Alcohol, Tobacco, and Hypertension
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SUMMARY In many studies of diverse populations it has been found that persons drinking relatively large amounts of alcohol tend to have higher blood pressures. In the Kaiser-Permanente study of about 87,000 persons, this alcohol-blood pressure association was not attributable to demographic characteristics, adiposity, reported salt use, smoking, or coffee consumption, nor could it be explained by underreporting of alcohol consumption. If the relationship is a causal one, the pathogenesis is not yet established; direct mechanisms or the effects of withdrawal from alcohol are possible explanations. The Kaiser-Permanente data suggest that about 5% of hypertension in the general population may be due to the consumption of three or more alcoholic drinks per day. Alcohol use shows a positive relation to some sequelae of hypertension but not others; the outstanding exception is coronary heart disease which is negatively related to alcohol intake, probably through different mechanisms. In most studies, cigarette smokers have shown similar or slightly lower blood pressures than nonsmokers. The degree to which this is due to the thinner body build of smokers, on the average, is not well established; nor is the degree to which a stronger negative relation of smoking to blood pressure might be masked by concomitant alcohol use. (Hypertension 4 (supp III): III-143-III-150, 1982)

KEY WORDS • blood pressure • ethanol • epidemiology • cigarette smoking

THE relation of alcohol use to hypertension and to other forms of cardiovascular disease has become a major question for both medical and public health research in recent years. Cigarette smoking is associated with alcohol use in our society. Since cigarette smoking has pharmacological effects that might be expected to influence blood pressure, the question of a smoking-blood pressure association also deserves exploration.

In this investigation we consider:
1. The epidemiological evidence linking alcohol use to blood pressure
2. The associated or potentially confounding factors that must be considered
3. Possible mechanisms or explanations involved in the alcohol-hypertension link
4. The effects of alcohol on the outcomes of hypertension
5. Potential public health implications
6. The relation between smoking and blood pressure.

Alcohol and Hypertension: Epidemiologic Evidence

It has been found in many studies, both in this country and in others, that persons in the higher range of alcohol consumption tend to have higher blood pressures.1-10 We shall not present a complete review of the literature, but a few examples will be illustrative.

The well-known Framingham study has involved a careful initial evaluation and long-term follow-up of over 5000 middle-aged men and women, initially aged 30-59 years. Although the overall correlation between alcohol intake and blood pressure in this study population was small, the prevalence of hypertension, defined as a systolic pressure ≥ 160 or diastolic ≥ 95 mm Hg, was about twice as high among persons drinking 60 ounces or more of ethyl alcohol per month as among those drinking less than 30 ounces per month. These lighter drinkers had slightly lower blood pressures than nondrinkers.1

In a study of 922 “problem drinkers” among employees of the E.I. du Pont de Nemours and Co., Inc., and matched comparison subjects consisting of nondrinkers and moderate drinkers, hypertension defined as above was over twice as frequent in the problem drinkers. Interestingly, the relation appeared to be partially reversible in that recovered problem drinkers had a lower prevalence of hypertension than those whose drinking problem was current.5
A different type of study population in a different setting consisted of 70 male twin pairs in Sweden who were discordant for drinking, i.e., either only one twin in each pair used substantial amounts of alcohol, or one twin drank much more than the other. Here again the users of more alcohol had higher casual and basal blood pressures on the average than their co-twins. Although most of the twins were dizygotic, the findings suggested at least some independence of the alcohol-blood pressure relation from genetic factors.

On the other side of the world, 85 active working men in Tasmania showed a direct correlation of both systolic and diastolic blood pressures with alcohol consumption up to 240 g/week. Multivariate analysis showed alcohol consumption to be an important predictive variable for blood pressure.

To our knowledge, our own cross-sectional investigation of this question has involved the largest study group, 87,000 ambulatory adult subscribers to the Kaiser-Permanente Medical Care Program, a prepaid health care plan. Our subjects lived in the San Francisco Bay area and received a multiphasic health checkup between 1964 and 1968 at the program's medical center in Oakland or San Francisco. In this checkup, alcohol use data (as assessed by questionnaire) was computer-stored along with many other health-related variables including blood pressure measured by an automated device. The study population was characterized by ethnic and socioeconomic diversity.

Figure 1 shows the age-adjusted mean systolic and diastolic blood pressure in each race-sex group according to usual number of drinks per day as reported on questionnaire. Among both men and women, there was a steady rise in blood pressure as consumption increased beyond two drinks per day except among blacks where the maximum blood pressure was usually found in the three- to five-drink category. Among the women of all races, there was a clear tendency for consumers of two or fewer drinks per day to have lower blood pressures than nondrinkers. In general, the trends in systolic pressure were more pronounced than those in diastolic pressure.

Not shown in figure 1 was a further breakdown of the 6+ drink group into a 6-8 drink and 9+ drink subgroup. The 9+ subgroup was relatively small and did not show an increase in mean blood pressure beyond that of the 6-8 drink subgroup.

We also determined the prevalence of hypertension in each alcohol consumption category (fig. 2), again using the 160/95 breakpoint. In white men and women, the prevalence of hypertension was approximately twice as great in the 6+ drink group as in the combined two or fewer and nondrinking categories. Among blacks, the 3+ drink group had an approximately 50% higher prevalence of hypertension.

**Potentially Confounding Factors**

**Adiposity**

Adiposity has proven to be an important predictor or determinant of hypertension. It is one of the first characteristics that should come to mind when trying to...
explain an association between the ingestion of any item and hypertension. In this conference concerned with aspects of nutrition in relation to blood pressure, we should always ask ourselves whether a higher level of intake of any particular item might be merely an index for higher overall food intake in relation to energy expenditure, rather than the culprit itself. On the other hand, since the relation of obesity to blood pressure is not fully understood, we should also be open to the possibility that obesity is a marker for the consumption of one or more of the key substances that raise blood pressure.

At any rate, we did subdivide our study population into thirds according to their body mass (Quetelet) index (wt/ht²). In each tertile of adiposity, the association of alcohol intake with mean blood pressure was apparent (fig. 3).

Reported Indiscriminate Salt-Shaker Use

An item on the multiphasic questionnaire read as follows: "Do you usually salt your food before tasting it?" It is debatable whether "yes" answers always represent higher levels of salt intake, but lacking salt excretion measurements we felt that, on the average, people who affirmed this behavior ingested more salt than those who denied it. We also found that there was a strong association of "yes" answers to this question with alcohol intake. Among subjects subdivided by their answers to this salt intake question, the relation of alcohol intake to blood pressure persisted (fig. 4). It can be seen that not only did this questionnaire item fail to explain away the alcohol-blood pressure relation but it has no discriminating ability for blood pressure level.

Other Characteristics

As has been shown, the alcohol-blood pressure relation was present in each race and sex, and our data were age-adjusted. Thus, the demographic characteristics of age, sex, and race were not responsible for the observed correlation. We also subdivided the study group by smoking habits and coffee consumption, two characteristics associated with alcohol use; we found that the alcohol-blood pressure relation persisted.

Possible Explanations

Underreporting Alcohol Use

We bring up underreporting of alcohol consumption as a potential source of bias, first to dispense with it as soon as possible, since it is repeatedly and incorrectly cited as a reason for disbelief of our findings. We all know that some persons consuming relatively large quantities of alcohol are embarrassed by this and report lesser amounts. But what kind of bias does this introduce into our studies?

Let us first consider the hypothetical case in which there is no real relation of alcohol use to blood pressure. For simplicity we will assume that there are only two categories of drinkers, heavy and light. If, because of false reporting, some truly heavy drinkers are incorrectly placed in the light category, the mean blood pressure among these truly heavy drinkers will be lower than the mean of the actual heavy drinkers from whom they were diverted. Similarly, the mean among the truly light drinkers will be higher than the mean of the actual light drinkers to whom they were diverted.
pressure of the heavy group will not change, and since heavy drinkers incorrectly placed in the light drinking category had the same blood pressure as the light drinkers, the mean blood pressure of the light drinking group will not change. Thus, the heavy and light drinking groups will have the same mean blood pressure even after some of the truly heavy drinkers put themselves in the light drinking group. Only if false reporting is associated with low blood pressure, which to our knowledge has never been demonstrated, would this kind of misclassification produce as an artifact the association that has been observed.

Figure 3. Age-adjusted mean systolic and diastolic blood pressure according to degree of adiposity: white men and women. (Reproduced, by permission, from the New England Journal of Medicine (see ref. 9).

Figure 4. Age-adjusted mean systolic and diastolic blood pressure according to whether the subjects (white men and women) salt their food before tasting it.
Now consider there to be a true difference in blood pressure between heavy and light drinkers. If some heavy drinkers erroneously put themselves in the light drinking category, the reported light drinkers will become more like the remaining reported heavy drinkers. That is, the apparent difference between the two groups will be reduced.

Thus, if any bias is introduced by the underreporting of alcohol consumption, this will not be in the direction of creating an association with blood pressure that is not really there, but will rather be to reduce a true association. We wish that many of those who raise this question would not just assume that large population studies relying on questionnaire assessments of alcohol use are invalid, but would follow the results of the likely reporting errors to their logical conclusion.

We believe that in our relatively nonjudgmental medical setting, the degree of underreporting is not great. Our questionnaire data produced an alcohol-drinking distribution similar to that observed in a national survey based on careful personal interviews. For those who still do not believe that what people say is valid unless backed up by laboratory data, there is an interesting recent report from Belgium of a study of blood pressure in over 9000 men who not only reported their alcohol consumption but also had measurements of gamma-glutamyl transpeptidase, a biochemical index of alcohol consumption. Both the consumption reported and the enzyme measure were associated with blood pressure. Two studies from the Glasgow, Scotland area in 1977 also showed associations of liver function test abnormalities with hypertension and there was evidence that alcohol consumption was the common explanatory factor.

Withdrawal

Another question that has been raised is whether the higher blood pressures in drinkers are largely due to withdrawal from alcohol rather than alcohol itself. It may be that a substantial number of heavy drinkers abstain from alcohol for a day or so in preparation for our multiphasic checkup or the other study examinations that have found this association. We agree that this is an important question that has not yet been answered. For example, the Lipid Research Clinics presented preliminary data purportedly supporting the withdrawal hypothesis; drinkers who, in a separate dietary recall, reported drinking alcohol during the prior 24 hours had slightly higher blood pressures than drinkers of similar amounts who denied intake in the prior 24 hours (difference not statistically significant). It is not clear to us that the former group was more withdrawn from alcohol than the latter when examined.

The withdrawal issue was put into sharper focus by the study of Saunders et al. of 96 alcoholics admitted to medical wards in Birmingham, England, for detoxification. At the time 48% were hypertensive and the blood pressure level was related to the severity of alcohol-withdrawal symptoms. Most of the hypertensives became normotensive when the withdrawal symptoms cleared. After discharge, blood pressure rose only in those who started drinking again. These investigators suggested that alcohol-related hypertension may be the result of the alcohol-withdrawal syndrome possibly due to increased "noradrenergic" activity. Thus, we can now see that the withdrawal question is really twofold. One question has to do with whether the alcohol-blood pressure association is really an artifact: Could it be that alcohol drinkers do not really have higher blood pressures most of the time, but only when they are temporarily in a state of withdrawal at medical examinations? The second question is: Given a true association, is withdrawal the underlying mechanism? It may be that withdrawal does raise blood pressure both acutely as in the detoxification ward, and, of greater concern, chronically through the long-term effects of repeated transient blood pressure increases leading to sustained hypertension. This should be a fruitful area of clinical research in the years to come.

Psychological Stress

It is also reasonable to ask whether psychological or emotional stress is not the underlying factor. Perhaps this leads both to increased alcohol intake and to raised blood pressure. This is an area in need of further investigation. If it turns out that emotional factors are important in producing sustained blood pressure elevations in the general population, the role of alcohol in this outcome would still need to be defined. Is increased alcohol use merely a marker for psychological problems, having no direct effect on blood pressure, or does emotional stress raise blood pressure, at least in part, through an intervening pharmacological effect of alcohol?

Possible Direct Mechanisms

The acute effects of small doses of ethanol (in the range of 30 to 75 ml of alcohol or 2½ to 6 ounces of 80 proof liquor), were studied decades ago. These produce a slight transient increase in heart rate and cardiac output with some increase in blood pressure, particularly systolic. Significant effects on blood pressure are not always found, however. Although cutaneous vascular resistance decreases, producing a flushed appearance, there is little change in total peripheral resistance because of a compensatory vasoconstriction in the viscera. Large doses, sufficient to produce central respiratory depression, also depress the cardiovascular system leading to hypotension and bradycardia through a combination of reflex mechanisms and myocardial effects. The chronic physiologic effects of alcohol are not well understood and need further study. At this stage it is difficult to muster physiological evidence either to support or refute the notion that chronic alcohol use raises blood pressure. Possible mechanisms that have been suggested are those mediated by the central or autonomic nervous systems, direct effects on the heart or blood vessels, chronic increases in corticosteroid levels, and a chronic hypermetabolic state.
Alcohol and the Outcomes of Hypertension

Two of the main complications of hypertension, coronary heart disease and stroke, have been studied in relation to alcohol consumption. The results have been of great interest in recent years, particularly for coronary disease. Most of these studies have found a negative association between alcohol use and coronary heart disease. This may at first seem surprising when it is remembered that alcohol is positively associated with two major coronary risk factors, cigarette smoking and hypertension. There are various epidemiologic methods for dissecting out relationships, and when these are applied the confounding effects of smoking and hypertension can be removed.

First, consider the outcome of our study of four groups of multiphasic examinees. We attempted to match each of the 2084 persons in our original large study group who reported six or more drinks per day with one who drank three to five drinks per day, one who drank two or fewer drinks per day and a non-drinker. Matching was for age, sex, race, cigarette smoking, and date of examination. Hospital follow-up was accomplished by computer search of magnetic tapes containing the 1971–1976 hospital records of all hospitalizations in our prepaid plan in Northern California.

The incidence of hospitalization for hypertension, all coronary heart disease, acute myocardial infarction and stroke, according to alcohol category, is shown in table 1. The trend of hospitalization for hypertension generally corresponded to that which we observed for hypertension at the multiphasic examination in our cross-sectional data. An inverse relation was observed for coronary disease and one of its major components, acute myocardial infarction. For stroke the correlation with alcohol consumption was direct, as it was for hypertension itself.

We also performed a mortality follow-up on the same subjects. Here the trends for death from complications of hypertension were not as simple as they were for hospitalization. For coronary heart disease, there was a U-shaped curve with the highest death rate observed in the nondrinkers and the lowest in the two-or-fewer drink group. For stroke, the curve was an inverted U with the highest death rate in the 3-5 drink group and the lowest in both the zero and 6+ drink groups. In another mortality study that we conducted, in which the primary focus was on smoking, alcohol drinking was positively correlated with total mortality but showed a slight negative correlation with mortality from coronary heart disease that was not statistically significant.

The Honolulu Heart Program, which has been studying men of Japanese birth or ancestry, also found an inverse correlation of alcohol consumption with the incidence of and mortality from coronary heart disease. This persisted when cigarette smoking, relative weight, and other important coronary risk factors were included in a multivariate analysis. The Honolulu group subdivided their stroke incidence and mortality data into hemorrhagic and thromboembolic categories.

## Table 1. Incidence of Hospitalization for Selected Cardiovascular Conditions in Relation to Reported Alcohol Consumption

<table>
<thead>
<tr>
<th>Reported no. of drinks per day</th>
<th>0</th>
<th>≤ 2</th>
<th>3-5</th>
<th>6+</th>
</tr>
</thead>
<tbody>
<tr>
<td>Person years of observation, 1971–1976</td>
<td>8436</td>
<td>8610</td>
<td>8244</td>
<td>7920</td>
</tr>
<tr>
<td>Number of persons hospitalized</td>
<td>33</td>
<td>40</td>
<td>47</td>
<td>59</td>
</tr>
<tr>
<td>Hypertension</td>
<td>112</td>
<td>84</td>
<td>80</td>
<td>74</td>
</tr>
<tr>
<td>Coronary heart disease</td>
<td>54</td>
<td>46</td>
<td>36</td>
<td>33</td>
</tr>
<tr>
<td>Acute myocardial infarction</td>
<td>26</td>
<td>29</td>
<td>31</td>
<td>33</td>
</tr>
<tr>
<td>Stroke</td>
<td>3.9</td>
<td>4.6</td>
<td>5.7</td>
<td>7.4</td>
</tr>
<tr>
<td>Incidence; persons hospitalized per 1000 person-years</td>
<td>13.3</td>
<td>9.8</td>
<td>9.7</td>
<td>9.3</td>
</tr>
<tr>
<td>Hypertension</td>
<td>6.4</td>
<td>5.3</td>
<td>4.4</td>
<td>4.2</td>
</tr>
<tr>
<td>Coronary heart disease</td>
<td>3.1</td>
<td>3.4</td>
<td>3.8</td>
<td>4.2</td>
</tr>
</tbody>
</table>

Alcohol consumption showed a clear positive correlation with the subsequent development of hemorrhagic stroke but not with the thromboembolic variety. It seems reasonable to interpret this as reflecting the more exclusive relation to hypertension of hemorrhagic than of thromboembolic stroke. Although blood pressure is important in thrombotic strokes, alcohol's metabolic effects may exert a counterbalancing protective influence against the occlusive atherosclerotic process as it might for coronary heart disease. The Honolulu investigators suggested that the stronger relation to hemorrhagic stroke was not entirely explained by effects of alcohol on blood pressure.

Although population studies have suggested a negative relation and possibly protective effect of use of small or intermediate amounts of alcohol against coronary heart disease, the finding is not unanimous with respect to heavier drinking. Most contrasting evidence comes from studies of so-called "problem drinkers," those whose alcohol consumption is relatively quite high and associated with antisocial behavior or difficulties in employment. Problem drinkers studied have been identified among U.S. employees of a public utility and a chemical company and among Swedish twins and a population sample in Sweden. Unfortunately, many of these studies did not control for cigarette smoking. There may be other reasons for this contradiction yet to be identified. In studies of the outcome of alcohol drinking we advocate classifying the subjects by amount of alcohol consumed per day or other time interval whenever possible and avoiding judgmental terms such as "heavy," "moderate," or...
"excessive." Presence or absence of problem behavior is also a characteristic worth noting whenever possible.

**Potential Public Health Implications**

If we assume that it will eventually be shown that alcohol does raise blood pressure chronically, it is worthwhile considering what proportion of hypertension would be due to alcohol ingestion. We can do this in two ways. First, we can determine the proportion of alcohol drinkers who have hypertension because of alcohol, and, second, we can determine the proportion of the entire population who have hypertension because of alcohol.

Altogether, persons drinking three or more drinks per day have a relative risk (r) of having hypertension of about 1.5 compared to persons drinking two or fewer (including none) drinks per day. The fraction of disease in the 3+ drinkers that is attributable to drinking, assuming causality, may be estimated\(^9\) by

\[
\frac{r - 1}{r} = \frac{1}{3}.
\]

Thus, also assuming reversibility, about one-third of hypertensives among these heavier drinkers might benefit if they drank two or fewer drinks per day. From the population standpoint, with p being the proportion of the population drinking three or more drinks per day, the proportion of the entire population who have hypertension because of alcohol might benefit if they drank two or fewer drinks per day. The fraction of the population drinking three or more drinks per day. From the population standpoint, with p being the proportion of the population drinking three or more drinks per day, the proportion of the entire population who have hypertension because of alcohol might benefit if they drank two or fewer drinks per day. The fraction of hypertension attributable to this level of drinking is estimated\(^9\) by

\[
\frac{p(r - 1)}{pr + (1 - p)} = \frac{0.1(1.5 - 1)}{0.15 + 0.9} = 0.05.
\]

Thus, about 5% of the hypertension in our study group might be due to increased alcohol intake. If one has the courage to generalize these estimates, then perhaps over 1 million cases of hypertension in the United States are due to higher levels of alcohol use.

It should be emphasized that the calculations above are based on the assumption, not fully proven, that alcohol is a cause of hypertension rather than being associated in a secondary way. Furthermore, the fact that alcohol may have an opposite effect on a major outcome of hypertension, i.e., coronary disease, must also be factored into estimates of public health effects. Further, this estimate of the so-called population attributable risk is an average; there would be marked variation among different age-sex groups. For example, in our study group about 25% of the white men in their fifties drank three or more drinks per day. Given the same relative risk of 1.5, their population attributable risk would be 11%, or over twice as great as for all subjects combined. Finally, any underestimation of drinking habits would make these estimates conservative by reducing apparent relative risks.

**Cigarette Smoking and Blood Pressure**

Experimental studies on man and animals have shown that an acute effect of smoking or nicotine administration is to produce small increases in blood pressure and heart rate, presumably due to the resulting release of catecholamines.\(^3\) Nevertheless, most epidemiologic studies have shown that the blood pressure of cigarette smokers is similar to or even slightly lower than that of nonsmokers.\(^3\) Perhaps some of this has to do with the fact that cigarette smokers tend to be thinner than nonsmokers, but it should also be remembered that smokers tend to consume more alcohol than nonsmokers, so that a blood-pressure-lowering effect of cigarette smoking, if actually present, might be masked as a result.

Our own data\(^2\) support the paucity of a relation between smoking and blood pressure (table 2). The mean diastolic blood pressure was 1 mm Hg higher in nonsmokers than smokers in each sex-race group. Among women, the mean systolic measure was 1 or 2 mm Hg higher in nonsmokers; among men, it was 1 or 2 mm Hg lower in nonsmokers. In comparing persons who quit with persons who persisted in cigarette smoking, the mean blood pressure changes between successive examinations showed only small disparities that were in different directions in various race-sex groups.\(^3\) To better assess the net chronic effects of smoking on blood pressure, more studies are needed in which the associated factors, alcohol use and body build, are taken into account.

Of interest is the possibility that chewing tobacco may have a different effect on blood pressure than smoking it. In a study of male college students in Texas,\(^3\) 22 tobacco chewers had mean systolic and diastolic pressures respectively, 9 and 4 mm Hg higher than 69 nonchewers of tobacco (p < 0.01). Drinking habits were not mentioned. If we may conclude by briefly digressing from science to esthetics, it is reassuring to note that California, not Texas, is the main source of social trends among U.S. youth.

### Table 2. Age-Adjusted Mean Blood Pressure (mm Hg) by Race, Sex, and Smoking Status

<table>
<thead>
<tr>
<th></th>
<th>Black women</th>
<th>White women</th>
<th>Black men</th>
<th>White men</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>S</td>
<td>N</td>
<td>S</td>
<td>N</td>
</tr>
<tr>
<td>Total number</td>
<td>1000</td>
<td>1546</td>
<td>4359</td>
<td>7956</td>
</tr>
<tr>
<td>Systolic BP</td>
<td>136</td>
<td>137</td>
<td>128</td>
<td>130</td>
</tr>
<tr>
<td>Diastolic BP</td>
<td>87</td>
<td>88</td>
<td>80</td>
<td>81</td>
</tr>
</tbody>
</table>

S = smokers; N = nonsmokers; BP = blood pressure.
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