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Sudden cardiac death and myocardial ischemia: A reappraisal

Sudden Cardiac Death (SCD) is a major health problem. The SCD patients referred to here are those apparently healthy persons who drop dead suddenly without experiencing any symptoms and are unable to call for help. The basic underlying mechanism causing death is often the sudden onset of ventricular fibrillation (VF), but the precise mechanism responsible for precipitating the VF remains unknown. The prevailing opinion is that the VF is caused by myocardial ischemia because these patients commonly have underlying coronary atherosclerosis. However, there are many reasons to question myocardial ischemia as the precipitating cause of VF, and these will be discussed.

We have performed a comprehensive post mortem examination of a large number of SCD patients as well as many who died of other acute coronary syndromes. Various types of plaques and coronary pathology will be presented to support our hypothesis that the sudden onset of VF is precipitated by plaque toxins released at the time of spontaneous plaque rupture. These toxins circulate downstream to various portions of the conduction system (CS) causing sudden, severe, and direct, but transient injury, to the CS, precipitating VF. These toxins are quickly neutralized and/or washed away by flowing blood, and explain why immediate cardioversion is often successful, why the VF does not immediately recur after successful cardioversion, and why resuscitated patients often do not have or develop subsequent acute myocardial infarction.

Biography

Frink is the principal investigator of the Heart Research Foundation of Sacramento. He received his training at the University of Iowa, the Mayo Clinic and the University of Alabama in Birmingham. He practiced invasive cardiology in Sacramento, California for 35 years and established a laboratory to study the post-mortem heart. He has published approximately 25 research papers and a book, *Inflammatory Atherosclerosis: Characteristics of the Injurious Agent*, detailing the pathologic findings in patients who died of acute coronary disease. The primary focus of his work has been the pathogenesis of atherosclerosis and the mechanism responsible for sudden cardiac death.

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