as ≤21 beats during the first minute of recovery in a sitting position and found that abnormal HRR is sensitive with regard to the diagnosis of coronary artery disease (CAD) (76.1%) but does not exhibit good specificity (41.3%). We suggest that the presence of abnormal HRR (≤21 beats) in treadmill exercise testing should be considered an additional diagnostic criterion for the presence of CAD, and therefore, we agree that HRR should be incorporated into the interpretation of treadmill exercise testing (TET), in addition to other significant parameters, such as ST-segment depression, typical chest pain, or hypotensive response.

Normal parasympathetic reactivation is needed for the rapid decrease in heart rate following the cessation of exercise. Therefore, slow HRR after exercise has prognostic value for predicting cardiovascular mortality, regardless of the extent of coronary disease (2). However, several risk factors for atherosclerosis, especially metabolic syndrome components (3), advancing age (4), and chronic obstructive pulmonary disease (5), are important factors of decreased HRR. Because the risk factors mentioned above are also strongly associated with CAD, the calculation of HRR, as well as traditional markers of ischemic response during TET, could provide additional diagnostic information about the presence of CAD.

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Assessment of serum hepcidin levels in patients with non-ST elevation myocardial infarction

To the Editor,

We read the article, entitled "Assessment of serum hepcidin levels in patients with non-ST elevation myocardial infarction," by Altun et al. (1) published in Anatolian J Cardiol 2014; 14: 515-8. Serum hepcidin levels were comparable among NSTEMI patients and control subjects. Also, its concentration did not change 6 hours after admission. The authors concluded that hepcidin could not be used as a marker of myocardial necrosis in NSTEMI patients. We thank the authors for drawing attention to a very important and challenging field of cardiology: markers in acute coronary syndromes. However, in their study, we think that there are some important questions that need to be answered.

The peptide hormone hepcidin is the main conductor of systemic iron hemostasis (2). The expression of the hepcidin gene has been shown to be regulated by hypoxia and inflammation (3). According to this finding, Suzuki et al. (4) argued that the human heart might also react to ischemia, and they measured serum hepcidin levels in patients with acute myocardial infarction. They found an elevated serum hepcidin level within 4 hours after the heart attack and showed that hepcidin levels decreased to normal levels in 7 to 14 days. In the present study of Altun et al. (1) the time interval between the onset of the symptoms and blood sampling was not mentioned. Additionally, the authors retested serum samples of the NSTEMI patients only 6 hours later. However, hepcidin levels are detectable after several days following myocardial injury (4). The racial and genetic differences between the study population of Suzuki et al. (4) and Altun et al. (1) can explain the negative result of the latter study. The authors did not mention anything regarding coronary artery lesions of the study population; control subjects were aged between 50 and 70 years, and they can also have coronary atherosclerosis. Hence, it is an important limitation of this study if hepcidin might reflect destabilization of the coronary plaques, as expected from an inflammatory biomarker. The authors provided that CRP levels were increased in NSTEMI patients. In this point, performing a correlation analysis between CRP and hepcidin levels is very essential. In the case of showing this relationship, it could be argued that hepcidin might be a surrogate marker of inflammation, although plasma kinetics were not identified properly. Moreover, since this biopeptide is not a structural element of the myocardial cell like cardiac troponin I, it naturally might not be elevated at the same time. Finally, serum levels of hepcidin in patients and in control subjects were unevenly distributed: 24.55±32.13 and 23.67±33.62 ng/mL. It can be concluded that there are many extreme cases in the laboratory results, which can affect all analyses and interpretations in this small-sized study.

Therefore, we think that although the study conducted by Altun et al. (1) draws attention to a very important and interesting subject, there are several points in the study design and data evaluation that need to be discussed, and the study results should be interpreted with caution.

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Author`s Reply

To the Editor,

We thank you for your comments on our study published in the September 2014 issue of The Anatolian Journal of Cardiology entitled "Assessment of serum hepcidin levels in patients with non-ST elevation myocardial infarction (NSTEMI)." (1). They have raised some questions. Hepcidin is produced mainly in the liver and increases in response to inflammation, and its expression is regulated by anemia, hypoxia, and inflammation (2). In this single-center study, we evaluated whether the level of hepcidin increased in the acute phase in NSTEMI, known as acute inflammatory aggravation of a chronic atherosclerotic process.

There are conflicting results for hepcidin in coronary artery disease patients (3, 4). The first remark was about blood sampling time and symptom onset. We did not investigate hepcidin kinetics in this study; our aim was fundamentally to use hepcidin as a new cardiac marker instead of troponin. Another remark was about the time interval between the onset of the symptoms and blood sampling. According to our study design, we aimed to compare hepcidin levels with troponin levels in the diagnosis of NSTEMI. It is important that the hepcidin levels did not increase; meanwhile, the levels of troponin were increased in NSTEMI patients in the acute phase. The observed differences in these parameters, performed simultaneously from the same patients, forced us to think that there was no need to take the time interval between the onset of symptoms and blood sampling. The other remark was about the study of Suzuki et al. (4). The patient population and the design of the two studies were different, as the authors (4) studied ST elevation myocardial infarction patients, but we did not. Also, the sample of their study was extremely low, and their aim was also different. As stated in the criticism, if we performed a correlation analysis between CRP and hepcidin levels, it should have corroborated our results, showing hepcidin as a surrogate marker of inflammation.

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Transcatheter closure of antegrade pulmonary blood flow with Amplatzer septal occluder after Fontan operation

To the Editor,

We read the article of Karagöz et al. (1), entitled "Transcatheter closure of antegrade pulmonary blood flow with Amplatzer muscular VSD occluder after Fontan operation," published in The Anatolian Journal of Cardiology 2014; 14: 565, with great interest. Recently, in our clinic, we closed residual antegrade pulmonary blood flow with an Amplatzer septal occluder device after Fontan operation.

Our patient's initial diagnosis was unbalanced complete atrioventricular septal defect and double outlet right ventricle with D-transposed great arteries. His first surgery was a pulmonary banding operation when he was 2.5 months old. When he was 6.5 years old, a bi-directional Glenn operation was performed (with antegrade flow). He underwent an extracardiac Fontan operation at the age of 11 years in our clinic. During his hospital stay, 10 days after the Fontan procedure, massive pleural effusion, edema, and ascites were detected. Echocardiography revealed significant antegrade flow to the pulmonary artery. The patient underwent cardiac catheterization to close the antegrade flow. Mean pulmonary artery pressure was 33 mm Hg. The right ventriculogram and main pulmonary artery angiogram showed normally branched pulmonary arteries, with a narrowing in the main pulmonary artery owing to his first operation-pulmonary banding. The narrow part of the pulmonary artery was 9 mm, and the proximal and distal sides of this narrow part were 24.3 mm and 21.5 mm, respectively. An 11-mm Amplatzer septal occluder (AGA Medical, MN, USA) device was deployed at the narrow region. After deployment of the device, the mean pulmonary artery pressure decreased to 26 mm Hg, which was also high but at least lower than the pre-intervention pressure.

Residual forward flow from the ventricle to the pulmonary artery, via either a native pulmonary outflow tract or a previously banded or ligated main pulmonary artery, leads to ineffective even hazardous pulmonary blood flow and unnecessary ventricular volume overload in Fontan patients. This in turn can lead to persistent pleural effusions or ventricular failure, especially in patients with transposed great arteries, in whom surgical dissection of the main pulmonary artery during the Fontan procedure would be difficult or hazardous. At least 5 of 8 patients from the Desai et al. (2) series had transposed great arteries. Similarly, our case had transposed great arteries. It may be difficult to locate and close pulmonary antegrade flow due to the anatomy of the