

## Effects of Drinking Patterns on the Relationship between Alcohol and Coronary Occlusion

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### **Abstract:**

Previous reports have described an inverse relationship between alcohol consumption and the prevalence of myocardial infarction or the extent of coronary artery occlusion. The study reported here explored the relationship between patterns of alcohol intake and coronary occlusion in 526 male patients who have had coronary arteriography. Patients were characterized as regular drinkers, occasional drinkers and non-drinkers. Regular drinkers were further characterized as drinking relatively consistent amounts or variable amounts. The inverse correlation between amounts of alcohol consumed and coronary occlusion found in previous studies was reaffirmed. It was also observed that the pattern of alcohol intake was related to the degree of occlusion. Higher levels of occlusion were found among non-drinkers, occasional drinkers, and regular drinkers with patterns of variable intake, while significantly lower levels of occlusion were observed for regular drinkers who drank relatively consistent amounts ( $P = 0.014$ ). Furthermore, while occlusion scores were inversely correlated with amounts consumed by regular drinkers with consistent intake ( $P = 0.019$ ), drinkers with variable drinking patterns had higher occlusion scores regardless of amounts consumed. Analyses of serum lipids according to drinking patterns showed a significant association between the total/HDL cholesterol ratio and drinking patterns. These findings suggest that whatever attenuating effect alcohol consumption might exert on coronary occlusion, it appears to be reversed by a variable or sporadic pattern of alcohol intake.

**Key words:** Alcohol consumption — Coronary occlusion — Myocardial infarction

### **Article:**

#### **Introduction**

Moderate alcohol consumption (30-90 ml ethanol per day) has been related to lower rates of coronary heart disease (CHD) and coronary artery occlusion in a number of studies [1-6]. Similar reductions in atherosclerosis have been reported for rabbits on diets supplemented with alcohol [7]. The relation of CHD and coronary occlusion to heavier alcohol consumption (over 100 ml ethanol/day) has been less well studied. Some evidence has been reported which suggests that heavy drinkers experience an inverse association between alcohol consumption and CHD, similar to moderate drinkers, but other findings have been presented to dispute this interpretation [8-10].

Recently, an analysis of mortality experience among British civil servants showed that total mortality was lower among men with moderate alcohol intake, while cardiovascular mortality was greater in non-drinkers and non-cardiovascular mortality was greater in heavier drinkers [11].

In all of these previous studies, total or average rates of consumption were measured but the pattern of alcohol use was not. Some of the inconsistencies in these results might have occurred because the drinking patterns of regular drinkers were incompletely characterized. For example, occasional heavier drinking superimposed on a more moderate usual consumption pattern might alter an individual's risk front what it would be if only total or average consumption was considered.

In the study reported here, we measured coronary occlusion in relation to the total amount of alcohol consumed and in relation to the pattern of alcohol intake. The results indicate that although alcohol consumption was

inversely associated with coronary occlusion, the pattern of alcohol intake was also an important factor in determining the degree of occlusion.

## **Materials and Methods**

### ***Study sample***

A group of 526 volunteer male patients who were referred for diagnostic angiographic examination to two Milwaukee hospitals between 1974 and 1979 were studied. Their mean age was 54 years, with ages ranging between 31 and 80. Informed consent for their participation in the study was obtained according to the principles of the Declaration of Helsinki.

### ***Coronary occlusion***

The criteria for the referral of these men were unstable angina pectoris, moderate-to-severe stable angina, previous myocardial infarction, and recurrent chest pain of unknown etiology. The coronary arteriography was carried out by the technique of either Sones and Shirey [12] or Judkins [13]. The angiograms were reviewed independently and by consensus, by a cardiologist and a radiologist, and were recorded according to a uniform protocol. The extent of coronary artery occlusion was graded, as suggested by Rowe et al. [14], except that the scale was inverted with the occlusion scores ranging from 0 (no occlusion) to 300 (complete occlusion). In this method, patients are considered to have three main coronary arteries: right, left anterior descending, and circumflex. If there was an occlusion, an estimate was made of its extent in increments of 25%. For instance, if the left anterior descending was considered 75% occluded and the other two vessels were normal, the occlusion score would be 75. If disease was found in a branch of one of the main vessels, the size of the branch was estimated as compared to that of its parent vessel, the degree of occlusion of the branch was estimated, and the overall effect of the occlusion approximated. This figure was added to the total score.

### ***Alcohol consumption***

Information on the amount, kind, and frequency of alcohol intake was obtained by self-administered questionnaire, based on the method of Khavari [15]. In this method respondents were asked a series of questions about their drinking behavior, including: (1) how much of an alcoholic beverage they usually drank; (2) how frequently; (3) if they ever drank more than their usual amount, what is the most they drank; and (4) how often. These questions were repeated three times—once each for beer, wine, and liquor. Alcohol intake was converted to equivalent ounces of ethanol using values of 4% for beer, 12% for wine and 43% for liquor.

The ability of the Khavari alcohol test (KAT) to discriminate between groups with different consumption levels has been demonstrated [15]. The reported reliability of this instrument for measuring individual alcohol consumption, based on test–retest correlations, was 0.92. The KAT questionnaire and its scoring instructions are available from the Ralph G. Connor Alcohol Research Reference Files (CARRF), Center of Alcohol Studies, Rutgers University, New Brunswick, NJ 08903.

The alcohol questions comprised a portion of an extensive questionnaire given to all hospitalized patients on the day prior to their coronary angiography. Thus, the extent of coronary occlusion in any patient was not known at the time the questionnaire was administered.

From responses to the Khavari questionnaire, three types of drinking patterns were characterized. Men who drank alcoholic beverages with a frequency of at least once-per-week were classified as regular drinkers. Men who drank less often than once-per-week were classified as occasional drinkers, and those who reported they did not drink any alcoholic beverages were non-drinkers.

The patterns of regular drinkers were further characterized by the degree of variability in the quantity of alcohol consumed on different occasions. This variability was measured by the ratio of the maximum to the usual amounts consumed by each individual. Men with low variability ratios (minimum = 1.0) had only small differences between their usual and maximum amounts, whereas those with higher ratios had larger differences, and thus more variable drinking patterns.

### Serum lipids

Fasting blood] samples were collected after an overnight fast at the time of a post-arteriography follow-up visit. The samples were analyzed for total plasma cholesterol and plasma triglyceride levels using automated procedures [16,17] quality control monitored by the Lipid Standardization Program, Center for Disease Control, Atlanta, GA. Plasma high density lipoproteins (HDL) were measured after the heparin—manganese precipitation of the low and very low density lipoproteins (LDL and VLDL) [18].

The procedure was standardized with serum samples of known HDL cholesterol content supplied by courtesy of Dr. G.R. Cooper, Center for Disease Control, Atlanta, GA.

### Smoking history

Information on cigarette smoking history was obtained by direct questioning of the patient. A five-point smoking scale (1 = non-smokers; 5 = heaviest smokers) was constructed as reported in a previous publication [19].

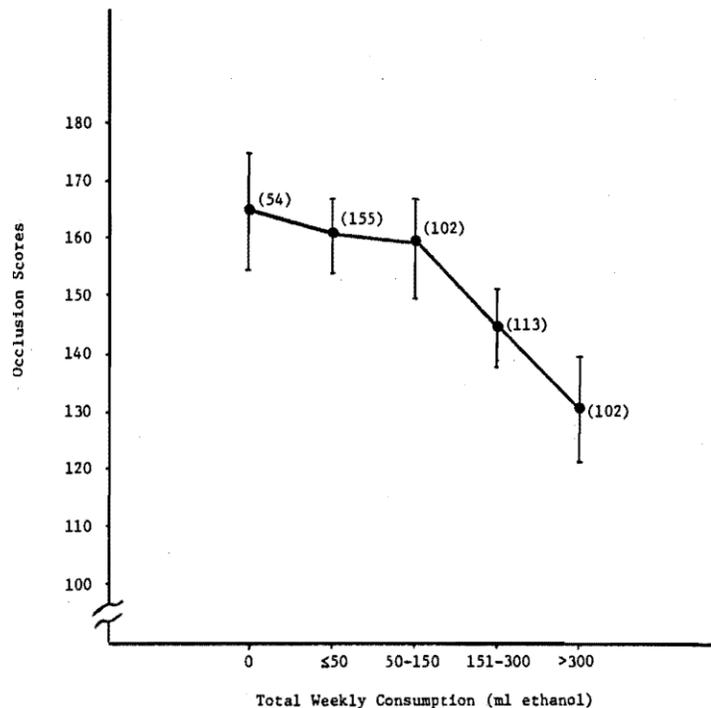


Fig. 1. Coronary occlusion scores for all 526 men in study, according to weekly alcohol consumption. Values shown are means ± SE.  $F=9.771$ ,  $P=0.0019$ . Numbers of patients in each group are given in parentheses.

### Results

For the entire group of men in the study, significantly lower coronary occlusion scores were observed for higher levels of total alcohol consumption (Fig. 1). The highest degree of occlusion was observed among the non-drinkers, and the lowest occlusion scores were among the heaviest drinkers (> 300 ml ethanol weekly). Separate analyses of amounts of alcohol consumed per occasion and the frequency of alcohol intake showed both to be significantly and inversely related to occlusion scores, similar to the trend for total alcohol consumption. As in previous studies, this inverse relationship between alcohol consumption and coronary occlusion was independent of age or smoking [4].

Comparisons between men with different drinking patterns showed regular drinkers to have significantly lower occlusion scores than either occasional drinkers or non-drinkers (Table 1). Regular drinkers also had significantly higher total alcohol consumption, but there were no significant differences in age or smoking history between any of these groups.

TABLE 1

COMPARISONS BETWEEN REGULAR, OCCASIONAL, AND NON-DRINKERS BY OCCLUSION SCORE, ALCOHOL CONSUMPTION, AGE, AND CIGARETTE SMOKING

	Drinking patterns <sup>a</sup>		
	Regular	Occasional	Non-drinker
Number	309	163	54
Occlusion score *	145.1± 4.9	160.6±6.4	165.4±9.7
Weekly alcohol consumption (ml ethanol) **	300.3±17.9	23.8±1.5	-
Age (years)	53.7± 0.5	54.9±0.5	54.7±0.9
Smoking scale	3.5± 0.1	3.3±0.1	3.2±0.2

<sup>a</sup> See text for definitions of patterns. Values given are means±SE.

\* Differences between groups significantly different based on F-ratios:  $P < 0.025$  for occlusion scores, and  $< 0.01$  for alcohol consumption.

Figure 2 shows the occlusion scores for regular drinkers according to their variability ratios. Significantly higher occlusion scores were observed for men with higher ratios, most notably for those with ratios over 2.0 ( $P = 0.014$ ). However, no significant relationships were evident between variability ratio and weekly alcohol consumption, age or smoking history. Additional univariate analyses (comparing men with ratios above and below 2.0), and multivariate regression analyses con-firmed that the direct correlation between variability ratio and occlusion score was independent of these other variables.

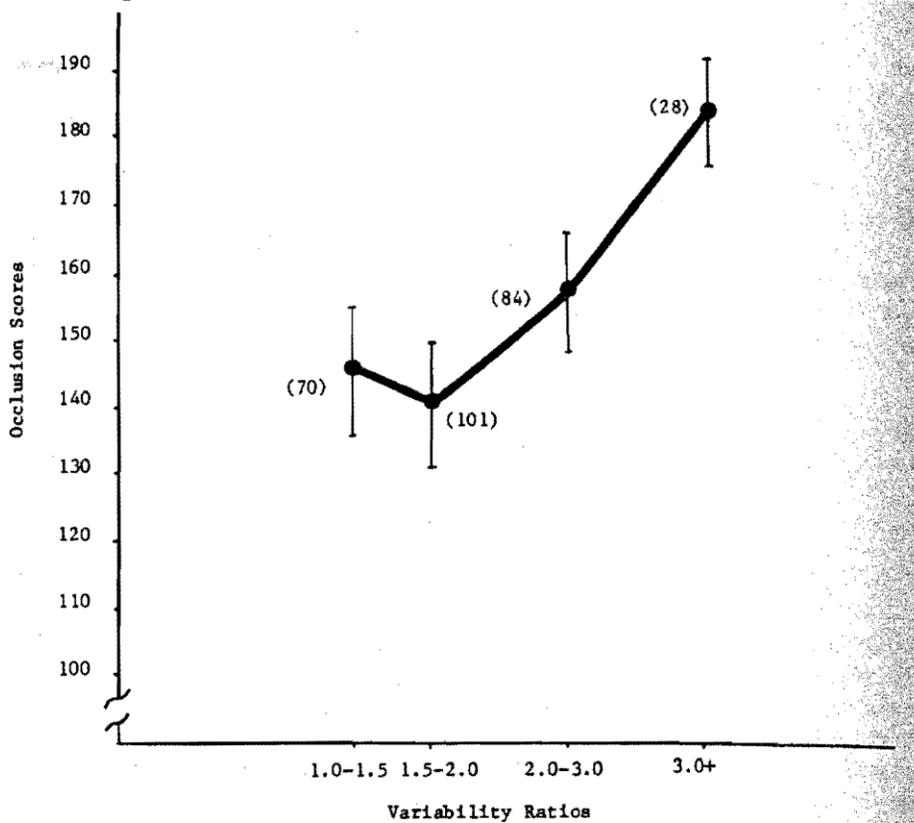


Fig. 2. Adjusted coronary occlusion scores for regular drinkers by variability ratios, (minimum ratio=1.0). Scores are adjusted for smoking history, age, and total alcohol consumption. Values given are means±SE.  $F=2.792$ ,  $P=0.041$ . Twenty-six patients were excluded from this analysis because of incomplete data, yielding a total n of 283. Numbers of patients in each group are given in parentheses.

Variability ratios also affected the association between coronary occlusion and total alcohol consumption. Men with more variable drinking patterns (variability ratios  $\geq 2$ ) had higher occlusion scores, regardless of the total amount of alcohol consumed (Table 2), while occlusion scores were negatively related to the amount of alcohol consumed by drinkers with lower variability ratios ( $\leq 2$ ).

TABLE 2

## OCCLUSION SCORES FOR WEEKLY ALCOHOL CONSUMPTION BY VARIABILITY RATIOS OF REGULAR DRINKERS

Numbers of patients are given in parentheses.

Variability ratio	Total weekly amounts (ml ethanol)			P-value *
	< 100	100-300	> 300	
>2	172 (23)	158 (60)	160 (35)	0.596
≤2	169 (36)	143 (71)	129 (66)	0.019
Total	170 (59)	150 (131)	139 (101)	0.021

\* P-values are based on F-ratios for linear trends in occlusion scores with increasing weekly amounts.

The results of the serum lipid determinations are given in Table 3. Total cholesterol, HDL cholesterol, and the ratio of total to HDL cholesterol varied significantly with drinking patterns. HDL cholesterol levels as well as total cholesterol levels were higher for regular drinkers than for occasional drinkers or non-drinkers, but there were only small differences in these levels between regular drinkers with high or low variability ratios. However, the lowest total/HDL cholesterol ratio was seen among regular drinkers with low ( $\leq 2$ ) variability ratios ( $P < 0.05$ ). This indicated that the fraction of total cholesterol accounted for by the HDL component was significantly higher among regular drinkers with low variability ratios than among those with high variability ratios.

TABLE 3

## RESULTS OF SERUM LIPID DETERMINATIONS ACCORDING TO DRINKING PATTERNS

Values given are means  $\pm$  SE.

	Non-drinkers (n=54)	Occasional drinkers (n=163)	Regular drinkers		P-value *
			Ratios $\leq 2$ (n=173)	Ratios $> 2$ (n=118)	
Triglycerides (mg/dl)	185.8 $\pm$ 51.7	160.8 $\pm$ 9.8	154.3 $\pm$ 7.7	158.4 $\pm$ 10.7	0.67
Total cholesterol (mg/dl)	215.9 $\pm$ 9.1	219.0 $\pm$ 3.6	228.5 $\pm$ 2.8	234.0 $\pm$ 4.6	0.02
HDL cholesterol (mg/dl)	43.8 $\pm$ 2.0	43.9 $\pm$ 1.3	49.5 $\pm$ 0.9	47.8 $\pm$ 1.4	<0.01
Total/HDL cholesterol ratio	6.1 $\pm$ 0.7	5.4 $\pm$ 0.2	4.9 $\pm$ 0.1	5.3 $\pm$ 0.2	0.02

\* P-values are based on F-ratios for the differences between groups.

## Discussion

The results of this study confirm previous findings that coronary occlusion is inversely associated with total alcohol intake [4]. However, more significantly the data indicate that the pattern of alcohol consumption also affects the degree of coronary occlusion. Lower occlusion scores were observed among regular drinkers who drank relatively consistent amounts, while higher scores were observed among non-drinkers, occasional drinkers, and regular drinkers who drank varying amounts.

A further interesting finding was that the inverse relationship between amount of alcohol consumed and degree of coronary occlusion existed for regular drinkers with relatively consistent intake patterns, but not for regular drinkers with more variable intake patterns. The implication of these findings is that whatever protective effect alcohol consumption might convey for coronary occlusion (and presumably CHD), it appears to be reversed by a variable or sporadic consumption pattern.

At this time there is no definite metabolic explanation for the effects of different drinking patterns on coronary occlusion. There have been several reports linking alcohol intake to elevated levels of HDL cholesterol [20,21]. We have previously investigated the interrelationship of coronary occlusion, HDL cholesterol, and alcohol [22],

and found that HDL cholesterol accounted for at least part of the alcohol—coronary occlusion association. The data presented here indicate that studies of serum lipids in relation to patterns of alcohol consumption may provide further insight to the metabolic factors involved. An elevated HDL cholesterol fraction was evident for regular drinkers with low variability ratios. The possibility of differential changes in HDL subfractions was not looked at in this study, but should also be considered.

The findings of this study indicate the importance of drinking patterns to the relationship between alcohol intake and coronary occlusion. However, generalization of these findings to other populations may be limited by the select nature of our study group which consisted of men with symptoms suggestive of heart disease, although there is no evidence of bias that could alter our conclusions. Little information is available on the drinking habits or other risk factors of these men prior to the time of angiography, but a preliminary study of the drinking habits indicates considerable long-term constancy. Also, despite the apparent selection bias, our results agree with those of other investigators who found inverse relationships between alcohol intake and CHD in less selected populations (c.f. [1,3,5,6]).

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