Beginning at the turn of the 20th century, death rates attributed to coronary heart disease (CHD) in the United States rose continually, reaching epidemic proportions by the mid-1960s. Since that time, the mortality rate due to CHD has markedly decreased.1-4 Internationally, inconsistent trends in CHD mortality rates have taken place. Favorable declines in these death rates have been seen since the early 1960s in Australia, New Zealand, Canada, France, Japan, Switzerland, and Italy, whereas other countries have actually experienced an increase in CHD mortality rates from this time until the late 1970s (e.g., Ireland, Sweden, Austria, France, and Denmark).

Although the declining death rates of CHD in the United States and several other industrialized countries have provided rich fodder for explanation by cardiovascular epidemiologists and clearly provide encouragement in the battle against CHD, reasons for the dramatic decline remain uncertain, particularly from a population-based perspective.5-9 Moreover, CHD remains a major cause of death and disability in the United States and other industrialized countries. Temporal declines in the incidence rates of the clinical manifestations of CHD, including sudden cardiac death, acute myocardial infarction, and angina pectoris, would suggest that the mortality decline is due at least partly to measures of primary prevention and changes in the prevalence and levels of the major coronary risk factors. These secular changes would include the increased detection, control, and prevention of elevated blood pressure, decreased cigarette smoking, and declining population serum cholesterol levels. Evidence from national, as well as individual investigator-initiated, studies supports such improving trends.10-14 In addition, several population-based investigations have provided supportive evidence of declining incidence rates of acute myocardial infarction.15-18 On the other hand, if the incidence rates of CHD are remaining steady or even increasing during this period of declining cardiovascular mortality, a second and more plausible explanation for the observed downward trend in cardiovascular mortality would be that it is due to efforts directed at the secondary prevention of CHD and improvements in medical care resulting in a lower case fatality rate in patients with acute manifestations of CHD. These advances would include such measures as the establishment and more effective utilization of coronary care units, more refined development and increased application of short-term and long-term (after hospital discharge) therapeutic interventions and greater access to, and use of, emergency medical services.

Two investigations have thoughtfully attempted to sort out the contributions of changes in lifestyle characteristics from those of secondary prevention during the period of declining CHD mortality rates from 1968 to 1976.8,9 Although the investigators were aware of the difficulty in carrying out such an assessment and of the lack of precision with regard to the availability of selected data and estimates thereof, efforts of both primary and secondary prevention were shown to have important impacts on declining CHD death rates. A particularly important influence on these declining death rates was attributed to favorable alterations in the major coronary predisposing factors.

In a recent issue of Circulation, Gillum19 furthers our insights into recent (1980–1985) temporal trends in the occurrence of sudden coronary death from a more broad-based, demographically mixed, national perspective. With routinely collected epidemiologic data, albeit nonvalidated, from the National Center for Health Statistics, deaths due to heart disease occurring out of the hospital or in hospital emergency rooms were examined to provide proxy representations for the occurrence of sudden coronary death. These data, assembled from 40 states and representing approximately 70% of the nation’s population, were drawn from cause of death information reported on the death certificate for persons aged 35–74 years who were further classified by race and sex.

The results confirm earlier cornerstone studies showing that approximately half of all deaths due to

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ischemic heart disease (IHD) either occur outside of the hospital setting or in emergency rooms. The results reported by Gillum further extend these observations to a wider geographic foundation and to recent time periods. Trends of persistently higher percentages of IHD deaths occurring out of hospital in blacks than in whites were seen. In examining secular trends in these sudden death rates, encouraging and consistent declines were seen in out-of-hospital and emergency room deaths between the study years of 1980 and 1985 for the various race and sex subgroups. For example, age-adjusted out-of-hospital plus emergency room IHD deaths declined by 19% in white men, 16% in white women, 20% in black men, and 19% in black women. Further analysis of these death rates revealed that the out-of-hospital death rates sharply decreased over time, whereas the death rates in emergency rooms actually increased, with some differences in these trends observed for race and sex. As expected, the death rates due to IHD increased with age, most notably among men; the greatest observed decline in these mortality rates were seen in the oldest age groupings and across all subgroups examined.

Given the focus of this important contribution, what available information do we have at present concerning the mechanisms of sudden coronary death, identification of individuals at risk for this event, impact of preventive and therapeutic strategies, and secular changes in its occurrence?

In his pioneer description and review of the syndromes of obstructive coronary artery disease, Herrick drew attention to the clinical and pathologic features of myocardial infarction and sudden cardiac death. However, it was not until a half century later that the full importance of sudden death as a public health problem was realized, based on the results of a community-wide study of sudden deaths in Baltimore by Kuller and colleagues. This landmark effort placed the magnitude of sudden death in a more proper perspective and called attention to the unique features of this disease and rapidity of death soon after the onset of symptoms. Based on these findings, a strong argument was made for the combined efforts of preventive medicine, emergency medical care, and in-hospital treatment to reduce the community burden from sudden cardiac death.

Efforts to comprehensively understand sudden coronary death and its underlying mechanisms, treatment, and ultimately prevention have been hampered by the multiplicity of definitions used to characterize its occurrence. Some investigations have included only those deaths occurring instantaneously, whereas others have included deaths within 1, 2, or 24 hours of the onset of acute symptoms. Some studies have included only those cases in which the death was witnessed or was unexpected or in which the victim was not known to have underlying coronary disease. Most sudden deaths occur within 2 hours of the onset of symptoms and are witnessed, and about one half of the decedents have a known history of heart disease.

In terms of its mechanisms, the principle cause of sudden coronary death is ventricular fibrillation superimposed on significant obstructive coronary atherosclerosis. It has also been consistently shown that only a minority of successfully resuscitated sudden death victims have any evidence of acute myocardial infarction.

Despite the general agreement that the mechanism of sudden death in patients with CHD is ventricular fibrillation, the precipitating events leading to this arrhythmia have not been clearly delineated. The sudden onset or extension of myocardial ischemia with a concomitant reduction in the ventricular fibrillation threshold has been postulated as a likely precursor to sudden coronary death; alternatively, selective necrosis of myocardial cells may produce irritable ventricular foci leading to electrical instability of the myocardium. Coronary spasm superimposed on an obstructed segment of a coronary artery may lead to extensive or global myocardial ischemia with resultant ventricular fibrillation, and persistent spasm may lead to coronary arterial thrombosis, plaque fissuring, and hemorrhage. Alterations in neural activity and neurohumoral factors have long been implicated in the genesis of sudden cardiac death, and cigarette smoking may act as an acute precipitator of sudden coronary events.

What is the profile of persons at increased risk for sudden death? From an epidemiologic perspective, the risk factors for sudden coronary death consist of essentially the same factors associated with an increased risk for CHD. This shared constellation of risk factors for CHD and sudden death is particularly seen in men, and somewhat different operative factors are seen in women. Extensive occlusive coronary artery involvement, with or without accompanying clinical symptoms, places individuals at particularly increased risk for sudden coronary death; this risk is further magnified by the presence of left ventricular dysfunction, left ventricular aneurysm, or high grade ventricular ectopy. Survivors of ventricular fibrillation accompanied by acute myocardial infarction are at particularly high risk for recurrence as are those individuals exhibiting sustained ventricular tachycardia during programmed ventricular stimulation in the electrophysiology laboratory. The inevitable conclusion of these pathologic, clinical, and epidemiologic studies is that the prevention of sudden coronary death is synonymous with the prevention of coronary atherosclerosis.

A variety of strategies have been suggested for the prevention of sudden coronary death as well as for its early identification and application of acute intervention strategies. Given the well-established link between the major coronary risk factors and occurrence of sudden coronary death, considerable efforts are being expended to correct these risk
factors through both hygienic and pharmacologic measures with the ultimate goal of preventing coronary atherosclerosis.

In reviewing the major primary prevention trials of CHD, the pooled findings from these trials failed to support a statistically significant effect of the various trial interventions on the incidence rates of sudden cardiac death; the overall trends of these trials were nonetheless in a positive direction. In examining the principal, randomized secondary prevention trials in patients with myocardial infarction, a consistently beneficial effect on the subsequent occurrence of sudden coronary death was seen for \( \beta \)-blocking agents with little effect observed for antiplatelet or cholesterol-lowering agents.

Additional strategies for preventing the occurrence of sudden coronary death include those designed to prevent or impede the progression of coronary atherosclerosis, to detect and treat coronary atherosclerosis in its early, potentially modifiable stages, and to prevent the development of ventricular fibrillation in patients with clinically manifest coronary disease through the use of therapeutic agents. The improved delivery and utilization of community-based emergency medical services, training of emergency medical technicians in advanced cardiac life support and early defibrillation, use of recent technologic advances including automatic external defibrillators, citizen-based CPR training, and the encouragement of CPR training for the immediate family members of patients with known CHD have been proposed and used as approaches in the treatment of sudden coronary death.

What evidence has been collected from a population-based standpoint to suggest that improvements over time in the out-of-hospital death rates due to CHD are occurring? Despite the use of different criteria to define out-of-hospital deaths due to CHD, we and others have shown consistent declines in the incidence rates of out-of-hospital deaths due to CHD, notably sudden cardiac death. In the Worcester Heart Attack Study, we observed a consistent decline in the age-adjusted incidence rates of out-of-hospital deaths due to CHD between calendar years 1975, 1978, 1981, and 1984 for an overall decrease in these mortality rates of 44% between 1975 (265 per 100,000) and 1984 (148 per 100,000). This study also showed a decline in the incidence rates of initial acute myocardial infarction between the anchor years studied. Moreover, there was a consistent improvement in in-hospital survival throughout the four study periods. Declines in the occurrence rates of sudden death have also been observed in the Minneapolis–St. Paul metropolitan area between the two study years of 1970 and 1978, in Rochester, Minnesota, between the late 1950s and the early 1970s, in Allegheny County, Pennsylvania, between 1970 and 1981, in Auckland, New Zealand, between the study years of 1974 and 1981, and among DuPont employees between 1972 and 1983.

Given the vagaries inherent to the death certificate, possible coding and misclassification errors and lack of validating information from other sources such as postmortem reports, medical records and next-of-kin interviews, appropriate caution should be exercised in the interpretation of the published data of Gillum as fully acknowledged by the author. The author’s encouraging findings also beg for further analyses of the available data as well as of the previously described investigations where such information may exist. To more fully describe changes over time in the attack rates of sudden coronary death, first or incident events should be distinguished from those in which a history of heart disease was present before cardiac arrest. Whether or not these declines in the death rates of sudden death have been uniform or differ by region of the country, urbanized or rural areas, and by sociodemographic and socioeconomic status would provide further insight into those factors associated with sudden coronary death and high-risk populations. Reasons for the increased propensity to sudden death in men compared with women and for the considerable sex differences in the risk profile for sudden cardiac death remain unexplained and only speculative at the present time. Moreover, given the relative paucity of available data describing changes over time in the time interval between onset of acute symptoms suggestive of CHD and activation of the medical care system, data on these trends are sorely needed to identify further health education needs and techniques to mobilize public health efforts. Identification of the precipitators of sudden coronary death and the means by which such precipitating factors might be modified remains critically needed. Given recent suggestive evidence of the occurrence of circadian variation in acute myocardial infarction and sudden cardiac death, further explanation of this phenomenon and its association with acute coronary event triggers should be carried out. The role of psychosocial factors, including stress, and the occurrence of sudden cardiac death should be further explored given the recently observed relation between such factors and sudden cardiac death in discharged hospital survivors of acute myocardial infarction. Lastly, further data need to be collected and strategies evaluated by which emergency medical services may be more efficiently used in urban and rural settings with the systematic application of promising intervention strategies.

The information contained in Gillum’s timely article provides a rich descriptive perspective of declining national IHD death rates. Furthermore, this study targets areas for future research and improvements from the point of view of both the patient and health care provider. Irrespective of the reasons for these declining death rates, these findings provide further encouragement for the appa-
ent benefits of favorable lifestyle habits, medical care interventions, and for “doing the right things.”

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