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Editorial

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Editorial: Simple Maneuver for Estimating the Depth of the Focal Activation Source in Myocardium

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► See the article "Simple Maneuver for Estimating the Depth of the Focal Activation Source in Myocardium" in volume 50 on page 420.

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In the current issue, Oh et al.[1] suggested that the depth of origin of triggered ventricular arrhythmia (VA) can be predicted by using the new mapping and pacing technique introduced in this study. First of all, using a rational mathematical formula was a valuable idea. I believe it is a meaningful article in terms of the concept to find the actual depth of VA trigger origin in the myocardium to improve radiofrequency (RF) ablation outcomes.

For instance, we had some patients who survived from sudden cardiac death with the implantable cardioverter-defibrillator implantation, thereby needing additional RF ablation of ventricular tachycardia (VT). Furthermore, if the endocardial and epicardial 3-dimensional voltage mapping shows normal voltage area in patients with scars revealed by imaging modalities and an epicardial origin of VA is supposed in surface electrocardiogram (ECG), the intramural lesion should be suspected.

Recently, RF ablation can be performed successfully for most of the idiopathic VAs in the endocardial or epicardial sites. An epicardial origin of VAs can be predicted by using surface ECG, then the procedure is performed with the consideration of the epicardial origin in advance. However, most of the failure cases of RF ablation are intramural origin VAs, which are usually found in patients with structural heart diseases such as ischemic/non-ischemic cardiomyopathy (CMP) and hypertrophic CMP, etc. In addition, the VAs in the patients with structural heart diseases are rather complicated in terms of its direction and sites, due to complex cell-to-cell relationship and structure. Recently, new treatment techniques such as intramural needle ablation and noninvasive radioablation therapy have been developed to improve success rates of RF ablation for intramural scar origin VAs.

In 1984, Cassidy et al.[2][3] presented that the electrograms recorded may simply reflect the myocardial disease regardless of the type. The size and depth of the actual anatomical circuit has not been ascertained with the currently available techniques. Recordings obtained during endocardial catheter mapping may reflect activity outside this circuit, thereby rendering such information is less useful. In addition, tissue anisotropy in diseased myocardium rather creates fractionated electrograms.[4] Finally, conduction velocity and activation patterns during sinus rhythm at the recorded site of origin of VA may not be simple, so the assumption that such electrograms are a marker of the slow conduction pathway necessary for reentry may not be valid.

Wetzel et al.[5] presented in 2003 that post-infarction VT is an arrhythmia based on reentry circuits in the complex 3-dimensional structure of normal and abnormal myocardial fibers within the scar area itself and its border zone. On simplified examination, the scar area contains surviving fibers acting as the so-called central common pathway allowing the reentry to occur. But the "reality" of scar-related VT is much more complex with branching and merging of surviving myocytes separated by connective tissue, leading to a high degree of inhomogeneous anisotropy with zigzag routes of activation. This could be demonstrated by several mapping studies and in histologic evaluations of myocardial scar tissue.[6]

To quote these papers, the results may be different as to what the author suggested in this study. In other word, the conduction velocity can be different even with the similar depth according to the extent of myocardial disease. So, we currently have no other option but to use endocardial and epicardial mapping to find reentry circuit excluding the intramural origin VA. In company with the mapping tools, we use intracardiac echocardiography or cardiac MRI to find the depth of scar, besides intramural scar.

Nonetheless, I consider this study is valuable as it suggests an idea for the development of new mapping catheter to predict the depth of VA even though its immediate use is not possible in clinical practice.

REFERENCES

1. Oh S. Simple maneuver for estimating the depth of the focal activation source in myocardium. *Korean Circ J* 2020;50:420-9.
CROSSREF
2. Cassidy DM, Vassallo JA, Marchlinski FE, Buxton AE, Untereker WJ, Josephson ME. Endocardial mapping in humans in sinus rhythm with normal left ventricles: activation patterns and characteristics of electrograms. *Circulation* 1984;70:37-42.
PUBMED | CROSSREF
3. Cassidy DM, Vassallo JA, Buxton AE, Doherty JU, Marchlinski FE, Josephson ME. The value of catheter mapping during sinus rhythm to localize site of origin of ventricular tachycardia. *Circulation* 1984;69:1103-10.
PUBMED | CROSSREF
4. Fenoglio JJ Jr, Pham TD, Harken AH, Horowitz LN, Josephson ME, Wit AL. Recurrent sustained ventricular tachycardia: structure and ultrastructure of subendocardial regions in which tachycardia originates. *Circulation* 1983;68:518-33.
PUBMED | CROSSREF
5. Wetzel U, Hindricks G, Dorszewski A, et al. Electroanatomic mapping of the endocardium. Implication for catheter ablation of ventricular tachycardia. *Herz* 2003;28:583-90.
PUBMED | CROSSREF
6. de Bakker JM, Coronel R, Tasseron S, et al. Ventricular tachycardia in the infarcted, Langendorff-perfused human heart: role of the arrangement of surviving cardiac fibers. *J Am Coll Cardiol* 1990;15:1594-607.
PUBMED | CROSSREF

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