A 2F epicardial electrode–guided ablation from left coronary cusp for substrates of left ventricular summit tachycardia

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Introduction

The summit of the left ventricle (LV) is defined as the most superior potion of LV epicardium bounded by bifurcation between left anterior descending and left circumflex coronary artery.[1] LV summit is bisected laterally by the great cardiac vein (GCV) dividing the triangular area into the superior portion, adjacent to the major coronary arteries, and the inferior portion, which is amenable for epicardial catheter ablation.[2] Epicardial radiofrequency (RF) ablation either inside coronary veins or from the pericardial space using Sosa technique confers potential risk of vein perforation and/or major coronary artery injury.[3] Previous studies have shown that LV summit ventricular tachycardia (VT) exit sites occasionally clustered in close proximity to the communication of the GCV and anterior interventricular vein.[4,5] Development of a 2F multipolar electrode catheter allows epicardial mapping in the LV summit by engaging small tributaries of the coronary veins.[6] Eradication of the epicardial local abnormal ventricular activities (LAVAs) representing scar-reentry VT substrates reduced recurrent VT episodes in the long-term follow-up.[7,8] Komatsu and colleagues[9] first reported that epicardial LAVAs could be modified or eliminated by delivering RF energy transmurally from the endocardial surface of the facing site in ischemic cardiomyopathy and arrhythmogenic right ventricular cardiomyopathy scar–related VT but not in the region of the LV summit.

KEY TEACHING POINTS

- Engaging a 2F electrode catheter in small tributaries of the epicardial coronary veins around the region of the left ventricular summit under coronary sinus venographic guidance is feasible.
- Epicardial or subepicardium local abnormal ventricular activities (LAVAs) can be recorded inside small tributaries of the great cardiac vein
- Left ventricular summit arrhythmias can be terminated and suppressed by radiofrequency energy application above or below the left aortic cusp abutting the facing site of epicardial LAVAs under fluoroscopic guidance.
- Epicardial LAVAs at the left ventricular summit can be modified or eradicated by delivering radiofrequency energy above or below the left aortic cusp.
- Coronary vein mapping in the vicinity of the left ventricular summit serves as a new landmark for the ablation target of the left ventricular summit arrhythmias with a measurable procedural endpoint.

KEYWORDS Ablation; Delayed potential; Epicardium; Left coronary cusp; Left ventricular summit; Ventricular tachycardia

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Case report

A 74-year-old man presented with sustained monomorphic VT (cycle length 414 ms, QRS duration 170 ms) exhibiting right inferior axis, positive concordance in precordial leads, presence of small q wave in lead I with intrinsicoid deflection in V_2 of 160 ms, and maximum deflection index of 0.69, suggestive of epicardial LV summit origin,[10–12] as shown in Figure 1 (patient #1). All VT episodes were successfully terminated by a single burst of antitachycardia pacing from an implantable cardioverter-defibrillator. One year ago, he had the first VT with QRS morphology identical to this

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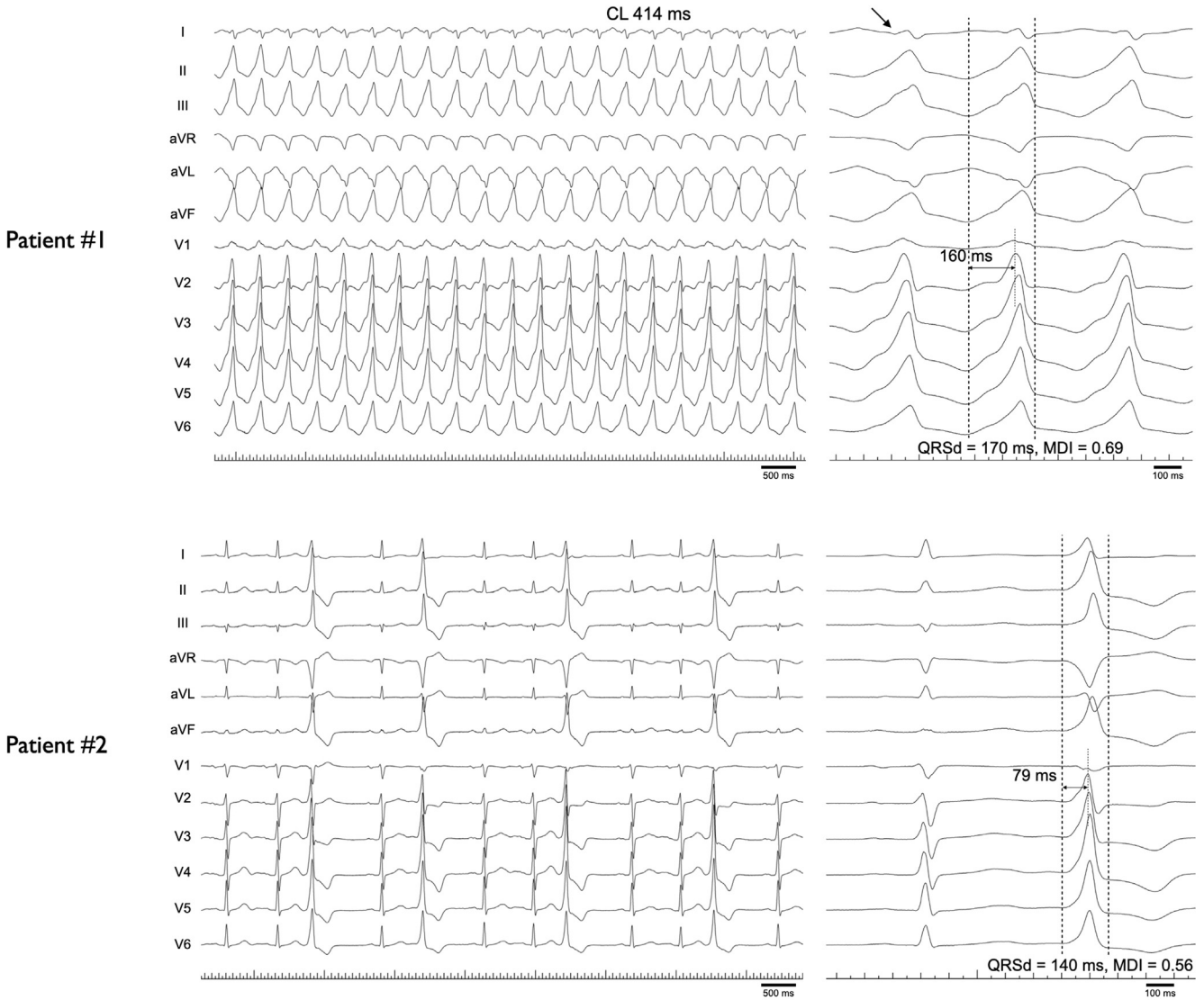

Figure 1 Patient #1: Sustained monomorphic ventricular tachycardia exhibited right bundle branch block–like pattern (monophasic R in V_1), positive concordance, and right inferior axis. Initial q wave was noted in lead I (*arrow*). Cycle length (CL) = 414 ms. QRS duration (QRSd) = 170 ms. Intrinsicoid deflection time in V_2 = 160 ms. Maximum deflection index (MDI) = 0.69. Patient #2: Sinus rhythm with frequent ventricular premature contractions (VPCs) manifesting left bundle branch block–like pattern (qrS complex in V_1, R/S transition in V_2) and inferior axis. QRS duration = 140 ms. Intrinsicoid deflection time in V_2 = 79 ms. MDI = 0.56. The first beat of VPCs was fusion.

episode. Coronary angiogram yielded 90% stenosis of atrioventricular nodal branch (#4AV). Percutaneous coronary intervention with a drug-eluting stent was successfully performed. Transthoracic echocardiogram showed anteroseptal aneurysm and hypokinesia of lateral to posterior wall. Left ventricular ejection fraction was 50%. Cardiac positron emission tomography did not demonstrate area of myocardial inflammation. Endomyocardial biopsy showed no remarkable findings. Electrophysiologic study was performed after discontinuation of all antiarrhythmic drugs for at least 5 half-lives. Coronary angiogram revealed no in-stent restenosis. A 6F decapolar electrode catheter with inner lumen (EPstar CS with lumen; Japan Lifeline, Tokyo, Japan) was engaged into the coronary sinus. Retrograde coronary sinus venography via the inner lumen depicted the GCV and its small tributaries. A 2F catheter with 1.3-mm electrode length, 5-mm interelectrode spacing (EPstar Fix; Japan Lifeline, Tokyo, Japan) was introduced into the lumen of the 6F catheter. Distal electrodes of the 2F catheter were positioned inside a small tributary of the GCV at the epicardial LV summit, searching for early abnormal local potentials during VT (Figure 2, patient #1). LV endocardial bipolar voltage mapping (CARTO 3 Version 6.0; Biosense Webster, Diamond Bar, CA) during sinus rhythm using a multipolar high-density mapping catheter (PentaRay®; Biosense Webster, Diamond Bar, CA) showed a low voltage area at mid anterior wall without LAVAs (Supplemental Figure 1). Clinical VT was induced by programmed ventricular stimulation. During VT, the distal bipolar electrode of the 2F catheter (CSd 1-2) recorded sharp low-amplitude, high-frequency potentials 60 ms earlier than QRS onset. During sinus rhythm, low-voltage late signals were observed on CSd 1-2, clearly

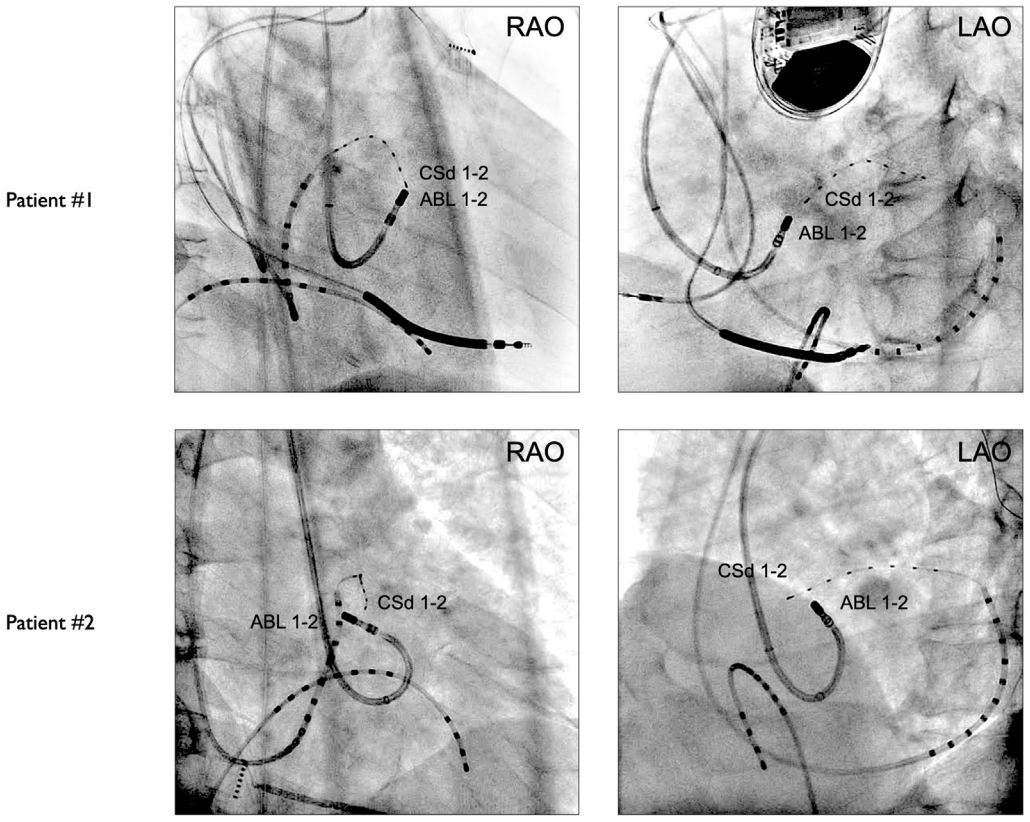

Figure 2 Right and left anterior oblique fluoroscopic views depicting position of 2F catheter in the small tributaries of the great cardiac vein in the vicinity of left ventricular summit and tip of ablation catheter above (patient #1) and under (patient #2) the left coronary cusp. ABL 1-2 = distal bipolar electrode of the ablation catheter; CSd 1-2 = distal bipolar electrode of the 2F catheter.

separated from far-field ventricular potential with a peak-to-peak bipolar amplitude of less than 1.5 mV, meeting criteria of LAVAs originally described by Jaïs and colleagues[7] (Figure 3, patient #1). Pace mapping at the lowest capture threshold (10–16 V @ 1 ms) from CSd 1-2 produced identical QRS morphology to clinical VT with a stimulus-to-QRS interval of 60 ms (Supplemental Figure 2). Left coronary ostium was tagged by CARTOUNIVU™ (Biosense Webster, Diamond Bar, CA) combining fluoroscopic image of left coronary angiogram and 3D mapping system (Supplemental Figure 3). An 8F 3.5-mm irrigated-tip ablation catheter (THERMOCOOL SMARTTOUCH® SF; Biosense Webster, Diamond Bar, CA) was placed above the left aortic cusp by retrograde transaortic approach abutting the epicardial LAVAs site recorded inside the coronary vein (Figure 2, patient #1). Abnormal potentials were not seen at the tip of the ablation catheter and pace mapping with decremental energy output did not produce a good pace map score. RF energy was delivered by using power-controlled mode with temperature limit at 50°C. VT was slowed and terminated. RF ablation was continued for 50 seconds with the second and third RF energy applications for 90 and 64 seconds, respectively. Epicardial LAVAs were delayed and abolished, followed by noninducibility of VT (Figure 3, patient #1). There were no complications. VT had not recurred at 3-month follow-up.

Another case involved a 76-year-old man without structural heart disease presenting with frequent symptomatic ventricular premature contractions (VPCs). Ambulatory electrocardiogram monitoring revealed single morphology of 33,000 VPCs in 24 hours with left bundle branch block–like morphology (qrS pattern in V_1, R/S transition in V_2) (Figure 1, patient #2). During VPCs, CSd 1-2 recorded presystolic potentials 78 ms preceding QRS onset distinct from far-field ventricular signal. LAVAs on CSd 1-2 displayed 2 components during sinus rhythm: initial fractionated signals buried in the far-field ventricular potentials and split late spiky potentials (Figure 3, patient #2). Pacing from CSd 1-2 (15 V @ 1 ms) yielded poor pace map score with very short stimulus-to-QRS interval of 26 ms. When pacing output was decreased to 5 V @ 1 ms, QRS morphology was changed toward that of clinical VPCs with longer stimulus-to-QRS interval of 62 ms. The ablation catheter was positioned below the left coronary cusp using a retrograde transaortic approach facing the LAVAs site on CSd 1-2 under fluoroscopic guidance (Figure 2, patient #2). There were no LAVAs during sinus rhythm or presystolic potentials during VPCs on the tip of the ablation catheter. However, pace mapping (10 V @ 1 ms) rendered QRS morphology resembling that of clinical VPCs with stimulus-to-QRS interval of 58 ms. RF energy application under the left cusp immediately suppressed VPCs. After

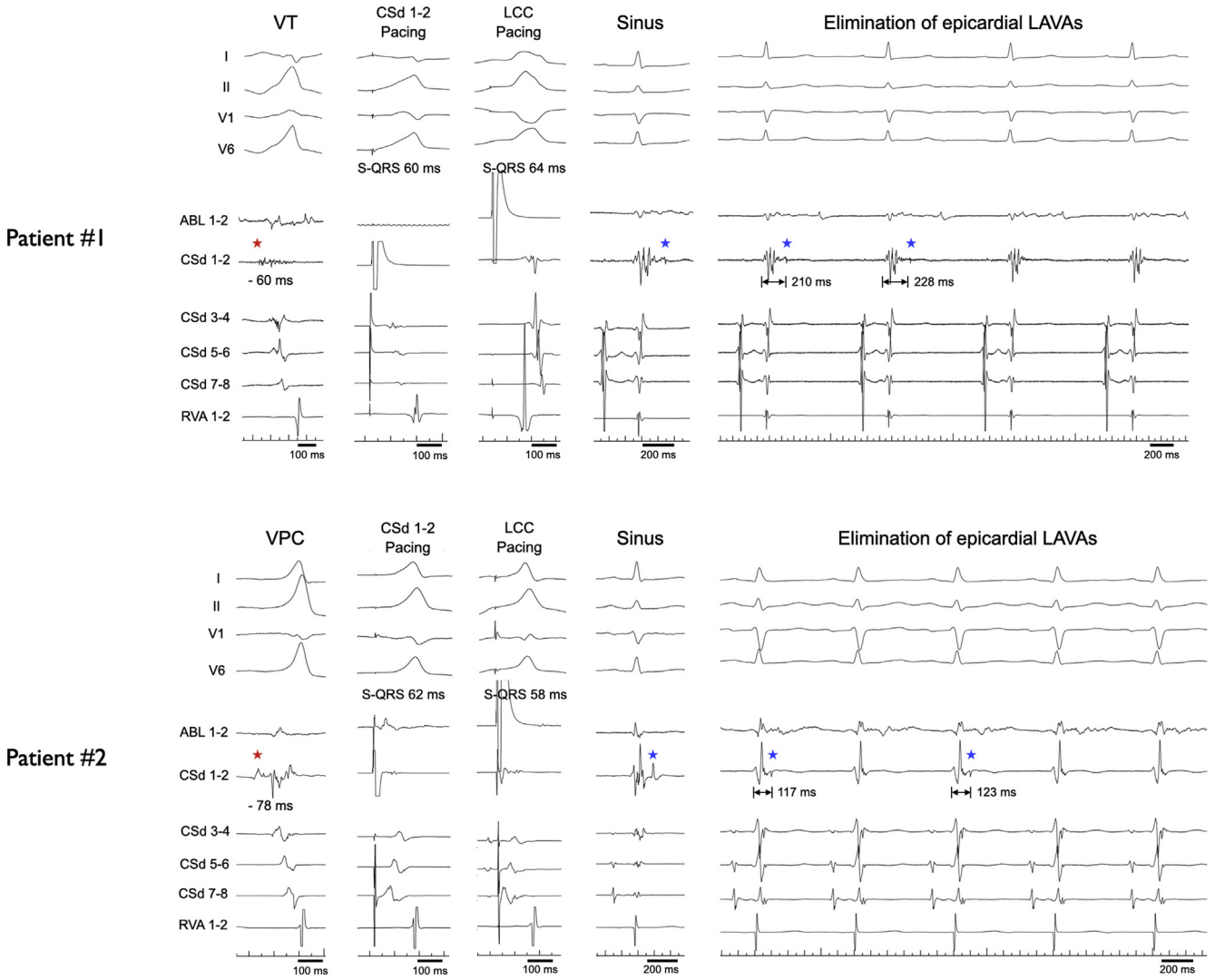

Figure 3 Patient #1: Epicardial local abnormal ventricular activities (LAVAs; *red star*) displayed at CSd 1-2 as presystolic potentials 60 ms preceding QRS onset distinct from far-field ventricular signal during ventricular tachycardia and split late potentials (*blue star*) during sinus rhythm. Pace mapping from CSd 1-2 yielded good pace map score with S-QRS identical to LAVAs-to-QRS interval. LAVAs were not seen at the tip of ablation catheter and pace mapping did not produce a good pace map score. Epicardial LAVAs were delayed and then abolished after radiofrequency (RF) ablations. Patient #2: Epicardial LAVAs (*red star*) displayed at CSd 1-2 as presystolic potentials 78 ms preceding QRS onset during ventricular premature contractions (VPCs) distinct from the far-field ventricular signals. Late potentials during sinus rhythm had 2 components: initial fractionated signals buried in far-field ventricular potentials and split late spiky signals. LAVAs were not seen at the tip of the ablation catheter. Pace mapping from CSd 1-2 and left coronary cusp (LCC) produced similar, but not identical, QRS configuration to VPCs. After RF ablations, late potentials became 2:1, delayed, and completely eradicated. ABL = ablation catheter; CSd = distal coronary sinus catheter; RVA = right ventricular apex; S-QRS = stimulus-to-QRS interval.

repeat ablation, initial fractionation disappeared. Eventually, late potentials became 2:1, delayed, and completely eradicated after successive attempts of RF energy application under the left cusp (Figure 3, patient #2). There were no spontaneous or inducible clinical VPCs after RF application. At 2-year follow-up, VPCs had not recurred.

Discussion

We reported that epicardial LAVAs could be recorded in small tributaries of the GCV with a 2F microcatheter in ischemic cardiomyopathy (patient #1) and structurally normal heart (patient #2), and served as a novel landmark for ablation target with a measurable endpoint in LV summit VT ablation. In patient #1, LAVAs on CSd 1-2 were completely distinct from far-field ventricular signals. Local pacing from CSd 1-2 selectively captured LAVAs and gave off nearly identical QRS morphology to the clinical VT. The ablation catheter above the left cusp facing opposite to the LAVAs site did not record LAVAs and failed to render an excellent pace map score. LAVAs represented an arrhythmogenic substrate confined to the epicardial region of the LV summit. While we did not perform tachycardia entrainment or other specific pacing maneuvers to elucidate the exact mechanism of VT, our speculation was scar reentry based on the following observations: (1) LAVAs normally

represented survival cell potentials surrounded by myocardial scar; (2) selective LAVAs capture produced excellent pace map score, with stimulus-to-QRS identical to electrogram-to-QRS interval; and (3) all VT episodes were successfully terminated by a single burst of antitachycardia pacing from an implantable cardioverter-defibrillator prior to mapping and ablation. In patient #2, LAVAs were the earliest potentials preceding QRS onset of the clinical VPCs and displayed as 2 components during sinus rhythm. Decremental output pacing from CSd 1-2 selectively captured LAVAs and rendered QRS morphology almost similar to VPCs. The ablation catheter under the left cusp in close approximation to CSd 1-2 did not record LAVAs or presystolic potentials, but pacing from this site produced nearly identical QRS morphology of clinical VPCs. Arrhythmogenic substrate might reside in the subepicardial or intramural region of the LV summit. However, there was no definite proof of the mechanism of VPCs. Our ablation strategy was to position the tip of the ablation catheter endocardially to the closest anatomical approximation to the epicardial LAVAs site and deliver RF energy transmurally. In both cases, VT and VPCs were totally suppressed. Repeat ablation at the left cusp delayed and completely abolished LAVAs during sinus rhythm, followed by noninducibility of VT and VPCs.

Komatsu and colleagues[6] targeted the ablation site of the LV summit VT by using the earliest local abnormal potentials inside the communicating vein between aortic and pulmonary annuli as a site of VT origin. Earliest signals were not LAVAs and all 31 patients had no structural heart disease. Liao and colleagues[13] recently proposed "abrupt transition in V3" (ATV3) as a specific electrocardiogram pattern for VT originating from the septal margin of the LV summit. Most of the patients with ATV3 pattern in their study had earliest activation and successful ablation sites at the right-left interleaflet triangle. However, some of them had the earliest potentials recorded in the GCV or anterior interventricular vein by using a customized guidewire-tip duodecapolar catheter and left coronary cusp ablation successfully terminated and suppressed LV summit VT. On the contrary, VT in our first patient had monophasic R in V_1 and VPCs in the second patient had a left bundle branch block–like pattern with qrS in V_1 and R/S transition in V_2 without ATV3 pattern. Successful ablation sites were above and below the left coronary cusp lateral to the right-left interleaflet triangle. Anatomical proximity of the left coronary semilunar cusp insertion site and subepicardial or intramural site of origin possibly dictates successful ablation. We proved that epicardial or subepicardial substrate recorded by a 2F catheter inside epicardial coronary veins in the region of the LV summit could be modified and eradicated transmurally by RF energy application above and below the left coronary cusp.

Conclusion

Mapping epicardial LV summit substrates using a 2F catheter engaging into the small tributaries of the GCV is feasible. RF energy application above and below the left coronary cusp on the facing site of recorded epicardial LAVAs terminated ventricular arrhythmias and completely eradicated the substrates. Coronary vein mapping in the vicinity of the LV summit serves as a new landmark for the ablation target with a measurable procedural endpoint.

Appendix

Supplementary data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrcr.2021.10.011.

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