The Pressor and Metabolic Effects of Alcohol in Normotensive Subjects

JOHN F. POTTER, ROBERT D. S. WATSON, WENDY SKAN, AND D. GARETH BEEVERS

SUMMARY Changes in blood pressure, pulse rate, and plasma catecholamines, renin activity, cortisol, and calcium were studied in 16 normotensive subjects (eight with a family history of hypertension) for 5 hours following ingestion of alcohol-free and alcohol-loaded beer. Both systolic and diastolic blood pressure rose after alcohol consumption; maximum responses occurred at peak blood alcohol concentrations and were significantly higher than those seen after placebo. Pulse rate was also significantly higher after alcohol ingestion and continued to rise throughout the study. There was no difference in the pressor response to alcohol between the groups with and without a family history of hypertension. No difference was found in plasma norepinephrine or epinephrine levels between alcohol and placebo phases. However, subjects with no family history of hypertension had significantly higher plasma norepinephrine levels (p<0.01) than did those with a family history during both the alcohol and placebo phases, although baseline blood pressures were not significantly different. Plasma epinephrine level was similar in both groups. Plasma renin activity was unchanged throughout, but plasma cortisol fell during both phases. Plasma calcium showed a small but significant fall with alcohol consumption in both groups (p<0.001). These results indicate that in normotensive subjects alcohol ingestion causes a rise in systolic and diastolic blood pressure that is not influenced by a family history of hypertension. This effect does not appear to be sympathetically mediated but may be due to a direct vasoconstrictor effect of alcohol, possibly with an alcohol-induced shift in intracellular calcium. (Hypertension 8: 625-631, 1986)

KEY WORDS • alcohol • hypertension • genetic factors • catecholamines • calcium • cortisol • renin activity

Both epidemiological and clinical studies have confirmed a close relationship between alcohol consumption and blood pressure. This effect appears to be independent of age, race, sex, social class, and body mass index. In previous work, we have shown that alcohol consumption causes a rise in blood pressure in moderate-drinking hypertensive subjects. This pressor response differs from the alcohol withdrawal-associated elevation of pressure seen in alcoholic patients during detoxification. Studies of the mechanisms of the alcohol–blood pressure link in nonalcoholics are conflicting, possibly because of differences in the type of subjects investigated, the amount of alcohol given, the lack of placebo control, and the duration of the study. We have therefore examined, in normotensive subjects, the acute effects of alcohol and placebo on blood pressure in relation to the possible pressor mechanisms. We examined the changes in blood pressure with the rise and fall in blood alcohol levels to determine whether these changes were due to a direct effect of alcohol or were related to a withdrawal response.

Subjects and Methods

Sixteen male medical students (mean age, 22 years; range, 20–30 years) who normally drank up to 200 g (4.3 mol) alcohol per week (mean intake, 92 g/week assessed by self-administered 2-week retrospective diary) were studied on two occasions at least 1 week apart. All were healthy and taking no medication. Family histories of hypertension, determined by blood pressure measurement of all parents in their homes after 5 minutes of rest, were obtained either by a physician or, if not possible, by the student himself. Subjects were considered to have a family history of hy-
pertension if one or both parents had ever received treatment for hypertension or had a resting blood pressure of 160/95 mm Hg or more. Eight subjects thus were considered to have a family history of hypertension.

All subjects abstained from alcohol, caffeine, and tobacco for 24 hours (although diet was otherwise unrestricted), and all fasted for 12 hours before each part of the study. Procedures took place after micturition in the morning, in a quiet room (temperature, 20–24°C), with the students wearing light clothing. A forearm vein cannula was inserted into each subject, and blood pressure was measured using a Hawksley (Lancing, England) random zero sphygmomanometer (diastolic pressure phase V); the mean of three readings was taken at each time point. Pulse rates were recorded for 1 minute. After subjects had been supine for 1 hour, unbuffed blood samples were taken for packed cell volume, blood alcohol levels, plasma renin activity, and plasma concentrations of calcium, cortisol, nor-epinephrine (NE), and epinephrine (E). Each subject then drank 600 ml of Barbican, an alcohol-free lager (Canada Dry Rawlings, Northants, UK) kept at room temperature, with or without the addition of 50% alcohol in a double-blind, random-order crossover design. The mean weight of the students was 77.0 kg (range, 67–96 kg), and the amount of alcohol they received was related to body weight (0.75 g/kg body weight). Supine blood pressure and pulse rate were recorded at 15-minute intervals for the first hour after ingestion and then every 30 minutes for the next 4 hours. Blood samples were taken for NE and E in 14 subjects (7 in each family history group) after 0, 0.5, 1, 2, 3, and 4 hours, and hourly samples were taken for all other estimations. Urine samples were collected hourly after blood sampling and at least 25 minutes before the next blood pressure reading. Subjects were asked to consume the drink within 15 minutes while semirecumbent. At the end of each part of the study subjects were asked to indicate whether they thought they had received the placebo (alcohol-free beer) or placebo with added alcohol.

All blood samples were kept on ice and centrifuged at 4°C within 30 minutes of collection. Plasma catecholamines were taken into chilled tubes containing ethylene glycol bis(β-aminoethyl ether)-N,N,N′,N′-tetraacetic acid and glutathione and stored at −20°C; paired samples from each subject were analyzed together within 6 weeks. Plasma NE and E were measured by the radioenzymatic assay of Peuler and Johnson. The intraassay coefficient of variation was 7% for NE and 13% for E. Sensitivity for NE and E was 15 and 5 pg/ml, respectively. Plasma electrolytes and calcium were assayed by Technicon autoanalyzer (Tarrytown, NY, USA). Total plasma calcium was measured by the method of Kessler and Wolfman with a coefficient of variation of 2% and corrected for serum albumin concentrations. Plasma cortisol and plasma renin activity were measured by radioimmunoassay, and blood alcohol was measured by the enzymatic method (Sigma Chemical, St. Louis, MO, USA). The study was approved by the hospital ethical committee.

Data are presented as means ± SEM. The differences within treatments were analyzed by paired Student’s t tests and between groups by Wilcoxon’s two-sample test and were considered significant when p was less than 0.05 (two-tailed analysis). The effects of treatment and time were tested by two-way repeated-measures analysis of variance, and the student Newman-Keuls test was applied to test for significant differences between treatments and between times. Differences between the family history groups with alcohol ingestion were also analyzed by analysis of variance.

Results

Subjects were blinded to the order of treatment, and only eight of the 16 subjects recognized both stages correctly, indicating that the blinding procedure was reasonably successful. Blood alcohol levels reached a maximum of $84 ± 6$ mg/dl (18 mmol/L) at 1 hour and fell to $18 ± 3$ mg/dl (0.4 mmol/L) after 5 hours. No subjects reported any discomfort either during or after alcohol ingestion.

Systolic blood pressure rose after alcohol ingestion in parallel with the rise in blood alcohol concentration, but no changes were seen with placebo ingestion; this was reflected in a significant time-treatment interaction ($df = 12, 180, F = 3.6, p < 0.001$; Figure 1). There was a significant difference between treatments at 45 and 60 minutes at the time of peak blood alcohol levels ($p < 0.05$, student Newman-Keuls). Diastolic blood pressure showed a similar rise with alcohol ingestion ($df = 12, 180, F = 6.2, p < 0.001$), and again a significant difference was seen between treatments at peak blood alcohol levels. Pulse rates rose immediately with the increase in blood alcohol ($df = 12, 180, F = 13.9, p < 0.001$), and there was a significant time-treatment interaction ($df = 12, 180, F = 5.9, p < 0.001$). Pulse rate continued to rise throughout the alcohol phase, and ingestion of placebo had no effect (see Figure 1).

Two subjects in the group with a family history of hypertension had parents who were both hypertensive, and six had one hypertensive parent; two parents in the group were receiving antihypertensive treatment. The difference in systolic and diastolic blood pressure between subjects with and without a family history of hypertension did not reach statistical significance (Table 1). The changes in blood pressure with alcohol ingestion were similar in both groups (Figure 2), suggesting that a family history of hypertension has no effect on the response to alcohol ingestion.

Plasma NE and E levels were not altered significantly by treatment or time during the 4 hours of study (Figure 3). Subjects with a family history of hypertension had significantly lower baseline levels of NE (but not E) than did those with no family history ($p < 0.01$ by Wilcoxon’s two-sample test; values for the baseline measurements of catecholamines were taken for each subject for both alcohol and placebo phases; see Table 1). These baseline differences in NE between the two family history groups were maintained throughout both the alcohol and placebo phases ($p < 0.01$ by analysis of variance). There was no between-group differ-
TABLE 1. Baseline Differences in Systolic and Diastolic Blood Pressure and Plasma Norepinephrine and Epinephrine Levels in Normotensive Subjects With and Without a Family History of Hypertension

<table>
<thead>
<tr>
<th>Variable</th>
<th>Family history (n = 8)</th>
<th>No family history (n = 8)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic BP (mm Hg)</td>
<td>127 ± 11 (117–142)</td>
<td>118 ± 10 (114–126)</td>
</tr>
<tr>
<td>Diastolic BP (mm Hg)</td>
<td>79 ± 9 (67–90)</td>
<td>69 ± 9 (58–80)</td>
</tr>
<tr>
<td>Norepinephrine (pg/ml)</td>
<td>182 ± 18</td>
<td>263 ± 27*</td>
</tr>
<tr>
<td>Epinephrine (pg/ml)</td>
<td>31 ± 6</td>
<td>33 ± 5</td>
</tr>
</tbody>
</table>

Values are means ± SEM. Range for each group is shown in parentheses. BP = blood pressure.

* p < 0.01, compared with values in subjects with a family history of hypertension (Wilcoxon's two-sample test).

in 1 hour, although no significant change was seen in plasma phosphate or albumin levels and no changes were seen with the ingestion of placebo.

Plasma cortisol fell during both alcohol and placebo phases (df 5, 75, F = 3.1, p < 0.01; see Table 2), although there was no difference between treatments. Plasma renin activity showed no variation with either time or treatment during the study.

During the alcohol phase, there was a significant, 1-kg weight loss by 5 hours (p < 0.01), but no change was seen in the placebo phase (Table 3). This weight loss was accounted for by an ethanol-induced diuresis, which started by 1 hour (p < 0.01) and was 700 ml greater than that seen with placebo ingestion by 5 hours (p < 0.01). However, there was no significant difference in urinary sodium and potassium output between alcohol and placebo ingestion.

Discussion

In this study a modest amount of alcohol, comparable to that consumed during social drinking, caused a transient rise in blood pressure of 5 to 7 mm Hg in normotensive subjects. This effect occurred immediately after alcohol consumption. The highest pressure occurred with peak blood alcohol levels; however, blood pressure then began to fall while the blood alcohol levels remained elevated. Similar acute pressor effects of alcohol have been reported by Grollman and Orlando et al. while Zsoter and Sellers reported a rise only in diastolic blood pressure and Ireland et al. only in systolic blood pressure. These results indicate that alcohol has a short-lived direct pressor effect in normotensive subjects. From the design of the study it is not possible to determine whether the rise in blood pressure was due to an increase in cardiac output, an increase in total peripheral resistance, or a combination of these factors. However, these mechanisms are different from those responsible for the elevation in pressure during alcohol withdrawal in alcoholics, in whom pressures rise as blood alcohol levels fall.

Alcohol caused a modest increase in heart rate that
FIGURE 2. Effect of alcohol on systolic (SBP) and diastolic blood pressure (DBP) in eight normotensive subjects with a family history of hypertension (*) and eight subjects with no such history (O) over 5 hours. Values are means ± SEM.

FIGURE 3. Effect of alcohol (*) and placebo (O) on plasma norepinephrine and plasma epinephrine levels in 14 normotensive subjects over a 4-hour period. Values are means ± SEM. Became more marked after 3 hours. This response is likely a direct chronotropic effect of alcohol that is independent of β-adrenergic receptor blockade and may be due to acetaldehyde (an alcohol metabolite), releasing myocardial catecholamines. The more marked rise toward the end of the study probably represents a reflex response to the decrease in plasma volume after the ethanol-induced diuresis.

Genetic factors may be important in the development of hypertension. Normotensive subjects with a family history of hypertension respond differently to dietary manipulation of potassium, mental stress, and isometric exercise. Subjects with a family history of hypertension have higher catecholamine levels during stress and show enhanced pressor response to exogenous catecholamines. Bianchetti et al. noted lower mean levels of plasma NE in normotensive subjects with a history of hypertension than in those without such a history, though these differences did not reach statistical significance. Beilin et al. found higher levels of E but not NE in high normal compared with low normal blood pressure groups, although no account was taken of a family history of hypertension. Although our study showed no difference in blood pressure or catecholamine response to alcohol ingestion between subjects with and without a family history of hypertension, resting basal NE levels were significantly lower in the group with a family history, a finding consistent with the results of Bianchetti et al.
TABLE 2. Changes in Plasma Electrolytes, Cortisol, Plasma Renin Activity, Packed Cell Volume, and Weight in 16 Normotensive Subjects over 5 Hours of Alcohol or Placebo Administration

<table>
<thead>
<tr>
<th>Variable</th>
<th>Time (hr)</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium (mEq/L)</td>
<td>A</td>
<td>140 ± 1</td>
<td>139 ± 1</td>
<td>140 ± 1</td>
<td>141 ± 1</td>
<td>141 ± 1</td>
<td>143 ± 1</td>
</tr>
<tr>
<td></td>
<td>P</td>
<td>140 ± 1</td>
<td>140 ± 1</td>
<td>139 ± 1</td>
<td>140 ± 1</td>
<td>141 ± 1</td>
<td>142 ± 1</td>
</tr>
<tr>
<td>Potassium (mEq/L)</td>
<td>A</td>
<td>4.7 ± 0.1</td>
<td>4.8 ± 0.1</td>
<td>4.5 ± 0.1</td>
<td>4.6 ± 0.1</td>
<td>4.4 ± 0.1</td>
<td>4.3 ± 0.2</td>
</tr>
<tr>
<td></td>
<td>P</td>
<td>4.7 ± 0.1</td>
<td>4.7 ± 0.1</td>
<td>4.5 ± 0.1</td>
<td>4.5 ± 0.1</td>
<td>4.7 ± 0.1</td>
<td>4.4 ± 0.5</td>
</tr>
<tr>
<td>Urea (mg/dl)</td>
<td>A</td>
<td>33.0 ± 1.2</td>
<td>29.4 ± 1.2</td>
<td>28.2 ± 1.2</td>
<td>27.0 ± 1.2</td>
<td>27.0 ± 1.2</td>
<td>27.0 ± 1.8</td>
</tr>
<tr>
<td></td>
<td>P</td>
<td>31.2 ± 1.2</td>
<td>31.2 ± 1.2</td>
<td>30.6 ± 1.2</td>
<td>30.6 ± 1.2</td>
<td>28.8 ± 1.2</td>
<td>29.4 ± 1.8</td>
</tr>
<tr>
<td>Calcium (mEq/L)</td>
<td>A</td>
<td>4.52 ± 0.06</td>
<td>4.42 ± 0.06</td>
<td>4.38 ± 0.08*</td>
<td>4.34 ± 0.06*</td>
<td>4.38 ± 0.06*</td>
<td>4.42 ± 0.06*</td>
</tr>
<tr>
<td></td>
<td>P</td>
<td>4.42 ± 0.06</td>
<td>4.46 ± 0.06</td>
<td>4.42 ± 0.06</td>
<td>4.42 ± 0.06</td>
<td>4.48 ± 0.06</td>
<td>4.46 ± 0.06</td>
</tr>
<tr>
<td>Cortisol (µg/dl)</td>
<td>A</td>
<td>11.0 ± 0.7</td>
<td>7.7 ± 0.7†</td>
<td>7.0 ± 0.7†</td>
<td>7.6 ± 0.8†</td>
<td>8.7 ± 0.7†</td>
<td>8.3 ± 0.6</td>
</tr>
<tr>
<td></td>
<td>P</td>
<td>10.8 ± 0.9</td>
<td>8.4 ± 0.8†</td>
<td>8.2 ± 0.9</td>
<td>8.5 ± 0.9</td>
<td>10.4 ± 1.8</td>
<td>9.2 ± 0.8</td>
</tr>
<tr>
<td>PRA (nmol/L/hr)</td>
<td>A</td>
<td>1.8 ± 0.3</td>
<td>1.9 ± 0.3</td>
<td>2.3 ± 0.6</td>
<td>1.8 ± 0.4</td>
<td>2.6 ± 0.8</td>
<td>2.7 ± 0.5</td>
</tr>
<tr>
<td></td>
<td>P</td>
<td>2.2 ± 0.3</td>
<td>2.1 ± 0.5</td>
<td>2.1 ± 0.4</td>
<td>2.2 ± 0.6</td>
<td>2.6 ± 0.6</td>
<td>2.6 ± 0.5</td>
</tr>
<tr>
<td>PCV (%)</td>
<td>A</td>
<td>42.2 ± 0.7</td>
<td>42.5 ± 0.9</td>
<td>42.9 ± 0.9</td>
<td>42.6 ± 0.9</td>
<td>42.7 ± 0.9</td>
<td>42.2 ± 0.6</td>
</tr>
<tr>
<td></td>
<td>P</td>
<td>41.5 ± 0.8</td>
<td>41.7 ± 0.8</td>
<td>41.2 ± 0.8</td>
<td>41.3 ± 0.8</td>
<td>41.2 ± 0.9</td>
<td>41.0 ± 0.8</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>A</td>
<td>77.1 ± 2.2</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>76.1 ± 2.1</td>
</tr>
<tr>
<td></td>
<td>P</td>
<td>76.8 ± 2.3</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>76.8 ± 2.2</td>
</tr>
</tbody>
</table>

Values are means ± SEM. A = alcohol; P = placebo; PRA = plasma renin activity; PCV = packed cell volume. *p < 0.01, †p < 0.05, compared with values at Time 0.

This finding suggests that the group with a family history may have an increased cardiovascular end-organ sensitivity to NE. Subjects with a family history of hypertension have been found to have higher plasma albumin levels than those with no family history, suggesting that they have a decreased plasma volume, although no such differences were found in our study. These results must be interpreted with caution, however, in view of the small number of subjects involved and the possibility of misclassification of subjects with and without a family history of hypertension. All parents labeled as hypertensive were found to be so by their general practitioner, though we have no further information on those parents labeled normotensive. As blood pressure and NE changes were small and consistent between the two groups, it is unlikely that misclassification affected the results, though this cannot be completely excluded.

Our results on the NE changes with alcohol are at variance with some studies that have shown a small but significant elevation and in accord with others that failed to show any rise. This difference may be due to the length of the study, as most observers have only investigated changes for 2 hours following alcohol ingestion. Ireland et al. noted that alcohol ingestion had no effect on plasma E levels but a small increase in plasma NE levels in normotensive moderate alcohol consumers and that these changes lagged behind the rise in blood pressure. In a further similar study, control levels of NE were higher than those seen following alcohol ingestion. Peripheral venous blood NE levels may be a relatively insensitive index of sympathetic nervous activity since plasma concentrations of catecholamines depend on the rate of release as well as on plasma clearance. Eisenhofer et al. have shown that alcohol decreases plasma clearance of NE, and this mechanism may explain the raised levels reported previously. Release of NE is also calcium-dependent, but to our knowledge the effect of alcohol on plasma calcium and therefore NE release has not been taken into account in previous work.

TABLE 3. Urinary Volume and Sodium and Potassium Excretion in 16 Subjects During Alcohol and Placebo Ingestion

<table>
<thead>
<tr>
<th>Variable</th>
<th>Time (hr)</th>
<th>0–1</th>
<th>0–5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Volume (ml)</td>
<td>A</td>
<td>475 ± 56</td>
<td>1459 ± 41</td>
</tr>
<tr>
<td></td>
<td>P</td>
<td>220 ± 42*</td>
<td>760 ± 29*</td>
</tr>
<tr>
<td>Sodium (mEq)</td>
<td>A</td>
<td>17.1 ± 2.1</td>
<td>59.9 ± 1.7</td>
</tr>
<tr>
<td></td>
<td>P</td>
<td>11.5 ± 2.4</td>
<td>53.9 ± 2.2</td>
</tr>
<tr>
<td>Potassium (mEq)</td>
<td>A</td>
<td>7.4 ± 0.7</td>
<td>23.8 ± 0.7</td>
</tr>
<tr>
<td></td>
<td>P</td>
<td>12.2 ± 4.1</td>
<td>32.6 ± 2.5</td>
</tr>
</tbody>
</table>

Values are means ± SEM. A = alcohol; P = placebo. *p < 0.01, compared with values during alcohol ingestion (Student’s t test).
Alcohol can cause vasodilation of skin vasculature, which is probably sympathetically mediated. However, it has a vasoconstrictor effect on the arterioles in muscle beds that is unaffected by sympathectomy, suggesting that vasoconstriction is a direct effect of alcohol. The magnitude of this effect is dependent not only on alcohol concentration (levels seen during moderate intoxication can cause spasm of cerebral arterioles) but also on environmental temperature. Altura et al. showed that alcohol had a direct vasoconstrictor action on certain arterioles and venules that could be prevented by the use of calcium entry blockers. We have noted a fall in total plasma calcium levels with alcohol ingestion, but this hypocalcemic effect lasted longer than the elevation in blood pressure. The effects of alcohol on plasma calcium levels are not well recognized in humans, but similar findings have been reported in animals. The hypocalcemic action of alcohol may be due to the movement of calcium from the blood to tissues with a rise in intracellular calcium. Although urinary calcium levels were not measured in the present study, others have found no rise in urinary calcium after alcohol ingestion. At the cellular level, alcohol acutely inhibits Na+, K+-adenosine triphosphatase activity, leading to an increase in intracellular sodium, which could in turn lead to an inhibition of the slow Na+-Ca2+ pump and to an increase in intracellular calcium. Even a small increase in cytosolic calcium ion activity might facilitate smooth muscle contraction, either directly or by potentiating the vasoconstrictor action of circulating neurohumoral agents such as NE. At low concentrations, ethanol potentiates the action of NE and may therefore act synergistically with changes in intracellular calcium to cause the initial pressor action of alcohol.

In the present study, plasma urea fell after alcohol ingestion, but no change was seen with placebo. Gill et al. noted similar changes in urea and suggested that the alcohol-induced diuresis may have led to increased urea clearance. Linkola et al. noted an initial suppression of plasma renin activity following alcohol ingestion and found that plasma renin activity rose only during the hangover period. Changes in the renin-angiotensin-aldosterone axis do not appear to account for the rise in blood pressure with alcohol ingestion. Plasma cortisol levels fell with both treatments in the present study. A similar fall has been noted by other workers, and levels usually do not rise until the withdrawal period. The acute, alcohol-induced elevations of plasma cortisol have been reported to be related to the degree of discomfort and usually are not encountered with blood alcohol levels below 100 mg/dl. No increase in plasma cortisol was noted in the present study.

Our results indicate that the acute pressor effect of alcohol in normotensive subjects is small, short-lived, independent of family history of hypertension, and not directly sympathetically mediated. Alcohol likely has a direct vasoconstrictor effect that could result from calcium ion fluxes in arteriolar smooth muscle cells, but this requires further investigation. It is still uncertain whether the mechanisms underlying hypertension in regular drinkers are due to similar changes or whether sympathetic overactivity is responsible.

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