Cardiopulmonary resuscitation and direct cardiac injury: Evidence of fractured coronary arteries and his bundle hemorrhage

Richard J. Frink
Heart Research Foundation of Sacramento, USA

Background: The heart is believed to escape serious injury during cardiopulmonary resuscitation (CPR). However, no comprehensive study of the coronary arteries or the conduction system has been performed to see if these structures might be injured during CPR. Direct injury to the heart may contribute to the poor prognosis of patients successfully resuscitated from sudden cardiac death.

Materials and Methods: One hundred five human hearts, 83 males and 22 females, were extensively studied at autopsy. Eighty three died of acute coronary disease and 22 of noncoronary causes. Eighty patients, 62 males, mean age 53 and 18 females, mean age 70 received CPR. Sixty patients died in the hospital.

Results: Evidence consistent with direct injury was found in 35 (44%) of patients who received CPR. These structural changes were characterized by fractures of the coronary arteries and or hemorrhage in the region of the HIS bundle. These injuries were not related to the site where CPR was performed, nor to the duration of CPR.

Conclusions: CPR is associated with evidence of direct blunt injury to the coronary arteries and/or the HIS bundle. These lesions may influence the outcome of resuscitation efforts as well as the ultimate prognosis.

Biography

Richard J. 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.

rjfrink@surewest.net