

nism of AF is multiple, and the damage of the atrial tissue, presence of inflammation, or decrease in the mechanical function of atria can influence the homogeneity of atrial conduction without atrial enlargement. However, using this echocardiographic technique to measure atrial EMD may be doubtful for patients with normal size of atria because atrial dilatation is the major cause for the increase in ACT. The gold standard measure for atrial EMD is direct measurement with electrophysiological study (EPS). If it is possible to demonstrate the increase of EMD in different conditions with EPS findings, it may be helpful in clarifying the issue.

Burcu Demirkan, Yeşim Güray, Esra Gücük İpek¹
Clinic of Cardiology, Türkiye Yüksek İhtisas Hospital, Ankara-Turkey
¹**Department of Cardiology, Johns Hopkins University School of Medicine, Baltimore-USA**

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Address for Correspondence: Dr. Burcu Demirkan
 Türkiye Yüksek İhtisas Hastanesi
 Kardiyoloji Kliniği, Samanpazarı, Ankara-Türkiye
 E-mail: burcume@gmail.com

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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 about our study entitled "Assessment of atrial conduction times in patients with mild diastolic dysfunction and normal atrial size" published in November issue of The Anatolian Journal of Cardiology 2015; 15: 925-31 (1).

I conducted my study on the basis of the hypothesis that electrical remodeling can occur before structural remodeling in diastolic left ventricular dysfunction (2), and I adopted the evaluation of atrial conduction times as a marker for atrial electrical remodeling (3).

Since then, I have received comments from my dear colleagues. According to these comments, an increase in atrial electromechanical delays (EMDs) occurs when left atrial enlargement reaches a certain level. There is some evidence supporting this hypothesis. Tsang et al. (4) demonstrated that when left atrial size reaches $>27 \text{ mL/m}^2$, the probability for the first episode of atrial fibrillation increases in the presence of left ventricular diastolic dysfunction. However, the question remains as to what is the critical point in left atrial size. To my knowledge, it has yet to be defined through new research. On the other hand, several pathological processes such as structural and electrical remodeling with multiple etiologies underlie the occurrence of atrial fibrillation. It has been suggested that atrial size is an index of structural remodeling and that atrial conduction times are markers of structural and electrical remodeling (3). In another part of these comments, it was cited that tissue Doppler echocardiography is not a reliable method for the evaluation of atrial EMDs in subjects with a normal atrial size. There is one study (5) that compared atrial conduction times as evaluated by tissue Doppler echocardiography and electrophysiological studies, and this study showed a weak association between the two methods regarding inter-atrial EMD, a moderate association with respect to left intra-atrial EMD, and no association in terms of right intra-atrial EMD. Left atrial size in that study was normal. Nevertheless, it should be considered that in that study, a high right atrial signal was used instead of a tricuspid annulus signal. It can be cause of these weak associations found in that study. Consequently, although there are some doubts with respect to the measurement of atrial EMDs by tissue Doppler echocardiography, the existing literature lacks a well-designed study that compares results between electrophysiological study and tissue Doppler echocardiography. Moreover, there is no evidence for the shortcomings of tissue Doppler echocardiography in the evaluation of atrial EMDs in a normal atrial size. These are, therefore, queries that merit future research on the feasibility of tissue Doppler echocardiography in the evaluation of atrial EMDs.

Ali Hosseinsabet
Department of Cardiology, Tehran Heart Center, Tehran University of Medical Sciences, Tehran-I.R.Iran

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Address for Correspondence: Ali Hosseinsabet, MD
Tehran Heart Center, Karegar Shomali Avenue, Tehran-I.R.Iran
Phone: +98 218 802 97 31 Fax: +98 218 802 97 31
E-mail: Ali_Hosseinsabet@yahoo.com

Obstructive sleep apnea and cardiovascular disease: Is mean platelet volume one of the links?

To the Editor,

We read with great interest the excellent review entitled "Obstructive sleep apnea and its effects on cardiovascular diseases: a narrative review" by Rivas et al. (1) on the cardiovascular comorbidities of patients with obstructive sleep apnea (OSA) published. Indeed, it is increasingly being appreciated that patients with OSA are at a higher risk of coronary artery disease, congestive heart failure, stroke, and atrial fibrillation. Treatment with continuous positive airway pressure (CPAP) reduces these comorbidities (1).

A novel important, though less widely used, marker of the severity of OSA is mean platelet volume (MPV), as shown by Varol et al. (2, 3) and us (4). Again, CPAP treatment has been reported to reduce MPV (3). Given its role as a marker of vascular disease and a predictor of acute vascular events (5), it appears that MPV also links OSA with cardiovascular disease. Specifically, in patients with OSA, MPV is also associated with atrial fibrillation (5).

In conclusion, it is now established that OSA poses patients at an increased risk of cardiovascular disease (1). MPV may prove useful as a marker of the latter in patients with OSA (4, 5); therefore, it should be more widely utilized for this purpose.

Nikolaos Papanas, Dimitri P. Mikhailidis¹, Paschalis Steiropoulos*
Diabetes Center Second Department of Internal Medicine, and
*Pneumology, Medical School, Democritus University of Thrace,
Alexandroupolis-Greece

¹Department of Clinical Biochemistry (Vascular Disease Prevention Clinics), Royal Free Hospital campus, University College London Medical School, University College London (UCL), London-UK

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Address for Correspondence: Nikolaos Papanas, MD
Diabetes Center, Second Department of Internal Medicine
Democritus University of Thrace, 68100 Alexandroupolis-Greece
Fax: +302551074723 E-mail: papanasnikos@yahoo.gr
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Author's Reply

To the Editor,

We appreciate Dr. Nena's comments about our review article entitled "Obstructive sleep apnea and its effects on cardiovascular diseases: a narrative review," published in *Anatol J Cardiol* 2015; 15: 944-50, and her suggestion that mean platelet volume (MPV) may have prognostic importance as a risk factor for cardiovascular events and therapeutic importance as an indicator of a response to CPAP management in patients with obstructive sleep apnea (OSA) (1). MPV is a marker for thrombocyte activation. Larger platelets contain more granules and thromboxane A₂ and express more glycoprotein receptors. Therefore, these platelets aggregate more quickly and adhere more strongly to collagen, and this potentially leads to either an increased frequency or severity of thromboembolic events. Because patients with OSA have an increased frequency of atrial fibrillation and stroke and because OSA has adverse effects on outcomes in patients with other cardiovascular disorders, measuring MPV may help classify patients into risk categories and identify patients who might need additional therapy.

One important issue in studies using MPV as an indicator of vascular events is whether to consider MPV as a continuous variable or as a categorical variable, which is of interest only if it is above the upper limit of normal or some other critical value based on outcome studies. Another important issue is the study population. Is it more important to study patients with underlying risk factors for cardiovascular disease or to study patients without any obvious evidence of cardiovascular disease? Karakaş et al. (2) analyzed MPV in controls and in patients with OSA with mild, moderate, and severe increases in apnea-hypopnea index (AHI). They found that it was significantly higher in patients with severe OSA than in control subjects. However, the reported values appeared to be within the normal range, and absolute differences were small (8.6 versus 7.8 femtoliters). They did find significant correlations