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Author`s Reply

To the Editor,

We enthusiastically read the letter regarding our article titled "Arterial stiffness evaluation in patients with irritable bowel syndrome: role of antihypertensive drugs and statins" published in Anatol J Cardiol 2014; 14: 525-30 (1).

Increased arterial stiffness reflecting decreased arterial compliance is an important marker of vascular aging (2). We demonstrated that carotid-femoral pulse wave velocity (PWV), the current gold standard measure of arterial stiffness did not differ between patients with irritable bowel disease and healthy control subjects (1). Arterial stiffness is mainly associated with aging and hypertension (3). As the authors kindly mentioned, antihypertensive drug groups tend to have different effects on arterial stiffness besides blood pressure-lowering effects. Angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, calcium channel blockers, and mineralocorticoid antagonists decrease PWV, whereas diuretics and β-blockers (except nebivolol) have neutral or negative influence (4). The effect of statins on arterial stiffness is still controversial due to conflicting results (5, 6). Although, 23% of patient population and 37% of control group had hypertension in our study, there was no significant difference between the groups. Unfortunately, we did not record antihypertensive drug groups at inclusion; thus, we do not have the relevant data. We excluded patients on β-blocker treatment due to the impact on heart rate variability. Due to the facts that the percentage of hypertensive patients was not different statistically, exclusion of β-blocker treatment, and having only one patient on statin treatment within each group, we do not think these presumed drug associations would have influenced our results. We thank the authors for their scrutiny and valuable remarks.

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Can epicardial adipose tissue predict coronary artery plaque?

To the Editor,

We read with great interest the manuscript written by Çullu et al. (1) titled "Does epicardial adipose tissue volume provide information about the presence and localization of coronary artery disease?" published in the May 2015 issue of Anatol J Cardiol 2015; 15: 355-9. In that study, authors investigated the relationship between the epicardial adipose tissue (EAT) volume and the atherosclerotic coronary artery plaques evaluated by computed tomography (CT). In this study, EAT volumes were found to be significantly higher in patients with coronary plaques than in patients without plaques. Furthermore, the left anterior descending (LAD) artery and multivessel located coronary atheromatous plaques were associated with higher EAT volumes than other coronary artery locations as well as with the absence of coronary plaques. One of the most important finding in this study is that the frequency of

diabetes mellitus (DM), hypertension (HT), and dyslipidemia were found to be significantly higher not only in cases with plaque but also in cases with increased EAT volume.

Similar results were shown in studies that evaluated the relationship between EAT and DM, HT, and hyperlipidemia (2-4). However, it is not clear whether EAT volume could predict the presence of plaque in coronary arteries in the current study (1). Both EAT volumes and risk factors for atherosclerosis, including DM, HT, hyperlipidemia, and age, are higher in patients with coronary plaque. Thus, in that case, multivariate regression analysis should be made to adjust for the confusing effects of these risk factors. It is impossible to say that "EAT volumes predict the presence of coronary plaque and plaque-involved vessels." If the EAT volume is found as an independent predictor for coronary plaque after regression analysis, the ROC analysis can be used to determine the cut-off value. Otherwise, it would be more appropriate to say that EAT volume is a "risk factor" for coronary plaque. Finally, coronary artery calcium (CAC) scores were written as mean±standard deviation, such as 53.4±138 and 80±163, in Table 1. We think that CAC score does not show the normal distribution; therefore, it should be represented as median with minimum and maximum range.

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Author`s Reply

To the Editor,

Thank you for your interest in our article titled "Does epicardial adipose tissue volume provide information about the presence and localization of coronary artery disease?" published in the May 2015 issue of Anatol J Cardiol 2015; 15: 355-9 by Çullu et al. (1). We have read your letter. In previous articles, it was stated that the EAT volume was the predictor of coronary artery plaque existence (2, 3). Firstly, multivariate regression analysis was performed in our study. EAT volume was found as an independent predictor in estimating the existence of coronary artery plaque (p=0.001). Secondly, the CAC score distribution does not statistically exhibit normal distribution. We agree with the reader in this regard. The CAC score median (min–max) values are 0.0 (0.0–5.0) and 32.1 (0.0–940.8), respectively, in the existence and absence of coronary artery plaque.

Thank you for the contribution you have made to our article.

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Preoperative oral pentoxifylline in case of coronary artery bypass grafting with left ventricular dysfunction (ejection fraction equal to/less than 30%)

To the Editor,

We want to congratulate Mansourian et al. (1) on their interesting and original manuscript titled "Preoperative oral pentoxifylline in case of coronary artery bypass grafting with left ventricular dysfunction (ejection fraction equal to/less than 30%)" published in Anatol J Cardiol Dec 31, 2014.

As pentoxifylline has a reducing effect upon inflammation, it is known that the increased plasma levels of TNF-alpha and interleukin (IL)-6 will decrease when pentoxifylline is used during inflammation (2). The section of the manuscript that raises a question in our minds is the unexpected difference in the TNF-alpha and interleukin levels of oral pentoxifylline, which was started 3 days before the operation, in the blood samples obtained preoperatively from the control and pentoxifylline groups. The mean preoperative levels of TNF-alpha and IL-6 in the control group were