Alcohol and cardiovascular disease

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Epidemiological evidence which has accumulated over the past three decades indicates that the 20th-century epidemic of coronary disease is to a great extent a product of a faulty life-style promoted by the industrialization and urbanization of modern society (Report of Intersociety Commission for Heart Disease Resources, 1984). This atherogenic life-style is characterized by a diet excessive in saturated fat, cholesterol, energy and salt; unrestrained weight gain, a sedentary existence, smoking cigarettes and, perhaps, stress. Because life-style plays such a large part in the development of atherosclerotic cardiovascular disease, the potential victim must accept responsibility for his own preventive maintenance under the guidance of his physician. The public health official must also contribute by providing the necessary health education and taking measures to alter the ecology to one more favourable to cardiovascular health.

There is evidence that the atherogenic life-style promotes traits such as hypertension, dyslipidaemia, increased fibrinogen and glucose intolerance. There is also some evidence that alcohol intake affects blood lipids, blood pressure, uric acid, thrombogenesis, and coronary heart disease (CHD) and stroke rates.

Heavy intake of alcohol is a leading cause of preventable mortality, second only to cigarette smoking in industrialized countries (Oulett *et al.* 1979). Alcoholism in the USA is far too common, estimated at 5–17 million people (Hennekens, 1983). Alcohol is implicated in more than 40% of all fatal traffic accidents, in 25% of all general hospital admissions, in liver and upper gastrointestinal cancers, suicides, sex crimes, industrial accidents, robbery and murder, and fetal alcohol syndrome. In the context of promotion of disease or death there is evidence to suggest that moderate alcohol intake reduces risk of CHD, the leading cause of death in most affluent societies. It therefore is important to establish the amounts of alcohol which confer benefit and harm and the effect of alcohol on specific cardiovascular disease outcomes.

CHD morbidity and mortality

The influence of alcohol on development of CHD is complex and may be beneficial or harmful depending on the amount of alcohol consumed. There are now some seven studies of CHD mortality which consistently indicate that intakes of large amounts of alcohol are associated with an increased death rate (Hennekens, 1983). In each of these studies of alcoholics or 'problem drinkers', mortality risk ratios exceeding 1·0 have been reported, ranging from 30 to 260% (see Table 1). Thus, heavy drinking confers a substantial health disadvantage for CHD as well as other illnesses.

In sharp contrast, a variety of studies of alcohol consumption in relation to development of CHD have strongly suggested a benefit. These population studies in which light to moderate intakes predominate have shown fairly consistent results. Studies correlating CHD mortality with per capita alcohol consumption around the world have consistently shown an inverse relation (Brummer, 1969; Hennekens, 1983)(Fig. 1). Case-control experimental and prospective studies have tested the hypothesis generated by these international comparisons. Some five case-control studies consistently showed an inverse relation with substantially reduced fatal and non-fatal CHD events in light to

Table 1. *Studies of alcoholics and problem drinkers in relation to mortality from coronary heart disease in men (From Hennekens, 1983)*

Study	Location	Study population	Relative risk, drinkers:non-drinkers
Sunby (1967)	Norway	Alcoholics	1·3
Schmidt & Delint (1972)	Canada	Alcoholics	1·7
Thorarinsson (1979)	Iceland	Alcoholics	1·9
Robinette *et al.* (1979)	United States	Alcoholics	1·2
Davies (1965)	United States	Problem drinkers	2·6
Pell & D'Alonzo (1973)	United States	Problem drinkers	1·9
Dyer *et al.* (1977)	United States	Problem drinkers	3·6

moderate drinkers (Klatsky *et al.* 1974; Stason *et al.* 1976; Rosenberg *et al.* 1981; Hennekens *et al.* 1978) (Table 2). A recent study indicates that this applies to women as well as men (Rosenberg *et al.* 1981). With few exceptions, prospective cohort studies have also indicated an inverse association between development of CHD and light to moderate drinking (Table 3).

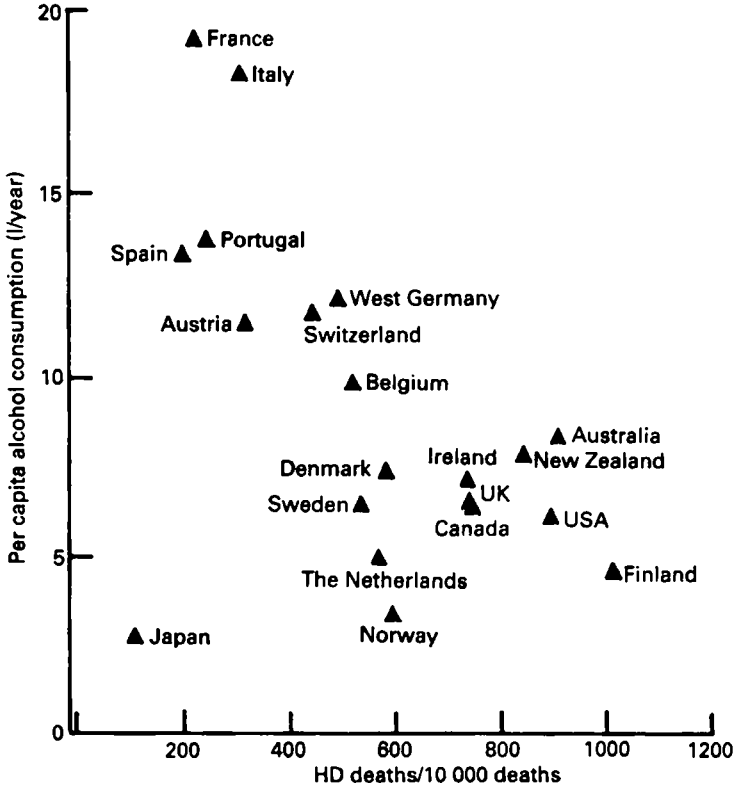

Fig. 1. Relation of per capita alcohol consumption with 1972 heart disease (HD) death rates in men aged 55–64 years in twenty countries. (From La Porte *et al.*1980.)

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Table 2. *Case-control studies of alcohol consumption and coronary heart disease (From Hennekens, 1983)*

Study	Size of population	Level of consumption	Relative risk, drinkers:non-drinkers
Klatsky *et al.* (1974)	661 Cases*	≤2 drinks/d	0·7
	661 Controls	3–5 drinks/d	0·7
		6+ drinks/d	0·4
Stason *et al.* (1976)	339 Cases†	<6 drinks/d	1·0
	2486 Controls	6+ drinks/d	0·6
Hennekens *et al.* (1978)	568 Cases‡	25 g alcohol/d	0·4
		50 g alcohol/d	0·7
Petitti *et al.* (1979)	Not given†	Any	0·3
Rosenberg *et al.* (1981)	513 Cases†	Any	0·7
	918 Controls		

*Both fatal and non-fatal.
†Non-fatal.
‡Fatal.

Table 3. *Cohort studies of alcohol intake and incidence of coronary heart disease (From Hennekens, 1983)*

Study	Sample size	Trend by intake	Comments
Stason *et al.* (1976)	4265	Inverse	–
Yano *et al.* (1977)	7591	Inverse	Japanese–American men
Kozararevic *et al.* (1980)	11121	Inverse	Yugoslavians
Dyer *et al.* (1980)	1872	U-shaped	–
Marmot *et al.* (1981)	1422	Inverse	–
Klatsky *et al.* (1981)	8060	U-shaped	For smokers only
Cullen *et al.* (1982)	2209	Inverse	For non-smokers only
Kozararevic *et al.* (1982)	11121	Inverse	For urban not rural
Gordon & Kannel (1983)	5127	Inverse	Not for male smokers
Kittner *et al.* (1983)	9150	Inverse	For high income
Friedman & Kimball (1986)	4745	Inverse	Not in light smokers

Experimental investigations in non-human primates fed on a high-cholesterol diet confirm the epidemiological findings. Monkeys given alcohol developed stenosis in only 8% of their arteries compared with 48% in those assigned to a placebo (Hennekens, 1983).

Small amounts of alcohol weekly are beneficial only if they are spread out over the week. Binge drinking of the entire week's allocation is harmful. The degree of coronary artery occlusive disease seen on coronary angiography is greater in binge drinkers for the amount of alcohol consumed (Gruchow *et al.* 1982). Hence frequent small amounts seem more protective than the same amount taken in one dose.

The information on the influence of the type of alcoholic beverage on CHD occurrence is inconsistent. A French study (St Leger, 1979) found only wine to be protective; a study of Hawaiian Japanese (Yano *et al.* 1977) indicated that only beer protects whereas a study by Hennekens (1983) in the USA found comparable effects for beer, wine and spirits. Taken together it seems likely that all forms of alcoholic beverages are protective if used daily in small amounts.

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Autopsy and angiographic studies have also indicated anti-atherogenic benefits of alcohol. Some autopsy studies have found no relation between alcohol consumption and extent of atherosclerosis (Sackett *et al.* 1968; Hennekens, 1983). Other work suggests an inverse relation between severity of atherosclerosis and alcohol intake (Lifsic, 1976; Rhoads *et al.* 1978; Hennekens, 1983). Arteriographic studies of the living were consistent with these later autopsy studies; both showed a significant inverse relation between alcohol intake and extent of coronary atherosclerosis, involving 2500 people (Anderson *et al.* 1978; Hennekens, 1983). These inverse relations persisted even on adjusting for cigarette smoking, blood lipids and hypertension.

Causality

The consistency of reports from international comparisons, case-control studies, prospective cohort studies and animal experiments, indicating an inverse relation of moderate alcohol intake to CHD, in studies using a wide range of definitions of exposure makes a causal relation likely (Table 3). However, there has been insufficient control for other risk factors, apart from cigarette smoking, that might confound the relation. In particular obesity, exercise and type A behaviour may be responsible. Also, the effect may be mediated by high-density-lipoprotein (HDL)-cholesterol.

A causal relation is more likely if strong and dose-related. Relative mortality risks of only 30–40% of those in non-drinkers indicate a strong effect. Also the protective effect seems dose-related, until heavy intake is reached. A causal relation is more likely if consistent and here too findings are impressively consistent despite different study designs, different cultural and geographic settings and whether prospective or retrospective. There are also plausible biological mechanisms whereby benefit could be conferred.

Anti-atherogenic mechanisms

A number of studies have indicated that alcohol raises HDL-cholesterol, a protective factor against CHD (Belfrage, 1977; Castelli *et al.* 1977; Hulley *et al.* 1977; Hennekens, 1983). However, there is some disagreement as to the relevance of this as the HDL fraction raised is HDL_3 which may not be the protective one. However, there is a dispute as to whether or not HDL_2, the protective factor, is also raised by alcohol (Haffner *et al.* 1982).

There is little question that alcohol raises blood pressure, which should accelerate atherogenesis. However, although heavy drinking certainly raises blood pressure (Dyer *et al.* 1977; Wallace *et al.* 1981), drinkers of moderate amounts have been reported to have lower average blood pressures than abstainers (Dyer *et al.* 1977). These findings are consistent with an adverse effect of heavy drinking and a protective effect of light drinking on CHD incidence. This is also consistent with higher risks of stroke found in heavy drinkers (Ashley & Rankin, 1979; Hennekens, 1983).

Alcohol may also protect against CHD by inhibiting platelet aggregation and potentiating the effect of aspirin (Ashley & Rankin, 1979). However, the influence of small amounts of alcohol on thrombogenesis is not well established (Hennekens, 1983). It has been reported that moderate drinkers eat less major atherogenic nutrients than do light or heavy drinkers which may account for some of the protection (Jones *et al.* 1982).

Quadratic relations

A number of case-control comparisons and prospective epidemiological studies suggest that moderate alcohol consumption may be associated with lower morbidity and mortality than either abstention or heavy drinking (Camacho *et al.* 1987). Discovery of this U-shaped (or J-shaped) association in which moderate drinkers are at lowest risk,

Table 4. *Coronary heart disease mortality by alcohol consumption; 24 year follow-up, Framingham study (From Wolf* et al. *1983)*

Age-adjusted percentage mortality

Men			Women		
Alcohol intake (g/week)	Non-smokers	Heavy smokers	Alcohol intake (g/week)	Non-smokers	Heavy smokers
None	16·3	28·3	None	5·5	6·3
25	14·8	16·0	25	5·4	3·7
50–75	14·6	14·4	50–75	1·9	3·0
100–175	7·8	14·0	100–225	6·5	4·6
200–500	5·7	13·1	250+	5·9	5·0
550+	7·4	12·5			

especially from CHD, has led to the proposal that moderate amounts of alcohol may be protective (Camacho *et al.* 1987). Review of the epidemiological findings from various sources suggests a quadratic relation of alcohol intake to cardiovascular morbidity and mortality, with a possible excess of mortality at both extremes. This holds more strongly for overall (all cause) mortality than for CHD mortality (Marmot *et al.* 1981; Camacho *et al.* 1987). Studies of CHD mortality in the Framingham cohort appear to show an inverse relation in men and a possible quadratic relation in women (Table 4). Camacho *et al.* (1987) examined this proposition in the Alemeda County study, comparing abstainers and light or moderate drinkers. They concluded that neither abstainers nor other drinkers were at a significantly higher risk of death from CHD than were light drinkers after adjustment for eleven psychosocial covariates of alcohol consumption in addition to age.

Quitting

Information on quitting has generally shown ex-drinkers to be at very high risk of mortality. Because ex-drinkers may stop because of declining health, their assignment to the abstainer category might account for the suggestion of excess risk in this group, compared with light drinkers. Findings from Camacho *et al.* (1987), looking at quitters and adjusting for age and alcohol covariates, showed no statistically significant differences, but in men there was a lower all-cause and CHD mortality in quitters than stable abstainers. This suggests a sustained benefit of quitting and eliminates confounding because of ill-health. However, in women there was a suggestion of excess morbidity particularly for CHD mortality (Camacho *et al.* 1987).

Alcohol and blood lipids

CHD incidence is strongly related to serum total cholesterol, the incidence rising over a fivefold range within the limits of usual cholesterol values (Report of Intersociety Commission for Heart Disease Resources, 1984). This serum total cholesterol, however, reflects a two-way traffic of lipid being transported into the arterial intima in the low-density-lipoprotein (LDL) fraction and being removed via the HDL fraction (Kannel, 1983).

Alcohol affects these and other blood lipids, at least acutely and probably chronically. Experiments have shown that mean levels of triglyceride, total HDL-cholesterol, HDL_2 and HDL_3 cholesterol and apolipoproteins A-I and A-II are higher during alcohol intake

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(Masarei *et al.* 1986). There appears to be an effect of alcohol on both major subfractions of HDL and its major apolipoproteins. Epidemiological studies of young men have shown a substantial impact of alcohol on HDL-cholesterol along with physical activity and smoking (Salonen *et al.* 1985).

The effect of alcohol on the HDL_2 and HDL_3 subfraction is uncertain. Alcohol consumption correlates with both reduced CHD and increased HDL-cholesterol. However, the relation of moderate alcohol intake which protects against CHD and the HDL_2 and HDL_3 fractions is currently obscure. Haskell *et al.* (1984) found an increase in HDL-cholesterol and HDL_3 mass, but not HDL_2 on resumption of moderate drinking. These findings suggest that the association of alcohol with lower CHD rate may not be mediated by HDL_2 and that HDL_3 may not be inert in coronary atherogenesis. It is also possible that other mechanisms unrelated to HDL may be responsible for the protective effect of alcohol.

Cross-sectional descriptive studies of several population samples indicate a strong and consistent dose–response pattern relating HDL-cholesterol to alcohol intake (Hennekens, 1983). Social drinkers have 33% higher HDL-cholesterol levels than abstainers. Cross-sectional multivariate analysis has indicated the HDL–alcohol association to be independent of associated smoking and body-weight and longitudinal analysis indicates that it is also not a consequence of other confounding factors (Hulley & Gordon, 1981). Glueck *et al.* (1981) found in children that HDL-cholesterol was positively related to alcohol intake, independent of other variables, and that HDL-cholesterol was 0·194 mg/l and 0·367 mg/l higher for every gram of alcohol intake in males and females respectively. In women, low-density-lipoprotein (LDL), cholesterol, triglycerides and very-low-density-lipoprotein (VLDL)-cholesterol were all positively related to alcohol intake.

The chief and most pronounced effect of alcohol on plasma lipids is on triglycerides in association with higher concentrations of VLDL. Elevations of VLDL, both acutely and chronically, have been noted in persons imbibing either moderately or heavily. Those who have a type 4 plasma lipoprotein pattern are especially susceptible to alcohol-induced hypertriglyceridaemia (Mendelson & Mello, 1973). Triglyceride has not been shown to make an independent contribution to risk of CHD, but it is positively related to CHD incidence and is involved in the intermediary metabolism of HDL and LDL.

The mechanism whereby ethanol ingestion raises HDL and VLDL is poorly understood. It decreases oxidation of fatty acids leading to release of VLDL consequent on increased hepatic free fatty acid esterification. Also, intestinal synthesis of both triglycerides and VLDL is increased by alcohol, and HDL, also produced by intestinal cells, may be likewise affected. There is also a possibility that HDL is raised as a toxic effect of alcohol, much like that produced by exposure to chlorinated insecticides (Carlson & Kolmodin-Hedman, 1972).

Alcohol and blood pressure

Habitual alcohol consumption is associated with hypertension. Several studies suggest a threshold of alcohol intake above which blood pressure elevations occur (Fig. 2). Klatsky (1982) found that men and women of all races taking three or more drinks per day had significant elevations of systolic and diastolic blood pressure. Other studies have corroborated this finding. Increased plasma cortisol, renin, aldosterone, arginine, vasopressin and adrenergic activity after alcohol ingestion may explain the alcohol-induced blood pressure rise. Since the blood pressure usually returns to normal when the alcohol intake is stopped, a direct causal relation seems likely.

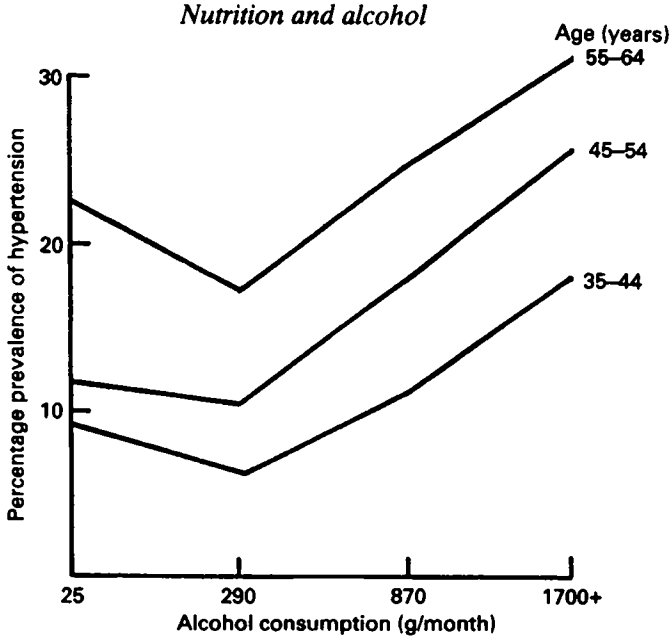

Fig. 2. Percentage prevalence of hypertension in men, by alcohol consumption, Framingham study. (From Wolf *et al.* 1983.)

Although in most studies regular intakes of small amounts of alcohol are not associated with hypertension, a precise threshold has not been defined. However, the alcohol–blood pressure relation does not appear to be attributable to obesity, salt use, coffee, smoking or demographics.

The positive association between alcohol and blood pressure appears to be quadratic, with those abstaining and those on heavy intakes having higher pressures than those on low intakes (Fig. 2). The effects on blood pressure may be more pronounced in those sensitive to hypertension (Malhotra *et al.* 1985). Recent evidence suggests that the effect of alcohol is direct and not due to alcohol withdrawal (Potter & Beevers, 1984*a*,*b*). Alcohol shows a positive association with the stroke sequela of hypertension, but not with CHD, where a protective effect is generally noted.

Alcoholic arrhythmia

Alcohol may increase myocardial irritability. However, although the 'holiday heart syndrome' characterized by rhythm disturbances after acute alcohol ingestion is well known, the arrhythmogenesis of alcohol is not well established. It has been shown that alcohol in modest doses can produce rhythm disturbances such as atrial or ventricular tachyarrhythmias, in patients with a history of chronic alcohol consumption and heart disease (Klatsky, 1982). Vulnerability to atrial fibrillation has also been examined in alcohol abusers without cardiomyopathy or manifest cardiac failure (Engel & Luck, 1983). Possible mechanisms include heightened or non-uniform sympathetic discharge, either peripherally or centrally, related to the euphoria of being drunk (Luck, 1983).

Alcohol and stroke

Heavy alcohol ingestion is associated with a number of cardiovascular effects which enhance risk of a stroke. These include arrhythmia, impaired ventricular function, increased blood pressure, coagulation and platelet disorders among others (Gorelick, 1987). Habitual alcohol consumption is also associated with reduced regional cerebral

blood flow (Gorelick, 1987) especially when other stroke risk-factors are present. This decrease in flow is usually attributed to the toxic effects of chronic and excessive alcohol intake on cerebral metabolism and not to enhanced atherogenesis. Alcohol has a direct vasospastic effect which can be inhibited by calcium antagonists (Gorelick, 1987).

Autopsy studies have noted cerebral infarction more commonly and at an earlier age in alcoholics (Walbran *et al.* 1981). In a controlled study of normotensive patients under 50 years of age, Lee (1979) found a history of excessive alcohol intake more frequently in patients with thrombotic strokes.

Prospective epidemiological studies have generally linked alcohol intake to the occurrence of stroke, but are inconsistent with respect to whether this increased risk is confined to haemorrhagic stroke and whether it is largely mediated through inducement of hypertension. Regular alcohol intake has been associated with fatal and non-fatal intracerebral haemorrhage, cerebral infarction and increased stroke mortality (Gorelick, 1987). Alcohol may also be a risk factor for subarachnoid haemorrhage (St Leger, 1979). Some evidence suggests that acute intoxication may occasionally precipitate a cerebral infarction in young and middle-aged men and women.

The Honolulu Heart Study showed that alcohol intake increased the risk of both fatal and non-fatal intracranial haemorrhage but not thrombo-embolic strokes (Kagan *et al.* 1981). The Kaiser–Permanente Study found that heavy drinking may be related to haemorrhagic stroke and that it is mediated through elevated blood pressure (Klatsky, 1982). On the other hand, abstenance from alcohol was associated with a higher risk of occlusive strokes suggesting a protective effect. The Framingham study findings suggest an association between alcohol intake and occurrence of stroke in general and atherothrombotic brain infarction in particular, but only in men (Wolf *et al.* 1983).

In the Yugoslavian cardiovascular disease study, alcohol was linked to stroke mortality (Kozararevic *et al.* 1980). In Finland, brain infarction in the young has been linked to alcoholic intoxication (Gorelick, 1987). Drinking within 24 h of hospitalization was found more commonly in young stroke patients (under 50 years of age) than in matched controls (Gorelick, 1987). This suggests that acute alcohol ingestion may be a stroke risk-factor.

Alcoholic cardiomyopathy

There is a great deal of evidence that there is a direct toxic effect of alcohol or one of its metabolites on the myocardium (Klatsky, 1982). Alcoholic cardiomyopathy is an accepted clinical condition, but it is based largely on circumstantial evidence (Anderson *et al.* 1978). A large proportion of patients found to have unexplained heart disease have been noted to use large amounts of alcohol (Walbran *et al.* 1981). A few well-documented case reports exist and more frequent regression of clinical abnormalities has been noted in abstainers compared with those who continue to drink. Skeletal muscle damage, both acute and chronic, has been noted with alcohol abuse (Walbran *et al.* 1981), which resembles the cardiac abnormalities seen in cardiomyopathy (Salonen *et al.* 1985). Autopsy studies have shown abnormalities in a large proportion of alcoholics with no other clinical evidence of heart disease.

There is increasing evidence that alcohol can cause serious cardiac metabolic problems, so that ethanol abuse is likely to be an important contributing factor for cardiac morbidity and mortality. Unfortunately, a direct causal relation between the biochemical dysfunctions produced by ethanol and the alcoholic cardiomyopathies has not been elucidated. The alcohol-induced myocardial damage cannot be clearly distinguished from that produced by other causes of dilated cardiomyopathy. Further long-term studies are needed to assess the benefits of abstinence from alcohol on sudden

death rates and occurrence and progression of alcoholic cardiomyopathy. Ethanol may be an underestimated cause of non-coronary cardiac disease. Echocardiographic studies indicate that chronic alcohol abuse is an important independent risk factor for cardiac dilatation which may be an early marker for alcoholic cardiomyopathy. There is evidence that metabolic injury induced by alcohol occurs involving mitochondria, contractile proteins and Ca fluxes and storage (Van Theil *et al.* 1985).

Cardiac arrhythmias occurring during weekend or holiday drinking sprees are sometimes symptoms of cardiomyopathy. Early stages of alcoholic cardiomyopathy may be reversible but cardiac failure and arrhythmias occur with continued alcohol consumption (Demakis *et al.* 1974). Necropsy findings in alcoholic cardiomyopathy include ventricular hypertrophy and dilatation, mural thrombi and even myocardial infarction without significant coronary disease (Regan *et al.* 1975).

Other cardiovascular conditions

There is little evidence to suggest a relation between alcohol and aortic and peripheral arterial atherosclerosis (Klatsky, 1982). It has been suggested that alcohol can induce vasospastic angina but this does not appear to be a common occurrence (Klatsky, 1982). Myocardial infarction without evidence of atherosclerotic or thrombotic occlusion, possibly as a consequence of scarring associated with cardiomyopathy, has been reported in alcoholics (Regan *et al.* 1975). A higher incidence of venous phlebitis and varicose veins has been reported in association with substantial alcohol use (Klatsky, 1982). Heavy maternal drinking has been reported to be associated with congenital cardiac anomalies such as septal defects and patent ductus arteriosis in offspring (Klatsky, 1982).

Unresolved issues

More information is needed to ascertain whether alcohol is protective against CHD. Other related risk factors must be taken into account and interactions looked for. A more accurate determination of the levels of consumption most beneficial and most harmful is needed. We also need a more accurate assessment of the very long- and short-term effects. More information is needed on non-cardiac detrimental effects of moderate alcohol consumption, particularly mortality from stroke, cancer and accidents (Kozararevic *et al.* 1980; Marmot *et al.* 1981; Klatsky, 1982; Taylor & Coombs-Orme, 1982). The effects of alcohol in moderation and in excess on haemorrhagic stroke seem established but effects on atherothrombotic brain infarction are unclear. The entity of alcoholic cardiomyopathy needs to be better defined and the dose relation better established. More information is needed on the cardiovascular effects of alcohol in women.

The possibility that small amounts of alcohol protect against hypertension needs further investigation and explanation.

Preventive implications

There are clearly a number of disease-promoting life-styles which need to be avoided or corrected if we are to further curb the spread of cardiovascular disease. Public health officials would do well to promote the needed changes in the national diet, to promote physical activity as part of daily living and to curb cigarette smoking. The practicing physician needs to encourage his high-risk patients to modify their life-styles. Whereas recommendations about diet, exercise, weight control and cigarette smoking are well founded, and not intrinsically harmful, those involving use of alcoholic beverages are less clear and may be harmful.

In moderation alcohol appears to be harmless and may be beneficial against CHD and atherothrombotic strokes. In excess it may increase CHD mortality, raise blood pressure, cause arrhythmias, damage the myocardium and promote thrombogenesis. Whatever the mechanism involved, CHD risk appears to be lowered by moderate alcohol intakes despite adverse effects on blood pressure, triglycerides and cardiac rhythm. Alcohol may also promote haemorrhagic strokes by raising blood pressure, particularly when abused.

The information on hand does not allow firm conclusions about protection, but does not indicate a need to restrict moderate alcohol intake in social drinkers who have no problems such as hypertension, hyperuricaemia, cardiac rhythm disturbances or impaired ventricular function. It can seldom, therefore, be recommended in high-risk coronary candidates who often have hypertension, left ventricular hypertrophy and hyperuricaemia. Also, because of the severe psychosocial and other complications of alcohol abuse, it is not wise to prescribe alcohol for abstainers, even for the purpose of avoiding CHD.

Conclusions

Epidemiological evidence developed over the past three decades indicates that the major atherosclerotic cardiovascular diseases are largely a product of a faulty life-style in susceptible persons. This atherogenic life-style is typified by a diet excessive in fat, energy, cholesterol and salt; sedentary habits, unrestrained weight gain and smoking cigarettes. The role of alcohol is complex since it may, depending on the amount consumed and the particular disease outcome, be beneficial or hazardous. Whereas, compared with abstention, moderate alcohol intake may be beneficial, heavy intake is harmful.

Most studies clearly indicate a protective effect of moderate intake (not exceeding two drinks per day) against CHD, but there are indications that heavy intake may increase CHD mortality. For stroke, alcohol seems to increase the risk of the haemorrhagic variety while possibly protecting against atherothrombotic brain infarction.

Alcohol appears to improve certain atherogenic factors while worsening others. It seems to improve blood HDL and LDL while raising triglyceride. Effects on thrombogenesis are variable depending on intake. Moderate intake may reduce blood pressure whereas heavy intake raises it. It can, when abused, damage the myocardium, impair its function and make the heart more irritable and prone to arrhythmia. It raises uric acid values and affects the blood sugar and fluid balance.

Thus, moderate or light intake appears to be beneficial as regards all-cause mortality, CHD and possibly atherothrombotic stroke, while carrying a penalty in haemorrhagic stroke. It can, when abused, produce disabling cardiomyopathy.

Prescription of alcohol to abstainers cannot be justified as a means for avoiding atherosclerotic cardiovascular disease. However, there is no reason for restricting alcohol intake in light social drinkers for the purpose of avoiding coronary disease unless they have hypertension, hyperuricaemia or are prone to arrhythmia.

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