We owe the present average life span of approximately 72 years to medical and public health advances which have decreased infant and childhood morbidity and mortality. However, longevity has not actually increased in the last century since chronic diseases continue to doggedly plague technologically advanced societies. These diseases include coronary and hypertensive cardiovascular disease, diabetes, obesity and cancer.

Coronary heart disease (CHD) incidence appears to have plateaued and decreased in the last decade. However, CHD remains a current public health problem and hypertension remains the paramount cause of death among blacks. The prevalence of cardiovascular risk factors in western societies also remains impressive. Approximately one third of all adult males have serum cholesterol levels above 260 mg/dl and are exposed to a six-fold increased risk of having a premature myocardial infarction. According to the Ten-State Nutrition Survey, 10–50% of the total population is obese; 10–30% of adolescents are obese; 20% of all Americans are hypertensive. The concept of risk factors as identified by the Framingham investigators may in fact be a major medical and health discovery of the century, as important as the “one germ-one disease” theory of Pasteur. The elucidation of host and environmental factors which interdigitate and interact in a complex “mosaic” manner remains a
most fascinating model of coronary and hypertensive heart disease as a “chronic disease.”

It may in fact appear strange to consider CHD as a chronic disease since its clinical manifestations may be as acute as sudden death. However, the metabolic and subcellular pathogenic progression of this disease may occur from infancy or even intrauterine life. Atherosclerosis appears to have a long incubation period; hence, our justified concern with lifestyle and infant feeding practices.

We postulate a “tri-composite” concept of atherosclerosis. One part relates to the aging process and is an expression of the mortality of man yielding to the inexorable press of time, the result of a universal genetic endowment which sets intracellular biological “time clocks” to the Biblical average life span of “three score and ten.” The second component is also inherited and can be exemplified by the specific genetic penetrance which results in familial types of hyperlipidemia, i.e. hypercholesterolemia and/or hypertriglyceridemia and results in the acceleration of the atherosclerotic process. Approximately 8% of the population exhibits Type IV hyperlipidemia and from 2–4%, Type II hyperlipidemia. Hyperlipoproteinemia can be identified in childhood and has a prevalence rate of about 1 in 200 newborns in U.S.A. populations. The disease in the homozygote state is associated with premature heart disease before the age of 20, while in the heterozygote state is associated with coronary heart disease in the fourth or fifth decade. Diabetes mellitus, another inherited disorder, has a prevalence of from 2–4% in the general population and also constitutes a considerable public health problem.

The third part of our concept of atherosclerosis is potentially more amenable to control and relates to a complex interplay of host and environmental factors. Against a background of the variable genetic penetrance of the familial or acquired hyperlipidemias, the presence of “national dietary hypercholesterolemia” or “universal hyperlipidemia” results when the nutritional environment or national diet pattern is high in calories, saturated fat, dietary cholesterol, oligosaccharides and alcohol. It is this third component which may be primarily responsible for the widespread elevated lipid levels found in middle-aged man.

Most serum lipid disorders which induce atherosclerosis have their roots in childhood. Some are inborn errors of metabolism and others are acquired or associated with various diseases (hypothyroidism, nephrotic syndrome, hepatic disease, etc.). Changes in infant feeding practices such as bottle feeding vs. breast feeding, early introduction of solid foods, infant overfeeding and baby foods high in sodium and oligosaccharides, may be important contributors to cardiovascular risk factors operating silently in infancy and childhood.

In intrauterine life and infancy, cholesterol and lipids are important participants in the cellular structure and function of the central nervous