A variety of cardiovascular disorders have been associated with chronic alcohol use. These range from heart failure and sudden death to hypertension and stroke. In contrast to alcoholic cirrhosis where clinical and laboratory evidence readily leads to appropriate diagnosis, the relationship of the cardiovascular system to chronic alcohol use is more difficult to identify. The variation of organ involvement is considered to be genetically determined, but nongenetic factors presumably contribute to the process.

Heart failure has been associated with alcohol use since the middle of the last century. However, the causative relationship of alcohol was not clearly defined since it was often complicated by nutritional deficiency. Most characteristic was thiamine deficiency, which produced the high cardiac output state of beri-beri. However, in the last three decades a series of clinical and experimental reports indicate that drinking alcohol is the major determining factor of heart failure in these patients. Moreover, alcoholism is the most frequent identifiable cause of heart disease, ranging up to 32 percent in reports for referral centers. The incidence would be expected to be higher at institutions where there is high frequency of alcohol addiction.

Major factors supporting the role of alcohol in causing heart failure include observations in individuals with clinically normal hearts who demonstrate a relatively high incidence of cardiac abnormalities in testing. The second factor is that abstinence results in a reduction of cardiac abnormalities in those with heart failure and an improved mortality rate when compared with subjects who remain actively alcoholic.

In the patient without symptoms a variety of techniques have been used to show that alcohol use is associated with a reduction in the heart's ability to function. Small but crucial increases of arterial pressure have been used to assess cardiac function in alcoholics with clinically normal hearts. Abnormal elevation of left ventricular pressure has been observed without a corresponding increase in producing stroke. Furthermore, the ability of the left ventricle to contract was found to be significantly reduced in the alcoholic. This systolic function was reduced to a level that was halfway between normal and that for patients with heart failure.

Of particular interest is a study of a group of relatively similar subjects that were employed and living with family. Detailed evaluation of their nutritional state revealed no evidence of malnutrition. Radionuclide ventriculograms showed left ventricular dysfunction in as many as one-third of these chronic alcoholics. Abnormal skeletal muscle performance was present in almost 50 percent of the subjects.

The cardiac status of patients with cirrhosis is of interest since they are generally considered to be resistant to congestive heart disease. Thirty-seven patients with evidence of alcoholic cirrhosis, but no symptoms or signs of cardiac involvement, have been investigated. Two distinct patterns of left ventricular function status were observed. Cardiac output while at rest was reduced in more than 50 percent. Left ventricular functional responses were further depressed during increased activity. The remaining subjects had a high cardiac output at rest which was considered to be secondary to diminished peripheral resistance and not related to a primary hypercontractile state.
Reduced resistance of the vascular system in patients with cirrhosis has been considered to be a potential basis for the infrequent occurrence of congestive heart disease. However, since the majority of alcoholics with cirrhosis have a normal peripheral vascular resistance, such an explanation appears unlikely. Important nutritional deficiencies may occur as liver disease progresses. These may slow down the appearance of some of the components of heart disease.

The presence of irregularity in the rhythm of the heartbeat is a phenomenon associated with mechanical dysfunction that is not detectable by clinical tests. A variety of irregularities in heartbeat have been described in alcoholic subjects without overt heart disease. These have usually occurred during acute intoxication.

In a report of patients with 32 symptomatic irregular heartbeat episodes requiring hospitalization, all drank habitually and had ingested heavily just before the episode. The rhythm was restored spontaneously in some but usually required therapeutic intervention.

In a health maintenance clinic, the prevalence of irregular heartbeat was determined in 1322 persons reporting six or more drinks per day. These were compared to 2644 subjects who reported drinking at least monthly, but less than daily. Relative risk in the former group was at least doubled for several kinds of heart problems.

In view of the observation on the "holiday heart," it is not unexpected that the monitoring of patients admitted to the hospital for alcohol withdrawal revealed a relatively high incidence of irregular heartbeat in addition to excessively rapid heartbeat. However, no evidence of excessively rapid heartbeat was observed despite isolated displaced beats.

Although this may be an accurate estimate of prevalence, a rapid heartbeat in alcoholic patients may progress to irregular heartbeat too rapidly for medical diagnosis. Several reports from medical examiners have indicated a higher incidence of sudden death in subjects who abuse alcohol than previously realized.

A study of sudden death at the Pathology Institute in Moscow revealed that 17 percent of all cases were related to alcohol abuse and these were predominantly in patients less than 50 years of age, without clinical evidence of heart disease. Inclusion in the study required that the event be witnessed and that death occur within 30 minutes of symptoms; thus the question of sleep apnea was not an issue. While significant coronary disease was absent in these patients, evidence of heart disease was present in specimens taken within hours of death. In addition, analysis of heart muscle indicated reduced activity of mitochondrial enzymes, particularly succinic dehydrogenase. This finding was thought to be relatively specific for alcoholic heart disease since it was absent in the variety that had no known cause. No data were provided in terms of the relative purity of the alcohol consumed, since contaminants may contribute to cardiac pathophysiology.

In Sweden, registration with the temperance board for alcohol problems was associated with increased risk of sudden cardiac death. In a study in which 50-year-old men in Uppsala were followed for 10 years, half of those who suddenly died had been registered at the temperance board. Moreover, men who chose not to participate in a primary prevention trial were more often registered for alcohol problems and had a higher incidence of "coronary" deaths. The higher death rate in non-participants was largely accounted for by sudden cardiac deaths.

A five-year Finnish study of 4532 men aged 40-64 revealed a reduced incidence of sudden death in abstainers, regardless of the presence of heart disease, and was true for both smokers and nonsmokers. A case control study of sudden, unexpected death in women showed that 40 percent of cases had a diagnosis of alcoholism, compared to 7 percent of heart patients and 3 percent of a control group.

A small rise of systolic pressure often occurs with social drinking. In subjects who habitually imbibe heavily the pressure rise may be substantial.

A study was undertaken in alcoholics without heart disease to assess the effects of inebriation and the post-intoxication period on the level of arterial pressure. Cardiac function was determined and compared with recovery levels. The hypertension was not related to a high-output state, since peripheral arterial resistance was substantially elevated. High plasma levels of aldosterone and renin, as well as urinary catecholamines, correlated with this response of constriction of the blood...
vessels. A decline of these hormones as blood pressure spontaneously normalized is compatible with this interpretation. However, changes in the intrinsic nerve activity of the arterial smooth muscle or its ionic composition may affect this response.

It is noteworthy that the incidence of chronic hypertension in those who remain abstinent for a year is similar to that in age-matched controls: approximately 10 percent. In contrast, hypertension in the active alcoholic appears less responsive to therapeutic interventions.

Despite the observation that these alcoholic subjects can have a chronic hypertensive response during intoxication, the left ventricular dimensions were normal in the acute hypertensives as well as in the alcoholic group without hypertension. Thus, it would appear that this periodic peripheral arterial response may not be associated with clinically significant heart disease. Although temporary pressure elevations are not considered to give rise to heart disease, if substantial elevations do occur in advanced undetected heart disease, one cannot entirely exclude a potential role for this phenomenon in the development of congestive heart disease in the chronic alcoholic.

At some stages in the development of alcoholic heart disease, the enlargement of the heart may simulate the response to chronic hypertension. Thus, the anatomic changes of the heart in some chronic alcoholics before symptoms appear may include 10 to 20 percent increments of thickness in the wall of the heart. However, fractional shortening of the left ventricle was maintained at normal levels. In uncomplicated essential hypertension, such abnormal enlargement is associated with a reduction in the velocity of circumferential shortening and the ejection fraction.

An inability of the heart to maintain adequate circulation occurs typically in men between 30 and 55 years of age who have drunk at least 80 g of alcohol on most days for a minimum period of ten years. When heart disease progresses to low cardiac output heart failure, lung congestion may lead to difficulties with breathing during exertion or at nighttime. With mild degrees of breathing difficulty, altered lung function must be considered, because impairment has been described that is more than can be attributed to cigarette smoking alone. Weakness and fatigue, presumably resulting from reduced cardiac output, are also common.

Physical signs of the heart's inability to maintain circulation are consistent with other causes of congestive heart disease. Most patients having an element of this inability have an irregular heart rhythm. In others, who do not have congestive heart failure, the finding of an irregular rhythm represents an early clue to the presence of heart disease. When heart failure is long sustained, or after repeated episodes, pulmonary hypertension and right heart failure may become evident. If the patient has not been seen in the earlier stages of the disease, the picture of primary pulmonary hypertension may be mimicked. However, in the patient with left ventricular disease, pulmonary hypertension commonly develops secondary to long-standing, severe left ventricular failure.

Unless there are complications, enlargement of the heart may be moderate in extent. Heart rate may revert to near normal after lung congestion is corrected during the initial episode of the heart's inability to maintain circulation. Obstructions of the lungs and the peripheral arteries are common features of this disease and sometimes represent the initial manifestation. These obstructions may be caused by the enlargement of the heart, congestive failure from any cause, or thrombophlebitis. These clinical events frequently seem to be caused by intensified drinking, but recurrent illness may occur after a period of abstinence in some individuals.

Laboratory experiments suggest important effects that may ultimately affect the study of disease. Thus, alcohol initially interacts primarily with the watery regions of the membrane surface rather than the bilayer. Each mole of alcohol is bound tightly to each mole of protein comprising the Ca-ATPase pump, but the additional interaction with protein are not known. In the sarcoplasmic reticulum, impaired uptake and binding of calcium have been observed in chronic alcoholics one
day after the last exposure to alcohol. When examined two days after alcohol withdrawal, however, the speed of calcium uptake by the isolated sarcoplasmic reticulum and whole-heart homogenates did not differ from control in a rat model fed 23 percent alcohol for 17 weeks. These data suggest a time dependency related to the last period of alcohol ingestion.

A decline in the ability of the heart to contract was observed after only four years of alcohol treatment in a canine model. A shorter term study of approximately two years concluded from analysis of a glycerinated heart muscle preparation that the force-velocity relation (V\text{max}) was significantly reduced. The mechanism of altered contractility in the long-term alcoholic groups is not clear. High energy phosphate levels do not appear to be diminished.

Inhibition of sodium-potassium adenosine triphosphatase (ATPase) has been described in several organs as a result of the long-term feeding of alcohol. However, the steady-state gain of sodium by the heart was not associated with potassium loss in contrast to the situation in which myocardial ATPase is inhibited by digitalis. In addition, accumulation of water contrasts with the typical response to inhibition of this enzyme. Previous observations have indicated an altered fatty acid incorporation as well as composition of phospholipid in animals fed alcohol for a long period. One of the membrane properties limiting permeability of the cell to sodium and water may thus be affected by the phospholipid alteration without affecting the normal transcellular gradient of potassium.

Long-term studies in dogs fed 36 percent of calories as alcohol produced an early decrease in left ventricular flexibility. These animals exhibit collagen increments in the heart muscle that are located in perivascular loci and between myofibers as judged from histochemical studies. Presumably, the accumulation of extracellular fibrous protein was the basis for the diminished diastolic compliance. Thus, collagen deposits in heart muscle occur relatively early during alcohol feeding similar to the liver alterations. Lack of progression in the animals fed alcohol for four years suggested that after early accumulation, the synthesis and removal of this protein are relatively equal. Larger increments may depend on a longer exposure to alcohol, or genetic or dietary factors that have not been delineated.

Early studies had indicated that protein synthesis in the cardiac cell as measured by lysine incorporation in the isolated perfused rat heart was significantly reduced by acetaldehyde, while alcohol had no direct effect. However, in experiments to determine the ability of alcohol to affect the process of enlargement of the heart muscle during chronic aortic pressure of overload in the same species, the enlargement response was unimpaired in the experimental animals. Moreover, ribonucleic acid, a determinant of protein synthetic rate, was not found to be diminished in the alcoholic animals over a period of 14 weeks. Data of the activity of lysosomal enzymes in this experimental study of heart muscle are not yet available.

Over the past decade there have been several studies which have suggested that low to moderate alcohol intake reduces the risk of coronary heart disease. However, factors other than the moderate use of alcohol may be involved. Patients in this group tend to have lower blood pressure and body weight. Furthermore, they appear to have a lower intake of total saturated fat and a higher intake of polyunsaturated fat. A persistent flaw has been the composition of the control group. It is believed that there are sufficient ex-drinkers in this group to compromise its validity, since they share a number of characteristics that increase their cardiovascular risk. These include a higher prevalence of cigarette smoking, obesity, and hypertension compared with those who were never drinkers. Moreover, they are likely to be unmarried and to work in manual occupations. In the final analysis, if modest alcohol ingestion proves to have a preventive effect, its usefulness as a general public health measure would be limited by potential addictive consequences and the availability of other interventions that do not have the potential for serious health consequences.

References


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