atrial fibrillation" published in Anatol J Cardiol 2017;18:15-22, by Kupczynska et al. (1) with great earnest and wish to commend the authors for their interesting work on this new and potentially impactful subject. The study investigated the link between transthoracic echocardiographic markers of left atrial function, including novel ones obtained through speckle-tracking echocardiography, and the presence of left atrial appendage thrombus on transoesophageal echocardiography.

Speckle-tracking echocardiography is a novel technique, which uses dedicated software that analyses the motion of specific segments of the myocardium to determine their fractional shortening, but it is fraught with technical issues. One of them is related to heart rate variability because strain and strain rate values are directly influenced by the length of diastole, and as such, this technique is designed for patients with regular heart rhythms (2). In this regard, the authors' approach of using speckle-tracking echocardiography in patients with atrial fibrillation is brave and their solution of using an indexed beat with the smallest R-R variability compared with previous beats could be a very practical solution.

A specific limitation of atrial strain is the dependence of atrial function on left ventricular function, and in this regard, the study groups are markedly different. The patients with left atrial appendage thrombi had a reduced left ventricular ejection fraction compared with those without a thrombus, with 53% of them having a severely reduced ejection fraction, perhaps owing to the increased prevalence of coronary artery disease. Although this is somewhat expected, it translates into increased ventricular filling pressures and increased atrial filling pressures, a fact demonstrated by the significantly increased mean indexed left atrial area and volume of the thrombus group compared with those in the no thrombus group (12 vs. 14 cm^2/m^2 and 28 vs. 34 mL/m^2, respectively). For evaluating this interdependence, the evaluation of left ventricular diastolic function would be useful, but the authors did not present this data.

However, the results of the study are very interesting because they identify left atrial longitudinal strain rate as a better predictor of left atrial appendage thrombus than the CH2ADS2-VASc score in this study population. Although this score is not used for predicting a left atrial appendage thrombus, it uses clinical variables known to be associated with thrombus formation and can be a good indicator of its presence (3). In this sense, speckle-tracking echocardiography cannot replace clinical evaluation, but it can provide additional information to improve risk assessment, as demonstrated by the improvement in predictive power of the model that uses both the score and atrial strain developed by the authors.

The added value of atrial longitudinal strain measurements brought to the CH2ADS2-VASc score in thrombus prediction proves that it is a valuable tool, and this study lays the groundwork for future prospective studies that can provide more proof of its usefulness in these patients.

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Author`s Reply

To the Editor,

We would like to thank Dr. Nistor (1) for his interest in our recent article entitled "Association between left atrial function assessed by speckle-tracking echocardiography and the presence of left atrial appendage thrombus in patients with atrial fibrillation." published in Anatol J Cardiol 2017; 18: 15-22. regarding the association between left atrial function assessed by speckle-tracking echocardiography and the presence of left atrial appendage thrombus in patients with atrial fibrillation and for his insightful comments concerning our study.

Indeed, the heart rate variability poses a problem for reliable speckle-tracking echocardiography analysis during atrial fibrillation, and to handle this concern we have implemented the previously proposed method and have validated it for the assessment of left ventricular strain during atrial fibrillation (2).

With regard to the difference in left ventricular function between patients with and without left atrial appendage thrombi, we agree that it could have influenced the difference in atrial function, but at the same time, we would like to emphasize that left atrial function parameters (average left atrial longitudinal early diastolic strain rate, average left atrial longitudinal systolic strain rate, and average peak positive longitudinal systolic atrial strain) were independently associated with the presence of left atrial thrombi in the multivariate analysis.

We greatly appreciate Dr. Nistor's comment regarding the need for further prospective clinical studies. We fully agree that additional data are essential to clarify the diagnostic role of the

left atrial strain and strain rate in the choice of proper treatment for patients with atrial fibrillation.

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Ganglionated plexi interactions with atrio-ventricular node and right vagus nerve

To the Editor,

We read with great interest the article titled "Vagal denervation in atrial fibrillation ablation: A comprehensive review" by Aksu et al. (1) published in Anatol J Cardiol 2017; 18: 142-8. It is an excellent narrative review about ganglionated plexi (GP), vagal denervation, and atrial fibrillation. We have some commentaries about GP interactions with atrio-ventricular node (AVN) and right vagus nerve.

There is a large interactive network among different GP; this network serves as an "integrated center" of the cardiac autonomic innervation (2). Vagus nerve exerts its influence on the AVN through the epicardial fat pads that are primarily located on the posterior wall of the left atrium (2). The inferior vena cava-left atrium fat pad (namely also right inferior GP) located around the coronary sinus mainly provides vagal innervations and selectively innervates the AVN in humans (2, 3). It was shown that high-frequency stimulation of the right anterior (or superior right atrial vagal GP) and left superior GP (or superior left atrial vagal GP) could also influence the AVN (3). In addition, the influence of the right anterior GP on the AVN appears to be more important than its influence on the left superior GP (2, 3).

A functional neural pathway between the right vagus nerve and the AVN was identified (2), and the integrity of the GP seems to represent a mandatory interconnected network (3). In this study by Xhaet et al. (3), the absence of any alteration in the ventricular rate in response to high-frequency stimulation of the right vagus nerve after the ablation of GP suggests that the right vagus nerve is not directly connected to the AVN and that the integrity of the GP is required to produce vagal effects on the AVN (3). Probably, there is no direct pathway between both the right and left vagus nerves and the AVN.

The long-term influence of GP ablation on the electrophysiology of the AVN is not known. However, the incomplete GP ablation can increase the vulnerability of the atria to atrial fibrillation and denervation is likely transient (4, 5). In addition, GP ablation that led to parasympathetic denervation of the AVN could play a role in the high ventricular rate response of atrial tachycardia after atrial fibrillation ablation. Therefore, GP interaction with the AVN and right vagus nerve could provide new insights on that particular mechanism.

Figure 1 is very interesting from the anatomical point of view, but there are two GC without any GB. The alignment of the GP nomenclature is also obviously required.

In conclusion, there are GP interactions with the AVN and right vagus nerve with possible important consequences on vagal denervation in atrial fibrillation ablation. However, the role and influence of the GP on the complicated vagal innervation of the heart still needs to be clarified.

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