Alcohol: effect on blood pressure and predisposition to hypertension

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Summary

1. The association between alcohol consumption and blood pressure was studied in 491 Government employees. The men, aged 21–45 years, volunteered to complete a health questionnaire and submitted to standardized measurements of blood pressure, heart rate and body size.

2. Average weekly alcohol consumption correlated with systolic pressure \( r = 0.18, P < 0.001 \) but not with diastolic pressure. Systolic pressure increased progressively with increasing alcohol consumption with no obvious threshold effect. The effect of alcohol was independent of age, obesity (Quetelet's index) or cigarette smoking.

3. Results indicate that alcohol ranks close to obesity as a preventable cause of essential hypertension in the community.

Key words: alcohol consumption, hypertension.

Introduction

An association between alcohol consumption and hypertension has been found in several general population studies [1, 2, 3]. Data from the Kaiser–Permanente study involving more than 80,000 persons [3] showed that men and women who consumed three or more drinks per day (approximately 45 g of ethanol or more) had higher mean systolic blood pressures and a substantially higher prevalence of hypertension. In this population the association of drinking and blood pressure was independent of age, sex, race, smoking, coffee use, former ‘heavy’ drinking, educational attainment and adiposity, strongly suggesting that the regular consumption of alcohol at this level may itself be a risk factor for hypertension. Three studies [1, 2, 3] found that people drinking one or two drinks per day had approximately the same or slightly lower pressures than teetotallers. Recently Mitchell et al. [4] found a strong linear correlation between alcohol consumption and systolic and diastolic pressures. In the population they studied, alcohol was the major environmental factor determining blood pressure.

This study examines the extent to which drinking alcohol contributes to the incidence of hypertension in a working population. It also examines the interrelationship between alcohol consumption and other environmental factors, such as obesity and smoking, implicated in the development and aggravation of hypertension or vascular disease.

Methods

Caucasian male Government employees (491) aged between 20 and 45 years took part in the study. These men were asked to complete an 18 page questionnaire and a 24 h dietary record form and to participate in a health screening programme conducted at the Government offices. The questionnaire was used to assess demographic, socio-economic, dietary and personality factors. It also collected information about drug use (including smoking habits) and the incidence of diseases within the immediate family which may predispose the individual to hypertension.

Alcohol consumption was assessed in two ways. The men were asked to write down the type and amount of alcohol-containing beverages they drank each day of the previous week. If their
alcohol consumption in the previous week differed from their usual consumption, they were also asked to write down what they would drink in an average week. 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 analyses.

The height and weight, pulse rate, mid-arm girth and skinfold thickness of the men were measured in a rigorously standardized screening programme. Blood pressure measurements were made in both the seated and the standing positions by personnel trained with the taped Korotkoff method of Prineas [5] using the London School of Hygiene sphygmomanometer.

Results

Of the 76% of drinkers, 74% drank beer only; 53% of men drank three or more drinks per day (one drink contains 10 ml of pure ethanol).

Alcohol consumption correlated with systolic pressure ($r = 0.18, P < 0.001$) but not with diastolic pressure. Systolic pressure increased progressively with increasing alcohol consumption with no obvious threshold effect (Fig. 1). Even men drinking one or two drinks per day had significantly higher systolic pressures than teetotallers ($t = 1.74, P < 0.05, d.f. = 230$). Men consuming six or more drinks per day had a mean systolic pressure 5.1 mmHg higher than teetotallers ($t = 3.81, P < 10^{-4}, d.f. = 242$).

Of 260 men drinking more than three drinks per day 10.4% had systolic pressures >140 mmHg, compared with 2.6% for 117 teetotallers ($\chi^2$ for linear trend between alcohol consumption and systolic hypertension $P < 0.005$). When teetotallers were subclassified into lifelong non-drinkers and ex-heavy drinkers (defined as men who in the past had consumed more than 350 ml of ethanol per week 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 mmHg and 120.3/73.3 mmHg respectively).

Age and obesity were correlated with increasing diastolic pressure, whereas cigarette smokers had lower diastolic pressures and increased heart rates compared with non-smokers. Of the 260 men drinking over 160 ml of ethanol per week 10% had diastolic pressures >90 mmHg, compared with 3.4% of 117 teetotallers ($\chi^2$ for linear trend $P < 0.025$).

Men consuming six or more drinks per day were more obese (using Quetelet’s index as the unit of measurement) than teetotallers ($t = 3.19, P < 0.001, d.f. = 242$). Together adiposity and alcohol consumption accounted for 6.5% of the

FIG. 1. Mean (±SEM) systolic/diastolic blood pressures of men with known drinking habits. Means adjusted for age and Quetelet’s index by analysis of covariance. Analysis of variance for an effect of alcohol on sitting systolic pressure: $F = 4.418$, d.f. = 3, $P < 0.006$. $t$-test vs non-drinkers: $+P < 0.05; \dagger P < 0.005$. 

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Men consuming six or more drinks per day were more obese (using Quetelet’s index as the unit of measurement) than teetotallers ($t = 3.19, P < 0.001, d.f. = 242$). Together adiposity and alcohol consumption accounted for 6.5% of the
Variation in systolic pressure (multiple $r = 0.26$, $F = 17.02$, $P < 0.001$). Adiposity and alcohol consumption were the only two environmental factors found to influence systolic pressure substantially.

No correlation was found between alcohol consumption and age. Of teetotallers 15% smoked cigarettes, and 50% of men drinking six or more drinks per day smoked cigarettes ($X^2 = 33.88$, $P < 10^{-5}$). Men drinking six or more drinks per day had higher systolic pressures than teetotallers, irrespective of smoking habits. Men who drank heavily and smoked still had significantly higher pressures than teetotallers ($t = 2.90$, $P < 0.005$, d.f. = 309), but had significantly lower pressures than men who drank heavily and did not smoke ($t = 1.90$, $P < 0.05$, d.f. = 124).

Smoking, independent of age, level of obesity and alcohol consumption, significantly reduced diastolic pressure ($t = 2.49$, $P < 0.02$, d.f. = 489). Of the smokers, those drinking three or more drinks per day had significantly higher diastolic pressures than teetotallers ($t = 2.18$, $P < 0.02$, d.f. = 111).

Smokers had higher heart rates than non-smokers ($t = 4.40$, $P < 10^{-4}$, d.f. = 487).

Coffee and tea drinking, educational level and regular sporting activity had no effect on blood pressure, although sport was associated with significantly lower heart rates.

**Discussion**

Alcohol consumption and obesity were the only two environmental factors found to influence systolic blood pressure substantially. Obesity was only a slightly better determinant of systolic pressure than was alcohol consumption.

No threshold effect described in previous papers was observed and even men drinking one or two drinks per day had significantly higher pressures than teetotallers. The relationship between drinking alcohol and blood pressure is independent of age and adiposity (with Quetelet as the unit of measurement). The incidence of systolic hypertension (pressures over 140 mmHg) was four times greater in men drinking three or more drinks per day than in teetotallers and that of diastolic hypertension increased threefold.

Cigarette smoking was associated with increased heart rates and lower diastolic blood pressures in teetotallers and drinkers.

It cannot be inferred with certainty from this cross-sectional study that the relationship between alcohol and elevated blood pressure is one of cause and effect. However, in favour of a causal relationship are the ‘linear dose–response’ between alcohol and systolic pressures, which was seen at various levels of obesity, and the normal blood pressures of ex-heavy drinkers.

Assuming that alcohol _per se_ was largely responsible for the three- to four-fold excess of hypertension in the moderate to heavy drinkers who comprised 53% of our study population, then the overall contribution of alcohol to hypertension in the male community must be considerable.

As a corollary, a reduction in both so-called moderate ‘social’ as well as heavy drinking could result in a substantial fall in the incidence of hypertension and its serious sequelae.

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**References**


