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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 plague. 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 plague 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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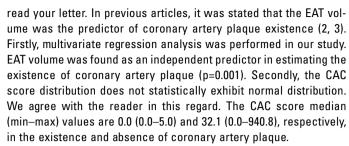
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## Author's Reply

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



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

### Neşat Çullu

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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



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139.0 and 133.4, respectively, whereas the corresponding levels in the group treated with pentoxifylline were in contrast to the expected values, 472.0 and 195.0, respectively; As opposed to the expected results, these markers of inflammation were found to be significantly higher in the pentoxifylline group. How can we explain this paradox? On the other hand, as seen in Table 2, the TNF-alpha and IL-6 levels were observed to be significantly decreased following surgery in the control group. Considering the inflammation-triggering effect of surgery, how can the decreased inflammation in the control group be explained?

Pentoxifylline is known to be a non-selective phosphodiesterase (PDE) inhibitor that is used in the treatment of peripheral arterial disease. It produces changes in red blood cells, decreases blood viscosity, and most importantly, it inhibits platelet aggregation (3). It was emphasized in your manuscript that on comparing of the two groups, one treated with drugs to promote platelet aggregation and the other being the control group, bleeding and requirement for transfusion was found to be significantly lower in the group treated with pentoxifylline than in the control group. Were there any differences between the two groups in terms of antiplatelet and anticoagulant use? How did you reach the conclusion that the use of this drug for platelet aggregation resulted in a significantly lower rate of bleeding and requirement for blood transfusion in the group treated with pentoxifylline than that in the control group?

It is well documented that drugs such as statins, renin—angiotensin—aldosterone system antagonists, and carvediol, which are in frequent use prior to a cardiopulmonary bypass, have very significant positive effects on inflammation. Naturally, we think that when comparing groups, the possibility that drugs may affect the results and whether or not any absolute differences exist in the distribution of the groups should be mentioned (4, 5). In addition, it would be appropriate to compare the cardiovascular risk factors such as diabetes, hypertension, and dyslipidemia between the two groups.

Pentoxifylline was reported in this study as having a positive effect on left ventricular ejection fractions. It is well known that image quality is low in transthoracic echocardiography following bypass surgery, and difficulties are encountered while obtaining images of sufficient quality to determine the endocardial borders in a quantitative analysis. The study failed to mention how the LVEF is evaluated in the methods section. Was a visual method or the Simpson method used in this evaluation?

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# **Author's Reply**

To the Editor,

We want to appreciate you and the author(s) of the manuscript who had precisely read our article by Mansourian et al. (1) 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 2014 Dec 31, (1). The results that are mentioned in the tables of the article are the same as those that had been carefully collected. Considering that this study was a randomized controlled trial, which is the gold standard of clinical research, we had seriously considered the randomization or allocation rules. We paid attention to some different values; therefore, we have considered it in our statistical analysis, and we conducted our analysis for the changes between the groups and not just for the crude results. This has also been indicated under the title of "Study Limitations" in the article. Moreover, the patients in the two groups did not show significant statistical differences among their major coronary artery disease (CAD) risk factors, including hypertension, hyperlipidemia, diabetes mellitus, and cigarette smoking. These parameters were eliminated from the tables just for simplicity. Furthermore, all the patients were under similar medication protocols in terms of antihyperlipidemic, antiplatelet, and anticoagulant use. I hope that these explanations are now clear and can resolve the gray areas.

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