The influence of chronic high alcohol intake on blood pressure, plasma noradrenaline concentration and plasma renin concentration

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Summary

1. Sixteen 44-year-old males with chronic high alcohol intake were investigated. Seventeen 44-year-old males with low alcohol intake from the same population served as controls.

2. Plasma noradrenaline concentrations did not differ significantly between individuals with high and low alcohol intake, neither at rest nor after acute stimulation induced by ambulation for 15 min. However, 63% (10 out of 16) of the individuals with high intake showed resting values within the upper quartile range for individuals with low intake.

3. Plasma renin concentration was twice as high ($P < 0.01$) in the group with high alcohol intake as in the group with low intake.

4. Systolic as well as diastolic blood pressure was significantly higher ($P < 0.01$) in the group with high intake compared with the group with low intake.

5. Sympathetic nerve activity, as defined from measurements of plasma noradrenaline concentration, is not uniformly increased in individuals with chronic high alcohol intake. The mechanisms behind the increased plasma renin level as well as the possible role of the renin–angiotensin system in alcohol-induced hypertension remain unsettled.

Key words: alcohol, noradrenaline, renin.

Abbreviations: PA, plasma adrenaline; PNA, plasma noradrenaline; PRC, plasma renin concentration; SGOT, serum glutamic–oxaloacetic transaminase.

Introduction

It is well recognized that alcohol abuse is associated with high blood pressure [1–4]. Furthermore, it has been shown that blood pressure decreases when the high alcohol consumption is discontinued [4]. Increased noradrenergic tone has been proposed as one of the possible mechanisms implicated in alcohol-induced hypertension [4]. Alcohol intake and alcohol abstinence have been found to increase urinary catecholamine excretion, but experimental data are few and controversial [5].

We have previously reported that plasma noradrenaline concentration at rest supine as well as after acute stimulation does not differ between 40-year-old hypertensive and normotensive individuals [6, 7]. Our finding is to some extent at variance with other investigations comparing plasma noradrenaline concentration in hypertensive and normotensive individuals [8]. In our previous study all individuals with a daily alcohol intake of more than 40 g (approximately more than four drinks/day) and/or abnormal liver-function test were excluded.

Since alcohol intake is high among individuals with high blood pressure and alcohol may increase sympathetic tone this might be one of the explanations for the discrepancies found in the literature. As far as we know, alcohol consumption has never been considered in earlier investigations.

The purpose of the present study was to investigate the influence of alcohol on blood
pressure, plasma noradrenaline concentration and plasma renin concentration.

Methods
All the individuals were born in 1936 and had participated in a population survey at the age of 40 years [6, 7]. Based upon their history of alcohol intake at that time and again 3 years later, by using a questionnaire, all males with a consistently high alcohol intake (≥35 drinks/week) were invited to the study. Sixteen individuals accepted the invitation and six declined. None of the individuals studied was severely disabled from their drinking habits. The alcohol consumption mainly consisted of beer, only two drinking predominantly spirits.

A sample of males with a low/moderate alcohol consumption from the same population was also studied. Seventeen individuals had a consistently low intake (<20 drinks/week). Five individuals formed a separate group with an estimated intake of 20–30 drinks/week.

None of the individuals had ever received antihypertensive treatment.

Blood pressure and heart rate were measured after 5 min rest in the sitting position and again after 10 min rest supine. Diastolic blood pressure was read at the disappearance of the Korotkoff sounds (phase V). The mean of three consecutive readings was used.

Blood samples for determination of plasma noradrenaline (PNA) and plasma adrenaline (PA) concentration [9] and plasma renin concentration (PRC) [10] were drawn after 10 min rest supine, 10 min after insertion of an indwelling needle. Blood samples for determination of serum glutamic–oxaloacetic transaminase (SGOT), blood glucose and blood ethanol concentration were also taken. PNA and PA were measured again after 15 min of quiet ambulation. The study was carried out in the morning at 08.30 hours with the individuals in the fasting state and they were not allowed to smoke. The statistical calculations were carried out by means of Wilcoxon’s test for paired differences and Mann–Whitney rank-sum test for unpaired observations. For correlation analysis Spearman’s rank test was used. Figures are presented as median values with ranges in parentheses.

Results
In the group with high alcohol intake (≥35 drinks/week) alcohol consumption was 50 drinks/week as a median figure (range 36–100). In the group with low intake (<20 drinks/week) median alcohol consumption was 11 drinks (range 0–18).

PNA did not differ significantly between individuals with high and low alcohol intake, neither at rest nor after acute stimulation was induced by ambulation (Table 1). However, 63% (10 out of 16) of the individuals with high intake showed resting values within the upper quartile range for individuals with low intake. The increase in PNA from resting to stimulated values was identical in the two groups. PA was identical in the two groups.

PRC was twice as high in individuals with high intake as in those with low intake (P < 0.01). Systolic as well as diastolic blood pressure was significantly higher in individuals with high alcohol intake as compared with the group with low intake (Table 1). Heart rate was also higher in the group with high intake.

SGOT was higher (P < 0.01) in individuals with high alcohol intake, 24 units/l (16–122), than

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**Table 1.** Plasma noradrenaline concentration (PNA), plasma renin concentration (PRC), systolic (SBP) and diastolic (DBP) blood pressure and heart rate (HR) in 44-year-old males with chronic high alcohol intake and in controls with low alcohol intake

<table>
<thead>
<tr>
<th></th>
<th>Individuals with high alcohol intake</th>
<th>Individuals with low alcohol intake</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>n = 16</td>
<td>n = 17</td>
</tr>
<tr>
<td>PNA (nmol/l)</td>
<td>Rest supine 1.40 (0.45–1.97)</td>
<td>N.S. 0.96 (0.64–1.85)</td>
</tr>
<tr>
<td></td>
<td>After ambulation 2.23 (1.15–3.57)</td>
<td>N.S. 1.85 (1.21–3.57)</td>
</tr>
<tr>
<td>PRC (m-i.u./l)</td>
<td>Rest supine 50 (23–99) &lt;0.01</td>
<td>25 (10–45)</td>
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<tr>
<td></td>
<td>After ambulation 132 (114–148) &lt;0.01</td>
<td>118 (102–137)</td>
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<tr>
<td></td>
<td>Systolic 131 (110–156) &lt;0.05</td>
<td>120 (96–134)</td>
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<tr>
<td></td>
<td>Supine 89 (76–98) &lt;0.01</td>
<td>80 (66–92)</td>
</tr>
<tr>
<td></td>
<td>Systolic 88 (72–96) &lt;0.01</td>
<td>80 (68–92)</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>Sitting 76 (58–108) N.S.</td>
<td>72 (56–84)</td>
</tr>
<tr>
<td></td>
<td>Supine 69 (54–88) &lt;0.02</td>
<td>64 (44–74)</td>
</tr>
</tbody>
</table>
in individuals with low intake, 12 units/l (11–24), and in five with high intake the figure was above the upper limit for normal values (40 units/l). None of the individuals with low intake had abnormal SGOT values.

The number of smokers was significantly higher in the group with high alcohol intake than in the group with low intake (15 vs 7).

Figures for the variables measured in the five individuals in the group with 20–30 drinks/week were not subjected to statistical calculations, but the values are included in the correlation analysis for all the individuals.

There was no significant correlation between alcohol intake and PNA, and blood pressure did not correlate to PNA. There were significant correlations between alcohol intake on the one hand and PRC \( (r_s = 0.64, P < 0.01) \), DBP \( (r_s = 0.41, P < 0.02) \) and SGOT \( (r_s = 0.55, P < 0.01) \) on the other hand. There was no correlation between PRC and blood pressure.

Fasting blood glucose concentration was normal in all individuals. Except for one single individual (blood ethanol concentration 0.5 g/l (11 mmol/l)), all blood samples were without any measurable amount of ethanol.

Discussion

The present investigation deals with 44-year-old males with a consistently high alcohol consumption during at least 4 years. As mentioned above none of them was severely socially disabled from their drinking habits. The controls were individuals from the same population with persistently low alcohol consumption during the same period. All were investigated in the fasting state after one night of alcohol abstinence.

In agreement with other investigations the blood pressure was higher in individuals with high alcohol intake than in those with low intake \([1, 2, 4]\). This finding is probably not explained by increased sympathetic tone since PNA was not significantly increased and no correlation was found between PNA and blood pressure. On the other hand, 63% of the individuals in the group with high intake showed resting PNA values within the upper quartile range for individuals with low intake.

Our initial proposal that high alcohol consumption might explain the high PNA values in some investigations comparing PNA in hypertensive and normotensive individuals is not clearly supported by the present data.

Heart rate was increased in individuals with high alcohol intake. This finding might be due to decreased vagal tone.

Although no clear cut increase was found in PNA, PRC was twice as high in the group with high intake as in the group with low intake. The mechanism behind the increased plasma renin levels needs further clarification \([11]\), and the possible role of the renin–angiotensin system in alcohol-induced hypertension remains unsettled.

References