

The New England Journal of Medicine

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VOLUME 341

NOVEMBER 18, 1999

NUMBER 21



LIGHT-TO-MODERATE ALCOHOL CONSUMPTION AND THE RISK OF STROKE AMONG U.S. MALE PHYSICIANS

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ABSTRACT

Background Several studies have shown U- or J-shaped relations between alcohol consumption and the risk of stroke. We evaluated the effect of light-to-moderate alcohol intake on the risk of stroke, with separate analyses of ischemic stroke and hemorrhagic stroke.

Methods Our analyses were based on a prospective cohort study of 22,071 male physicians, 40 to 84 years old, who were participating in the Physicians' Health Study. At base line, the participants reported that they had no history of stroke, transient ischemic attack, or myocardial infarction and were free of cancer. Alcohol intake, reported by 21,870 participants at base line, ranged from none or almost none to two or more drinks per day.

Results During an average of 12.2 years of follow-up, 679 strokes were reported. As compared with participants who had less than one drink per week, those who drank more had a reduced overall risk of stroke (relative risk, 0.79; 95 percent confidence interval, 0.66 to 0.94) and a reduced risk of ischemic stroke (relative risk, 0.77; 95 percent confidence interval, 0.63 to 0.94). There was no statistically significant association between alcohol consumption and hemorrhagic stroke. The overall relative risks of stroke for the men who had one drink per week, two to four drinks per week, five or six drinks per week, or one or more drinks per day were 0.78 (95 percent confidence interval, 0.59 to 1.04), 0.75 (95 percent confidence interval, 0.58 to 0.96), 0.83 (95 percent confidence interval, 0.62 to 1.11), and 0.80 (95 percent confidence interval, 0.64 to 0.99), respectively, in an analysis in which we controlled for major risk factors for stroke.

Conclusions Light-to-moderate alcohol consumption reduces the overall risk of stroke and the risk of ischemic stroke in men. The benefit is apparent with as little as one drink per week. Greater consumption, up to one drink per day, does not increase the observed benefit. (N Engl J Med 1999;341:1557-64.)

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STROKE is a leading cause of morbidity and mortality in many countries.¹ Among the risk factors for stroke, potentially hazardous but modifiable behavior such as alcohol consumption has drawn increasing attention in recent years, especially after a U-shaped relation was suggested between alcohol consumption and coronary heart disease.² Alcohol consumption is a modifiable behavior, and drinking moderate amounts of alcohol may have protective effects against subtypes of stroke.³⁻⁶ Although most studies show a positive correlation between drinking and the risk of hemorrhagic stroke,^{3,7-10} the relation with ischemic stroke is less clear.

Studies in North America and Europe have found a U- or J-shaped association, suggesting that moderate consumption of alcohol provides protection against ischemic stroke.^{6,7,9,11-15} However, the definition of moderate consumption has differed substantially among studies. Some definitions were based on the frequency of alcohol consumption¹⁶ and others on the amount (in grams or units per day).¹⁵ In various studies, the categories associated with the lowest risk of ischemic stroke were 1 to 150 g per week,¹⁵ 1 to 33 g per day,¹⁷ 1 to 10 units per week,¹⁸ two drinks per day,^{4,6} and consumption of alcohol less than once a day.¹⁶ The reports of a protective effect of drinking with respect to cardiovascular and cerebrovascular dis-

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ease were criticized, since most of the studies compared alcohol drinkers with nondrinkers. The latter group might have included people who abstained from alcohol because of poor health.^{3,19-21} However, subsequent studies that excluded "sick quitters" from the analysis found the same J-shaped relation.²²⁻²⁴

Using data from the Physicians' Health Study, we prospectively examined the associations between alcohol consumption and stroke and evaluated potential modifications of these relations by other risk factors for stroke.

METHODS

Study Design

The Physicians' Health Study²⁵ was a randomized, double-blind, placebo-controlled trial designed to test the effect of low-dose aspirin on the risk of cardiovascular disease and the effect of beta carotene on the risk of cancer.²⁶ Briefly, 22,071 U.S. male physicians, who were 40 to 84 years of age at the time of enrollment in 1982, were randomly assigned to receive aspirin, beta carotene, both agents, or placebo according to a two-by-two factorial design. All participants reported at base line that they did not have a history of stroke, transient ischemic attack, myocardial infarction, active liver disease, or peptic ulcer disease. In January 1988 the aspirin component of the study was terminated early because of a statistically significant 44 percent reduction in the risk of a first myocardial infarction among physicians in the aspirin group.²⁵ The beta carotene component of the study continued until its scheduled termination on December 31, 1995. This report includes data available as of October 1995, when the participants had been followed for an average of 12.2 years. Follow-up data on morbidity and mortality were available for more than 99 percent of the study participants.

Information was collected at base line by means of a mailed questionnaire, which included questions about alcohol consumption; age; height; weight; systolic and diastolic blood pressure; history of angina pectoris, diabetes mellitus, and hypertension; and cigarette smoking. At base line, 21,870 of the physicians responded to the following question about alcohol consumption: "How often do you usually consume alcoholic beverages (beer, wine, or liquor)?" The possible responses were rarely or never, one to three per month, one per week, two to four per week, five or six per week, one per day, or two or more per day.²⁷ These responses were interpreted as the number of drinks consumed per unit of time. Alcohol consumption was reassessed at 84 months of follow-up, and a high correlation was found between these responses and the responses at base line. Systolic and diastolic blood pressure were self-reported blood-pressure levels at base line. Validation studies of physician-measured blood pressure show a high correlation with self-reports of systolic pressure ($r=0.72$) and diastolic pressure ($r=0.60$, $P<0.001$ for both comparisons).²⁸

Every six months for the first year and annually thereafter, the participants were mailed questionnaires requesting information about their compliance with the randomized treatment assignment and about newly diagnosed conditions, including stroke and transient ischemic attack. Deaths were usually reported by family members or by the postal authorities and were verified by a review of all available medical records, death certificates, and eyewitness accounts, if relevant. Only first cases of stroke were counted. A diagnosis of stroke was confirmed only after medical records and all other available information had been reviewed by the End Points Committee (consisting of two internists, one cardiologist, and one neurologist, who were unaware of the treatment assignments). A stroke was defined as a focal neurologic deficit of vascular mechanism that lasted more than 24 hours. Strokes were classified according to the probable mechanism (ischemic or hemorrhagic) on the basis of medical records, reports of brain imaging, and the judgment of the neurologist on the End Points Com-

mittee. There was a high level of interobserver agreement in the diagnosis of hemorrhagic and ischemic stroke throughout the study.²⁹ Cases of fatal stroke were documented by evidence of a cerebrovascular mechanism obtained from all available sources, including death certificates and hospital records.

Statistical Analysis

Since only 674 physicians reported that they consumed alcohol two or more times a day, this highest category of alcohol intake was combined with the second highest category (once a day). The categories rarely or never and one to three times a month were also combined to form a larger reference group. Cox proportional-hazards models³⁰ were used to estimate the relative risk of stroke associated with alcohol consumption. We calculated relative risks for the total group of participants with stroke (ischemic or hemorrhagic, hereafter referred to as total stroke) and for participants with each subtype of stroke when age, systolic blood pressure, smoking, body-mass index (defined as the weight in kilograms divided by the square of the height in meters), exercise, history of diabetes, current treatment for hypertension, and randomized treatment assignment were controlled for. Tests of linear trend were performed by using each category as an ordinal variable in the proportional-hazards models. The assumption of proportional hazards was tested and was not violated. Age, body-mass index, smoking, systolic blood pressure, and exercise were analyzed for their effect on the association between alcohol consumption and stroke. For this analysis, an interaction term for alcohol consumption and each variable was included in separate models.

RESULTS

During an average of 12.2 years of follow-up, 679 first strokes were reported (557 ischemic strokes, 88 hemorrhagic strokes, and 34 strokes of unknown type). Table 1 shows base-line characteristics of the participants according to categories of alcohol consumption. The mean systolic and diastolic blood pressures were lowest among participants who had one to four drinks per week and were highest among those in the two highest categories of alcohol consumption. The proportion of participants with hypertension was higher among those in the two highest categories of alcohol consumption than among those in the other categories. The percentage of men who had never smoked decreased consistently with increasing alcohol intake. The percentage of current smokers was significantly higher among the men who had at least one drink per day than among those who consumed less alcohol. The percentage of men who were overweight (body-mass index, ≥ 27.8) decreased with increasing alcohol intake.

The overall effect of alcohol consumption on the risk of total stroke, ischemic stroke, and hemorrhagic stroke is shown in Table 2. The men who had less than one drink per week were chosen as the reference group. After adjustment for major risk factors for stroke, there were significant reductions in the risk of total stroke (21 percent) and ischemic stroke (23 percent) among the men who had one or more drinks a week. No statistically significant association was observed between alcohol consumption and hemorrhagic stroke.

The relative risk of total stroke according to the level of alcohol consumption is shown in Table 3. After

TABLE 1. BASE-LINE CHARACTERISTICS OF THE STUDY PARTICIPANTS ACCORDING TO ALCOHOL CONSUMPTION.

CHARACTERISTIC	ALCOHOL CONSUMPTION (NO. OF DRINKS)				
	<1/WK (N=5696)	1/WK (N=3060)	2-4/WK (N=4900)	5 OR 6/WK (N=2771)	≥1/DAY (N=5443)
Mean age (yr)	53.2	51.6	51.9	52.7	55.5
Blood pressure (mm Hg)					
Systolic	125.6	125.3	125.3	126.0	128.1
Diastolic	78.6	78.5	78.4	78.9	79.7
Hypertension (%)*	11.4	10.7	10.2	12.3	14.7
Smoking (%)					
Never smoked	62.8	55.9	49.6	43.7	35.1
Smoked in the past	28.0	34.9	40.8	45.6	49.3
Currently smokes	9.2	9.2	9.6	10.7	15.6
Exercise frequency per week (%)					
<1 time	33.1	28.5	24.1	23.9	27.0
1-4 times	50.4	58.0	59.8	57.7	56.3
≥5 times	16.5	13.5	16.1	18.4	16.7
Body-mass index ≥27.8 (%)†	16.5	16.6	13.4	12.5	11.5
Diabetes (%)	3.8	2.5	1.4	1.2	2.3
Angina pectoris (%)	1.5	1.4	0.9	1.2	1.3
Random assignment to aspirin group (%)	49.2	51.2	49.9	50.8	49.9
Random assignment to beta carotene group (%)	49.3	51.0	49.4	52.0	49.7

*Hypertension was defined as self-reported systolic blood pressure of 160 mm Hg or higher, diastolic blood pressure of 95 mm Hg or higher, or current treatment for hypertension (regardless of blood pressure).

†Body-mass index was defined as the self-reported weight in kilograms divided by the square of the height in meters.

adjustment for age and treatment assignment, the largest risk reductions were found among the men who had one to four drinks per week. Further adjustment for systolic blood pressure, current treatment for hypertension, smoking, body-mass index, diabetes, and exercise revealed an L-shaped, almost flat association, with similar risk reductions of about 20 percent for all categories of alcohol consumption (Table 3 and Fig. 1).

The relative risk of subtypes of stroke, after adjustment for important confounders, is shown in Table 4. Reductions in the risk of ischemic stroke were similar in magnitude to those in the risk of total stroke and were of borderline significance. Alcohol intake was not significantly associated with the risk of hemorrhagic stroke.

In secondary analyses (data not shown), we examined the effects of other risk factors for stroke on the

TABLE 2. RELATIVE RISK OF ISCHEMIC OR HEMORRHAGIC STROKE (TOTAL STROKE) AND SUBTYPES OF STROKE ACCORDING TO ALCOHOL CONSUMPTION.*

ALCOHOL CONSUMPTION	TOTAL STROKE		ISCHEMIC STROKE		HEMORRHAGIC STROKE				
	NO. OF CASES	RELATIVE RISK (95% CI)	NO. OF CASES	RELATIVE RISK (95% CI)	NO. OF CASES	RELATIVE RISK (95% CI)			
		<i>adjusted for age and treatment†</i>	<i>multivariate adjusted‡</i>	<i>adjusted for age and treatment†</i>	<i>multivariate adjusted‡</i>	<i>adjusted for age and treatment†</i>	<i>multivariate adjusted‡</i>		
<1 drink/wk	206	1.00	1.00	168	1.00	1.00	26	1.00	1.00
≥1 drink/wk	473	0.83 (0.71-0.98)	0.79 (0.66-0.94)	389	0.84 (0.70-1.01)	0.77 (0.63-0.94)	62	0.84 (0.53-1.33)	0.92 (0.55-1.54)

*The mean period of follow-up was 12.2 years. CI denotes confidence interval. Men who consumed less than one drink per week served as the reference category.

†Values have been adjusted for age (in years) and treatment assignment (aspirin, yes or no; beta carotene, yes or no).

‡Values have also been adjusted for systolic blood pressure (continuous values), current treatment for hypertension, smoking (four categories), history of diabetes (yes or no), body-mass index (in quartiles), and exercise (four categories).

TABLE 3. RELATIVE RISK OF ISCHEMIC OR HEMORRHAGIC STROKE (TOTAL STROKE), ACCORDING TO ALCOHOL CONSUMPTION.*

ALCOHOL CONSUMPTION	NO. OF CASES	TOTAL STROKE			
		RELATIVE RISK ADJUSTED FOR AGE AND TREATMENT (95% CI)†	P VALUE‡	MULTIVARIATE ADJUSTED RELATIVE RISK (95% CI)§	P VALUE‡
<1 drink/wk	206	1.00	0.51	1.00	0.07
1 drink/wk	70	0.75 (0.57–0.98)		0.78 (0.59–1.04)	
2–4 drinks/wk	111	0.74 (0.59–0.94)		0.75 (0.58–0.96)	
5 or 6 drinks/wk	83	0.90 (0.70–1.16)		0.83 (0.62–1.11)	
≥1 drink/day	209	0.90 (0.74–1.09)		0.80 (0.64–0.99)	

*CI denotes confidence interval. Men who consumed less than one drink per week served as the reference category.

†Values have been adjusted for age (in years) and treatment assignment (aspirin, yes or no; beta carotene, yes or no).

‡P values are for linear trend across all categories of alcohol consumption.

§Values have also been adjusted for systolic blood pressure (continuous values), current treatment for hypertension, smoking (four categories), history of diabetes (yes or no), body-mass index (in quartiles), and exercise (four categories).

relation between alcohol intake and stroke. No interactions with age, body-mass index, or smoking were found. We found significant interactions with systolic blood pressure ($P=0.02$) and exercise ($P=0.03$). An analysis stratified according to systolic blood pressure (<130, 130 to 139, or ≥ 140 mm Hg) showed a risk reduction only among participants with blood pressure of 140 mm Hg or higher (relative risk for men who had one to six drinks per week, as compared with those who had less than one drink per week, 0.57; 95 percent confidence interval, 0.42 to 0.79; relative risk for men who had one or more drinks per day, 0.54; 95 percent confidence interval, 0.39 to 0.75). With respect to exercise, drinking had a beneficial ef-

fect only in the group of physicians who exercised at least once a week.

DISCUSSION

The focus of this analysis was the relation between light-to-moderate alcohol consumption and the risk of stroke. The Physicians' Health Study offered the opportunity to evaluate the relatively narrow range of alcohol intake of one to seven drinks per week (i.e., light-to-moderate intake, accounting for about 97 percent of our sample²⁷), corresponding to the beneficial part of the U- or J-shaped relation between alcohol consumption and stroke that other studies have suggested. We found an L-shaped association between alcohol intake and total stroke, with a risk reduction of about 20 percent after adjustment for important confounders. The shape of this relation corresponds to the first part of the J-shaped curve shown by other studies.^{3,6} The benefit was observed among physicians who had only one alcoholic drink per week, and the magnitude of this protective effect did not increase with increased alcohol consumption. The relation between alcohol consumption and ischemic stroke had the same flat shape, and the risk reductions were similar in magnitude.

The protective effect of light-to-moderate drinking with respect to total stroke or ischemic stroke has varied considerably, with risk reductions of 20 to 90 percent found in other studies.^{3,4} A recent population-based case-control study reported an odds ratio of 0.51 for ischemic stroke in the group with moderate alcohol consumption.⁶ Most cohort studies have reported risk reductions of 20 to 40 percent. Our result is similar in magnitude. However, our population dif-

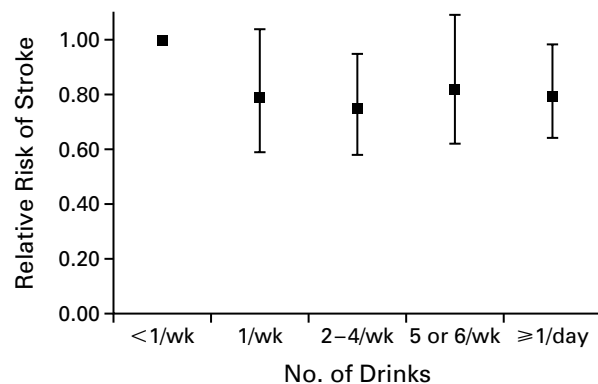


Figure 1. Relative Risk of Ischemic or Hemorrhagic Stroke (Total Stroke), According to Alcohol Consumption.

The reference category of alcohol consumption was less than one drink a week. Bars denote 95 percent confidence intervals.

TABLE 4. RELATIVE RISK OF SUBTYPES OF STROKE ACCORDING TO ALCOHOL CONSUMPTION.*

ALCOHOL CONSUMPTION	ISCHEMIC STROKE			HEMORRHAGIC STROKE		
	NO. OF CASES	RELATIVE RISK (95% CI)	P VALUE	NO. OF CASES	RELATIVE RISK (95% CI)	P VALUE
<1 drink/wk	168	1.00	0.10†	26	1.00	0.67†
1 drink/wk	54	0.73 (0.52–1.00)		13	1.17 (0.58–2.40)	
2–4 drinks/wk	91	0.74 (0.56–0.98)		14	0.70 (0.33–1.46)	
5 or 6 drinks/wk	68	0.81 (0.59–1.12)		11	1.00 (0.46–2.23)	
≥1 drink/day	176	0.79 (0.62–1.00)		24	0.90 (0.48–1.69)	

*Values have been adjusted for age (in years), randomized treatment assignment (aspirin, yes or no; beta carotene, yes or no), systolic blood pressure (continuous values), current treatment for hypertension, smoking (four categories), history of diabetes (yes or no), body-mass index (in quartiles), and exercise (four categories). CI denotes confidence interval. Men who consumed less than one drink per week served as the reference category.

†P values are for linear trend across all categories of alcohol consumption.

fers from that in other studies in that it consists of healthy men of high socioeconomic and educational status. Their risk-factor profile differs substantially from that of men in the general U.S. population. In particular, only 3.1 percent of the participants in our study reported that they had more than one drink per day,²⁷ confirming that heavy drinking was very rare in this population. For this reason, it is not possible to draw any conclusions about the participants who reported having two or more drinks per day.

Like other studies involving predominantly white populations, our study showed that the majority of strokes were ischemic; only 13 percent were hemorrhagic. This finding explains the similarity of the relations of alcohol consumption with total stroke and with ischemic stroke.

In prior studies, the beneficial effect of alcohol on the risk of stroke was observed over a rather broad range of alcohol intake (e.g., 1 to 150 g per week¹⁵), as a consequence of defining categories broadly or combining them to provide stable estimates in the analysis. In contrast, our analysis had the power to examine risks within five narrowly defined categories of alcohol consumption, with four of them corresponding to the beneficial part of the suggested associations. With this approach, we found that alcohol had a beneficial effect in men who had as little as one drink per week, a finding that would have been masked if we had used broader categories. The flat shape of the association with total stroke also suggests that the nadir of the observed benefit is not a single category of alcohol consumption but rather a range of consumption from one to seven drinks per week.

Our study has a number of limitations. First, there may have been some incorrect reports of alcohol consumption by the participants at base line, leading to a certain degree of misclassification of this variable.

However, since all information was assessed at base line, before the occurrence of stroke, any misclassification due to an incorrect report was nondifferential.³¹ In addition, studies have shown that the reliability of self-reported alcohol consumption in the general population, including health care professionals, is good^{32,33} and also that physicians accurately report their own cardiovascular risk factors.^{28,34} Furthermore, both high-density lipoprotein (HDL) cholesterol and blood pressure, measures known to be correlated with levels of alcohol use, had the expected associations with the reported amounts of alcohol consumption in the Physicians' Health Study,²⁷ providing further evidence of the reliability of self-reporting in this cohort.

Nonetheless, the possibility of systematic underreporting of alcohol consumption must be considered. If it occurred, the observed association between alcohol use and reduced risk of stroke would be artificially shifted toward lower drinking categories, leading to an erroneous, lower nadir for the relation between alcohol intake and the risk of stroke. However, since we observed a beneficial effect at a very low level of alcohol intake (one drink a week), a slight shift of the curve to the right would still reveal an effect over a range of moderate levels of alcohol intake.

Misclassification due to a change in the level of alcohol consumption after base line is also possible. However, if there was such an effect in this cohort, it was probably small, since we found a high correlation between alcohol consumption at base line and at 84 months.

The use of persons who never drank as the reference group in epidemiologic studies of the health effects of alcohol consumption is often questioned, because some of these abstainers might have given up drinking for reasons of health. However, the physi-

cians in our study reported at base line that they had no history of myocardial infarction, transient ischemic attack, or stroke and that they were free of active liver disease and peptic ulcer, conditions that could lead to abstinence from drinking alcohol.

Finally, the questionnaire asked about the frequency of drinking, but the participants may have consumed more than one drink on a particular occasion. However, since the nadir of the risk reduction was observed over a range of categories of moderate drinking, rather than a single category, the shape of the association would not be affected.

We could not assess the possibility of a differential effect of specific types of alcohol on the risk of stroke, as some studies have suggested,^{35,36} since the questionnaire did not ask about the type of beverage. We also had no information about drinking patterns. Since binge drinking may be a risk factor for stroke, especially for hemorrhagic stroke in young adults,³⁷⁻³⁹ this risk could not be specified. However, since our cohort consisted of middle-aged and elderly men of high socioeconomic status, a group with a low incidence of binge drinking,⁴⁰ any effect of such drinking would probably have been small. Finally, the possibility of residual confounding was substantially reduced by our use of a cohort of physicians that was homogeneous with respect to education and socioeconomic status.

The effects of alcohol on the body are complex and involve a number of biologic mechanisms. Moderate alcohol consumption has an inverse relation with arteriosclerosis of the large arteries of the circle of Willis and the carotid arteries^{41,42} but not with arteriosclerosis of the small, intracerebral arteries.⁴³ This effect could be due to a reduction of Lp(a) lipoprotein, since Lp(a) lipoprotein levels are related to the extent of carotid arteriosclerosis.⁴⁴ Elevated levels of Lp(a) lipoprotein are a risk factor for coronary heart disease,⁴⁵⁻⁴⁷ have been found to be elevated in survivors of stroke,⁴⁸ and are reduced by alcohol intake.⁴⁹ In addition, there is evidence that vascular endothelial factors, especially nitric oxide, provide protection against atherosclerosis.^{50,51} In experimental studies, the activity of nitric oxide at the vessel wall was enhanced by ethanol through the induction of the enzyme nitric oxide synthase.⁵²

Another explanation for the effect of alcohol on the risk of stroke is the finding that alcohol increases HDL cholesterol levels,^{53,54} which are inversely related to arteriosclerosis at many sites, including the carotid arteries.^{41,55} HDL cholesterol levels are lower in survivors of stroke than in controls.⁵⁶⁻⁵⁸ However, changes in HDL cholesterol levels only partially explain the inverse relation between alcohol consumption and death from cardiovascular causes.⁵³ Furthermore, a recent study⁶ suggested that the observed association between moderate alcohol consumption and a reduced risk of stroke was independent of the HDL cholesterol level.

These findings suggest that hemostasis plays a part in the protective effect of alcohol on the risk of stroke. Alcohol alters coagulation in a number of ways. It reduces platelet aggregation^{59,60} by increasing the ratio of prostacyclin to thromboxane,⁶⁰ and it decreases the aggregation and increases the deformability of red cells.⁶¹ In addition, alcohol decreases the risk of clotting by reducing fibrinogen levels and increasing the levels of tissue plasminogen activator.⁶²⁻⁶⁴ The latter effect is paradoxical, since elevated levels of tissue plasminogen activator are correlated with an increased risk of ischemic stroke,⁶⁵ yet alcohol consumption, although it reduces the risk of stroke, is associated with increased plasma levels of tissue plasminogen activator.⁶³ Although elevated levels of tissue plasminogen activator increase the risk of ischemic stroke, with moderate alcohol use the deleterious effect of tissue plasminogen activator is probably outweighed by the other beneficial effects of alcohol on HDL cholesterol, Lp(a) lipoprotein, platelet aggregation, and fibrinogen, resulting in a net benefit of alcohol use. Thus, the relative magnitude of the effect of the individual factors, both positive and negative, and their interplay probably determine the ultimate effect of alcohol on the risk of stroke.

In summary, light-to-moderate consumption of alcohol (one to seven drinks per week) reduced the risks of total stroke and of ischemic stroke in a cohort of healthy, predominantly white physicians. Although this finding may be important for persons who consume alcoholic beverages with low or moderate frequency, no generalizations about the benefits of a change in lifestyle should be drawn from the observation. The risk reduction associated with such a change is clearly smaller than that associated with medical interventions, such as treatment for hypertension. Moreover, aside from the serious morbidity and social problems related to alcohol use, it has been shown that in a large population, any increase in alcohol consumption is associated with a proportionate increase in heavy drinking.⁶⁶ Heavy drinking is a risk factor for hemorrhagic and ischemic stroke,^{3,6,17} as well as for morbidity and mortality from other causes; therefore, in the words of Marmot and Brunner, "Any public health recommendation that emphasizes the positive aspects of alcohol would be likely . . . to do more harm than good."²²

Supported by grants from the National Institutes of Health (CA 34944, CA 40360, HL 26490, and HL 34595) and by a grant from the German Academic Exchange Service (to Dr. Berger).

We are indebted to the staff of the Physicians' Health Study and to the dedicated and conscientious physicians who participated in this trial.

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