Ablation of ventricular arrhythmia originating at the papillary muscle using an automatic pacemapping module

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BACKGROUND Ventricular arrhythmia originating from the papillary muscle (VA-PM) is characterized by multiple exits and morphologic alternations. The conventional ablation strategy relies on activation mapping, but the results might be suboptimal.

OBJECTIVE The purpose of this study was to propose a novel pacemapping strategy aimed at multiple exits using high-output software as a complementary approach to the conventional strategy.

METHODS A consecutive 13 patients with VA-PM were enrolled in this study. Novel pacemapping based on an automatic matching algorithm and integrated electroanatomic mapping was used to quantify the morphology variation in these patients and to identify the potential exits of VA-PM. Complementary ablation targeting at the best matching site of each morphology was performed.

RESULTS Twelve of 13 patients (92%) experienced morphologic alternation, and a total of 34 morphologies were detected (2.6 ± 1.0 per patient). A total of 23 (68%) morphologies were detected as spontaneous pleomorphic ventricular premature complexes (VPCs) before procedure, and 4 morphologies (12%) were induced under isoproterenol infusion. Another 7 of 34 morphologies (21%) could be found only after radiofrequency ablation attempts. Exits with a high pacemapping correlation index for corresponding morphology would be mapped, so preferential exits could be identified. Mean interexit distance was 15.1 ± 5.9 mm. Acute success rate was 100%. During mean follow-up of 12.2 ± 6.9 months, only 1 case recurred with ventricular tachycardia. Although 3 cases recurred with different VPC morphologies, the VPC burden decreased from 16.3% ± 8.8% to 2.6% ± 1.7%.

CONCLUSION This novel pacemapping strategy could effectively eliminate multiple exits as a complementary approach to the conventional strategy.

KEYWORDS Ventricular arrhythmia; Papillary muscle; Radiofrequency ablation; Pacemapping

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Introduction

Ventricular arrhythmia originating from the papillary muscle (VA-PM) is known to propagate through multiple exits with various morphologies presented.[1] Activation mapping with identification of papillary prepotentials has been established as the conventional approach for catheter

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ablation.[2–9] However, the recurrence rate using the conventional approach remains high at 13%–58%.[2–4,6,10] Current strategy is suboptimal in many ways. First, low burden of arrhythmia may limit activation mapping and identification of papillary prepotential signals. Second, prepotentials may not be identified in all patients and could be difficult to differentiate from fascicular potentials in nearby structures.[2,4,7,8,10,11] Third, multiple morphologies may limit activation and conventional pacemapping.[10,12,13] Furthermore, multiple morphologies with multiple preferential exits could occur before and after ablation and make focal ablation difficult. Thus, the use of traditional pacemapping or activation mapping for patients with VA-PM and multiple morphologies would be impractical, especially when the exits change immediately after the initial ablation attempt.

In this study, we applied pacemapping score maps based on 3-dimensional (3D) mapping with the aim of identifying multiple exits of VA-PM. We hypothesized that the potential multiple exits of VA-PM could be identified, and that additional ablation targeting of multiple exits could serve as a complementary approach to the conventional strategy.

Methods

Patient characteristics

The study population was collected from a cohort of 246 patients (127 men, age 16–80 years) who had undergone catheter ablation for VA between January 2013 and August 2015. The enrollment criteria were as follows: (1) exclusion of substrate VA based on cardiac magnetic resonance imaging study, echocardiography, or coronary angiography; (2) ablation lesions applied along the PM; (3) exclusion of a reentrant mechanism by entrainment; (4) matched ECG morphology for VA-PM according to previously published criteria; and (5) contact mapping and novel pacemapping used during the procedure.[6,14] Thirteen patients (5.2%) who fulfilled the criteria were enrolled. The study was approved by the Institutional Review Board (VGH-IRB No. 2014-10-004BC).

Electrophysiologic study

All patients underwent electrophysiologic study and catheter ablation. Baseline morphologies of ventricular premature complexes (VPCs) or ventricular tachycardia (VTs) were collected before the invasive procedure (Figure 1). Standard multielectrode catheters were placed within the right ventricular apex. Programmed electrical stimulation with 2-ms width and twice diastolic threshold was applied to induce VT until a maximum of 3 extrastimuli were introduced after an 8-beat drive. If VA could not be induced, the same protocol was introduced again under a continuous 4 μg/min isoproterenol infusion, and the catheter was repositioned within the right ventricular outflow tract. All of the electrograms and surface ECGs were recorded using a BARD LabSystem Pro workstation (C.R. Bard, Murray Hill, New Jersey). Systemic anticoagulation with an activated clotting time >250 seconds was achieved with bolus intravenous heparin throughout the entire procedure. The anesthesia protocol is described in the Online Supplementary Material.

Electroanatomic mapping and catheter ablation

Step 1: Left ventricular geometry and activation mapping

The electroanatomic mapping was reconstructed using an irrigating ThermoCool ablation catheter and a CARTO 3.2 with UDM version and PaSo module (Biosense Webster, Diamond Bar, CA) The left ventricle (LV) was approached using a retrograde maneuver via the aorta. The transseptal approach was used in selected patients with aortic valve disease or torturous aorta. Bipolar and unipolar voltage mapping was reconstructed in sinus rhythm (SR). LV substrate mapping was completed when geometry and color-coded voltage mapping was shown. In the presence of VT and frequent monomorphic VPCs, activation mapping was performed after reconstruction of the geometry and SR substrate mapping (Figure 1). Papillary prepotentials were identified based on the description by Yamada et al[10] that (1) a spiky potential was noted before local ventricular activation during VPC, and (2) no Purkinje potentials were observed during SR at the same position. Papillary prepotentials and fascicle potentials were separated based on these EGM criteria and were recorded in the 3D mapping system.[1,6,10]

Intracardiac echocardiography (ICE) was used to examine the relative position of the ablation catheter and PM (SoundStar catheter and CartoSound module; Biosense Webster). The ICE catheter was advanced via the left femoral vein to the right ventricle. The anatomy of the PM was reconstructed accordingly. Both activation mapping and pacemapping were performed and integrated into 3D geometry by the built-in ICE module of the CARTO system. Optimal catheter contact was confirmed with fluoroscopy, the 3D mapping system, and real-time ICE imaging (Figure 2).

Step 2: Pacemapping

Initially the template from clinically documented VPC or VT before the invasive procedure was chosen for pacemapping to minimize the sympathetic tone change related to pacing and the isoproterenol infusion. The potential templates for VA included (1) spontaneously initiating VA, (2) different morphologies after the isoproterenol infusion and programmed electrical stimulation, and (3) the new morphologies emerged after catheter ablation. The selected template should be matched with the clinical VA documented with ECG or 24-hour Holter recording at the outpatient clinic. Pacing was delivered with a 2-ms pulse width and twice diastolic threshold at the pacing cycle length equal to the coupling interval of the VPC or VT. To avoid capturing the nearby myocardium, the pacing output was gradually titrated down if successful myocardium capture was detected. If no local capture was found, the pacing output was titrated up to the maximal pacing output at 20 mA. The pacing output was checked repeatedly to ensure the stimulus strength at the lowest level above the threshold. All of these pacing maneuvers should be completed smoothly to minimize the size of captured myocardium and to avoid misinterpretation of spontaneous or catheter-induced VPCs.[15]