JACC Vol. 3, No. 5
May 1984:1111-7

COOPERATIVE STUDIES

Timing, Mechanism and Clinical Setting of Witnessed Deaths in Postmyocardial Infarction Patients

SIDNEY GOLDSTEIN, MD, FACC, LAWRENCE FRIEDMAN, MD, RICHARD HUTCHINSON, MD, FACC, PAUL CANNER, PhD, DONALD ROMHILT, MD, FACC, ROBERT SCHLANT, MD, FACC, RAFAEL SOBRINO, MD, FACC, JOEL VERTER, PhD, ALAN WASSERMAN, MD, FACC and the ASPIRIN MYOCARDIAL INFARCTION STUDY RESEARCH GROUP

Bethesda, Maryland

The temporal distribution and mechanism of death were studied in a large multicenter secondary prevention trial (Aspirin Myocardial Infarction Study) in which acute witnessed death represented 72% (270 of 376) of the deaths due to arteriosclerotic heart disease. Instantaneous deaths represented 28.9% (78 of 270) of the acute witnessed deaths; 45.2% (122 of 270) occurred in the first hour after the onset of symptoms and were defined as sudden deaths. In the subsequent 23 hours, an additional 113 deaths (41.8%) occurred and were defined as intermediate deaths; 29 late deaths (10.7%) occurred after 24 hours. Cardiac arrhythmia was the mechanism of death in 83% (194 of 235) of deaths within 24 hours. Univariate analysis of baseline clinical and electrocardiographic characteristics indicates that a history of congestive heart failure, cardiomegaly, angina pectoris, multiple myocardial infarctions and therapy with digitalis and nitroglycerin were more common in those who died than in survivors, regardless of the timing of death.

A description of the timing and mechanism of early death occurring in patients with coronary heart disease can aid in our understanding of the pathophysiology of this disease. The recent development of a number of therapeutic interventions aimed at the prevention of both coronary heart disease mortality and sudden death requires an understanding of both of these factors. To test these interventions, a uniform definition of these factors is important to compare different modes of therapy (1). It is also possible that certain unique clinical characteristics exist in patients at risk of dying that will permit us to distinguish the sudden death victim from those who will not die suddenly.

The Aspirin Myocardial Infarction Study (AMIS) (2) provides an opportunity to examine deaths occurring in a large secondary prevention trial in a systematic manner. In previous studies (3–6) of similar groups, it has been noted that most of the deaths occur within the first hour of the onset of symptoms. The mechanism of death in patients dying of coronary heart disease is complex and often multifactorial. The witness to the event can often shed light on its mechanism. In this study, we related the timing of the event to the observation made by the witnesses of the death to arrive at the presumed mechanism of the event. In addition, the past medical history available in this group provided us with an opportunity to relate the baseline variables, collected in a prospective manner, to the mechanism and timing of death.

Methods

Source of data. Because there was no significant difference in the mortality rate in the treated (aspirin, 1g/day) and placebo groups of patients, all cardiac deaths occurring in the Aspirin Myocardial Infarction Study were included and treated equally in the analysis. These forms were completed by both clinical coordinators and physicians by collecting information from witnesses of the event when available. The forms were in a multiple question format, but also provided a narrative portion in which the physician was required to describe all the information available regarding the fatal event. In the 4,524 patients followed up for a mean

From the Aspirin Myocardial Infarction Study Research Group sponsored by the National Heart, Lung, and Blood Institute, Bethesda, Maryland. The principal investigators of this group are listed in the Appendix. Manuscript received June 27, 1983; revised manuscript received November 15, 1983, accepted November 28, 1983.

Address for reprints: Sidney Goldstein, MD, Division of Cardiovascular Medicine, Henry Ford Hospital, 2799 W. Grand Boulevard, Detroit, Michigan 48202.

0735-1097/84/$3.00

JACC Vol. 3, No. 5
May 1984:1111-7

of 38.2 months in this trial, 465 deaths were reported. Of these, 376 were due to arteriosclerotic heart disease and 270 were witnessed acute events and are the subject of this report (Table 1).

The forms were reviewed by a seven member Mortality Classification Committee that evaluated materials submitted by each clinic physician. The data included autopsy information, emergency vehicle and emergency room reports, hospital records, death certificates and interviews of next of kin and witnesses of the event. Each form was reviewed independently by two committee members. Discrepancies in interpretation were adjudicated in meetings of the entire committee.

Definitions

Deaths due to arteriosclerotic heart disease. The following set of definitions was established before the evaluation of these forms:

Arteriosclerotic heart disease death. Because all patients in this study were determined to have had a myocardial infarction secondary to atherosclerosis at entry, further substantiation of the presence of atherosclerotic heart disease was not required. All deaths except those due to trauma or other definite noncardiac disease were presumed to be caused by arteriosclerotic heart disease.

Acute arteriosclerotic heart disease death (with recent or acute event): Individuals who either experienced new symptoms suggesting acute myocardial infarction or ischemia such as chest pain or shortness of breath or who died within minutes without symptoms or whose death was unobserved.

Nonacute arteriosclerotic heart disease death (without recent or acute event): Death as a progressive manifestation of previous symptomatic coronary heart disease such as progressive heart failure.

Primary mechanisms of death. These were judged to be due to:

Definite cardiac arrhythmia: In patients with actual electrocardiographic evidence of cardiac arrhythmia. When an abnormal rhythm occurred in the setting of shock and congestive heart failure it was excluded as a primary event.

Table 1. Categories of Mortality Due to Ateriosclerotic Heart Disease (ASHD)

Acute witnessed ASHD Death	270
Sudden death < 1 hour	122
Instantaneous	78
Additional < 1 hour	44
Intermediate death (1 to 24 hour)	113
Late death (> 24 hour)	29
Incomplete information	6
Acute non-witnessed death	81
Nonacute ASHD death	25
Total	376

Presumed cardiac arrhythmia: In patients who died suddenly without evidence of cardiovascular or other symptoms. These events were usually instantaneous and were presumed to be due to cardiac arrhythmia in the absence of expression by the victim of any symptoms.

Cardiogenic shock: In patients with a blood pressure of less than 90 mm Hg, central nervous system deterioration and oliguria. When shock occurred after the development of a definite cardiac arrhythmia, it was excluded as a primary event.

Heart failure: In patients with symptoms of shortness of breath or orthopnea in the absence of cardiogenic shock or definite arrhythmia. When heart failure preceded cardiogenic shock or an arrhythmia it was coded as the primary mechanism of death; otherwise it was excluded as a primary event.

Cardiac surgery: In patients who died within 30 days of cardiac surgery. When patients experienced a myocardial infarction immediately before cardiac surgery, the myocardial infarction was considered a contributing and underlying cause of death and cardiovascular surgery was considered the primary cause.

Other definitions. The following additional terms were used:

Acute symptoms: New symptoms or marked changes in the chronic symptoms occurring before death. Symptoms were defined as chronic if they continued until death without change. In the presence of chronic symptoms, acute symptoms were determined to have occurred only if 1) chronic symptoms disappeared and suddenly reappeared before death; 2) stable chronic symptoms became more significant before the event; or 3) chronic symptoms had been stable or improving and significant new symptoms occurred 24 hours before the time of death. If the patient died while asleep and without apparent acute symptoms, the patient was considered to have died without symptoms.

Time of death: Time at which the victim was found to be without pulse and blood pressure and unresponsive by the witness, and before the onset of an unsuccessful cardiopulmonary resuscitation effort. If resuscitation was successful and the patient returned to normal function and was discharged from the hospital, a death event was not recorded. The timing and onset of symptoms was established by interviewing the witness of the event.

Hospital and out of hospital death: Death occurring in the emergency room was classified as a hospital death, even if the patient was not formally admitted to the hospital. If the patient was dead on arrival, the event was classified as an out of hospital death, even if the patient was pronounced dead in the hospital.

Sudden death: Death occurring within 1 hour of the onset of symptoms. This included instantaneous death (acute collapse without any symptoms) and death occurring within the subsequent hour after the onset of symptoms.

Intermediate death: Death occurring between 1 and 24 hours after the onset of acute symptoms.

Late death: Death occurring 24 or more hours after the onset of symptoms.

Witnessed death: Death occurring in the presence of a person who had visual or auditory contact with the victim at the moment of death.

Study Protocol

Data collection and analysis. For all patients, data on a wide variety of medical, historical, social, demographic and laboratory characteristics were available from the baseline examination at the beginning of the study. The baseline electrocardiogram at rest was recorded at the time of randomization; it was sent to the AMIS electrocardiographic reading center at The George Washington University Medical Center where it was mounted and labeled. Each electrocardiogram was coded by two specially trained technicians, working independently using the Minnesota Coding System (7). Premature beats were identified as to type and frequency. Disagreements were resolved by one of the investigators. The readings were made without knowledge of source, treatment group or previous electrocardiograms. Reports of all autopsies were collected. They were not performed using a standard protocol. Gross and microscopic reports were reviewed and classification of postmortem findings was established by the Mortality Classification Committee.

Statistical analysis. Analysis of this information on study patients was carried out in cooperation with the study coordinating center at the University of Maryland, and the Clinical Trials and Mathematical and Applied Statistics Branches of the National Heart, Lung, and Blood Institute. Differences between groups were assessed by the Student's *t* test for continuous variables and by the continuity corrected chi-square for discrete factors. The joint evaluation of risk factors for sudden and intermediate death was evaluated by multiple logistic regression analyses. The coefficients for these analyses were estimated by the method of weighted least squares (8). Because of the considerable number of nonindependent comparisons, we have noted only those differences with probability (p) values less than 0.01.

Results

Categories of mortality due to arteriosclerotic heart disease (Table 1). Of all such deaths, 72% (270 of 376) were acute and witnessed. Of these, 28.9% (78 of 270) were instantaneous deaths occurring without any apparent symptoms. An additional 44 patients died within the first hour after acute symptoms. Therefore, sudden deaths, defined here as death within 1 hour, represented 45.2% (122 of 270) of the witnessed deaths due to acute arteriosclerotic heart disease. During the next 23 hours, 113 deaths occurred (intermediate deaths) and an additional 29 late deaths occurred more than 24 hours after the onset of symptoms.

Mechanism of death (Fig. 1). Cardiac arrhythmia was the dominant mechanism in 83% (194 of 235) of both the sudden and nonsudden witnessed deaths. Of these arrhythmic deaths, 40% (78 of 194) were classified as due to definite and 60% (116 of 194) to presumed arrhythmia. Shock and congestive heart failure occurred with increased frequency in the nonsudden death group.

Clinical and electrocardiographic characteristics (Tables 2 to 4). Table 2 compares the baseline clinical characteristics of the victims of sudden and intermediate death and the study survivors. There were no significant differences between the sudden and intermediate death groups. However, each of these two groups differed from the survivors by having a history of congestive heart failure, cardiomegaly, angina pectoris, New York Heart Association functional class II and more than one myocardial infarction. They also were taking digitalis and more commonly used nitroglycerin tablets.

The baseline electrocardiographic characteristics in the study patients (Table 3), with the exception of premature ventricular complexes, did not distinguish patients with sudden from those with intermediate death. ST depression, T wave abnormalities and intraventricular conduction defects were more common in those who died in either manner than in survivors. Premature ventricular complexes were observed more frequently in the patients who died suddenly than in the intermediate death group and about twice as often as in those who were alive at the end of the study period. Comparison of instantaneous deaths with other deaths in the first hour and with intermediate deaths, in terms of both clinical and electrocardiographic variables, did not show any statistically significant differences. A multiple logistic regression analysis was also performed. Cardiomegaly, number of previous myocardial infarctions, history of angina, use of digitalis, presence of Q or QS wave abnormalities and premature ventricular complexes on the elec-

Figure 1. Mechanism of death related to duration of symptoms before death. CHF = congestive heart failure.

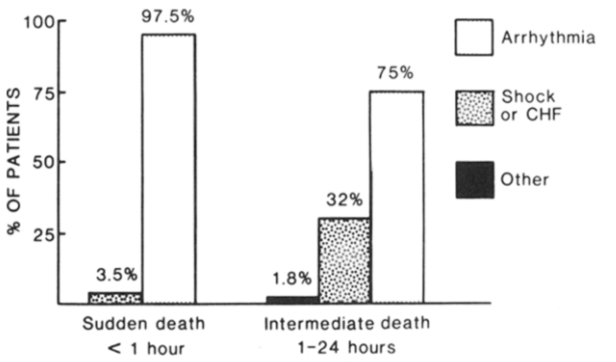

Table 2. Clinical Characteristics at Baseline of All Aspirin Myocardial Infarction Study Survivors and 235 With a Witnessed Death Due to Arteriosclerotic Heart Disease Within 24 Hours of Symptoms

	Sudden Death < 1 hour (%) (n = 122)	Intermediate Death 1 to 24 hour (%) (n = 113)	Alive (%) (n = 4,059)
Mean age (yr)	55.2	54.9	54.6
Male	94.3	92.9	88.4
White	88.5	88.5	92.1
Black	7.4	9.7	5.5
Hypertension†	18.0	17.7	13.4
Congestive heart failure	20.5*	25.7*	8.6
Cardiomegaly on X-ray film	23.8*	25.7*	7.4
NYHA class II	48.4*	46.9*	31.5
Angina pectoris	45.1*	46.9*	30.2
More than one myocardial infarction	25.4*	23.0*	10.4
Current cigarette smokers	28.7	32.7	26.3
Never smoked	18.0	23.0	19.8
Medical therapy at baseline			
Digitalis	36.9*	35.4*	15.6
Propranolol	10.7	13.3	12.0
Antiarrhythmic agents	13.9	14.2	9.1
Nitroglycerin	50.8*	50.4*	36.3

*$p < 0.01$ (sudden and intermediate death vs. alive); †hypertension = systolic pressure 140 mm Hg and diastolic pressure 80 mm Hg. Congestive heart failure, NYHA functional class and angina pectoris determined by clinic physician. NYHA = New York Heart Association.

trocardiogram were significantly related to sudden death (Table 4). Similar analyses for intermediate deaths showed that all of these factors except premature beats and Q or QS wave abnormalities were related to deaths occurring 1 to 24 hours after the onset of symptoms.

Acute symptoms and classification of death (Table 5). Chest pain and symptoms in general were more common ($p < 0.01$) in the intermediate than in the sudden death group. This is due in a large part to the many patients in the sudden death group who died instantaneously and who, therefore, by definition, had no acute symptoms. The occurrence of other symptoms was the same for both groups.

Place of death and activity at time of death (Table 6). There was a significant difference in the distribution of type of activities at the time of death and in the place of death of the intermediate and sudden death groups ($p < 0.01$). Proportionately more people were asleep or engaged in sedentary activities at the time of onset of acute symptoms in the intermediate death group than in the sudden death group. As would be expected, most of the witnessed sudden deaths occurred at home, whereas more than half of the witnessed intermediate deaths occurred in the hospital. Of the patients in the intermediate death group who died at home, 66% had chest discomfort (Table 7).

Postmortem examinations (Table 8). These were available in 50 individuals (18.5%) whose witnessed death

Table 3. Electrocardiographic Characteristics at Baseline of All Study Survivors and the 235 Patients Who Died Within 24 Hours of Symptoms

	Sudden Death < 1 hour (%) (n = 122)	Intermediate Death 1 to 24 hours (%) (n = 113)	Alive (%) (n = 4,059)
ST depression	40.4*	45.0*	23.6
T wave abnormalities	81.7*	80.0*	66.4
ST elevation	26.8	19.6	18.6
AV conduction abnormalities	5.0	2.7	3.0
Intraventricular conduction abnormalities	13.2*	11.6*	3.4
Q/QS pattern	90.0	87.0	80.2
Premature ventricular complexes	30.5†	19.6	14.6

*$p < 0.01$ (sudden and intermediate death versus alive); †$p < 0.01$ (sudden death versus alive). AV = atrioventricular.

Table 4. Logistic Regression Coefficients for Sudden and Intermediate Deaths

	Sudden Death < 1 hour	Intermediate Death 1 to 24 hours
Cardiomegaly on X-ray film	0.926†	1.017†
Number of prior myocardial infarctions	0.646†	0.698†
Use of digitalis	0.583†	0.547*
ST depression	0.142*	0.220†
Angina pectoris	0.470*	0.498*
Premature ventricular complexes	0.510†	0.062
Q/QS pattern	0.246*	0.127

*$p < 0.05$; †$p < 0.01$.

occurred in less than 24 hours from the onset of symptoms. Evidence of an acute myocardial infarction was seen in 35% of the postmortem examinations in the sudden death group. There were substantially more cases of left ventricular aneurysm and recent myocardial infarction in the intermediate than in the sudden death group, but these differences did not reach statistical significance in light of the small number of autopsies performed.

Discussion

The fact that most cardiac deaths occur suddenly with little warning has been the subject of renewed interest as new therapeutic interventions have become available. The temporal distribution of deaths observed in this study is similar to that of previous observations (3,4) and indicates that the case fatality rate is extremely high in the first hour of the onset of symptoms and decreases over the subsequent 24 hours. Most sudden deaths (64%) in this study were instantaneous, occurring without symptoms. Of the acute witnessed deaths in this study, 45% occurred within the first hour, similar to the experience reported by Hinkle and Thaler (6). Only 12% of the sudden deaths occurred in the hospital, 50% occurred at home. Many patients also have acute symptoms for more than 1 hour and die at home, outside the perimeters of medical or medical emergency care.

Table 5. Presence and Nature of Symptoms in Witnessed Deaths Occurring in Less Than 24 Hours

	Sudden Death < 1 hour (%) (n = 122)	Intermediate Death 1 to 24 hours (%) (n = 113)
Chest pain	18.0*	49.6
Fatigue	14.8	15.0
Shortness of breath	12.3	25.7
Abdominal pain	5.7	10.6
Palpitation	2.5	1.8
Syncope	1.6	5.3
Dizziness	3.3	5.3
Apprehension	4.1	12.4
Depression	3.3	4.4

*$p < 0.01$ (sudden death versus intermediate death).

Table 6. Place of Death and Activity of Subjects With Witnessed Deaths Occurring in Less Than 24 Hours

	Sudden Death < 1 hour (%) (n = 122)	Intermediate Death 1 to 24 hours (%) (n = 113)
Place*		
home	50.0	33.6
Public place	25.4	4.4
Hospital	12.3	53.2
Other	12.3	8.8
Activity*		
Asleep	8.2	19.5
Sedentary	42.6	61.1
Light work	30.3	8.8
Moderate work	12.3	2.7
Unknown	6.6	7.9

*$p < 0.01$ represents a difference in distribution characteristics between the sudden and intermediate deaths.

Roles of arrhythmia, cardiac failure, myocardial infarction and ischemia. The primacy of rhythm disturbance as a mechanism of cardiac death in the first 24 hours is also emphasized in this study. Although we cannot provide information regarding the relation of preexisting ischemia to rhythm disturbances, it is clear that cardiac arrhythmia is a primary clinical mechanism and the dominant mechanism of death in the first 24 hours. When cardiac failure manifested by congestive heart failure and shock does occur, it is a relatively late event and even then represents an unusual mechanism of cardiac death in this time frame. It is probable that ischemia or infarction, or both, often precedes both the arrhythmia and events due to left ventricular dysfunction. In previous studies (9) of patients who were resuscitated after out of hospital cardiac arrest due to arteriosclerotic heart disease, 78% of patients were observed to have either infarction or ischemia. In our study, postmortem evidence of myocardial infarction was observed in 35% of the sudden deaths and 75% of the nonsudden deaths in the small proportion of patients undergoing autopsy. Using a similar def-

Table 7. The Nature of Symptoms Before Intermediate Death (1 to 24 hours) Related to Place of Death

	Hospital (%)* (n = 60)	Home (%) (n = 38)
Chest pain	77	66
Shortness of breath	45	42
Fatigue	17	26
Indigestion/abdominal pain	10	5
Syncope	10	11
Palpitation	8	0
Dizziness	5	11
Other	20	24

*Data from one person are missing.

JACC Vol. 3, No. 5
May 1984:1111-7

Table 8. Significant Postmortem Cardiovascular Abnormalities in Witnessed Deaths Occurring in Less Than 24 Hours

	Sudden Death < 1 hour (%) (*n = 22)	Intermediate Death 1 to 24 hours (n = 28)
Recent myocardial infarction	35	75
Left ventricular aneurysm	10	32
Recent coronary artery occlusion by thrombus or embolus	30	54
Recent coronary artery occlusion by hemorrhage	5	14
Pulmonary edema	60	82

*n = the number of patients who had postmortem examination.

inition, Hinkle and Thaler (6), in a long-term follow-up study of individuals with mixed cardiovascular disease, observed that cardiac arrhythmia was the predominant feature in acute cardiac deaths. In that study, myocardial infarction and ischemia were observed at postmortem in 33% of the patient group. Although evidence of myocardial infarction was not detected by histologic techniques in 65% of the patients dying suddenly in that study, this does not indicate that microscopic evidence of ischemia did not precede the development of sudden cardiac arrhythmia. The pathologic evidence of myocardial infarction requires patient viability and survival for a period of time before the currently available pathologic characteristics of myocardial infarction can develop.

Premature ventricular complexes in prediction of sudden death. A number of studies (10–13) have attempted to identify certain characteristics that will predict sudden cardiac death. In previous studies, those characteristics that predicted sudden cardiac mortality also predicted all coronary deaths. Chiang et al. (10) observed that premature ventricular complexes and conduction abnormalities were frequently noted on the standard electrocardiogram in victims of sudden death in the Tecumseh study. In the Coronary Drug Project (14), premature ventricular complexes recorded on a standard electrocardiogram were predictive of both sudden death and death from all causes. Statistical analysis in that study suggested that premature ventricular complexes were uniquely associated with sudden death and were independent of other electrocardiographic or clinical characteristics. Over the 3 year follow-up of the Coronary Drug Project study, the mortality rate in the patients with premature ventricular complexes was twice that of the patients without such complexes. In a postcoronary care unit group, Moss et al. (15) studied comorbidity factors predictive of sudden and nonsudden death. The factors studied included premature ventricular complex frequency and clinical evidence of left ventricular dysfunction. They were unable to identify any features unique to the sudden death group. In contrast, Ruberman et al. (13) observed that premature ventricular complex frequency and complexity observed on a 1 hour electrocardiographic recording provide information on postmyocardial infarction patients to predict sudden death independently of clinical evidence of left ventricular dysfunction. In our study, premature ventricular complexes present on a single baseline electrocardiogram were more frequent in the patients who died suddenly than in those who died 1 to 24 hours after the onset of symptoms and the survivors of the study.

Implications. Our observations support the fact that most deaths from coronary heart disease occur within the first few hours of symptoms. With the exception of premature ventricular complexes, there were no obvious predictors of sudden rather than intermediate death. Within the first 24 hours, and particularly the first hour, rhythm disturbances were the most common mechanism of death. Whether these rhythm disturbances were primary or secondary to acute ischemia requires further investigation.

APPENDIX

Aspirin Myocardial Infarction Study (AMIS): Principal Investigators

Pantel S. Vokonas, MD, Boston City Hospital, Boston, Massachusetts; Stephen Scheidt, MD, Cornell University Medical Center, New York, New York; Robert C. Schlant, MD, Emory University, Atlanta, Georgia; Gary N. Wilner, MD, Evanston Hospital, Evanston, Illinois; Charles A. Laubach, Jr., MD, Geisinger Medical Center, Danville, Pennsylvania; Thaddeus E. Prout, MD, Greater Baltimore Medical Center, Baltimore, Maryland; Peter T. Kuo, MD, Heart Clinic, Raritan Valley Hospital, Rutgers Medical School, Piscataway, New Jersey; Sidney Goldstein, MD, Henry Ford Hospital, Detroit, Michigan; William L. Holmes, MD, Lankenau Hospital, Philadelphia, Pennsylvania; Paul Samuel, MD, Long Island Jewish-Hillside Medical Center, New Hyde Park, New York; David W. Richardson, MD, Medical College of Virginia, Richmond, Virginia; Marvin Segal, MD, Mount Sinai Hospital, Minneapolis, Minnesota; William H. Bernstein, MD, Mount Sinai Medical Center, Miami Beach, Florida; Olga Haring, MD, Northwestern University, Chicago, Illinois; C. Basil Williams, MD, Ogden Research Foundation, Ogden, Utah; J. Judson McNamara, MD, Pacific Health Research Institute, Honolulu, Hawaii; Bernard I. Lewis, MD, Palo Alto Medical Center and Research Foundation, Palo Alto, California; James A. Schoenberger, MD (Chairman, Steering Committee), Rush Presbyterian-St. Lukes Medical Center, Chicago, Illinois; David M. Berkson, MD, Saint Joseph Hospital, Chicago, Illinois; Allan H. Barker, MD, Salt Lake City Research Foundation, Salt Lake City, Utah; Elmer E. Cooper, MD, Santa Rosa

Medical Center Clinic, San Antonio, Texas; Nemat O. Borhani, MD, University of California at Davis, Davis, California; Noble O. Fowler, MD, University of Cincinnati Medical Center, Cincinnati, Ohio; Henry K. Schoch, MD, University of Michigan, Ann Arbor, Michigan; Richard G. Hutchinson, MD, University of Mississippi Medical Center, Jackson, Mississippi; Mario R. Garcia-Palmieri, University of Puerto Rico, San Juan, Puerto Rico; Jessie Marmorston, MD, University of Southern California, Los Angeles, California; J. Joanne Hoover, MD, University of Washington, Seattle, Washington; Philip H. Frost, MD, USPHS Hospital, San Francisco, California; Hugh R. Gilmore III, MD, Veterans Administration Hospital, Miami, Florida; William T. Friedewald, MD, National Heart, Lung, and Blood Institute, Bethesda, Maryland; Dayton T. Miller, PhD, Central Laboratory, Center for Disease Control, Atlanta, Georgia; Jorge C. Rios, MD, ECG Reading Center, George Washington University, Washington, D.C.; William F. Krol, PhD, Coordinating Center, University of Maryland, Baltimore, Maryland; Lawrence Friedman, MD, National Heart, Lung, and Blood Institute, Project Office, Bethesda, Maryland.

References

1. Goldstein S. The necessity of a uniform definition of sudden coronary death: witnessed death within 1 hour of the onset of acute symptoms. Am Heart J 1982;103:156–9.
2. Aspirin Myocardial Infarction Study Research Group: a randomized, controlled trial of aspirin in persons recovered from myocardial infarction. JAMA 1980;243:661–9.
3. McNeilly R, Pemberton J. Duration of attack in 998 fatal cases of coronary artery disease and its relation to possible cardiac resuscitation. Br Med J 1968;3:139–42.
4. Fulton, M, Julian D, Oliver M. Sudden death and myocardial infarction. Circulation 1969;39(suppl IV):182–191.
5. Kuller L, Lilienfield A, Fisher R. Epidemiological study of sudden and unexpected deaths due to arteriosclerotic heart disease. Circulation 1966;34:1056–68.
6. Hinkle L, Thaler H. Clinical classification of cardiac deaths. Circulation 1982;65:457–64.
7. Rose G, Blackburn H. Cardiovascular Surgery Methods. WHO Monograph Series No. 56. Geneva: WHO Press, 1968.
8. Walker SN, Duncan DB. Estimation of the probability of an event as a function of several independent variables. Biometrica 1967;54:167–79.
9. Goldstein S, Landis J, Leighton R, et al. Characteristics of the resuscitated out-of-hospital cardiac arrest victim with coronary heart disease. Circulation 1981;64:977–84.
10. Chiang B, Pearlman L, Fulton M, Ostrander L, Epstein F. Predisposing factors in sudden cardiac death in Tecumseh, Michigan: a prospective study. Circulation 1970;41:31–7.
11. Kannel W, Doyle J, McNamara P. Precursors of sudden coronary death. Factors related to the incidence of sudden death. Circulation 1975;51:606–12.
12. Ruberman W, Weinblatt E, Goldberg J, Frank C. Chaudhary B, Shapiro S. Ventricular premature complexes and sudden death after myocardial infarction. Circulation 1981;64:297–305.
13. Ruberman W, Weinblatt E, Goldberg J, Frank C, Shapiro S. Ventricular premature beats and mortality after myocardial infarction. N Engl J Med 1977;297:750–7.
14. The Coronary Drug Project Research Group. Prognostic importance of premature beats following myocardial infarction. JAMA 1973;223:1116–24.
15. Moss A, Davis H, DeCamilla J, Bayer L. Ventricular ectopic beats and their relation to sudden and non-sudden cardiac death after myocardial infarction. Circulation 1979;60:998–1003.