Effect of Alcohol Abstinence on Blood Pressure
Assessment by 24-Hour Ambulatory Blood Pressure Monitoring

María Teresa Aguilera, Alejandro de la Sierra, Antonio Coca, Ramón Estruch, Joaquín Fernández-Sola, Alvaro Urbano-Márquez

Abstract—Several studies have shown that cessation of alcohol drinking reduces blood pressure (BP). However, attempts to reproduce these findings by ambulatory BP monitoring (ABPM) have shown inconsistent results. The aim of the present study was to assess the effect of 1 month of proven abstinence from alcohol on the 24-hour BP profile in heavy alcohol drinkers. Forty-two men who were heavy drinkers (>100 g of pure ethanol per day) were consecutively admitted to a general ward for voluntary alcohol detoxification. On the day of admission, they received a total dose of 2 g/kg of ethanol diluted in orange juice in 5 divided doses, and a 24-hour ABPM was performed. A new 24-hour BP monitoring in the same environmental conditions was performed after 1 month of proven alcohol abstinence while the subjects were receiving the same amount of fluid but without the addition of alcohol. After 1 month of proven alcohol abstinence, BP and heart rate (HR) significantly decreased. The reduction was 7.2 mm Hg for 24-hour systolic BP (SBP) (95% CI, 4.5 to 9.9), 6.6 mm Hg for 24-hour diastolic BP (DBP) (95% CI, 4.2 to 9.0), and 7.9 bpm for HR (95% CI, 5.1 to 10.7). The proportion of alcoholic patients considered hypertensive on the basis of 24-hour BP criteria (daytime SBP $\geq$ 135 mm Hg or daytime DBP $\geq$ 85 mm Hg) fell from 42% during alcohol drinking to 12% after 1 month of complete abstinence. Abstinence did not modify either the long-term BP variability, assessed by SD of 24-hour BP, or its circadian profile. We conclude that abstinence in heavy alcohol drinkers significantly reduces BP assessed by 24-hour ABPM and that this reduction is clinically relevant. These results show that heavy alcohol consumption has an important effect on BP, and thus cessation of alcohol consumption must be recommended as a priority for hypertensive alcohol drinkers. (Hypertension. 1999;33:653-657.)

Key Words: hypertension, alcohol-induced, alcohol, blood pressure determination, blood pressure monitoring, ambulatory

Several cross-sectional epidemiological studies have shown a clear relationship between alcohol consumption, high blood pressure (BP), and the prevalence of hypertension. As early as 1915, Lian reported an association between alcohol drinking and high BP in French soldiers. Since then, large epidemiological studies performed in North America, Europe, Australia, and Japan have concluded that a clear relationship between alcohol drinking and both high BP and the prevalence of hypertension is likely to exist. This relationship has been shown in both sexes, at various ages, in several racial groups, and for drinkers of liquor, wine, or beer. Further, as the Intersalt Study has shown, the magnitude of such a relationship is similar to that observed for obesity and greater than that observed for salt intake.

A clear physiological mechanism for the alcohol-BP link has not yet been established. Genetic predisposition, increased cardiac output, abnormalities in the renin-angiotensin system or the sympathetic nervous system, and a direct vascular tone effect probably mediated through sodium and calcium transport alterations have all been suggested as possible mechanisms of alcohol-induced hypertension.

Intervention studies have clearly shown that cessation of alcohol drinking reduces office BP in both hypertensive and normotensive alcoholic patients. However, attempts to reproduce these findings by ambulatory BP monitoring (ABPM) have shown inconsistent results. Cessation of alcohol intake has been shown not to affect 24-hour BP in alcohol drinkers or even to increase BP at night, leading to speculation that alcohol ingestion does not produce sustained rises in BP but, rather, a transient increase in BP due to an alerting reaction when BP is measured in the clinical setting. These studies, however, have been conducted in moderate alcohol drinkers and after a period of only 4 to 7 days of abstinence.

The aim of the present study was to assess the effect of 1 month of proven alcohol abstinence on 24-hour BP profile in heavy alcohol drinkers (>100 g of alcohol per day).
Methods

Patient Selection
Because of different sensitivity to the effect of alcohol intake in both genders and because most patients attended in the alcohol unit were men, we decided to include only men in our study. Thus, 45 men aged 40±8 years (between 24 and 53 years) and with a mean daily alcohol intake of 216±72 g (range, 100 to 380 g) were consecutively recruited from patients admitted to the Alcohol Unit of the Hospital Clinic (Barcelona, Spain) for voluntary alcohol detoxification. None of the patients received any medication during the 4 weeks before their inclusion or during the month of the study.

Study Protocol
The study was approved by the Ethics Committee of the Hospital and the Spanish Health Authority (Protocol F.I.S. 93/0195). Written informed consent was obtained from all participants. Alcoholic patients were admitted into a general hospital ward, and alcohol intake was maintained at a dose of 0.4 g of pure ethanol (vodka) in 200 mL of orange juice per kilogram of body weight every 4 hours (except during sleeping time) (total dose, 2 g/kg). Plasma ethanol concentration was measured before each dose and averaged 0.26±0.21 g/L. None of the patients presented signs or symptoms of acute alcohol intoxication. Twenty-four-hour ABPM was performed between 8 AM and 9 AM. After this procedure was completed, alcohol intake was stopped and psychological support was initiated. Withdrawal symptoms were evaluated according to the Clinical Institute for Withdrawal Assessment (CIWA) scale, and none of the patients scored higher than 15, which is considered withdrawal syndrome. After 3 days of hospitalization, patients were discharged and continued follow-up and psychological support. Compliance with abstinence from alcohol drinking was assessed by direct interview of the patients and their relatives, repeated urinary alcohol determinations, and serum γ-glutamyltranspeptidase (GGTP) and erythrocyte mean corpuscular volume measurements. After 1 month of proven alcohol abstinence, 42 patients (3 patients relapsed and were excluded from the study) were readmitted to the hospital; a new 24-hour ABPM was performed in the same environmental conditions as those of the first measurement, with the patients drinking 200 mL of orange juice without alcohol addition every 4 hours.

BP Measurements
Twenty-four-hour ABPM was performed twice (during alcohol and placebo intakes) by use of an automated noninvasive oscillometric device (SpaceLabs 90207, SpaceLabs Inc). The appropriate cuff was placed on the nondominant arm, and BP was registered automatically at 20-minute intervals for a 24-hour period. The following parameters were obtained from each record in the 24-hour period as well as in daytime (8 AM to 10 PM) and nighttime (12 AM to 6 AM) periods: mean values and SD of systolic BP (SBP), mean BP (MBP), diastolic BP (DBP), and heart rate (HR). Finally, day/night ratio was obtained by dividing daytime by nighttime BP.

Statistical Analysis
Values are expressed by their mean±SD or by the 95% CI. Comparison of BP parameters during alcohol intake and after 1 month of abstinence was performed by paired Student’s t test.

Results
Table 1 shows mean values of SBP, MBP, DBP, and HR obtained during the entire 24-hour period, as well as during daytime and nighttime periods, in heavy alcohol drinkers during alcohol intake and after 1 month of abstinence. There was a significant decrease of BP and HR after 1 month of abstinence. The average decrease in these parameters during the 24-hour period was 7.2 mm Hg (5.4%) for SBP (95% CI, 4.5 to 9.9 mm Hg), 6.8 mm Hg (6.7%) for MBP (4.3 to 9.3 mm Hg), 6.6 mm Hg (7.6%) for DBP (4.2 to 9.0 mm Hg), and 7.9 bpm (9.1%) for HR (5.1 to 10.7 bpm).

Cessation of alcohol intake reduced BP without affecting its circadian pattern. As shown in Table 1, the reduction of daytime and nighttime BP was of the same magnitude. Further, the BP day/night ratio was not significantly modified after cessation of alcohol intake (Table 2). Figure 1 shows the circadian profile of BP obtained with average hourly BPs during both alcohol consumption and abstinence and shows that both profiles obtained are parallel and that the nocturnal fall of BP is preserved.

The effect of alcohol abstinence on long-term BP variability was examined by the SD of BP obtained during the 24-hour period as well as during daytime and nighttime periods. Although this measure of variability based on discontinuous measurements has poor accuracy, we did not find differences in BP variability during alcohol consumption and abstinence, as can be seen in Table 3. However, abstinence significantly reduced HR SD, particularly during the 24-hour period. The extent of this reduction was 1.9 bpm (95% CI, 0.9 to 2.9 bpm).

The decrease in BP after cessation of alcohol consumption was not significantly correlated with age, daily alcohol intake, or years of alcohol consumption. However, we observed a direct correlation between this decrease in BP and baseline BP obtained during alcohol consumption (Figure 2).

Table 1. Mean Values of ABPM Data in 42 Heavy Alcoholic Drinkers During Chronic Ingestion and After One Month of Abstinence

<table>
<thead>
<tr>
<th>Time</th>
<th>Alcohol Consumption</th>
<th>Abstinence</th>
</tr>
</thead>
<tbody>
<tr>
<td>24 Hours</td>
<td>SBP, mm Hg</td>
<td>125.3±14.5</td>
</tr>
<tr>
<td></td>
<td>MBP, mm Hg</td>
<td>95.0±10.7</td>
</tr>
<tr>
<td></td>
<td>DBP, mm Hg</td>
<td>80.3±9.3</td>
</tr>
<tr>
<td></td>
<td>HR, bpm</td>
<td>83.7±10.6</td>
</tr>
<tr>
<td>Daytime</td>
<td>SBP, mm Hg</td>
<td>128.2±15.3</td>
</tr>
<tr>
<td></td>
<td>MBP, mm Hg</td>
<td>97.5±11.3</td>
</tr>
<tr>
<td></td>
<td>DBP, mm Hg</td>
<td>82.7±9.7</td>
</tr>
<tr>
<td></td>
<td>HR, bpm</td>
<td>88.3±11.4</td>
</tr>
<tr>
<td>Nighttime</td>
<td>SBP, mm Hg</td>
<td>119.2±15.1</td>
</tr>
<tr>
<td></td>
<td>MBP, mm Hg</td>
<td>89.2±11.2</td>
</tr>
<tr>
<td></td>
<td>DBP, mm Hg</td>
<td>74.7±10.0</td>
</tr>
<tr>
<td></td>
<td>HR, bpm</td>
<td>73.1±9.6</td>
</tr>
</tbody>
</table>

*P<0.0001; †P=0.0002.

Table 2. Effect of Alcohol Consumption and Abstinence on BP Day/Night Ratio

<table>
<thead>
<tr>
<th>BP day/night ratio</th>
<th>Alcohol Consumption</th>
<th>Abstinence</th>
</tr>
</thead>
<tbody>
<tr>
<td>SBP</td>
<td>1.08±0.07</td>
<td>1.09±0.06</td>
</tr>
<tr>
<td>MBP</td>
<td>1.10±0.08</td>
<td>1.11±0.07</td>
</tr>
<tr>
<td>DBP</td>
<td>1.12±0.09</td>
<td>1.14±0.08</td>
</tr>
</tbody>
</table>
In fact, 18 (43%) alcohol drinkers exhibited ABPM values of high BP (ie, daytime SBP $\geq 135$ mm Hg and/or DBP $\geq 85$ mm Hg) during alcohol intake. Of this group, 13 (72%) had normalized their BP (daytime BP $< 135/85$ mm Hg) after 1 month of abstinence.

**Discussion**

The present study shows that cessation of alcohol drinking in heavily alcoholic subjects significantly reduces BP assessed by 24-hour ABPM. After 1 month of proven abstinence, the magnitude of this reduction on average was 7.2/6.6 mm Hg for SBP/DBP, respectively. In fact, 13 out of 18 heavy alcohol drinkers (72%) considered hypertensive on the basis of baseline ABPM recordings while drinking (daytime SBP $\geq 135$ mm Hg and/or daytime DBP $\geq 85$ mm Hg) became normotensive after cessation of alcohol drinking.

Several epidemiological, cross-sectional, and prospective studies have shown a clear relationship between heavy alcohol consumption and BP. In the first Kaiser Permanente study, 3 white men drinking 6 or more drinks per day had a BP 10.9/4.5 mm Hg higher than nondrinkers. These findings were confirmed in a second study from the same group as well as in other studies in different populations in North America, Europe, Australia, and Japan. Moreover, prospective studies have shown that alcohol drinking is associated with an increase in BP over time and with an increased risk of developing hypertension.

Intervention studies performed in moderate or heavy alcohol drinkers have uniformly shown a significant decrease of BP with alcohol reduction or abstinence. In the first study by Saunders et al., performed in 132 alcoholic patients, the prevalence of hypertension fell from 51.5% during alcohol drinking to 9% after detoxification. Potter and Beevers, in a crossover study performed in hypertensive drinkers, found a significant reduction of BP after 4 days of abstinence and a significant rise in BP after 4 days of alcohol reintroduction. These results were confirmed in treated or untreated hypertensive subjects, and in normotensive subjects, and in more moderate alcohol drinkers.

In contrast with these uniform results, more recent studies examining the effect of alcohol consumption on BP by use of ABPM have yielded negative results. Howes et al. did not find any significant change in 24-hour BP during 4 days of alcohol consumption or abstinence in healthy volunteers. Abe et al. observed that after 7 days of regular alcohol consumption at dinner, BP fell significantly during the first 6 hours after drinking and rose during the next 8 hours without affecting the average 24-hour BP. O’Callaghan et al. did not find any significant effect of alcohol in office or 24-hour BP in normotensive light drinkers. Finally, Maiorano et al. reported a slight but not significant decrease in SBP after 1 week of abstinence in heavy alcohol drinkers.

These negative results, in contrast to the previous findings showing significant decreases of BP with alcohol abstinence, have led to the hypothesis that, in fact, alcohol does not have a sustained effect on BP but rather induces a transient increase in BP under the stress of clinical measurement. Our results showing that abstinence significantly decreases BP by means of ABPM do not support this last hypothesis and are in agreement with the former studies.
There are several differences between the present study and previous studies using ABPM. First, the number of patients included in the present study is considerably greater than in previous ones. Second, the Australian studies and the Japanese studies included nondrinkers or moderate drinkers. Third, the Italian study, the only one including heavy alcohol drinkers, showed a decrease, although not statistically significant, of 7 mm Hg in 24-hour SBP. Fourth, in previous studies, ABPM was performed after a short period of abstinence (between 4 and 7 days), whereas the abstinence period in the present study lasted 1 month. It may be that some alcohol withdrawal symptoms may have influenced the ABPM recordings during the acute abstinence phase in the previous studies.

The results of the present study may be criticized in 2 respects. First, it is possible that the BP measured during alcohol intake may be influenced by some degree of alcohol withdrawal due to the difference between the amount of alcohol that patients were used to drinking and the amount that they drank during ABPM recording. This amount was adjusted to the weight of the patients and not to their usual alcohol intake before inclusion in the study. However, the total dose (2 g/kg in 24 hours) seems enough to assume that alcohol withdrawal symptoms, if present, were minimal. In fact, according to the CIWA scale, none of the patients fulfilled criteria of withdrawal syndrome. Moreover, there was no correlation ($R = 0.099$ and $R = 0.171$ for SBP and DBP, respectively) between the BP decrease after abstinence and the usual alcohol intake of the patients.

Second, it is possible to speculate that the second ABPM measurement (after alcohol detoxification) may have yielded low BP values because the patients had been familiarized with the ABPM procedure. This placebo effect, which is relevant in repeated office BP measurements, is almost irrelevant in ABPM measurements performed within a 4-week period when comparing average 24-hour BP values. Although a slight placebo effect has been described in ABPM measurements during the first hours of the procedure, it only affects the systolic component of BP and its magnitude is $< 3$ mm Hg. As shown in Figure 1, this is obviously not the case in the present study in which 24-hour curves during alcohol intake and after cessation of intake were completely parallel. It is not possible to exclude completely a regression to the mean phenomenon as partially responsible for the decrease in BP after cessation of alcohol intake. However, the inclusion of a control group (ie, alcohol drinkers maintained on alcohol intake during 1 month) would have been ethically questionable.

We were unable to show that alcohol consumption produces a change in circadian BP profile as has been suggested by Abe et al. However, in contrast with that study in which alcohol was administered in a single dose (1 g/kg) at dinner, we used 5 doses of 0.4 g/kg every 4 hours, except during sleep.

Finally, it is important to note that the magnitude of BP decrease after detoxification was directly correlated with the initial BP, as shown in Figure 2. Patients considered hypertensive on the basis of ABPM criteria (average daytime SBP $\geq 135$ or daytime DBP $\geq 85$ mm Hg) showed a decrease in BP of $\approx 12.2/10.6$ mm Hg for SBP/DBP, respectively, whereas in normotensive subjects, the decrease for SBP/DBP was $3.4/3.5$ mm Hg, respectively. Further, the prevalence of hypertension in heavy drinkers was 43%, a figure similar to that obtained by Saunders et al. After alcohol detoxification, BP was normalized in 14 (72%) of these patients, and the prevalence of sustained hypertension fell to 12%, a figure similar to that observed in the general population.

In conclusion, cessation of alcohol consumption in heavy alcohol drinkers significantly decreases BP, assessed by 24-hour ABPM. More than half the patients considered hypertensive may become normotensive after alcohol detoxification. These results show that heavy alcohol consumption has an important effect on BP, and they strengthen the recommendation of alcohol-intake reduction as a priority for those patients with essential hypertension who are usual alcohol drinkers.

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References

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