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Review

Substrate-based approach for ventricular tachycardia in structural heart disease: Tips for mapping and ablation

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ABSTRACT

Catheter ablation of scar-related ventricular tachycardia (VT) often relies on substrate-based approaches because of hemodynamic instability during VT, multiple VT morphologies, and poor reproducibility of VT-inducibility, rendering the VT unmappable. As substrate-guided ablation is performed in stable sinus rhythm, any VT can potentially be targeted regardless of its hemodynamic state. So-called "late potentials," conventionally defined as signals detected after the end of QRS, have been traditionally proposed as ablation targets. However, late potentials cannot be detected in up to 30% of patients with VT in the setting of ischemic and non-ischemic cardiomyopathy. Recently, a substrate-based approach that targets poorly coupled fibers surviving within the scar has been developed. These bundles generate local abnormal ventricular activities (LAVA) and are believed to be responsible for VT. Considering the limitations of late potential ablation, substrate homogenization with the aim of eliminating all identified LAVA appears to be an ideal procedural endpoint. This article reviews substrate-based approaches and tips for mapping and ablation of VT substrate.

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1. Introduction

Ventricular tachycardia (VT) most frequently occurs in the presence of structural heart disease, and it is an important cause of mortality and morbidity. The vast majority of cases of structural heat disease are associated with some degree of scarring that serves as the substrate for sustained VT. Implantable cardioverter defibrillators (ICDs) or cardiac resynchronization therapy defibrillators are used in patients at high risk of sudden death due to malignant ventricular arrhythmias. However, despite effective VT termination, recurrent VT, which gives rise to frequent ICD discharges, is associated with increased rates of mortality and

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congestive heart failure hospitalization as well as reduced patient quality of life [1–8]. Pharmacological therapies such as beta-blockers, amiodarone, and sotalol are used to reduce recurrent VT. The combined use of amiodarone and beta-blockers is more effective than sotalol or beta-blocker therapy alone [9]. However, it is important to recognize that these pharmacological therapies may have adverse effects on clinical outcomes. Amiodarone causes multiple organ toxicities that are associated with increased mortality in patients with New York Heart Association class III heart failure and no ICD [10]. The side effects of amiodarone restrict its long-term use in more than 20% of patients [9].

Catheter ablation is an effective therapeutic option in the management of scar-related VT [11,12]. Ablation targets a critical isthmus to interrupt the re-entrant circuit [11–14]. Ideally, VT will be reliably inducible, hemodynamically well tolerated, and of a single morphology, allowing for activation and entrainment mapping. However, this is the exception rather than the rule. Hemodynamic instability and non-inducibility throughout the procedure can render the VT unmappable [13,14]. Furthermore, most patients with scar-related VT have multiple VT circuits with multiple QRS morphologies on surface electrocardiograms. VT frequently changes QRS morphology (often with entrainment attempts). A previous multicenter study showed that 31% of patients had only unmappable VT and 38% patients had both mappable and unmappable VT morphologies targeted [13]. In these situations, a substrate-based approach is attractive. A substrate mapping and ablation strategy in sinus rhythm allows us to offer ablation therapy to patients with unmappable VT. Various ablation strategies such as targeting late potentials, endo- and epicardial scar homogenization, and eliminating local abnormal ventricular activities (LAVA) have been described previously [15–23]. In this article, we review substrate-based approaches and tips for mapping and ablation of VT substrate in patients with structural heart disease.

2. Substrate mapping in sinus rhythm

In previous studies using explanted human hearts, de Bakker et al. demonstrated that there are bridges of surviving myocardial cells within the post-infarction VT scar that connect opposite borders of the scar [24–26]. The surviving fibers surrounded by electrically unexcitable fibrosis serve as a slow-conducting pathway of VT re-entrant circuits. In non-ischemic dilated cardiomyopathy, replacement of muscle by fibrosis creates conducting channels that similarly facilitate a re-entrant circuit. Cassidy et al. noted that local electrograms at the sites of VT origin displayed lower amplitudes and longer electrogram durations than those at other sites, as obtained from intra-operative mapping in sinus rhythm [27]. Endocardial abnormal and fractionated electrograms can be eradicated by surgical subendocardial resection, with long-term arrhythmia-free rates of 60–80% in patients with a previous myocardial infarction [28].

The concept of substrate mapping and ablation evolved from these surgical experiences. Unlike surgeons who can visually identify the infarct region, electrophysiologists must locate the abnormal region using the guidance of local ventricular electrograms. Marchlinski et al. reported that voltage mapping in sinus rhythm using 3-dimensional electroanatomic mapping can characterize electroanatomic substrates [14]. A peak-to-peak bipolar amplitude <0.5 mV identifies areas with extremely abnormal signals (dense scars). The dense scar is typically surrounded by a border zone area with an electrogram amplitude of 0.5–1.5 mV. This voltage criterion was recently validated by using fluorodeoxyglucose positron emission tomography [29]. Delayed enhancement as depicted by contrast-enhanced magnetic resonance imaging can also exhibit a good correlation with the low-voltage region [30,31].

The surviving myocytes responsible for VT should form anatomically definable conduction channels within the dense background scar. Arenal et al. and Hsia et al. described that areas of relatively higher voltage corresponding to surviving bundles of myocardial cells within the scar tissue can function as conducting channels, which can be depicted on a color-coded electroanatomic voltage map [19,32]. They used different voltage cutoff values to define scars to identify conducting channels as corridors of continuous electrograms in the scar. By applying a stepwise reduction in the definition of abnormal voltage from 0.5 to 0.1 mV, most conducting channels were found to have voltage scar definitions of ≤ 0.2 mV. Recently, Mountantonakis et al. demonstrated the non-critical relationship between voltage-defined channels and VT critical isthmuses [33]. Voltage channels can be identified in 87% of patients with mappable ischemic VT by adjusting the voltage limits of bipolar maps; however, the specificity of those channels in predicting the location of VT isthmus sites is only 30%. The presence of late potentials inside the voltage channel significantly increases the specificity for identifying the VT isthmus (Fig. 1). This study indicates the importance of identifying both abnormal voltage areas and abnormal local electrograms as optimal targets of VT substrate ablation.

Because precise assessment of the arrhythmia substrate pre- and post-ablation is mandatory, substrate-based approaches using 3-dimensional electroanatomic mapping systems are dependent on high-density mapping of the VT substrate during the baseline rhythm. Whereas point-by-point mapping can be time consuming and sometimes operator-dependent, multielectrode mapping may produce a high-density map in a timely manner. This potentially overcomes failure due to unrefined mapping. High-density electroanatomic mapping is performed with a multipolar mapping catheter (PentaRay, Biosense Webster, Diamond Bar, CA, USA). The splines of this catheter are extremely soft and provoke very few mechanical ectopics. The greatest value of the PentaRay is in epicardial mapping, as recently described [34]. The elimination of epicardial arrhythmia substrates from endocardial ablation is feasible on using the PentaRay catheter. An epicardially placed PentaRay can be used for both high-density mapping and as a landmark of the target epicardial abnormal region, precisely guiding the operator to the facing endocardial site. It also enables real-time monitoring of the impact of endocardial ablation on epicardial arrhythmia substrates.

3. Substrate ablation strategies

The presence of surviving bundles of myocytes that were electrically uncoupled from the surrounding myocardium by interstitial fibrosis is indicated by abnormal signal characteristics within low-voltage scars. These abnormal characteristics include fractionation, prolonged duration, and late potentials [35]. There are several substrate-based ablation approaches to determine ablation targets and modify or eliminate the VT substrate.

3.1. Pace mapping

Pace mapping can provide useful information by demonstrating local tissue excitability, slow condition properties at the pacing site, and the surface QRS morphology during pacing. Soejima et al. marked areas with high pacing thresholds of more than 10 mA at a 2-ms pulse width as unexcitable scars [36]. Pacing non-capture may also indicate insufficient tissue contact by the pacing electrode.

Pace mapping deep within the scar can identify slow conduction by a long stimulus to the QRS interval (Fig. 2), although a long stimulus–QRS interval can also occur in bystander regions [37–39].