ALCOHOL-INDUCED HEART DAMAGE

J. Fernández-Solà
Alcohol Unit. Hospital Clinic. IDIBAPS. University of Barcelona

The heart is one of the most sensitive organs concerning the harmful effects of alcohol. Ethanol either in acute high-dose, binge or in chronic lifetime cumulated intake is able to produce a diversity of cardiac disturbances. These effects are directly produced by ethanol independently of type of beverage, vitamin, ionic or caloric deficiencies. Acute effects are characterized by induction of arrhythmias, depression of left-ventricle (LV) function and sudden death. Chronic effects induce progressive LV diastolic and systolic dysfunction, hypertension, dilated cardiomyopathy, heart failure and cardiac death. The possible beneficial effects induced by low-dose alcohol consumption in ischemic heart disease disappear at binge or high-dose intake. Currently they are in question since many confounding factors out of alcohol itself may justify this benefit. In addition, at the same level of benefit, they may also induce dependence, cancer or neurologic damage. Mechanisms of alcohol-induced cardiac damage are diverse, including functional and structural changes in myocyte membrane composition, Ca\(^{2+}\) and Na\(^{+}\) channel activities, disturbances in SR and sarcolemmal excitation-contraction coupling, \([i.e. Ca^{2+}]\) transient disruption, decrease in protein synthesis and increased catabolism, energetic and oxidative mitochondrial disturbances, changes in gene-control mechanisms and induction of apoptosis.

In front of this damage, the heart is usually able to establish regulatory and adaptation mechanisms. However, ethanol decreases itself the proliferative and regenerative myocyte capacity and disturbs reparative myokines (FGF, Metrnl), and growth factors (IGF-1, Myostatin) leading to an imbalance to progressive irreversible myocyte damage and death. Considering this overview, less alcohol intake is recommended for better heart health.