By Arthur L. Klatsky, MD

The Epidemiology of Alcohol and Cardiovascular Diseases

This article summarizes and evaluates current knowledge about the relation between drinking alcoholic beverages and several cardiovascular conditions. Both possible harmful and beneficial effects are discussed. Kaiser Permanente investigators have been active in this area for more than 20 years, and their studies are reviewed. There are disparities with respect to amount of alcohol used and with respect to various cardiovascular conditions. Conclusions about benefit or harm depend upon individual risk/benefit consideration.

Introduction

Disparity in the relation of alcohol consumption to various cardiovascular (CV) conditions has become evident.[1] Heavier drinking is related to higher prevalence of cardiomyopathy (CM), hypertension (HTN), hemorrhagic stroke, and cardiac arrhythmias. Lighter drinking is related to lower prevalence of coronary artery disease (CAD), ischemic stroke, and sudden cardiac death. The composite of these relations in several population studies of overall CV mortality is a U-shaped curve (lighter drinkers at lower risk than abstainers or heavier drinkers), although several other studies show all drinkers, lighter and heavier, at lower CV mortality risk than abstainers. Increased non-CV mortality among heavier drinkers is found in all studies, with a J-curve (heavier drinkers at highest; lighter drinkers at lowest risk) for the all-cause alcohol-mortality relation.

Definitions of Moderate and Heavy Drinking

Any definition of moderate drinking is arbitrary. The operational definition here used is based upon the level of drinking in epidemiologic studies above which net harm is usually seen. Thus, less than three drinks per day is called "lighter" or "moderate" drinking, and three or more drinks per day, "heavy" drinking. Sex, age, and individual factors lower the upper limit for some persons and raise it for others. In data based upon surveys, systematic "underestimation" (lying) probably tends to lower the apparent threshold for harmful alcohol effects.

Fortunately, the amount of alcohol in a standard-sized drink of wine, liquor, or beer is approximately the same. Since people think in terms of "drinks," not milliliters or grams of alcohol, it seems to this author best to describe alcohol consumption in terms of drinks per day or week. When talking with patients, health professionals should always remember the importance of defining the size of drinks.

Alcoholic Cardiomyopathy (ACM)

The concept of an independent, direct cardiotoxic effect of alcohol has become accepted.[1] The circumstantial evidence is substantial, but the absence of specific diagnostic tests seriously impedes epidemiologic study. Alcohol-associated CM cannot be distinguished clinically or pathologically from dilated CM of unknown cause(s). Historical episodes suggest synergistic myocardial toxicity of alcohol with arsenic and cobalt; other cofactors in alcoholic heart disease remain speculative. A role for thiamine deficiency in low-output chronic heart failure has never been established, although an interaction with alcohol cardiotoxicity might exist in malnourished persons.

The most convincing circumstantial evidence for ACM is the extensive data, in animals and humans, of nonspecific cardiac abnormalities related to alcohol. These include structural abnormalities in autopsy and biopsy studies and demonstration of acute and chronic functional and metabolic derangements by several techniques. A possible nonoxidative metabolic pathway for alcohol has been reported by Laposata and Lange[2] in the heart, muscle, pancreas, and brain, related to fatty acid metabolism. Accumulation of fatty acid ethyl esters was shown to be related to blood alcohol levels and to mitochondrial metabolism. A report by Urbano-Marquez et al[3] showed in alcoholics a clear relation of lifetime alcohol consumption to structural and functional myocardial and skeletal muscle abnormalities. The amounts of alcohol were large—the equivalent of 120 grams alcohol per day for 20 years.

As of 1997, a majority of all cases of CM are considered to be of unknown cause. The proportion of CM cases attributed to alcohol varies markedly in reports, probably due mostly to differences in the alcohol consumption habits of the populations under study. Thus, recent reports include alcohol-attributable proportions ranging from 3.4% at Johns Hopkins Hospital[4] to 41.9% at the Philadelphia VA Hospital.[5]

The lack of specific diagnostic tests for ACM necessitates exclusion of other CV conditions for diagnosis. However, the probability of synergistic damage includes possibly enhanced alcohol cardiotoxity in the presence of other myocardial damage. For this reason, persons with heart muscle impairment or major arrhythmias should be especially strongly advised to limit alcohol intake to less than three drinks per day.

Hypertension (HTN)

An association between heavier alcohol consumption and HTN reported by Lian in French servicemen in 1915[6] was largely ignored for the next 60 years.

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Since the mid-1970s, largely because of epidemiologic studies in developed countries, alcohol ingestion has joined other correlates of hypertension, such as obesity and salt intake, as a major focus in research about possible HTN risk factors. An alcohol-HTN link has been shown in almost all of more than 50 cross-sectional and 10 prospective population studies in ambulatory persons in a number of countries.[7,8] Studies differ about whether the alcohol-HTN link is linear or nonlinear in men (i.e., is a consumption threshold present?); in women, the curve which represents the relation is J-shaped, or present only at higher alcohol intake. Studies of hospitalized alcoholics or problem drinkers have been conflicting with respect to HTN. It is possible that chronic alcohol-related conditions such as malnutrition, cirrhosis, and cardiomyopathy lower blood pressure in some persons.

Two Kaiser Permanente studies[9,10] are among the largest of the cross-sectional population surveys. The first[9] showed a J-curve in women and a threshold relation in men, with higher blood pressures at three or more drinks per day in both sexes (Figure 1). The findings were independent of age, sex, and race and, by direct cross-classification (examination of the alcohol-HTN relationship in population subcategories), of smoking, coffee intake, reported past heavy drinking, education, adiposity, and habitual salt use. HTN (greater than 160/95 mmHg) prevalence was doubled in white men and women reporting consumption of six or more drinks per day. The second Kaiser Permanente study[10] showed similar findings in an analysis adjusted simultaneously for age, adiposity, smoking, coffee, tea, and seven blood tests (Figure 2). Ex-drinkers did not have higher blood pressure than lifelong abstainers. Study of drinking variability and intake in the week before examination suggested rapid regression of alcohol-associated HTN with abstinence.

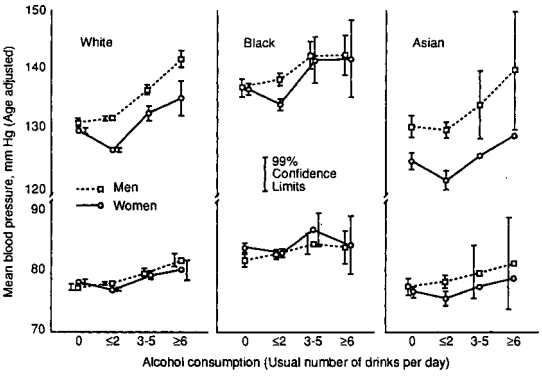

Figure 1. Mean systolic blood pressures (upper half) and mean diastolic blood pressures (lower half) for white, black, or Asian men and women with known drinking habits. Small circles represent data based on fewer than 30 persons. (From Klatsky AL, Friedman GD, Siegelaub AB, Gerard MJ. Alcohol consumption and blood pressure. Kaiser Permanente Multiphasic Examination data. N Engl J Med 1977;296:1194-1200. Used by permission.)

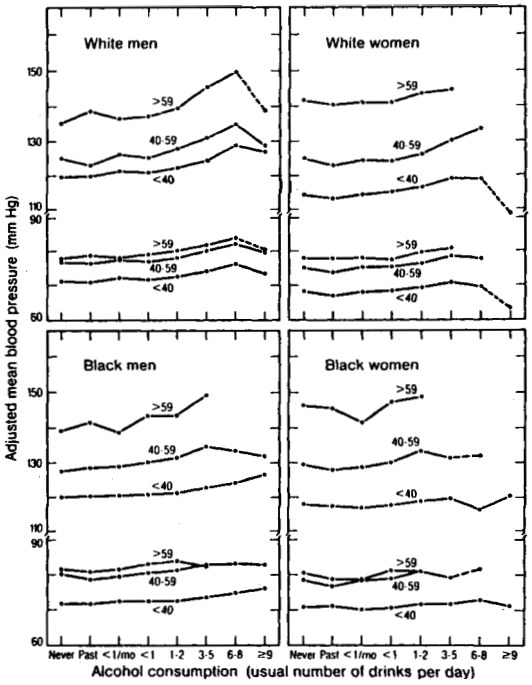

Figure 2. Adjusted mean systolic and diastolic blood pressures (mmHg) according to alcohol consumption by three age groups (top left, white men; lower left, black men; top right, white women; lower right, black women). Dashed lines and open circles indicate 10 < n < 25. Data omitted from figure for categories with n < 11 (white women aged 40 to 59 years, nine or more drinks per day and aged > 59 years, six to eight and nine or more drinks per day; black men aged > 59 years, six to eight and nine or more drinks per day; black women aged 40 to 59 years, nine or more drinks per day and aged >59 years, three to five, six to eight, and nine or more drinks per day). (From Klatsky AL, Friedman GD, Armstrong MA. The relationship between alcoholic beverage use and other traits to blood pressure: A new Kaiser Permanente study. Circulation 1986;73:628-636. By permission of the American Heart Association, Inc.

Several intervention studies suggest a short-term (develops in days to several weeks) pressor effect of three to eight alcoholic drinks per day, and decreases in blood pressure upon abstention or marked reduction in alcohol intake.[8] No elevations of blood pressure due to withdrawal have been seen in these studies. A few studies present data showing independence of the alcohol-blood pressure association from intake of salt, physical activity, and psychosocial stress. Even without confirmation in long-term trials, the intervention studies support a cause-effect relation between alcohol intake and HTN. Estimates of possible population-attributable risk (proportion of HTN due to alcohol) range from 5% to 30%.[8] Even if only 5% of HTN is attributable to alcohol, this may be the commonest cause of reversible HTN in developed societies.

The inconsistent acute effects of alcohol on blood pressure as reported in human and animal studies may not be directly relevant to the epidemiologic relation in humans.[8] There is no known animal model for chronic studies. There is no proof of a sustained effect in humans via the renin-angiotensin mecha-

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nism, cortisol, catecholamines, increased cardiac output, "hypermetabolic state," central nervous system actions, or autonomic nervous system effects. A recently reported experiment[11] in normal humans used intraneural microelectrodes to demonstrate increased sympathetic activation in response to I.V. alcohol with a delayed (second hour) blood pressure rise. Inhibition by dexamethasone suggested a central mechanism via corticotropin-releasing hormone. There is some current interest in a possible direct effect upon peripheral vascular tone via a calcium transport mechanism. Explanations for the alcohol-HTN association remain speculative; this fact is the major deficiency in the case for causality. Studies of HTN sequelae (coronary disease, stroke, congestive heart failure, renal insufficiency, etc.) are greatly complicated by the independent relations of alcohol use to several common hypertension sequelae.[8]

It is likely that the alcohol-HTN link is causal. Reduction of intake in some heavier drinkers is probably therapeutic, and avoidance of heavier drinking will probably prove to have an important role in primary prevention of HTN.[12]

Coronary Artery Disease (CAD)

Data showing that major CAD events are more likely to develop in abstainers than in alcohol drinkers include international comparisons, time-trend analyses, case-control studies, and longitudinal studies.[13,14] Most studies of CAD hospitalizations show heavier drinkers have a risk of CAD hospitalization similar to or lower than that of lighter drinkers (i.e., no U-shaped curve). Several population studies using CAD mortality as an endpoint also show a progressive inverse relation to amount of alcohol consumption, but others show a U-shaped curve. Those studies which separate lifelong abstainers from past drinkers suggest that both subsets of nondrinkers are at higher risk of CAD than drinkers, but some would still dispute this. Many population studies were not able to distinguish these subsets of nondrinkers. Where available, data about choice of type of alcoholic beverage suggest that beverage choice is a minor factor in CAD risk. Studies of sudden cardiac death, due mostly to CAD, also show an inverse relation to alcohol use.

There are plausible mechanisms by which alcohol drinking might protect against CAD.[13,14] These include a favorable effect on HDL cholesterol concentration (an increased level), a similarly favorable effect upon apolipoproteins, and an antithrombotic action. Controversy about protection persists, however, on the grounds that correlates of abstinence and lighter drinking could explain the higher risk of abstainers. For example, a much publicized hypothesis advanced by Shaper et al[15] suggested that movement of persons at high CAD risk into the abstainer referent group could explain the U-shaped curve shown in their work and in that of other investigators.

A prospective Kaiser Permanente study of alcohol habits in relation to CAD hospitalizations[16] showed that ex-drinkers and infrequent (less than 1/month) drinkers were at a risk similar to that of lifelong abstainers. A lower CAD risk was present among all other drinkers with no U-shaped curve, independent of a number of potential confounders (Table 1). These relations were independent of base line CAD risk at examination (Table 2) and beverage choice. The data suggested a protective effect of alcohol against risk of hospitalization for CAD.

In a prospective Kaiser Permanente study of total CV mortality,[17] ex-drinkers had higher age-adjusted CAD and overall CV mortality risk than lifelong abstainers, but the difference disappeared when adjusted for other traits. Among drinkers, there were U-shaped mortality curves relating amounts of alcohol and both CVD and CAD, with a nadir at one to two and at three to five drinks per day. Subsets free of baseline risk had similar alcohol-CAD and alcohol-CV mortality curves. The study demonstrated the expected disparities between alcohol and various CV conditions (Table 3). A number of features of the analysis argued against a spurious inverse alcohol-CAD relation, including:

1) independence from CAD risk at baseline examination;
2) absence of higher CAD risk among persons reducing alcohol intake for medical reasons;
3) evidence that the higher unadjusted CAD risk of ex-drinkers is due to confounding;

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Table 1. Relative Risk of Coronary Artery Disease Hospitalization* According to Alcohol Use

Alcohol Use	RR#	95% CI	p value
Nondrinkers			
Abstainers	1.0 (ref)	----	----
Ex-drinkers	1.0	(0.7, 1.4)	0.9
Drinkers			
<1 per month	0.9	(0.7, 1.2)	0.6
<1 per day, >1 per month	0.7	(0.5, 0.8)	<0.001
1-2 per day	0.6	(0.5, 0.7)	<0.0001
3-5 per day	0.5	(0.4, 0.8)	<0.001
6-8 per day	0.5	(0.3, 1.1)	0.1
9 per day	0.5	(0.2, 1.5)	0.2

* First for any CAD diagnosis (n=756)
# Computed from coefficients estimated by Cox proportional hazards model; covariates include sex, age, race, smoking, education, coffee
RR=relative risk; CI=confidence interval
(adapted from Klatsky AL, Armstrong MA, Friedman GD. Am J Cardiol 1986;58:710-4)

Table 2. Relative Risk of Coronary Artery Disease Hospitalization According to Alcohol Use Among Persons Free of Coronary Risk/Symptoms or Recent Major Illness*

Alcohol Use	RR#	95% CI	p value
Nondrinkers			
Abstainers	1.0 (ref)	-----	-----
Ex-drinkers	0.9	(0.6, 1.6)	0.8
Drinkers			
<1 per month	0.6	(0.6, 1.3)	0.8
>1 per month	0.6	(0.4, 0.9)	<0.01
1-2 per day	0.5	(0.3, 0.7)	<0.0001
3-5 per day	0.5	(0.3, 0.8)	<0.01
6-8 per day	0.7	(0.2, 1.8)	0.4
9 per day	0.5	(0.1, 3.8)	0.5

* First for any CAD diagnosis (n=336) among persons with no CHD risk/symptoms (12 items) or other major illness in the past year.
# Computed from coefficients estimated by Cox proportional hazards model; covariates include sex, age, race, smoking, education, coffee.
RR=relative risk; CI=confidence interval
(Adapted from Am J Cardiol 1986;58:710-714)

4) absence of a relation among ex-drinkers between CAD risk and maximal past intake;
5) absence of a relation between infrequent (less than 1/month) drinking and CAD risk;
6) similar reduction of CAD risk among drinkers of wine, liquor, and beer.

Another large prospective study among women free of CAD at examination[18] showed a progressive inverse relation of alcohol use to major CAD events, independent of prior reduction in alcohol intake and of nutrient intake (the latter was analyzed in detail). The relative risk of CAD events in women reporting daily alcohol intake of 25 or more grams per day was 0.4, similar to the findings for women in the Kaiser Permanente study. Further analysis of these data in women[19] demonstrated that net beneficial effects of moderate alcohol use in women was limited by adverse effects to persons clearly at above-average CAD risk (i.e., those above 50 years of age).

Large prospective studies in men also confirm the lower CAD risk of drinkers, independent of confounders or disease when alcohol habits were determined.[20,21] The American Cancer Society Study[20] was a 12-year prospective mortality study of 276,802 white men; there was a U-shaped curve for CAD mortality, with a RR of 0.8 (vs. abstainers) at one to two drinks per day. The Health Professional Followup Study of 51,529 men[21] was well controlled for dietary habits; newly diagnosed CAD was inversely related to increasing alcohol intake. A study in both sexes, the Auckland Heart Study,[22] was designed to study the hypothesis that persons at high CAD risk are likely to become nondrinkers; the analysis showed that moderate drinkers had lower CAD risk than both lifelong abstainers and ex-drinkers, thus supporting the hypothesis that alcohol protects against CAD.

Reduced risk of CAD is present at various ages, although the impact upon total mortality in a Kaiser Permanente study was clearest in older age brackets and the adverse effects of alcohol were greater among younger persons.[23] Among persons > 60 years of age, overt or latent CAD may play a role in risk of death from causes other than CAD.[23]

The hypothesis that the apparent protective effect of alcohol against CAD is mediated by higher HDL cholesterol levels in drinkers has been examined quantitatively in three separate studies.[24-26] All three analyses yielded similar findings suggesting that higher HDL levels in drinkers mediated about half of the lower CAD risk. One of these studies[26] suggests that both HDL2 and HDL3 are involved. HDL3 may be more strongly related to lighter alcohol intake but is probably related as strongly as HDL2 to lower CAD risk. There are no similar data about protective mechanisms other than the HDL link, but some data support several possible antithrombotic mechanisms.[13,14,26] Thus, multiple mechanisms may play a role.

International comparison studies[27-29] suggest that wine confers more protection against CAD than beer or liquor. The "French paradox" concept has arisen from these data; it refers to the fact that France tends to be an outlier on graphs of mean dietary fat intake vs. CAD mortality, unless adjusted for wine alcohol intake.[28,29] Reports of nonalcohol antioxidant phenolic compounds[30-32] or antithrombotic substances[33-36] in wine, especially red wine, have appeared. Inhibition of oxida-

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Table 3. Relative Risk* of Death of Various Cardiovascular Conditions and Cirrhosis by Alcohol Use

	Relative risk for each drinking category versus lifelong abstainers					
Condition (n deaths)	Ex-drinkers	<1 per month	>1 per day; <1 per month	1-2 per day	3-5 per day	6 or more per day
All CAD (600)	1.0	0.9	0.8(a)	0.7(b)	0.7(b)	0.8
AMI (284)	1.0	0.7	0.8	0.6(b)	0.5(b)	0.6
Other CAD (316)	0.9	1.0	0.7	0.8	0.7	1.0
Stroke (138)	1.0	0.8	0.8	0.8	0.7	1.4
Hemorrhagic (41)	1.4	1.5	1.6	1.8	1.3	4.7
Ischemic (34)	0.9	0.5	0.5	0.3	0.4	--(d)
Nonspecific (63)	1.1	0.7	0.9	1.0	1.0	1.2
Hypertension (64)	2.8	2.4	1.9	1.3	2.2	2.1
Cardiomyopathy (24)	3.4	8.5(a)	4.0	5.6	2.4	8.0
Syndromes (82)**	0.6	0.6	0.5	0.4(a)	0.6	1.0
Arterial (41)***	--(d)	1.1	1.6	0.4(a)	1.7	--(d)
Cirrhosis (42)	10.8(c)	1.4	1.0	4.3	8.1(b)	22.0(c)

* Computed from coefficients estimated by Cox proportional hazards model; covariates include sex, age, race, smoking, education, coffee; reference group is lifelong abstainers.
** Includes "symptomatic heart disease" (n=32); disorders of heart rhythm (n=22); and ill-defined heart disease (n=28).
*** Includes arteriosclerosis (n=15); aneurysms (n=23); peripheral vascular disease (n=2); and arterial embolism and thrombosis (n=1).
(a)=p<0.05; (b)=p<0.01; (c)=p<).001; (d)=insufficient cases for estimate.
CAD=coronary artery disease; AMI=acute myocardial infarction.
(Adapted from Klatsky AL, Armstrong MA, Friedman GD. Am J Cardiol 1990; 66:1237-42)

In Kaiser Permanente studies, all three major beverage types show evidence of protection against CAD ...[16,17]

tive modification of low-density-lipoprotein cholesterol is probably anti-atherogenic, although prospective clinical trials of antioxidant supplements are not yet conclusive.[37] Thus, antioxidant substances in wine are an attractive hypothetical explanation for CAD protection. However, the prospective population studies provide no consensus that wine has additional benefits, and various studies show benefit for wine, beer, liquor, or all three major beverage types.[13,14] In Kaiser Permanente studies, all three major beverage types show evidence of protection against CAD[16,17]; wine drinkers fare best with respect to CAD mortality, but drinkers of red and non-red wine fare equally well.[38] Because the beverages differ in user traits, with wine drinkers having the most favorable CAD risk profile,[39] a noncausal explanation was favored for the lower CAD risk of wine drinkers. Drinking-pattern differences among the beverage types are another hypothetical factor. The wine/liquor/beer issue is unresolved at this time, but it seems likely that ethyl alcohol is the major factor with respect to lower CAD risk.

It remains theoretically possible that lifelong abstainers could differ from drinkers in psychological traits, dietary habits, physical exercise habits, or some other way which could be related to CAD risk, but there is no good evidence for such a trait. The various studies indicate that such a correlate would need to be present in persons of both sexes, various countries, and multiple racial groups. Although it remains possible that other factors play a role, a causal, protective effect of alcohol is a simpler and more plausible explanation.[13,14,40,41]

Cerebrovascular Disease

Several reports suggest that alcohol use, especially heavier drinking, is associated with higher risk of stroke. Some studies examined only drinking sprees; others did not differentiate between hemorrhagic and occlusive strokes. Several studies have suggested that alcohol was related only to hemorrhagic stroke. The Nurse's Health Study[18] showed drinkers to be at higher risk of subarachnoid hemorrhage but at lower risk of occlusive stroke.

A Kaiser Permanente study looked at the relations between reported alcohol use and the incidence of hospitalization for several types of cerebrovascular disease.[42] Daily consumption of 3 or more drinks, but not lighter drinking, was related to higher hospitalization rates for hemorrhagic cerebrovascular disease, especially intracerebral hemorrhage. Higher blood pressure appeared to be a partial mediator of this relation. Alcohol use was associated with lower hospitalization rates for occlusive cerebrovascular disease; an inverse relation was present for both sexes, for whites and blacks, and for extracranial and intracerebral occlusive lesions (Table 4). The data suggest that heavier drinking increases the risk of hemorrhagic cerebrovascular events but that alcohol use may lessen the risk of occlusive lesions.

At this time there is no consensus about the relations of alcohol drinking to the various types of cerebrovascular disease and agreement only that more study of this important area is needed.[43]

Table 4. Relative Risk of Stroke Hospitalization* According to Alcohol Use and Other Traits

Trait	RR#	95% CI	RR	(95% CI)
Alcohol				
Abstainer	1.00 (ref)	-----	1.00 (ref)	-----
Ex-drinker	0.8	0.2-2.9	1.0	0.6-1.6
<1 per day	0.9	0.4-1.8	0.6(a)	0.5-0.9
1-2 per day	0.8	0.3-1.9	0.5(a)	0.4-0.8
3 per day	1.4	0.5-3.6(b)	0.4(a)	0.3-0.8
Age (per 10 years)	1.7(a)	1.4-2.1	2.4(a)	2.1-2.7
Sex (F vs M)	0.8	05-1.4	0.6(a)	0.5-0.7
Race (B vs W)	2.4(a)	1.4-4.0	1.0	0.8-1.4
Smoking (>1ppd vs never)	1.9	0.8-4.1	3.1(a)	2.0-4.8
BMI (per 0.1 unit)**	1.0	0.9-1.0	1.0	0.97-1.0
Systolic BP (per 10 mmHg)	1.2(a)	1.1-1.4	1.2(a)	1.1-1.2
Baseline CAD risk (yes/no)***	1.2	0.7-2.0	1.8(a)	1.4-2.2

* Computed from coefficients estimated by Cox proportional hazards model.
** Quetelet's
*** Any "yes" to 12 history/risk items
(a) indicates significantly different from 1.0 ($p<0.05$)
RR=relative risk; CI=confidence interval; BMI=body mass index; CAS=coronary artery disease
(adapted from Klatsky AL, Armstrong MA, Friedman GD. Stroke 1989;20:741-746)

Cardiac Arrhythmias

Increased ventricular ectopic activity has been documented after ingestion of substantial amounts of alcohol, although epidemiologic studies have not shown a higher risk of sudden death in drinkers. Various atrial dysrhythmias have been reported to be associated with spree drinking. A Kaiser Permanente study[44] compared atrial dysrhythmias in 1,322 persons reporting six or more drinks per day to dysrhythmias in 2,644 light drinkers. The relative risk in the heavier drinkers was at least doubled for atrial fibrillation, atrial flutter, supraventricular tachycardia, and atrial premature complexes (Table 5).

Table 5. Relative Risk* of Supraventricular Arrhythmia in Persons with High versus Low Daily Alcohol Intake

	Persons with Arrhythmia					
	6+ per day (n=1,332)		<1 drink per day (n = 2,664)			
Rhythm	No.	%	No.	%	RR (6+/<1)	p value
Atrial Fibrillation	15	1.1	13	0.5	2.3	0.02
Atrial Flutter	8	0.6	6	0.2	3.0	0.05
SVT	5	0.4	2	0.1	5.0	0.03
APB's	43	3.3	32	1.3	3.0	<0.01
Fibrillation, flutter, or SVT	21	1.6	19	0.7	2.3	<0.01

* Relative risks and p values estimated using McNemar's method for matched pairs

RR = relative risk; SVT = supraventricular tachycardia; APB's = atrial premature beats.
(Adapted from Cohen EJ, Klatsky AL, Armstrong MA. Am J Cardiol 1988;62:971-973)

Table 6. Relation of Alcohol Drinking to Cardiovascular Conditions			
Condition	Amount of Alcohol Drinking		Comment
	Small	Large	
Dilated Cardiomyopathy	no relation	probably causal	? unknown cofactors
Beri-beri	no relation	no relation	thiamine deficiency
(As) Co--beer Disease	no relation	synergistic	examples of cofactors
Hypertension	little or no relation	probably causal	mechanism unknown
Coronary Disease	protective	? protective	via HDL, anti-thrombotic effects; beverage type minor factor
Arrhythmia	? none	probably causal	? susceptibility factors
Hemorrhagic Stroke	? increased risk	increased risk	via high BP, anti-thrombotic actions
Ischemic Stroke	protective	? protective	complex interactions with other conditions

Conclusion

This brief survey documents the evidence for disparity in the relations of alcohol and CV disorders. Published reviews are available.[1,46] Table 6 summarizes the relations, with emphasis on the disparity between the overall favorable relations of lighter drinking and the overall unfavorable relations of heavier drinking. ❖

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"At the still point of the turning world ... there the dance is."
T.S. Eliot, Four Quartets