Effects of Alcohol Use and Other Aspects of Lifestyle on Blood Pressure Levels and Prevalence of Hypertension in a Working Population

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SUMMARY The relationship between alcohol consumption and blood pressure was studied in 491 males, ages 20–45 years, who volunteered to complete a health questionnaire and submit to standardized measurements of blood pressure, heart rate and body size. Average weekly alcohol consumption correlated with systolic pressure (r = 0.18) but not diastolic pressure. Systolic pressure increased progressively with increasing alcohol consumption, with no obvious threshold effect. In moderate and heavy drinkers (53% of the population studied), the prevalence of systolic hypertension (≥ 140 mm Hg) was four times that of teetotalers. The effect of alcohol on systolic blood pressure was independent of the effects of age, obesity, cigarette smoking and physical activity. Ex-heavy drinkers had blood pressures similar to those of teetotalers, suggesting that the effect of alcohol is reversible. Cigarette smokers had lower diastolic pressures than nonsmokers, an effect independent of obesity. The linear correlation between alcohol consumption and systolic blood pressure and the lower blood pressures in exdrinkers suggest a cause-and-effect relationship. The results indicate that alcohol ranks close to obesity as a potentially preventable cause of hypertension in the community.

AN ASSOCIATION between alcohol consumption and hypertension has been found in several studies of general populations. Kannel and Sorlie1 and Clark, Chapman and Coulson2 found a modest positive relationship between alcohol consumption and systolic pressure that was independent of weight. Data from the Kaiser Permanente Study,3 which involved more than 80,000 persons, showed that men and women who consumed three or more drinks per day (approximately 45 g or more of ethanol) had higher mean systolic blood pressures and a substantially higher prevalence of hypertension. In that population, the association of drinking and blood pressure was independent of age, sex, race, smoking, coffee use, former heavy drinking, educational achievement and adiposity. All three studies found that people who drink one or two drinks per day had approximately the same or slightly lower blood pressures than teetotalers. These studies complement earlier work that suggests an increased prevalence of hypertension in heavy drinkers and alcoholics.4–7

In the present study we examine further the association between alcohol drinking and blood pressure in an employed population of men. In particular, the relationships between alcohol consumption and factors...
such as obesity and smoking, which may also be involved in the development and aggravation of hypertension or vascular disease, are analyzed.

**Methods**

Four hundred ninety-one Caucasian male government employees, ages 20–45 years, volunteered for the study. None of these men were receiving treatment for high blood pressure. Four percent took painkillers once per week or more often. Seventy-two percent had been born in Australia (64% in Western Australia) and 18% had been born in Britain. Forty-three percent were professionals (such as engineers, accountants or architects), 26% were clerks and 25% were tradesmen (such as fitters or turners), representing a wide range of educational and social backgrounds. Twenty-nine other non-Caucasians and three men taking antihypertensive drugs were, at the outset, excluded to avoid confounding effects of race and treatment.

The men were asked to complete an 18-page questionnaire and participate in a health screening program conducted at the government offices between 8 a.m. and 12 noon from December 1979 to April 1980. To avoid adding to the stress of the survey, they were allowed normal breakfasts and were not advised to stop smoking before this visit. The questionnaire was used to assess demographic, socioeconomic and dietary factors and to collect information about drug use, smoking habits and the prevalence of cardiovascular or renal disorders within the immediate family.

Alcohol consumption was measured in accordance with recommendations from a workshop on methods of measurement of alcohol consumption. The men were asked the amount of alcoholic beverages they drank each day of the previous week and when they drank. If their beverage consumption in the previous week differed from their usual consumption, they were also asked what they drank in an average week. In this study the correlation coefficient between alcohol consumption as calculated from a previous week's drinking and that calculated from an average week's drinking was 0.9. Alcohol consumption as calculated from the previous week's drinking was used in all subsequent analyses. Beverage consumption was converted to an estimate of pure alcohol (ethanol) consumed by use of conversion factors for beer, wine and spirits recommended for use in Australia.

For analytical purposes, participants were grouped as follows: group 1 — teetotalers; group 2 — men consuming 1–160 ml (1–125 g) of ethanol per week (equivalent to up to two drinks per day); group 3 — men consuming 161–350 ml (126–275 g) of ethanol per week (equivalent to three to five drinks per day); group 4 — men consuming more than 350 ml (more than 275 g) of ethanol per week (equivalent to more than five drinks per day).

Of all drinkers, 74% drank only Western Australian beer containing 4.8% alcohol by volume; therefore, one drink was calculated as containing 10 ml of pure ethanol.

Duplicate blood pressure measurements were made after 5 minutes sitting, and again after 1 minute standing, by personnel trained by the method of Prineas using a London School of Hygiene sphygmomanometer and a 28 × 13-cm inflatable cuff. Disappearance of the sounds (Korotkov phase V) was used as the measure of diastolic pressure. Standing pressures were recorded with the arms supported at the level of the heart. Height and weight were measured by use of calibrated scales, the pulse rate was counted at the wrist for 30 seconds, and ambient temperature was recorded every 30 minutes. Statistical analysis was performed by computer using the Statistical Package for the Social Sciences.

The men were derived in approximately equal numbers from two local government concerns. Analysis of the data from the two groups separately showed a similar relationship between alcohol and blood pressure, so populations were pooled for all further analyses.

Where analysis of covariance was used to adjust group mean values, the validity of this procedure was established by determining the linear regression of the dependent on the independent variables, and comparing the regression slopes for the subgroups being studied. Sitting and standing blood pressures were analyzed separately in relation to the other factors; relationships were essentially similar; hence, only data relating to sitting pressures are presented.

**Results**

A summary of the drinking habits of the men is shown in table 1. One hundred seventeen of 491 men (24%) were currently teetotalers; of those, 74 (63%) had been lifelong teetotalers. The drinkers were fairly evenly separated into groups 2–4.

In one of the two local government concerns, a direct approach to individuals was made; the volunteer rate was 80% of the eligible work force. In the other, larger organization, volunteers were sought by poster, and the rate was 10%, giving similar absolute numbers in each subgroup. Despite the difference in volunteer rates, the relationship between blood pressure and alcohol consumption was similar in both subgroups, and comparison with census statistics for Western Australia indicated that the population studied was representative of the population of young men at large in terms of occupation. It therefore seems reasonable to extrapolate the results of this study to the wider community.

**Alcohol Intake and Blood Pressure**

For the population as a whole, alcohol consumption correlated significantly with systolic blood pressure \( r = 0.18, p < 0.001 \) but not with diastolic blood pressure \( r = -0.01 \). Systolic pressure increased progressively with increasing alcohol consumption, with no obvious threshold (fig. 1) \( F = 4.148, p < 0.006 \) for an effect of alcohol). Men who drank 1–160 ml/week of ethanol had a significantly higher mean systolic pressure than teetotalers (two-tailed t test \( p <\)
TABLE 1. Known Drinking Categories of 491 Government Employees

<table>
<thead>
<tr>
<th>Amount of ethanol consumed per week (ml)</th>
<th>n</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Teetotalers</td>
<td>117</td>
<td>24</td>
</tr>
<tr>
<td>Lifelong teetotalers</td>
<td>74</td>
<td>15</td>
</tr>
<tr>
<td>Exdrinkers</td>
<td>43</td>
<td>9</td>
</tr>
<tr>
<td>Drinkers</td>
<td>374</td>
<td>76</td>
</tr>
<tr>
<td>1–160</td>
<td>114</td>
<td>23</td>
</tr>
<tr>
<td>161–350</td>
<td>134</td>
<td>27</td>
</tr>
<tr>
<td>&gt;350</td>
<td>126</td>
<td>26</td>
</tr>
<tr>
<td>Total</td>
<td>491</td>
<td>100</td>
</tr>
</tbody>
</table>

0.051), and men who consumed more than 350 ml/week of ethanol had a mean systolic pressure 5.1 mm Hg higher than teetotalers (two-tailed t test \( p < 0.0001 \)). Analysis of variance showed no significant effect of alcohol on diastolic blood pressures (\( F = 1.297 \)). When teetotalers were subclassified into lifelong nondrinkers and ex-heavy drinkers (defined as men who in the past had consumed more than 350 ml/week of ethanol for at least 3 months), no significant differences between systolic or diastolic pressures of these two subgroups were found (blood pressure 119.2/71.4 mm Hg and 120.3/73.3 mm Hg respectively).

Prevalence of Hypertension

Increasing alcohol consumption was associated with a greater prevalence of both systolic (\( \geq 140 \) mm Hg) and diastolic (\( \geq 90 \) mm Hg) hypertension. Overall, there were significant differences in the proportions with systolic (\( p < 0.05 \)) and diastolic (\( p < 0.025 \)) hypertension in each drinking class. There were also significant linear trends toward increasing prevalence of systolic (\( p < 0.001 \)) and diastolic (\( p < 0.025 \)) hypertension with increasing alcohol consumption (fig. 2).

Twenty-seven of the 260 men (10.4%) drinking more than 160 ml/week of ethanol (equivalent to three or more glasses of beer per day) had systolic pressures over 140 mm Hg, compared with three of the 117 teetotalers (2.6%). The prevalence of systolic hypertension was therefore four times greater in moderate and heavy drinkers than in nondrinkers (fig. 2). Twenty-six of the 260 men (10.0%) who drank more than 160 ml/week of ethanol had diastolic pressures \( \geq 90 \) mm Hg, compared with four (3.4%) of the 117 teetotalers (fig. 2).

Characteristics of Men According to Their Drinking Habits

The differences in age between drinking categories (table 2) were not statistically significant. Drinkers, however, were significantly more obese (\( p < 0.001 \)) than nondrinkers, as measured by the Quetelet index (weight/height\(^2\)).

Cigarette smoking and regular physical exercise were both significantly associated with drinking alcohol. Eighteen percent of teetotalers smoked, compared with 50% of men who drank more than 350 ml/week of ethanol (\( \chi^2 = 33.8, p < 0.00001 \)). Forty-six percent of teetotalers said they exercised regularly throughout the year, compared with 65% of men who drank more than 350 ml/week of ethanol (\( \chi^2 = 16.42, p < 0.01 \)).

Alcohol, Obesity and Blood Pressure

Multiple linear regression showed that alcohol intake and level of obesity were the only factors that significantly influenced systolic pressure independent of the others (\( r = 0.18 \) and 0.20, respectively, \( p < 0.001 \)). Other factors included in the analysis were age, number cigarettes smoked per day, weeks of regular exercise each year, educational attainment and coffee and tea consumption.

Obesity and age were the major determinants of diastolic pressures (\( r = 0.30 \) and 0.27, respectively, \( p < 0.01 \)). As the majority of subjects were nonsmokers, the effect of smoking cigarettes is analyzed in the next section.

Data were cross-classified by alcohol use and systolic pressure according to the Quetelet index (fig. 3). A relationship between ethanol and blood pressure is seen within each adiposity subgroup. The results confirm that an association between alcohol intake and systolic pressure is present independent of the effect of adiposity on blood pressure.

We tried to determine which factors would correctly classify subjects at the upper and lower extremes of
systolic pressure, arbitrarily $\geq 140$ mm Hg and $\leq 110$ mm Hg, using a linear stepwise discriminant function. The effects of age, alcohol consumption, obesity, cigarette smoking, coffee, exercise and education were examined. A discriminant function, with alcohol consumption and obesity as the two independent variables, could correctly classify 70.6% of cases into these two groups. The standard canonical discriminant coefficient for alcohol consumption in this function was 0.77 and that for obesity was 0.59, indicating that with the group selected, alcohol intake was a slightly stronger determinant of systolic pressure than was obesity.

A similar discriminant function analysis was carried out for the extremes of diastolic pressure $\geq 90$ mm Hg and $\leq 60$ mm Hg. This showed that age and obesity as independent variables could correctly classify 76.3% of cases into these groups. The standard canonical discriminant coefficient for age was 0.55 and that for obesity was 0.72.

As a standard sphygmomanometer cuff was used for all subjects, the possibility that differences in arm girth might have influenced the results on the relation between alcohol and blood pressure was examined by correcting arterial pressures for arm girth using tables from Pickering. With this adjustment, the correlation between systolic pressure and alcohol consumption persisted ($r = 0.15$, $p < 0.005$) and the relation be-

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**Table 2. Characteristics of Men with Known Drinking Habits**

<table>
<thead>
<tr>
<th>Alcohol consumption (ml of ethanol/week)</th>
<th>0 (n = 117)</th>
<th>1-160 (n = 114)</th>
<th>161-350 (n = 134)</th>
<th>&lt;350 (n = 126)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Obesity (Quetelet index g/cm²)</td>
<td>2.414</td>
<td>2.445</td>
<td>2.477</td>
<td>2.496*</td>
</tr>
<tr>
<td>Age (years)</td>
<td>31.6</td>
<td>31.4</td>
<td>30.9</td>
<td>30.1</td>
</tr>
<tr>
<td>% cigarette smokers</td>
<td>18</td>
<td>28</td>
<td>33</td>
<td>50</td>
</tr>
</tbody>
</table>

Values are mean ± SD.

*Effect of alcohol on Quetelet index, $p<0.001$.

χ² for linear trend for increased prevalence of smoking with increasing alcohol consumption, $p<0.001$.
between obesity and systolic pressure disappeared, while that between diastolic pressures and obesity remained ($r = 0.13, p < 0.002$).

**Alcohol-Smoking Interaction**

Because alcohol consumption and smoking cigarettes were related (table 2), the effects of these factors were considered separately (fig. 4). Systolic and diastolic pressures and heart rates were compared in smokers and nonsmokers (including ex-smokers) in the population as a whole and in each of the four categories of alcohol consumption. Values were adjusted by covariance analysis for effects of age and adiposity (fig. 4). Overall, smokers had slightly lower mean diastolic pressures (2.8 mm Hg) than nonsmokers ($F = 7.0, p < 0.008$). Teetotalers who smoked had mean diastolic pressures 5.8 mm Hg lower than those who did not, and heavy drinkers (< 350 ml/week of ethanol) who smoked had diastolic pressures 4.9 mm Hg lower than those who did not smoke. The effect of smoking on systolic pressures was not significant, although for three of the four levels of alcohol consumption, the mean systolic pressure was lower in smokers than nonsmokers. Smokers had significantly higher heart rates (5 beats/min) than nonsmokers ($p < 0.0001$) (fig. 4).

The effect of alcohol on systolic blood pressures was present in both smokers and nonsmokers (fig. 4).

**Sport and Other Factors**

Physical activity was assessed in two ways: first, by a score based on statements of the number of days in the average week different types of vigorous exercise were taken outside working hours; second, according to the answer to the question, "How many weeks of the year do you regularly play sport?" When the population was divided into tertiles of physical activity on the basis of both these criteria, regular exercise had the greatest effect on heart rate, with slower heart rates related to increased regular exercise ($F = 8.8; \text{df} = 2; p < 0.001$; table 3). As this suggested that regular exercise was the better discriminator of physical fitness at the time of the survey, this measure was used to analyze for effects on blood pressure. Analysis of vari-

![Figure 4. Mean systolic and diastolic blood pressures (± SEM) and heart rates by drinking and smoking habits. Means have been adjusted for age and adiposity (Quetelet index) by analysis of covariance. Analysis of variance shows significant effects of smoking on diastolic pressure ($p < 0.008$) and heart rate ($p < 0.0001$).](image-url)
Discussion

Although we cannot infer from this cross-sectional study that the relationship between alcohol and elevated blood pressure is one of cause and effect, in favor of a causal relationship are the linear dose-response relationship between alcohol and systolic pressure levels, which was seen at varying levels of obesity in the present study, and the normal blood pressures of those who were formerly heavy drinkers and now abstain.

A threshold for the effect of alcohol on blood pressure found in three studies was not observed in our population. Even men who drank 1–160 ml/week of ethanol (equivalent to two or fewer drinks per day) had significantly higher systolic pressures than teetotalers. Systolic pressure increased 1 mm Hg for every glass of alcohol-containing beverage drunk per day. The prevalence of systolic hypertension (≥ 140 mm Hg) was several times higher in moderate-to-heavy drinkers than teetotalers. Moreover, alcohol consumption was the most powerful predictor of extremes of systolic pressure in a discriminant-function analysis. Although very heavy drinkers were more obese than teetotalers, the relationship between alcohol consumption and systolic pressure was seen at all levels of adiposity. Moreover, if blood pressures were arithmetically corrected for the differences in arm girth, the relationship between alcohol and systolic pressures remains.

The data thus provide further evidence that the higher systolic pressures among those who consume more than 160 ml/week of ethanol cannot be explained by confounding effects of age, level of obesity, amount of physical exercise and cigarette smoking.

Given the error in normal variability of blood pressure measurements, and the difficulties in obtaining a reliable assessment of alcohol consumption, the extent to which alcohol consumption and obesity could predict those at the upper and lower extremes of systolic pressure with 70% accuracy using a discriminant-function analysis is striking.

Age and obesity were the predictors of extremes of diastolic pressure. Although the population as a whole showed no significant correlation between alcohol intake and diastolic pressures, the prevalence of diastolic hypertension (≥ 90 mm Hg) was three times greater in the moderate-to-heavy drinkers than teetotalers. Two reports in which a correlation was found between alcohol intake and diastolic pressure included populations older than ours. Age may account for this difference; elderly subjects are more prone to elevation of both systolic and diastolic pressures through the cumulative effect of adaptive and degenerative changes in the cardiovascular system due to longer exposure to elevated mean pressures.

The systolic and diastolic pressures of lifelong teetotalers were not significantly different from those of ex-heavy drinkers. Thus, the increase in blood pressure associated with drinking alcohol, whether it be a direct effect of ethanol intake or dependent on some factor not considered in this study, appears to be reversible when drinking ceases. If the association is due to a confounding factor, this factor must vary with alcohol consumption, and is thus unlikely to be one of the more permanent characteristics of the individual, such as a personality attribute. There are no adequately controlled prospective studies of the effects of long-term cessation of moderate-to-heavy alcohol consumption, although earlier reports, reviewed by Ashley and Rankin, suggested that alcoholic hypertensives who abstain tend to normalize their blood pressure. This is supported by a recent study of very heavy drinkers who abstained.

How alcohol might increase blood pressure is not clear. A direct action on the central nervous system provides the most attractive explanation: In susceptible persons, alcohol may have a sustained pressor action by interfering with central inhibitory pathways controlling vasomotor centers, in a manner analogous to its action on higher cortical function leading to loss of emotional control. Pressor effects might then be mediated by neural and humoral mechanisms similar to those involved in the acute "stress" reaction induced by alcohol. However, as alcohol has widespread biologic effects, its action on blood pressure control mechanisms may be multiple and complicated by interactions with other factors. Thus, a central pressor influence of alcohol may be countered to some extent by its action as a peripheral vasodilator, perhaps accounting for its predominant effect on systolic rather than diastolic pressures in our relatively young population.

In addition to a direct pressor effect of alcohol, continued heavy drinking may contribute indirectly to hypertension by virtue of its calorie content predisposing to obesity. Moreover, as shown in this report, those who are both obese and drinkers have higher pressures than those who demonstrate either one of these characteristics.

The possibility that the blood pressure elevation in drinkers is a withdrawal phenomenon due to temporary abstinence before examination should be considered. Acute hypertension after cessation of alcohol has so far only been described in problem drinkers in whom blood pressures decrease with continuing abstinence. A milder form of the same phenomenon in persons drinking one or two drinks per day seems less plausible than a more direct pressor effect of alcohol; however, whether repeated blood pressure elevations arise from a direct effect, or indirectly from repeated withdrawal episodes, the result is likely to be progressive resetting of blood pressure to higher levels.

Cigarette smokers overall had slightly lower diastolic pressures (2.8 mm Hg) than nonsmokers despite a higher heart rate. The effect of smoking on diastolic pressure was present at all levels of alcohol consumption, and was more marked in teetotalers and heavy drinkers (> 350 ml/week). Smokers may have lower
blood pressures primarily because of their tendency to be less obese than nonsmokers. Our results show that a depressor effect of smoking is still present when obesity level is held constant, suggesting an additional mechanism. One possibility is selective mortality of smoking hypertensives, which would leave behind a population of smokers with lower-than-average blood pressures. However, this selection probably would not be much in evidence by the age of 45 years, the upper age limit in this survey. Blood pressure reduction due to alleviation of stress by smoking would be more likely.

The case for an overall reduction in alcohol consumption as a public health measure can be made on many grounds besides its relation to hypertension. Reports that alcohol may lower the incidence of coronary deaths must be considered in the broader context of the effects of alcohol on health. Thus, in a prospective study of causes of deaths in 11,000 Yugoslav men ages 35–62 years, Koizaradvice et al. reported that alcohol consumption was inversely related to coronary heart disease mortality, but the overall risk of dying increased with alcohol intake, due to stroke, cancer and accidental or violent deaths. They concluded that an effect of alcohol on blood pressure was mainly responsible for the increased incidence of stroke deaths. Similar results have been reported from Honolulu. A recent study of mortality of civil servants ages 40–64 years in London showed lowest cardiovascular mortality in mild and moderate (1–34 g/day) drinkers and a U-shaped curve for overall mortality in relation to alcohol consumption. In that study heavy drinkers (>34 g/day) had higher systolic and diastolic pressures than nondrinkers.

On the basis of mortality statistics, Mathews proposed that alcohol usage may be an explanation for socioeconomic and occupational differentials in mortality from hypertension and coronary heart disease in England and Wales. He also suggested that if the relationship between alcohol and blood pressure levels were causal and linear, then 10–20% of hypertension could be due to alcohol in countries with a per capita consumption similar to that in Australia (i.e., 10 liters of absolute alcohol annually). The present study provides further evidence for linearity and causality in the relationship. Moreover, as blood pressures of individuals tend to retain their rank order in relation to one another with increasing age, even a relatively small pressor effect of alcohol in early adult life may be significant with continued exposure, particularly if coupled with progressive weight gain.

Presuming that alcohol per se was largely responsible for the three- to fourfold excess of hypertension in the moderate and heavy drinkers (53% of our study population), the overall contribution of alcohol to hypertension in the male community must be considerable. As a corollary, a reduction in both moderate social and heavy drinking could result in a substantial fall in the prevalence of hypertension.

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