Heart Disease

Following are two of the papers presented at a session devoted to heart disease during the September 1957 meeting of the American Statistical Association in Atlantic City, N. J.

Methods of Studying the Ecology of Coronary Heart Disease

MORTON ROBINS, M.S.P.H., and ALFRED M. STEINMAN, M.D.

DURING the past 2 years we have witnessed a tremendous surge of interest in the ecology of coronary heart disease. A conference on the epidemiology of atherosclerosis and hypertension (*1*), a symposium on measuring the risk of coronary heart disease (*2*), and numerous papers and articles on this leading cause of death (*3–5*)—all have served to whet the appetite of both the medical and public health profession for more definitive data. The public is looking for widespread programs for control or prevention, but while our knowledge of coronary heart disease has increased and many leads on etiological factors have been obtained, preventive programs have not yet been recommended. Emphasis is still on the need for additional research into causation.

The development of coronary heart disease is generally agreed to be the result of complex, unidentified interactions between the human host, with his variable susceptibility, either genetic or acquired, and his total environment.

The study of such interactions is called "human medical ecology" (*6*) or "scientific epidemiology" (*7, 8*). Ecology emphasizes the nature of multiple causation and seeks to integrate the diversity of factors involved in disease and to synthesize hypotheses on causation. The ecologic approach has rarely been used in epidemiological investigations of infectious diseases since these studies have been concerned principally with identification of the causative agent—the micro-organism. Interest in medical ecology was revived when epidemiological inquiry was broadened to encompass the noninfectious chronic diseases of unknown etiology. Thus, medical ecology and epidemiology are blood brothers.

The ecologic approach is new, complicated, and expensive. Because of the shortages of trained investigators—at times requiring a team composed of clinician, epidemiologist, biostatistician, and experts from the basic and applied sciences—only a small number of epidemiological studies are currently under way. There is therefore a need to examine our resources and choose our studies with circumspection.

Considering the variation in age-specific incidence rates, the difficulty of accurate diagnosis, and the chronic nature of the disease, which of the alternative methods of data collection is best

Mr. Robins is chief of the Biometrics and Evaluation Division, and Dr. Steinman, chief of the Clinical Studies Division, in the Department of Medicine and Surgery, Veterans Administration.

suited to study causation? An evaluation of the various data collection methods may give some perspective in making a proper choice. A review of ecologic factors may also reveal opportunities for field studies.

Ecologic Factors

Coronary heart disease is ubiquitous, with increasing morbidity and mortality in both young and old. Physicians are able, to some extent, to treat the result of the disease, but they are unable to prevent it. Prevention must hinge upon a better understanding of the basic role that each of the many known, suspected, and as yet unknown factors play in the creation of a thrombus in a coronary artery which has become atherosclerotic.

Factors incriminated as causes of the disease have been identified or evaluated in monographs and texts on coronary heart disease (*9*, *10*). An excellent summarization of our current state of knowledge concerning the pathogenesis of the disease is found in a recent paper by Miller and associates (*4*), and in the report of the Conference on Epidemiology of Atherosclerosis and Hypertension (*1*). In order to serve as a backdrop for the discussion of study methods, a brief description follows of what we believe to be the factors which require special investigation.

Experimental studies designed to produce atherosclerosis deliberately in animals have led to implications currently being utilized in planning clinical studies in humans, for example, hormone and diet studies. However, the experimental lesions produced in animals are rarely associated with thrombosis, which leads to the suspicion that blood coagulability may be altered.

Another approach to identification of etiological factors is through correlative, fact-finding studies. In essence, most of these studies seek to establish the association of a given physical feature, habit pattern, or result of a physiological or chemical test with the presence of the disease.

The clinician is aware of certain seemingly hereditary factors which appear to be of some importance. Coronary heart disease is observed more frequently in mesomorphs than in other somatotypes (*11*). In many instances, the disease is seen in each generation of a given family (*12*). This suggests the probability that certain basic differences—anatomical, psychological, metabolic, or mechanical—exist among members of such families as compared with other groups. This feeling is strengthened by the observation that a synergistic relationship exists between coronary heart disease and certain other diseases with familial concentrations, that is, diabetes, xanthomatosis, hypercholesterolemia, and hypertension (*13*, *14*). These conditions are considered significant when present in males under age 40, and perhaps even more so, in females.

Since some people are nearly entirely free of atheromata while others have dramatic depositions, some basic metabolic defect must be suspected which produces the atheromatous substances. Atherosclerotic plaques may be formed not only in the coronary arteries but in the vessels of the brain, legs, and other body organs and sites. A further question arises from the lack of uniformity in the distribution of atherosclerosis—the presence of plaques at one site bears no necessary relationship to their presence or absence at other principal locations. In addition, in hens, estrogens inhibit the incidence of experimental coronary but not aortic atherosclerosis (*15*).

Other factors, categorized as environmental influences, also play a role. The effect of climatic changes, ultraviolet light, and smoking have been incriminated. Nutrition probably has an important relationship to the metabolic dysfunction possibly involved in the pathogenesis of the disease. However, specific dietary factors have not as yet been "validated." The relative role of physical activity and the reactions to mental, physical, and biological stress are other significant variables that need further study and quantification.

In regard to the direction of concentrated research in the future, the group of patients under age 40 with an accelerated form of the disease may provide the best source of information ecologically. How do those with manifestations of the disease differ from others in their genetic makeup and habit patterns, including

diet, occupation, mode of living, and associated disease? If significant differences are discovered, are they coexistent or correlated?

The answer to these and many other questions will lead to newer ones. These in turn will lead to the attack upon what we believe is the underlying process: a metabolic defect which produces either atherosclerotic or thrombotic changes, or both, in the coronary arteries, accelerated by such factors as diet, hormonal changes, and habit patterns.

Epidemiological Approaches

Let us now consider the epidemiological approaches in the search for causes of coronary heart disease. We can classify these methodologies into six broad categories: mortality data, hospital statistics, morbidity surveys, morbidity reporting, longitudinal studies, and selected patient cohorts.

Mortality Data

Information on death certificates has traditionally been analyzed to supply clues on statistical associations between demographic characteristics of the population and the incidence of disease. Inferences from analyses of mortality figures have often been substantiated by findings from other epidemiological studies. A large number of statistical studies of coronary heart disease mortality data have been made, and significant differences have been reported for such factors as age, sex, marital status, income, occupation, ethnic group, height, and weight (*1*). The relationship between dietary fat consumption and coronary heart disease mortality reported by Keys has recently been challenged by Yerushalmy and Hilleboe (*16*).

It has been suggested that mortality analyses should be pursued because the source data are readily accessible and the study costs are low compared with other epidemiology approaches (*1*). Moreover, more intensive studies can be designed to increase the epidemiological significance of observed mortality differentials. These mortality studies would start with facts on death certificates and then add information by means of field investigations or by questionnaires to the attending physician and relatives of the decedent. In addition to gathering facts on events leading to death, the studies might provide valuable information on the accuracy of cause of death certification. More profitable coronary heart disease mortality data can also be derived from studies in selected localities where death certification practices are believed to be fairly uniform and accurate.

The major weaknesses of mortality statistics on coronary heart disease as a source of ecologic data are: first, variation in the completeness and accuracy in medical certification of deaths due to the disease; second, questionable correlation between specific mortality rates and incidence of the disease; and third, the impossibility of analyzing from routine tabulations of mortality data many of the ecologic factors believed to be significant in the pathogenesis of the disease.

Accuracy and completeness of cause of death certification depend on the quantity and quality of available physicians' services, and vary with concepts held by physicians concerning the

Guide for Avoiding Arteriosclerosis

A statement advising laymen on how to resist arteriosclerosis, coronary thrombosis, and brain thrombosis has been issued by the National Health Education Committee, Inc., of New York City.

According to the physicians who signed the statement, Dr. Paul D. White, Dr. Howard B. Sprague, Dr. Samuel A. Levine, and Dr. Frederick J. Stare, all of Boston, there are five predisposing factors. They are listed as heredity, overweight, elevated cholesterol level, elevated blood pressure, and excessive cigarette smoking. Hard work in itself, they maintained, cannot be considered a factor.

Persons with a strong hereditary background of arteriosclerosis are particularly cautioned to minimize the effects of the other factors.

The statement lists a number of documents referring to the factors listed. Stressing the importance of arteriosclerosis of the heart and brain, the statement noted that heart and circulatory disease caused the largest number of deaths in the United States. Of the 843,410 deaths in 1956 in this category, most were due to arteriosclerosis.

manifestations of coronary heart disease. These differences in death reporting practices in various subgroups of the population can alter the actual association of coronary heart disease mortality and a particular demographic characteristic (*17*). Yerushalmy and Hilleboe's study of heart disease mortality statistics for different countries indicates that large numbers of coronary heart disease deaths are reported variably, and are classified as "degenerative heart disease" or as "other diseases of heart" (*16*).

A basic epidemiological question is whether differential mortality can be used as an index of the relative incidence of initial coronary heart disease attacks among specific population groups, since the disease is chronic and most initial attacks are not fatal. Attacks often recur over a period of many years before the fatal attack occurs. Thus, the age distribution of deaths from the disease may be markedly different from that of patients having an initial attack. Moreover, survivors of an initial attack frequently change their occupation to compensate for their residual disability or from fear of physical exertion. These facts should be recognized in evaluating the epidemiological significance of the mortality differentials of the disease by age, income, occupation, socioeconomic status, and place of residence.

Notwithstanding their limitations, mortality data provide an index of the size and seriousness of the problem. In the absence of specific morbidity and case fatality data, analyses of mortality trends supply indirect evidence of changes in incidence or advances in therapeutic procedures.

Hospital Statistics

The growth of hospital facilities accompanying the expansion of hospitalization insurance coverage and medical care for indigents suggests the increased usefulness of hospital statistics as a measure of coronary heart disease morbidity. Some merit is attached to proposals for a statistical study of coronary heart disease admissions or discharges from a group of hospitals that supply all the inpatient care requirements of a particular area. An example of integrated hospital statistics for a known population is provided by the experience of the Saskatchewan Hospital Services Plan (*18*). In the United States, the Commission on Professional and Hospital Activities, Inc. (Ann Arbor), has assembled detailed statistics on patients discharged since January 1, 1953, from hospitals comprising the Southwestern Michigan Hospital Council.

Hospital statistics are considered by some to represent, at worst, a compromise between mortality statistics and complete morbidity data. Extensive hospital morbidity surveys were conducted in New York City in 1933 (*19*), in Oxford, England, in 1943 (*20*), and in Ontario, Canada, in 1951 (*21*). More recently, a large pilot study was conducted in New York City to demonstrate the feasibility of a hospital reporting system as a source of providing useful information on morbidity and for planning medical care programs (*22*).

While the data on coronary heart disease in tabulations of hospital records suggest differentials in morbidity, their validity has not been ascertained. The question still unanswered is whether the differentials in hospital admission rates are comparable to those based on total incidence of coronary heart disease according to sex, age, race, occupation, and other demographic characteristics. Studies are needed to correlate hospital statistics for a community with estimates of morbidity. In such studies analysis should be made separately for each of the manifestations of coronary heart disease, that is angina pectoris, abnormal electrocardiogram not related to other disease states, and myocardial infarction. Until these questions are answered, indexes of morbidity from hospital admission rates require confirmation by other epidemiological studies.

Detailed clinical, laboratory, and medical history data on hospitalized coronary heart disease patients may also supply leads for other statistical associations related to ecologic factors.

Morbidity Surveys

Morbidity surveys on the health status of a population may take two forms: interviews in sample households, or a complete canvass of physicians and hospitals in a community.

To our knowledge, such a complete survey of physicians and hospitals has not been applied

to the study of coronary heart disease morbidity, but it has been used to obtain morbidity data on cancer. The canvass method is expensive and requires protracted negotiations with medical societies, hospital associations, and health departments. Although the method has been practicable occasionally for cancer morbidity surveys, its results would be highly questionable when applied to coronary heart disease. As contrasted with cancer where pathological laboratory records, hospital records, and mortality reports form a large part of the total morbidity picture, the canvass method applied to coronary heart disease morbidity would rely primarily on reports by cooperating physicians. Retrospective reporting of coronary heart disease is not, therefore, likely to yield a complete picture of its incidence during a prior year.

Morbidity surveys using data from household interviews of a scientifically selected sample of a community, a State, or the Nation have provided epidemiological data on coronary heart disease. An appraisal of heart disease morbidity derived from the National Health Survey of 1935–36 was made by Collins (*23*) in 1949. More recently, findings have been reported based on morbidity surveys conducted in Hunterdon County, N. J. (*24*), Baltimore, Md. (*25*), and the State of California (*26*). All of these reports generally agree on the uncertain validity of the computed prevalence rates for such chronic diseases as coronary heart disease. Incidence rates derived from data obtained in household surveys are even more tenuous.

Comparisons of coronary heart disease prevalence as measured by household interviews and clinical examinations indicate that the interview data provide minimum estimates. It would also appear that the reported prevalence rates for various subgroups of the surveyed population are subject to varying correction factors of uncertain magnitude.

Despite these limitations, valuable demographic and epidemiological data are byproducts of household morbidity surveys. Estimates are obtained of ecologic characteristics not otherwise available. Such data are useful in assessing concurrent data from morbidity reporting projects, and hospital and mortality studies of the same community. The survey may also supply rosters of individuals presumably free of coronary heart disease. These randomly selected persons can serve as controls for study patients identified by other study methods.

Morbidity Reporting

Ideally, voluntary reporting by physicians of all new cases of coronary heart disease as they are diagnosed provides the cheapest and most direct approach for acquiring data on incidence. In an appeal in 1930 for voluntary reporting by New York State physicians of certain facts relating to heart disease, Dr. J. V. DePorte, of the New York State Department of Health, said, "In this day, when the immutability of even chemical elements is no longer an axiom, the rigid grouping of diseases into communicable and noncommunicable seems to be altogether artificial. . . . A group of diseases which incapacitate about 300,000 persons in the State is certainly a matter that cannot be excluded from the field of legitimate public health activities by the mere fiat of our individualistic tradition (*27*)." No action was ever taken on his proposal.

In 1956, Dr. P. D. White called attention to the increased collaboration between the epidemiologist, the cardiologist, and the family doctor as illustrated by their participation in an epidemiological survey of coronary heart disease in the Grand Forks, N. Dak., area (*28*). During this survey all physicians in the area notified a central committee of each new case of coronary heart disease diagnosed. The reports were supplemented by a household survey giving descriptive data of the population in terms of ethnic background, diet, exercise, habits, stress, smoking habits, and other characteristics. Rosters of the surveyed group are matched against cases of coronary heart disease reported and act as controls in followup studies of both groups. A similarly organized study started in January 1956 in Middlesex County, Conn.

Despite this and other evidence of increased community and physician interest in coronary heart disease, it does not appear likely that voluntary reporting is a practical approach in many localities.

More realistic perhaps is the use of this ap-

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proach to determine the incidence of coronary heart disease among more circumscribed groups. Valuable ecologic data can be assembled, for example, on persons covered by comprehensive medical care programs where both physician services and medical records are integrated.

Longitudinal Studies

In prospective or followup studies of coronary heart disease, a cohort of the general population, considered to be free of the disease, is observed over a long period of time to determine the natural history of the disease process. Information is sought on signs or symptoms considered to be precursors to clinical manifestations of the disease. The incidence of conditions can be related to multiple characteristics of the study population which are ascertained by interview, medical examination, or diagnostic tests. Prognosis can be measured in terms of progression of clinical signs and symptoms with reference to the characteristics of each case. In a longitudinal study, provision can also be made after the study has started to introduce a new study variable meriting investigation.

The major limitations of longitudinal studies are: (*a*) technical difficulties in organizing the study and high costs over the long period required for data collection; (*b*) attrition of the study group due to noncooperation or movement out of the study area; (*c*) change of observers throughout the course of the study; and (*d*) changes in the normal living patterns of the study subjects when they are conscious of the existence of precursors to clinical coronary heart disease.

The first two limitations lead to the question of how to select the study population. Should the study group be a sample of the general population? Or should the study group include all or a sample of a specially constituted group? Obviously, study of the general population permits more valid generalization. However, studies of specially constituted groups, such as industrial employees, Veterans Administration beneficiaries, and participants of pension, disability, or health insurance plans, have the advantages of lower attrition rates and greater cooperation of the study group. Moreover, study costs are appreciably lower, since medical and laboratory facilities are available and medical records are generally maintained for other medical care purposes.

While selection factors may be present with such variables as age, sex, race, activity status, and income, the observed experience of selected groups can be adjusted by available biometric techniques. In this connection, approximately the same average annual incidence of coronary heart disease, 7 per 1,000, was observed among male civil service employees in the age group 40–54 in Albany, N. Y. (*29*), and in Los Angeles, Calif. (*30*), as was found in a sample of the total male residents of Framingham, Mass. (*31*), in the same age group.

Selected Patient Cohorts

Coronary heart disease patients, or persons having a disease believed to be synergistic in the pathogenesis of heart disease, provide special cohorts for ecologic study. Ideally, these study groups should be scientifically selected from the general universe of patients with the disease. Practical considerations, however, commonly lead to a decision to select hospitalized patients or outpatients as a study unit.

How representative such a group is of the general class of patients from which it is drawn and the methodology of the study plan are concerns of the statistician. The restrictions on the interpretation of findings and the procedures necessary to avoid either erroneous or spurious statistical associations have already been described by Berkson (*32*), Kraus (*33*), Lilienfeld (*34*), and Moore (*35*). With proper concern for the study design, patient cohorts furnish excellent case material for the study of metabolic defects and specific environmental influences as they relate to the incidence of coronary heart disease.

Retrospective study of coronary heart disease cases generally consists of inquiry into their medical history for suspected antecedent events. Comparison with similar data from a control population provides an indirect method for estimating differential incidence rates. However, when hospitalized patients are used as the study group, the validity of procedures for estimating relative risks (*36*) is dependent on the degree to which the hospital patients

and the selected controls are representative of these same groups in the general population.

Generally speaking, the longitudinal rather than the indirect approach is recommended for the study of diseases with relatively high incidence. While the total incidence of coronary heart disease is considered to be sufficiently high for longitudinal studies, the incidence among adults under 40 years of age has been found to be so low that the use of the retrospective approach is suggested for this age group. The study of coronary heart disease among these young adults, particularly those without evidence of hypercholesterolemia, hypertension, or diabetes, is of special ecologic interest. This group of patients represents an accelerated form of atherosclerosis.

"Recovered" coronary heart disease patients also serve as cohorts for prospective studies to evaluate the effectiveness of control or prophylactic measures in preventing recurrent attacks. These exploratory studies may provide an understanding of at least one of the components of the causative complex. The advantages of using a cohort of patients instead of a sample of the general population are fairly obvious.

Prospective, longitudinal studies of individuals considered to be particularly susceptible to coronary heart disease, such as diabetics and hypertensives, should also be pursued more intensively than in the past. The size of such cohorts need not be as large as those for longitudinal studies of the general population since the expected differential incidence rates are higher. Moreover, longitudinal studies of these patient groups may possibly suffer from less attrition because of greater interest in their disease. Determination of the environmental factors which trigger symptomatology among these highly susceptible individuals would add significantly to our understanding of the ecology of the disease.

Summary

Public health authorities, who are confronted with the task of reducing the incidence of coronary heart disease, must base their policies and programs on knowledge of the causes. A review of ecologic factors indicates that future preventive programs will probably be related to the control of the causes of coronary atheroma and of the environmental influences which accelerate atheromatous depositions and coronary thrombosis.

Epidemiological data are needed to define the differential incidence of the disease among persons with different characteristics and living under different environmental conditions. Such data, when added to pertinent knowledge derived from clinical sources, animal experimentation, and laboratory sciences, will eventually enable the medical ecologist to synthesize a pattern of the multiple causative factors involved.

From this review it is evident that no one all-encompassing field study is likely to settle the epidemiology of coronary heart disease. Significant evidence on causation can be derived from one approach which is not within the capabilities of another. However, because of the short supply of technicians needed for epidemiological research, it is necessary to proceed piecemeal in order to fill the important gaps in our knowledge.

Hypotheses should be intensively investigated by the most rigorous epidemiological method available, despite known limitations in our ability to generalize from the findings. This recommendation does not imply indiscriminate collection of information nor disregard for the principles of validity and reliability.

Three approaches are suggested for future epidemiological studies of coronary heart disease:

1. Routine reporting for a limited time period of initial manifestations of coronary heart disease among adult populations whenever practicable.

2. Longitudinal studies of selected groups whose physical, physiological, and psychological characteristics are determined at the beginning of the observation period.

3. Retrospective and prospective studies of patient populations to determine the relationship between underlying metabolic defects and environmental influences and the incidence of coronary heart disease.

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Some Observations on the Epidemiology of Heart Disease

TAVIA GORDON

CERTAINLY, the problems in studying heart disease are complex and difficult. To discuss them in detail is obviously impossible here. I will therefore confine myself to a few simple observations.

The first is that the study of heart disease is to a considerable degree still a study of deaths. All too often the first indication we have that a person's coronary artery isn't all it ought to be is when he dies. The final evaluation of an attack of coronary artery disease requires an analysis of changes in the coronary artery and this can be done only by autopsy. There is no equivalent to the biopsy in the study of heart disease.

The second observation is that we must make a distinction between the age group under 65 and the age group over 65 years. When we speak of the alarming increase in heart disease in this country we are referring to the rise in mortality among white men aged 45–64. When we speak of the difficulties of diagnosis we are referring primarily to events after age 65 and secondarily to events among middle-aged women, which are apt to be equivocal.

The study of heart disease among old people is really for the future. I doubt whether medical science is far enough advanced at present to adequately describe the complexity of chronic illness at advanced ages. Medical pathology is certainly of little help. Usually, the pathologist's report clearly indicates that the person was extensively diseased; the wonder is that he lived as long as he did; but it is difficult to delimit from the multiplicity of defects present any specific, well-defined etiology. Nor is the clinical picture much more help. As has been pointed out, all the organ systems fail at death—the lungs, the liver, the kidneys, and so forth, as well as the heart—but in the absence of a clear-cut etiology the failure of the heart will tend to dominate the picture. If, however, we confine our attention to the study of coronary artery disease among middle-aged men I think we are in a good position to tag our cases and to investigate the epidemiology of the disease.

Unfortunately, vital statistics has gotten itself into a dilemma in the reporting of deaths among older people. If a doctor, faced with a complex and poorly defined pathology, reports a death as due to "old age," the local registrar will in all likelihood request a more definite cause of death. Any student of medical ecology knows what happens next. After a while the doctor starts giving definite answers even when he has only the vaguest notion of the cause of death. And then, of course, the vital statistician becomes understandably skeptical about the reporting of cause of death.

This skepticism about the reporting of cause of death, which is practically an occupational disease of vital statisticians, seems to me grossly exaggerated. I think much of it would evaporate if the skeptics ever attempted to reassign the deaths attributed to heart disease. There are just too many of them. Either they represent a substantial reality or some other diseases represent a public health problem of much greater magnitude than anyone has previously suggested.

It must be granted that the difficulties in the reported death statistics are considerable, but few of them can be resolved by contemplation.

Mr. Gordon is statistician with the National Heart Institute, National Institutes of Health, Public Health Service.