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Hearts Too Good to Die — Problems in Acute Myocardial Infarction*

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WHEN we appreciate the vast number of disorders and diseases which take human life, it is a shocking fact that the lives of so many Americans depend finally on the health of two small arteries. Disease of the coronary arteries represents the one greatest threat to life in the American male, ages 40-60 years, and was responsible for more than one-half million deaths in the United States in 1964.

We, as physicians, are much too complacent about the problem of acute coronary heart disease. This is in part because we have many drugs and increasingly new gadgets to treat the hospitalized patient with acute myocardial infarction. It is also partly due to the fact that we are not really enthusiastic about preventive medicine for adults.

Coronary artery disease is now of epidemic proportions in the United States.[1] Hospital statistics show mortality rates of five to twenty per cent.[2] These statistics inadvertently mask the deadlines of acute coronary heart disease. A more realistic picture of the fatality rate in acute coronary heart disease is provided by investigations such as that reported in a United States military population;[3] epidemiologic investigations of coronary artery disease such as the Framingham study,[4,5] and long-term investigations in an employed population in a major industry.[6] These studies indicate a mortality rate of about 40 per cent for individual attacks of acute coronary heart disease!

The nature of this mortality problem is well illustrated in Figure 1. It is shown that after a

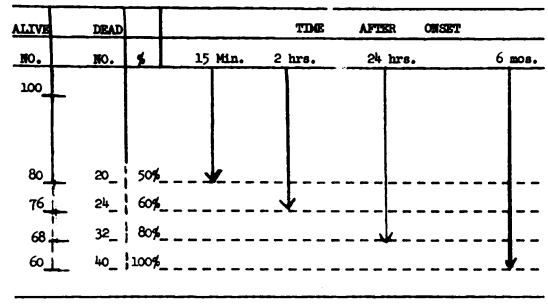

FIG. 1.—DURATION OF TERMINAL ILLNESS IN FATAL CASES OF ACUTE CORONARY DISEASE.

six-month period from the onset of the acute attack, 40 of each 100 cases are dead. Twenty-four of these individuals will be dead in the first two hours. Note, however, that 20 of these deaths (50 per cent) occur within the first 15 minutes. If this half of the acute coronary patients who die within the first 15 minutes ever reach a hospital, usually they will be "dead on arrival." They can never benefit from the refinements of hospital

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treatment and they do not appear in the hospital mortality statistics. For these 20 people, only prophylaxis and prevention might have been life saving. Who are these people? How can they be found in advance in order to get them into a program of prevention?

THE CORONARY PROFILE

The recognition of the "coronary prone" individual is now more nearly a realistic possibility. The major factors which increase the risks of acute coronary heart disease are presented in Table 1,

TABLE 1.—RISK FACTORS INFLUENCING CORONARY HEART DISEASE.

Male Age 50	4 x risk of male age 30
Serum cholesterol over 240 mg%	3 x risk of male with cholesterol below 200 mg%
B. P. greater than 160	4 x risk of male with B. P. 120 or less
Pathological E. C. G.	2—4 x risk of normal E.C.G.
Cigarette smoker	2 x risk of non-smoker

as modified from studies reported in the literature. These factors include hypertension, hypercholesterolemia, pathological electrocardiogram, and cigarette smoking. Combinations of these factors compound the risk as seen in Figure 2.[7] Although final proof is not yet available from long-term studies, preliminary evidence suggests that favorable alteration of these risk factors will be helpful in the prevention of the acute attack. Blakeslee and Stamler have written a small monograph on this subject which is an excellent review, entitled, Your Heart Has Nine Lives.[8]

These risk factors are easily detectable. Age and sex cannot be materially altered, but all the others can now be favorably influenced, thus making it possible theoretically, to remove the coronary prone subject from the high-risk group. It would seem, without question, that every male above the age of 40 years should have the necessary screening procedures done as a part of the comprehensive examination to determine his "coronary proneness". If he falls in a high-risk group, it is the responsibility of the physician to urge the patient to accept a program of prevention with regular follow-up. Most patients are sensible about their hearts and will make the necessary financial sacrifice for honest diagnostic study and preventive therapy with reference to acute coronary heart disease. Only the physician can give the patient his chance.

THE CAUSE OF DEATH IN ACUTE CORONARY HEART DISEASE

Inquiry into the causes of death in patients with

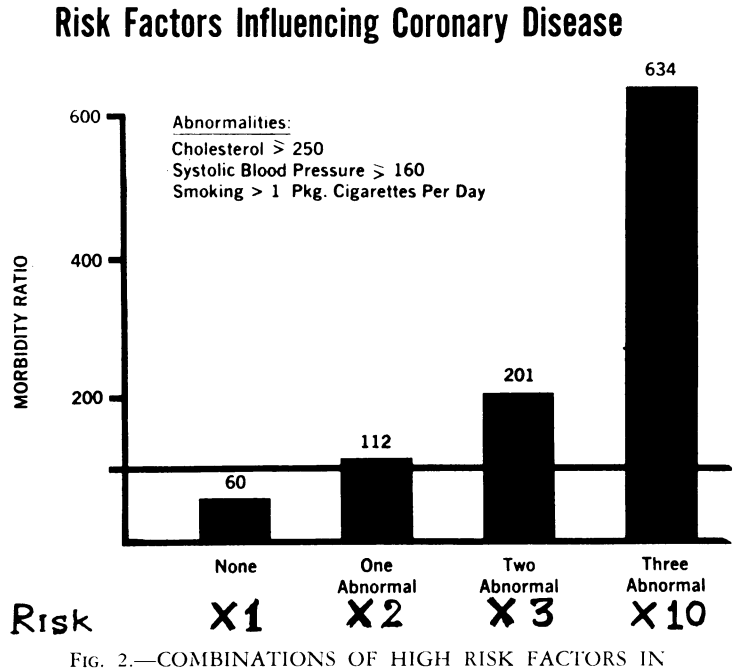

FIG. 2.—COMBINATIONS OF HIGH RISK FACTORS IN CORONARY HEART DISEASE—MODIFIED FROM DAWBER ET AL.

acute coronary heart disease provides important information about the lethal complications of this disorder. These causes are listed below in descending order of frequency:

Causes of Death	*Comments*
Fatal Arrhythmias	*Greatest cause of death.* Morphine accentuates sudden death response. Use atropine with morphine for the severe pain (morphine is vagomimetic).
Shock	*Second most frequent cause.* Etiology debatable. Therapy — Vasoconstrictors plus alpha receptor blockade, steroids.
Heart Failure	*Much improvement in treatment possible here.* Digitalis, saluretics, antihypertensive drugs for the hypertensives.
Thrombo-embolism	Good anticoagulant control required. Problem is one of case selection.
Cardiac Rupture	Occurs in first two weeks. Control hypertension, avoid straining.

The influence of shock and heart failure on the mortality rate is illustrated in Table 2. Note that the mortality rate of acute infarction in the presence of shock is 10 times as high as the compensated myocardial infarction patient with normal blood pressure.

TABLE 2.—MORTALITY RATE IN ACUTE MYOCARDIAL INFARCTION WITH REFERENCE TO THE COMPLICATIONS OF HEART FAILURE, HYPOTENSION, AND CARDIOGENIC SHOCK.[15]

Clinical State	Patients No.	Deaths No.	Deaths %
Compensated—Normal B.P.	169	13	7.7
Heart Failure and/or Hypotension	188	74	39.4
Cardiogenic Shock	32	25	78.1
Total	389	112	28.8

Serious arrhythmias, however, represent the greatest cause of death in acute coronary disease. It is tragic indeed that the human heart, capable of beating 30 million times a year with perfect regularity, can with slight but fortuitous damage, succumb to a chaotic fatal arrhythmia within a matter of minutes although the heart muscle as a pump, was still "too good to die." When promptly diagnosed, these arrhythmias usually can be corrected by the use of quinidine, procainamide, and lidocaine, by the use of cardiac pacemakers, and by direct current cardioversion. The most important recent concept in the prevention of fatal cardiac arrhythmias in patients hospitalized with acute coronary heart disease is the Coronary Care Unit.

THE CORONARY CARE UNIT

A Coronary Care Unit may be defined as a circumscribed, technically well-planned hospital area of four or more beds, specially staffed, and specially equipped for the minute by minute surveillance and treatment of definite or suspected acute myocardial infarction patients. In essence, the unit should be looked upon as an intensive care recovery room for acute myocardial infarction patients, set up in a manner to detect and provide definitive therapy for complications anticipated in the first three to five days following the acute coronary attack.

The work of Zoll[9] and Lown[10] with external defibrillators; that of Jude et al.[11] in the development of the technique of closed-chest cardiac resuscitation; and the recent advances in electronic physiologic monitoring apparatus, all together, made possible the implementation of the Coronary Care Unit concept. From the initiation of the first unit by Dr. Hughes Day[12] at Bethany Hospital in Kansas City, Kansas, in 1962, the number has grown to more than 200 Coronary Care Units in operation at the present time.

The Coronary Care Unit has exhibited great life-saving potential for the acute myocardial infarction patients, particularly for those who suddenly develop unexpected episodes of lethal or potentially lethal cardiac arrhythmias, such as ventricular tachycardia, ventricular fibrillation, and ventricular standstill. Patients with "mild" or severe myocardial infarction have been dramatically rescued from chaotic heart mechanisms by the early detection and aggressive treatment which can best be instituted in a Coronary Care Unit.

The unit is equipped for continuous electrocardiographic monitoring for detection of arrhythmias. Other equipment basic to the unit includes resuscitation apparatus in the form of bed boards and mechanical compressors for external cardiac massage, external and internal electrical pacemakers, and direct current defibrillators. Supportive equipment in the unit consists of wall outlets for oxygen and suction, positive pressure breathing apparatus, endo-tracheal tubes and other emergency equipment. To achieve the optimal physical and psychological well being of the patient, the

Coronary Care Unit should be designed to provide privacy for the individual patient in a tranquil, climate-controlled environment, yet with ready visibility of each patient by the nursing staff.[13]

By far the most important part of the Coronary Care Unit, however, is the personnel—nurses and physicians. Nurses are the single most important personnel group in the unit. The successful operation of Coronary Care Units is dependent upon the skills, judgement, vigilance, experience and training of the cardiovascular nurse, as well as upon newer techniques for detection and treatment of cardiac arrhythmias. The monitoring devices do not replace the nurse by any means, but assist her in widening the horizon of her observations. The nurses of the unit must be specially trained in use of the monitors, arrhythmia detection, the use of electric countershock, cardiopulmonary resuscitation, drug regimens for cardiac emergencies; and should have a broad base of cardiovascular nurse training and experience. They must be taught to detect readily significant complications arising in the acute myocardial infarction patient. As soon as the complication is detected, the nurses sound the alarm for help, make ready the previously prepared emergency drugs and anticipated cardiopulmonary apparatus, and institute supportive measures until the physicians on emergency call arrive. In Coronary Care Units where the cardiovascular nurses are highly trained, they may be authorized to initiate lifesaving measures including direct current defibrillation. The role and training of the Coronary Care Unit nurse has been detailed in a recent manual.[14]

One conspicuous illustration of the lifesaving value of the Coronary Care Unit is seen in the salvage rate of myocardial infarction patients who develop unexpected cardiac arrest. Data on cardiac arrest from Coronary Care Units in five different hospitals are summarized in Table 3. Note that cardiac arrest in patients with acute myocardial

TABLE 3.—THE INCIDENCE OF CARDIAC ARREST AND SURVIVOR RATE IN ACUTE MYOCARDIAL INFARCTION.[15]

Hospital	Patients No.	No.	%	Cardiac Arrests Survivors Observed No.	Expected No.
A	143	18	12.5	5	0
B	96	19	19.8	2	0
C	150	28	18.7	7	0
D	300	42	14.0	23	0
E	234	14	5.9	11	0
Total	923	121	13.1	48	0

infarction is not infrequent, occurring in 13% or 2 of each 15 patients. The other important feature is that of the 121 cardiac arrests there are 48 survivors! The survivor rate was twice as great in patients with ventricular fibrillation as in those with ventricular standstill.[15] Under routine ward management no survivors would have been expected.

SPECIFIC TREATMENT OF ARRHYTHMIAS IN MYOCARDIAL INFARCTION

The management of lethal cardiac arrhythmias is both preventive, by treatment of precursors of

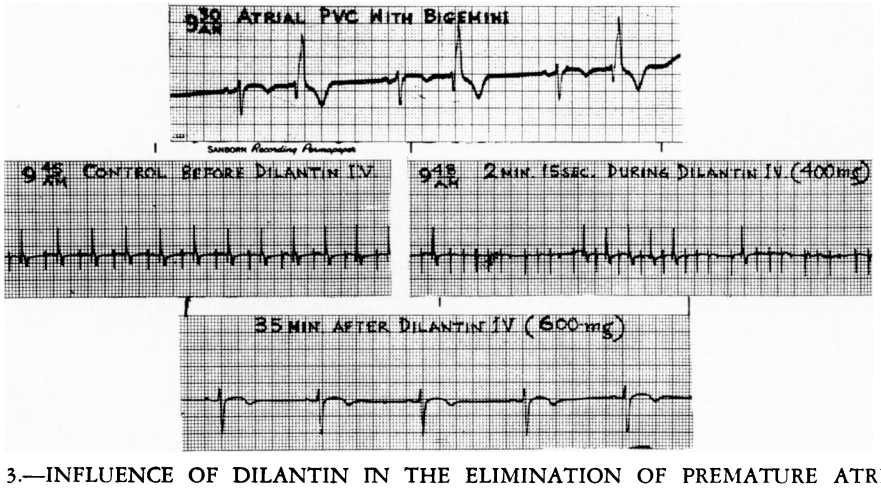

FIG. 3.—INFLUENCE OF DILANTIN IN THE ELIMINATION OF PREMATURE ATRIAL IMPULSES WITH ABBERRANT CONDUCTION.

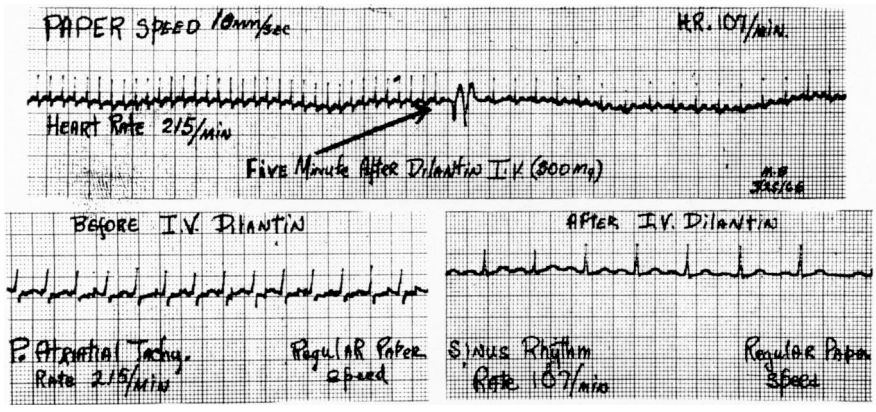

Fig. 4. Influence of Dilantin in the termination of paroxysmal atrial tachycardia.

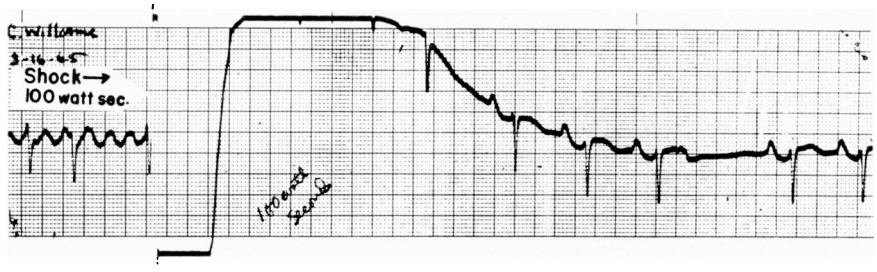

FIG. 5.—ESTABLISHMENT OF SINUS RHYTHM WITH DIRECT CURRENT COUNTER-SHOCK (100 WATT-SECONDS) IN A PATIENT WITH ATRIAL FLUTTER.

serious arrhythmias, and resuscitative by treatment of life-threatening arrhythmias already established. If the myocardial infarction patient shows persistence of frequent premature atrial or premature ventricular impulses, or a run of three or more successive premature ventricular impulses, prophylactic therapy should be instituted.

A variety of drugs is available for prophylactic use, such as the intravenous administration of quinidine, procainamide hydrochloride (Pronestyl), lidocaine (Xylocaine), or diphenyl-hydantoin (Dilantin sodium). If any of these is effective, it may be followed by procainamide, 1½ to 2 grams daily as needed.

The effectiveness of Dilantin sodium is illustrated by the case of a 66-year-old male with frequent premature atrial impulses. In Figure 3 note the disappearance of the premature atrial impulses during the intravenous administration of 600 mg of Dilantin. Figure 4 shows the dramatic effect of intravenous Dilantin sodium in terminating paroxysmal atrial tachycardia.

Lidocaine received some attention as an anti-arrhythmic drug in clinical medicine as far back as 1950.[16] Recently, lidocaine has been shown theoretically to be superior to procainamide and quinidine as an anti-arrhythmic agent and is now under more intensive study. Its use has been recommended primarily in the treatment of rapid ventricular arrhythmias and prophylactically in acute myocardial infarction cases with frequent premature ventricular impulses. Its effect on the myocardium is transitory. A recommended method of administration is as follows: 1 milligram per kilo body weight as a stat dose intravenously while the patient is under continuous monitoring. This is followed by 500 milligrams in intravenous drip of 5 per cent glucose given at a rate of 1 to 4 milligrams per minute.[17] The patient is then changed to oral procainamide, 1.5 grams or more per 24 hours until the critical point is passed.

Atrial flutter, atrial fibrillation and other tachyarrhythmias which threaten the life of the acute myocardial infarction patient may be corrected immediately by means of cardioversion. Figure 5 represents cardioversion in a patient with atrial flutter in whom sinus rhythm has just been restored following 100 watt-seconds.

SUMMARY

Acute coronary heart disease appears to be in epidemic proportions in the United States and is responsible for more than a half-million deaths annually; many of its victims are men at the height of their powers, between ages 40 and 60 years. The mortality rate for the individual attack, including those dead within the first half-hour, is about 40 per cent. Indeed, about half of those destined to die with the individual attack will be dead in the first half-hour. For them the only hope might have been prophylaxis and prevention.

We have reviewed the "risk factors" influencing acute coronary heart disease and described the coronary profile, as widely agreed on by epidemiologists. The obligation of the physician to his patients in attempting to get them out of the high risk group is inescapable.

For the acute coronary victim who reaches the hospital, cardiogenic shock and heart failure are potentially lethal complications; however, the greatest threat to the lives of these patients is cardiac arrhythmias.

The most important recent development for the detection and prevention of chaotic lethal arrhythmias is the Coronary Care Unit and the prompt utilization of direct current cardioversion, pacemakers, and such anti-arrhythmic drugs as procainamide, lidocaine, Dilantin sodium, and quinidine. A brief review of these important advances has been presented.

It now seems possible that with an adequate program of prevention, continuous monitoring and with a prompt aggressive approach to the prevention and ablation of serious cardiac arrhythmias, fewer acute coronary patients will be dead with "hearts too good to die."

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