

PREFACE TO THE SECOND EDITION

During the last 15 years the need for a second edition of this work has become increasingly necessary for several reasons.

Firstly, a basic understanding of the cause, nature, and natural course of atherosclerosis is not sufficiently recognized or understood by the general public and by many physicians.

Secondly, in spite of numerous epidemiological studies, lipid research studies and clinical trials there is still no unequivocal scientific evidence that reduction of cholesterol levels or any of its fractions by diet or drugs influences the pathological process or lowers the risk of atherosclerotic vascular disease in the total population. Critical analysis of these studies reveals statistical flaws, improperly drawn conclusions, and unwarranted extrapolations. Statistical associations per se do not constitute scientific proof of causal relations, and statistical significance does not always correlate with clinical significance. Questions are answered not by authority but by experiment. One crucial experiment can demolish all statistics and speculation. Yet, the National Institutes of Health, the American Heart Association, the National Cholesterol Education Program, the media, the drug companies, the food industry and others in the medical establishment are “selling” the American people this medical fallacy, claiming to relate diet and cholesterol in a causative sense to coronary heart disease.

Thirdly, ongoing studies of blood flow patterns, vascular geometry, and the endothelium have established the effect of the laws of fluid mechanics—hemodynamics—as the primary causative factor in the localization, inception and progressive development of atherosclerosis.

The time has come to change course and to place the cholesterol-heart disease hypothesis in a holding pattern while more promising directions for atherosclerosis research—hemodynamic, molecular, cellular, immunological, and

heredity—are explored.

We may look forward to the control or modification of blood velocity and other relevant hydraulic conditions that cause atherosclerosis.

We may also look forward to control or modification of the response of the endothelium at the cellular level to the hydraulic factors that cause atherosclerosis. We may then retard the development of atherosclerotic vascular disease and consequently extend the human life span.

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