Idiopathic Ventricular Arrhythmias Originating From the Vicinity of the Communicating Vein of Cardiac Venous Systems at the Left Ventricular Summit

See Editorial by Muser and Santangeli

Yuki Komatsu, MD
Akihiko Nogami, MD
Yasutoshi Shinoda, MD
Keita Masuda, MD
Takeshi Machino, MD
Kenji Kuroki, MD
Hiro Yamasaki, MD
Yukio Sekiguchi, MD
Kazutaka Aonuma, MD

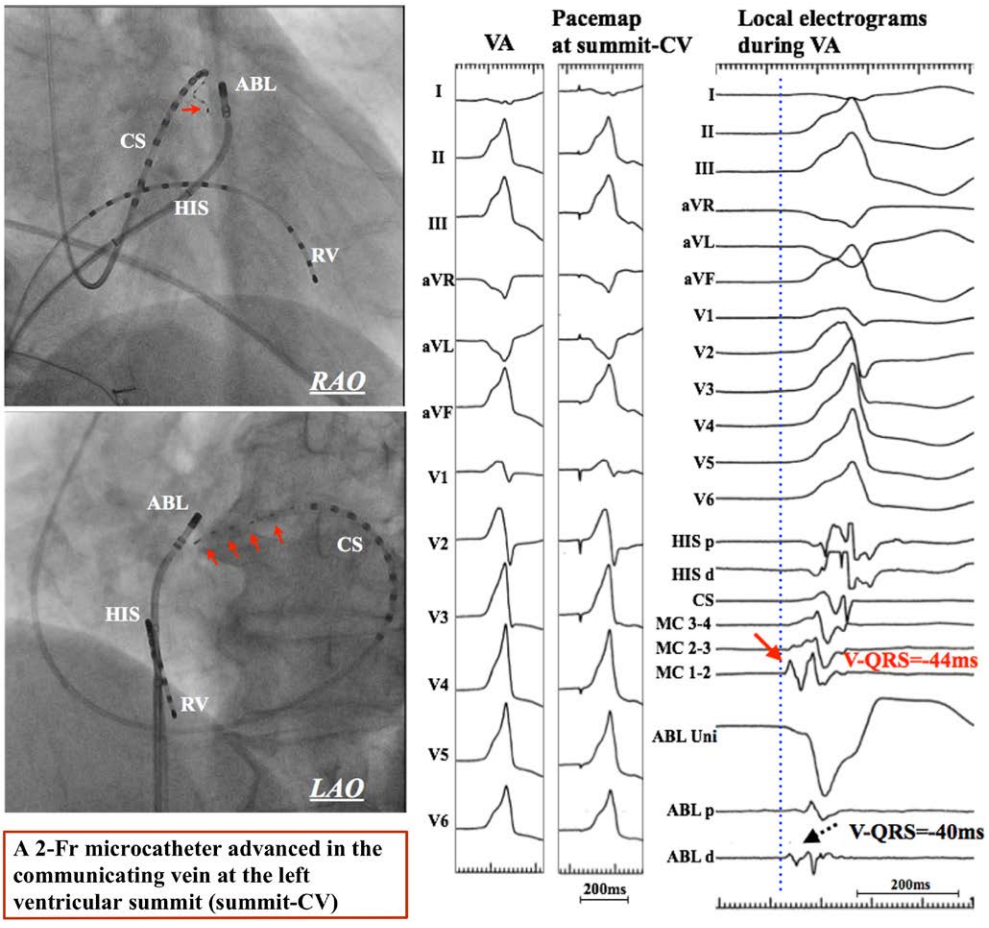
BACKGROUND: The communicating vein (CV) between the great cardiac vein and small cardiac venous systems passes between the aortic and pulmonary annulus and is located in close association with the left ventricular summit (summit CV).

METHODS AND RESULTS: Thirty-one patients with idiopathic ventricular arrhythmias (VAs) underwent mapping of the left ventricular summit by using a 2F microcatheter introduced into the summit CV with coronary sinus venographic guidance. Of these, 14 patients were found to have summit-CV VAs. The remaining 17 patients (control group) had VAs originating from the right ventricular outflow tract and the aortic cusps. In patients with summit-CV VAs, the earliest activation during VAs in the summit CV preceded QRS onset by 34.1±5.3 ms. The summit-CV VAs exhibited inferior axis, negative polarity in lead I, deeper QS wave in lead aVL than aVR, and nonspecific bundle branch block morphology with an R/S ratio in lead V_1 of 0.67±0.33, which could be distinguishable from VAs originating from the right ventricular outflow tract and the right coronary cusp. Because of the inaccessibility of the summit CV to ablation catheter, ablation of summit-CV VAs was attempted at adjacent structures where an excellent pacemap was rarely obtained. Overall ablation success was achieved in 10 (71%) patients with summit VAs and 15 (88%) patients in control group (P=0.24).

CONCLUSIONS: The myocardium near the summit CV can be the source of idiopathic VAs. Direct monitoring of the summit CV is helpful for identifying the site of origin and provides a landmark of the ablation target, which may facilitate ablation through adjacent structures.

Correspondence to: Yuki Komatsu, MD, Department of Cardiology, University of Tsukuba, 1-1-1 Tennodai, Tsukuba 305–8575, Japan. E-mail yk.komat@gmail.com

Key Words: catheter ablation ■ coronary sinus ■ electrocardiography ■ electrophysiology ■ ventricular arrhythmia



A 2-Fr microcatheter advanced in the communicating vein at the left ventricular summit (summit-CV)

WHAT IS KNOWN?

- Catheter ablation of idiopathic ventricular arrhythmias originating from the summit of the left ventricle is often challenging.
- The communicating vein between the great cardiac vein and small cardiac venous systems passes between the aortic and pulmonary annulus and is located in close association with the left ventricular summit.

WHAT THE STUDY ADDS?

- The myocardium in the vicinity of the communicating branch at the left ventricular summit can be the source of idiopathic ventricular arrhythmias.
- Mapping within the communicating vein by using a 2F microcatheter with coronary sinus venographic guidance is feasible and helpful for identifying the site of origin.
- Direct monitoring of the local electrogram in the communicating vein serves as a landmark of the ablation target, which may facilitate ablation of the targeted arrhythmia through adjacent structures.

Radiofrequency catheter ablation is a widely accepted therapy for idiopathic ventricular arrhythmias (VAs). However, ablation of VAs originating from the summit of the left ventricle (LV) can be challenging because of the possibility of an intramural or subepicardial site of origin that is inaccessible to an ablation catheter.[1–5] Moreover, the close proximity of the coronary vessels and overlying epicardial fat may preclude effective ablation through a percutaneous epicardial approach to the site of these arrhythmias.[2]

McAlpine[6] described that the LV summit is the epicardial LV outflow region bounded by the bifurcation between the left anterior descending artery and left circumflex artery and transected by the great cardiac vein (GCV) at its junction with the anterior interventricular vein. The communicating veins (CVs) between the GCV and conus branch that drains to the small cardiac vein have great individual variability. However, there may be a distinct CV that is located between the aortic and pulmonary annulus. Because the CV anterior to the aortic annulus but posterior to the pulmonary annulus is located in close association with the superior portion of the LV summit (summit CV),[5–7] detailed mapping inside the CV has

the potential to localize the precise site of origin of the challenging VAs for ablation. This study was undertaken to evaluate the ECG and electrophysiological features and ablation outcome of VAs originating from the vicinity of the CV at the superior portion of LV summit (summit-CV VAs) by using a 2F microcatheter introduced into the summit CV with coronary sinus venographic guidance.

METHODS

The data and methods will be made available to other researchers from the corresponding author on reasonable request.

Study Population

The study patients were drawn from 229 consecutive patients who underwent catheter ablation for idiopathic VAs between January 2013 and August 2016 at our institution. This study evaluated 31 patients (22 men; age, 59±13 years) who underwent mapping of the LV summit by using a 2F microcatheter (1.3-mm electrode with 5-mm interelectrode spacing; EPstar Fix 2F; Japan Lifeline, Tokyo, Japan) introduced into the summit CV. Of these, 14 patients were found to have summit-CV VAs. The site of origin was confirmed to be near the summit CV when the mapping catheter positioned in the summit CV showed the earliest activation time during arrhythmia with an excellent matching pacemap (≥11/12 leads) obtained by pacing at the location. A control group consisted of the remaining 17 patients with VAs originating from the right ventricular outflow tract (RVOT; n=7) and the aortic cusps (n=10). In these 17 patients, the earliest activation site during VAs was not found at the summit CV but at the RVOT or aortic cusps. The site of origin in the aortic root was confirmed by the fluoroscopic views with aortogram and intracardiac echocardiography (SoundStar; Biosense Webster, Diamond Bar, CA). There was no evidence of structural heart disease based on echocardiography, coronary angiography, or cardiac magnetic resonance imaging. All patients signed a written informed consent of catheter ablation. The institutional review board approved review of these data.

Electrophysiological Study

An electrophysiological study was performed after discontinuation of all antiarrhythmic drugs. Patients presented to the cardiac electrophysiology laboratory in the fasting state. Multipolar electrode catheters were placed at the His-bundle region and the right ventricular apex. A 6F decapolar catheter with inner lumen (4-mm interelectrode spacing; Inquiry LumaCath Fixed Diagnostic Catheter; St. Jude Medical, Saint Paul, MN) was inserted from the internal jugular vein or the subclavian vein and placed in the coronary sinus. Small branches of the coronary venous system were visualized by retrograde venography through the inner lumen of the decapolar catheter placed in the coronary sinus (Figure 1). After retrograde venography, a 2F microcatheter was introduced through the lumen of the 6F decapolar catheter and advanced into the CV between the aortic and pulmonary annuli (Figure 2). An irrigated-tip mapping/ablation catheter (NAVISTAR Thermocool; Biosense Webster) was advanced to the right ventricle and coronary venous system via transvenous approach and to the LV via retrograde aortic approach. The RVOT, LV endocardium, aortic cusp, and distal portion of the GCV were mapped with the ablation catheter to identify the earliest site of ventricular activation during VAs. Pacemapping was performed at a pacing cycle length equal to the coupling interval of the spontaneous VAs. In all patients, a 3-dimensional electroanatomical

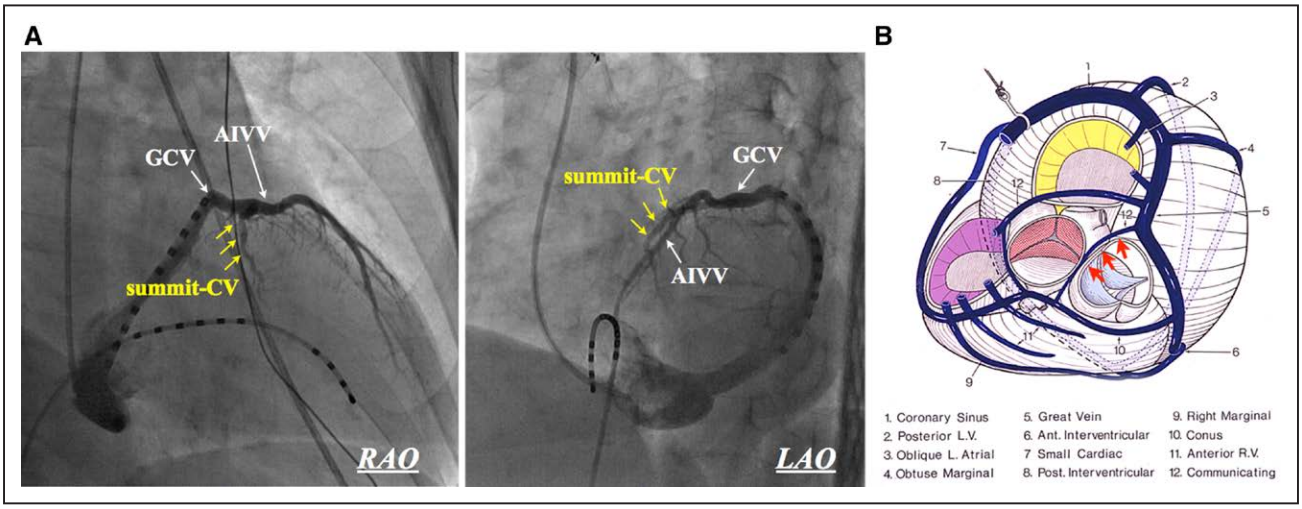


Figure 1. Coronary venography depicting the communicating vein (CV) at the left ventricular (LV) summit. **A**, Coronary venography by retrograde injection through the inner lumen of the decapolar catheter placed in the coronary sinus allows clear visualization of the coronary venous architecture and depicts a small distinct CV that is located between the aortic and pulmonary annuli. **B**, The CV between the great cardiac vein (GCV) and conus branch are typically comprised of branch posterior to the aortic annulus near the noncoronary sinus of Valsalva and branch between the aortic and pulmonary annulus. The CV between the aortic and pulmonary annulus (red arrows) is located in close association with the superior portion of the LV summit (summit CV). Cover illustration courtesy of the UCLA Cardiac Arrhythmia Center, Wallace A. McAlpine MD collection, reproduced with permission. Copyright ©, UCLA Cardiac Arrhythmia Center. AIVV indicates anterior interventricular vein; LAO, left anterior oblique; RAO, right anterior oblique; and RV, right ventricle.

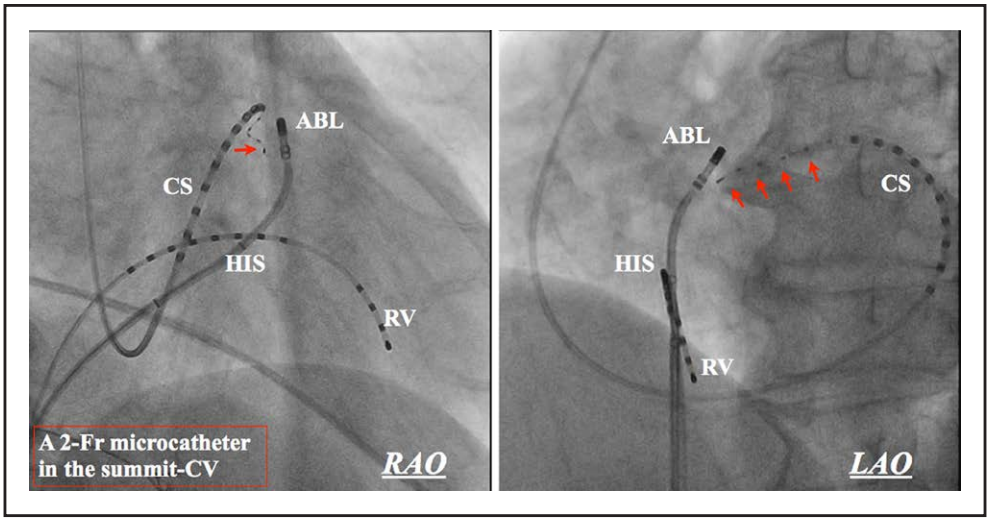


Figure 2. A 2F microcatheter placed in the communicating vein (CV).
After visualization of the small branches of the coronary venous system by retrograde venography, a 2F microcatheter (EPstar Fix 2F; Japan Lifeline, Tokyo, Japan) is introduced through the lumen of a 6F decapolar catheter and advanced into the CV to allow direct monitoring of the CV. The 2F microcatheter in the CV was used as a landmark of the ablation sites in the nearby endocardial structures. ABL indicates ablation catheter; CS, coronary sinus; HIS, His bundle; and RV, right ventricle.

mapping system was used for activation mapping (CARTO; Biosense Webster, Diamond Bar, CA). If few clinical VAs were observed at the beginning of the electrophysiological study, burst pacing from the right ventricular apex or right atrium was performed with the addition of an intravenous isoproterenol (3.0–5.0 μg/min) or epinephrine (5–10 μg) infusion to provoke the clinical arrhythmia.

Radiofrequency Ablation

Radiofrequency energy was delivered using an irrigated-tip catheter with a maximum temperature of 42°C and a maximum power of 40 W, targeting an impedance drop of 10 to 15 Ω. When performing ablation in the coronary venous system, power exceeding 25 W was avoided. Coronary angiography was performed before and after ablation to delineate the ostium and the course of the left and right coronary artery. Ablation was performed first at the site of earliest activation. If the site of earliest activation was the summit CV and was not reached by the ablation catheter, ablation was attempted at adjacent sites in the closest proximity to the earliest activation site. The 2F microcatheter placed into the vein was used as a landmark of the facing ablation sites. After ablation, atrial and ventricular burst pacing with and without intravenous isoproterenol followed by epinephrine was performed to assess arrhythmia inducibility. Acute success was defined as the absence of any targeted arrhythmia at the end of the procedure.

Postablation Management

Patients were monitored for at least 24 hours in hospital before discharge. No antiarrhythmic drugs were prescribed after the ablation procedure. Patients were followed through regular clinic visits for the first month and every 3 months thereafter. A 24-hour Holter recording was obtained at approximately yearly intervals. Arrhythmia recurrence was assessed by patient interview, 12-lead ECG, and Holter recording.

ECG Analysis

Twelve-lead ECG was recorded digitally at a sweep speed of 100 mm/s for offline analysis. The 12-lead ECG findings were analyzed according to the previously reported measurements applied to the ventricular outflow tract regions.[8,9] The QRS duration was measured as the interval between the earliest onset of the QRS complex in any lead to the latest offset in any lead on the 12-lead ECG. The duration of pseudodelta wave (the interval from the onset of QRS complex to the earliest fast deflection in any precordial lead) and the intrinsicoid deflection time (the interval from the QRS onset to the maximum deflection in any of the precordial leads) were measured. The maximum deflection index was calculated by dividing the intrinsicoid deflection time by the QRS duration.[8] R–S in lead I was measured by subtracting S-wave amplitude from R-wave amplitude in lead I.

Statistics

Categorical variables were expressed as numbers and percentages. Continuous data were expressed as mean±SD. Procedural outcomes in patients with summit-CV VAs were compared with those in control group. Categorical variables were compared using the Pearson χ^2 test or the Fisher exact test, as appropriate. Continuous data were compared using the independent sample Student *t* test. A probability value of <0.05 was considered statistically significant. Statistical calculations were performed with SPSS software (SPSS, version 21.0; IBM, Chicago, IL).

RESULTS

Patient Characteristics

Patient characteristics are shown in Table 1. There were no significant differences in clinical characteristics, including age, sex, type of clinical symptoms, nature of

Table 1. Patient Characteristics

	All (N=31)	Summit-CV VAs (n=14)	Control (n=17)
Age, y	59±13	54±14	63±11
Men/women	22 (71%)	12 (86%)	10 (59%)
Clinical symptoms			
Palpitations	10 (32%)	5 (36%)	5 (29%)
Presyncope	3 (10%)	1 (7%)	2 (12%)
Fatigue	12 (39%)	7 (50%)	5 (29%)
Asymptomatic	6 (19%)	1 (7%)	5 (29%)
Ventricular arrhythmia burden, %	21±13	23±13	18±13
Nonsustained ventricular tachycardia	14 (45%)	7 (50%)	7 (41%)
LV ejection fraction, %	60±15	59±13	62±17
LV ejection fraction <50%	6 (19%)	3 (21%)	3 (18%)
BNP, pg/mL	45.6±47.6	42.3±46.0	48.2±50.2
Medications			
β-Blockers	25 (81%)	12 (86%)	13 (76%)
Antiarrhythmic drug	16 (52%)	7 (50%)	9 (53%)

Data are presented as mean±SD or as n (%). BNP indicates brain natriuretic peptide; CV, communicating vein; LV, left ventricle; and VA, ventricular arrhythmia.

clinical arrhythmia, LV ejection fraction, VA burden, or brain natriuretic peptide.

Mapping and Ablation Outcome

Mapping and ablation outcomes are shown in Table 2. In patients with summit-CV VAs, the local ventricular activation in the summit-CV during targeted arrhythmias preceded the QRS onset by 34.1±5.3 ms. Ablation of summit-CV VAs was attempted at adjacent structures (ie, the RVOT, LV endocardium, aortic cusps, or GCV) because of the inaccessibility of the small vein to the ablation catheter (Figure 3). The earliest activation site where ablation was performed among the adjacent structures preceded the QRS onset by 22.3±7.6 ms. Pacemap 12-lead ECG at ablation sites matched the targeted VA morphology in only 2 patients (14%) with summit-CV VAs.

Table 2. Procedural Outcomes

Variables	Summit-CV VAs (n=14)	Control (n=17)	*P* Value
Activation time during VA, ms			
CV in the LV summit	−34.1±5.3	−7.2±7.2	<0.001
Earliest ablation site	−22.3±7.6	−28.4±5.5	0.015
Excellent pacemap from ablation site	2 (14%)	12 (71%)	0.002
Procedure time, min	267±70	212±71	0.040
Total radiofrequency time, min	31±13	16±15	0.010
Complications	1 (7%)	0 (0%)	0.53
Acute success	8 (57%)	15 (88%)	0.060
No recurrence during follow-up	10 (71%)	15 (88%)	0.24

Data are presented as mean±SD or as n (%). CV indicates communicating vein; LV, left ventricle; and VA, ventricular arrhythmia.

In control patients, the local activation in the CV also preceded the QRS onset by 7.2±7.2 ms. In patients with VAs originating from the aortic cusp, the earliest activation site was the right coronary cusp in 4 patients and the left coronary cusp (LCC) in 6 patients, as confirmed by the fluoroscopic views with aortogram and intracardiac echocardiography. In patients with VAs originating from the RVOT, the earliest activation site where the ablation successfully eliminated the targeted arrhythmias was the RVOT posteroseptum in 6 patients and the RVOT anteroseptum in 1 patient.

Procedure duration and radiofrequency energy delivery time in patients with summit-CV VAs were significantly longer than in control patients. Acute success was achieved in 8 patients (57%) with summit-CV VAs. Tamponade occurred in 1 patient and was managed successfully by percutaneous pericardiocentesis without further fluid reaccumulation. Venography of the coronary sinus showed no perforation of the coronary venous system. Coronary angiography after ablation showed no coronary artery stenosis.

ECG Characteristics

Figure 4 shows the QRS morphology of summit-CV VAs. All had an inferior axis with monomorphic R pattern in inferior leads and exhibited nonspecific bundle branch block morphology (neither right nor left bundle branch block morphology). All summit-CV VAs displayed a QS pattern in leads aVR and aVL with a Q-wave ratio in lead aVL/aVR of 1.33±0.42. The precordial R-wave transition occurred earlier than lead V_3 in 12 (86%) patients. All summit-CV VAs had an R wave in lead V_1 with an R-wave/S-wave ratio of 0.67±0.33. An S wave in leads V_5 and V_6 was absent in all patients.

ECG characteristics of summit-CV VAs, aortic cusps (the left and right coronary cusps), and RVOT are summarized in Table I in the Data Supplement. Several ECG characteristics differentiated summit-CV VAs from VAs originating from the RVOT and right coronary cusp. The R-wave amplitude ratio in lead III/II, Q-wave amplitude ratio in leads aVL/aVR, and ratio of R wave/S wave in lead V_1 were higher in summit-CV VAs than in the RVOT foci and the right coronary cusp foci. On the contrary, ECG characteristics in summit-CV VAs were similar to those in VAs originating from the LCC. However, the presence of an initial Q wave in lead I (summit-CV VAs in 5/14 [36%] versus LCC foci in 0/6 [0%]), an initial R wave in lead V_1 (summit-CV VAs in 14/14 [100%] versus LCC foci in 3/6 [50%]), and a notch on the descending limb in lead

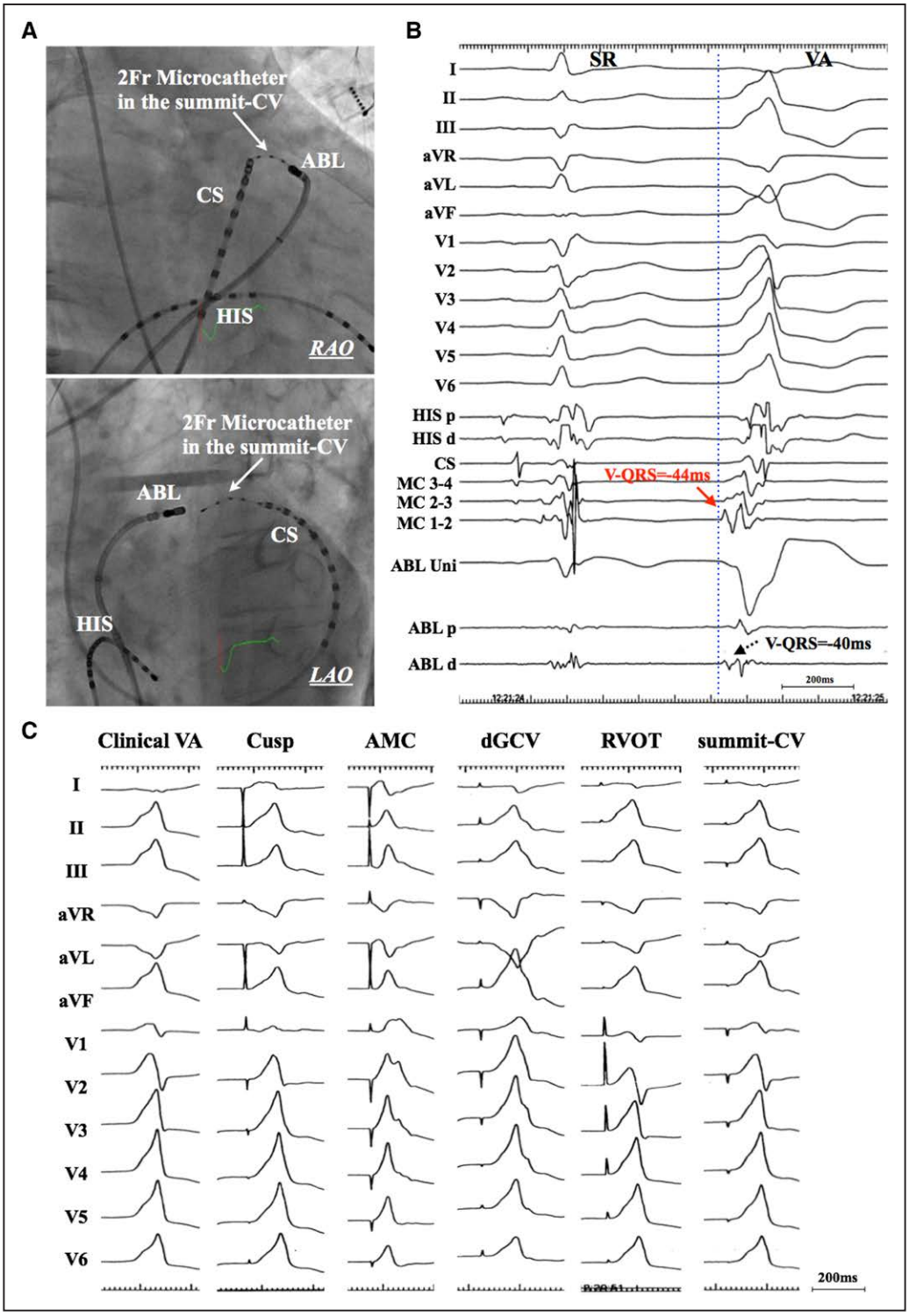


Figure 3. A case of successful summit–communicating vein (CV) ventricular arrhythmic (VA) ablation from the right ventricular outflow tract (RVOT; patient No. 7).

A, A 2F microcatheter was advanced into the CV at the left ventricular summit (summit CV). The ablation catheter was positioned at the RVOT posteroseptum that was the facing the microcatheter in the CV. **B**, The local ventricular activation time recorded at the summit-CV preceded the onset of the QRS complex by 44 ms. The local activation recorded by the ablation catheter was not sharp, however, and preceded the QRS onset by 40 ms. The interval between the activation time at the summit CV and the ablation site was only 4 ms. Ablation at this site eliminated the target arrhythmia. **C**, A perfect pacemap was obtained at the summit CV. The ECG pacemap obtained in the RVOT was similar to that of clinical VA but not identical (with slightly different morphology in lead I). AMC indicates aortomitral continuity; dGCV, distal great cardiac vein; and MC, microcatheter.

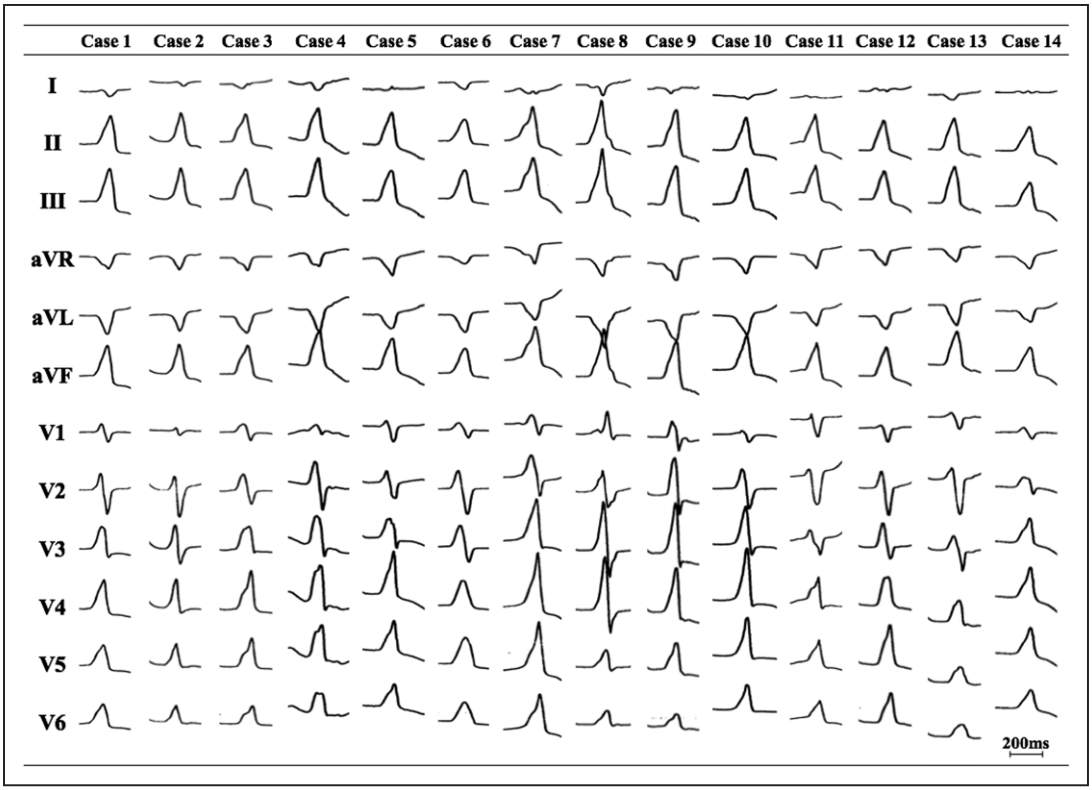


Figure 4. **Twelve-lead ECG of summit–communicating vein ventricular arrhythmias.**

V_1 (summit-CV VAs in 0/14 [0%] versus LCC foci in 2/6 [33%]) differentiated summit-CV VAs from the LCC foci.

Comparison Between Successful and Unsuccessful Ablation of Summit-CV VAs

ECG characteristics in patients who achieved successful elimination of summit-CV VAs were similar to those in patients who failed to eliminate summit-CV VAs (Table II in the Data Supplement). The earliest activation site among the adjacent structures was the RVOT posteroseptum in 7 patients, the aortic cusp in 4 patients, the LV endocardium in 2 patients, and the distal GCV in 1 patient (Table 3). The distribution of the earliest activation site was also similar between groups. Among ablation sites, the earliest activation preceded the surface QRS onset by 22.5±7.8 ms. The earliest local activation time at the adjacent sites in the successful group preceded the surface QRS onset by 26.1±7.1 ms. Of note, the activation interval from the summit CV to the adjacent ablation sites was shorter in the successful group compared with the unsuccessful group (7.6±3.3 ms in the successful group versus 17.3±5.0 ms in the unsuccessful group).

Follow-Up

In 2 patients with summit-CV VAs who failed to achieve acute success, the delayed efficacy of ablation was observed. The targeted VAs disappeared in the day after the procedure in 1 patient. One patient had a reduction in the VA burden on 24-hour Holter monitoring (from 15.3% to 1.1%) during follow-up and has remained off antiarrhythmic drug therapy. Overall, 10 patients (71%) with summit-CV VAs and 15 patients (88%) in control group were free from recurrent VAs

Table 3. **Mapping Data in Patients With Summit-CV Ventricular Arrhythmias**

Variables	Overall (N=14)	Acute Success (n=8)	Acute Failure (n=6)
Activation time, ms			
CV in the LV summit	−33.9±5.5	−33.8±4.7	−34.5±6.4
Earliest ablation site	−22.5±7.8	−26.1±7.1	−17.2±5.0
Interval between summit-CV and the earliest ablation site	11.5±6.6	7.6±3.3	17.3±5.7
Earliest ablation site			
RVOT	7 (50%)	3 (38%)	4 (66%)
Aortic cusp	4 (29%)	4 (50%)	0 (0%)
LV endocardium	2 (14%)	1 (12%)	1 (17%)
Distal GCV	1 (7%)	0 (0%)	1 (17%)
Procedure time, min	267±70	239±56	304±74
Total radiofrequency time, min	31±13	29±16	34±9

Data are presented as mean±SD or as n (%). CV indicates communicating vein; GCV, great cardiac vein; LV, left ventricle; and RVOT, right ventricular outflow tract.

during a mean follow-up of 12 months (P=0.24). Among 4 patients (29%) who experienced recurrence of summit-CV VAs, 3 were successfully managed with antiarrhythmic drugs, including amiodarone, and redo procedures were planned for one.

DISCUSSION

Main Findings

This is the first study demonstrating the ECG and electrophysiological features of idiopathic VAs originating from the vicinity of the communicating branch at the LV summit as assessed by a 2F microcatheter allowing direct monitoring of the specific region at the LV summit. The summit-CV VAs are characterized as follows:

1. ECG of summit-CV VAs was characterized as an inferior axis with monomorphic R pattern in all inferior leads, a negative polarity in lead I, a QS pattern in leads aVR and aVL with a Q-wave ratio in lead aVL/aVR of 1.33±0.42, nonspecific bundle branch block morphology with an R/S ratio in lead V_1 of 0.67±0.33, and no S wave in leads V_5 and V_6.
2. Because of the inaccessibility of the CV to the ablation catheter, the ablation attempt from adjacent structures is performed at the earliest activation site or closest site to the microcatheter in the CV.
3. An excellent pacemap by pacing at the adjacent ablation sites is rarely obtained.
4. Although the elimination of summit-CV VAs is challenging, a shorter activation interval between the summit-CV and ablation sites during the targeted arrhythmia may be associated with higher success rate.

Diagnosis of Summit-CV VAs

The myocardium near the communicating branch at the superior portion of the LV summit can be a source of idiopathic VAs. However, these specific regions are usually inaccessible to an ablation catheter. A 2F microcatheter can be introduced into the vein under the guidance of coronary venography, which allows direct monitoring of the inaccessible regions. In previous reports, the diagnosis of VAs arising from the LV summit or the intramural LV outflow tract was mainly based on the ablation response.[10,11] This study demonstrates that direct monitoring of the summit CV can provide additional electrophysiological information that aids in the diagnosis of VAs arising from these regions. A recent report demonstrated a similar concept, whereby a straight 0.14-inch wire placed in the septal perforator vein provides activation information in the small branches, which efficiently directs the next mapping efforts accordingly.[3]

ECG Characteristics of Summit-CV VAs

The summit-CV VAs have several ECG features, including nonspecific bundle branch block morphology with initial R wave in lead V_1, transition zone, negative polarity in lead I, and QS pattern in leads aVL and aVR with more negative polarity in lead aVL than aVR. These ECG features can differentiate summit-CV VAs from the RVOT foci and the rightward cuspal VAs. However, these ECG features are also observed in idiopathic VAs originating from the leftward aortic cusps. The presence of an initial Q wave in lead I and an initial R wave without a notch in lead V_1 may differentiate summit-CV VAs from the leftward cuspal VAs. The summit-CV VAs did not exhibit an R-wave pattern break in lead V_2, suggesting that summit-CV VAs can be distinguished from the site of origin at the LV epicardium close to the anterior interventricular sulcus.[12]

In comparison with previous reports, the Q-wave amplitude ratio in aVL/aVR in summit-CV VAs was comparable with that in patients with VAs from the intramural origin between the aortomitral continuity and the distal portion of the GCV[10] and that in patients with unsuccessful ablation through a percutaneous epicardial approach.[2] These observations suggest that the summit-CV VAs in our study cohort may be originating from rather the intramural foci than the subepicardial origin at the LV summit.

The CV anterior to the aortic annulus may be confused with the coronary venous septal perforator vein. Although the septal perforator vein is a small branch of the anterior interventricular vein and runs into the intraventricular septum, the CV is located distal to the transitional area between the GCV and anterior interventricular vein and passes between the aortic and pulmonary annuli.[5–7] A previous report on intramural VAs,[13] wherein the site of origin was confirmed by using a microcatheter in the septal perforator vein, demonstrated that the subset of VAs arising in the intraventricular septum exhibited a Q wave in lead V_1. On the contrary, all summit-CV VAs in this study cohort exhibited an initial R wave in lead V_1. Figure 5 shows the QRS morphology during pacemapping in the septal perforator vein. The pacemap obtained in the septal perforator vein exhibited no R wave in lead V_1 and monophasic R wave in lead I. These ECG characteristics were not observed during pacemapping at the summit CV.

Ablation of Summit-CV VAs

A single previous study reported 3 cases of idiopathic VAs originating from near the summit CV that were successfully eliminated by radiofrequency application directly at the extended tributary of distal GCV.[7] Presumably, these previously reported cases involved VAs originating from the proximal portion of the communicating branch near the GCV-anterior interventricular vein

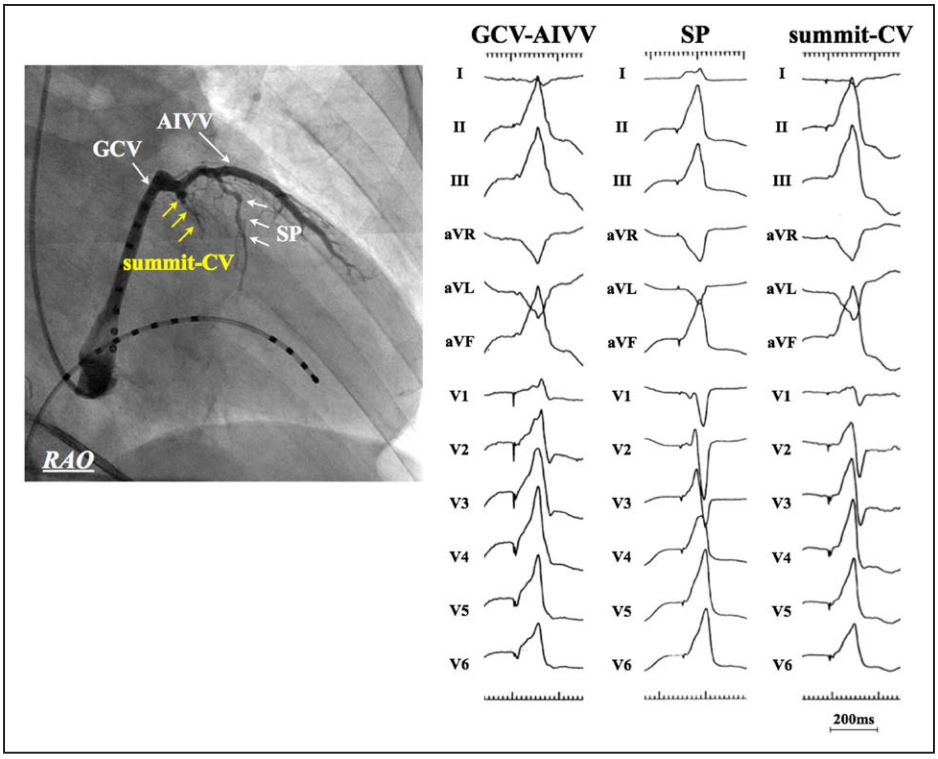


Figure 5. Representative ECG morphologies obtained by pacemap in the great cardiac vein (GCV)–anterior interventricular vein (AIVV), septal perforator vein (SP), and summit communicating vein (CV).
Coronary venography and schema depict the summit CV, which is located distal to the transitional area between the GCV and the AIVV and passes between the aortic and pulmonary annulus. The SP is a small branch of the AIVV and runs into the intraventricular septum. Pacemap obtained at the junction between the GCV and the AIVV exhibited right bundle branch block configuration with wide QRS duration and negative polarity in lead I. The pacemap obtained in the SP exhibited left bundle branch block configuration with no R wave in lead V_1 and relatively narrow QRS duration. In addition, it had monophasic R wave in lead I. These ECG characteristics of the SP pacemap indicate the septal intramural foci. The pacemap obtained in the summit CV exhibited nonspecific bundle branch block morphology with an RS wave in leads V_1 and V_2.

junction. In this study, it was not possible to advance a standard irrigated-tip ablation catheter to the summit CV, and ablation was attempted at adjacent structures.

Because the earliest local electrograms are not always sharp, high-frequency signals probably because of the nonsuperficial endocardial location of the arrhythmia origin, careful mapping at the regions adjacent to the communicating branch is needed. Mapping of the vein by using a 2F microcatheter has the potential to serve as a landmark to the target of radiofrequency ablation and facilitate successful ablation. A first ablation attempt is performed from the RVOT, LV endocardium, or aortic cusps, whichever is earliest or closest to the microcatheter in the communicating branch.

The elimination of summit-CV VAs requires ablation at multiple sites at adjacent structures. In this study, detailed mapping indicated that the earliest activation during arrhythmia was most frequently recorded at the RVOT posteroseptum. However, it is difficult to determine whether radiofrequency delivery at the RVOT was likely to be most effective. In fact, a subset of patients in whom the targeted arrhythmia was not eliminated during the ablation procedure experienced a significant reduction in the arrhythmia burden without antiarrhythmic drugs at the postablation follow-up. In these patients, effective ablation sites cannot be determined. Thus, uncertainties remain regarding the appropriate procedural end point when arrhythmias cannot be completely suppressed. A recent report demonstrated that delayed efficacy in eliminating VAs originating from the LV outflow tract could be observed after moderate ablation from the endocardial structures.[14] When multiple radiofrequency applications elicit positive responses, such as decreased arrhythmia frequency, apparent change in VA morphology, and prolongation of coupling interval, the need for further extensive ablation should be carefully considered.

A short activation interval between the summit CV and adjacent ablation sites during the targeted arrhythmia may be associated with successful ablation, which suggests that a closer anatomic relation between the summit CV and the region accessible to the ablation catheter may be associated with a higher success rate. This finding is consistent with a previous study reporting that the successful ablation of VAs originating from the distal GCV may be associated with a short GCV–non-GCV interval.[4]

Study Limitations

This study involved a retrospective descriptive case series with a small number of patients. The comparison of procedural outcomes between summit CV and control group was limited by small sample size.

The precise prevalence of summit-CV VA is unknown because of the following reasons. First, not a few patients who were referred to our institution during the study period had previously failed ablation. Second, because the microcatheter was used at the discretion of the operator during the study period, there might have been unrecognized cases in which VAs were eliminated by ablation at the left or right outflow tract alone without monitoring electrograms in the summit CV. Third, there is a possibility of geographic variation.

In this study, no patient developed coronary artery stenosis as confirmed by coronary angiography after radiofrequency ablation. However, delayed presentation of coronary artery injury was not examined. The possibility of mild stenosis that did not aggravate symptom cannot be excluded, although no patient presented with chest pain or ischemic ECG changes during follow-up.

Conclusions

The myocardium near the communicating branch at the LV summit can be the source of idiopathic VAs. Mapping within the small coronary venous systems by using a microcatheter is feasible and helpful for identifying the site of origin. Although ablation of arrhythmia originating from these regions is often challenging, direct monitoring of the local electrogram recorded in the CV may serve as a landmark of the ablation target, which has the potential to facilitate ablation of the targeted arrhythmia through adjacent structures.

ACKNOWLEDGMENTS

We thank Kalyanam Shivkumar, MD, PhD, UCLA Cardiac Arrhythmia Center, Los Angeles, CA, for his helpful discussion.

DISCLOSURES

Dr Nogami has received honoraria from St. Jude Medical and an endowment from Medtronic and Johnson & Johnson. Dr Sekiguchi has received an endowment from St. Jude Medical. The other authors report no conflicts.

AFFILIATIONS

From the Department of Cardiology, University of Tsukuba, Japan.

FOOTNOTES

Received April 24, 2017; accepted November 28, 2017.

The Data Supplement is available at http://circep.ahajournals.org/lookup/suppl/doi:10.1161/CIRCEP.117.005386/-/DC1.

Circ Arrhythm Electrophysiol is available at http://circep.ahajournals.org.

REFERENCES

1. Yamada T, McElderry HT, Doppalapudi H, Okada T, Murakami Y, Yoshida Y, Yoshida N, Inden Y, Murohara T, Plumb VJ, Kay GN. Idiopathic ventricular arrhythmias originating from the left ventricular summit: anatomic concepts relevant to ablation. *Circ Arrhythm Electrophysiol*. 2010;3:616–623. doi: 10.1161/CIRCEP.110.939744.
2. Santangeli P, Marchlinski FE, Zado ES, Benhayon D, Hutchinson MD, Lin D, Frankel DS, Riley MP, Supple GE, Garcia FC, Bala R, Desjardins B, Callans DJ, Dixit S. Percutaneous epicardial ablation of ventricular arrhythmias arising from the left ventricular summit: outcomes and electrocardiogram correlates of success. *Circ Arrhythm Electrophysiol*. 2015;8:337–343. doi: 10.1161/CIRCEP.114.002377.
3. Enriquez A, Malavassi F, Saenz LC, Supple G, Santangeli P, Marchlinski FE, Garcia FC. How to map and ablate left ventricular summit arrhythmias. *Heart Rhythm*. 2017;14:141–148. doi: 10.1016/j.hrthm.2016.09.018.
4. Nagashima K, Choi EK, Lin KY, Kumar S, Tedrow UB, Koplan BA, Michaud GF, John RM, Epstein LM, Tokuda M, Inada K, Couper GS, Stevenson WG. Ventricular arrhythmias near the distal great cardiac vein: challenging arrhythmia for ablation. *Circ Arrhythm Electrophysiol*. 2014;7:906–912. doi: 10.1161/CIRCEP.114.001615.
5. Li YC, Lin JF, Li J, Ji KT, Lin JX. Catheter ablation of idiopathic ventricular arrhythmias originating from left ventricular epicardium adjacent to the transitional area from the great cardiac vein to the anterior interventricular vein. *Int J Cardiol*. 2013;167:2673–2681. doi: 10.1016/j.ijcard.2012.06.119.
6. McAlpine WA. *Heart and Coronary Arteries*. New York, NY: Springer-Verlag; 1975.
7. Chen YH, Lin JF. Catheter ablation of idiopathic epicardial ventricular arrhythmias originating from the vicinity of the coronary sinus system. *J Cardiovasc Electrophysiol*. 2015;26:1160–1167. doi: 10.1111/jce.12756.
8. Daniels DV, Lu YY, Morton JB, Santucci PA, Akar JG, Green A, Wilber DJ. Idiopathic epicardial left ventricular tachycardia originating remote from the sinus of Valsalva: electrophysiological characteristics, catheter ablation, and identification from the 12-lead electrocardiogram. *Circulation*. 2006;113:1659–1666. doi: 10.1161/CIRCULATIONAHA.105.611640.
9. Berruezo A, Mont L, Nava S, Chueca E, Bartholomay E, Brugada J. Electrocardiographic recognition of the epicardial origin of ventricular tachycardias. *Circulation*. 2004;109:1842–1847. doi: 10.1161/01.CIR.0000125525.04081.4B.
10. Yamada T, Doppalapudi H, Maddox WR, McElderry HT, Plumb VJ, Kay GN. Prevalence and electrocardiographic and electrophysiological characteristics of idiopathic ventricular arrhythmias originating from intramural foci in the left ventricular outflow tract. *Circ Arrhythm Electrophysiol*. 2016;9:e004079. doi: 10.1161/CIRCEP.116.004079.
11. Yamada T, Doppalapudi H, Litovsky SH, McElderry HT, Kay GN. Challenging radiofrequency catheter ablation of idiopathic ventricular arrhythmias originating from the left ventricular summit near the left main coronary artery. *Circ Arrhythm Electrophysiol*. 2016;9.e004202.
12. Hayashi T, Santangeli P, Pathak RK, Muser D, Liang JJ, Castro SA, Garcia FC, Hutchinson MD, Supple GE, Frankel DS, Riley MP, Lin D, Schaller RD, Dixit S, Callans DJ, Zado ES, Marchlinski FE. Outcomes of catheter ablation of idiopathic outflow tract ventricular arrhythmias with an R wave pattern break in lead V2: a distinct clinical entity. *J Cardiovasc Electrophysiol*. 2017;28:504–514. doi: 10.1111/jce.13183.
13. Yokokawa M, Good E, Chugh A, Pelosi F Jr, Crawford T, Jongnarangsin K, Latchamsetty R, Oral H, Morady F, Bogun F. Intramural idiopathic ventricular arrhythmias originating in the intraventricular septum: mapping and ablation. *Circ Arrhythm Electrophysiol*. 2012;5:258–263. doi: 10.1161/CIRCEP.111.967257.
14. Ding L, Hou B, Wu L, Qiao Y, Sun W, Guo J, Zheng L, Chen G, Zhang L, Zhang S, Yao Y. Delayed efficacy of radiofrequency catheter ablation on ventricular arrhythmias originating from the left ventricular anterobasal wall. *Heart Rhythm*. 2017;14:341–349. doi: 10.1016/j.hrthm.2016.12.006.

Idiopathic Ventricular Arrhythmias Originating From the Vicinity of the Communicating Vein of Cardiac Venous Systems at the Left Ventricular Summit

Yuki Komatsu, Akihiko Nogami, Yasutoshi Shinoda, Keita Masuda, Takeshi Machino, Kenji Kuroki, Hiro Yamasaki, Yukio Sekiguchi and Kazutaka Aonuma

Circ Arrhythm Electrophysiol. 2018;11:
doi: 10.1161/CIRCEP.117.005386
Circulation: Arrhythmia and Electrophysiology is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231

Print ISSN: 1941-3149. Online ISSN: 1941-3084

SUPPLEMENTAL MATERIAL

Supplemental Table 1. ECG characteristics

Variable	Summit-CV (n=14)	LCC (n=6)	RCC (n=4)	RVOT (n=7)
Duration, ms				
QRS duration	150±13	148±11	148±15	146±8
IDT	82±11	76±17	70±9	69±11
MDI	0.54±0.06	0.51±0.08	0.47±0.08	0.46±0.05
PdW	49±12	44±18	45±9	28±8
Limb leads				
Initial Q in lead I	5 (36%)	0 (0%)	0 (0%)	0 (0%)
R - S in lead I, ms	-0.11±0.25	-0.13±0.27	0.26±0.22	0.13±0.20
Mean R amplitude in inferior leads, mV	2.09±0.31	2.27±0.41	1.57±0.32	1.85±0.64
Ratio R in III/II	1.09±0.12	1.12±0.10	0.92±0.15	0.94±0.06
Q in aVL, mV	1.17±0.32	1.36±0.22	0.68±0.30	0.82±0.40
Q in aVR, mV	0.92±0.23	1.08±0.22	0.85±0.18	0.97±0.32
Ratio Q in aVL/aVR	1.33±0.42	1.30±0.27	0.85±0.44	0.82±0.16
Precordial transition				
≤V1	2 (14%)	1 (17%)	0 (0%)	0 (0%)
V1< and ≤V2	3 (21%)	2 (33%)	0 (0%)	0 (0%)
V2< and ≤V3	7 (50%)	2 (33%)	4 (100%)	1 (14%)
V3< and ≤V4	2 (14%)	1 (17%)	0 (0%)	6 (86%)
Precordial leads				
Initial R in lead V1	14 (100%)	3 (50%)	3 (75%)	5 (71%)
Notching in lead V1	0 (0%)	2 (33%)	1 (25%)	0 (0%)
Ratio R/S in V1	0.67±0.33	0.63±0.40	0.30±0.24	0.09±0.05
Ratio R/S in V2	0.73±0.34	0.88±0.46	0.61±0.34	0.14±0.06

Data are presented as mean±SD or as n (%).

Supplemental Table 2. ECG characteristics of successful and unsuccessful ablation of summit-CV VAs

Variable	Acute success (N=8)	Acute failure (N=6)
Duration, ms		
QRS duration	152±15	148±9
IDT	81±12	82±11
MDI	0.53±0.06	0.55±0.05
PdW	54±11	42±12
Limb leads		
Initial Q in lead I	2 (25%)	3 (50%)
R - S in lead I, ms	-0.06±0.33	-0.18±0.07
Mean R amplitude in inferior leads, mV	2.17±0.36	1.99±0.20
Ratio R in III/II	1.08±0.15	1.10±0.07
Q in aVL, mV	1.26±0.39	1.05±0.13
Q in aVR, mV	1.01±0.22	0.80±0.19
Ratio Q in aVL/aVR	1.31±0.51	1.37±0.30
Precordial transition		
≤V1	1 (13%)	1 (17%)
V1< and ≤V2	2 (25%)	1 (17%)
V2< and ≤V3	4 (50%)	3 (50%)
V3< and ≤V4	1 (13%)	1 (17%)
Precordial leads		
Ratio R/S in V1	0.73±0.38	0.59±0.25
Ratio R/S in V2	0.80±0.42	0.64±0.17
Ratio R/S in V3	1.91±1.31	1.74±0.61

Data are presented as mean±SD or as n (%).