The Heart and Alcohol

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The pathological effects of chronic alcoholism are usually considered primarily to be the result of associated nutritional deficiencies. Peripheral neuritis, pellagra, central nervous system disturbances and cirrhosis of the liver are the most well recognized complications in chronic alcoholics. The cardiovascular effects of both acute and chronic alcoholic intake are often overlooked except for the well known entity of beriberi heart disease. The rate of oxidation of alcohol is directly proportional to the integrity of hepatic function as well as being intimately related to the speed of absorption from the gastrointestinal tract. In persons with impaired liver function the toxic effects of alcohol are increased. This is an important factor in the production of degenerative changes in several other organs, including the heart.

When moderate amounts of alcohol are consumed there is a quickening of the pulse rate; the rate then becomes slower as the dosage is increased, owing to a depressant effect in the vasoconstrictor centers which causes a lowering of the blood pressure. In addition a vagal and a direct depressant effect on the heart muscle occurs; anoxia induced by toxic effects on the respiratory center is also involved. Visceral vasoconstriction with peripheral vasodilatation occurs with moderate dosage and the net increase in peripheral resistance is demonstrable as a rise in mean arterial blood pressure. Because the systolic pressure elevation exceeds that of the diastolic, the stroke volume output is increased. Pulmonary arterial pressure and blood carbon dioxide tension increase in conjunction with a decrease in pulmonary aeration. The clinical cardiac manifestations of acute alcoholic intake in relation to progressively rising blood concentrations are: (1) Tachycardia and systemic arterial hypertension; (2) Cardiac arrhythmias; and (3) Anoxia with bradycardia, hypotension and ultimate asystole which is caused by asphyxia rather than a direct myocardial effect.

Prolonged alcoholism brings about various cardiac abnormalities which are related not only to the amount and duration of the ingestion but also to the associated nutritional habits and hepatic status. Three well defined types of heart disease in chronic alcoholics have been observed:

1. Alcoholic myocarditis, which is unrelated to vitamin deficiency or liver disease and is characterized by electrocardiographic changes giving evidence of abnormal left ventricular repolarization simulating a so-called "digitalis effect" and/or evidence of left ventricular enlargement. These changes were present in 57.4 per cent of a group of 94 chronic alcoholic persons whose average age was 42 years and who had no other manifestations of heart disease as observed by clinical or laboratory diagnostic methods. These abnormalities were not present in electrocardiograms made several days after alcohol intake was discontinued.

2. The abnormal electrocardiographic patterns were considered to be the result of the cumulative effects of ethanol on cardiac muscle, inasmuch as acute alcoholism is not associated with these changes. Awareness of these electrocardiographic concomitants can be of considerable importance in the diagnosis of otherwise occult chronic alcoholism. Likewise, recognizing the relationship will obviate the misinterpretation of such tracings as being caused by digitalis, left ventricular hypertrophy or both.

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2. Nutritional heart disease which is associated with malnutrition and progressive liver disease was described in 1950 by Suzman and later by Gillanders following a study of Bantu natives in South Africa. Dyspnea, anasarca and peripheral neuritis in conjunction with high venous pressure, low pulse pressure, cardiac enlargement, hepatic engorgement and gallop rhythm were noted clinically in these patients. Circulation time was prolonged, kidney function was normal and x-ray films of the chest showed pulmonary venous engorgement and globular shaped cardiac enlargement. Decreased pulsation was observed fluoroscopically. Electrocardiograms showed left ventricular enlargement in 70 per cent of patients, right ventricular hypertrophy in 5 per cent and right bundle branch block in 10 per cent; in the remainder there were no predominating patterns.

When adequate diets were given, the cardiac manifestations subsided usually within one month in the less severe cases. In the seriously affected patients the condition progressively worsened and resulted in death. Cardiac glycosides were of no value in treatment in spite of the apparent low output type of cardiac failure. Biopsy of specimens of liver showed extensive fatty and pigmentary cirrhosis in all patients regardless of the outcome of the cardiac lesions. In the cases of death from cardiac insufficiency there was demonstrable hypertrophy of myocardial muscle fibers and interstitial fibrosis without hydropic degeneration.

3. Beriberi heart disease which has been well known since Wenckebach's description in 1934 is the most severe form of cardiac involvement that results from chronic alcoholism. It is characterized by anasarca, generalized cardiac enlargement, high-output failure with increased pulse pressure, dilatation of peripheral arterioles, tachycardia, rapid circulation time, low arteriovenous oxygen difference, enlargement of the pulmonary artery and electrocardiograms showing low voltage. Circulatory collapse and sudden death are common and the prognosis is generally poor in the alcoholic type in spite of excellent care and presumably specific therapy.

The possibility that these three entities represent only different degrees of severity of a single pathologic process has been considered. However, the diagnostic aspects and prognostic implications are sufficiently distinctive to warrant their segregation for clinical purposes.

The therapeutic uses of alcohol are primarily related to the cerebral effects of the drug, in which dosage plays the most important part. Beginning with small quantities the responses are, progressively, sedation, hypnosis, analgesia and ultimately anesthesia. The beneficial effects of alcohol in syncope are attributable to the pharyngeal and esophageal irritative reactions resulting in reflex stimulation of breathing and production of tachycardia through local action, and are unrelated to absorption. In the treatment of acute pulmonary edema 50 per cent ethanol by inhalation has proved an effective "wetting agent," usually used in conjunction with oxygen by mask or nasal catheter. The tonic effects of alcohol are based upon the response of increased gastric secretion and motility plus the relaxing and fatigue-suppressing cerebral effects. In addition the caloric value, approximately 100 calories per ounce of whiskey, is to be considered.

As a vasodilator ethyl alcohol has been reputed to be of value in coronary artery disease since 1772 when Heberden suggested its use in angina pectoris. Convincing experimental proof that the drug has any visceral vasodilating effects is lacking. Beneficial peripheral vascular responses have been repeatedly demonstrated, but not an effect upon cerebral or coronary arterial blood flow. Low to moderate concentrations of alcohol in the blood have been shown to produce no change in either cerebral blood flow or oxygen consumption and it is for this reason that any beneficial therapeutic effects of the drug in cerebrovascular disease must be considered more psychic than physiologic. When

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### TABLE 1.—Physiologic Effects of Moderate Alcohol Intake

| 1. | Increase in pulse rate. |
| 2. | Increase in mean blood pressure (systolic more than diastolic). |
| 3. | Peripheral vasodilatation with visceral vasconstriction (net increase in peripheral resistance). |
| 4. | Increase in stroke volume output. |
| 5. | Increase in pulmonary arterial pressure. |
| 6. | Decrease in pulmonary aeration with rise of blood carbon dioxide tension. |

### TABLE 2.—Effects of Excessive Acute Intake of Alcohol

| 1. | Bradycardia (vagal and direct cardiac depressing effect). |
| 2. | Depression of vasoconstrictor center with hypotension. |
| 3. | Depression of respiratory center, anoxia and secondary asystole. |

Excessive chronic intake produces:

1. Primary myocardial fatty infiltration and degeneration with subsequent dilatation.
2. Secondary manifestations of nutritional alterations (hepatic, edema and beriberi).

### TABLE 3.—Clinical Cardiac Manifestations of Alcoholism

**Acute:**

1. Tachycardia and increased blood pressure.
2. Cardiac arrhythmias.
3. Anoxia, bradycardia and hypotension.

**Chronic:**

1. Without vitamin deficiency (alcoholic myocardosis).
2. With hepatic insufficiency (nutritional heart disease).
high blood levels are attained, there is an increase in cerebral blood flow with significant deleterious fall in oxygen consumption. It has been suggested that similar phenomena occur in the myocardium in regard to both coronary blood flow and oxygen utilization.

In patients having coronary artery disease the moderate use of alcohol as a relaxant because of its cerebral effects is acceptable. Excessive dosage leading to euphoria with the probability of prolonged or strenuous physical stresses such as dancing or demonstration of feats of great strength should be avoided.

As a prophylactic agent for arteriosclerosis there is no valid evidence that alcohol is effective. Contrariwise it has not been shown to induce or hasten the process of atherosclerosis.  

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REFERENCES