Effects of Alcohol and Caloric Restrictions on Blood Pressure and Serum Lipids in Overweight Men

I.B. Puddey, M. Parker, L.J. Beilin, R. Vandongen, and J.R.L. Masarei

We have examined the independent and combined effects on blood pressure and blood lipids of alcohol restriction and weight loss in overweight male drinkers with a view to assessing overall effects on cardiovascular risk of two widely promoted nonpharmacological approaches for hypertension. Eighty-six men with a mean age of 44.3 years, a mean regular alcohol intake of 440 ml/wk (five or six standard drinks per day), a mean blood pressure of 137.4 mm Hg systolic and 84.8 mm Hg diastolic, and a mean body mass of 92.5 kg entered a controlled two-way factorial study. The subjects were randomly assigned to four groups for an 18-week intervention in which members of two groups drank only low-alcohol beer, thereby reducing their alcohol intake by 374 ml/wk, while those of the other two groups continued their normal alcohol intake. Within the low and normal alcohol intake groups subjects either continued their usual diet or reduced their caloric intake by 4,200–6,300 kJ/day (1,000–1,500 kcal/day) (with protein, fat, and carbohydrate provided as 15%, 30%, and 55% of total calories, respectively). Calorie reduction and alcohol restriction caused weight losses of 7.5 (p<0.001) and 2.1 (p<0.01) kg, respectively. Calorie reduction and alcohol restriction were associated with decreases in systolic blood pressure of 5.4 (p<0.001) and 4.8 (p<0.01) mm Hg, respectively, and in diastolic blood pressure of 4.2 (p<0.001) and 3.3 (p<0.01) mm Hg, respectively. The combined measures had additive effects on blood pressure reduction, giving decreases of 10.2 mm Hg systolic and 7.5 mm Hg diastolic supine and 14.3 mm Hg systolic and 7.5 mm Hg diastolic standing. With alcohol restriction, total cholesterol concentration fell 0.49 mmol/l (p<0.05), the triglyceride level fell 0.63 mmol/l (p<0.01), and the high density lipoprotein (HDL)-cholesterol concentration fell 0.15 mmol/l (p<0.001). With caloric reduction alone, total cholesterol concentration fell 0.40 mmol/l (p<0.07) and the triglyceride level fell 0.81 mmol/l (p<0.001), but the HDL-cholesterol concentration rose 0.11 mmol/l (p<0.05). The combined measures prevented a potentially adverse alcohol-related decrease in the HDL-cholesterol level and had additive effects in reducing total cholesterol and triglyceride concentrations. We conclude that the combination of reduced alcohol consumption and weight loss from calorie restriction leads to substantial and sustained reductions in blood pressure and to improved blood lipid profiles. These combined strategies offer a valuable approach to modification of cardiovascular risk in overweight male drinkers. (Hypertension 1992;20:533–541)

KEY WORDS • hypertension, essential • alcohol drinking • weight loss • blood pressure

A pproximately one third of Australian men and women are overweight,1 and 80% of Australian men regularly consume alcohol, with 14% consuming five or more drinks per day.1 These figures are similar to those found in other industrialized nations, and given such high rates of both obesity and excessive alcohol intake it is not surprising that these two aspects of lifestyle are thought to make a substantial overall contribution to the development of hypertension in Western communities.1-2 As a result, recommendations to reduce weight and cease heavy alcohol consumption are now becoming the cornerstones of current initial management of mild hypertension.3 Their relative role as public health measures in both the treatment of mild hypertension and the prevention of subsequent development of high blood pressure (BP) is often met with widespread pessimism about long-term compliance. Such pessimism may be misplaced, however, with sustained weight reduction through caloric restriction being both clinically achievable and resulting in predictable and worthwhile declines in BP in hypertensive patients.4-7 In several controlled trials, restriction of alcohol intake in moderate-to-heavy drinkers has also led to consistent and clinically relevant reductions in BP in both normotensive and hypertensive subjects.8-11

In the present study the independent and combined effects of these two nonpharmacological approaches on both BP and the serum lipid profile have been explored in overweight male drinkers whose BP was either mildly elevated or at the upper limit of the normotensive range. The primary aim was to assess whether a combined approach using both caloric and alcohol restric-
tions would have potentially additive or synergistic effects in lowering BP in such subjects. Second, we wished to determine whether the fall in BP with alcohol restriction, previously demonstrated only during short-term interventions (4–6 weeks), persists in the longer term. Finally, previous studies of alcohol restriction have found not only a fall in BP but a potentially disadvantageous fall in serum high density lipoprotein (HDL) cholesterol levels. In contrast, weight loss caused by caloric reduction has been reported to increase HDL cholesterol concentrations. We therefore postulated that using a hypocaloric diet in association with alcohol restriction would ameliorate or reverse the fall in HDL cholesterol levels seen with alcohol restriction alone.

Methods

Subjects

Overweight and moderately drinking men participated in an 18-week study of the combined effects of weight reduction and alcohol restriction on BP. Of the 527 respondents to media advertisements, 295 satisfied entry criteria of age between 25 and 70 years; minimum alcohol intake of 210 ml/wk (approximately three standard drinks per day); body mass index of >25 kg/m² or current weight greater than 120% of ideal weight for age; no current use of antihypertensive or nonsteroidal anti-inflammatory drugs; and no history of renal or hepatic disease, diabetes mellitus, myocardial infarction or coronary artery surgery, stroke, or substantial weight loss (>10 kg) in the preceding 12 months. On the basis of responses to a questionnaire seeking further demographic information, medical history, height and weight, and cigarette and alcohol consumption 261 subjects were seen in the clinic. Of these, 141 were excluded because they did not meet BP entry criteria (systolic BP >130 mm Hg and <160 mm Hg, diastolic BP >80 mm Hg and <105 mm Hg). BP was averaged from two sets of five readings taken at 2-minute intervals 1 week apart using an automatic oscillometric device (Dinamap 845XT, Critikon Inc., Tampa, Fla.). Of the remaining 120 subjects, 22 declined to participate further and 12 dropped out during the 2-week familiarization period, leaving 86 entering the intervention phase of the study.

Study Design

During an initial 2-week familiarization period, subjects continued their usual alcohol and caloric intake. They were then stratified and matched as closely as possible for age, body mass index, alcohol consumption, and BP before random assignment to one of four groups. Subjects in two groups continued their usual diet while subjects in the other two groups were given an individual dietary program to reduce caloric intake by 4,200–6,300 kJ/day (1,000–1,500 kcal/day). Subjects in one of the unchanged and one of the low calorie groups continued their usual alcohol intake, while those in the other two groups reduced their alcohol consumption by substituting a low-alcohol beer (0.9% alcohol by volume, Swan Special Light Lager, Bond Brewing, Perth, Australia) for their usual drink. To aid compliance, subjects in all four groups were supplied with four 750-ml bottles per fortnight of either low-alcohol beer or normal lager (5% alcohol by volume, Swan Lager). The diet/alcohol intervention continued over 18 weeks.

Dietary Compliance

At an initial interview with a diettian, subjects were given written and verbal instructions on how to keep detailed and accurate food records, with food weight and volume being measured whenever possible. Dietary intake was subsequently monitored by the same dietitian throughout the study, with completion of a 3-day diet record every fortnight. At each fortnightly interview this record was individually checked for clarity and reliability.

At a second interview and after review of the initial diet records, those subjects who had been allocated to the calorie-restricted diet received (with their wives present when possible) advice on reducing their intake by 4,200–6,300 kJ/day (1,000–1,500 kcal/day), with the aim of providing protein, fat, and carbohydrates as 15%, 30%, and 55% of energy, respectively. This was achieved by substituting low-fat alternatives for typical high-fat foods, increasing fruit and vegetable consumption, and substituting complex carbohydrates like whole-grain bread and cereals for refined carbohydrates. The subjects were encouraged to work with a target weight loss (approximately 7–8 kg) in mind and to adopt the whole program as a lifestyle change rather than a quick weight loss diet. The importance of involvement of spouses and families was also emphasized, particularly since the dietary changes were to a large extent dependent on the meal-preparer, most often the wife. The keeping of detailed and frequent food records together with plots of fortnightly progress on individual charts further aided compliance. All subjects were advised to make no major changes to sodium intake. Subjects in the unchanged diet groups were also seen fortnightly by the diettian, who determined both from the 3-day diet records and by interview whether there had been any alterations to their usual eating habits and reinforced at each visit that no changes were to occur. These subjects were offered a weight loss program on completion of the study.

The 24-hour urinary sodium, potassium, calcium, and creatinine excretions were monitored during the familiarization period and at weeks 4, 8, 12, and 16 during the intervention and assayed on a SMAC II multichannel autoanalyzer (Technicon Inc., Sydney, Australia).

Alcohol Compliance

Alcohol intake was assessed by three 7-day retrospective diaries completed during the familiarization period and at fortnightly intervals during the intervention and converted using standard tables to alcohol consumption in milliliters of ethanol per week. The plasma γ-glutamyl transpeptidase (γ-GT) concentration and the erythrocyte mean corpuscular volume were measured at weeks 0, 9, and 18 as possible biomarkers of a change in alcohol intake.
Blood Pressure Measurement

Heart rate and BP were measured with the Dinamap device at approximately the same time of day on 3 days during the familiarization period, fortnightly during the intervention, and on 3 days during the last 2 weeks of the study. A cuff size appropriate to the subject's arm size was selected, and the same cuff size was used thereafter for each subject. Subjects abstained from cigarettes, coffee, and vigorous exercise for at least 2 hours before the BP measurement. BP was averaged from 10 supine readings taken at 2-minute intervals for 20 minutes and five standing readings taken at 1-minute intervals for 5 minutes. The mean difference between the averaged BP during the 2 weeks of familiarization and the last 2 weeks of the intervention was used for comparisons between groups. For those subjects withdrawing before completion of the study, mean differences were based on the average BP during their final 2–4 weeks.

Serum Lipid-Lipoproteins

At weeks 0, 9, and 18, after an overnight fast and 20 minutes of supine rest, blood was sampled for cholesterol, triglyceride, and HDL cholesterol concentrations. Serum for determinations of the total cholesterol and triglyceride concentrations was analyzed enzymatically on a Cobas Mira analyzer, using reagents from Abbott, North Ryde, Australia, with coefficients of variation of 1.4% at 4.9 mmol/l and 3.6% at 0.9 mmol/l. The HDL cholesterol concentration was assayed on a heparin-manganese chloride supernatant with a coefficient of variation of 3.1% at 1.1 mmol/l.

Other Variables

A health and lifestyle questionnaire was completed the day before the intervention commenced, and a shorter questionnaire was completed at week 9 and at the end of the trial to assess possible changes in tea and coffee consumption (average number of cups consumed per day or week), smoking habit (cigarettes per day), or physical activity (score computed from the number of days per week the subject participated in five separate categories of vigorous physical exercise). Height was measured at baseline by means of a stadiometer, and body weight was recorded at every visit using a calibrated beam balance.

Statistical Analysis

A two-way analysis of variance model with interaction was used to compare changes in BP, weight, and serum lipid-lipoproteins among the four groups. All subjects who entered the intervention phase of the study were analyzed on an intention-to-treat basis in evaluating the independent contributions of alcohol restriction and low calorie diet to any change seen in BP or serum lipid-lipoproteins. Within-group comparisons were made using Bonferroni's method, with the t statistic calculated as the mean square error term from the two-way analysis of variance. One-way analysis of variance and χ² tests were used to test for differences in characteristics between the groups at baseline. An a posteriori calculation of the study had 93% and 96% power, respectively, at α=0.05 to detect an alcohol restriction or caloric reduction main effect on systolic BP of 5 mm Hg. Results are expressed as mean (95% confidence interval).

Results

After random assignment the four study groups remained well matched, with no significant differences in any baseline parameter with the exception of HDL cholesterol concentration (Table 1). Approximately 35% of all subjects were found to have a mean supine systolic BP of >140 mm Hg during the familiarization phase, while 26% had a mean supine diastolic BP of >90 mm Hg, and 17% had elevation of both systolic and diastolic BP. In addition, 64% and 45% of the subjects were outside the National Heart Foundation of Australia's recommended upper limits of 5.5 mmol/l and 1.8 mmol/l for serum cholesterol and triglycerides, respectively.

Subjects in the two groups assigned to consume low-alcohol beer during the intervention reported a drop in their usual alcohol intake of 374 (311–436) ml/wk, while in those continuing their usual alcohol intake there was a nonsignificant increase in consumption of 29 (2–59) ml/wk (Figure 1). There was no significant change in the weight of subjects maintained on their usual caloric and alcohol intake (Figure 2). Those who restricted their alcohol intake but made no change to their usual diet had a significant reduction in weight of 1.7 (0.4–3.0) kg (p<0.05), which was apparent within the first 6 weeks of the intervention and did not fall any further. Reducing the caloric intake but continuing the usual alcohol intake caused a weight loss of 7.0 (5.4–8.5) kg (p<0.001), while 9.6 (7.9–11.3) kg (p<0.001) was lost in subjects reducing their intake of both calories and alcohol. Relative to the baseline value, these losses within individual groups translated into a 2.1 (0.8–3.5) kg weight reduction attributable to alcohol restriction (p<0.01) and a 7.5 (6.1–8.8) kg weight loss attributable to caloric reduction (p<0.001) during the 18-week intervention. The majority of subjects in the calorie-restricted groups reached a steady state at a lower weight from approximately week 10 to week 14 of the intervention, weight having fallen from 94.3 (90.5–98.1) kg at baseline to 85.9 (81.8–90.1) kg at week 12. Mean weight continued to fall slightly, however, to 84.9 (80.7–89.1) kg at week 16 and 84.2 (79.9–88.5) by week 18 (Figure 2).

Both caloric reduction and alcohol restriction resulted independently in decreases in both systolic and diastolic BP (Figure 3). These decreases were evident after 4 weeks. The main effects (relative to baseline) of caloric reduction on supine BP were −5.4 (−7.9 to −2.9) mm Hg systolic (F₁,₁₂=12.1, p<0.001) and −4.2 (−6.2 to −2.2) mm Hg diastolic (F₁,₁₂=12.3, p<0.001). Larger changes were observed in standing BP: −9.2
(−12.2 to −6.2) mm Hg systolic \( F_{1,82}=24.4, p<0.001 \) and −4.8 \((-7.00 \text{ to } -2.6) \) mm Hg diastolic \( F_{1,82}=11.9, p<0.001 \). The main effects of alcohol restriction on supine BP were −4.8 \(-7.3 \text{ to } -2.3) \) mm Hg systolic \( F_{1,82}=9.8, p<0.01 \) and −3.3 \(-5.2 \text{ to } -1.3) \) mm Hg diastolic \( F_{1,82}=7.4, p<0.01 \). The changes in standing BP were similar: −5.1 \(-8.1 \text{ to } -2.1) \) mm Hg systolic \( F_{1,82}=7.3, p<0.01 \) and −4.2 \(-5.0 \text{ to } -0.6) \) mm Hg diastolic \( F_{1,82}=4.2, p<0.05 \).

The effects of the combined measures were additive (Figure 3), giving reductions of 10.2 mm Hg systolic and 7.5 mm Hg diastolic in supine BP and 14.3 mm Hg systolic and 7.5 mm Hg diastolic in standing BP relative to baseline values in subjects who participated in both interventions.

The effects of alcohol on supine systolic, but not diastolic, BP remained significant \( F_{1,81}=4.3, p<0.05 \) and \( F_{1,81}=3.0, p=0.087 \), respectively) when the effects of weight loss induced by alcohol restriction alone were taken into account by repeating the two-way analysis of variance with change in weight entered as a covariate.

Of the subjects with elevated diastolic BP (>140 mm Hg) identified at baseline, 100% of those who decreased both their caloric intake and alcohol consumption developed normal diastolic BP during the intervention, compared with 80% of those reducing calories alone, 75% of those restricting alcohol intake alone, and 57% of those making no changes. The corresponding results for those with elevated systolic BP (>140 mm Hg) were 80%, 40%, 63%, and 43%, respectively.

There was a significant fall in supine, but not standing, heart rate with both caloric reduction alone, 75% of those restricting alcohol intake alone, and 57% of those making no changes. The corresponding results for those with elevated systolic BP (>140 mm Hg) were 80%, 40%, 63%, and 43%, respectively.

Baseline supine systolic and diastolic BP was negatively correlated with the change in systolic and diastolic BP during the intervention \( R=0.41, p<0.001 \) and \( R=0.33, p=0.002 \), respectively. Baseline alcohol con-

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**TABLE 1. Initial Characteristics of Subjects After Random Assignment Into Four Groups**

<table>
<thead>
<tr>
<th></th>
<th>Normal alcohol intake</th>
<th></th>
<th>Reduced alcohol intake</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Normal diet (n=20)</td>
<td>Hypocaloric diet (n=22)</td>
<td>Normal diet (n=21)</td>
<td>Hypocaloric diet (n=23)</td>
</tr>
<tr>
<td>Age (years)</td>
<td>Mean 95% CI</td>
<td>Mean 95% CI</td>
<td>Mean 95% CI</td>
<td>Mean 95% CI</td>
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<td>45.4</td>
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<td>Weight (kg)</td>
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<td>87.4-99.7</td>
</tr>
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<td>27.5-29.9</td>
<td>29.6</td>
<td>28.2-30.9</td>
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<tr>
<td>Smokers (No.)</td>
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<td>5</td>
<td>4</td>
<td>5</td>
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<td>Alcohol consumption (ml/wk)</td>
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<td>328-578</td>
<td>393</td>
<td>309-477</td>
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<tr>
<td>Urinary Na⁺ excretion (mmol/day)</td>
<td>158</td>
<td>130-186</td>
<td>189</td>
<td>152-226</td>
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<tr>
<td>Systolic BP (mm Hg)</td>
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<td>130.2-141.8</td>
<td>136.8</td>
<td>133.1-140.5</td>
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<tr>
<td>Diastolic BP (mm Hg)</td>
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<td>80.7-88.2</td>
<td>85.0</td>
<td>81.9-88.1</td>
</tr>
<tr>
<td>Heart rate (bpm)</td>
<td>70.8</td>
<td>67.0-74.5</td>
<td>69.3</td>
<td>65.0-73.7</td>
</tr>
<tr>
<td>Cholesterol (mmol/l)</td>
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<td>5.4-6.3</td>
<td>5.7</td>
<td>5.2-6.2</td>
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<td>Triglyceride (mmol/l)</td>
<td>1.9</td>
<td>1.3-2.5</td>
<td>1.7</td>
<td>1.2-2.1</td>
</tr>
<tr>
<td>High density lipoprotein cholesterol (mmol/l)*</td>
<td>1.22</td>
<td>1.09-1.35</td>
<td>1.18</td>
<td>1.06-1.29</td>
</tr>
</tbody>
</table>

CI, confidence intervals; BP, blood pressure; bpm, beats per minute.

*p<0.05 by one-way analysis of variance.

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**FIGURE 1.** Line graph shows mean±SEM alcohol consumption for four study groups. ● ● ●, normal alcohol intake/normal caloric intake (n=20); ○ ○ ○, normal alcohol intake/low caloric intake (n=22); • • •, low alcohol intake/normal caloric intake (n=21); ○—○, low alcohol intake/low caloric intake (n=23).

**FIGURE 2.** Line graph shows mean±SEM change in weight for four study groups. ● ● ●, normal alcohol intake/normal caloric intake (n=20); ○ ○ ○, normal alcohol intake/low caloric intake (n=22); ○—○, low alcohol intake/normal caloric intake (n=21); ○—○, low alcohol intake/low caloric intake (n=23).
sumption and self-reported change in alcohol intake correlated with changes in systolic ($R=0.23$, $p<0.05$ and $R=0.44$, $p=0.003$, respectively) and diastolic ($R=0.27$, $p<0.05$ and $R=0.37$, $p<0.05$, respectively) BP during the intervention. Baseline weight correlated with change in neither systolic nor diastolic BP, but change in weight was a strong correlate of change in both systolic and diastolic BP ($R=0.49$, $p<0.001$ and $R=0.49$, $p<0.001$, respectively).

Restricting alcohol consumption and caloric intake resulted independently and additively in decreases in total cholesterol concentration (Figure 4). The main effect with alcohol restriction was $-0.49$ ($-0.85$ to $-0.12$) mmol/l ($F_{1,38}=5.04$, $p<0.05$) and that with caloric reduction was $-0.40$ ($-0.76$ to $-0.03$) mmol/l ($F_{1,38}=3.34$, $p=0.07$). Similar independent and additive reductions in the triglyceride level occurred (Figure 4), the main alcohol effect being $-0.66$ ($-1.05$ to $-0.27$) mmol/l ($F_{1,38}=7.92$, $p<0.01$) and the main caloric effect $-0.78$ ($-1.17$ to $-0.39$) mmol/l ($F_{1,38}=11.32$, $p<0.001$).

After correcting for the effects of weight loss induced by alcohol restriction, an independent effect of alcohol on total cholesterol concentration was no longer demonstrable ($F_{1,38}=2.25$), and the effect on the triglyceride concentration was of borderline significance ($F_{1,38}=3.65$, $p=0.06$).

Reducing alcohol consumption and caloric intake affected HDL cholesterol levels (Figure 4) in opposite directions, resulting in changes of $-0.15$ ($-0.22$ to $-0.08$) mmol/l ($F_{1,38}=12.13$, $p<0.001$) and $0.11$ ($0.04$–$0.18$) mmol/l ($F_{1,38}=6.25$, $p<0.05$), respectively. The fall in the HDL cholesterol level with alcohol restriction was fully established at 9 weeks, whereas the increase in the HDL cholesterol level with caloric restriction continued to rise with further weight loss. In those subjects assigned to both alcohol restriction and caloric reduction there was no change in the HDL cholesterol concentration (Figure 4). The total cholesterol to HDL cholesterol ratio did not change with alcohol restriction but fell substantially after caloric reduction (Figure 5).

The $\gamma$-GT concentration correlated with self-reported alcohol intake, but not weight, at baseline ($R=0.37$, $p<0.001$), and decreases in the $\gamma$-GT level during the study were due to independent and additive influences of both alcohol restriction and caloric reduction (Figure 5). The change attributable to alcohol restriction was $-15.1$ ($-22.9$ to $-7.2$) units/l ($F_{1,38}=10.2$, $p<0.01$) and that attributable to caloric reduction $-9.0$ ($-16.9$ to $-1.2$) units/l ($F_{1,38}=3.7$, $p=0.058$). The erythrocyte mean corpuscular volume was unaffected by either alcohol restriction or caloric reduction.
Several other lifestyle factors that could potentially have influenced the outcome were carefully monitored during the study. Smokers reported no change in the number of cigarettes smoked. The 24-hour urinary excretion of sodium was unchanged in subjects in the low alcohol groups but reduced in those continuing their usual alcohol intake, with an alcohol main effect of 40.5 (9.7–71.3) mmol/day \( (F_{1,48}=4.65, p<0.05) \). A main effect of hypocaloric diet of 14.3 (–16.5 to 45.1) mmol/day was not significant \( (F_{1,48}=0.58) \) (Figure 6).

In the regular coffee drinkers there was a trend to drink more coffee among those who decreased their alcohol intake, with an alcohol main effect of 3.8 (0.6–7.1) cups per week \( (F_{1,48}=3.72, p=0.059) \). A similar trend with the low calorie diet was not significant, with a main effect of 2.4 (–0.9 to 5.7) cups per week \( (F_{1,48}=1.53, p=0.22) \).

Data on physical activity available from 68 of the 73 subjects completing the study indicated that this was decreased in those drinking less alcohol relative to subjects maintaining their usual drinking habits. The alcohol main effect was \(-2.3\) (–3.6 to –1.0) days on which vigorous exercise was performed each week \( (F_{1,48}=8.59, p=0.005) \). There was no significant influence of hypocaloric diet on level of physical activity.

**Discussion**

There have been few randomized controlled intervention studies to determine whether two or more nonpharmacological strategies result in a greater fall in BP than any single strategy pursued alone. The outcome of such studies has immediate clinical relevance given that compliance with nonpharmacological regimens may be affected if too many constraints are placed on a patient. In men with a moderate-to-heavy alcohol intake, we have previously confirmed a BP-lowering effect of alcohol restriction alone but were unable to demonstrate any additional BP reduction by combining this measure with 4 weeks of salt restriction\(^{10}\) or 4 weeks of aerobic exercise.\(^{11}\) In the present study, however, an 18-week low calorie diet in conjunction with a substantial reduction in alcohol intake had additive effects to lower BP in overweight men. Moreover, the combined approach resulted in additive decreases in serum total cholesterol and triglyceride and prevented the fall in serum HDL cholesterol that was seen with alcohol restriction alone.

Of the subjects identified as mildly hypertensive at baseline, normal diastolic BP developed in all who decreased both their caloric intake and alcohol consumption compared with 75% of those reducing calories alone, 80% of those restricting alcohol intake alone, and 57% of those making no changes. The remainder of the subjects had a baseline BP that was in the upper end of the normal range. Because BP is positively related to risk of stroke and coronary artery disease not only among hypertensive individuals but also in those usually considered normotensive,\(^{16}\) BP consistently in the "high" normotensive range has a major impact on cardiovascular outcome. Intervention to lower BP with drug therapy in these subjects is not cost-effective, but the substantial decreases in BP achieved with both weight reduction and alcohol restriction suggest that these should be promoted at a public health level to

**FIGURE 5.** Bar charts show mean±SEM change in serum total cholesterol to high density lipoprotein cholesterol ratio (Cholesterol/HDL-C) (top) and plasma γ-glutamyl transpeptidase (γGT) at weeks 9 and 18 of intervention in each of four study groups. *p<0.05, **p<0.001 different from 0 by t test (Bonferroni's method).

**FIGURE 6.** Line graph shows mean±SEM change in 24-hour urinary sodium (Na) excretion in four study groups.

\[\text{normal alcohol intake/normal caloric intake (n=18)}; \]
\[\text{normal alcohol intake/low caloric intake (n=17)}; \]
\[\text{low alcohol intake/normal caloric intake (n=17)}; \]
\[\text{low alcohol intake/low caloric intake (n=18)}.\]
study has demonstrated that the major nutrient changes the calorie restriction regimen followed in the present ratio. Our previous experience (unpublished data) with potent effects of potassium, a vegetarian diet, and constituents, particularly total fat intake. The major hypertensive effects of sodium chloride and the hy-

whether practical weight-reducing dietary advice given to overweight subjects is additive to the effects of alcohol restriction in lowering BP, and if we had restricted calories alone but not altered the dietary mix the generalizability of any results obtained would have to be seriously questioned. This is because nearly all attempts to lose weight in a clinical setting entail not only a reduction in calories but also a change in dietary constituents, particularly total fat intake. The major dietary influences on BP identified to date include the hypertensive effects of sodium chloride and the hypotensive effects of potassium, a vegetarian diet, and possibly decreased dietary fat when this entails an increase in the polyunsaturated to saturated fat (P/S) ratio. Our previous experience (unpublished data) with the calorie restriction regimen followed in the present study has demonstrated that the major nutrient changes seen are a decrease in the total fat intake, with similar reductions in the intakes of saturated, monounsaturated, and polyunsaturated fats (i.e., no change in the P/S ratio), a decrease in total carbohydrate intake, and a decrease in the intakes of starch, sugars, phosphate, iron, and calcium. No significant changes are seen in intakes of the major vitamins, total protein, fiber, or potassium, but carotene intake increases significantly. Salt intake from food sources usually drops, and this was anticipated as the major potential confounding factor in the current study. However, despite an initial trend toward lower 24-hour urinary sodium excretion in subjects in the calorie-restricted groups during the first 4 weeks, this pattern was not maintained.

In contrast, and for unexplained reasons, a significant decrease in salt intake (as reflected in a decreased 24-hour urinary excretion of sodium) was seen in subjects who were asked to continue their usual alcohol intake. Our relatively crude questionnaire instruments also detected trends for change in other lifestyle variables during the intervention. Subjects assigned to reduce their alcohol intake also reduced their level of physical activity and increased their coffee intake relative to those who continued their usual drinking habits. There were no differences between calorie-restricted subjects and those continuing their usual diet in any of these lifestyle parameters, and their combined influence, if any, would have served to blunt any overall impact of alcohol restriction on BP. This does not appear to have been the case, however, and the fall in BP with alcohol restriction was similar in magnitude to that seen in our previous intervention studies in normotensive and treated hypertensive subjects. These observations highlight the necessity of closely monitoring all aspects of lifestyle when any attempt is made to modify one or more lifestyle parameters over a prolonged period.

There were also additive decreases in serum total cholesterol and triglyceride with the combination of caloric reduction and alcohol restriction. Such an outcome has important prognostic significance. In the Goteborg Primary Prevention Trial, in which subjects with mild hypertension were treated with either a diuretic or a β-blocker, analysis of cardiovascular morbidity demonstrated that reductions in both BP and the cholesterol concentration were necessary to achieve a substantial reduction in morbidity. Similar results have been obtained from the Australian National Blood Pressure Study and the Multiple Risk Factor Intervention Trial, in which favorable cardiovascular outcomes were more likely in subjects with lower initial cholesterol levels. Extrapolating from our study, a clearly favorable influence on serum lipid-lipoproteins can be anticipated in overweight drinkers who reduce their excessive calorie and alcohol intake.

The major effects of caloric reduction and alcohol restriction were on serum triglyceride, with a predicted fall of 1.4 mmol/l over 18 weeks. Given the ongoing controversy over whether hypertriglycerideremia constitutes an independent risk factor for coronary artery disease, the question of whether the decreases in triglyceride levels achieved in this study, by either weight reduction or alcohol restriction, reduce risk remains to be answered.
There is little controversy, however, concerning low HDL cholesterol levels and high total cholesterol to HDL cholesterol ratios as important predictors of coronary risk. The well-established effect to raise the HDL cholesterol concentration has been used to explain alcohol’s putative coronary protection in light-to-moderate drinkers. This gives rise to the theoretical concern that there may be little to be gained from the fall in BP if there is a simultaneous fall in the HDL cholesterol level when drinkers reduce their alcohol intake. Previous attempts to address this problem by the prescription of regular exercise during reduction of alcohol consumption were unsuccessful in preventing an alcohol-related fall in HDL cholesterol levels. In contrast, in the present study calorie restriction and the associated weight reduction completely prevented the fall in HDL cholesterol levels seen with alcohol restriction alone. An inverse association between HDL cholesterol levels and body weight has been described, and the expected increase in the HDL cholesterol concentration with weight reduction has been reported previously but has not been a consistent result. Our findings suggest that any inconsistencies in previous reports could well have been due to unaccounted changes in alcohol intake with calorie restriction.

A recent meta-analysis of nine major prospective observational studies of the impact of BP on stroke and coronary artery disease estimated that a reduction in diastolic BP of 5–10 mm Hg could result in a 34–56% decrease in stroke and a 21–37% decrease in coronary artery disease. This has been achieved with stroke, whereas the reduction in coronary artery disease has been just over half that expected. Although no prospective trial of the effects of BP reduction by nondonor measures on cardiovascular disease outcome has yet been reported, an approach that reduces weight and alcohol-related fall in HDL cholesterol levels. Plain alcohol’s putative coronary protection in light-to-moderate drinkers. This gives rise to the theoretical concern that there may be little to be gained from the fall in BP if there is a simultaneous fall in the HDL cholesterol level when drinkers reduce their alcohol intake. Previous attempts to address this problem by the prescription of regular exercise during reduction of alcohol consumption were unsuccessful in preventing an alcohol-related fall in HDL cholesterol levels. In contrast, in the present study calorie restriction and the associated weight reduction completely prevented the fall in HDL cholesterol levels seen with alcohol restriction alone. An inverse association between HDL cholesterol levels and body weight has been described, and the expected increase in the HDL cholesterol concentration with weight reduction has been reported previously but has not been a consistent result. Our findings suggest that any inconsistencies in previous reports could well have been due to unaccounted changes in alcohol intake with calorie restriction.

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The high prevalence of obesity and excess alcohol consumption, and their ready modification with simple intervention strategies, suggests that greater emphasis should be given to both weight reduction and moderation of alcohol intake in attempts to reduce cardiovascular risk in Western communities.

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