Author’s Reply

To the Editor,

We recently demonstrated decreased heart rate variability (HRV) values in patients with irritable bowel disease (IBS) in our study entitled “The effect of irritable bowel syndrome on carotid intima-media thickness, pulse wave velocity, and heart rate variability” published in the September issue of The Anatolian Journal of Cardiology 2014; 14: 525-30 (1). We read the letter entitled “Heart rate variability can be affected by gender, blood pressure, and insulin resistance” with great interest. As the authors kindly mentioned, HRV is a valuable tool for assessing autonomic dysfunction. Decreased HRV is associated with coronary artery disease, myocardial infarction, and cardiovascular mortality in patients with diabetes (2). Interestingly, insulin resistance and obesity, the prerequisites of diabetes mellitus, are also related to autonomic dysfunction (3). Our study included 30 women with IBS and 30 healthy control subjects. Although numeric differences existed in the prevalence of hypertension and diabetes mellitus compared with the control subjects, these were not statistically significant. Moreover, body mass index, fasting plasma glucose, and blood pressure values were not different between groups. Therefore, we do not believe that an important difference is present, which would have influenced our results with, regard to insulin resistance and obesity between the control and patient groups.

References

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Author’s Reply

To the Editor,

The authors state an important bias about studies comparing different graft types in different regions entitled “Long-term patency of autogenous saphenous veins vs. PTFE interposition graft for prosthetic hemodialysis access.” published in Anatol J Cardiol 2014; 14: 542-6. (1). As mentioned in the study, the selection of the anastomosis region was based on the calibration of the arteries and veins (1). Both PTFE and saphenous vein grafts were
Carotid blood flow and the coverage of the coronary ostia by the opening aortic valve leaflets during cardiac systole. Elevation of cardiac troponin levels has also been reported in atrial myxomas, all of which were secondary to the coronary artery embolization (4, 5).

Interestingly, however, we examined 10 patients (age: 49±13 years; six females) with atrial myxoma and normal coronary arteries by angiography and normal ECG but with elevation of cardiac enzymes. Cardiac troponin and CK-MB levels were measured on admission; these markers were elevated in six patients (four females; normal value of cardiac troponin: I=0.4 ng/mL; increased values in our six patients: 0.70, 1.10, 2.35, 0.86, 1.67, and 1.45 ng/mL, respectively), all of whom had normal coronary arteries, based on angiography findings and normal ECG findings, and had no accompanying chest pain. Patients were further investigated for exclusion of other reasons for elevated cardiac troponin levels, including renal failure, sepsis, pulmonary emboli, tachy, or bradyarrrhythmias. These findings suggest that atrial myxoma increases cardiac markers without involvement of coronary arteries. Actually, we think such constitutional symptoms (fever, weight loss, or symptoms resembling connective tissue disease) are due to cytokine (interleukin-6) secretion; cardiac markers could be secreted in cardiac myxomas as well. Moreover, cardiac myxomas could be considered as the differential diagnosis for the diseases with elevated cardiac enzymes. However, further studies are required to reveal this association.

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References