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Transmural dispersion of repolarization: a complementary index for cardiac inhomogeneity

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To the editor

We read the article written by Nuis, *et al*.[1] The authors investigated the effects of transcatheter aortic valve implantation (TAVI) on corrected QT dispersion (cQTD) in severe aortic stenosis patients. They concluded that, 40% of patients had defective cQTD recovery at six months after the procedure and this was associated with increased late mortality risk. We would like to contribute to the study from a different methodological perspective.

In this study, the percentages of defective cQTD patients were higher than we expected. This findings were interesting because previous studies had shown that, aortic balloon valvuloplasty and surgical aortic valve implantation were associated with cQTD recovery.[2,3] Moreover, Erkan, *et al*.[4] have reported that TAVI procedure reduced QT dispersion six months after the intervention. This inconsistency may be attributed to measurement differences. We appreciate the magnification of ECG recordings to 50 mm/s and evaluating with two different observers. However, according to our experiences, manual ECG measurement is extremely difficult. It would have been better if the authors had transferred ECG recordings to the digital platform in order to obtain more precise data.

QT dispersion is the most frequently used parameter to determine ventricular homogeneity and arrhythmia risk. However, there are some technical difficulties of QT dispersion measurement. For example, it has low reproducibility, thereby the inter- or intra-observer variabilities are very high. In addition, determination of the end of the T wave in some ECG leads such as V1, V2 or aVF is frequently difficult. Alternatively, transmural dispersion of repolarization (TDR) have been using for ventricular arrhythmia prediction over past decade. Briefly, myocardium consists of three different cell layers: endocardial, epicardial and midmyocardial M cells.[5] M cells exhibits the longest action potential duration. Moreover, M cells are more susceptible to action potential prolongation when exposed to external factors. On surface ECG, the repolarization of epicardial layer ends at the peak of T-wave but M cells' repolarization continues until the end of T wave.[5] Thus, it is possible to determine the M-cells repolarization period by measuring the time between the peak and end of the T wave, which is called as Tp-e interval and reflects transmural dispersion of repolarization. Some studies have shown that, Tp-e interval are associated with ventricular arrhythmias, sudden cardiac death, Brugada syndrome, short and long QT syndromes and ST elevated myocardial infarction.[5-7] It would have been better if the authors had evaluated these parameters in order to obtain more comprehensive data. Thus, it would be reasonable adding TDR parameters to the study which would support the results of TAVI on the life threatening arrhythmias. We believe that, the study of Nuis, *et al*.[1] will inspire to the researchers for future investigations.

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Authors' reply

We thank Dr Dogan and his team for his thoughtful comments and suggestions. The first comment to our study relates to the percentage of defective cQTD patients that was higher in our study as compared to previous studies.[1–3] We would like to point out that defective cQTD occurred in 40% while recovery of cQTD occurred in the majority of patients (60%). Also, the mean cQTD in the overall population significantly decreased after transcatheter aortic valve implantation (TAVI) (47 to 40 ms, $P = 0.049$). We do agree, however, that cQTD determination to assess ventricular inhomogeneity and arrhythmia risk suffers from (technical) measurement difficulties resulting in increased inter- and intra-observer variabilities and imperfect estimates of the true ventricular repolarization inhomogeneity. Therefore, a different methodological perspective such as measurement of T-peak to T-end wave dispersion should also be investigated in patients undergoing TAVI albeit that this method is also likely to suffer from measurement difficulties when assessing the end of the T wave (especially in lead V1, V2 or aVF). We agree that further research is indeed needed to investigate whether repolarization dispersion measurements correlate with sudden death and arrhythmia risk as suggested by Dogan *et al*. with methods that include or correct for the different electrical properties of the different myocardial cells.

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