Author`s Reply

To the Editor,

Thank you very much for Yiğiner et al. (1) interest related to the article entitled "Influence of the left ventricular types on QT intervals in hypertensive patients" in this issue. We are very glad that you gave us the opportunity to answer the letter. Here is our response to the letter.

One of the first studies (published in 2001) showed that the Tpeak-Tend interval (Tp-e) was not affected by left ventricular hypertrophy (LVH). The main effect was an increase in QT peak dispersion, resulting from an increase in the maximum QT peak interval (but not in the minimum QT peak interval) (2). Prolonged transmural dispersion of repolarization (TDR) is associated with the induction as well as spontaneous development of ventricular tachycardia in higher risk patients (3). The Tp-e/QT ratio is probably a better predictor of adverse outcomes, particularly after successful primary percutaneous coronary intervention (PCI) in patients with STEMI. In a previous study, patients with a Tp-e/QT ratio of >0.29 showed elevated rates of hospital death, main adverse cardiac events, all-cause death, and cardiac death after discharge (4). We intended to exclude patients who had organic heart disease, hypertrophic cardiomyopathy without hypertension, diabetes, and several other diseases that could influence the occurrence of arrhythmias, as mentioned in the methods section. Only patients with essential hypertension were included in the study. Such rigorous inclusion criteria demand a long period of selection of appropriate patients. Only few published studies have revealed TDR as a marker of proarrhythmic risk

in patients with hypertensive LVH (2, 5). The majority of studies investigating Tp-e and Tp-e/QT ratio as markers of TDR are related to the LQT syndrome, Brugada syndrome, or influence of drugs on TDR.

In our work, LVH in ECG was determined according to two criteria: Sokolow-Lyon and left ventricular strain criterion. The majority of our patients had complex morphology of T waves and it was difficult to determine Tp-e manually, as has been mentioned by other studies (5, 6). The most expressed changes were exactly in the lateral leads that view the electrical field across the ventricular wall. In one study, a close correlation was found between the QT interval and T-wave variables in hypertensive patients (5). Therefore, it is expected that Tp-e is prolonged in patients with LVH, and investigation of TDR parameters would probably result in non-significant results. We did not measure TDR. It can be assumed that Tp-e in our patients would be in correlation with the QT interval and QT dispersion.

Investigation of TDR in hypertensive patients with LVH in relation to the different patterns of LVH can be the topic of some further investigations.

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