Evidence for a Direct Effect of Alcohol Consumption on Blood Pressure in Normotensive Men
A Randomized Controlled Trial

IAN B. PUDEY, LAWRENCE J. BEILIN, ROBERT VAN DONGEN, IAN L. ROUSE, AND PENNY ROGERS

SUMMARY A direct pressor effect of alcohol is proposed as the basis for the association between regular alcohol consumption and an increase in blood pressure found in population studies. To examine this further, a randomized controlled crossover trial of the effects of varying alcohol intake on blood pressure in 46 healthy male drinkers was conducted. From an average of 336 ml of ethanol per week, alcohol consumption was reduced by 80% for 6 weeks by drinking a low alcohol content beer alone. This reduction was associated with a significant reduction in systolic and diastolic blood pressure (p < 0.001 and p < 0.05 respectively). The mean difference in supine systolic blood pressure during the last 2 weeks of normal or low alcohol intake was 3.8 mm Hg, which correlated significantly with change in alcohol consumption (r = 0.53, p < 0.001). Reduction of alcohol intake also caused a significant decrease in weight (p < 0.001). After adjustment for weight change, an independent effect of alcohol on systolic but not diastolic blood pressure was still evident, with a 3.1 mm Hg fall predicted for a decrease in consumption from 350 ml of ethanol equivalent per week to 70 ml per week (p < 0.01). Systolic blood pressure rose again when normal drinking habits were resumed. These results provide clear evidence for a direct and reversible pressor effect of regular moderate alcohol consumption in normotensive men and suggest that alcohol may play a major role in the genesis of early stages of blood pressure elevation. (Hypertension 7: 707-713, 1985)

KEY WORDS • alcohol intake • blood pressure • hypertension

ALTHOUGH an association between alcohol consumption and increased blood pressure levels has been described in a number of population studies, the nature of this relationship remains uncertain.1-4 On the one hand, there is accumulating evidence that alcohol is a direct cause of blood pressure elevation,5-9 while on the other, there is the suggestion that some common constitutional trait predisposes to both alcohol use and hypertension.10

In an attempt to resolve this uncertainty, we have conducted a randomized crossover trial of the effects of varying alcohol intake on blood pressure in normotensive moderate drinkers. The results show highly significant and reversible changes in blood pressure with changes in alcohol consumption and provide what we believe is the most definitive evidence to date of a direct blood pressure raising effect of regular moderate alcohol consumption in normotensive healthy men.

Subjects and Methods

Seventy-one subjects responded to advertisements seeking men aged 25 to 55 years who were regular drinkers and prepared to participate in a trial designed to assess the effect of moderation of normal alcohol intake on blood pressure. They completed a questionnaire that assessed demographic details, medical history (including medications), and use of alcohol and tobacco. Alcohol consumption was estimated from a 7-day retrospective diary of both the previous week's and an average week's intake11 and was expressed as the equivalent in milliliters of absolute ethanol per week. Fourteen subjects were excluded because their consumption was less than 210 ml per week. Three respondents outside the specified age range and two taking β-blockers were also excluded. Of the 52 invit-
ed to participate, four declined, leaving 48 subjects at entry.

All subjects entered a 2-week familiarization period during which they were stratified and matched as closely as possible for age, Quetelet's index (height/weight), blood pressure, and alcohol consumption. They were then randomly allocated to one of two experimental groups; the first group substituted for their normal drinking habits a low alcohol content beer (Swan Lager: alcohol content 5.0% vol/vol) for each of the 6 weeks, but no other alcoholic drink was permitted. At the end of this period, normal drinking habits were resumed for a further 6 weeks during which six 750-ml bottles of normal alcohol content beer (Swan Lager: alcohol content 5.0% vol/vol) were provided each week.

The second group participated in a reverse design: they continued their normal drinking habits during the first 6 weeks and drank low alcohol beer for the second 6 weeks. Six bottles of normal or low alcohol content beer per week were provided in each of these periods as for the first group. Low alcohol beer is prepared commercially by vacuum distillation of normal alcohol content beer, which leaves the organic constituents essentially unaltered (Table 1).

At weekly intervals throughout the 14-week study, systolic and diastolic blood pressure and heart rate were measured with an automatic oscillometric recorder and printer (Dinamap, Critikon, Inc., Tampa, FL, USA).12 Readings were taken after 2, 4, and 6 minutes of supine rest and after 2, 4, and 6 minutes of standing. The same nursing sister was in attendance, and laboratory temperature was maintained at 23 to 25 °C. At each visit, the subjects completed a retrospective diary of the preceding week’s consumption. The investigators were not blinded as to whether subjects were in the low or normal alcohol intake periods.

A more extensive assessment was carried out at the completion of the 2-week familiarization period and at 3, 6, 9, and 12 weeks. After the subject had been weighed and blood pressure measurements obtained, a heparinized needle was inserted into a forearm vein and blood sampled for a full blood picture and liver function tests. High density lipoprotein cholesterol levels were assayed at Weeks 0, 6, and 12. During the familiarization period and at the end of each 6 weeks, subjects completed a questionnaire assessing changes in diet, tea and coffee consumption, smoking habit, and physical activity. Changes in anxiety levels were monitored by the A-state anxiety inventory of Spielberger and colleagues.13

Results are summarized as means ± sd. The effect of change in alcohol intake on blood pressure, heart rate, and biochemical indicators of alcohol intake were evaluated using the method of Hills and Armitage14 for analysis of two-period crossover designs. Average individual blood pressure and heart rates at the end of each intervention period were calculated as the mean of the three determinations made in the final 2 weeks. The significance of treatment-period interaction, period effects, and treatment effects (low or normal alcohol intake) was assessed using the methods described by Hills and Armitage.14 Significance levels were evaluated from pooled estimates of the variance and reference to the t distribution.

Differences in blood pressure, heart rate, and other baseline variables between groups were evaluated by unpaired t tests, and association between variables was evaluated by Pearson’s product-moment correlations. Changes within groups were evaluated using paired t tests.

The relationship between change in blood pressure and heart rate and the change in body weight, alcohol intake, and a number of objective measures of alcohol intake were examined using correlation and multiple regression analyses.

### Results

Forty-five subjects completed the study; 2 subjects from Group 2 withdrew during the familiarization period and 1 subject from Group 1 withdrew 2 weeks before the conclusion of the study. Initial characteristics of all subjects are presented in Table 2 and reveal no substantial differences between the groups. The mean reported alcohol consumption for all subjects was 336.2 ± 137.7 (sd) ml/week with 35% drinking predominantly beer; 44%, beer and wine; 6%, beer and spirits; and 15%, beer, wine, and spirits. There was no correlation between weekly alcohol intake during the familiarization period and systolic or diastolic blood pressure.

#### Changes in Blood Pressure

Reduction in alcohol intake by both groups was associated with falls in supine systolic and diastolic blood pressure (Figure 1), mostly during the first 2 weeks, and was maximal by 5 weeks. Recommencement of normal drinking habits by the first group resulted in systolic blood pressure increasing once again to levels higher than those recorded during the familiarization period.

A significant reduction in systolic and diastolic blood pressure recorded after 2, 4, and 6 minutes of supine rest was evident when the average readings during the last 2 weeks of low or normal alcohol intake

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**Table 1. Comparative Analysis of Normal and Low Alcohol Content Lager**

<table>
<thead>
<tr>
<th>Constituent</th>
<th>Light</th>
<th>Normal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alcohol (% vol/vol)</td>
<td>0.90</td>
<td>5.00</td>
</tr>
<tr>
<td>Carbohydrate (g/100 g)</td>
<td>3.6</td>
<td>4.4</td>
</tr>
<tr>
<td>Energy (kJ/L)</td>
<td>800</td>
<td>1720</td>
</tr>
<tr>
<td>Calcium (mg/L)</td>
<td>45</td>
<td>40</td>
</tr>
<tr>
<td>Sodium (mg/L)</td>
<td>78</td>
<td>60</td>
</tr>
<tr>
<td>Potassium (mg/L)</td>
<td>400</td>
<td>350</td>
</tr>
<tr>
<td>Protein (g/L)</td>
<td>3.40</td>
<td>3.75</td>
</tr>
</tbody>
</table>
Table 2. Characteristics of Subjects Before Study

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group 1 (n = 24)</th>
<th>Group 2 (n = 22)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>34.8±7.6</td>
<td>35.3±8.3</td>
</tr>
<tr>
<td>Quetelet's index (kg/m²)</td>
<td>25.1±2.7</td>
<td>25.0±3.0</td>
</tr>
<tr>
<td>Alcohol consumption (ml/wk)</td>
<td>330.8±158.7</td>
<td>342.3±112.6</td>
</tr>
<tr>
<td>Supine systolic BP (mm Hg)*</td>
<td>133.7±10.3</td>
<td>132.5±7.4</td>
</tr>
<tr>
<td>Supine diastolic BP (mm Hg)*</td>
<td>75.4±8.8</td>
<td>75.8±6.4</td>
</tr>
<tr>
<td>Supine heart rate (beats/min)*</td>
<td>66.6±7.7</td>
<td>70.0±10.1</td>
</tr>
<tr>
<td>Tea and coffee consumption (cups/wk)</td>
<td>35.2±3.1</td>
<td>32.4±4.2</td>
</tr>
<tr>
<td>Smoking status</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Smoker</td>
<td>7</td>
<td>6</td>
</tr>
<tr>
<td>Exsmoker</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Nonsmoker</td>
<td>12</td>
<td>11</td>
</tr>
<tr>
<td>Cigarette consumption (cigarettes/day)</td>
<td>15.6±5.2</td>
<td>13.3±4.0</td>
</tr>
</tbody>
</table>

Values (except smoking status) represent means ± SD. BP = blood pressure. *Mean of three values recorded during 2-week familiarization period, each recorded after 6 minutes of supine rest.

The blood pressure lowering effect of reducing alcohol intake was greatest for systolic blood pressure (6 min supine, 3.8 mm Hg; 2 min erect, 4.4 mm Hg); the corresponding falls in diastolic pressure were smaller (1.4 mm Hg and 2.5 mm Hg respectively; see Table 3). Changes in both systolic and diastolic blood pressure were significantly correlated (r = 0.53, p < 0.001 and r = 0.30, p < 0.05 respectively) with reported changes in alcohol consumption, as well as changes in the biochemical and hematological markers of alcohol consumption (Table 4).

The changes in blood pressure were unaffected by the order in which low alcohol content beer was administered, and there was no evidence of a period-treatment interaction. A familiarization effect was evident, however, in Group 2 with significant falls in supine systolic and diastolic (6 min) blood pressures (p < 0.05 and p < 0.01 respectively) and erect systolic (2 min) blood pressure (p < 0.05) while normal drinking habits were continued (Table 5).

The fall in blood pressure with reduction of alcohol consumption, though highly significant for the group as a whole, was not uniform, and 15 of the 46 participants showed either no change or modest increases in blood pressure during reduction of alcohol consumption.

Analyses of questionnaire responses revealed no change in the level of physical activity, diet, smoking habits, or anxiety state score. There was a significant weight loss, however, with an average 0.7-kg reduction during restriction of alcohol intake (p < 0.001; see Table 3). This significant reduction was not evident until 6 weeks, and no change was evident at 3 weeks.
TABLE 3. Effect of Change in Alcohol Intake on Body Weight, Blood Pressure, and Heart Rate

<table>
<thead>
<tr>
<th>Variable</th>
<th>Familiarization (n = 46)</th>
<th>Low alcohol (n = 46)</th>
<th>Normal alcohol (n = 45)</th>
<th>Average treatment effect* (n = 45)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight (kg)</td>
<td>78.9 (11.9)</td>
<td>78.0 (11.8)</td>
<td>78.7 (12.2)</td>
<td>-0.77</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Systolic BP (mm Hg)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Supine</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 min</td>
<td>136 (8.5)</td>
<td>131.3 (7.2)</td>
<td>133.9 (8.3)</td>
<td>-2.55</td>
<td>0.0018</td>
</tr>
<tr>
<td>4 min</td>
<td>134.2 (8.6)</td>
<td>128.9 (7.4)</td>
<td>131.6 (8.5)</td>
<td>-2.58</td>
<td>0.0027</td>
</tr>
<tr>
<td>6 min</td>
<td>133.1 (9.0)</td>
<td>126.5 (7.7)</td>
<td>130.4 (7.9)</td>
<td>-3.80</td>
<td>0.0006</td>
</tr>
<tr>
<td>Erect</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 min</td>
<td>130.6 (9.6)</td>
<td>123.5 (7.6)</td>
<td>128.0 (9.2)</td>
<td>-4.39</td>
<td>0.0002</td>
</tr>
<tr>
<td>4 min</td>
<td>130.2 (8.7)</td>
<td>124.9 (7.7)</td>
<td>126.7 (8.3)</td>
<td>-1.69</td>
<td>NS</td>
</tr>
<tr>
<td>6 min</td>
<td>131.2 (9.6)</td>
<td>125.2 (7.3)</td>
<td>127.0 (8.2)</td>
<td>-1.78</td>
<td>NS</td>
</tr>
<tr>
<td>Diastolic BP (mm Hg)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Supine</td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>2 min</td>
<td>75.4 (8.1)</td>
<td>72.2 (6.4)</td>
<td>74.3 (8.0)</td>
<td>-1.88</td>
<td>0.019</td>
</tr>
<tr>
<td>4 min</td>
<td>75.6 (7.7)</td>
<td>71.5 (6.9)</td>
<td>73.6 (7.7)</td>
<td>-1.99</td>
<td>0.009</td>
</tr>
<tr>
<td>6 min</td>
<td>75.6 (7.6)</td>
<td>71.6 (6.8)</td>
<td>73.2 (7.8)</td>
<td>-1.43</td>
<td>0.044</td>
</tr>
<tr>
<td>Erect</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 min</td>
<td>80.5 (8.9)</td>
<td>77.0 (7.4)</td>
<td>79.5 (8.9)</td>
<td>-2.49</td>
<td>0.003</td>
</tr>
<tr>
<td>4 min</td>
<td>81.0 (9.7)</td>
<td>78.1 (7.0)</td>
<td>78.4 (8.8)</td>
<td>-0.14</td>
<td>NS</td>
</tr>
<tr>
<td>6 min</td>
<td>81.7 (9.6)</td>
<td>78.3 (6.7)</td>
<td>79.2 (8.5)</td>
<td>-0.84</td>
<td>NS</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Supine</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 min</td>
<td>67.8 (9.8)</td>
<td>64.5 (9.4)</td>
<td>67.5 (8.0)</td>
<td>-2.75</td>
<td>0.002</td>
</tr>
<tr>
<td>4 min</td>
<td>67.9 (9.5)</td>
<td>64.8 (9.7)</td>
<td>67.3 (8.4)</td>
<td>-2.14</td>
<td>0.023</td>
</tr>
<tr>
<td>6 min</td>
<td>68.3 (9.0)</td>
<td>65.0 (9.3)</td>
<td>67.9 (8.0)</td>
<td>-2.60</td>
<td>0.003</td>
</tr>
<tr>
<td>Erect</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 min</td>
<td>77.5 (9.7)</td>
<td>77.0 (9.2)</td>
<td>78.7 (10.7)</td>
<td>-1.88</td>
<td>NS</td>
</tr>
<tr>
<td>4 min</td>
<td>78.5 (9.7)</td>
<td>77.4 (8.9)</td>
<td>78.7 (8.6)</td>
<td>-1.41</td>
<td>NS</td>
</tr>
<tr>
<td>6 min</td>
<td>78.7 (10.1)</td>
<td>78.1 (8.3)</td>
<td>79.7 (9.2)</td>
<td>-1.67</td>
<td>NS</td>
</tr>
</tbody>
</table>

Results are means (so given in parentheses) based on the averages of three readings during the last 2 weeks of each treatment period. p values are for significance of the average treatment effect, according to the method of Hills and Armitage.4

BP = blood pressure; NS = not significant.

*Average treatment effect = (dA + dB)/2, where dA = mean difference between Periods 1 and 2 in Group 1 and dB = mean difference between Periods 2 and 1 in Group 2.

when a blood pressure fall was already present. The change in weight between normal and low alcohol intake was significantly (but weakly) correlated with change in blood pressure (p < 0.05; see Table 4). The change in blood pressure was not related to age, initial Quetelet's index, or baseline alcohol consumption.

Multiple linear regression analysis demonstrated a highly significant effect of change in alcohol consumption on change in supine (6 min) systolic blood pressure, which was independent of the change in weight (F1,41 = 10.05, p = 0.003). An independent effect of alcohol consumption was not demonstrated for supine (6 min) diastolic blood pressure (F1,41 = 0.88, p = 0.355). Based on the regression coefficients, the change in supine systolic blood pressure was estimated to be 1.1 mm Hg for each 100 ml/week change in alcohol consumption. When the readings obtained after 2, 4, and 6 minutes of supine rest were averaged and the analysis repeated, similar results were obtained, with a significant independent effect of alcohol on systolic (F1,41 = 7.86, p = 0.008), but not diastolic (F1,41 = 2.35, p = 0.133), blood pressure.

A similar pattern emerged with analysis of the readings obtained after the subject had been standing for 2 minutes, with an independent effect of alcohol on systolic (F1,41 = 6.79, p = 0.013), but not diastolic (F1,41 = 0.67, p = 0.419), blood pressure. When the readings obtained after 2, 4, and 6 minutes of standing...
were averaged, an independent effect of alcohol on erect blood pressure was no longer demonstrated (systolic $F_{1,41} = 2.83, p = 0.10$; diastolic $F_{1,41} = 2.52, p = 0.120$).

A significant fall in the heart rate obtained after 2, 4, and 6 minutes of supine rest was evident during reduction of alcohol intake; in contrast, no change was noted in the corresponding erect readings (see Table 3).

**Monitoring of Compliance**

Retrospective weekly diaries revealed an approximate 80% reduction in alcohol intake when low alcohol beer was substituted (Figure 2). There were only eight reported protocol violations (by 6 subjects), where an additional alcoholic beverage was consumed. During continuation of normal drinking habits, an increase in alcohol consumption relative to the familiarization period was seen: familiarization, 336.3 ± 137.7 ml/wk; sixth week of normal alcohol intake, 425.8 ± 146.9 ml/wk (paired t test, $p < 0.05$; see Figure 2). Significant falls in γ-glutamyl transferase levels ($p < 0.001$), mean corpuscular volume ($p < 0.01$), and high density lipoprotein cholesterol levels ($p < 0.001$) accompanied consumption of low alcohol beer alone (see Figure 2) and were highly correlated with the self-reported change in alcohol consumption calculated from retrospective weekly diaries (see Table 4). The change in these indices was also significantly but less strongly correlated with change in blood pressure (see Table 4).

**Discussion**

Despite accumulating epidemiological and clinical evidence that alcohol use may increase blood pressure, this relationship has not been widely accepted because of a lack of any adequately controlled trials of the effect of reducing moderate alcohol consumption on blood pressure. The present study provides evidence that important and reversible changes in blood pressure occur when normotensive men change their alcohol intake by approximately 80% over 6 weeks.

How reliably did subjects adhere to modification of their alcohol intake? Evidence for a substantial reduction in alcohol consumption during the low alcohol period is provided by the fall in γ-glutamyl transferase levels, mean corpuscular volume, and high density lipoprotein cholesterol levels. The correlation of these changes with the self-reported reduction in alcohol intake, and their reversal with resumption of normal drinking habits, provides further support.

**Table 4.** Correlation Matrix: Changes in Blood Pressure, Heart Rate, Weight, and Indices of Alcohol Consumption from the End of Period 1 to the End of Period 2

<table>
<thead>
<tr>
<th>Variable</th>
<th>Weight change</th>
<th>RD</th>
<th>MCV</th>
<th>HDL</th>
<th>GGT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Supine (6 min)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic BP</td>
<td>0.33*</td>
<td>0.53†</td>
<td>0.30*</td>
<td>0.28</td>
<td>0.52†</td>
</tr>
<tr>
<td>Diastolic BP</td>
<td>0.30*</td>
<td>0.30*</td>
<td>0.16</td>
<td>0.32</td>
<td>0.42*</td>
</tr>
<tr>
<td>Heart rate</td>
<td>0.33*</td>
<td>0.43‡</td>
<td>0.15</td>
<td>0.12</td>
<td>0.22</td>
</tr>
<tr>
<td>Erect (2 min)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic BP</td>
<td>0.36*</td>
<td>0.50‡</td>
<td>0.48†</td>
<td>0.33*</td>
<td>0.43‡</td>
</tr>
<tr>
<td>Diastolic BP</td>
<td>0.31*</td>
<td>0.40‡</td>
<td>0.38‡</td>
<td>0.32*</td>
<td>0.38‡</td>
</tr>
<tr>
<td>Heart rate</td>
<td>0.22</td>
<td>0.20</td>
<td>-0.05</td>
<td>-0.01</td>
<td>0.05</td>
</tr>
<tr>
<td>RD</td>
<td>0.54†</td>
<td>-</td>
<td>0.57†</td>
<td>0.63†</td>
<td>0.58†</td>
</tr>
</tbody>
</table>

Values represent Pearson's product-moment correlation coefficient.

RD = self-reported consumption in a weekly retrospective diary; MCV = mean corpuscular volume; HDL = high density lipoprotein cholesterol; GGT = γ-glutamyl transferase; BP = blood pressure.

*p ≤ 0.05, †p ≤ 0.01, ‡p ≤ 0.001, §p < 0.01, two-tailed test of significance.

**Table 5.** Effect of Change in Alcohol Intake on Blood Pressure (BP) and Heart Rate for Individual Groups

<table>
<thead>
<tr>
<th>Variable</th>
<th>Familiarization (n = 24)</th>
<th>Low alcohol (n = 24)</th>
<th>Normal alcohol (n = 23)</th>
<th>Familiarization (n = 22)</th>
<th>Normal alcohol (n = 22)</th>
<th>Low alcohol (n = 22)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Supine (6 min)*</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic BP (mm Hg)</td>
<td>133.7 ± 10.3</td>
<td>127.2 ± 8.1†</td>
<td>131.9 ± 7.5‡</td>
<td>132.5 ± 7.4</td>
<td>128.9 ± 8.2§</td>
<td>125.8 ± 7.2</td>
</tr>
<tr>
<td>Diastolic BP (mm Hg)</td>
<td>75.4 ± 8.8</td>
<td>71.5 ± 7.4‡</td>
<td>72.9 ± 8.5</td>
<td>75.8 ± 6.4</td>
<td>73.4 ± 7.3j</td>
<td>71.7 ± 6.3</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>66.6 ± 7.7</td>
<td>64.5 ± 9.3</td>
<td>66.5 ± 8.4</td>
<td>70.0 ± 10.1</td>
<td>69.3 ± 7.6</td>
<td>65.6 ± 9.5§</td>
</tr>
<tr>
<td>Erect (2 min)*</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic BP (mm Hg)</td>
<td>129.2 ± 10.3</td>
<td>123.7 ± 7.7†</td>
<td>127.4 ± 8.9§</td>
<td>132.2 ± 8.7</td>
<td>128.6 ± 9.8§</td>
<td>123.2 ± 7.8†</td>
</tr>
<tr>
<td>Diastolic BP (mm Hg)</td>
<td>80.3 ± 9.4</td>
<td>77.8 ± 6.6§</td>
<td>79.4 ± 8.7</td>
<td>80.8 ± 8.7</td>
<td>79.7 ± 9.4</td>
<td>76.1 ± 8.2‡</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>75.7 ± 8.6</td>
<td>76.6 ± 9.3</td>
<td>78.0 ± 12.5</td>
<td>79.5 ± 8.5</td>
<td>79.5 ± 8.5</td>
<td>77.4 ± 9.3</td>
</tr>
</tbody>
</table>

Values represent means ± sd.

*Average of three readings during familiarization period and last three readings from Periods 1 and 2.

†p ≤ 0.001, †p ≤ 0.05, ‡p ≤ 0.01 (paired t test): familiarization to low alcohol intake (Group 1) or normal alcohol intake (Group 2).

§p < 0.01, ‡p ≤ 0.05 (paired t test): low alcohol intake to normal alcohol intake.
In this study, the effect of alcohol appeared to be independent of other factors that might influence blood pressure for several reasons. The low alcohol beer had 50% less energy content than normal beer and not unexpectedly led to a small but significant decrease in body weight. However, this weight loss alone was not sufficient to explain the fall in systolic blood pressure. Furthermore, the largest fall in blood pressure was observed within 2 weeks of reducing alcohol intake, before there was any change in weight. Another confounding variable may have been stress, which has been postulated to predispose to both alcohol use and hypertension. Using the anxiety inventory of Spielberger and colleagues, no change in anxiety levels was seen during moderation of alcohol intake.

It has been suggested that the correlation between alcohol and blood pressure may reflect the drinker’s increased salt intake in the form of salted snacks or, alternatively, because of the salt content in beer itself. Both beers were of equivalent sodium content; the low alcohol beer was widely available in bars and restaurants, and it is unlikely that intake of salty foods altered substantially. The possibility that alcohol dulls taste appreciation and leads to increased salting of food has not been ruled out. No other major consistent dietary alteration was evident from analysis of the questionnaires.

The possibility exists that participants may have guessed the hypothesis under investigation and influenced the outcome. Participants were not informed of our expectations, and the use of an automatic device to record blood pressure eliminated observer bias. However, complete objectivity would have been possible only with a double-blind design, which would pose considerable difficulty in a study like this.

The fall in blood pressure after reducing alcohol intake was of early onset; the largest fall occurred within the first 2 weeks. This finding is consistent with results from a recent uncontrolled study in hypertensive subjects, in which the pressor effect of alcohol was found to be rapidly reversible. In that study, follow-up after cessation of alcohol intake was for a short period only, whereas we have shown that the fall in blood pressure was sustained for at least 5 weeks. Resumption of normal drinking habits led to an increase in blood pressure within 2 weeks; however, changes in alcohol consumption did not affect blood pressure in all subjects, which suggests important individual susceptibility.

During the low alcohol period, lower erect systolic blood pressure was only observed after 2 minutes of standing and not after 4 or 6 minutes of standing. This apparent anomaly may reflect increased efficiency of the baroreceptor mechanism with reduction of alcohol intake, with a more rapid return of systolic blood pressure toward supine values.

The importance of the fall in blood pressure after reduction in alcohol intake is best appreciated when comparisons are made with our findings in a cross-sectional study of male drinkers and teetotalers. In that study, systolic blood pressure was found to increase 1 mm Hg for each 70 ml of ethanol consumed per week and in men drinking more than 160 ml of ethanol/week, the prevalence of systolic hypertension was four times greater than that in nondrinkers. No independent effect of alcohol on diastolic blood pressure was demonstrated. In the present study, in subjects who drank a minimum of 210 ml of ethanol per week (3 drinks per day), the finding of a predominant effect on systolic blood pressure with a fall of 1.1 mm Hg for every 100-ml reduction in ethanol intake is therefore consistent with our previous observations.

A predominant effect of alcohol on systolic blood pressure has been found consistently in other cross-sectional studies and has also been observed in a recent short-term prospective trial in hypertensive drinkers. These studies demonstrated a lesser but significant influence on diastolic blood pressure as well. This difference in systolic and diastolic blood pressure response to regular alcohol use may be a clue to the underlying pressor mechanism and may imply more of an effect on cardiac output or fluid volume than on
vascular resistance. In this respect, we observed a fall in heart rate after alcohol consumption was decreased, and this may have been relevant to the finding of an effect on systolic rather than diastolic blood pressure.

The fall in blood pressure occurred in normotensive subjects, and given the increased prevalence of hypertension in drinkers, it is anticipated that hypertensive subjects would show an even greater blood pressure response to a similar change in alcohol intake. As the fall in blood pressure was directly correlated with the change in alcohol consumption, heavier drinkers would also be expected to have larger falls. Our previous observation that, in the age range 20 to 44 years, former heavy drinkers have similar blood pressure to life-long teetotalers suggests that the fall is likely to be sustained.

There is good evidence that relatively small decreases in the level of blood pressure in the community may be translated into substantial falls in both coronary artery and cerebrovascular disease risks, and it has been estimated that a downward shift of the whole blood pressure distribution by only 2 to 3 mm Hg would have the same impact on mortality as current antihypertensive therapy. If alcohol consumption is reduced from 350 ml/week to 70 ml/week — a target easily achieved by most participants in the present study — we predict from the regression coefficients an average fall of 3.1 mm Hg in systolic blood pressure, independent of the accompanying weight loss. Therefore, although the fall in blood pressure achieved by reducing alcohol intake may appear modest, it could have major implications for both the prevalence of hypertension and hypertension-associated morbidity.

The effects of reducing alcohol consumption on the risk of coronary vascular disease remain controversial, as theoretically the advantage of a fall in blood pressure might be offset by the disadvantage of a fall in high density lipoprotein cholesterol levels.

In conclusion, this study has provided firm evidence for a direct pressor effect of long-term alcohol consumption, by demonstrating a significant fall in blood pressure when normotensive men substituted a low alcohol beer for their normal "moderate" drinking habits. Encouragement of social use of beverages of lower alcohol content may have a substantial impact on the prevalence of hypertension and other alcohol-related disorders.

Acknowledgment

We thank Dr. J. Masarei for assistance with several of the biochemical analyses.

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Hypertension. 1985;7:707-713
doi: 10.1161/01.HYP.7.5.707

Hypertension is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0194-911X. Online ISSN: 1524-4563

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