response to standing up, and 5) blood pressure response to a sustained handgrip.

There are other approaches for evaluating the autonomic nervous system by heart rate variability (HRV) parameters from short- or long-term monitoring (2). We agree that HRV and bedside autonomic function tests for evaluating the autonomic nervous system provide complementary information regarding autonomic regulatory mechanisms in health and disease. However, the bedside autonomic function tests were more feasible for us during the study.

We also considered that spectrum bias may account for differences in the reported results between the investigations.

Therefore, in order to point out the difference between the cardiovascular autonomic function of RA patients and general population, we will design another prospective cohort study with complementary and more sensitive tests.

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Restless leg syndrome and slow coronary flow. Is it inflammation or autonomic nervous system?

To the Editor,

Erden et al. (1) recently published a very interesting paper in the Anatolian Journal of Cardiology 2014; 14: 612-6 entitled "Association between restless leg syndrome and slow coronary flow," which suggests an association between the coronary slow flow (CSF) phenomenon and restless leg syndrome. The article showed that patients with the CSF phenomenon were more likely to suffer from restless leg syndrome compared to subjects with normal coronary flow. Although, I appreciate the authors for their work, there are some issues that need to be clarified in order to glean more data from the article. The definition of CSF relies upon TIMI frame count (TFC), which varies depending on the image acquisition rate. The authors chose a recording speed of 25 frames/s. Still, they defined CSF according to the criteria based on the reference values of Gibson et al. (2). "a TFC greater than two standard deviations from the normal range for a particular coronary artery." Gibson used a frame rate of 30/s. Thus, the authors could have underestimated TFC. I believe that they need to multiply their corrected TFC with a factor of 1.2 in order to find the real corrected TFC, which may render some of their normal patients into a group of CSF (2, 3). It would be appropriate to know the mean cTFC values of patients with the CSF phenomenon and those of the normal patients in this regard. Even though the authors reported the overall prevalence of restless leg syndrome, it would be useful to know how many patients with and without the CSF phenomenon had restless leg syndrome. In our current practice, we do not come across patients having both the CSF phenomenon and restless leg syndrome, thus, they may have mild symptoms. Was there any association with symptom severity and TFC? We previously showed that patients with the CSF phenomenon had attenuated heart rate recovery, suggesting impaired vagal activation of the cardiovascular system (4). Therefore, we agree with the authors that the common link between the CSF phenomenon and restless leg syndrome is the probably autonomic nervous system.

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Author`s Reply

To the Editor,

We thank to Tekin for her interest in our investigation entitled "Association between restless leg syndrome and slow coronary flow" published in Anatol J Cardiol 2014; 14: 612-6 (1).

Some standard recommendations are made for the quantitative analysis of epicardial blood flow. Pérez de Prado et al. (2) reported that imaging speed should ideally be 25 frames/s. Nevertheless, the corrected TIMI frame count (cTFC) can be calculated at any recording speed, and subsequently it can be expressed in seconds or adjusted to the recommended speed. The images obtained by cineangiography in

the study by Gibson et al. (3) were recorded at the rate of 30 frames/s. When we evaluated our findings according to the rate of 30 frames/s, there was no statistically significant change in the association between restless legs syndrome (RLS) and coronary slow flow (CSF).

Ohayon et al. (4) reported RLS prevalence in the general adult population.

1) A symptom only: ranged from 9.4% to 15%,
2) A set of symptoms meeting the minimal diagnostic criteria of the international RLS study group: ranged from 3.9% to 14.3%,
3) Meeting minimal criteria accompanied with a specific frequency and/or severity: ranged from 2.2% to 7.9%,
4) A differential diagnosis: ranged from 1.9% and 4.6%.

In our study, 33 subjects (38%) had RLS with the CSF phenomenon, and 15 (17%) had RLS without the CSF phenomenon (1). The prevalence of RLS in our control group was slightly higher than the prevalence of Ohayon's (4) study. Previously, we found that (5) the prevalence of RLS in hypertensive patients was more than twice as frequent as that in normotensive individuals (35.3 vs. 17.2%, respectively, $p<0.01$).

Additionally, there were significant but weak correlations between mean TFC ($r=0.268$, $p<0.001$), LAD TFC ($r=0.322$, $p<0.001$), and RCA TFC ($r=0.117$, $p=0.02$) and severity of RLS. There was no significant correlation between Cx TFC and severity of RLS.

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Ultrasound-assisted catheter-directed thrombolysis for pulmonary embolism

To the Editor,

We have read through the case report article with great interest, entitled "Combined catheter thrombus fragmentation and percutaneous thrombectomy in a patient with massive pulmonary emboli and acute cerebral infarct," by Uğurlu et al. (1) and published in Anatol J Cardiol 2015; 15: 69-74. For the last two years, ultrasound-assisted catheter-directed thrombolytic (USAT) has been used as an alternative method for treatment in selected cases (2). We believe that massive pulmonary embolism can be a life-saving treatment option in experienced centers of the percutaneous intervention. However, we have some concerns about employing it in "intermediate-high" group patients. In this article, we would like to present a successful USAT on a patient to whom a prior thrombolytic treatment had been applied. However, this initial thrombolytic treatment had ended with failure and a bleeding complication had developed.

A 75-year-old female patient with hemiplegia showed thrombus in bilateral main pulmonary arteries in CT pulmonary angiogram (CTPA) performed at another center, and developed respiratory and cardiac failures. The patient was given thrombolytic treatment; however, her hypoxemia got deeper in spite of anticoagulant treatment. The patient whose thrombolytic treatment was in the "intermediate-high" category with respect to mortality risk, pulmonary embolism severity index was 175, and Wells bleeding risk score was 4, was admitted to the intensive care treatment. Since the probability of mortality was determined as 10–25% within the first 30 days, systemic thrombolytic treatment failed, and since the bleeding risk remained high, USAT was planned. Angiography for USAT was performed under local anesthesia during invasive mechanical ventilator support. Mean pulmonary artery pressure was found to be 53 mm Hg. 5 mg tPA bolus was administered through each catheter to maintain the patency of catheters and receive an immediate response. Following a total 10 mg push, a continuous tPA infusion was initiated as 1 mg/h dose for the first 5 hours, and 0.5 mg/h dose for the following 10 hours time. In addition to tPA, the patient was administered systemic unfractionated heparin. Echocardiographic evaluation on the fifth day of treatment revealed that pulmonary artery pressure and right ventricular functions were back to normal. CTPA showed almost complete resolution of thrombi within the pulmonary arteries.

According to Uğurlu et al. (1), percutaneous intervention is a life-saving treatment option in massive PE treatment. USAT treatment was found to be especially effective at the right ventricular dilatation without causing any hemorrhage, compared with unfractionated heparin infusion in patients diagnosed with intermediate-risk PE (3). In conclusion, our case indicates that USAT is a safely usable option for treating massive and sub-massive PE's with high-risk of bleeding and is unresponsive to systemic thrombolytic treatments.

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