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Inflammatory Atherosclerosis: characteristics of the injurious agent

The injurious agent (IA) responsible for causing atherosclerosis remains unknown. Atherosclerosis is a complex, chronic inflammatory disease, characterized by a series of highly specific cellular and molecular responses, generally believed to be caused by multiple IA's. Atherosclerotic lesions however are non-specific. There are no pathognomonic histologic features that distinguish one IA from another. If atherosclerosis is caused by multiple agents, then many different agents produce highly specific, cellular and molecular responses. The development of identical or similar lesions in response to multiple different IA's can only be explained if arterial cells respond in the same way, a non-specific way, to all IA's, whether that be hypoxia, chemical agents, infectious agents etc. This view is not only questionable; it is not consistent with the pathogenesis of a highly specific, complex disease. Cardiovascular risk factors are considered to be "accelerators", but not the primary IA itself. What is the source of energy that sustains or replenishes the IA? What evolutionary purpose is served by the retention of cholesterol and other lipids? Is atherosclerosis an auto-immune disease? In the final analysis, atherosclerosis is basically a destructive disease. The aim in this presentation is to characterize the IA involved in the initiation and progression of atherosclerotic plaques that lead to acute coronary disease. Histologic evidence from the coronary arteries of patients who died of acute coronary disease will be presented to support our hypothesis that the IA is a single infectious agent.

Biography

Dr. Frink is the Principal Investigator of the Heart Research Foundation of Sacramento. He received his training at Iowa, Mayo Clinic and Alabama. He practiced invasive cardiology in Sacramento for 35 years and at the same time established a histology photographic laboratory to study the post-mortem heart. He performed extensive post-mortem examinations of the hearts of over 150 of patients who died of acute coronary disease, as well as hearts of pigs and sheep. He has published approximately 25 research papers and a book detailing the pathologic findings in patients who died of acute coronary disease.