To the Editor:

In their recent article, Ghosh et al demonstrated the feasibility of noninvasive localization of accessory pathways in patients with Wolff-Parkinson-White syndrome through high-resolution body surface ECG mapping. These results nicely confirm data from our group that demonstrated a high accuracy of noninvasive ECG imaging as compared with electroanatomical invasive mapping (CARTO) in localizing the accessory pathway in patients with Wolff-Parkinson-White syndrome. The localization error in our study was 18±6 mm. However, a direct comparison of these two studies is difficult because Ghosh et al were not able to calculate the localization error. This inability was due to the fact that they did not use invasive electroanatomical 3-dimensional mapping, which constitutes the current gold standard in electrophysiology.

In contrast to our approach, the ECG imaging method used by Ghosh et al enables imaging of repolarization and thus analysis of cardiac memory following radiofrequency ablation. However, the data presented are somewhat confusing. For patient W1, the first ventricular activation occurs at the insertion of the accessory pathway in a left posterior position (Figure 3A). The beginning of ventricular repolarization is observed at the same location (Figure 3B), and the longest activation recovery interval (ARI) is observed at the accessory pathway insertion site. From Figures 3C and 6A, we estimated that repolarization at the accessory pathway insertion occurs about 130 ms later than at the apex. Furthermore, concluding from the color coding in Figures 6B and 6D, the ARI shortens by 60 ms within 1 month after ablation. In an apparent contradiction, the interval from Q-wave onset to T-wave end prolongs in the epicardial electrograms plotted in the same figures. Similar observations can be made for other patients.

Perhaps these contradictions are due to the fact that the authors used a static computer model for their reconstructions, which did not take into account the myocardial deformation in the contracting heart. Furthermore, local myocardial necrosis after delivery of radiofrequency energy may also result in regional changes in repolarization. This effect strongly depends on the amount of necrosis. Therefore, we question whether changes in the ARI are solely due to the change in the activation sequence after the ablation or whether they may also be influenced by the ablation procedure itself. Therefore, we are interested in the number, duration, and power of the radiofrequency impulses that were delivered and the temperature that was reached. Have the authors performed any measurements for quantification of myocardial damage after ablation, such as troponin levels?

Disclosures

None.

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References

Letter by Berger et al Regarding Article, "Cardiac Memory in Patients With Wolff-Parkinson-White Syndrome: Noninvasive Imaging of Activation and Repolarization Before and After Catheter Ablation"
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