Acute Depressor Effect of Alcohol in Patients With Essential Hypertension

Yuhei Kawano, Hitoshi Abe, Shunichi Kojima, Terunao Ashida, Kaoru Yoshida, Masahito Imanishi, Hiroki Yoshimi, Genjiro Kimura, Morio Kuramochi, and Teruo Omae

To investigate the time course of the effects of alcohol on blood pressure, we studied the response of ambulatory blood pressure, neurohumoral variables, and hemodynamics to a single moderate dose of alcohol in hypertensive patients. Sixteen Japanese men (22-70 years old) with essential hypertension who were habitual drinkers were examined under standardized conditions. On the alcohol intake day, they ingested 1 ml/kg ethanol (vodka) at dinner, and on the control day they consumed a nonalcoholic beverage. The order of the two periods was randomized. Mean ambulatory blood pressure was lower in the alcohol intake period than in the control period (125±3/74±2 versus 132±4/78±2 mm Hg, p<0.05), and the significant depressor effect of alcohol lasted for up to 8 hours after drinking. Blood pressure on the next day did not differ with or without alcohol intake. The acute hypotensive effect of alcohol was associated with an increase in heart rate and cardiac output and with a decrease in systemic vascular resistance as determined by echocardiography. Plasma catecholamine levels and renin activity rose significantly at 2 hours after dinner, whereas vasopressin and potassium levels fell on the alcohol day. Blood glucose and serum insulin levels were comparable between the two periods. Three patients with marked alcohol-induced flush had greater hypotensive and tachycardic responses than those who did not show an alcohol-induced flush. The change in mean blood pressure induced by alcohol was negatively correlated with age, the baseline blood pressure, and the change in plasma norepinephrine. These results indicate that the major effect of acute alcohol intake is to lower blood pressure through systemic vasodilatation in hypertensive subjects. Ambulatory blood pressure monitoring may be useful for assessing blood pressure in habitual drinkers. (Hypertension 1992;20:219-226)

Key Words • alcohol drinking • blood pressure monitoring, ambulatory • hemodynamics • catecholamines • electrolytes • hypertension, essential

The relation between alcohol and hypertension is well known. A positive association between the level of habitual alcohol intake and blood pressure has been shown in many reports,1-5 and this relation has been observed in whites, blacks, and Asians. It has also been reported that abstinence from alcohol results in a decrease of blood pressure in drinkers.6-9 However, these observations depended on casual blood pressure measurements taken in the daytime, while most habitual drinkers consume alcohol in the evening and at night. Alcohol has an acute vasodilatory action,10,11 and it has been our experience that many hypertensive patients note a decrease in their blood pressure after alcohol ingestion at home. It has been recommended that the relation between the time of the last alcohol intake and that of blood pressure measurement should be considered,7 but this has generally been ignored. Inconsistent results have been reported about the acute effect of alcohol on blood pressure,12-22 but most of these studies were carried out under conditions unrelated to normal drinking patterns and had a relatively short observation period.

Ambulatory blood pressure monitoring is now widely accepted as a useful method for the evaluation of hypertensive subjects because it provides multiple blood pressure recordings during the whole day and eliminates the observer’s bias and the “white coat” effect on blood pressure.23,24 In the present study, we examined the effect of a single moderate dose of alcohol on the ambulatory blood pressure in hypertensive patients under standardized conditions that were designed to mimic the usual drinking pattern. We also studied the effects of alcohol on neurohumoral variables and hemodynamics in the same patients.

Methods

Subjects

Sixteen Japanese men with hypertension who were habitual drinkers participated in this study. Informed consent was given by each patient, and the study protocol was approved by the Ethics Committee of the National Cardiovascular Center. All the patients were diagnosed as having mild-to-moderate essential hypertension (average diastolic blood pressure, 90-114 mm Hg) after routine examinations at the Hypertension...
Measurements

Ambulatory blood pressure and heart rate were measured every 30 minutes until noon the next day by an oscillometric method using ABPM 630 recorders (Nippon Colin, Komaki, Aichi, Japan). This device uses a silent cuff inflation system operated by compressed CO$_2$ gas. The accuracy of each recorder was checked by simultaneous measurement with a mercury sphygmomanometer, and all recorders showed a difference of less than 10 mm Hg. The same recorder was used for the alcohol intake and control days in each individual patient for at least 7 days before the study. Six patients had never been treated for hypertension and the remaining 10 patients were asked to discontinue their antihypertensive medication for at least 7 days before the study. Six patients were examined, and special attention was taken to avoid misleading angulation of the left ventricular long axis in relation to the ultrasound beam. All echocardiograms were taken by an experienced examiner and were read independently by two investigators. Left ventricular dimensions and wall thickness were measured using the leading edge technique, according to the recommendations of the American Society of Echocardiography. Left ventricular volumes were estimated by the cube-function formula from the end-diastolic and end-systolic left ventricular internal dimensions and were used to estimate the stroke volume and cardiac output.

Data Analysis

All results are expressed as the mean±SEM. Data were analyzed using repeated-measures analysis of variance with Fisher's test. Student's $t$ test was used for the comparison of two groups. Linear regression analysis and stepwise regression analysis were used for the evaluation of relations between the measured variables. A value of $p<0.05$ was considered statistically significant.

Results

Figure 1 shows the time course of ambulatory blood pressure at 30-minute intervals, and Figure 2 shows 2-hourly means of ambulatory blood pressure and heart rate from 4 PM to noon of the next day in 13 of the 16 hypertensive patients. Data for three patients were missing.

Subject for analysis of the ambulatory blood pressure, every 2-hour segment of the record was averaged. M-mode echocardiograms were recorded at an equivalent paper speed of 50 mm/sec with two-dimensional monitoring using a phased-array ultrasonic sector scanner and a 2.5 or 3.5 MHz transducer (model 77020A, Hewlett-Packard, Waltham, Mass.). The patients were in a partial left lateral decubitus position when they were examined, and special attention was taken to avoid misleading angulation of the left ventricular long axis in relation to the ultrasound beam. All echocardiograms were taken by an experienced examiner and were read independently by two investigators. Left ventricular dimensions and wall thickness were measured using the leading edge technique, according to the recommendations of the American Society of Echocardiography. Left ventricular volumes were estimated by the cube-function formula from the end-diastolic and end-systolic left ventricular internal dimensions and were used to estimate the stroke volume and cardiac output.

Serum electrolyte and total protein levels as well as the blood glucose were determined using a biochemical analysis system (TBA-80S, Toshiba, Tokyo). Hematocrit was measured by a microhematocrit method. Plasma levels of renin activity (PRA), aldosterone, and vasopressin, as well as serum levels of cortisol and insulin were determined by radioimmunoassay. Plasma catecholamines were assayed by high-performance liquid chromatography and trihydroxyindole fluorometry.

Data Analysis

All results are expressed as the mean±SEM. Data were analyzed using repeated-measures analysis of variance with Fisher's test. Student's $t$ test was used for the comparison of two groups. Linear regression analysis and stepwise regression analysis were used for the evaluation of relations between the measured variables. A value of $p<0.05$ was considered statistically significant.

Results

Figure 1 shows the time course of ambulatory blood pressure at 30-minute intervals, and Figure 2 shows 2-hourly means of ambulatory blood pressure and heart rate from 4 PM to noon of the next day in 13 of the 16 hypertensive patients. Data for three patients were missing.
excluded because the ambulatory recordings were not available for the entire period. Systolic and diastolic blood pressure in the evening hours of the alcohol intake day were significantly lower than those on the control day. The hypotensive effect of alcohol was most prominent at 3-4 hours after its intake (alcohol day, 115.8±3.0/68.5±2.1 mm Hg; control day, 139.4±4.6/80.3±2.9 mm Hg, \( p<0.05 \)) and lasted for up to 8 and 4 hours for systolic and diastolic blood pressure, respectively. Heart rate reciprocally increased for 8 hours after alcohol ingestion. Blood pressure and heart rate were similar in the early and late morning of the next day after both the alcohol and control days.

The average 20-hour ambulatory blood pressure in the alcohol period was also lower than that in the control period (systolic blood pressure, 124.8±2.5 versus 131.9±3.5 mm Hg, \( p<0.01 \); diastolic blood pressure, 73.7±1.8 versus 77.6±2.4 mm Hg, \( p<0.05 \)). Mean ambulatory blood pressure values for the individual subjects are shown in Figure 3. Eleven of the 13 patients showed a reduction in mean blood pressure during the alcohol period (systolic blood pressure, 124.8±2.5 versus 131.9±3.5 mm Hg, \( p<0.01 \); diastolic blood pressure, 73.7±1.8 versus 77.6±2.4 mm Hg, \( p<0.05 \)). Mean ambulatory blood pressure values for the individual subjects are shown in Figure 3. Eleven of the 13 patients showed a reduction in mean blood pressure during the alcohol period (systolic blood pressure, 124.8±2.5 versus 131.9±3.5 mm Hg, \( p<0.01 \); diastolic blood pressure, 73.7±1.8 versus 77.6±2.4 mm Hg, \( p<0.05 \)).

Figure 4 shows neurohormonal variables for the 16 patients at 5 PM (before dinner), at 7 PM (after dinner), and at 8 AM the next day (before breakfast). There were no differences in plasma norepinephrine, epinephrine, PRA, aldosterone, vasopressin, and serum cortisol between the alcohol intake and control days at 5 PM. After alcohol ingestion (7 PM), the norepinephrine, epinephrine, and PRA levels were elevated significantly (norepinephrine, 412±58 versus 295±29 pg/ml; epinephrine, 51±14 versus 25±5 pg/ml; PRA, 3.1±0.7 versus 1.9±0.4 ng/ml/hr, alcohol intake versus control days), whereas the vasopressin level was reduced (0.6±0.1 versus 1.1±0.2 pg/ml). A significant decrease in plasma epinephrine level was noted after dinner on the control day. By the next morning, there were no differences in the measured variables. Serum cortisol levels showed their normal circadian changes in both periods.

Serum sodium levels were comparable between the alcohol intake and control days (Figure 5), but the serum potassium level decreased after alcohol intake (3.58±0.06 versus 3.98±0.06 at 7 PM, \( p<0.05 \)). Serum protein and hematocrit tended to be higher on the alcohol intake day at 7 PM, although the difference was not significant.

Blood glucose and serum insulin levels were determined at 5 PM, 6 PM, and 7 PM in 10 patients (Table 3). These levels before and after dinner were not different between the alcohol intake and control days. The blood ethanol level at 7 PM on the alcohol intake day ranged

<table>
<thead>
<tr>
<th>Variables</th>
<th>Control day</th>
<th>Alcohol intake day</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1 ((n=7))</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP (mm Hg)</td>
<td>134±4</td>
<td>127±3*</td>
</tr>
<tr>
<td>DBP (mm Hg)</td>
<td>80±3</td>
<td>76±3*</td>
</tr>
<tr>
<td>Group 2 ((n=6))</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP (mm Hg)</td>
<td>129±5</td>
<td>122±4*</td>
</tr>
<tr>
<td>DBP (mm Hg)</td>
<td>76±2</td>
<td>72±2*</td>
</tr>
</tbody>
</table>

SBP, systolic blood pressure; DBP, diastolic blood pressure.

*\( p<0.05 \) vs. the control day.
FIGURE 4. Bar graphs show plasma levels of various hormones at 5 PM (before dinner), 7 PM (after dinner), and 8 AM (next morning) on the alcohol intake and control days. NE, norepinephrine; E, epinephrine; PRA, plasma renin activity; Aldo, aldosterone; Cort, cortisol; VP, vasopressin. *p<0.05 vs. 5 PM.

from 0.6 to 0.9 mg/ml (0.7±0.1 mg/ml), which is considered to be within the range that produces mild ethanol intoxication.

Reliable echocardiographic records were obtained in eight patients. Resting hemodynamic variables before dinner were similar in the two periods (Table 4). The decrease in blood pressure and increase in heart rate after alcohol intake were associated with a significant increase in the cardiac index and a large decrease in the peripheral vascular resistance. An increase in left ventricular fractional shortening and a decrease in left ventricular end-systolic wall stress were also observed in the alcohol intake period.

The change in mean blood pressure induced by alcohol intake (mean blood pressure at 7 PM minus mean blood pressure at 5 PM) correlated positively with the mean blood pressure before drinking, the age, and the change in plasma norepinephrine (Figures 6 and 7). The relation between the change in mean blood pressure and the baseline blood pressure or the variation in norepinephrine was also significant by stepwise regression analysis. The change in mean blood pressure did not correlate significantly with alterations of the heart rate, plasma epinephrine, PRA, or vasopressin. There was also no relation between the change in mean blood pressure and the baseline levels of the various neurohormonal variables.

The effect of alcohol on postural changes in blood pressure was examined in 14 patients. As shown in Table 5, the hypotensive effect of alcohol in the standing position was comparable to that in the supine position, and the heart rate response was also similar in both positions.

Marked facial flushing developed in three subjects after alcohol intake; such alcohol-induced flushing was mild or absent in the remaining 13 subjects. The subjects with marked flushing showed greater alcohol-induced hypotension and tachycardia than those without flushing (Table 6). However, hypotension and tachycardia were also evident in the nonflushing group.

**Discussion**

The present study showed that a single moderate dose of alcohol lowered the blood pressure in hypertensive patients who were habitual drinkers. The alcohol was taken with dinner, and the hypotensive effect was evident in evening and night hours, but blood pressure the next day was not influenced by the single dose of alcohol. The average 20-hour ambulatory blood pressure value was also lower in the alcohol intake period than in the control period. In our study, the order of alcohol intake day and control day was randomized to minimize the influences of various factors including the order effect. Although food intake itself may induce postprandial hypotension, the decrease in blood pressure after dinner was much greater in the alcohol intake day than the control day despite similar changes in blood glucose and serum insulin.

Earlier studies have provided inconsistent results about the acute effects of alcohol on blood pressure, with some studies showing an increase and others showing a decrease or no change. Blood pressure was usually measured for short periods and only occasionally for longer periods in these earlier studies.

**Table 3. Blood Glucose and Serum Insulin Levels on the Alcohol Intake and Control Days in Ten Hypertensive Patients**

<table>
<thead>
<tr>
<th>Variables</th>
<th>5 PM (mg/dl)</th>
<th>6 PM (mg/dl)</th>
<th>7 PM (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose</td>
<td>Control</td>
<td>91.1±1.8</td>
<td>144.5±9.6</td>
</tr>
<tr>
<td></td>
<td>Alcohol</td>
<td>90.4±1.7</td>
<td>131.0±8.5</td>
</tr>
<tr>
<td>Insulin</td>
<td>Control</td>
<td>12.2±2.6</td>
<td>43.7±5.4</td>
</tr>
<tr>
<td></td>
<td>Alcohol</td>
<td>9.8±3.0</td>
<td>40.7±7.1</td>
</tr>
</tbody>
</table>

**Table 4. Blood Glucose and Serum Insulin Levels on the Alcohol Intake and Control Days in Ten Hypertensive Patients**

<table>
<thead>
<tr>
<th>Variables</th>
<th>5 PM (microunits/ml)</th>
<th>6 PM (microunits/ml)</th>
<th>7 PM (microunits/ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose</td>
<td>Control</td>
<td>91.1±1.8</td>
<td>144.5±9.6</td>
</tr>
<tr>
<td></td>
<td>Alcohol</td>
<td>90.4±1.7</td>
<td>131.0±8.5</td>
</tr>
<tr>
<td>Insulin</td>
<td>Control</td>
<td>12.2±2.6</td>
<td>43.7±5.4</td>
</tr>
<tr>
<td></td>
<td>Alcohol</td>
<td>9.8±3.0</td>
<td>40.7±7.1</td>
</tr>
</tbody>
</table>
TABLE 4. Hemodynamic Variables on the Alcohol Intake and Control Days in Eight Hypertensive Patients

<table>
<thead>
<tr>
<th>Variables</th>
<th>Control day</th>
<th>Alcohol intake day</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>5 PM</td>
<td>7 PM</td>
</tr>
<tr>
<td>MBP (mm Hg)</td>
<td>109±4</td>
<td>100±4</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>54±2</td>
<td>56±2</td>
</tr>
<tr>
<td>CI (/min/l)</td>
<td>2.6±0.2</td>
<td>2.9±0.3</td>
</tr>
<tr>
<td>PVR (dyne·sec·cm⁻¹)</td>
<td>2,061±208</td>
<td>1,791±225</td>
</tr>
<tr>
<td>LVFS (%)</td>
<td>34±2</td>
<td>37±2</td>
</tr>
<tr>
<td>LVESWS (10³ dyne/cm²)</td>
<td>67±10</td>
<td>56±7</td>
</tr>
</tbody>
</table>

MBP, mean blood pressure; HR, heart rate; bpm, beats per minute; CI, cardiac index; PVR, peripheral vascular resistance; LVFS, left ventricular fractional shortening; LVESWS, left ventricular end-systolic wall stress.

*p<0.05 vs. the control day; †p<0.05 vs. 5 PM on the alcohol intake day.

An increase in blood pressure has been observed within 30 minutes of the intake of alcohol in healthy young men, and a significant reduction in blood pressure occurred at 90 to 180 minutes after drinking in healthy volunteers. Howes and Reid reported a biphasic response of blood pressure to acute alcohol loading at a dose of 0.9 g/kg in healthy male subjects. Blood pressure increased initially at 15 to 30 minutes, then decreased at 4 to 8 hours after drinking. Stott et al also observed a tendency for a reduction in blood pressure up to 8 hours after the intake of 1.3 g/kg alcohol in healthy volunteers. In their study, transient elevations in blood pressure were seen in both the alcohol intake and control periods. However, there have been few studies on the acute effect of alcohol on blood pressure in hypertensive patients. Potter et al reported that moderate drinkers but not mild drinkers with hypertension showed an increase in blood pressure at 30 minutes after the intake of 0.75 g/kg of ethanol.

These data taken together suggest that the increase in blood pressure after a single dose of alcohol is transient and terminates within 60 minutes, whereas the decrease...
in blood pressure induced by alcohol appears to begin after 60 minutes and to last for several hours. In our current study, only a decrease in blood pressure was observed after alcohol intake. The acute pressor effect of alcohol might have been masked because we analyzed the blood pressure data as averages for each 2-hour period from measurements made at 30-minute intervals. Intake of alcohol together with a meal may be another reason for the lack of blood pressure elevation in the present study, since the transient pressor effect of ethanol may be due to gastric irritation or stress induced by rapid ingestion of this substance in the laboratory setting.  It has been shown that the blood alcohol concentration is maximal at 60–120 minutes after intake, and then falls gradually over the subsequent several hours. Therefore, our present study and earlier studies suggest that a single moderate dose of alcohol mainly acts to lower blood pressure in humans.

The chronic effect of alcohol on blood pressure appears to differ from its acute effect. Most epidemiological studies have demonstrated a positive relation between the habitual level of alcohol intake and blood pressure, with a high prevalence of hypertension being found in regular drinkers. This positive relation has been seen in white, black, and Asian populations. It has also been shown that abstinence or restriction of alcohol lowers blood pressure in hypertensive and normotensive subjects. However, blood pressure measurement was carried out in the daytime in these studies, despite the fact that alcohol is usually consumed in the evening hours. Thus, it is possible that the pressor effect of chronic alcohol consumption has been underestimated in earlier studies. Using ambulatory blood pressure monitoring, we have observed that repeated alcohol intake lowers nighttime blood pressure and elevates early morning blood pressure without changing the average 24-hour blood pressure in hypertensive patients. Therefore, the effect of alcohol on blood pressure may be more complex than was realized.

A rise in blood pressure has been observed as one manifestation of the withdrawal syndrome in alcoholic patients. During alcohol withdrawal, there is excess central nervous system excitability and adrenergic discharge, and plasma norepinephrine level peaks 12–24 hours after the cessation of alcohol intake. The withdrawal hypothesis has been considered as a mechanism of the alcohol-related hypertension. In the present study, blood pressure did not rise the day after alcohol intake, suggesting that a single moderate dose of alcohol may not be enough to cause a significant increase in blood pressure in relation to the withdrawal phenomenon.

The hypotensive effect of alcohol was pronounced in the patients who had a higher baseline blood pressure in this study. It is difficult to determine the exact influence of the basal blood pressure level, but it seems possible that vascular responsiveness may be altered in patients with advanced hypertension. In our preliminary study, the hypotensive effect of alcohol on the ambulatory blood pressure was also seen in normotensive subjects, although it was not significant (unpublished data). Many antihypertensive agents are effective in lowering blood pressure in hypertensive humans and animals but have little effect on normotensive humans and animals. The effect of alcohol on blood pressure may also be influenced by age, since there was a negative correlation between age and the alcohol-related change in blood pressure. However, this relation was not confirmed to be significant by stepwise regression analysis. The marked reduction in nocturnal blood pressure produced by alcohol ingestion in patients with advanced hypertension or elderly patients is potentially harmful, and further long-term studies of this subject are required.

The fall in blood pressure after alcohol intake was associated with an increase in heart rate and cardiac output and with a decrease in peripheral vascular resistance, indicating that the hypotensive effect of alcohol is due to peripheral vasodilatation. It has been shown that alcohol and its metabolites, acetaldehyde and acetate, have a vasodilatory effect on isolated blood vessels. This action may be related to interference with the movement or translocation, or both, of calcium across the vascular smooth muscle cell membrane. The increase in heart rate that we have noted was consistently observed in earlier studies. This tachycardia appears to be due to sympathetic activation and contributes to the increase in cardiac output. Although alcohol acts to depress myocardial function, this effect can be masked by autonomic responsiveness. Vasodilation in cardiac output may prevent a further reduction in blood pressure since the hypotensive effect of alcohol seems to be stronger in patients with heart failure or autonomic failure.

The significant depressor effect of alcohol observed in this study could be related to the alcohol flush syndrome that is common in Orientals. Alcohol flushing is caused by the accumulation of acetaldehyde due to a genetic deficiency of aldehyde dehydrogenase. It has been reported that Japanese subjects with an alcohol flush show marked cardiac stimulation and peripheral vasodilation. In our current study, the hypotensive and
tachycardic effects of alcohol were greater in the subjects with flushing than in those without it. Since the characteristics of subjects may explain the different blood pressure responses between this and earlier studies. Kupari et al. showed that the blood acetaldehyde level after alcohol intake is high in subjects with alcohol flushing, but it is too low to detect in those without flushing. However, the hypotensive effect of alcohol may not be produced by the accumulation of acetaldehyde alone, since the subjects without flushing also showed a significant fall in blood pressure after drinking. Although acetaldehyde has a vasodilatory action, the dilator effect of alcohol appears to be greater than acetaldehyde, at least in certain vascular beds.

In the present study, plasma norepinephrine and epinephrine levels increased while blood pressure decreased at 2 hours after alcohol intake. The increase in plasma norepinephrine was marked in patients who showed a large decrease in mean blood pressure. Thus, the sympathetic nervous system appears to be activated by the vasodilatory effects of alcohol and may prevent the worsening of hypotension. An alcohol-induced increase in plasma catecholamines has been shown in many studies. The sympathetic nervous system may also contribute to the early transient rise in blood pressure after alcohol intake, since Grassi et al. observed an increase in muscle sympathetic nerve activity and blood pressure after alcohol ingestion in normotensive subjects. The increase in plasma norepinephrine seems to reflect a change in the activity of central and peripheral noradrenergic neurons, although reduced norepinephrine clearance might partly be involved.

It has been shown that alcohol influences autonomic reflexes and blood pressure responses. Single or repeated doses of alcohol attenuate the pressor responses to the cold pressor test, handgrip exercise, and intravenous methoxamine in healthy volunteers. Howes and Reid have observed a relatively larger fall in the erect blood pressure after drinking in normotensive men, but standing and supine blood pressure fell similarly in our present study, suggesting that the influence of a single dose of alcohol on orthostatic blood pressure regulation is not significant.

As in other reports, PRA increased and vasopressin decreased after alcohol intake in our study. The increase in PRA may reflect activation of the sympathetic nervous system and may prevent severe hypotension. The decrease in vasopressin does not appear to play a role in alcohol-induced hypotension, since vasopressin has little effect on blood pressure at the plasma levels observed. However, the suppression of vasopressin release probably contributed to the relatively higher serum protein and hematocrit levels by stimulating diuresis.

Serum potassium levels decreased significantly after alcohol intake, confirming the report by Puddey et al. This fall in serum potassium may be due to an intracellular shift induced by activation of the sympathoadrenal axis. Insulin did not appear to play a role in this hypokalemia since the changes in blood glucose and serum insulin were similar between the alcohol and control days. It is possible that such hypokalemia coupled with sympathetic activation and cardiac changes could participate in the development of alcohol-induced arrhythmias.

Finally, the present study indicated that the major effect of acute alcohol consumption was to lower the blood pressure in hypertensive subjects. This depressor effect of alcohol was due to peripheral vasodilatation and was accompanied by reflex tachycardia, an increase in cardiac output, and activation of the sympathetic nervous system. Since the alcohol-induced hypotensive effect appeared to be more pronounced in individuals with a higher basal blood pressure or in elderly patients, it should be taken into account in the treatment of hypertension. The ambulatory blood pressure monitoring may be useful for assessment of the blood pressure in habitual drinkers. It is possible that the pressor effect of alcohol has been underestimated, and the depressor effect has been underestimated by measuring only the daytime blood pressure.

References

18. Kawano Y, Abe H, Yoshida K, Ashida T, Kuramoto M, Omae T: Acute cardiovascular and neurohumoral effects of alcohol in...
226 Hypertension Vol 20, No 2 August 1992


Acute depressor effect of alcohol in patients with essential hypertension.
Y Kawano, H Abe, S Kojima, T Ashida, K Yoshida, M Imanishi, H Yoshimi, G Kimura, M Kuramochi and T Omae

Hypertension. 1992;20:219-226
doi: 10.1161/01.HYP.20.2.219

Hypertension is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1992 American Heart Association, Inc. All rights reserved.
Print ISSN: 0194-911X. Online ISSN: 1524-4563

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://hyper.ahajournals.org/content/20/2/219

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Hypertension can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Hypertension is online at:
http://hyper.ahajournals.org//subscriptions/