Noncontact mapping to guide ablation of right ventricular outflow tract arrhythmias

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BACKGROUND There is limited data on outcomes after noncontact mapping (NCM)–guided right ventricular outflow tract (RVOT) ventricular arrhythmia (VA) ablation.

OBJECTIVES To assess outcomes of NCM-guided RVOT VA ablation in a large cohort with extended follow-up, to determine optimal ablation site, and to analyze limitations of conventional mapping techniques.

METHODS In consecutive patients undergoing RVOT VA ablation, 2 sites of early activation—earliest activation (EA) and breakout (BO) sites—were identified on NCM maps. Pace mapping and activation mapping were performed at both sites. The area of depolarized myocardium during the first 10 ms of spontaneous VA and pacing was measured. The initial site of ablation was randomized to either EA or BO sites, with crossover to the alternate site if ablation was not successful.

RESULTS In 136 patients, prematurity of local activation and pace maps were similar at EA and BO sites. More myocardium was depolarized 10 ms after pacing than during spontaneous VA (12.9 ± 7.8 cm^2 vs 5.3 ± 3.9 cm^2; $P < .01$). Clinical success was more likely achieved when initial ablation was directed toward the EA site ($P < .05$). A wider EA-BO separation was associated with acute procedural failure ($P < .01$). With a follow-up of 36.2 ± 17.5 months, the success rate after a single procedure without antiarrhythmic agents was 86.8%.

CONCLUSIONS NCM-guided RVOT VA ablation is highly effective, and clinical success is best achieved by ablating the EA site. Broad regions of early activation are associated with worsened clinical outcomes. Spatial resolution of activation and pace mapping is limited by rapid electrical propagation in the RVOT.

KEYWORDS Right ventricular outflow tract; Ventricular arrhythmias; Noncontact mapping; Pace mapping; Activation mapping; Catheter ablation

ABBREVIATIONS **BO** = breakout; **EA** = earliest activation; **ECG** = electrocardiographic; **MEA** = multielectrode array; **NCM** = noncontact mapping; **PCM** = pace-captured myocardium; **PVCs** = premature ventricular contractions; **RVOT** = right ventricular outflow tract; **SAM** = spontaneous activated myocardium; **VA** = ventricular arrhythmias; **VT** = ventricular tachycardia

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Introduction

Right ventricular outflow tract (RVOT) ventricular arrhythmias (VA) can result in symptoms associated with hemodynamic compromise such as syncope, reversible tachycardia-mediated cardiomyopathy, and trigger electrical storms in vulnerable patients with concomitant channelopathies.[1–3] For medically refractory RVOT VA, catheter ablation is highly effective and often a curative therapy.[2–7] However, conventional mapping and ablation of RVOT VA can be technically challenging, requiring significant fluoroscopic and procedural times.

A noncontact mapping (NCM) system incorporating a multielectrode array (MEA), capable of recording and displaying global electrical activation of any single beat within a single cardiac chamber, has been validated as an accurate tool to guide mapping and ablation of a variety of arrhythmias.[8–10] Hence, the NCM system has been used routinely for several years as part of our local clinical protocol for the mapping and ablation of RVOT VA. To date, the clinical utility of NCM to guide RVOT VA ablation has been reported in small cohorts with limited follow-up. The objectives of this study were severalfold: first, to determine the optimal site of ablation on the basis of NCM data; second, to better understand the limitations of conventional techniques of pace mapping and activation mapping; and finally, to assess the long-term clinical outcomes after

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NCM-guided RVOT VA ablation in a large cohort of more than 130 patients, with mean extended follow-up of more than 3 years.

Methods

Patients

The study was conducted from May 2006 to December 2011. The inclusion criteria included patients with symptomatic ventricular tachycardia (VT) or premature ventricular contractions (PVCs) with left bundle branch block morphology, inferior axis, and precordial lead transition zone $\geq V_3$ undergoing their index procedure. For patients with frequent PVCs, PVCs burden was greater than 20% on 24-hour Holter monitoring.[2] RVOT VAs were refractory to β-blockers and class Ic or III antiarrhythmic agents. Patients with structural heart disease, polymorphic VA, or VA originating outside the RVOT were excluded from this study. A total of 170 consecutive patients with VA electrocardiographic (ECG) morphology fulfilling the inclusion criteria underwent ablation. In these 170 patients, 20 had VA originated from the pulmonary artery trunk origin, 10 had left ventricular outflow tract, and 4 had the junction between the distal great cardiac veins. After exclusion of these patients, a total of 136 patients form the study cohort. The study protocol conforms to the ethical guidelines of the 1975 Declaration of Helsinki as reflected in a prior approval by the institution's human research committee.

Electrophysiological procedure

All antiarrhythmic drugs except amiodarone were discontinued for at least 5 half-lives before the procedure.[11] After written informed consent was obtained, the electrophysiological study was performed in the fasting state under conscious sedation. A quadripolar electrode catheter was positioned in the right ventricular apex. Ablation catheters with 4-mm distal electrode and 2-5-2 mm interelectrode spacing (Cordis Webster Inc, Diamond Bar, CA, or EP Technologies Inc, San Jose, CA) were inserted into the RVOT. Spontaneous VT or PVCs at baseline was marked for analysis. In patients without spontaneous VA, programmed stimulation was performed from 2 right ventricular sites at drive trains of 2 different cycle lengths, with up to 3 extra stimuli, with and without the use of isoproterenol. In addition, incremental burst pacing up to cycle lengths of 280 ms was performed. Systemic heparinization maintained an activated clotting time of 250–300 seconds during the procedure.

Noncontact mapping

The NCM system (Ensite Array, St Jude Medical, St Paul, MN) was deployed, with the MEA placed at the optimal mapping position such that the earliest activation (EA) site of VT/PVCs to the center of MEA (defined as R value) value was not more than 35 mm.[12] The 3-dimensional geometry of the RVOT was designed.

Definitions of landmarks on noncontact activation maps

On each NCM activation map, EA and breakout (BO) sites were marked. The EA site was defined as the site with the earliest unipolar deflection from baseline during spontaneous VA, forming a single spot on the isopotential map, as well as characterized by a QS pattern of noncontact unipolar electrogram (Figure 1). The BO site was marked as the site along the depolarization pathway identified by the color-coded activation map where rapid centrifugal electrical propagation originated from and local unipolar electrograms exhibited the maximum negative dV/dt (Figure 1).[13] For the identification of EA and BO sites, a broad color band setting was used with color high (defined as unipolar electrogram baseline) at −0.1 mV and color low at −2 mv. The virtual unipolar high-pass filter was set to 4 Hz. Local activation at EA and BO sites based on contact bipolar electrograms from the mapping catheter was also recorded. The endocardial area of the spontaneous activated myocardium (SAM) was determined by measuring the area of the RVOT geometry (Figure 2) activated in the first 1, 5, and 10 ms after the onset of EA during VA (Figure 3).

Pace mapping

Bipolar pace mapping was performed at EA and BO sites (Figure 4). Pacing was performed at twice the diastolic threshold and 0.5-ms pulse width, with the pacing cycle length equal to the coupling interval of the PVCs or VT cycle length. The endocardial area of the pace-captured myocardium (PCM) was measured by using the same technique as used for measuring the area of the SAM at 1, 5, and 10 ms after the onset of endocardial activation (Figure 3). The pace-map score (maximum score of 24; a score of 1 was acquired for each morphology and amplitude of the QRS wave in each lead if it matched well to the clinical PVCs) was evaluated by 4 blinded physicians. A score of more than 22 was defined as a match to the clinical PVCs.

Identifying the optimal site of radiofrequency ablation on the basis of NCM data

Preliminary data demonstrated that local activation during VA or degree of ECG match during pace mapping was similar at both BO and EA sites. To prospectively investigate the optimal site for radiofrequency ablation, the site of initial ablation was randomized to either BO or EA sites. If initial ablation was unsuccessful at the EA site, then the radiofrequency ablation target was switched over to the BO site and vice versa. Randomization was performed by using customized computer program.

Ablation was performed by delivering radiofrequency energy in the temperature-control mode, with the power output measured as high as 40 W to achieve a target temperature of 50–60°C for 60 seconds. Ablation was considered to be acutely successful if the VT or PVCs was eliminated during ablation and/or became noninducible with programmed electrical stimulation and use of isoproterenol infusion.