

PREFACE TO THE FIRST EDITION

In 1954 I wrote a book entitled *Heart Disease and Industry* (Texon, 1954), which consisted of my clinical experience with 100 consecutive workmen's compensation cases of individuals who claimed that their heart disease was related in a causative sense to industrial conditions or incidents as described. In 78 of the 100 cases the diagnosis included arteriosclerotic heart disease due to coronary atherosclerosis (Texon, 1959). After critical analysis of the pertinent factors and findings in these cases, I concluded that coronary atherosclerosis occurs in the working population in the same manner that it occurs in the entire population.

The compelling conclusions were also drawn that the radix malorum is atherosclerosis which frequently pursues its natural course to produce myocardial infarction as a result of progressive coronary occlusive atherosclerotic disease, and that atherosclerosis is not significantly influenced by external factors such as rest, exertion, emotional stress, or nonpenetrating chest trauma. I recommended that a nonoccupational accident and sickness disability benefits insurance law be applied to heart disease so that all questions of time, place, and causal relation could be eliminated (Burchell, 1966). In 1954, after presenting a paper on heart disease and industry at the Second World Congress of Cardiology and the American Heart Association in Washington, D.C., I was invited by Dr. Paul D. White to become a member of the Committee on the Effect of Strain and Trauma on the Heart and Great Vessels of the American Heart Association, serving as a member of the subcommittee on physiology and pathology. In order to study coronary heart disease firsthand, I became a member of the Department of Forensic Medicine at the New York University Medical Center and an Assistant Medical Examiner of the City of New York, under the direction of Dr. Milton Helpern, the late Professor and Chairman of the Department of Forensic

Medicine and Chief Medical Examiner of the City of New York.

My duties as a Medical Examiner included investigations at the scene of death in the many and varied circumstances that come under the jurisdiction of the Medical Examiner's office. My duties also included the pathological examination of hearts and coronary arteries of individuals of all ages who had died of heart disease as well as those who had died of other causes. I visited the laboratories of Dr. Stanley Durlacher in New Orleans and Dr. James Paterson in London, Ontario to observe their methods of tissue preparation and examination. I spent long sessions in discussions with Dr. Paul D. White, Dr. Howard Sprague, and others on the committee. My studies at the autopsy table included blood vessels in all areas of the circulation—gross observations, microscopic sections, innumerable serial sections, varied histological staining techniques, and photographs of both gross and microscopic specimens. The basic pathological findings in the atherosclerotic lesions were identified as progressing from the earliest intimal thickening to the occlusive plaque, including the variations resulting from fibroblastic proliferation, lipids, intramural hemorrhage, and thrombosis.

In pondering the bewildering mass of clinical and pathological data (Rindfleisch, 1872), I found atherosclerosis in both men and women, in relatively young as well as elderly persons, in hypertensive as well as normotensive persons (Moyer, 1971), and in lean as well as obese individuals. Notwithstanding available studies of the statistical association of atherosclerosis with lipids, diet, age, sex, hypertension (Brest and Moyer, 1974), race, occupation, smoking (Astrup and Kjeldsen, 1974), nutritional status, trace elements, enzyme systems (Zemplenyi, 1974), hormones, and emotional stress, my data indicated that the causal relation of these factors, either singly or in any combination, to atherosclerosis was not thereby proved or demonstrated. A statistical association per se does not constitute scientific proof of a causative mechanism. I became convinced that the primary causative factor or mechanism for atherosclerosis is a common denominator operating in all cases and that it determines the presence as well as the absence of atherosclerosis in all cases and in any given case.

In the fall of 1955, all the parts of the puzzle fell into place when I attempted to explain the localization of the atherosclerotic plaque. I returned to the autopsy specimens and reviewed my data from the standpoint of the laws of fluid mechanics in relation to the forces generated by flood flow, with emphasis on the biologic response of vessels with various geometric vascular configurations and different patterns of blood flow.

The puzzle was solved. I found that atherosclerosis occurs at the segmental zones of diminished lateral pressure produced by the forces generated by the

flowing blood. I accumulated more specimens to demonstrate the lesions at zones of curvature, branching, bifurcation, tapering, and external attachment. In December 1955, after completing the routine autopsy work of the day, I presented my findings to Dr. Helpert and the staff at an informal conference. I emphasized and demonstrated the hydraulic conditions and the basic laws of fluid mechanics that are relevant to the development of atherosclerosis. I was encouraged to continue accumulating more specimens and anatomical proof for the hemodynamic basis of atherosclerosis. In April 1957 I presented the concept in the Ether Dome of the Massachusetts General Hospital at Medical Grand Rounds with an introduction by Dr. White. The first publication (Texon, 1957) appeared in March 1957. In 1958 the research was awarded the Hektoen Silver Medal of the American Medical Association for an exhibit at the annual meeting held in San Francisco.

The relation of the laws of fluid mechanics to human circulation and in particular to the development of atherosclerosis was becoming increasingly apparent. I consulted with Dr. Richard Skalak, James Kip Finch Professor of engineering mechanics and Chairman of the Department of Civil Engineering and Engineering Mechanics at Columbia University. Mathematical analysis of blood flow and computer studies of velocity, wall shear stress were instituted for various patterns of flow. Localized areas of diminished lateral pressure in the theoretical models uniformly correlated with the localization of atherosclerotic lesions found in the human circulatory system.

It occurred to me that if a normally straight vessel were altered to become a curvature with other conditions held constant, additional support for the hemodynamic basis of atherosclerosis could be achieved. I then consulted with Dr. André Cournand and was referred to Dr. Jere R. Lord, Jr., who in turn referred me to Dr. Anthony M. Imparato. The surgical competence of Dr. Imparato led to a series of experiments in which atherosclerotic changes were produced in dogs by surgical alteration of their vascular configurations.

The hemodynamic basis of atherosclerosis has become a subject for reports, papers, chapters, meetings, and symposia both in this country and abroad. This book is the record and product of my research effort in identifying the effect of the laws of fluid mechanics as the primary causative factor in the development of atherosclerosis.

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