route does not influence mortality rates (5). We think that this situation may be related to experiences of the heart team and operators.

Secondly, after graft insertion to the left iliac artery, the patient was transferred to the catheterization laboratory immediately. Therefore, the patient underwent anaesthesia stress once. However, this procedure increases infection risk due to graft operation. The rate of graft infections is expected to be low (6).

In conclusion, we presented an alternative technique for patients with an unsuitable anatomy. Improvements and further trials are needed to compare different routes.

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Evaluation of heart rate recovery index in heavy smokers

To the Editor,

I read the article entitled "Evaluation of heart rate recovery index in heavy smokers" by Erat et al. (1), which has been recently published in Anatolian Journal of Cardiology 2016; 16: 667-72, with great interest. The authors have successfully manifested a statistically significant relationship between smoking and the heart rate recovery index (HRRI) even though the study population was small in number.

HRRI, which is indicator of the autonomic nervous system (ANS), is not routinely evaluated in daily clinical practice even though it is an independent risk factor for cardiovascular (CV) diseases. Several studies have shown that HRRI plays an important role in all-cause mortality and CV events (2, 3). The authors have done a good job by investigating the relationship between HRRI and smoking because the potential harmful effects of smoking on the autonomic nervous system apart from those on the vascular biology needed to be proved. HRRI calculation is a simple and beneficial way to evaluate autonomic nervous system function. Therefore, this trial will help us understand the harmful effects of smoking on ANS using HRRI.

To our knowledge, HRRI is calculated by extracting the heart rate during the 1st, 2nd, 3rd, and 5th minutes after finalizing the test from the patient's maximum heart rate during exercise. However, the authors have described HRRI in the "Introduction" section as being calculated by extracting the maximum heart rate from the heart rate in the 1st, 2nd, 3rd, and 5th minutes in the post-exercise period. In case of this type calculation, the study results will change, and it will forward us wrongly. I wonder if it was miswritten or miscalculated in this article. I wanted to emphasize on the importance of right usage of medical formulas.

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Author`s Reply

To the Editor,

We thank the author for the great interest in our study entitled "Evaluation of heart rate recovery index in heavy smokers"

published in Anatol J Cardiol 2016; 16: 667-72. (1).

The prognostic value of the slow heart rate recovery index (HRRI) after exercise in predicting cardiovascular disease and mortality has been established (2). Our study determined that the 1st-, 2nd-, 3rd-, and 5th- minute HRRIs after maximum stress testing were statistically significantly lower in the heavy smoker group than in the nonsmoker healthy control group.

HRRI is calculated by extracting the heart rate during the 1st, 2nd, 3rd, and 5th minutes after finalizing the test from the patient's maximum heart rate during exercise. In our study, we used this formula and mentioned it in the "Method" section. However, in the "Introduction" section, the definition was incorrect. Therefore, we thank the author for bringing this to our attention. In summary, HRRI was calculated correctly in our study.

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Analysis of platelet-to-lymphocyte ratio requires methodological consideration

To the Editor,

I read the article by Kundi et al. (1) entitled "Relationship between platelet-to-lymphocyte ratio and the presence and severity of coronary artery ectasia" published in Anatolian J Cardiol 2016;16: 857-62. The authors aimed to investigate the relationship between the platelet-to-lymphocyte ratio (PLR) and coronary artery ectasia in the adult population. They found that PLR values in patients with isolated coronary artery ectasia were significantly higher than those in patients with obstructive coronary artery disease and the control group with normal coronary artery angiograms. I have a few comments:

PLR is calculated as the ratio of the platelet to lymphocyte count from the same complete blood count, which is a widely available, automated, inexpensive, and easy-to-do test, and it can be used as a marker of systemic inflammation in coronary artery disease and cardiovascular events (2). However, the standardized laboratory methods are crucial with regard to PLR analysis. Kundi et al. (1) did not mention from where blood samples were obtained, what kind of sample tubes were used, or when blood samples were analyzed after venipuncture in each patient. First, the platelet count obtained from citrate-anticoagulated blood samples has been reported to be higher than that obtained from EDTA-anticoagulated blood samples (3). Second, EDTA-induced pseudothrombocytopenia due to platelet agglutination because of EDTA-induced alteration of surface glycoproteins and anionic phospholipids is an important issue when using EDTA-anticoagulated samples (4). EDTA-induced pseudothrombocytopenia should be checked by a peripheral blood smear. Because of the factors I have mentioned above, it may be deceptive to make an interpretation based on results of the study by Kundi et al. (1) regarding the relationship between PLR and coronary artery ectasia.

In addition to PLR, the mean platelet volume (MPV) or platelet distribution width (PDW) can be also used as a marker of inflammation, which is obtained from the same blood sample (5). Thus, one can speculate about a relationship among MPV, PDW, and PLR in patients with coronary artery ectasia. Analysis of MPV and PDW also requires methodological consideration, as I have stated previously.

In conclusion, I think that it will be more helpful to design a prospective study considering the methodological details mentioned above to determine the relationship between PLR and coronary artery ectasia.

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