Alcohol-Associated Hypertension
When One Drinks Makes a Difference

Arthur L. Klatsky

The presentation by Stranges et al in this issue of Hypertension contributes importantly to several of these issues. This is one of a series of analyses from this Western New York State group characterized by unusually meticulous collection of data about drinking patterns. The analysis confirms the known risk of HTN associated with recent daily intake of ≥2 drinks per day, which was more than doubled compared with abstainers. The report’s most noteworthy feature is the robust independent relationship found for alcohol taken separately from meals. The 12% of drinkers whose predominant drinking pattern was ingestion separate from food had 64% greater risk of HTN.

Although persons drinking predominantly liquor had borderline higher HTN risk versus predominant beer/wine drinkers, the independent effect of beverage type observed in the Stranges et al report is not impressive. The analysis is especially interesting on this point because wine is the beverage type most often consumed with food. Of historic interest is the fact that the first report of an alcohol–HTN association involved French soldiers who were heavy wine drinkers. Wine’s modest potassium content offers hypothetical benefit for HTN, and wine drinkers have lifestyle traits favorable for HTN risk. Yet, in agreement with this report, most available data suggest no important role of beverage choice in HTN risk.

Weekend-only drinking, independent of total amount of alcohol, had little apparent independent BP effect in the analysis. Although related, weekend-only drinking and binge drinking are not synonymous. As Stranges et al point out, there are inconsistencies in BP relationships to weekend drinking in previous reports. One relevant ecological study showed higher BPs on Mondays in Ireland, with a heavy weekend drinking pattern, but no relation to day of week in France, with a steadier drinking pattern. One might hypothesize that repeated pressor effects of alcohol might ultimately lead to irreversible changes.

Data from controlled clinical experiments verify an alcohol–HTN association and support a causal interpretation. The landmark experiment of Potter and Beevers used a crossover design in hospital in men with HTN and usual intake of ∼4 pints of British beer. The data suggested both a pressor effect of alcohol in hypertensive men and short-term BP reductions after alcohol withdrawal, with changes requiring several days to appear and not present in all subjects. No withdrawal symptoms or blood pressure rebound (overshoot) was observed. Australian reports of controlled trials of alcohol restriction in ambulatory men with HTN and normotensives showed lower pressures during alcohol withdrawal. Other clinical trials suggest that regular drinking may antagonize efficacy of drug treatment and weight reduction, that alcohol

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he high prevalence of systemic hypertension (HTN) and its progressive association with multiple adverse sequelae confer great potential importance on any modifiable trait influencing blood pressure (BP). Established modifiable factors include obesity and sodium intake. Less solidly established are physical inactivity and low potassium or calcium intake. With a relationship to HTN seen nearly unanimously in a substantial number of observational cross-sectional and prospective studies, alcohol drinking has become a major focus of interest in recent decades as a modifiable HTN risk factor. Even low estimates of 5% or 7% of HTN attributable to alcohol imply that there are more patients with HTN caused by alcohol than by conventional causes of remediable secondary HTN.

Present in both sexes and several ethnic groups, the alcohol–HTN association is independent of a number of potential confounders including nutritional factors. Available evidence suggests substantial regression of HTN in several days with alcohol abstinence. Although heavier alcohol intake (≥2 drinks daily) has been more consistently related, an alcohol–BP relationship with lighter drinking in some studies raises a possibility of great potential public health importance. Most observational analyses used average amount of alcohol consumed daily as the alcohol measure, with presumed underestimation of alcohol amount by some heavier drinkers impairing definition of a threshold dose level. To add further confusion, a few studies show lower pressures in light drinkers than in abstainers, perhaps especially in women.

Lack of a proven mechanism limits causal interpretation of the alcohol–HTN link. There are other unresolved issues, including: (1) is the relationship linear at all drinking levels or is there a threshold alcohol dose; (2) is choice of wine, liquor, or beer a factor; (3) is drinking pattern a factor; (4) what is the time sequence in drinking–BP changes; (5) are diet and/or behavioral factors confounders; and (6) does alcohol-associated HTN result in the usual HTN sequelae? All of these queries are relevant to the role of reduction of alcohol intake in HTN treatment or prevention.

The opinions expressed in this editorial are not necessarily those of the editors or of the American Heart Association.

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restriction may lower BP more than exercise, and that alcohol restriction may be more effective than Na restriction. All of these studies suggest a therapeutic benefit of alcohol withdrawal in some patients with HTN.

The BP increase does not seem to be an immediate effect of alcohol. Japanese investigators, as cited in a review, used ambulatory monitoring in hypertensives to demonstrate a short-term depressor effect of a dinner time alcohol dose of 1 mL/kg, with persistence of lower BP for up to 8 hours, but higher pressures the next morning. They also examined morning and late evening home BP for 4 weeks in a usual alcohol consumption period and for 4 weeks in alcohol restriction, finding a depressor effect of alcohol on the evening BP from day 1 to week 4, and a pressor effect on the morning BP from week 2 regardless of the order of the 2 periods. These results suggest a short-term biphasic effect of alcohol on BP. Unfortunately, these ambulatory monitoring data only involve alcohol with food. The time sequence of the alcohol–BP effect indicates that higher pressures on Monday do not necessarily represent withdrawal effects.

The “slow” (days) alcohol–BP effect raises questions about the relevance of experiments dealing with acute (minutes/hours) effects. Other issues in experiments include alcohol’s diverse effects on various target organs, differences in health and disease, and aspects related to rate, dose, route of alcohol administration, time interval to BP measurement, and, probably, psychic factors. Acute and chronic studies in humans and animals show inconsistent immediate results with respect to BP and fail to convincingly demonstrate a mechanism in terms of effects on catecholamines, renin, cortisol, norepinephrine, central nervous system effects, peripheral vascular muscle tone, calcium transport, erythrocyte cell membrane changes, insulin sensitivity, and magnesium depletion.

Because the overall pathophysiological basis for the alcohol–BP relationship remains undefined, it follows that attempts to define a basis for greater effects of alcohol without food are “speculative.” Stranges et al present an admirable list of speculations that properly includes possible confounding by other lifestyle/health traits. It would be unfortunate if the apparent advantage of taking alcohol with food were spurious, because the phenomenon so readily translates into practical advice. The cited effects of alcoholic beverages on fibrinolysis, blood lipids, and lipid peroxidation seem more likely related to protective effects against atherothrombotic conditions, but the idea of relative benefit for HTN via these mechanisms is intriguing. Of the explanations offered, beneficial effects of food–alcohol interaction on absorption and metabolism of alcohol seem the most plausible.

It is always appropriate to attempt integration of new findings into current practical guidelines. Guidelines about alcohol drinking must always emphasize the need for individual risk/benefit evaluation, the harms of heavy drinking (including increased HTN risk), and the fact that many persons should not drink at all. A balanced view considers the probable protective effect of light-to-moderate alcohol intake for coronary heart disease, a benefit that extends to those with HTN. Patterns of alcohol drinking relevant to coronary risk include increased risk from drinking outside of meals and data indicating that frequency of drinking may be more important than amount or beverage choice as a predictor of lower coronary risk.

For those who choose to be light-to-moderate drinkers, a US government guideline advises “take with meals to slow alcohol absorption.” With relevant health outcome data still relatively sparse, the recommendation was presumably based largely on hypothetical considerations and common sense. Although definitive answers about alcohol and HTN still lie ahead, the detailed consideration of the role of drinking pattern in the present study indicates that alcohol–health research is coming of age. It is gratifying to find common sense so resoundingly supported. To minimize risks and maximize benefit of light/moderate consumption of alcohol, a small amount most days with meals is the way to go.

References

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