). They were more likely to be taking diuretic therapy at the time of collapse ($p < 0.01$) although there was no significant difference in the serum potassium level at the time of admission to hospital.

Ventricular fibrillation recurred in hospital in eight of these patients, and three patients received intravenous thrombolytic therapy on admission.

Eight patients were seen by cardiologists during their hospitalisation and four of them underwent cardiac catheterisation while in hospital; two of the remainder were readmitted some weeks after discharge from hospital for routine coronary angiography. Only two patients were referred for electrophysiological studies. In 10 of the 17 patients who did not see a cardiologist the only investigations carried out were serial resting electrocardiogram recordings and estimates of serum levels of cardiac enzyme activity. Patients in group 3 were significantly more likely ($p < 0.01$) to be discharged on an antiarrhythmic drug than those in group 1; 11 of 13 taking amiodarone.

**Discussion**

The characteristics of the patients discharged from hospital following resuscitation from out-of-hospital ventricular fibrillation are similar to previous reports [1,10]. A minority of cardiac arrests in these patients are classified as secondary to an acute myocardial infarction, while the remainder are due either to acute ischaemia without progression to infarction [11], or to arrhythmias independent of an acute ischaemic process (primary arrhythmic events) [12]. In patients experiencing chest pain before collapse, the proportion of cases of acute myocardial infarction [4] is likely to be greater. The expected mortality rates of survivors of out-of-hospital ventricular fibrillation are greater than the expected mortality rates of the general population or of patients discharged following acute myocardial infarction [13]. Dickey et al report 1, 2, 5 and 10-year survival rates of 76%, 66%, 41%, and 27% in patients resuscitated from ventricular fibrillation by a mobile coronary care unit [4]. Factors significantly associated with increased long-term mortality were:

- patient age greater than 60 years;
- ventricular fibrillation due to ischaemic heart disease without infarction;
- a history of previous myocardial infarction;
- a history of hypertension;
- digoxin and diuretic therapy before ventricular fibrillation;
- digoxin as discharge medication;
- failure to stop smoking after discharge from hospital.

A review of the Seattle data [14] revealed the following univariate predictors of recurrent cardiac arrest in resuscitated survivors of ventricular fibrillation: abnormal left ventricular function (associated with previous myocardial infarction or history of congestive heart failure), angiographic evidence of extensive coronary artery disease, ventricular fibrillation without new Q-wave development, male gender, advanced age, exercise-induced angina or hypotension, complex ectopy on Holter monitoring, inducibility of ventricular tachycardia at electrophysiological studies, and continuing smoking.

Although we do not present long-term follow-up data for our patient groups, it can be assumed that these patients, particularly those in group 3, with no evidence of acute myocardial infarction at the time of the pre-hospital cardiac arrest, are at high risk of further cardiac arrest and death. Furthermore, recurrent cardiac events often occur within the first six to twelve months after discharge from hospital [1,4,15]. Assessing these patients adequately and planning logical treatment for them is therefore best performed during the initial hospitalisation [16]. This should ideally include an assessment of cardiac anatomy and left ventricular function (by echocardiography and exercise testing), coronary anatomy and ischaemic burden (by coronary angiography and exercise testing), and residual arrhythmic substrate (by electrocardiogram monitoring, signal-averaged electrocardiograms [17] and electrophysiological studies [6]).

It has been argued that nearly all survivors of out-of-hospital ventricular fibrillation who have not suffered an acute myocardial infarction should be investigated by cardiac catheterisation to seek significant coronary lesions suitable for intervention [16,18,19], and that
probably all patients should undergo electrophysiological testing [16].

In a study of the management of 36 patients with late (48-hour to 12-year) post-infarct ventricular tachycardia presenting to a district general hospital with close liaison to a regional cardiothoracic centre specialising in electrophysiology, 12 patients were treated empirically and 24 underwent electrophysiological studies [20]; coronary angiography was undertaken in 23 of the latter group. As a result of the studies, 16 patients were treated with antiarrhythmic drugs, three by anti-ischaemic measures (drugs/coronary bypass grafting), and five by non-pharmacological antiarrhythmic treatments. Electrophysiological assessment of 37 survivors of cardiac arrest not associated with acute myocardial infarction, resulted in the implantation of 12 cardioverter defibrillators; four patients underwent cardiac surgery, and 11 were prescribed antiarrhythmic drugs [21].

In our study, 13 of 25 survivors with no evidence of acute myocardial infarction were discharged taking empirical antiarrhythmic therapy (mainly amiodarone). Such empiric antiarrhythmic treatment does not affect total mortality and may, in some cases, be associated with an increased frequency of sudden death [22], though this survey did not study the use of amiodarone. However, up to 40% of patients on long-term amiodarone therapy will develop drug-related adverse effects over a five-year period, requiring discontinuation of treatment [23].

In our series, limited use was made of non-invasive and invasive investigations during the initial hospitalisation. Less than one-third of high-risk patients (group 3) were reviewed by a cardiologist, and only four of 25 underwent cardiac catheterisation; only two of them were referred for electrophysiological studies. The underuse of investigations could be related to the lack of full cardiological facilities in some or all of the hospitals, though echocardiography and Holter monitoring were available in all hospitals. Two of the district general hospitals had no cardiologists on the staff; therefore, for patients to be assessed by a cardiologist during hospitalisation would have required an inter-hospital transfer; none took place. Moreover, of those high-risk patients who were reviewed by a cardiologist, only 25% (two of eight) were referred for electrophysiological studies. This relatively low percentage may reflect the lack of this facility within the region and the need for them to be performed in a specialist centre outside the region. This experience is not unique to our hospitals. For example, only 13% of Scottish patients surviving pre-hospital primary ventricular fibrillation underwent electrophysiological studies [24].

The increasing deployment of ambulance paramedics and defibrillators will lead to more patients being admitted to hospital after resuscitation from pre-hospital ventricular fibrillation. Their subsequent management is variable and does not utilise all available investigations that might help to assess the likelihood of recurrent ventricular fibrillation. We believe that clinicians involved in the care of these patients should have guidelines so that they can offer more logical therapy.

ECG = electrocardiogram;

Fig 1. Options for management

CK = creatine kinase;
AMI = acute myocardial infarction;
VF = ventricular fibrillation;
LV = left ventricle;
MUGA = multiple uptake gated angiogram;
NMR = nuclear magnetic resonance imaging;
CABG = coronary artery bypass grafting;
PTCA = percutaneous transcoronary angioplasty;
EPS = electrophysiological study.

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Errata

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Pages 111–5. West: A look at the statistical overview

The legends to Figs 1 and 3 were interchanged.

Fig 1 shows a statistical overview of seven cohort studies in acute myeloblastic leukaemia.

Fig 3 gives a statistical overview of 17 trials of rehabilitation after myocardial infarction.

Page 172. Moxon: Microbes, molecules and man

Fig 5 — the second sentence of the legend should read: ‘The *bex* genes (A,B,C,D, indicated by light grey rectangles) are provisionally considered to be required for export of the polysaccharide from across the cytoplasm membrane.’