An unmappable ventricular tachycardia in the arrhythmogenic right ventricular cardiomyopathy: elucidation of critical isolated delayed components with high-resolution electroanatomical mapping

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Unmappable ventricular tachycardia (VT) is a challenge in the management of arrhythmogenic right ventricular cardiomyopathy (ARVC). We report a feasible strategy for a curative ablation. In the present case with ARVC, the clinical VT showed a single morphology of left bundle branch block with inferior axis. Neither activation mapping nor entrainment mapping could be done because of instability of the haemodynamics. Furthermore, pace mapping could not be obtained due to electrically unexcitable scars covering with the RV. We found isolated delayed components (IDCs) in the diastolic phase recorded within the scar areas. Electroanatomical mapping (CARTO) with tiered decreasing voltage definition revealed that IDCs were delineated on the narrow conducting channels along or between the complete scars (amplitude <0.1 mV). Isolated delayed components on the narrow channels were targeted under the guidance with CARTO. After 11 radiofrequency applications, the clinical VT was eliminated. Moreover, epsilon waves recorded on the 12-lead electrocardiogram disappeared. No ventricular tachyarrhythmia was recognized at 6-month follow-up.
Isolated delayed component ablation with high-resolution CARTO map was feasible and provided a curative approach in the treatment of an unmappable VT in ARVC.

Introduction
Arrhythmogenic right ventricular cardiomyopathy (ARVC) is characterized by left bundle branch block (LBBB) type ventricular arrhythmia and a specific RV pathology that demonstrates replacement of the myocardium by fatty and fibrous tissues. We present a case of successful treatment of unmappable ventricular tachycardia (VT) in a patient with ARVC solely by ablating isolated delayed components (IDCs) during sinus rhythm.

Case presentation
A 60-year-old female diagnosed with ARVC was admitted to our institution for recurrent syncopal episodes due to monomorphic VT. Since clinical VT was refractory to antiarrhythmics, she was referred to the electrophysiology laboratory for the curative ablation. Clinical VT (Figure 1A, cycle length = 240 ms) showed a single morphology of LBBB with inferior axis that suggested an RV outflow tract (RVOT) origin. Right ventriculogram demonstrated a bizarre aneurysmal formation in the RVOT area (Figure 1B).

Since the unstable VT required external cardioversion for its termination, entrainment mapping or activation mapping failed. Further, pace mapping was not feasible as well because the entire RVOT free wall was unexcitable even at the maximum output (9.9 V, 2.0 ms). With the detailed mapping, we found several IDCs recorded in the aneurysmal free wall of the RV during sinus rhythm. Isolated delayed components had presented fractionation and reversal of sequence on the occasion of ventricular extrasystole or extrastimuli (Figure 1C). We constructed the RV map with CARTO electroanatomical mapping system (Biosense-Webster, Inc. Diamond Bar, CA, USA) during sinus rhythm. The bipolar voltage map was constituted by a total of 151 points taken with a NaviStar catheter (Biosense-Webster, Inc.). During the initial phase of the study, we defined the normal tissue as a bipolar voltage of $\geq 1.5 \text{ mV}$ and the dense scar as a bipolar voltage of $< 0.5 \text{ mV}$ in accordance with the published data for structural heart disease. However, this map failed to provide the useful information for ablation because entire RV chamber was visualized as a single extensive scar (indicated by black arrows) and the yellow dot indicates discrete delayed potentials. The brown dot represents the radiofrequency (RF) application site.

Isolated delayed component ablation with high-resolution CARTO map was feasible and provided a curative approach in the treatment of an unmappable VT in ARVC.

Figure 2 (A) Electroanatomical mapping of the RV chamber (RAO cranial view). When the voltage scar definition was $\leq 0.5 \text{ mV}$, almost the entire RV chamber was covered by the scar. (B and C) The adjusted voltage scar definition ($\leq 0.1 \text{ mV}$ as an entire scar and $0.1–0.5 \text{ mV}$ as a dense scar) revealed the narrow isthmus between the complete scars (‘high-resolution map’). The IDC (indicated by black arrows) were mainly located at the narrow channels or near the edge of the ‘complete’ scar defined by the high-resolution map. The blue dot represents dull or fractionated delayed potentials and the yellow dot indicates discrete delayed potentials. The brown dot represents the radiofrequency (RF) application site.
as a red area in Figure 2A). Subsequently, the colour range was adjusted with a bipolar voltage change of 0.1–0.5 mV. This ‘high-resolution scar map’ revealed the distribution of complete (≤0.1 mV) and dense (0.1–0.5 mV) scar areas (Figure 2B), and also elucidated the localization of IDC around or between the complete scars. A total of 33 points of IDCs were identified on the CARTO map. The mean amplitude of IDC was 0.14 ± 0.09 mV and the mean duration from the onset of QRS to IDC was 173 ± 36.2 ms. Isolated delayed components between the complete scars were targeted and ablated on the basis of the idea that they were presumably on the protected isthmus within the scar. After 11 RF applications (40 W and 50°C for 60 s for each application), most of the IDC as well as epsilon waves on the surface electrocardiogram (ECG) disappeared (Figure 3). Clinical VT turned to be no longer inducible. No ventricular tachyarrhythmia was recorded on her implantable cardioverter-defibrillator at the 6-month follow-up.

Discussion
Isolated delayed components are considered to be an alternative target for unmappable VT. Arenal et al.² defined that IDCs are distinct ventricular electrograms separated by ≥40 ms by an isoelectric interval or very low amplitude signal. Nogami et al.³ reported 89% of freedom from VT recurrence at 12 months by IDC-oriented ablation for VT with ARVC. However, in these reports, pace mapping was indispensable to decide the target IDC. In the present case, the pace mapping was not feasible. A possible explanation is the occurrence of local depolarization without global capture of the myocardium in the RVOT area. Under the circumstances, the differentiation of optimal target electrograms is crucial to the curative ablation. The high-resolution scar map elucidated the distribution of the complete scars and IDC, and facilitated differentiation of the optimal electrograms for ablation from bystander electrograms. In making a high-resolution map, it is critical to adjust the voltage definition. We adopted 0.1 mV as a complete scar definition for clarifying the distribution of viable corridors within the extended dense scar area.⁴ Such adjustments facilitated the curative ablation of unmappable VT.

Another debatable issue is the endpoint of ablation. Since, in ARVC, we considered that the ablative therapy for VT was adjunctive, non-inducibility of clinical VT was considered to be an optimal endpoint. Further, we speculated that the disappearance of epsilon waves reflected the elimination of critical IDCs that are related to the VT. Consequently, our strategy provided successful outcome.

Conflict of interest: none declared.

References