Use of a novel fragmentation map to identify the substrate for ventricular tachycardia in postinfarction cardiomyopathy

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BACKGROUND Substrate ablation is commonly performed in patients with postinfarction cardiomyopathy and ventricular tachycardia (VT). Recognition of fragmented and late potentials during sinus rhythm is a tedious process subject to operator fatigue.

OBJECTIVE The purpose of this study was to assess the value of automated analysis to quantify electrogram fragmentation and to determine the relationship of fragmented regions to the VT isthmus.

METHODS Detailed left ventricular (LV) mapping was performed in 2 groups: (1) 14 patients with previous myocardial infarction and tolerated VT and (2) 14 controls with structurally normal hearts. In patients with VT, mid-isthmus sites were identified using entrainment mapping. Sinus rhythm endocardial LV electrograms underwent time– and frequency–domain analysis and were displayed as fragmentation or frequency maps. The region of fractionated electrograms and their relation to the VT isthmus sites were determined.

RESULTS Cutoffs for abnormal electrogram fragmentation were ventricular fractionation index ≥ 7 and fast Fourier transform ratio $\geq 14\%$, respectively. In the time domain, LV surface area with fractionated electrograms was significantly smaller than the total scar surface area (27.3% $\pm$ 7.1% vs 42.1% $\pm$ 12.3%, $P < .001$), yet contained 100% of VT isthmus sites. In the frequency domain, areas of abnormal fractionation occupied 9.7% $\pm$ 6.9% of total LV surface area and included only 60% of the VT isthmus sites.

CONCLUSION Automated electrogram fractionation analysis represents an objective tool to rapidly quantify electrogram fragmentation and guide substrate-based ablation of VT. Empiric ablation of these regions may be a new strategy for substrate-guided VT ablation.

KEYWORDS Ventricular tachycardia; Fragmentation; Mapping; Ablation; Cardiomyopathy; Fourier transform

ABBREVIATIONS **A-VFI** = abnormal ventricular fragmentation index; **FFT** = fast Fourier transform; **FFTr** = fast Fourier transform ratio; **H-FFTr** = high fast Fourier transform ratio; **HF-VFI** = highly fragmented ventricular fragmentation index; **LV** = left ventricle; **VFI** = ventricular fragmentation index; **VT** = ventricular tachycardia

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Introduction

Entrainment mapping is the gold standard for identifying the critical isthmus of ventricular tachycardia (VT) during catheter ablation procedures. However, because the majority of VTs are poorly tolerated, substrate modification has emerged as a valuable technique for identifying the abnormal substrate distribution and exit sites of VT in patients with ischemic cardiomyopathy. Such techniques involve a detailed point-by-point acquisition of bipolar voltage information throughout the chamber of interest and display on a 3-dimensional electroanatomic mapping system. Cutoffs for abnormal voltage reflecting damaged myocardial tissue or scar have been established in prior studies and are widely accepted.[1] Once the location of scar is identified, pace-mapping around the scar border can be used to locate the presumed exit site of the induced VT. Then, linear ablation through these exit sites can render the VT noninducible.

Although such substrate modification techniques opened up a new era in the ablation of poorly tolerated VT, the efficacy in completely eliminating recurrent VT has been limited.[1–5] Therefore, other surrogates of slow conduction within scar that might harbor a VT isthmus, such as fractionated and late potentials, are also often empirically targeted for ablation. Such sites are often manually tagged

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on an electroanatomic map during acquisition and then targeted later for ablation.[6,7] The detail of such maps requires laborious acquisition, the labeling of these sites is quite subjective, and cumulative labeling of all fractionated/late signals depends on the number of points acquired and operator diligence. The frustration with such detailed mapping has led some to advocate empiric ablation of all tissue within the scar, a laborious process that may increase the risk of complications and create more slow conduction through incomplete substrate ablation.[8]

Therefore, in addition to display of "activation time" and "voltage" information on an electroanatomic map, we hypothesized that automated display of a "fractionation map" could be useful for guiding catheter ablation. Using this rubric, after acquisition of a sinus rhythm voltage map, a "fractionation map" could be displayed and all abnormal areas targeted for ablation, thus limiting the ablated area to that of abnormal electrograms. Therefore, we studied patents with an postinfarction cardiomyopathy and tolerated entrainable VT, together with a population of patients with idiopathic VT and normal endocardial voltage maps in order to determine (1) cutoffs for normal and abnormal electrogram fractionation, (2) percentage of scar attributed to areas of abnormal electrogram fractionation, and (3) relationship between regions of abnormal fractionation to the VT isthmus in patients with tolerated VT.

Methods

Study population

Detailed left ventricular (LV) electroanatomic data were obtained from 2 groups of patients undergoing ablation of ventricular arrhythmias: (1) patients with a history of remote myocardial infarction and spontaneous VT, and (2) a reference group of patients with structurally normal hearts undergoing ablation of idiopathic ventricular premature depolarizations or VT. All patients provided written informed consent. All of the procedures were clinically indicated, and data collection was approved by the human research committee.

1. Postinfarction cardiomyopathy: Fourteen patients (13 men, mean 67 ± 8 years) with a history of remote myocardial infarction undergoing detailed endocardial LV electroanatomic mapping and catheter ablation of VT in whom at least 1 critical isthmus of a hemodynamically tolerated and mappable monomorphic sustained VT had been accurately identified using entrainment maneuvers were included in the study. In all cases, VT terminated with ablation at the critical isthmus site. Fractionation analysis of the electroanatomic maps was performed offline after the procedure.
2. Reference population: Fourteen patients (10 men, mean age 42 ± 13 years) with structurally normal hearts who underwent detailed endocardial LV mapping for catheter ablation of idiopathic ventricular premature depolarizations/VT and had normal bipolar endocardial voltage served as the reference group to determine the reference value of abnormal bipolar electrogram fractionation. Absence of structural heart disease was confirmed by transthoracic echocardiography, stress testing, and/or coronary angiography.

Endocardial electroanatomic substrate mapping

Patients were studied in the postabsorptive state under conscious sedation or general anesthesia. Detailed maps of the endocardial LV surface were obtained during sinus rhythm before radiofrequency ablation from the 28 patients in the study using an electroanatomic mapping system (CARTO, Biosense Webster, Diamond Bar, CA) and a 3.5-mm open irrigated-tip catheter (NaviStar ThermoCool, Biosense Webster), which contains a 2-mm ring electrode with a 1-mm interelectrode distance. Bipolar electrograms (sampled at 1 kHz and bandpass filtered at 30–500 Hz) were recorded, displayed at 200 mm/s sweep speed, and digitally stored for offline analysis. A detailed assessment of each electrogram was made to exclude premature or paced beats and noisy signals. Valvular sites were identified, and intracavitary and poor contact points were deleted and excluded from the analysis. To ensure adequate sampling density and a complete representation of voltage distribution, a fill threshold $\leq$15 mm was maintained with an emphasis on fully defining the scar and border zone of the infarcted tissue. Normal bipolar endocardial electrogram voltage was defined as a peak-to-peak amplitude $\geq$1.5 mV, and "dense scar" was defined as a peak-to-peak amplitude $<$0.5 mV.[1] Peak-to-peak amplitude was measured automatically. A retrograde transaortic approach was used to access the LV endocardium in all except 2 cases (1 in each group of patients) in whom transseptal access was used to map the LV endocardium.

VT mapping and catheter ablation

Once the endocardial LV bipolar voltage map during sinus rhythm was completed, ventricular programmed stimulation applying up to 3 extrastimuli at $\geq$2 basic cycle lengths was performed. Induced VTs were analyzed for cycle length and morphology using the CardioLab recording system (GE, Houston, TX). If an induced VT was hemodynamically tolerated, standard entrainment maneuvers were performed by pacing from the mapping catheter at a cycle length 20–30 ms faster than the tachycardia cycle length and observing the response to entrainment. A site was considered a VT mid-isthmus only if it demonstrated (1) concealed fusion on all 12 ECG leads during entrainment, (2) postpacing interval within 30 ms of the VT cycle length, (3) stimulus-to-electrogram interval within 30 ms of the electrogram–QRS interval following entrainment, and (4) local electrogram-to-QRS interval between 30% and 70% of the VT cycle length. Confirmed VT isthmus sites were annotated on the map after VT termination in sinus rhythm. Radiofrequency ablation was performed using an open-irrigated ablation catheter (NaviStar ThermoCool) with power of 30–50 W. For the purpose of this analysis, only isthmus sites that resulted in