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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 evaliation 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

[^0]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.

In particular, rules of internal consistency for mortality statistics seem to me very slippery. If the mortality sex ratio is high among the white population and low among the Negro population, it may be simply that the death rate for arteriosclerotic heart disease is higher among Negro women than among white. We know that serious arteriosclerotic heart disease is very uncommon among middle-aged women unless they have diabetes or some similar metabolic defect. Conceivably such conditions are more common among Negro women than white. Mortality statistics certainly suggest that. Similarly, mortality sex ratios for arteriosclerotic heart disease are much lower in some countries than they are here. Does that mean those countries are reporting some other disease? Or does it mean that there is a real difference in the balance of factors controlling the appearance of the disease? The suggestion of Edward A. Lew of the Metropolitan Life Insurance Co. that there is a strong environmental component in the sex differentials for arteriosclerotic heart disease seems well taken. I think it is also true that there is an environmental component in the age differentials for this disease.

The question of what factors are involved in the development of coronary artery disease, which is the question that epidemiological studies in this field are concerned with, is a very vexed one. Numerous factors have been implicated, with or without evidence, but none of the really important issues has yet been settled. This is not peculiar to heart disease, however. All diseases may be said to involve a complex interrelation of factors, but the use of a simplified approach still has an obvious utility in the investigation of these factors. It is certainly not necessary that all of the critical factors be recognized or understood for a disease to be controlled, and it is conceivable that one or another of the presently considered factors may provide the key to the control of heart disease. Perhaps diet will prove to be that critical factor. Certainly, there is an increasing weight of evidence suggesting this possibility.

Unfortunately, it has become increasingly difficult to make epidemiological studies of diet in the American population. More and more people are dieting, think they are dieting, or
think they ought to be dieting, and as a result their account of what they eat is apt to be confused and unreliable. In such circumstances the controlled nutrition experiments become more and more critical for our understanding of the role of diet. Currently, such experiments most frequently take the following form: some factor or factors, usually the kind of fat, are manipulated within a more or less carefully controlled regimen, while the remainder of the diet is kept the same or adjusted to maintain a constant caloric intake. The effect of this is measured by some index-total cholesterol or some other serum lipid, usually, since there is some evidence that the serum lipids are associated in a rough way with the risk of overt coronary artery disease.

In this kind of investigation the statistician can be of considerable assistance in designing economical inquiries and rationalizing their results. The usual result of these experiments is that the index goes up and down as the diet is altered, but there are no clear-cut criteria for deciding when the effect of one regimen has given way to the effect of another or (put another way) when we have reached an index level characteristic of a specified regimen. How do you decide when you have reached a steady state and when do you decide that this steady state is not transitory? Furthermore, there are individual differences in response, and while these are no doubt very important they are difficult to evaluate. At the moment this is a very complex but a very promising area of research.

Investigation of other factors in heart disease epidemiology can, at this stage, still be undertaken by field studies. The most serious problem in such studies is the low level of response. You are lucky to get two-thirds of any reasonable sample you select to participate in your study. No one seems to have turned up a way of improving the situation. Going to special groups, instead of to a general population sample, does not seem to help. Captive populations are much less cooperative than might be expected.

The problems of bias arising from nonresponse are certainly serious; and admitting them, unfortunately, does not make them any
easier. About all you can do that is worth doing is to find out as much about the nonrespondent group as possible. Prospective studies have some advantages in this regard, because there is a reasonable expectation that after a group has been followed for a time the initial bias will tend to diminish, but there is no gainsaying that reliable estimates of the prevalence of cardiovascular conditions in the general population are going to be hard to come by.

Unfortunately, the morbidity survey, which has proved useful in getting prevalence data for many diseases, has not worked out very well for the chronic diseases in general or for heart disease in particular. Contrary to some im-
pressions it is neither cheap nor easy. What is worse it is exceedingly ineffective in identifying persons who would be considered to have heart disease on the basis of a careful clinical examination. No one will dispute that all available epidemiological techniques ought to be considered for the study of heart disease, but there is little advantage in dispersing our substance in a search for shortcuts. This is a real hazard: We are all impatient for answers to our questions about heart disease, and the routine clinical devices for accumulating pertinent epidemiological information are painfully slow and laborious. But here, as elsewhere, the slow and careful way may yet prove the quickest.

# Shaw Promoted to Assistant Surgeon General 



Dr. James R. Shaw, chief of the Public Health Service's Division of Indian Health, has been promoted to the grade of Assistant Surgeon General.

When the Public Health Service took over the Indian health program from the Department of the Interior in 1955 , Dr. Shaw was made chief of the newly created Di vision of Indian Health. In that capacity, Dr. Shaw is responsible for the administration of medical and public health services for approximately 380,000 American Indians and natives of Alaska.
A career officer of the Public Health Service, Dr. Shaw has been commissioned since 1938. After assignments in Public Health Service hospitals and the U.S. Coast Guard, he became medical officer in charge of the U. S. Marine Hospital, Detroit, in 1949. In 1952 he was named chief of the Service's Division of Hospitals and served in that post until he began his duties in Indian health in 1953 with the Department of the Interior.


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