WHERE DO WE GO FROM HERE ?

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Health problems are essentially a byproduct of civilization, and changes in the levels of that civilization reflect changes in the patterns of diseases indigenous to the populace of the community. Thus, in the United States the infectious and communicable diseases that were once mass killers are now no longer a threat.

The Traditional Concept

The ideology that generated the control of these infectious and communicable diseases emerged from a single basic epidemiologic concept comprising the host, the agent, and the environment (fig. 1) advanced by Gordon (*1*). Under optimum conditions, the interactions between these components resulted in disease that ranged from a single isolated case to epidemics depicted in figure 1's black area.

Once these interactions were identified and the pathogenetic factors understood, the ability to control disease became easier. The physicians, microbiologists, engineers, and personnel of the paramedical disciplines individually and collectively sought ways to make the host more resistant and the agent less available to the susceptibles. The resistance of the host against a disease was increased by various immunologic procedures.

Most efforts to control mass diseases, however, were vested in alteration of the environment, which was the weakest link in the

Figure 1. Traditional epidemiologic concept of interactions of the host, the agent, and the environment

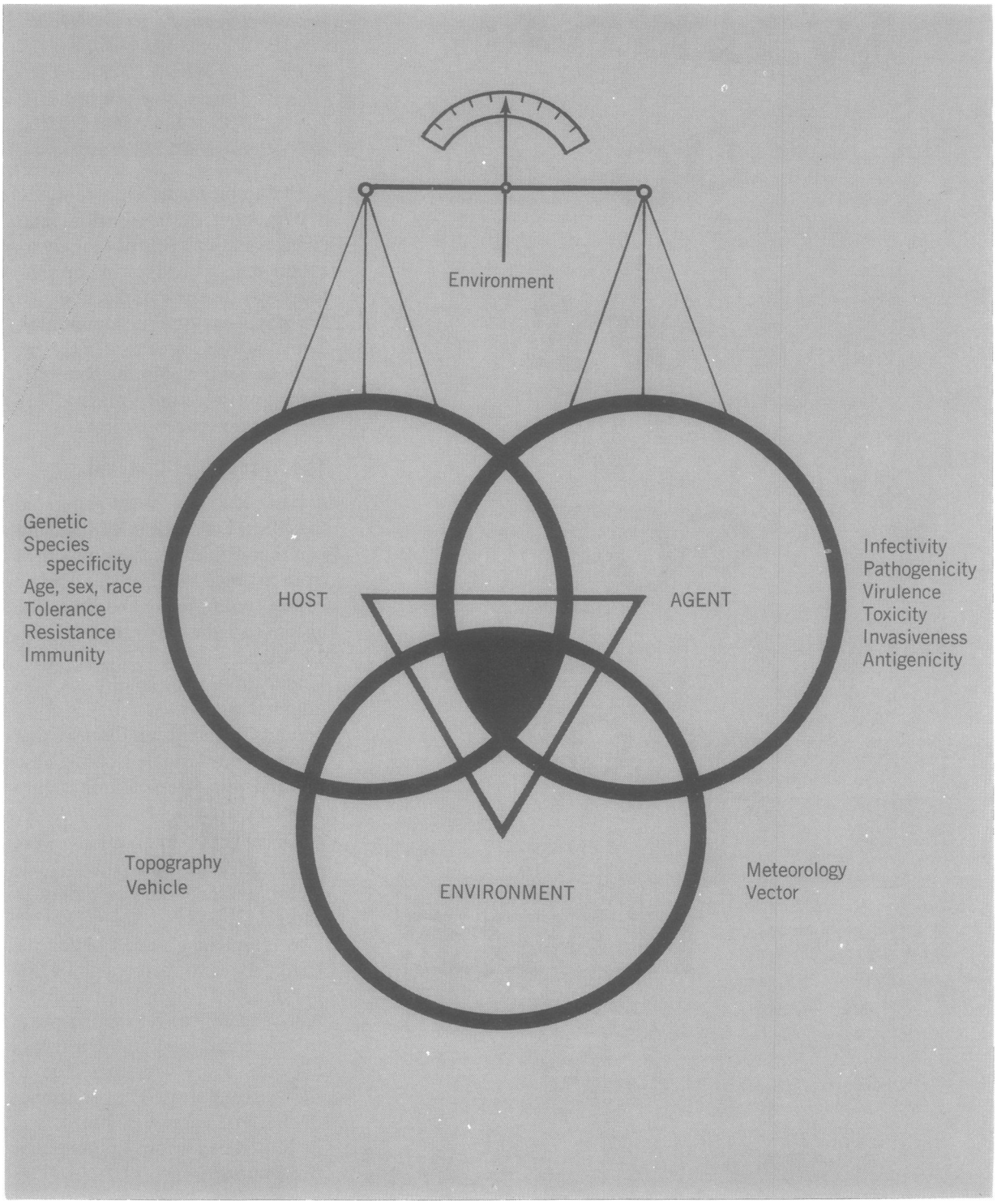
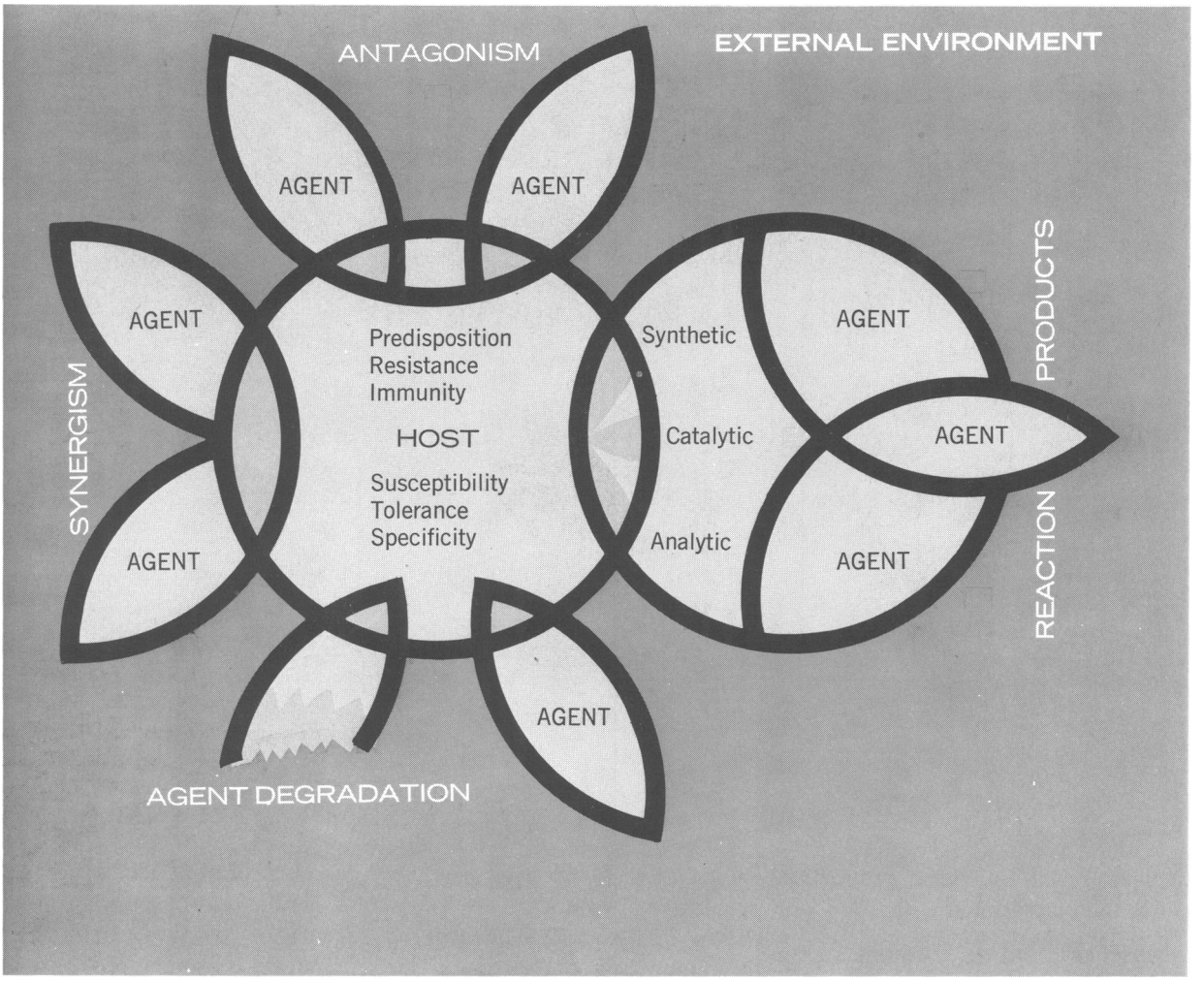


Figure 2. Epidemiologic concept of interactions of the host, the agent, and the environment relating to the chronic degenerative diseases



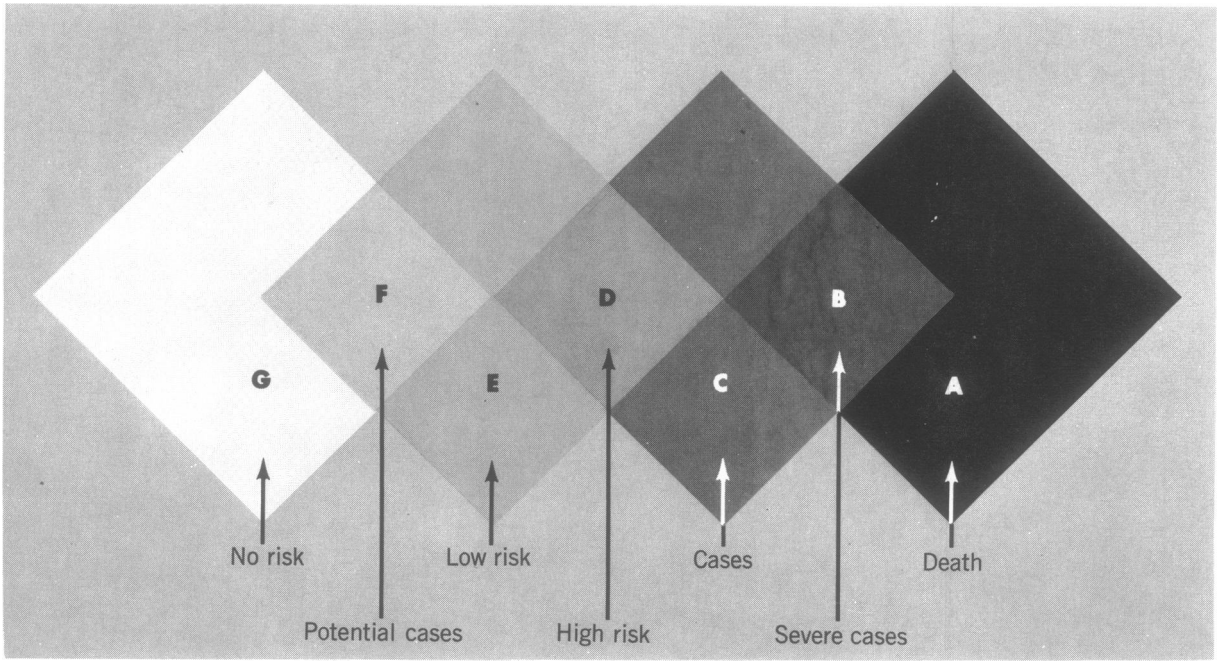
chain of events, was economically feasible, and was readily acceptable by the communities. Thus purification of water, pasteurization of dairy products, and attacks on rodents and vectors, without directly involving people, produced positive results. In figure 1 the bottom circle, representing the environment, thus could be taken out of the interactive mechanism and placed in top position as a tool of surveillance and as a predictor of the occurrence of infectious and communicable diseases.

Unlike the diseases that could be controlled sufficiently by attacking the environment, tuberculosis, a major communicable and infectious disease, has persisted although reduced from epidemic to endemic proportions. Venereal diseases, similarly, still exist and cause concern in public health. Indeed, alleviation of environmental conditions such as crowding and host factors such as poverty, reflecting malnutrition, have helped to reduce the incidence of these diseases.

Casefinding, however, is by far the single most important aspect of the public health movement in controlling these diseases and requires active participation by the host. The ideology of casefinding and host participation concerning the control of tuberculosis and venereal diseases can be easily extended to areas of today's more prevalent diseases, including chronic, degenerative, nonspecific, noninfectious diseases such as neoplasia and cerebrovascular and cardiovascular diseases.

Active cooperation and participation of all members of a society will provide knowledge in depth about these maladies, which

Figure 3. Population segments depicted as successive sources of epidemiologic knowledge



could result in sound programs for their control.

Host as Central Element

Unlike the traditional epidemiologic patterns of infectious and communicable diseases, which in essence separately describe in a triangle the host, the agent, and the environment, the pattern of chronic diseases depicts the host as the central element in the generation and progression of the disease (fig. 2).

Since the etiologies of most contemporary diseases are unknown, the environment ill-defined, and the host factors obscure, the weakest link in the chain of events that precipitates a disease is difficult to find. An outcome of the need to develop and to modify the traditional epidemiologic approach to the problems of modern disease is shown in figure 2. The host in this figure is a central one. The characteristics of the host—internal environment, genetic make-up and predisposition, natural and artificial resistance, susceptibility, immunity, and tolerance—are paramount.

Specificity in the host refers to specific anatomical sites where specific diseases, such as duodenal ulcer, coronary thrombosis, gout, carcinoma of the breast, and so on, generate. The external environment, which in essence is composed of food, water, and air, is modified by socioeconomic, psychosocial, rural, and urban complexes, and is further complicated by the person's habits and hobbies, such as smoking, use of alcohol, use of drugs, and so forth. The obscurities of the agents of today's diseases can therefore be unmasked only by full understanding of the synergistics, antagonistics, and effects of the end products of the interactions of all the external and internal environments of the host.

Keeping figure 2 in mind let us, for example, examine the complexities of heart disease—the number one cause of death in the United States. During 1960–62, the Department of Health, Education and Welfare (*2*) reported that of 111.1 million adults in the United States some 14.6 million had definite heart disease and nearly the same number had suspect heart disease. Of every 100 persons aged 18 to 79 years, 13.2 had definite heart

disease, while an additional 11.7 had suspect heart disease. These diseases included hypertensive, coronary, rheumatic, congenital, syphilitic, and other categories. For further discussion, I shall dwell on coronary heart disease.

Population Patterns

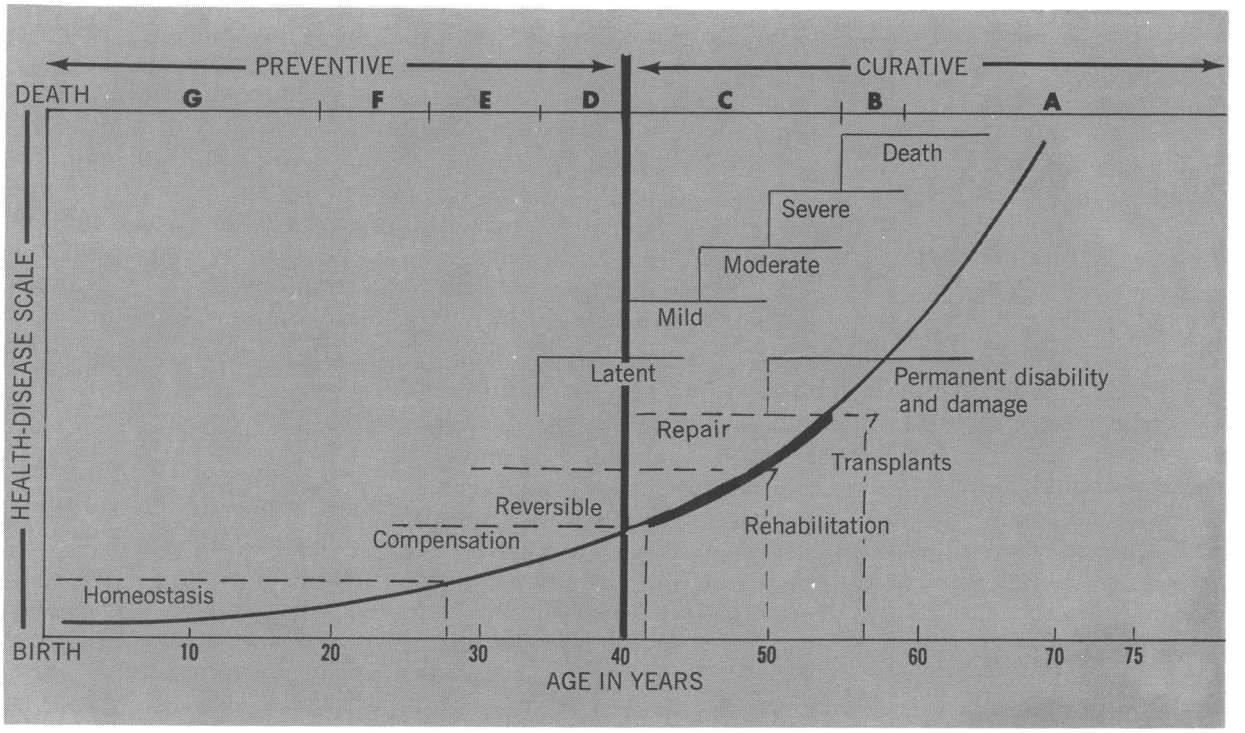
How much do we know about coronary heart disease in terms of the host, the environment, and the agent? My answer is that we know a great deal about the characteristics of this disease after the event has occurred but not enough to prevent it from occurring. At one time all heart diseases were simply clinical concepts, labeled as "heart attacks." The various names now given them are descriptions of the clinical manifestations, derived primarily from necropsy examinations. Advents and advances in knowledge have since been forthcoming from the populations conceptualized in figure 3.

The first knowledge of the few characteristics of coronary disease, in addition to and in some instances apart from the mere clinical concepts of the disease, came from population segment A (people who died of heart attacks) on the basis of their morbid anatomy. In retrospect this knowledge was coupled with the symptoms observed in these people before death. Similar circumstances in living patients, then, could be used as prognostic of a heart attack in population segments B and C. The challenge was to acquire more precise knowledge about the disease from these groups.

A biochemical variable, such as serum cholesterol, in severely ill persons was found to be elevated and related to coronary heart disease. Similarly, a physiological condition (elevated blood pressure) was found to be related to the disease. Other information concerning habits and hobbies also was gathered and associated with the disease. Whereas electrocardiograms and X-rays were used as diagnostic tools, the biochemical and physiological factors were termed "risk factors."

The biochemical variable of elevated serum cholesterol was probably the first of its kind to be closely associated with a significant probability of a future coronary in an otherwise healthy person. Numerous hypotheses have been generated concerning the role of hypercholesterolemia and coronary disease, and many

Figure 4. Progressive stages in the generation and progression of chronic degenerative diseases. (Segments A-G represent the populations of figure 3.)



of these ideas were designed to meet a singular causality. Anticoronary groups, on the premise that hypercholesterolemia (reflecting consumption of saturated fats in the diet) espouses coronary disease, have leaped into action to control and prevent such events.

The success of these groups is being gauged by the reduction and maintenance of reduced weight among their otherwise overweight patients, and by lower than initial levels of cholesterol. The parameter of obesity, so far ill defined, has in many epidemiologic studies been directly or indirectly related to hypercholesterolemia and to mortality from atherosclerotic heart disease.

The total community studies initiated in the past two decades, in countries on both sides of the Atlantic Ocean, have provided information that has resulted in defining many risk factors and thus has accelerated that lurking concept of multifactorial causes of coronary disease by centralizing the host. It is now possible to study the effect of combined factors, confounding factors, related factors, and interacting factors. With these concepts one could perhaps separate the diseased from the healthy and possibly further categorize the healthy as a high or a low risk for the coronary disease (D and E, fig. 3).

Present-day knowledge of the interactions of these risk factors, however, is inadequate because it does not include the definitive prognostic potential for individual cases; that is, it lacks precision albeit it contains the issue of probability. The meaning of probability must be unequivocally understood by all those who wish to use this language. It is not possible to state that a probability of 0.05 is an improbability of 0.95. The latter contention has not usually been tested (*3*).

The rationale for prevention is developed on the basis of probability indeed, but prevention is instituted on the premise that if it is predicted but does not accrue at least additional knowledge would have developed. Thus the use of cholesterol-lowering diets and drugs have had little success in preventing coronary disease; and for other risk factors there is valid doubt as to whether intervention and treatment would provide positive results.

Cornfield and Mitchell (*4*) stated: "We would be farther along in our knowledge of the effects achievable by intervention if we had relied less on statistical procedures of doubtful applicability and more on quality and quantity of observation." According to these scientists, the application of current knowledge about coronary disease is inadequate to reduce the incidence of or mortality from coronary heart disease.

What this means is that we should look for additional factors, as yet unknown, in persons of all ages. Perhaps we could then taxonomize them according to sets of risk factors so that changes in these sets would be of prognostic value and could result in prevention, cure, and rehabilitation. These results, in turn, could be translated to the quality of survival rather than to simple survival time.

It is well known that the end results of chronic degenerative processes are a function of time. No one specifically knows when and where they begin; however, long periods of time may elapse before they become clinically evident as a severe health threat. Thus a man in his fifties has four times the risk of a heart attack as a man in his thirties. If prevention of heart attacks, then, is to be sought successfully, research in this area must be oriented to understanding the phenomena that trigger the onset, and these reasons must be found before age as such becomes a risk in itself.

Many times, by many people, it has been said that an old man will die, if not of heart attack or neoplasia, of old age alone. But we must not forget that time has changed the concept of old age. What used to be old age no longer is old age, and what today is regarded as old age may still be considered young if the mass killers of this era, akin to those mastered in the past, can be reduced. This thought reflects the optimism of a young epidemiologist who would like to see the research attitudes of scientists diverted from studying people over 40 years of age (populations A–C, fig. 3) to intensively studying those under age 40 (populations D–G, fig. 3).

Knowledge of physical fitness, in terms of "normality" for the young, is plentiful, and the general attitudes of everyone concerned (relatives, friends, neighbors, physicians, and scientists alike) are unanimous in one respect; that is, young men must be fit. But we have no knowledge about the normality of physical fitness for the middle-aged and the old. We often measure their normalcy or abnormality by the same measure with which we gauge the physical fitness of the young. I must agree that the best normal values are derived individually, and this fact shifts me to my central theme: the host who must volunteer to be observed and tested medically, periodically, in order that the trigger mechanism which generates the coronary disease may be under-

stood. A central depository of these periodic examinations can provide much knowledge, and until such knowledge is available we will not be able to attack the problem of coronary disease in such magnitude as to claim success in its control.

The task at hand, therefore, is to centralize the information and evaluate the tendencies and deviations in young people to which attention may be usefully directed. On the basic foundations of the knowledge at hand, we must proceed toward age zero in such a way that each step, though it may seem backward, will in reality be a forward one, adding to our progress in understanding the disease. We may even be able to get to the root of it all!

Degenerative Processes

Hatch (*5*) has projected these ideas through his impairment disability curve, which I have modified a bit in terms of health and disease versus age in reference to the chronic degenerative processes. Figure 4 represents an epidemiologic structure of what the tentative steps might be in the generation and progression of these diseases. Needless to say, the figure is not without exceptions to the general rule—whatever the general rule is—but it attempts to identify logically the various stages that may follow in precipitating an event on the axis of age.

Thus up to about age 30 the human body has the vigor of the homeostatic faculties operating within it. This is then followed by other compensatory phenomena until about age 40 to 45. But compensation, by its implied meaning, occurs to counterbalance some deviate function, which suggests a latent condition or abnormality not yet recognized either by the host or by a physician, perhaps because of inadequate knowledge about such deviations.

At this very point, distinction is being made between preventive and curative medicine since the host now is a potential victim and can abruptly show signs and symptoms of the disease anywhere from mild to severe. If the disease is recognized as mild, it is often reversible, dictated by individual circumstances and the nature of the deviation. So, too, is the damage accrued by moderate affliction repairable by surgical or other therapeutic regimen, again depending on individual circumstances and the nature of the deviation. Somehow it seems to me at this time, within the discipline of curative medicine, that rehabilitation of the patient can be most meaningful to the health of the host.

Once the disease becomes a severe threat to life and permanent damage of an organ ensues, there is little that can be done except what is being done; namely, organ transplant—again depending on the extent of the damage. The vices and virtues of organ transplants are difficult to understand and assess at this time because knowledge is lacking in this area. Rehabilitation of the disabled likewise should be the premise of an expert, and must consequently fall into the lap of curative medicine rather than preventive medicine. Needless to say, research in these areas is mandatory to achieve optimum recompense and indirectly may add to the predictive and thus preventive aspects of the disease.

Preventive Care

In the past few years more and more emphasis has been placed on physical exercise as a means of rehabilitating the heart patient. The beneficial effects of exercise have been amply substantiated by reports on the training of fit and unfit subjects. The attainment and maintenance of high levels of physical fitness, however, have not been proved conclusively to be effective in preventing the occurrence of coronary disease in "normal" subjects or progression in patients with coronary disease.

With the assets of modern technology and electronics, which have already landed men on what once used to be a celestial body, the moon, we can learn about the total force (biological, physical, chemical, and social) that operates on the basic genetic endowment of the host and generates the chronicity of the diseases of this era. The multifactorial concept in the incipience and progression of these diseases cannot be ignored, but it seems possible that certain weak links may be discovered within the complex and make it possible to prevent the onset of these diseases.

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