a trigger for PoAF (3, 4). In conclusion, these confounding factors will probably explain this association, but many of them were not taken into consideration in the analysis.

Ali Rıza Akyüz, Levent Korkmaz
Department of Cardiology, Trabzon Ahi Evren Cardiovascular and Thoracic Surgery Research and Application Center, Saglik Bilimleri University; Trabzon-*Turkey*

References

1. Geçmen Ç, Güler GB, Erdoğan E, Hatipoğlu S, Güler E, Yılmaz F, et al. SYNTAX score predicts postoperative atrial fibrillation in patients undergoing on-pump isolated coronary artery bypass grafting surgery. Anatol J Cardiol 2015 Nov 18. Epub ahead of print.
2. Lau DH, Alasady M, Brooks AG, Sanders P. New-onset atrial fibrillation and acute coronary syndrome. Expert Rev Cardiovasc Ther 2010; 8: 941-8. [Crossref]
3. Ichiki H, Orihara K, Hamasaki S, Ishida S, Oketani N, Iriki Y, et al. The role of infection in the development of non-valvular atrial fibrillation: up-regulation of Toll-like receptor 2 expression levels on monocytes. J Cardiol 2009; 53: 127-35. [Crossref]
4. Gedikli O, Örem C, Baykan M, Karahan C, Küçükosmanoğlu M, Şahin S, et al. Association between serum C-reactive protein elevation and atrial fibrillation after first anterior myocardial infarction. Clin Cardiol 2008; 31: 482-7. [Crossref]

Address for Correspondence: Dr. Ali Riza Akyüz
Sağlık Bilimleri Üniversitesi, Ahi Evren Göğüs ve Kalp Damar Cerrahisi Sağlık Uygulama ve Araştırma Merkezi ; Trabzon-*Türkiye*
Phone: +90 462 231 41 14 Fax: +90 462 231 24 20
E-mail: dralirizaakyuz@gmail.com

DOI:10.14744/AnatolJCardiol.2016.7084

Author`s Reply

To the Editor,

We are pleased with the authors' interest in our article titled "SYNTAX score predicts postoperative atrial fibrillation in patients undergoing on-pumping isolated coronary artery bypass grafting surgery," which was published in Anatol J Cardiol 2015 Nov 18 Epub ahead of print (1), and we would like to thank them for their contribution. As the authors have mentioned systemic inflammation caused by cardiopulmonary bypass, atrial inflammation might contribute to the occurrence of postoperative atrial fibrillation (PoAF). Bruins et al. (2) reported that an elevation in C-reactive protein-complement complexes was greater in patients who developed AF. Soluble vascular cell adhesion molecule-1 (VCAM-1) is an emerging biomarker for inflammation and endothelial activation. In another study, Verdejo et al. (3) reported that in patients undergoing coronary artery bypass surgery, elevated VCAM-1 levels predict a higher risk for PoAF. However, inflammatory markers were not included in our patient data, and we think that the patient population is too small to add these variables in the analysis. There would be too many variables for a small group, and this could disrupt the results. With the inclusion of these data, our hypothesis can be further tested in a bigger patient population.

Çetin Geçmen
Department of Cardiology, Kartal Koşuyolu Heart and Research Hospital; İstanbul-*Turkey*

References

1. Geçmen Ç, Güler GB, Erdoğan E, Hatipoğlu S, Güler E, Yılmaz F, et al. SYNTAX score predicts postoperative atrial fibrillation in patients undergoing on-pump isolated coronary artery bypass grafting surgery. Anatol J Cardiol 2015 Nov 18. Epub ahead of print.
2. Bruins P, te Velthuis H, Yazdanbakhsh AP, Jansen PG, van Hardevelt FW, de Beaumont EM, et al. Activation of the complement system during and after cardiopulmonary bypass surgery: postsurgery activation involves C-reactive protein and is associated with postoperative arrhythmia. Circulation 1997; 96: 3542-8. [Crossref]
3. Verdejo H, Roldan J, Garcia L, Del Campo A, Becerra E, Chiong M, et al. Systemic vascular cell adhesion molecule-1 predicts the occurrence of post-operative atrial fibrillation. Int J Cardiol 2011; 150: 270-6. [Crossref]

Address for Correspondence: Dr. Çetin Geçmen
Kartal Koşuyolu Kalp Araştırma Hastanesi, Kardiyoloji Bölümü
34846, Kartal; İstanbul-*Türkiye*
Fax: +90 216 500 15 00 E-mail: drcetingecmen@hotmail.com

Effects of energy drinks on blood pressure, heart rate, and electrocardiographic parameters: an experimental study on healthy young adults

To the Editor,

I have read the article entitled "Effects of energy drinks on blood pressure, heart rate, and electrocardiographic parameters: an experimental study on healthy young adults" by Hajsadeghi et al. (1), which was recently published in the Anatolian Journal of Cardiology 2016; 16: 94-9, with great interest. The investigators reported that energy drink consumption could contribute to heart rate decrease and ST-T changes in healthy young adults. In addition, systolic and diastolic BP and other ECG parameters do not significantly change after the energy drink consumption (1).

There were conflicting results about the relationship between heart rate response and energy drink consumption (2, 3). Authors implied that the possible mechanism underlying the heart rate decrease was related to an increase in the stroke volume and enhancement of the myocardial contractility after the energy drink consumption (1). Authors claimed that excessive catecholamine release after energy drink consumption is the mechanism underlying the significant ST-T changes (1). It is well known that heart rate

increase is an essential effect of catecholamine release. The possible reason underlying these conflicting results is related to the follow-up duration and amount of energy drink consumed (2, 3).

In addition, there was no data regarding smoking history or current smoking status. The vasopressor and tachycardia effects of smoking are associated with an increase in the plasma catecholamine concentration (4). Likewise, there was a strong relationship between heart rate variability and smoking (5).

In the light of this knowledge, authors should mention regarding the smoking habits of participants. Moreover, the follow-up duration and amount of energy drinks consumed should be standardized.

Levent Cerit
Department of Cardiology, Near East University, Nicosia-*Turkish Republic of Northern Cyprus*

References

1. Hajsadeghi S, Mohammadpour F, Manteghi MJ, Kordshakeri K, Tokazebani M, Rahmani E, et al. Effects of energy drinks on blood pressure, heart rate, and electrocardiographic parameters: An experimental study on healthy young adults. Anatol J Cardiol 2016; 16: 94-9.
2. Geiss KR, Jester I, Falke W, Hamm M, Waag KL. The effect of a taurine-containing drink on performance in 10 endurance-athletes. Amino Acids 1994; 7: 45-56. [Crossref]
3. Steinke L, Lanfear DE, Dhanapal V, Klaus JS. Effect of "energy drink" consumption on hemodynamic and electrocardiographic parameters in healthy young adults. Ann Pharmacother 2009; 43: 596-602.
4. Grassi G, Seravalle G, Calhoun DA, Bolla GB, Giannattasio C, Marabini M, et al. Mechanisms responsible for sympathetic activation by cigarette smoking in humans. Circulation 1994;90:248-53.
5. Bianchim MS, Sperandio EF, Martinhão GS, Matheus AC, Lauria VT, da Silva RP, et al. Correlation between heart rate variability and pulmonary function adjusted by confounding factors in healthy adults. Braz J Med Biol Res 2016 Mar 3. Epub ahead of print. [Crossref]

Address for Correspondence: Dr. Levent Cerit
Near East Hospital University Hospital, Nicosia-*Turkish Republic of Northern Cyprus*
Phone: +90 392 675 10 00 E-mail: drcerit@hotmail.com

DOI:10.14744/AnatolJCardiol.2016.7184

Author`s Reply

To the Editor,

We thank the authors for their great interest in our work entitled "Effects of energy drinks on blood pressure, heart rate, and electrocardiographic parameters: an experimental study on healthy young adults" published in Anatol J Cardiol 2016; 16: 94-9 (1). In addition to our discussion, they also notified the incoherent results of different studies on the heart rate (HR) response after energy drink consumption. Authors of the letter, however, stated that an HR increase is an essential effect of catecholamine release and then considered the combination of "HR decline" and "catecholamine release" as conflicting. Although we agree that an HR increase is an effect of situations with pure catecholamine release, it must be emphasized that a combination of "HR decline and catecholamine release" is also possible in some conditions, of which the most well-known is the Cushing reflex. As we supposed in the article, the HR decrease after energy drink consumption is possibly due to direct central stimulation of the vagus nerve by caffeine (2). A similar mechanism has been previously described for the Cushing reflex where concurrent hypertension (owing to sympathetic activation) and bradycardia (owing to the vagus nerve stimulation) are seen (3).

Given the known effects of smoking on sympathetic activation (4), as noted by the authors of the letter, we excluded all smokers from our study to avoid its possible confounding role on results. This was mentioned in the paper by excluding those with a history of "substance abuse." According to the valid definitions, including Diagnostic and Statistical Manual of Mental Disorders-Fifth Edition (DSM-5) (5), tobacco consumption is an example of substance abuse.

Finally, as we discussed in the abovementioned article (1), factors such as different types of energy drinks and durations of BP monitoring after energy drink consumption are among the possible reasons underlying the conflicting results of different studies on hemodynamic effects of energy drinks. Thus, we agree with the authors of the letter that considering follow-up duration and amount of energy drinks are of great importance in comparing the results of different investigations on energy drinks.

Shokoufeh Hajsadeghi
Department of Cardiology, Rasoul-e-Akram Hospital, Faculty of Medicine, Iran University of Medical Sciences; Tehran-*Iran*

References

1. Hajsadeghi S, Mohammadpour F, Manteghi MJ, Kordshakeri K, Tokazebani M, Rahmani E, et al. Effects of energy drinks on blood pressure, heart rate, and electrocardiographic parameters: An experimental study on healthy young adults. Anatol J Cardiol 2016; 16: 94-9.
2. Whitsett TL, Manion CV, Christensen HD. Cardiovascular effects of coffee and caffeine. Am J Cardiol 1984; 53: 918-22. [Crossref]
3. Hackett JG, Abboud FM, Mark AL, Schmid PG, Heistad DD. Coronary vascular responses to stimulation of chemoreceptors and baroreceptors:evidence for reflex activation of vagal cholinergic innervation. Circ Res 1972; 31: 8-17. [Crossref]
4. Grassi G, Seravalle G, Calhoun DA, Bolla GB, Giannattasio C, Marabini M, et al. Mechanisms responsible for sympathetic activation by cigarette smoking in humans. Circulation 1994; 90: 248-53.
5. Hasin DS, O'Brien CP, Auriacombe M, Borges G, Bucholz K, Budney A, et al. DSM-5 criteria for substance use disorders: recommendations and rationale. Am J Psychiatry 2013; 170: 834-51. [Crossref]

Address for Correspondence: Shokoufeh Hajsadeghi, MD,
Department of Neurology, Boston Children's Hospital
Harvard Medical School, 1 Autumn St, Boston, MA 02115-*USA*
Phone: (617) 919-6232 E-mail: Fatemeh.touserkani@childrens.harvard.edu