EDITORIAL COMMENT
“The Older the Broader”

Electrogram Characteristics Help Identify the Critical Isthmus During Catheter Ablation of Postinfarct Ventricular Tachycardia*

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During the past 10 years, significant progress has been achieved in the treatment of supraventricular as well as ventricular tachycardia (VT) with radiofrequency (RF) catheter ablation. In particular, RF catheter ablation became the treatment of choice for atrioventricular-nodal re-entrant tachycardia, atrioventricular re-entry tachycardia, ectopic atrial tachycardia, atrial flutter, and idiopathic right or left VT. In contrast to a high success rate accomplished in the aforementioned tachycardias, ablation of atrial fibrillation and postinfarct VT is far less successful and still a matter of intensive research. The reason for may be the heterogeneous structural abnormalities of the underlying tissue from which the electrical abnormality originates, the lack of a fixed anatomic structure that is involved in the onset or maintenance of a macro-re-entrant or micro-re-entrant circuit, the possibility of multiple tachycardia morphologies using different exit or entry points within a large “arrhythmogenic” area and, most importantly, a continuous structural remodeling of the atrial or ventricular myocardium caused by the underlying disease process (1,2).

Postinfarction VTs arise in areas of fibrosis that contain surviving myocardial strands, producing a “zig-zag” course of activation leading to inhomogeneous anisotropy (3). Hidden in the arrhythmogenic area is the common central pathway, the critical isthmus, causing slowing of impulse conduction, allowing re-entry to occur. The isthmus itself may be surrounded by “dead ends” or branching of impulses that do not participate in the common pathway of the leading re-entrant circuit and may be misinterpreted when recorded with roving mapping electrodes (4).

In the early studies of patients with postinfarct VT who underwent intraoperative “point-by-point” passive mapping of endocardial and epicardial myocardium, bipolar electrograms within the infarcted area demonstrated abnormally broadened and fractionated electrograms as well as delayed isolated potentials and double potentials during sinus rhythm (5). They were considered as “arrhythmogenic,” and by either removing or encircling them, it was hoped that surgical techniques such as large endocardial resection would abolish VT (6,7).

With the introduction of catheter ablation for VT, new mapping strategies to identify the critical isthmus within or at the border of the infarcted area were designed. The target site of ablation is identified by activation mapping and stimulation techniques using concealed entrainment criteria in an area where mid-diastolic potentials are recorded (8). This, however, requires induction of the “clinical” VT, which may not be inducible or give rise to many “nonclinical” VTs. Often, the induced VT is hemodynamically unstable, and thorough activation mapping becomes impossible. New computer-assisted mapping tools, such as the three-dimensional electroanatomic (CARTO, Biosense Webster, Diamond Bar, California) (9) or the noncontact mapping system (EnSite 3000, Endocardial Solutions Inc., St. Paul, Minnesota) (10), have been developed to either facilitate activation mapping or to create voltage maps to delineate the arrhythmogenic substrate (i.e., the low-voltage zone during sinus rhythm looking for very low-amplitude electrograms, fractionation, and late potentials) (11). Applying linear lesions with RF energy along an identified border between scar and normal voltage tissue or anatomic boundaries encircles or isolates the critical arrhythmogenic substrate (12). This approach is most promising, and identifying only abnormal electrograms during sinus rhythm within the infarcted area may be more successful than sophisticated entrainment mapping to pinpoint one or multiple critical isthmus spots of potential re-entrant circuits. Activation mapping in postinfarct VT could probably be replaced by “substrate” mapping.

The findings presented by Bogun et al. (13) in this issue of the Journal support the importance of electrographic characteristics in patients with postinfarct VT. Sites with the broadest electrograms or isolated potentials detected during sinus rhythm mapping were identical with the critical isthmus of the re-entrant circuit during induced VT. The application of RF energy was most successful when delivered within the area of abnormal electrograms.

Most interesting in the study by Bogun et al. (13) was the fact that they found a significant positive correlation between infarct age and the duration of the broadest endocardial electrograms (>200 ms) during sinus rhythm mapping in the peri-infarct zone. The older the infarct, the broader the electrograms, the longer the delay between the electrogram and the isolated potentials. There was no correlation with left ventricular ejection fraction, number of old infarctions, the infarct location, and the area of infarct size.

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However, other hemodynamic parameters or ventricular dilation parameters were not assessed. The authors explain their time-dependent findings by a continuous remodeling process after the acute infarction event with gradual formation of areas with surviving myocardial strands separated by fibrous tissue deposition. There are experimental data to confirm this (14), and clinical experience also tells us that growing time after myocardial infarction increases the likelihood of developing sustained VT or life-threatening ventricular tachyarrhythmias (15).

A word of caution is necessary not to oversimplify their interesting and important findings. In the report of Bogun et al. (13), the time of first infarction to the time of the electrophysiologic study and ablation procedure ranged from 1 to 31 years, with a mean of 16 years after the index event. The reported series included only 23 patients, but in 5 patients the time of infarction could not be determined, and 10 patients had a second infarction before the study. Is there a continuous uninterrupted remodeling process over the years to come after myocardial infarction, and when is the formation of the arrhythmogenic substrate finished to originate a sustained VT? If the formation of the arrhythmogenic substrate is time dependent, how do we explain that some patients may develop VT after a few weeks, some after many years after myocardial infarction, whereas others will never experience VT episodes although suffering from the same amount of ventricular dilation and ventricular dysfunction?

Remodeling after myocardial infarction is a multiformal and complex mechanism involving molecular and cellular components, causing early and late changes in ventricular size, architecture, function, and electrical stability in infarcted but also noninfarcted areas (14). Experimental data from healing myocardial infarction are mostly derived from early postinfarct periods (15), and information on the late genetic and molecular process that perpetuates remodeling and may cause arrhythmias is scarce (16,17).

The major limitation of the study is that we have no insight into the electrogram characteristics in patients with similar functional remodeling late after myocardial infarction who did not develop VT. They may have the same electrogram characteristics.

The specificity of the described electrogram changes is unknown, and further studies with a larger patient cohort are needed to prove the authors’ conclusion that the older the myocardial infarction, the broader the potentially arrhythmic electrogams, and the easier it will be to use them as target sites for ablation.

To widely apply the described technique of VT ablation by targeting the sites of broadest electrogams during sinus rhythm, a generally accepted definition of electrogram duration, fractionation, and isolated potentials is mandatory. Defined cut-off points of beginning and ending of electrograms as well as agreement on signal filtering, amplitude, amplification, and sampling rate of bipolar signals from low voltage areas are needed.

Because we did not learn from Bogun et al. (13) how many broad electrogram sites were ablated with RF energy until the clinical VT became uninducible, further studies are needed to demonstrate if encircling the areas of broad electrogams with linear lesions is sufficient or if each critical isthmus site must be destroyed separately. If, however, broad electrogams are the consequence of a perpetuating remodeling process after myocardial infarction, there is little hope that permanent suppression of VT may be achieved because new critical isthmus sites will arise unless the remodeling process can be stopped by other approaches such as drugs, revascularization, or non-pharmacologic techniques. Applying sophisticated entrainment techniques and mapping the earliest breakthrough during induced VT may become unnecessary in the future, if we target the broadest electrogams within the low-voltage infarction zone during sinus rhythm (18). Applying efficient energy with the appropriate electrode tool that is able to delineate and destroy the most critical arrhythmogenic electrogams, either endocardially or epicardially, will certainly increase the success rate of currently still unsatisfactory results of VT ablation.

Because structural remodeling is the basic underlying mechanism of both VT and atrial fibrillation, it is time to look for similar electrographic characteristics in patients with atrial fibrillation to improve results of extensive and time-consuming ablation procedures in patients with persistent or chronic atrial fibrillation (19).

References


