Acute cardiovascular responses elicited by consumption of beer in healthy people

Authors: Karolina Adamska, Tomasz Krauze, Przemysław Guzik, Jarosław Piskorski, Krzysztof Klimas, Andrzej Wykrętowicz

Article type: Research letter

Received: April 20, 2018

Accepted: May 8, 2018

Published online: May 16, 2018

ISSN: 1897-9483

This is an Open Access article distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 International (CC BY-NC-SA 4.0) License (http://creativecommons.org/licenses/by-nc-sa/4.0/), allowing third parties to copy and redistribute the material in any medium or format and to remix, transform, and build upon the material, provided the original work is properly cited, distributed under the same license, and used for noncommercial purposes only. For commercial use, please contact the journal office at pamw@mp.pl.
Acute cardiovascular responses elicited by consumption of beer in healthy people

Karolina Adamska¹, Tomasz Krauze¹, Przemysław Guzik¹, Jarosław Piskorski²,³, Krzysztof Klimas¹, Andrzej Wykrętowicz¹

¹Department of Cardiology-Intensive Therapy, Poznan University of Medical Sciences, 49 Przybyszewskiego, 60-355 Poznan, Poland
²Department of Physics, University of Zielona Gora, Zielona Gora, Poland
³Institute of Physics, University of Zielona Gora, 4a Szafrana, 65-516 Zielona Gora, Poland

Correspondence to:
Andrzej Wykrętowicz,
Klinika Intensywnej Terapii Kardiologicznej i Chorób Wewnętrznych
Uniwersytet Medyczny w Poznaniu,
49 Przybyszewskiego,
60-355 Poznań,
Poland
Email: awykreto@ptkardio.pl
Tel: +48618691391

Running title: Beer and cardiovascular system

Disclosures of conflicts of interest: None.
1. Introduction

The regular consumption of alcohol is associated with increased blood pressure, which may be reversed during abstinence [1]. The blood pressure-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 a regular intake [2,3].

World beer consumption is growing each year 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.

2. Material and methods

Twenty one healthy volunteers (13 men, 8 women, mean (SD) age 26 (3) years, mean body mass index 23 (3) kg/m², were studied in cross-over design investigation. All the subjects were normotensive and none was taking any medication. The University Ethics Committee approved the study protocol and informed consent was obtained from all the participants.

Hemodynamic measurements

Subjects were studied in the supine position after 20 minutes of rest. After the baseline assessment, each subject drank 500 ml of tap water at room temperature, over 15 min while remaining in a semisupine position. Having finished the water the study subjects returned to the supine position and subsequent measurements were continued for 60 minutes. A second session with the same subjects was performed 48 - 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).

Non-invasive beat-to-beat finger arterial blood pressure (BP) was recorded continuously with the use of a volume-clamp photoplethysmograph (Portapres 2, FMS, The Netherlands) with the sensor on the middle finger of the right hand. Calculations of mean blood pressure, heart rate, systemic vascular resistance (SVR) were made using the Modelflow algorithm [4].

Baroreflex sensitivity (BRS)

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

3. Statistical analysis

The continuous anthropometric data are presented as mean and standard deviation (SD). The continuous data of hemodynamic measurements are presented as the mean (SEM), (standard error of the mean). One-way ANOVA for repeated measures was used (followed by Bonferroni’s test for multiple comparisons against baseline) for their analysis. Two-way ANOVA, followed by Bonferroni’s test for multiple comparisons, was used for the assessment of differences between groups. Only p < 0.05 was considered significant. All tests were 2-tailed. Tests were performed using GraphPad Prism version 5.00 for Windows (GraphPad Software, USA)
4. Results

Blood pressure responses

a) Systolic BP

The mean pre-ingestion basal systolic BP was 122 (4) mmHg and rose significantly after water ingestion \((P < 0.0001, \text{one-way ANOVA})\). Values were elevated after 20 minutes [134 (4) mm Hg, \(P < 0.001\)], 30 minutes, [133 (4) mm Hg, \(p<0.001\)], 40 and 50 minutes[134 (4) mmHg, 135 ± 4 mm Hg, \(P < 0.001, P < 0.001 \text{ respectively}\)] up to [139 (4) mm Hg in 60 min \((P < 0.001)\)], (one-way ANOVA, followed by Bonferroni’s test for multiple comparisons against baseline).

During the second experiment involving beer consumption, the basal systolic BP was 120 (4) mm Hg and did not change significantly after beer consumption \((P = 0.05, \text{one-way ANOVA})\), (Figure 1A).

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

b) Diastolic BP, mean BP, heart rate response, systemic vascular resistance

Two-way ANOVA revealed that the diastolic BP, mean BP and SVR during consumption of water or beer did not differ significantly between study groups (water vs beer) at any of the time points (data not shown).
Baroreflex sensitivity

The basal BRS prior to the ingestion of water was 15.5 (2) ms/mm Hg and changed significantly after water consumption (Figure 1B, \( P = 0.03 \), one-way ANOVA). BRS increased significantly after 20 minutes [19.1 (1) ms/ mm Hg, \( P < 0.05 \), one-way ANOVA, followed by Bonferroni’s test for multiple comparisons against baseline].

Prior to the beer ingestion the basal BRS was 13.5 (1) ms/mm Hg and it did not change significantly after the beer had been consumed (\( P = 0.39 \), one-way ANOVA), (Figure 1B).

Two-way ANOVA revealed that 30 and 40 minutes after beer consumption BRS was significantly lower in comparison to values obtained at similar intervals following water ingestion [18.8 (1) vs 13.8 (1) ms/mm Hg and 18.0 (1) vs 12.6 (1) ms/mm Hg respectively, \( P < 0.05 \), \( P < 0.05 \) respectively].

5. Discussion

Water consumption by healthy people is associated with a pressor effect [5,6]. The ingestion of water in controlled clinical studies was followed by an increase in both systolic and diastolic blood pressure, 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].

Our results are in keeping with those observed by others, namely, the ingestion of 500 ml of tap water was associated with a significant rise in systolic blood pressure [8]. This elevation of BP was significant after 20 min of water ingestion and reached a maximum increase after 60 min. In assessing muscle sympathetic neural activity in healthy subjects, it
has been shown that water ingestion increases sympathetic nerve traffic, leading to peripheral vasoconstriction [9]. In healthy subjects an increase in vascular resistance and rise in BP is followed by a BRS-mediated reduction in the 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 blood pressure responses depends on the amount of alcohol ingestion, the pattern of drinking (daily versus occasionally), and the type of beverage, all of which are confounded by different factors, including dietary habits. Recently Zilkens et al. [3] showed that 4 weeks consumption of red wine or beer by normotensive subjects increased awake systolic and diastolic blood pressure as determined from 24-hour ambulatory blood pressure monitoring. De-alcoholized red wine did not affect the BP in the population studied. Neither red wine, de-alcoholized red wine nor beer affected vascular function as assessed by flow-mediated dilatation [3]. These data suggest that red wine polyphenolics do not influence nitric-oxide mediated arterial dilatation and do not mitigate the blood pressure-elevating effects of alcohol, at least in the model employed by Zilkens [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 blood pressure [10].

In our present study, the ingestion of a 500 ml of beer by healthy subjects was associated with increased systolic as well as diastolic and mean blood pressure (data not shown). However, the overall changes in BP and vascular resistance response did not differ from those observed after water consumption. The baroreflex did not improve significantly after beer drinking in contrast with the observation noticed after water consumption. Our findings are to some extent in agreement with previous reports showing reduced baroreflex sensitivity after alcohol administration [11]. Explanation for the mechanism by which alcohol
affects BRS range from direct interference with central regulation of the reflex to local carotid vasodilatation with decreased shear stress at receptor sites [12].

An important new finding in our present study is that the significant rise in blood pressure observed after beer consumption in healthy subjects did not differ from that observed after water ingestion. Although Zilkens et al. [3] were unable to demonstrate that wine or beer enhance vasodilatation through nitric-oxide mediated mechanism, Tawakol [10] showed that alcohol increased arterial dilatation through a NO-independent pathway. Thus, it may be argued that the immediate effect of beer consumption on BP was mitigated by as yet unidentified vasodilatatory properties of alcohol or other beer components.

Study limitations

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

Conclusions

In summary, this study of healthy, normotensive people has demonstrated that the ingestion of beer promptly elevates blood pressure, although this effect was no different from that observed after tap water ingestion. However, consumption of beer, in contrast to water ingestion was not accompanied by a significant improvement of BRS.
6. References


Figure 1

Figure (part A) shows changes in systolic (SBP) and (part B) in baroreflex sensitivity (BRS). * indicates significant difference between groups in two-way ANOVA comparison. Data are presented as mean ± SEM.