RESEARCH LETTER

Acute cardiovascular responses elicited by consumption of beer in healthy people

Karolina Adamska[1], Tomasz Krauze[1], Przemysław Guzik[1], Jarosław Piskorski[2], Krzysztof Klimas[1], Andrzej Wykrętowicz[1]

1 Department of Cardiology-Intensive Therapy and Internal Diseases, Poznan University of Medical Sciences, Poznań, Poland
2 Faculty of Physics and Astronomy, University of Zielona Gora, Zielona Góra, Poland

Introduction The regular consumption of alcohol is associated with increased blood pressure (BP), which may be reversed during abstinence.[1] The BP-raising effect of alcohol may depend on the type of alcoholic beverage consumed and the pattern of consumption: the influence of episodic drinking of alcohol on the cardiovascular system may differ from that of regular intake.[2,3]

Beer consumption is growing each year worldwide, but there are almost no data on the acute effects of beer on the cardiovascular system. Therefore, we decided to assess the acute hemodynamic and neural cardiovascular responses elicited by the consumption of beer in healthy individuals.

Patients and methods A total of 21 healthy volunteers (13 men and 8 women; mean [SD] age, 26 [3] years; mean [SD] body mass index, 23 [3] kg/m^2) were investigated in a crossover study. All patients had normal BP, and none of them were on medication. The local ethics committee approved the study protocol, and written informed consent was obtained from all the participants.

Hemodynamic measurements Participants were studied in a supine position after 20 minutes of rest. After the baseline assessment, each individual drank 500 ml of tap water at room temperature over 15 minutes, while remaining in a semisupine position. Having finished the water, participants returned to the supine position and subsequent measurements were continued for 60 minutes. A second session with the same participants was performed 48 to 72 hours later under identical conditions, except for the substitution of water by a commercially available beer (500 ml, with an alcohol content of 5.3%; Kompania Piwowarska, Poland).

A noninvasive measurement of beat-to-beat finger BP was performed continuously with the use of a volume-clamp photoplethysmograph (Portapres 2, FMS, Enschede, the Netherlands) with the sensor on the middle finger of the right hand. Mean BP, heart rate, systemic vascular resistance (SVR) were made using the Modelflow algorithm.[4]

Baroreflex sensitivity Baroreflex sensitivity (BRS) was measured in consecutive 5-minute segments recorded before and after water or beer consumption. The BRS was calculated by the cross-correlation method, which computes a time-domain sequential BRS on spontaneous BP and RR interval variability for fixed windows of 10 seconds in length.[5] The geometric mean of the series of BRS estimates obtained from each 5-minute segment was obtained for further analysis.

Statistical analysis The continuous anthropometric data were presented as mean and SD. The continuous data of hemodynamic measurements were presented as mean and SEM. One-way analysis of variance (ANOVA) for repeated measures was used (followed by the Bonferroni test for multiple comparisons with baseline values) for their analysis. Two-way ANOVA, followed by the Bonferroni test for multiple comparisons, was used to assess differences between groups. A P value of less than 0.05 was considered significant. All tests were 2-tailed. Tests were performed using GraphPad Prism version 5.00 for Windows (GraphPad Software, La Jolla, California, United States).

Results **Systolic blood pressure response** The mean (SD) systolic BP at baseline was 122 (4) mm Hg and increased significantly after water ingestion (P <0.0001, 1-way ANOVA). It remained elevated after 20 minutes (mean [SD], 134 [4] mm Hg, P <0.001], 30 minutes (mean [SD], 133 [4] mm Hg, P <0.001], 40 and 50 minutes (mean [SD], 134 [4] mmHg and 135 [4] mm Hg, both

Correspondence to:
Prof. Andrzej Wykrętowicz, MD, PhD, Katedra i Klinika Intensywnej Terapii Kardiologicznej i Chorób Wewnętrznych, Uniwersytet Medyczny w Poznaniu, ul. Przybyszewskiego 49, 60-355 Poznań, Poland, phone: +48 61 869 13 91, email: awykreto@ptkardio.pl
Received: April 20, 2018.
Revision accepted: May 8, 2018.
Published online: May 16, 2018.
Conflict of interests: none declared.
Pol Arch Intern Med. 2018; 128 (6): 400-402
doi:10.20452/pamw.4266

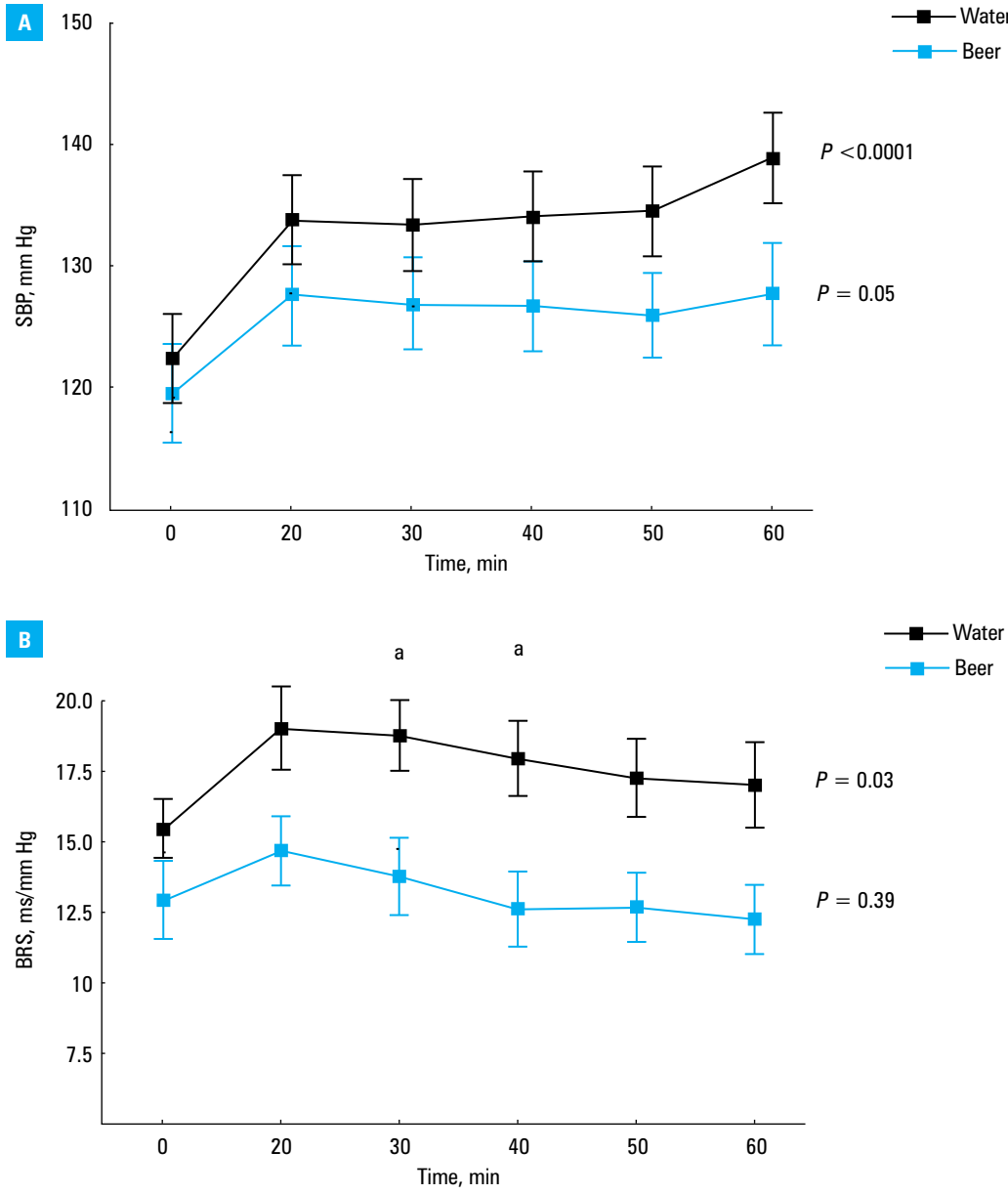

FIGURE 1 Changes in systolic blood pressure (SBP) (A) and baroreflex sensitivity (BRS) (B)
a Difference between groups; 2-way analysis of variance. Data are presented as mean (SEM).

P <0.001), and up to 60 minutes (mean [SD], 139[4] mm Hg, P <0.001; 1-way ANOVA followed by the Bonferroni test for multiple comparisons with baseline values).

During the second experiment involving beer consumption, the mean (SD) systolic BP at baseline was 120 (4) mm Hg and did not change after beer consumption (P = 0.05, 1-way ANOVA) (FIGURE 1A).

Two-way ANOVA did not reveal significant differences between groups in systolic BP during the consumption of water or beer at any of the time points.

Diastolic blood pressure and heart rate responses, systemic vascular resistance Two-way ANOVA revealed that diastolic BP, mean BP, and systemic vascular resistance during water or beer consumption did not differ significantly between study groups at any of the time points (data not shown).

Baroreflex sensitivity The mean (SD) BRS at baseline was 15.5 (2) ms/mm Hg and changed after water consumption (P = 0.03, 1-way ANOVA, FIGURE 1B). It increased after 20 minutes (mean [SD], 19.1 [1] ms/mm Hg, P <0.05; 1-way ANOVA).

The mean (SD) BRS before beer consumption was 13.5 [1] ms/mm Hg and did not change after the consumption (P = 0.39, 1-way ANOVA, FIGURE 1B).

The mean (SD) BRS was lower 30 and 40 minutes after beer consumption compared with the values after water ingestion: 18.8 (1) ms/mm Hg vs 13.8 (1) ms/mm Hg, respectively, at 30 minutes, and 18.0 (1) ms/mm Hg vs 12.6 (1) ms/mm Hg, respectively, at 40 minutes; P <0.05 for both comparisons.

Discussion Water consumption by healthy people is associated with a pressor effect.[5,6] In controlled clinical studies, the consumption was followed by an increase in both systolic and diastolic BP, a decrease in heart rate, and an increase in peripheral resistance. Water drinking improves orthostatic tolerance and may constitute an effective prophylaxis against vasovagal reactions.[7]

The pressor effect of oral water intake is particularly prominent in patients with autonomic failure.[8]

We observed that the ingestion of 500 ml of tap water was associated with a significant rise in systolic BP, which is in line with those observed by other authors.[8] This elevation of BP was significant at 20 minutes and reached a maximum increase at 60 minutes. Assessment of muscle sympathetic neural activity in healthy individuals showed that water ingestion increases sympathetic nerve traffic, leading to peripheral vasoconstriction.[9] In healthy individuals, an increase in vascular resistance and rise in BP is followed by a BRS-mediated reduction in heart rate and a compensatory adjustment of the pressor response. We found that water ingestion was followed by a significant increase in BRS activity.

The relationship between alcohol consumption and BP responses depends on the amount of alcohol, the pattern of drinking (daily versus occasional), and the type of beverage, all of which are confounded by different factors, including dietary habits. Recently, Zilkens et al[3] showed that 4-week consumption of red wine or beer by individuals with normal BP increased awake systolic and diastolic BP as determined from 24-hour ambulatory BP monitoring. Dealcoholized red wine did not affect the BP in the population studied. Red wine, dealcoholized red wine, and beer did not affect vascular function as assessed by flow-mediated dilatation.[3] These data suggest that red wine polyphenolics do not influence nitric oxide–mediated arterial dilation and do not mitigate the BP-elevating effects of alcohol, at least in the model employed by Zilkens et al.[3] Tawakol et al[10] evaluated the direct effect of ethanol on human vascular function and showed that it acutely induces vasoconstriction at rest. Moreover, intra-arterial infusion of ethanol did not increase mean BP.[10]

In our present study, the ingestion of 500 ml of beer by healthy subjects was associated with increased systolic, diastolic, and mean BP (data not shown). However, the overall changes in BP and vascular resistance response did not differ from those observed after water consumption. BRS did not improve significantly after beer drinking in comparison with the effects of water consumption. Our findings are to some extent in agreement with previous reports showing reduced BRS after alcohol administration.[11] The mechanisms underlying the effect of alcohol on BRS range from direct interference with central regulation of the reflex to local carotid vasodilation with decreased shear stress at receptor sites.[12]

An important new finding in our present study is that the significant rise in BP observed after beer consumption in healthy individuals did not differ from that observed after water ingestion. Although Zilkens et al[3] were unable to demonstrate that wine or beer enhance vasodilation through nitric oxide–mediated mechanism, Tawakol et al[10] showed that alcohol increased arterial dilation through a nitric oxide–independent pathway. Thus, it may be argued that the immediate effect of beer consumption on BP was mitigated by as yet unidentified vasodilatory properties of alcohol or other beer components.

A relatively small sample size of the studied group and thus a lack of possibility to exclude sex differences in the observed responses are limitations of our study.

In summary, this study of healthy people with normal BP demonstrated that the ingestion of beer results in prompt elevation of BP, although this effect did not differ from that observed after tap water ingestion. However, unlike water ingestion, consumption of beer was not accompanied by a significant improvement of BRS.

REFERENCES

1 Puddey IB, Beilin LJ, Vandongen R, et al. Evidence for a direct effect of alcohol consumption on blood pressure in normotensive men: a randomized controlled trial. Hypertension. 1985; 7: 707-713.

2 Klatsky AL, Friedman GD, Armstrong MA. The relationships between alcoholic beverage use and other traits to blood pressure: a new Kaiser Permanente study. Circulation. 1986; 73: 628-636.

3 Zilkens RR, Burke V, Hodgson JM, et al. Red wine and beer elevate blood pressure in normotensive men. Hypertension. 2005; 45: 874-879.

4 Harms MP, Wesseling KH, Pott F, et al. Continuous stroke volume monitoring by modelling flow from non-invasive measurement of arterial pressure in humans under orthostatic stress. Clin Sci (Lond). 1999; 97: 291-301.

5 Jordan, J, Shannon JR, Grogan E, et al. A potent pressor response elicited by drinking water. Lancet. 1999; 353: 723.

6 Jordan J, Shannon JR, Black BK, et al. The pressor response to water drinking in humans. Circulation. 2000; 101: 504-509.

7 Lu CC, Diedrich A, Tung CS, et al. Water ingestion as prophylaxis against syncope. Circulation. 2003; 108: 2660-2665.

8 Cariga P, Mathias CJ. Haemodynamics of the pressor effect of oral water in human sympathetic denervation due to autonomic failure. Clin Sci (Lond). 2001; 101: 313-319.

9 Scott EM, Greenwood JP, Gilbey SG, et al. Water ingestion increases sympathetic vasoconstrictor discharge in normal human subjects. Clin Sci (Lond). 2001; 100: 335-342.

10 Tawakol A, Omland T, Creager MA. Direct effect of ethanol on human vascular function. Am J Physiol Heart Circ Physiol. 2004; 286: H2468-H2473.

11 Fazio M, Bardelli M, Macaluso L, et al. Mechanics of the carotid artery wall and baroreflex sensitivity after acute ethanol administration in young healthy volunteers. Clin Sci (Lond). 2001; 101: 253-260.

12 Zhang X, Abdel-Rahman AR, Wooles WR. Impairment of baroreceptor reflex control of heart rate but not sympathetic efferent discharge by central neuroadministration of ethanol. Hypertension. 1989; 14: 282-292.