Survival after Resuscitation from Out-of-Hospital Ventricular Fibrillation

By Robert S. Baum, M.D., Hernan Alvarez III, M.D., and Leonard A. Cobb, M.D.

SUMMARY
During the first three years in operating a comprehensive system for the management of out-of-hospital medical emergencies, 146 patients were resuscitated from ventricular fibrillation, hospitalized, and discharged home. The diagnosis of acute transmural myocardial infarction associated with the episode of ventricular fibrillation was confirmed in only 17% of the patients. The presence of myocardial necrosis, based on either evidence of new transmural infarction or LDH-isoenzyme criteria was established in 49.5% of the patients. During the follow-up period, averaging 418 days, 43 of the 146 patients died. Thirty-four of the 43 deaths occurred suddenly outside the hospital. Patients whose aborted sudden cardiac death was associated with acute transmural infarction had a mortality rate of 14% after two years of follow-up. In contrast, patients without evidence of acute myocardial necrosis had a high mortality rate — 47% at two years.

It is concluded that: 1) out-of-hospital ventricular fibrillation is common and treatable; 2) the phenomenon of sudden cardiac death should not be equated with acute myocardial infarction; 3) patients resuscitated from ventricular fibrillation without associated acute myocardial infarction are prone to sudden death — most likely from ventricular fibrillation.

Additional Indexing Words:
Sudden death Coronary heart disease Myocardial infarction
Mobile intensive care units

Sudden cardiac death is a health problem of enormous magnitude in several areas of the world, including the United States. Approximately two-thirds of patients who die from coronary artery disease do so before reaching medical care. These out-of-hospital sudden cardiac deaths were a major stimulus in the development of a comprehensive system for the management of out-of-hospital medical emergencies in Seattle. During the first three years of operation, 821 patients were unconscious, pulseless, and in ventricular fibrillation when medical assistance first arrived. In addition, 65 patients developed ventricular fibrillation after the arrival of the mobile units. This paper is a report on the follow-up of 146 long-term survivors of out-of-hospital ventricular fibrillation.

Methods
The system, referred to as Medic I, is operated through the Seattle Fire Department with medical supervision by Harborview Medical Center and the University of Washington. A single emergency telephone number (911) is utilized in this city which has a population of about 500,000 persons in an area of 92 square miles. Medic I provides a three-tiered response, with the first response time averaging about three minutes after dispatch. Initial response is provided by one of ten district aid units, staffed by firefighters trained as emergency medical technicians; in instances of major health crises, a fire engine may also be dispatched in order to ensure rapid response. The three secondary response units are staffed by advanced level paramedical personnel who have completed a 1,000 hour training program and are proficient in a wide variety of techniques and procedures appropriate for out-of-hospital emergencies. The secondary response units are dispatched in instances of suspected myocardial infarction, sudden death, drowning, multiple trauma and other life-threatening situations. When indicated, patients are transported to one of fourteen metropolitan hospitals selected by the patient, his physician, or his family. Appropriate hospital bed availability (i.e., coronary care unit bed) is determined before or during transportation of the patient.

Between March 7, 1970 and March 6, 1973, 143 patients were resuscitated from ventricular fibrillation and subsequently discharged home from the hospital. Three of these patients were successfully resuscitated from out-of-hospital ventricular fibrillation on two separate occasions, making a total of 146 patients followed up after hospital discharge. For the purposes of this study, follow-up was terminated on June 30, 1973; the duration of follow-up averaged 418 days.

From the Department of Medicine (Division of Cardiology), Harborview Medical Center and University of Washington, Seattle, Washington.
Supported by Contract NIH NOI-HL-12474 and Training Grant HL-05281-14 from the National Heart and Lung Institute. Dr. Baum was a Research Fellow of the Washington State Heart Association.
Address for reprints: Leonard A. Cobb, M.D., Cardiology Division, Harborview Medical Center, 325 Ninth Avenue, Seattle, Washington 98104.
Received June 14, 1974; revision accepted for publication August 20, 1974.

Circulation, Volume 50, December 1974

1231
and ranged from 22 days to 38.5 months in these 146 patients. The group included 122 patients who were in ventricular fibrillation when Medic I arrived and 24 patients who developed ventricular fibrillation after the arrival of medical aid. There were 124 males and 22 females; mean age was 60.4 years.

Laboratory measurements during the postresuscitation hospitalization included serial electrocardiograms (ECG) in 140 of the 146 patients and lactic acid dehydrogenase (LDH) isoenzyme determinations in 94 patients. From these, we determined the presence or absence of acute transmural myocardial infarction and myocardial necrosis associated with the episode of ventricular fibrillation. A diagnosis of acute transmural myocardial infarction was made when serial ECGs during the postresuscitation hospitalization showed the development of new pathologic Q waves. In patients with all other ECG findings, an attempt was made to establish the presence or absence of myocardial necrosis from LDH-isoenzyme determinations. Myocardial necrosis was confirmed when the percentage distribution of α-1 LDH was both increased and greater than α-2 LDH. In addition, all patients with acute transmural myocardial infarction were included in the myocardial necrosis group. Reliance on the diagnostic use of SGOT, CPK, and total LDH activities was usually impossible because cardiopulmonary resuscitation alone commonly results in elevated serum enzyme activities. However, in three patients in whom LDH isoenzyme activities were unavailable, normal SGOT and LDH activities were used to substantiate the absence of myocardial necrosis. In the assessment of historical data in all patients, prior cardiovascular disease was defined as a history of previous heart attack, or angina, or congestive heart failure, or hypertension. A history of coronary heart disease was restricted to knowledge of either previous heart attack or angina pectoris.

One- and two-year mortality rates were derived using the life table method of Cutler and Ederer. For statistical purposes, the three recurrences of successful resuscitation from out-of-hospital ventricular fibrillation were counted as follow-up deaths from the first episodes, and the patients were re-enrolled for follow-up. The Chi-square test was used for assessment of statistical significance.

Results

Acute Transmural Myocardial Infarction

Electrocardiographic evidence of acute transmural myocardial infarction associated with the episodes of ventricular fibrillation was established in 24 of 140 patients (17%). Serial ECGs were unavailable for the remaining six patients. Nineteen patients (14%) had myocardial infarction of indeterminate age, and 69 patients (49%) had only ST-T wave changes. In 26 patients (19%) the ECG remained unchanged from the admission tracing. Two patients (1%) showed changes compatible with confirmed hypokalemia.

Myocardial Necrosis

Serum LDH isoenzyme patterns were indicative of myocardial necrosis in 35 of 94 patients for whom such measurements were available (table 1). Forty-seven of 95 patients (49.5%) had evidence of myocardial necrosis, as judged by LDH isoenzyme pattern or by ECG evidence of acute transmural infarction. In 48 patients (50.5%) adequate data were available to rule out myocardial necrosis. In 51 patients the presence or absence of acute myocardial necrosis could not be determined (table 1). LDH isoenzyme patterns were equivocal for myocardial necrosis in 5 of the 51 patients and were not measured in 42 others; in 4 patients neither ECGs nor LDH isoenzyme levels were available.

Ventricular Fibrillation on Arrival vs Ventricular Fibrillation After Arrival

One hundred twenty-two patients were found in ventricular fibrillation when Medic I first arrived (table 1). ECGs in twelve of these patients (10%) subsequently showed evolution of a typical acute transmural infarction. Data necessary to confirm or exclude the presence of myocardial necrosis were available in 76 of these 122 patients and indicated that myocardial necrosis had occurred in association with the episode of ventricular fibrillation in 32 (42%) of the 76 patients. In contrast, ECGs in 12 of the 24 patients (50%) who developed ventricular fibrillation after arrival of Medic I were characteristic of acute transmural infarction following the aborted episode of sudden cardiac death: 15 of 19 patients (79%) had evidence of myocardial necrosis (P < 0.005).

Deaths

During follow-up, 43 patients (29.5%) died; all but

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Acute Transmural Myocardial Infarction and Myocardial Necrosis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Total episodes</td>
</tr>
<tr>
<td>VF on arrival</td>
<td>122</td>
</tr>
<tr>
<td>VF after arrival</td>
<td>24</td>
</tr>
<tr>
<td>Total</td>
<td>146</td>
</tr>
</tbody>
</table>

*Criteria for necrosis defined in methods.
Abbreviations: ATMI = acute transmural myocardial infarction; VF = ventricular fibrillation.
nine of the deaths were unexpected and occurred outside the hospital. Some form of long-term anti-dysrhythmic therapy had been prescribed for at least 26 of the 43 patients at the time of death. The distribution of the 43 deaths according to ECG criteria for transmural infarction is illustrated in figure 1. Only three of the 24 patients (13%) with acute transmural infarction died, whereas 39 of the 116 patients (34%) with other ECG abnormalities died.

The mortality rate for the entire group of 146 patients was 26% at the end of one year and 38% at the end of two years. Dividing the cohort according to the presence or absence of acute transmural infarction associated with the episode of ventricular fibrillation, a significant difference in mortality is apparent (fig. 2). In the group whose ECGs showed acute transmural infarction following ventricular fibrillation, none died during the ensuing 16 months, and at two years the mortality rate in this group was 14%. However, in the remaining patients with other ECG interpretations, the mortality rate for the first year was 32%, increasing to 43% by the end of two years. The difference in mortality rates between the two groups was statistically significant ($P < 0.005$).

Similarly, when mortality was examined in groups defined on the basis of myocardial necrosis associated with the episode of ventricular fibrillation, a difference was evident. The mortality rate in patients with myocardial necrosis was 14% at the end of one year and 25% at the end of two years. However, in patients without myocardial necrosis the calculated mortality rate was 30% at the end of one year and 47% at two years. The difference in mortality rates between these two groups, although substantial, is statistically less significant than that observed when the patients were grouped according to ECG evidence of acute transmural infarction ($0.10 < P < 0.20$).

**Prior History of Cardiovascular Disease**

Historical data necessary to confirm the presence or absence of previously known cardiovascular disease (history of angina, heart attack, hypertension, or congestive heart failure) were available in 194 patients. Forty-eight of these patients (36%) reported negative histories, thus making sudden cardiac death their first manifestation of cardiovascular disease. Mortality during follow-up was greater in patients with histories of cardiovascular disease and coronary heart disease (table 2). A history of coronary heart disease was obtained from 41% of patients who survived during follow-up, compared with 62% of those who died ($P < 0.05$); cardiovascular disease in these two groups had been previously noted in 57% and 84%, respectively ($P < 0.005$).

**Discussion**

Although coronary atherosclerosis is closely associated with out-of-hospital sudden cardiac death,
Table 2

<table>
<thead>
<tr>
<th>Patient Profiles Related to Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
</tr>
<tr>
<td>--------------------------------------</td>
</tr>
<tr>
<td>Number of patients</td>
</tr>
<tr>
<td>Average age ± so</td>
</tr>
<tr>
<td>History† of</td>
</tr>
<tr>
<td>Previous heart attack</td>
</tr>
<tr>
<td>Angina pectoris</td>
</tr>
<tr>
<td>Hypertension</td>
</tr>
<tr>
<td>Congestive heart failure</td>
</tr>
<tr>
<td>Known coronary heart disease‡</td>
</tr>
<tr>
<td>Known cardiovascular disease§</td>
</tr>
</tbody>
</table>

*Probability by Chi-square, except age by t-test.
†Patients with questionable or unknown histories have been excluded.
‡Coronary heart disease was defined as a history of heart attack or angina pectoris or both prior to the initial episode of ventricular fibrillation.
§Patients with one or more of the following were classified as having known cardiovascular disease: heart attack, angina pectoris, hypertension, congestive heart failure.

relatively little is known concerning mechanisms responsible for the terminal event.1, 8 Certainly, the abruptness of the syndrome suggests that dysrhythmias are causal. Since ventricular fibrillation is a well recognized complication of acute myocardial infarction, sudden cardiac death is commonly attributed to acute myocardial infarction, particularly when coronary atherosclerosis is demonstrated at autopsy. Indeed, death certificates of many, if not most, patients succumbing to sudden cardiac death indicate acute myocardial infarction as the underlying precipitating event. However, the observations reported here show that only 17% of patients who survived hospitalization following successful resuscitation from out-of-hospital ventricular fibrillation had an associated acute transmural myocardial infarction. (ECGs in an additional 14% showed Q waves of undetermined age.) One-half of our patients had associated myocardial necrosis, based on ECG evidence of new Q waves or LDH-isoenzyme patterns of myocardial damage. Furthermore, in an unknown number of these patients, myocardial necrosis may have been consequent to the cardiac arrest.

The applicability of these observations concerning the incidence of acute transmural myocardial infarction and myocardial necrosis to the large numbers of victims of sudden cardiac death deserves consideration. Liberthson and colleagues9 diagnosed acute myocardial infarction in 39% of 80 long- and short-term survivors of out-of-hospital ventricular fibrillation. As noted, the 146 patients in our study represent only a portion of nearly 900 patients treated for out-of-hospital ventricular fibrillation during this three-year period. It is difficult to assess the underlying pathophysiology in the unresuscitated patients. However, our observed 25% incidence of acute transmural infarction in a group of 53 patients who were resuscitated, but subsequently died in the hospital, approximates that of the 146 patients described in this report. Thus, these results appear to apply to at least the numbers of patients (approximately 45%) who can be initially resuscitated from ventricular fibrillation.

Although the calculated one-year mortality rate for all 146 patients was 26% at one year and 38% at two years after resuscitation, patients with acute transmural infarction or necrosis showed a relatively low mortality rate. Survival in this subgroup was similar to that reported following resuscitation from ventricular fibrillation complicating acute myocardial infarction in hospital coronary care units.10, 11 In contrast, patients who showed no evidence of infarction or necrosis in the postresuscitation period had a markedly greater mortality rate (fig. 2). Certainly, other studies on similar groups of patients are necessary to confirm these observations. Nevertheless, our data strongly suggest that patients resuscitated from ventricular fibrillation without associated acute myocardial infarction are prone to sudden death, most likely from ventricular fibrillation. Whereas the propensity for developing ventricular fibrillation at the time of acute infarction is of short duration, ventricular fibrillation in the absence of acute infarction may be related to a chronic process and therefore likely to recur. In this regard, myocardial fibrosis and ischemia represent possible precipitating factors.

It is of interest that sudden cardiac death in these 146 patients can be described in two general patterns (table 1): (1) a small group (N = 24) who developed ventricular fibrillation after arrival of medical aid and (2) a much larger group (N = 122) who were already in circulatory arrest when aid arrived. The former
group frequently (87%) experienced prodromal symptoms of chest pain or dyspnea, which in fact prompted the request for medical assistance. In this group we noted a relatively high incidence of acute transmural infarction (50%). On the other hand, in the larger group, loss of consciousness usually precipitated entry into the emergency system; only 20% of this group had prodromal chest pain or dyspnea. Acute transmural infarction was diagnosed in only 10% of the patients who were already in circulatory arrest when aid arrived. Thus “instantaneous” death occurring without warning symptoms was uncommonly due to acute transmural infarction in our patients.

Previous reports have dealt with survival from ventricular fibrillation occurring as a complication of acute myocardial infarction. Adgey and co-workers reported 27 long-term survivors from a group of 55 patients who had ventricular fibrillation and were resuscitated within four minutes. Twenty-four of these had “acute myocardial infarction.”12 Lawrie reported a 7% one-year mortality rate in a group of 53 long-term survivors of ventricular fibrillation due to acute myocardial infarction.10 Our study supports these findings, in that patients resuscitated from ventricular fibrillation associated with acute myocardial infarction had reasonably good prognosis for survival. However, it is clear that ventricular fibrillation without acute myocardial infarction contributes substantially to the numbers of sudden cardiac deaths.

At the present time, 25% of patients found in ventricular fibrillation in our community are resuscitated and discharged home. In addition, when delay is minimal, as in our patients who developed ventricular fibrillation under surveillance, a similar outcome was observed in 37% of instances. Thus the contention13 that little can be done to prevent patients dying within one hour after the onset of symptoms seems overly pessimistic.

References
Survival after Resuscitation from Out-of-Hospital Ventricular Fibrillation
ROBERT S. BAUM, HERNAN ALVAREZ III and LEONARD A. COBB

Circulation. 1974;50:1231-1235
doi: 10.1161/01.CIR.50.6.1231
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1974 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/50/6/1231

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/