long-term mortality in ST-elevation myocardial infarction" published in Anatol J Cardiol 2015 Feb 11. (1). In the study, we conducted ST elevation myocardial infarction (STEMI) in patients undergoing primary percutaneous coronary intervention (PCI). An admission serum potassium (sK) level of >4.5 mmol/L was found to be associated with short- and long-term mortality (1).

Firstly, all patients in the study were treated with the dual antiplatelet therapy (clopidogrel 75 mg/day and acetylsalicylic acid 100 mg/day) for at least one year (1). Even though ticagrelor and prasugrel are associated with better results in patients with STEMI, during the period the study was conducted, neither prasugrel nor ticagrelor was administered in our center (2, 3). The effect of ticagrelor or prasugrel was not evaluated in our study.

The effect of aldosterone antagonists was not evaluated. The global left ventricular ejection fraction did not significantly differ between groups. Even though we did not evaluate the effect of aldosterone antagonists, in our opinion, this could not affect the outcome between the groups. However, cumulative end points will probably be affected. The effect of aldosterone antagonists could be a part of another study.

Thirdly, all patients with STEMI underwent primary PCI in our center regardless of the admission creatinine level. The patients' blood at the time of admission was drawn at the emergency department without procedure delay. No significant correlation was found between admission sK level and door-to-balloon time (p=0.19).

In conclusion, despite the presence of many confounding factors, we thought that an sK level of >4.5 mmol/L is associated with short- and long-term mortality. The effect of aldosterone antagonists, prasugrel, and ticagrelor could be evaluated in different studies.

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References

1. Uluganyan M, Ekmekçi A, Murat A, Avşar Ş, Ulutaş TK, Uyarel H, et al. Admission serum potassium level is associated with in-hospital and long-term mortality in ST-elevation myocardial infarction. Anatol J Cardiol 2015 Feb 11. Epub ahead of print.
2. Steg PG, James S, Harrington RA, Ardissino D, Becker RC, Cannon CP, et al.; PLATO Study Group. Ticagrelor versus clopidogrel in patients with ST-elevation acute coronary syndromes intended for reperfusion with primary percutaneous coronary intervention: A Platelet Inhibition and Patient Outcomes (PLATO) trial subgroup analysis. Circulation 2010; 122: 2131-41. [CrossRef]
3. Montalescot G, Wiviott SD, Braunwald E, Murphy SA, Gibson CM, McCabe CH, et al.; TRITON-TIMI 38 investigators. Prasugrel compared with clopidogrel in patients undergoing percutaneous coronary intervention for ST-elevation myocardial infarction (TRITON-TIMI 38): double-blind, randomised controlled trial. Lancet 2009; 373: 723-31. [CrossRef]

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Mean platelet volume: When the size does matter

To the Editor,

Cardiovascular diseases are known to be associated with unstable atherosclerotic plaques matching with platelet reactions, which lead to thrombus formation and finally clinical events (1). Platelets are heterogeneous blood components, differing in size, density, and reactivity. It is recognized that several substances released from alpha-granules, dense granules, lysosomes, or the cytosol in larger platelets are either vasoactive and prothrombotic (thromboxane A2, coagulation factors), adhesion proteins (P-selectin), growth factors (TGF-beta), chemokines (platelet factor 4), or cytokine-like factors (CD40 ligand). These proteins act in a collaborative way to determine biological functions. In other words, activated platelets are larger, and the mean platelet volume (MPV), a measure of platelet size, could be an accurate and easily available marker of platelet activation. Several studies have reported an increasing MPV associated with the prognosis of either acute coronary syndromes (2) or cerebrovascular diseases (3). However, until this issue in which Kalkan et al. (4) entitled "Mean platelet volume is associated with aortic intima-media thickness in patients without clinical manifestation of atherosclerotic cardiovascular disease." published in Anatol J Cardiol 2015; 15: 753-8 report an association between MPV and the extent of subclinical thoracic aortic atherosclerosis in patients without a clinical manifestation of atherosclerotic cardiovascular disease, we did not know the role of this potential marker in the general population without cardiovascular events, namely, in people whose prothrombotic status is unknown or supposedly inactivated. The authors showed how the extent of thoracic aorta intima to media thickness, as a marker of diffuse atherosclerotic disease, is significantly related to an increasing MPV, supporting the role of systemic thrombocyte activation over the course of atherosclerosis, a relationship that has been previously reported, though in a different scenario such as coronary or carotid arteries (5) and again, in patients with atherosclerotic disease present.

The results reported by Kalkan et al. (4) are interesting, though some questions remain to be answered. MPV is uncomplicated and cheap to obtain, easy to elucidate, and is conventionally measured by automated cell counters. Its increase should suggest a careful assessment of cardiovascular risk; however, more studies are necessary in the general population to confirm the findings by Kalkan et al. (4), and new studies investigating the relationship of MPV with future cardiovascular events in healthy people beyond the wall of their arteries.

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References

1. Fitzgerald DJ, Roy L, Catella F, Fitzgerald A. Platelet activation in unstable coronary disease. N Engl J Med 1986; 315: 983-9. [CrossRef]
2. Kılıçlı-Çamur N, Demirtunç R, Konuralp C, Eskiser A, Başaran Y. Could mean platelet volume be a predictive marker for acute myocardial infarction? Med Sci Monit 2005; 11: CR387-92.
3. O'Malley T, Langhorne P, Elton RA, Stewart C. Platelet size in stroke patients. Stroke 1995; 26: 995-9. [CrossRef]
4. Kalkan GY, Gür M, Baykan AO, Uçar H, Elbasan Z, Şahin DY, et al. Mean platelet volume is associated with aortic intima-media thickness in patients without clinical manifestation of atherosclerotic cardiovascular disease. Anatol J Cardiol 2015; 15: 753-8. [CrossRef]
5. Arévalo-Lorido JC, Carretero-Gómez J, Villar-Vaca P. Mean platelet volume predicting carotid atherosclerosis in atherothrombotic ischemic stroke. Ir J Med Sci 2012; 181: 179-83. [CrossRef]

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DOI:10.5152/AnatolJCardiol.2015.6502

Author`s Reply

To the Editor,

There are large clinical data on the importance of mean platelet volume (MPV) in unstable patients; its importance in stable atherosclerotic disease is scarce. We reported that MPV is independently associated with subclinical thoracic atherosclerosis in the article entitled "Mean platelet volume is associated with aortic intima-media thickness in patients without clinical manifestation of atherosclerotic cardiovascular disease" published in Anatol J Cardiol 2015; 15: 753-8.

One of the main disturbances that play a role in atherosclerosis is increased platelet aggregation, and increased platelet volume is a marker of increased platelet activity (2). Recently, one meta-analysis showed that a larger MPV is associated with coronary artery disease (3). According to our results, we confirm that an increase in MPV may be an important biochemical marker for initial atherosclerosis.

Previous studies demonstrated that platelets play a critical role in carotid atherosclerosis and that P-selectin that is stored in platelet secretory granules is important for the development of atherosclerosis. Additionally, platelets directly affect the degree of plaque maturation, including the existence of smooth muscle cells and calcification (4). These findings comprise the rationale to our hypothesis.

As far as we know, our article is the first to report a relationship between thoracic aorta intima media thickness and the mean platelet volume in healthy subjects. Therefore, more studies are needed to confirm this finding. Our study is not a prospective clinical study, so we do not know whether the mean platelet volume is a predictor of future cardiovascular events in healthy subjects or not. Prospective clinical trials must be conducted to investigate the prognostic importance of the mean platelet volume.

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References

1. Yüksel Kalkan G, Gür M, Baykan AO, Uçar H, Elbasan Z, Şahin DY, et al. Mean platelet volume is associated with aortic intima-media thickness in patients without clinical manifestation of atherosclerotic cardiovascular disease. Anatol J Cardiol 2015; 15: 753-8. [CrossRef]
2. Haubelt H, Simon M, Anders CH, Hellstern P. Platelet function tests for monitoring of acetylsalicylic acid: clinical significance in antiplatelet treatment. Hamostaseologie 2004; 24: 196-202.
3. Sansanayudh N, Anothaisintawee T, Muntham D, McEvoy M, Attia J, Thakkinstian A. Mean platelet volume and coronary artery disease: a systematic review and meta-analysis. Int J Cardiol 2014; 175: 433-40. [CrossRef]
4. Burger PC, Wagner DD. Platelet P-selectin facilitates atherosclerotic lesion development. Blood 2003; 101: 2661-6. [CrossRef]

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Is atrial septal defect alone able to affect the cardiac autonomic function or are there different factors that influence this function?

To the Editor,

We read with a great interest the paper by Özyılmaz et al. (1) entitled "Heart rate variability improvement in children using transcatheter atrial septal defect closure" published in the Anatol J Cardiol 2015 Mar 4. The authors aimed to evaluate cardiac autonomic functions in children who underwent transcatheter closure of atrial septal defect (ASD) using analysis of heart rate variability (HRV) parameters. They concluded recovery of HRV indices approximately 6 months after transcatheter ASD closure.

ASD is a frequently seen congenital heart disease characterized with left-to-right shunting and dilation of the right cardiac chambers and pulmonary artery, which might result in heart failure, arrhythmia, and thromboembolic events as well as increased mortality. The enlarged right ventricle usually returns to normal size during the first 24 months after transcatheter device closure or surgical repair, although this normalization may persist for up to 5 years after defect closure (2). As mentioned in the article by Özyılmaz et al. (1), HRV impairment in patients with ASD has been attributed to right ventricular filling and right atrial tension due to left-to-right blood flow through ASD (3). However, in the study by Özyılmaz et al. (1), no data demonstrating dimensions of cardiac chambers before and after the transcatheter closure are available, and we do not know whether the initial dimensions are significantly different from those measured 6 months after transcatheter closure. In addition, the mean diameter of ASD as well as the range of the diameter of the defect in the study population is not mentioned in the article. With these additional data, we believe that readers of the journal can more easily understand whether the size of the defect and the dimensions of cardiac chambers have an effect on HRV parameters.

The interpretation of HRV analysis is not as simple as thought because of various factors that influence HRV indices, which might be affected by many variables such as hyperlipidemia and blood pressure (3-5). We think that it would be more helpful to demonstrate blood pressure levels and blood lipid profiles of the study population in terms of showing no variable affecting HRV parameters rather than ASD. Thus, one can understand whether ASD alone really impairs the cardiac autonomic function, which has a prognostic importance for survival (5).

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References

1. Özyılmaz I, Ergül Y, Tola HT, Saygı M, Öztürk E, Tanıdır IC, et al. Heart rate variability improvement in children using transcatheter atrial septal defect closure. Anatol J Cardiol 2015 Mar 4. Epub ahead of print.
2. Cansel M, Yağmur J, Ermiş N, Açıkgöz N, Taşolar H, Ataş H, et al. Effects of transcatheter closure of atrial septal defects on heart rate variability. J Int Med Res 2011; 39: 654-61. [CrossRef]