



Role of surface electrocardiogram in the era of high-resolution mapping and imaging systems—back to the future

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The last decade has witnessed a tremendous technological advance in the field of cardiac electrophysiology, which has transformed our understanding of the mechanisms underlying the development of the majority of cardiac ventricular arrhythmias (VAs).

As interest has shifted to more complex arrhythmias, sophisticated mapping and imaging systems have become of utmost importance to precisely diagnose, manage and treat cardiac rhythm disorders.

Nonetheless, surface electrocardiogram (ECG) still represents an important tool in the shed of the physician treating rhythm disorders (1). A detailed ECG analysis can provide useful, non-invasive, and inexpensive information on arrhythmia mechanisms and suggest the best ablation strategy to adopt in order to increase the probability of procedural success and prevent complications.

In this perspective, we have read with great interest the article by Andreu *et al.*, published on the October issue of the *Heart Rhythm Journal*, regarding a novel QRS axis-based algorithm to predict the origin of scar-related ventricular tachycardia (VT) in patients with structural heart disease (SHD) (2).

As a result of an abnormal substrate characterized by cellular damage, reactive and replacement fibrosis, the main

mechanism underlying the development of VAs in patients with SHD is re-entry around scar areas and through the conducting channels where the surviving tissues show slow and circuitous electrical conduction (3,4). On the basis of the exit site of these slow conduction pathways into the normal myocardium, the same scar area can manifest with several clinical VTs which can be mapped using conventional mapping techniques (e.g., activation mapping, pace-mapping and entrainment mapping). Additionally, substrate mapping during sinus rhythm allows to identify areas of normal myocardium, dense scar and border zone tissue.

Patients with SHD have a not-negligible risk of VAs which may result in sudden cardiac death, thus requiring antiarrhythmic drugs, the need for an implantable cardioverter defibrillator and catheter ablation procedures.

At the same time, these patients display a high prevalence of competing comorbidities and a remarkable intrinsic frailty. There may result in a higher risk of drug-related side effects and peri-procedural complications.

Catheter ablation is an effective therapeutic strategy for scar-related VTs. However, a tailored strategy is essential to optimize procedural duration, improve success rate and prevent or promptly treat any complication, especially in

patients with SHD.

A successful procedure requires a thorough pre-procedural analysis of the surface arrhythmic ECG. Specifically, predicting VT exit site before the procedure may help identify the optimal ablation strategy, including vascular and/or epicardial accesses, thus reducing related risks and duration.

Even though there are some general rules to help localize the VA site of origin (SoO), ECG interpretation in patients with SHD might be complicated by several factors, such as the size and distribution of myocardial scar.

In consideration of the above-mentioned advantages and limitations, different surface ECG-based algorithms have been proposed during the last decades.

A first algorithm developed by Miller *et al.* (5) correlated the VT exit site location identified during arrhythmia mapping with four electrocardiographic features of the index arrhythmia: location of the infarction, bundle branch block (BBB) type configuration, QRS axis quadrant and precordial R wave progression pattern.

The combination of these four features allowed to identify a specific ECG-predicted SoO that matched with the mapped SoO in 48% of total VTs.

Kuchar *et al.* (6) developed an algorithm based on ECG features derived from the analysis of QRS complex morphology during ventricular endocardial pace mapping, which was used as a surrogate of VT exit. The ECG features used to detect the VT SoO were: QRS configurations in leads V4-aVR to differentiate among apical and basal sites, leads II-III-V6 for anterior versus inferior exits and leads I-aVL-V1 for septal versus lateral origins. Although anterior, inferior, lateral, and septal regions were differentiated in approximately 90% of cases, Kuchar's algorithm correctly predicted the VT SoO in 39% of the patients on the basis of a triaxial mapping grid, composed by 24 possible endocardial sites. A key limitation of this approach was the use of electrocardiographic features based on findings derived from pace mapping, which is significantly less predictable in the setting of SHD, compared to patients with idiopathic VAs.

Additionally, the latter algorithm, as well as Miller's one, was created specifically for patients with SHD of ischemic aetiology and limited only to patients with previous anterior or inferior myocardial infarction (MI).

Several years later, Segal *et al.* (7) proposed a novel approach applicable to patients with all types of MI (not only anterior or inferior) of unknown location. VT exit sites detected via noncontact mapping were correlated with

specific QRS features, such as BBB pattern-type and limb lead polarity. The 3-dimensional LV endocardial surface was divided into 9 regions representing 9 different VT exit sites; epicardial mapping was not performed, thus excluding VTs exiting from sub-epicardial sites. The algorithm was applicable to 88% of patients and correctly predicted the SoO in 93% of cases.

The manuscript by Andreu *et al.* (2) describes a simple electrocardiographic algorithm for segmental localization of scar-related VTs using the left ventricle (LV) 17-segment model of the American Heart Association (AHA). The study included 108 patients with incessant VT, repetitive episodes of sustained monomorphic VT, appropriate implantable cardioverter-defibrillator (ICD) therapy or symptomatic drug-resistance premature ventricular complex. Patients with idiopathic VT or arrhythmogenic right ventricular dysplasia/cardiomyopathy were excluded.

The method is based on two simple steps. First, the QRS axis in the frontal plane is assessed; this allows to locate the VT origin in the LV short axis (inferior vs anterior, septal vs lateral). Second, the VT segment of origin (SgO) is localized in the longitudinal plane on the basis of the polarity in V3-V4 (a positive or negative concordance suggests a basal or apical origin, respectively, whereas discordant polarities identify a medial location).

The novel algorithm is short and nonquantitative, and may represent a handy and useful tool, especially during VT ablation procedures in patients with hemodynamic instability or incessant VTs.

Overall, the authors analyzed the electrocardiographic recordings of 149 successfully ablated VTs, whose exact SgO was localized via entrainment manoeuvres or accurate activation mapping (67.1%) or by means of pace mapping (32.9%).

Specifically, the study population included 31 (28.7%) patients with nonischemic cardiomyopathy and 77 (71.3%) patients with SHD with ischemic aetiology; among them, 29.9% had an anterior MI, 64.9% an inferior MI, 2.6% patients a septal MI and 2.6% a lateral MI.

The ECG-predicted SgO matched with the actual SgO in 82% of cases, and identified a segment adjacent to the actual SgO in 77.8% of mismatched cases.

Of note, the algorithm could not be used in only 2.6% of VTs owing to indeterminate axis. One of the major advantages of the proposed algorithm is its broad applicability. Unlike the previous algorithms (5-7), the one proposed by Andreu *et al.* (2) has been applied with no differences in accuracy on patients with either nonischemic

and ischemic cardiomyopathies; among the latter, patients with multiple scar locations were included, since the algorithm is not limited only to patients with previous anterior or inferior MI.

Additionally, a similar accuracy was documented for VTs of either endocardial and epicardial origin, an epicardial origin representing 24% of the total sample.

Although the presence of extensive ventricular scarring or multiple scar areas may affect impulse propagation and significantly alter the QRS contour, the present algorithm apparently overcame these limitations. As the authors suggest, the frontal plane axis might be less affected by scar-related activation disturbances, as the accuracy was similar among patients with large and small scars.

Another point worth discussing regarding the algorithm is the use of the 17-segment AHA model as the reference system for arrhythmia localization.

Non-invasive cardiac imaging techniques have become an integral part of electrophysiological procedures in the setting of different rhythm disorders, providing a consistent body of anatomical and functional information prior, during, and after ablation.

Data from echocardiography, computed tomography, and cardiac magnetic resonance can be easily integrated into electroanatomical mapping systems which can serve as a common platform to assist with electrophysiology procedures. A precise reconstruction of cardiac ultrastructure is crucial for a successful ablation strategy, in order to prevent complications, minimize radiation exposure and procedure time, and improve outcomes. Timing is particularly important in the setting of VT ablation of patients with SHD, as longer procedures are associated with a significantly high risk of peri- and post-procedural complications and hemodynamic instability (8).

An electrocardiographic algorithm built around a cardiac imaging segmentation system represents a step beyond electrocardiography, a valuable trait-d'union with different imaging modalities performed before and during ablation. The present algorithm based on the LV AHA 17-segment model simplifies integration of anatomical and electrophysiological data between electrocardiography and different imaging modalities, therefore facilitating localization of the scar segment responsible for the clinical VT before and during ablation.

This feature might be particularly attractive in the setting of substrate-based VT ablation. There is a strong body of evidence supporting the superiority of substrate modification compared to standard ablation in reducing the

risk of VA recurrences and all-cause mortality in patients with SHD (9,10). As an example, the VISTA randomized multicentre trial demonstrated that an extensive substrate-based ablation approach is superior to ablation targeting only clinical and stable VTs (10).

As our ablation target has moved from an arrhythmia-oriented approach toward a scar-based strategy, integrating electrocardiographic/electrophysiological data with anatomical information for substrate modification to reduce scar arrhythmogenicity is still of utmost importance for a more efficacious patient-tailored ablation.

One main limitation of the algorithm is that it has not been validated for VTs arising from the right ventricle. Specifically, patients with nonischemic cardiomyopathy and septal scar with a mid-myocardial pattern may display a right septal breakthrough with an ECG pattern very similar to an exit in the LV.

In conclusion, the authors are to be congratulated on a well-designed, easy-to-use QRS axis-based algorithm providing high-level accuracy for predicting scar-related VT SgO, regardless of the underlying heart disease, the distribution and extent of scar, and the epicardial vs endocardial origin. The use of the LV 17-segment AHA model encourages integration of ECG-based information with tissue imaging modalities and brings valuable information on the most optimal strategy to adopt for VA ablation.

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Footnote

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