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Author`s Reply

To the Editor,

We would like to thank the authors of the letter for their interest and criticism on our study entitled "Heart rate variability improvement in children using transcatheter atrial septal defect closure "published in Anatol J Cardiol 2015 Mar 4 (1).

Heart rate variability is a parameter used for the non-invasive evaluation of the neurohumoral control of the heart. One study reported reduced measurements of HRV in children with various congenital heart diseases (2). In another study, it has been shown that the dilatation of RV can decrease for up to 5 years after ASD closure (3). Some studies have published the normalization of RV size during the first 24 months after device closure (4). There may be other factors that affect the cardiac autonomic function besides atrial septal defect as the author mentions. However, Cansel et al. (4) found that the right ventricular diameter and pulmonary artery systolic pressure significantly decreased 6 months after transcatheter closure compared with values measured before transcatheter closure in patients with ASD. In our study, we concluded that HRV in children recovers approximately 6 months after transcatheter ASD closure. We did not report the dimensions of cardiac chambers before and after transcatheter closure. In our article, HRV after transcatheter ASD closure was compared with that of the control group. We did not declare that heart chambers reached normal values in 6 months. In our study, the 6th month HRV of patients who underwent transcatheter ASD closure approached the levels of the control group (1). HRV and reaching normal levels of right ventricular measurements are two different things. HRV could return to the normal range before the normalization of heart cavity due to hemodynamic improvement after transcatheter closure.

Our study was designed using the heart rate variability data of Holter ECG in the previously published "'Holter Electrocardiographic Findings and P-wave Dispersion in Pediatric Patients with Transcatheter Closure of Atrial Septal Defects"' study. A previously published part of this study was not used the heart rate variability data (5). Patient information [mean±SD, pulmonary artery pressure (mm Hg), Qp/Qs ratio, stretched diameter of ASD (mm), device defect ratio, device diameter (mm): 20.8±4.4, 2.1±0.4, 16.8±3.8, 1.3±1.4, 19±4.2, respectively] were not written again because they were declared in this previously published study (1). İsa Özyılmaz, Sinem Özyılmaz*, Yakup Ergül, Alper Güzeltaş Departments of Pediatric Cardiology, *Cardiology, Mehmet Akif Ersoy Cardiovascular and Thoracic Surgery Center, Training and Research Hospital; İstanbul-*Turkey*

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Potential benefits of oral pentoxifylline before coronary artery bypass surgery

To the Editor,

We read with interest the recent publication by Mansourian et al. (1) published in Anatol J Cardiol 2014 Dec 31 entitled "Preoperative oral pentoxifylline in case of coronary artery bypass grafting with left ventricular dysfunction (ejection fraction equal to/less than 30%)" on effects of preoperative oral pentoxifylline in a cohort of high-risk patients undergoing coronary artery bypass surgery. They reported a shorter ventilation time and intensive care unit stay, less frequent need for blood product transfusion along with a significantly lower TNF-alpha and insignificantly lower interleukin (IL)-6 levels postoperatively in patients who received oral pentoxifylline. An increase in the level of inflammatory cytokines has been shown after cardiac surgery (2). It has been reported in both offpump and on-pump CABG (3). Some studies reported a diminished activation of the inflammatory system after off-pump procedures, but surprisingly, this has not been reported to have a clinically relevant benefit (2). Pentoxifylline is a xanthine derivative, and its main mechanism is decreasing blood viscosity. This drug has been shown to inhibit inflammatory cytokine release in both oral and intravenous forms (4).

The authors stated that they excluded patients with recent myocardial infarction, but the preoperative troponin-T levels are well above the normal range. The reason for the increased cardiac biomarkers is not

clear. The levels of both TNF-alpha and IL-6 at the baseline are higher than the levels previously reported, which could be partially explained by the fact that the study is performed in a subgroup of high-risk patients with remarkable left ventricular dysfunction; however, the mean levels of the baseline TNF-alpha are approximately 10-fold higher than the baseline values of previous reports (2, 3). Also, the levels of both TNF-alpha and IL-6 show a decrease, though insignificant, following cardiopulmonary bypass, which has never been shown in previous studies that measured these levels immediately after surgery and later (2, 4). The explanation for this rather unexpected finding is not provided. The level of inflammatory cytokines is expected to rise when measured immediately after surgery and in subsequent time intervals, and the rise is expected to be lower in patients receiving pentoxifylline. In conclusion, though the paper aims to address the potential benefits of oral pentoxifylline in a high-risk subgroup of patients undergoing CABG, some clarifications needs to be made before drawing a conclusion.

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Author's Reply

To the Editor,

We appreciate you and the author(s) who accurately read and criticized our article entitled "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 gold standard of clinical research is a randomized controlled trial (2). We considered all rules and laws of allocation sequences in randomized trials. The levels of cytokines have been evaluated by laboratory-trained personnel using the same type of laboratory kit.

As you noticed, the study sample, a group of high-risk patients, may be one of the reasons for the higher level of cytokines before surgery. Also, we mentioned in our article that some novel results have been obtained that should be confirmed in further studies (1).

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Atrial fibrillation after cardiac surgery

To the Editor,

Postoperative atrial fibrillation (POAF) is mostly seen rhythm disturbance after coronary bypass surgery. POAF prolongs hospital care and increases hospital cost. It is a good indicator of a patient's morbidity and mortality. Studies aiming to investigate the pathogenesis of POAF show that inflammatory reactions and oxidative stress are the most important factors for the development of POAF. Inflammation changes the atrial transmission pathway, activates reentry mechanisms, and precipitates the development of POAF (1, 2). We read with great interest the article by Aydın et al. (3) entitled "Efficiency of postoperative statin treatment for preventing new-onset postoperative atrial fibrillation in patients undergoing isolated coronary artery bypass grafting: A prospective randomized study" published in Anatol J Cardiol 2015; 15: 491-5. The authors concluded that postoperative statin therapy seemed to reduce AF development after coronary bypass surgery. They also stated that CRP levels significantly decreased in patients undergoing coronary bypass surgery with early postoperative statin therapy.

In the results of this study, CRP levels showed no significant differences between the statin and non-statin groups on postoperative days 1 and 7. However, there was significant difference between the AF and non-AF groups. On postoperative day 14, the CRP levels showed significant differences between the statin and non-statin groups. There was also a significant difference between the AF and non-AF groups (Table 4). Due to high inflammation during the intraoperative period (extracorporeal circulation, cardiac ischemia-reperfusion injury, and oxidative stress) and postoperative period (pulmonary infections and cardiac deficiency), the inflammatory activity and CRP levels reach its