Ventricular arrhythmias from the coronary venous system: Prevalence, mapping, and ablation

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BACKGROUND The coronary venous system (CVS) is linked to the origin of idiopathic epicardial ventricular arrhythmias (VAs).

OBJECTIVE The purpose of this study was to identify the prevalence and effective mapping/ablation strategies for idiopathic VAs mapped to the CVS.

METHODS Detailed activation and pace-mapping of the right ventricle (RV), left ventricle (LV), CVS, and aortic cusps was performed, followed by attempted catheter ablation.

RESULTS Forty-seven of 511 patients with non–scar-related VAs (21 males, age 55 ± 15) had earliest activation in the CVS, 39 ± 18 ms before QRS. Twenty-five (53%) were in the great cardiac vein, 19 (40%) in the anterior interventricular vein, and 3 (7%) in the middle cardiac vein. We ablated inside CVS in 32 patients (68%) at the earliest activation site, in 18 patients at an adjacent CVS site, and in 14 patients because of an inability to advance the catheter in 4, inadequate power delivery in 2, and for safer distance from the coronary artery in 8. Proximity to coronaries precluded ablation inside the CVS in the remaining 15 patients (32%), who underwent ablation from adjacent left sinus of Valsalva, RV or LV endocardium, or LV epicardium. Success was achieved in 17 of 18 (94%) ablated at the earliest CVS site and in 16 of 29 (55%) ablated at adjacent CVS or non-CVS sites.

CONCLUSION Idiopathic VAs are occasionally (9%) linked to CVS. Although ablation at the earliest CVS site is effective, it is often (62%) precluded, mainly because of proximity to coronary arteries. Ablation at adjacent CVS and non-CVS sites can be successful in 55% of these anatomically challenging cases, for an overall ablation success rate of 70%.

KEYWORDS Ventricular tachycardia; Ventricular premature depolarization; Coronary venous system; Catheter ablation

ABBREVIATIONS **AIV** = anterior interventricular vein; **CS** = coronary sinus; **CVS** = coronary venous system; **GCV** = great cardiac vein; **LV** = left ventricle; **MCV** = middle cardiac vein; **RF** = radiofrequency; **RV** = right ventricle; **RVOT** = right ventricular outflow tract; **VA** = ventricular arrhythmia; **VPD** = ventricular premature depolarization; **VT** = ventricular tachycardia

(Heart Rhythm 2015;12:1145–1153)

Introduction

Recently there have been increasing reports of idiopathic ventricular arrhythmias (VAs) with earliest activation and successful ablation inside the branches of the coronary sinus (CS).[1–10] Whether the branches of the CS represent the origin of these arrhythmias or simply convenient access to the epicardium is unclear. We sought to examine the prevalence, strategies, limitations, and outcomes of catheter mapping and ablation of VAs with earliest activation inside the coronary venous system (CVS).

This study was funded in part by the Susan and Murray Bloom Research Fund. **Address reprint requests and correspondence:** Dr. Francis E. Marchlinski, Division of Cardiovascular Electrophysiology, Hospital of the University of Pennsylvania, 3400 Spruce St, Founder 9, Philadelphia, PA 19104. E-mail address: francis.marchlinski@uphs.upenn.edu.

Methods

Study population

We retrospectively analyzed 47 consecutive patients with frequent symptomatic ventricular premature depolarizations (VPDs; >7500 per 24 hours) or sustained VAs with site of earliest activation inside the branches of the CS after detailed activation mapping of the adjacent endocardium of the right ventricle (RV) and left ventricle (LV) as well as the aortic sinuses of Valsalva.

Electrophysiologic procedure

After providing written informed consent, patients were brought to the electrophysiology laboratory in the postabsorptive, nonsedated state in accordance with the University of Pennsylvania Health System's institutional guidelines. All data were prospectively entered in a database that was

1547-5271/$-see front matter http://dx.doi.org/10.1016/j.hrthm.2015.03.009

approved by the Institutional Review Board. Antiarrhythmic drugs were discontinued before the procedure.

Surface ECG leads were placed in the standard positions. Bipolar electrograms were recorded with a bandpass filter at 30 to 500 Hz using a 3.5-mm open irrigated-tip ablation catheter (ThermoCool, Biosense Webster, Diamond Bar, CA). Electrograms recorded on the ablation catheter were evaluated at 5000 or 10,000 gain (Prucka, Cardiolab, GE Healthcare, Waukesha, WI). A 6Fr quadripolar catheter was placed in the RV apex for pacing. An 8Fr intracardiac echocardiography catheter (AcuNav, Siemens Medical, Mountain View, CA) was advanced to the RV to guide catheter positioning, assess the distance from the coronary vasculature, monitor lesion formation, and assess for complications.

Mapping the branches of the CS

For patients with inferiorly directed VAs, a detailed activation map was initially performed in the right ventricular outflow tract (RVOT) using a 3-dimentional mapping system (CARTO, Biosense Webster; or NavX, St. Jude Medical, St. Paul, MN). Activation times were measured from the onset of the electrogram on the distant bipole on the ablation catheter to the earliest QRS onset on the surface ECG.

For cases in which earliest activation was <15 ms before the earliest onset of the QRS complex from the RVOT, activation mapping was also performed in the LV outflow tract and sinuses of Valsalva. Intravenous heparin was administered to maintain an activated clotting time >250 seconds during aortic cusp and/or LV endocardial mapping. If activation times in the aortic cusp region were not sufficiently early (by at least 15 ms) or if ablation in the RVOT and/or coronary cusps failed to eliminated the VAs, then additional mapping was performed in the great cardiac vein (GCV) and anterior interventricular vein (AIV). Mapping in the CS branches was also performed if the VA morphology was suggestive of an epicardial origin.[4,6,10,11] A 6Fr decapolar catheter (Bard Electrophysiology, Lowell, MA) was initially used to map the coronary veins, and if the catheter could not be successfully advanced, the 6Fr catheter was exchanged for a 4Fr decapolar catheter (St. Jude Medical). An occlusive venogram was performed in selected patients to facilitate positioning of the decapolar catheter in the branches of the CS. In addition, in selected cases a long sheath was used with the tip of the sheath at the CS ostium to facilitate manipulation of the mapping catheter inside the CVS. The decision to obtain epicardial access was based on operator discretion once ablation inside the coronary venous branches or adjacent endocardial sites could not be performed or was unsuccessful. The decision to map the middle cardiac vein (MCV) was based on basal, superiorly directed VA morphology with late activation from both the RV and LV endocardium. Pace-maps were performed from all locations at the lowest possible output that could achieve capture (starting at pacing amplitude of 1 V and pulse duration of 0.5 ms and gradually decreasing or increasing pacing amplitude until threshold output was reached) and were evaluated qualitatively by at least 2 observers in real time and offline.

Anatomic definitions

Coronary venous branches were defined using the right anterior oblique projection (Figure 1). The GCV was defined as the portion of the coronary vein traveling in the atrioventricular groove and the AIV as the portion traveling in the interventricular septum, anterior to the atrioventricular groove. Finally, the MCV was defined as the coronary venous branch traveling along the posterior interventricular groove.

Coronary angiography

Coronary angiography was performed before ablation in the CS branches or epicardium in all cases to assess the distance from the site of earliest activation to the coronary arteries. Ablation was not performed at sites within 10 mm of the closest major coronary artery. Coronary angiography was not repeated after the procedure unless it was clinically indicated based on symptoms or ECG changes.

Ablation

Ablation inside the CVS was performed with an irrigated catheter. Typical starting power was 20 W. If the VA terminated/suppressed, radiofrequency (RF) delivery was continued for a total of 60 seconds, gradually increasing

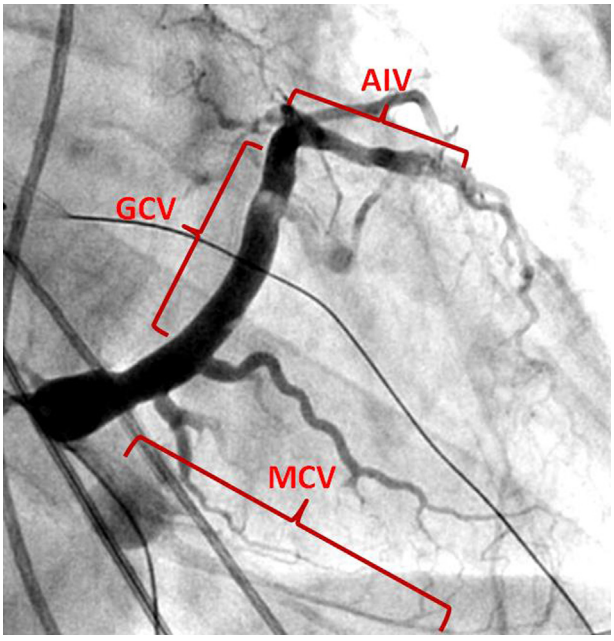


Figure 1 Coronary sinus venous system (CVS). Branches of the CVS were defined using the right anterior oblique projection. The great cardiac vein (GCV) is the portion of the CVS traveling on the atrioventricular groove. The anterior interventricular vein (AIV) is the CVS branch that leaves the atrioventricular groove to course anteriorly on the epicardial surface of the interventricular septum. The middle cardiac vein (MCV) can take off the proximal coronary sinus or have an independent ostium and travels along the posterior interventricular groove.

power as necessary to obtain a 10- to 15-Ω impedance drop with maximum temperature of 45°C. For cases in which the ablation catheter could not be advanced to the earliest site of activation or ablation was prohibited by proximity to a coronary artery, RF delivery was attempted at an adjacent site further away from the coronary artery either inside the CVS or from neighboring LV endocardium, sinus of Valsalva, or RVOT, based on activation and anatomic proximity. If power delivery was limited because of wedging of the catheter tip in the vein branch, then the catheter was withdrawn to a more proximal site where power delivery was improved. Ablation on the endocardial surface opposite the site of earliest activation was typically started at 30 W, and energy was titrated to achieve an impedance drop of 15 Ω. Duration of RF application from adjacent anatomic sites could be extended to 120 seconds in cases of late suppression within 60 seconds, but not immediate elimination of the VA. In case of no response after achievement of the target impedance drop, the ablation was redirected and a new lesion delivered. Epicardial access was obtained using the Sosa technique as previously described in selected patients when the above ablation strategy was unsuccessful.[12] Epicardial activation mapping was then performed, with ablation targeting the site of earliest activation if there was at least 10-mm distance from the nearest coronary artery. Similarly to the endocardial ablation, starting energy was 30 W with subsequent titration to achieve an impedance drop of 15 Ω. The rate of catheter open irrigation was typically decreased during epicardial ablation to 15–17 mL/min to avoid hemodynamically significant rapid fluid accumulation in the epicardial space.

Acute success was defined as the absence of spontaneous or inducible VAs with isoproterenol infusion (rate up to 20 μg/min) and burst pacing from the high right atrium and RV apex for 30 minutes after the last ablation lesion.

Follow-up

Patients underwent 24-hour full disclosure telemetry monitoring and transthoracic echocardiography after the procedure and were followed in the Arrhythmia Center of the Hospital of the University of Pennsylvania with routine ECGs at 6 weeks. Follow-up echocardiography and Holter monitoring were performed 4–12 months postablation.

Statistical analysis

Continuous variables are expressed as mean with SD, except for ablation times, which are expressed as mean with SEM, and categorical variables, which are expressed as percentages. The Student *t* test and Mann–Whitney *U* test were used to compare continuous variables. The Fisher exact test was used to compare dichotomous variables. Analyses were performed using SPSS software (version 18.0, SPSS Inc, Chicago, IL). $P \leq .05$ was considered significant.

Results

Between January 2007 and June 2012, 511 patients were referred to the Electrophysiology Section of the Hospital of the University of Pennsylvania for idiopathic VAs refractory to medical therapy. Three hundred twenty-seven were referred for frequent symptomatic VPDs and 184 for nonsustained or sustained ventricular tachycardia (VT). Activation mapping of the RV and LV endocardium as well as coronary venous branches was performed in 117 patients. Of these patients, 47 (9%, 21 males, age 55.4 ± 14.8 years) had earliest activation in the branches of the CS system (39 ± 18 ms pre-QRS). The dominant arrhythmia was symptomatic VPDs in 37 patients and sustained VT in 10 patients. Nonsustained VT of the same morphology with the dominant arrhythmia was also present in 21 of the 37 with VPDs and in 6 of the 10 with sustained VT. All 10 patients who were referred for sustained VT had structurally normal heart by echocardiography, cardiac magnetic resonance imaging, and stress test or cardiac catheterization. Of the 37 patients who were referred for ablation of symptomatic VPDs, 6 had coronary artery disease and 14 had depressed LV function. In all patients with cardiomyopathy, the origin of the clinical VPD was not related to the location of myocardial scar. This was determined by cardiac magnetic resonance imaging in 9 patients with cardiomyopathy that could not be explained by the presence of coronary artery disease and by nuclear imaging, echocardiography, and voltage mapping in the remaining patients.

In 25 patients (53%), VAs arose from the distal GCV, in 19 (40%) from the AIV, and in 3 (7%) from the MCV (Figures 2–4). VAs from the GCV or AIV had a characteristic right axis with rS or S morphology in lead I. Twenty-four patients (55%) had VAs with a right bundle branch block pattern in lead V_1 and 20 patients (45%) had a left bundle branch block pattern with precordial transition from negative to positive in lead V_2 (8 patients) or V_3 (12 patients). VAs from the GCV were more likely to have a right bundle branch block morphology or earlier precordial transition (lead V_2) than VAs from the AIV ($P = .029$). Amplitude in lead III was slightly higher than in lead II (III/II ratio 1.17 ± 0.15, $P = .001$). There were no statistically significant differences in axis, QRS width, and maximum deflection index between VAs arising from the GCV vs AIV. VAs with earliest activation at the MCV had a left bundle branch block morphology with rapid positive precordial transition in lead V_2. Axis was left (R wave in lead I) and superior, with S in lead III more negative than in lead II. Baseline characteristics are listed in Table 1.

In all cases, pace-maps generated from within the CVS generated a 12/12 match with the clinical VA and were superior to pace-maps from adjacent sites by qualitative analysis from at least 2 observers.

Ablation inside the CVS was performed in 32 patients (68%). In 14 of those patients (44%), ablation was not delivered to the earliest site but proximal to the earliest site in order to establish a 10-mm distance from the closest

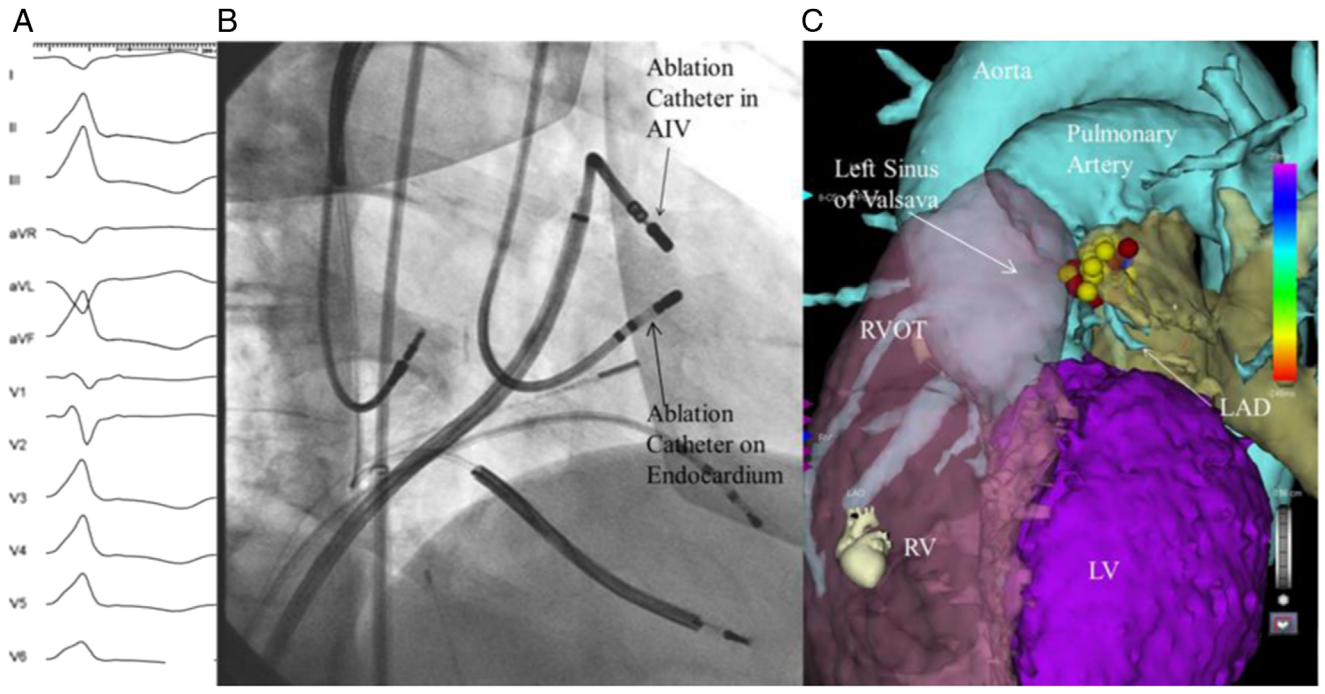


Figure 2 Ventricular premature depolarization (VPD) originating from the anterior interventricular vein (AIV). **A:** Twelve-lead ECG morphology of the clinical ventricular premature depolarization (VPD) (100 mm/s). The VPD has a left bundle branch block morphology with right inferior axis and precordial transition from negative to positive in lead V_3. **B:** Ablation catheter at the site of earliest activation in the AIV in the right anterior oblique projection. A second catheter is placed on the endocardial site opposite the site of successful ablation. **C:** Merged image between the created 3-dimensaional electroanatomic map and computed tomographic scan. The course of the AIV is indicated by *yellow points.* Note the relationship between the AIV, left sinus of Valsalva, right ventricular outflow tract (RVOT), and left anterior descending coronary artery (LAD). LV = left ventricle; RV = right ventricle.

coronary artery in 8 patients, due to an inability to advance the ablation catheter to the site of earliest activation in 4 patients, or because of inadequate power delivery in 2 patients. The decision to not deliver RF lesions inside the CVS was made in 15 patients (32%). In 13 of those patients the earliest site was in immediate proximity to a coronary artery (Figure 5), and in 2 patients ablation was first performed from the adjacent left coronary cusp and was successful.

Seventeen of 18 cases (94%) in which ablation was delivered at the earliest site in the CVS were successful. Of the 14 patients with ablation proximal to the site of earliest activation inside the CVS, ablation only in the CVS was successful in 4 (29%); with the addition of ablation in nearby

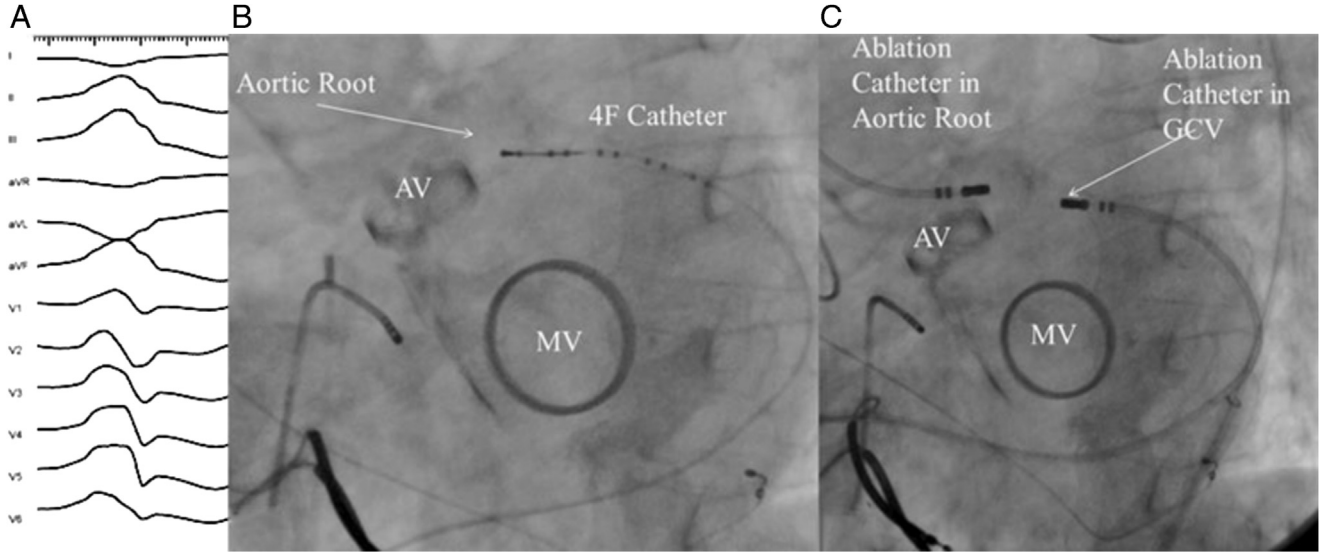


Figure 3 Ventricular premature depolarization (VPD) originating from the great cardiac vein (GCV). **A:** Ventricular tachycardia with earliest activation at the distal GCV in a patient with aortic and mitral valve replacement (100 mm/s). The tachycardia has a right bundle branch block morphology with right inferior axis and positive precordial concordance. **B:** The site of earliest activation and best pace-map was identified with a 4Fr catheter; however, the 7Fr ablation catheter could not be advanced as distally. **C:** Radiofrequency application from both the GCV and left sinus of Valsalva rendered the tachycardia noninducible. AV = aortic valve; MV = mitral valve.

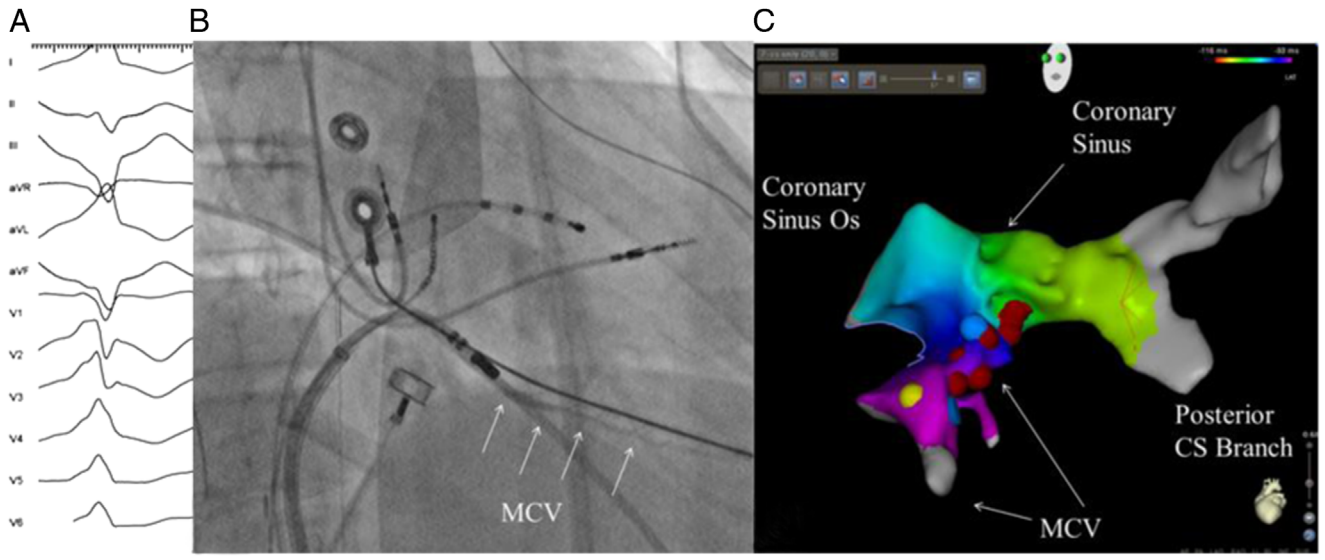


Figure 4 Ventricular tachycardia originating from the middle cardiac vein. **A:** Twelve-lead ECG morphology of the clinical ventricular tachycardia (100 mm/s). The tachycardia has a left bundle branch morphology with rapid positive precordial transition in lead V_2. Frontal axis is left (R wave in lead I) and superior, with S in lead III more negative than in lead II. **B:** Ablation catheter at the site of earliest activation in the right anterior oblique projection. Contrast that had been administered outlines the middle cardiac vein (MCV, *arrows*). **C:** Electroanatomic map of the proximal coronary sinus (CS)and MCV with ablation lesions.

sites outside the CVS, another 5 (36%) were successful. Mapping characteristics are listed in Table 2, and acute ablation outcomes based on the site of ablation are summarized in Figure 6.

All 15 patients who did not undergo ablation inside the CVS had ablation in adjacent anatomic sites, including the sinuses of Valsalva, LV endocardium, or RVOT based on anatomic proximity. Ablation in the left coronary cusp alone successfully eliminated VAs in 3 patients (20%), and ablation in the left coronary cusp followed by ablation on the LV endocardium eliminated the VAs in 4 patients (27%; Figure 6). Ablation times for cases in which the VAs were eliminated from the CVS alone were significant lower that those in which ablation was required in adjacent sites (307 ± 55 seconds vs 750 ± 97 seconds, $P = .002$).

Epicardial access was obtained in 7 patients (15%), in 5 because of proximity of the site of earliest activation in the CVS to a coronary artery and in 2 patients in whom the ablation catheter could not be advanced to the site of earliest activation in the CVS. Mapping in the epicardium was not successful in identifying a more favorable site of ablation in terms of activation or pace-mapping in all cases. All cases in which ablation was limited because of proximity to coronary arteries were equally limited in the epicardium, and epicardial ablation was not performed. Epicardial ablation was performed in only 1 patient after the VPD was initially suppressed with ablation inside the AIV and was subsequently successfully eliminated with ablation from the adjacent epicardium. Acute complications included a femoral artery pseudoaneurysm in 1 patient who did not require further intervention. There was 1 case of new pericardial effusion in a patient with earliest activation in the AIV, presumed secondary to CS perforation while mapping as no ablation was delivered within the CVS. This patient underwent

Table 1 Baseline characteristics

Baseline characteristic	Overall	GCV (n = 25)	AIV (n = 219)	MCV (n = 23)	*P* value*
Age (years)	55 ± 15	45 ± 16	63 ± 11	47 ± 20	**.004**
Male	21 (45%)	11 (44%)	9 (47%)	1 (33%)	.897
VT	10 (21.0%)	2 (9%)	5 (31%)	3 (100%)	**.001**
VPD/24 hours	29,042 ± 19,491	26,226 ± 22,317	34,227 ± 10,200	N/A	.508
% VPD	25 ± 12	19 ± 12	32 ± 8	N/A	.337
Multiple VPDs	16 (34%)	12 (48%)	4 (21%)	0 (0%)	.064
Previous ablation	22 (47%)	9 (36%)	10 (53%)	3 (100%)	1
LVEF	47 ± 14	46 ± 17	52 ± 17	52 ± 17	.383
LVDD (cm)	5.2 ± 0.9	5.2 ± 0.9	5.2 ± 0.8	4.5 ± 0.8	.891
LA size (cm)	3.9 ± 0.7	3.7 ± 0.8	4.5 ± 0.6	3.9 ± 0.4	.632
QRS duration (ms)	153 ± 26	153 ± 29	152 ± 23	156 ± 30	.923
MDI (%)	49 ± 7	47 ± 6	51 ± 8	48 ± 6	.131

AIV = anterior interventricular vein; GCV = great cardiac vein; LA = left atrium; LVDD = left ventricular diastolic dimension; LVEF = left ventricular ejection fraction; MCV = middle cardiac vein; MDI = maximum deflection index; VPD = ventricular premature depolarization; VT = ventricular tachycardia.
*Comparison between GCV and AIV.
Significant *P* values are in bold type.

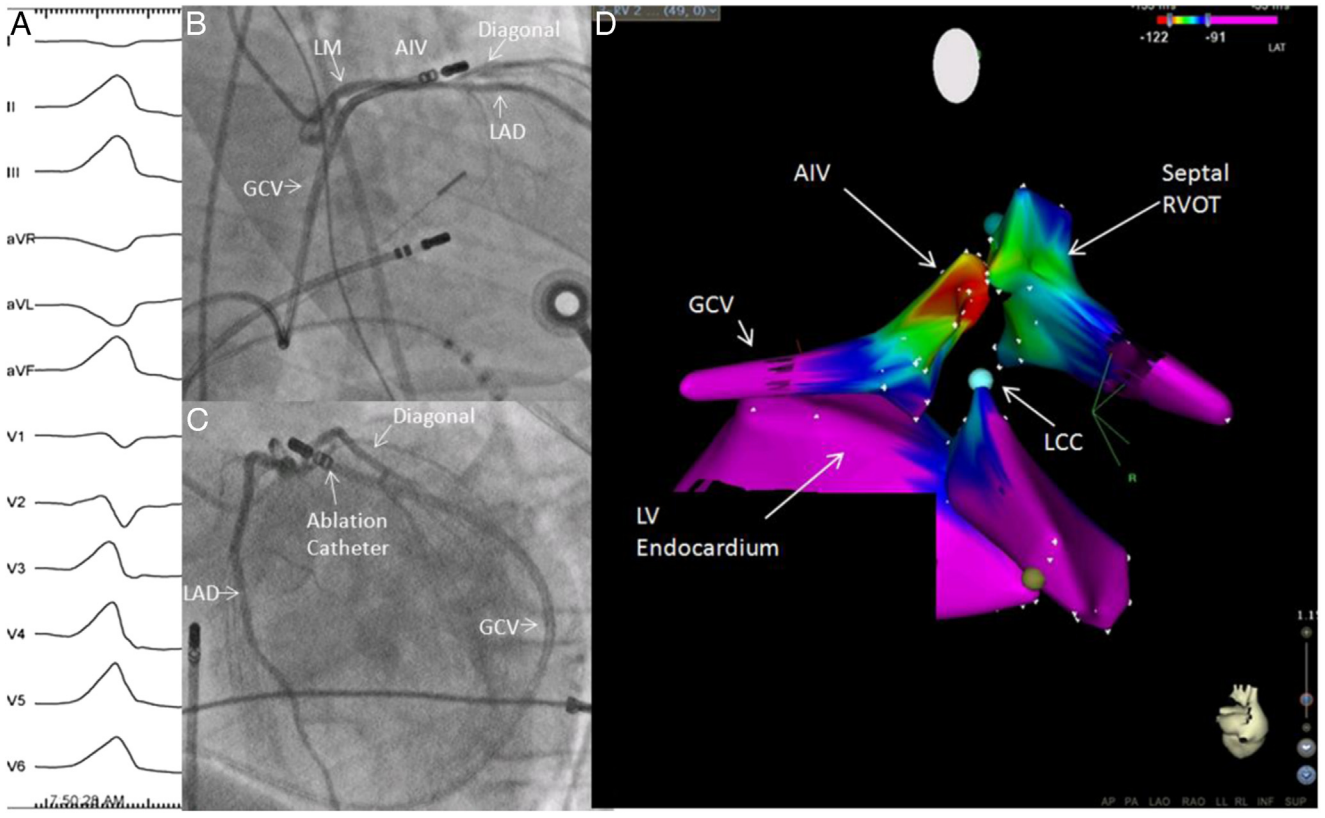


Figure 5 Ventricular premature depolarization (VPD) originating from the anterior interventricular vein with close proximity to a coronary artery branch. **A:** VPD (100 mm/s) has q left bundle branch block pattern with right inferior frontal axis and precordial transition from negative to positive in lead V_3. Coronary angiogram in right and left anterior oblique caudal views (**B** and **C,** respectively) demonstrated close proximity to a large diagonal coronary artery branch. **D:** Activation map from adjacent sites in the posterior view demonstrating the greatest anatomic proximity of the earliest site with the septal right ventricular outflow tract (RVOT). The VPD was ablated successfully from the RVOT. AIV = anterior interventricular vein; GCV = great cardiac vein; LAD = left anterior descending coronary artery; LCC = left coronary cusp; LM = left main coronary artery; LV endocardium = left ventricular endocardium.

pericardiocentesis to drain the effusion without recurrence. There were no signs or symptoms of acute ischemia, including ST-segment changes on ECG or wall-motion abnormality by intracardiac echocardiography in any patients. One patient presented at follow-up with nonspecific symptoms of fatigue and subsequent coronary angiography showed a 50% stenosis of the proximal circumflex coronary artery that was not present during angiography just before ablation. The stenosis was in close proximity (10 mm) to the initial RF energy application. The coronary abnormality was

Table 2 Procedural characteristics

Procedural characteristic	Overall	GCV (n = 25)	AIV (n = 19)	MCV (n = 3)	*P* value*
Activation time (ms)	39 ± 18	39 ± 18	40 ± 20	27 ± 7	.893
Presence of any limitation	29 (62%)	12 (48%)	16 (84%)	1 (33%)	**.025**
Proximity to coronaries	26 (55%)	10 (40%)	15 (79%)	1 (33%)	**.011**
Inability to advance ablator at the earliest identified site	4 (9%)	1 (4%)	3 (16%)	0 (0%)	.300
Inadequate energy delivery	2 (4%)	0 (0%)	2 (11%)	0 (0%)	.181
Ablation not performed in CVS	15 (32%)	9 (36%)	6 (32%)	0 (0%)	1
Elimination within CVS	21 (45%)	11 (40%)	8 (42%)	2 (66%)	1
Elimination from adjacent site	12 (26%)	8 (32%)	4 (21%)	0 (0%)	.148
Cusp/RVOT	6	5	1‡	N/A	
Cusp and/or endocardium	5	3	2	0	
Epicardium	1	0	1	0	
Total ablation time (seconds)†	589 ± 72	499 ± 104	674 ± 99	748 ± 320	**.014**
Overall success	33 (70%)	19 (76%)	12 (63%)	2 (66%)	.327

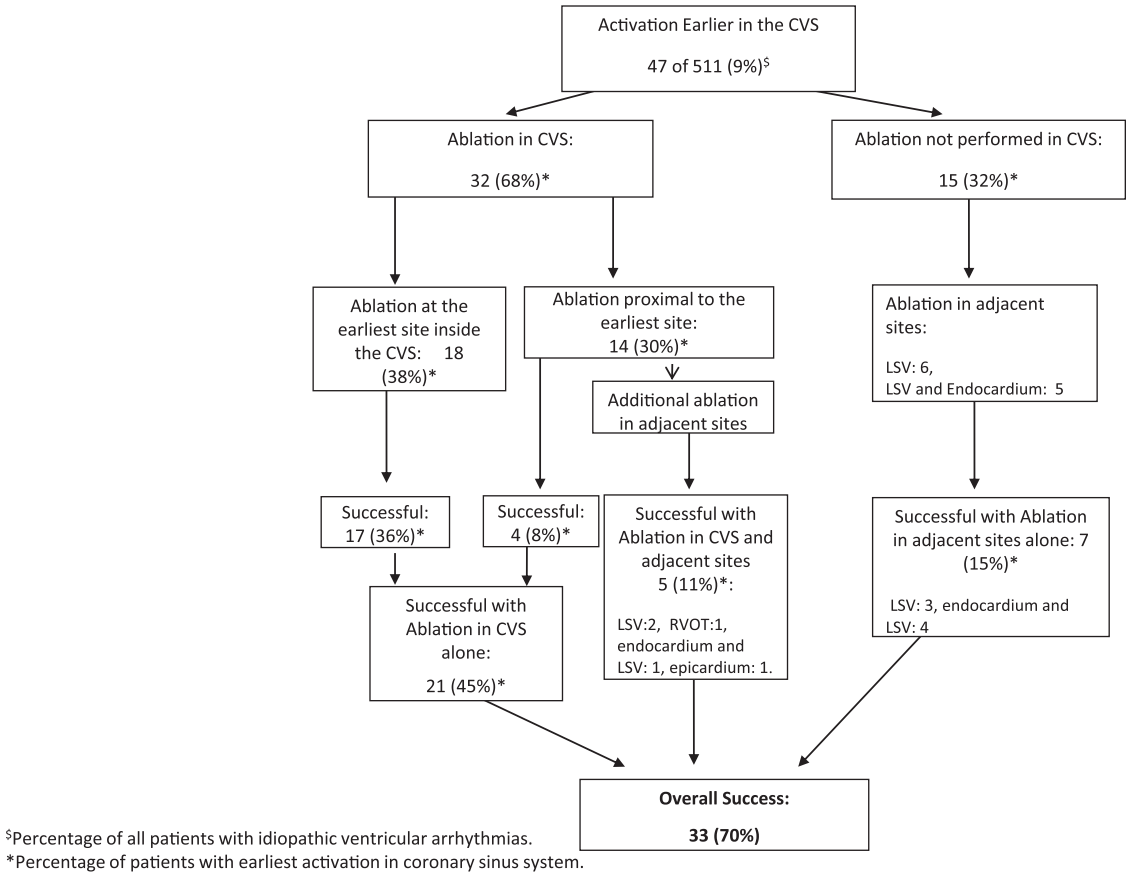
AIV = anterior interventricular vein; CVS = coronary venous system; GCV = great cardiac vein; MCV = middle cardiac vein; RVOT = right ventricular outflow tract.

*Comparison between GCV and AIV.

†Expressed as mean ± SEM.

‡Eliminated from RVOT.

Significant *P* values are in bold type.



$Percentage of all patients with idiopathic ventricular arrhythmias.
*Percentage of patients with earliest activation in coronary sinus system.
CVS : Coronary sinus system; LSV: Left sinus of Valsalva, RVOT: right ventricular outflow tract.

Figure 6 Acute ablation outcomes based on site of ablation.

not associated with ischemia on stress test and was not intervened upon.

Of the 29 patients with ongoing follow-up (4 lost to follow-up) after successful ablation, 7 (24%) had recurrence of premature ventricular complexes or VT. The remaining 22 of the 29 (76%) had no VT or premature ventricular complexes during follow-up of 30.7 ± 21.1 months.

Discussion

We report on the largest series of VAs with earliest activation inside the CVS. In our experience, these arrhythmias represented 9% of all idiopathic VAs. This incidence is in agreement with previous reports.[6] Ablation frequently could not be delivered at the site of earliest activation within the CVS (62% of cases). The most common limitation was proximity to coronary arteries (55%). Less common reasons were inability to access the earliest site of activation (as defined by the decapolar mapping catheter) with the ablation catheter (9%) and inadequate power delivery (4%).

A particularly challenging area to map and ablate in the CVS was the AIV. First, access is associated with technical difficulties because the ablation catheter needs to navigate anterior to the atrioventricular groove, overcoming an acute angle between the GCV and AIV. In our practice, this was facilitated in the majority of cases by the use of a long sheath, which was positioned at the ostium of the CS or even well inside the CS (Figure 2) to enhance maneuverability of the ablation catheter tip. Additionally, sites in the AIV were more frequently in proximity to the left anterior descending coronary artery (79%), as the 2 vessels course together in the anterior interventricular groove. Although proximity to coronary arteries was slightly less common in the GCV, it still occurred in 40% of cases. Therefore, we propose that coronary angiography should always be performed before delivering RF ablation inside the CVS. Finally, both cases of inadequate power delivery were in the AIV, likely because of smaller vein caliber compared to the GCV. Use of an open irrigated catheter in the patients of our series probably accounts for the relatively small incidence of inability to deliver adequate power inside the CVS.

Despite procedural challenges, our series demonstrates that ablation can be successful even when delivered from adjacent sites inside or outside the CVS. Specifically, 16 patients (34%) had their VAs successfully ablated from a site other than the site of earliest activation in the CVS. We believe that this is possible because of the complex anatomic relationships in this "left ventricular ostium"[5] with multiple anatomically distinct areas of interest in close proximity. The importance of multiple ablation sites in eliminating those VAs suggests that penetrating a midmyocardial origin from opposing sides occasionally is necessary. Whether such a

strategy was successful by means of conductive heating reaching the true site of origin or by blocking the exit of the VA is unclear. In terms of the specific origin, VAs from the distal GCV or proximal AIV were more likely to be successfully ablated from adjacent sites, particularly the left sinus of Valsalva. In contrast, VAs from the more distal AIV could not be eliminated from the sinuses of Valsalva. This is not surprising because the AIV resides further away from the atrioventricular groove and therefore is at a greater distance from the aortic root. Nevertheless, 1 case with earliest activation in the AIV was successfully eliminated from the anterior RVOT, as the outflow tract can extend more anteriorly and leftward than the sinuses of Valsalva (Figure 2).

Ablation on the endocardium opposite to the earliest CVS site was typically performed after ablation in the left sinus of Valsalva and/or CVS had failed. Our group previously described this strategy, and if the anatomic separation is minimal (<13 mm) from the site of earliest activation (<13 mm), ablation can be successful in up to 50% of cases.[10] Elimination from the endocardial surface opposite to the CVS site of earliest activation may require longer lesion duration, suggesting that further lesion depth was needed to penetrate to the true site of origin.

In our series, direct epicardial access via a subxiphoid approach was obtained in 7 patients (15%) and was helpful in VA elimination in only 1 patient because it failed to identify a superior site of origin and was equally limited by the proximity of the coronary arteries. This observation suggests that direct epicardial mapping might have small incremental value when a favorable site of origin (12/12 pace-map match with activation times greater than –30ms) has been identified by mapping inside the CVS. This is in agreement with recent reports in which direct epicardial access either failed to identify an earlier site than within the CVS[7] or was precluded from adjacent coronaries.[8] This low incremental rate of success may be due to the undesirable effect of epicardial fat insulating the arrhythmogenic tissue in the LV summit. Alternatively, the true site of origin for the majority of these VAs may be either in the CVS itself or immediately beneath the CVS making it inaccessible from the epicardium. Our data suggest that ablation at sites within the CVS or other adjacent sites be performed whenever possible rather than subjecting a patient to the additional risks of a percutaneous epicardial puncture if the site of origin appears to be located in the proximal CVS.

Study limitations

This is a retrospective analysis of a referral cohort with $>50\%$ of patients having undergone failed ablations at other institutions. Therefore, our cohort may differ from the general population with idiopathic VAs. However, the purpose of this study was to provide mapping and ablation strategies in this particularly challenging subset of patients. The methodology of mapping and ablation, including mapping in the sinuses of Valsalva and the decision to ablate in adjacent structures and the order in which ablation was performed, was not standardized. Rather, the ablation strategy was designed by the operator for each individual case after taking into account anatomic and electrophysiologic characteristics. The identification of coronary artery injury as a result of ablation in the CVS was based on clinical, electrocardiographic, and echocardiographic data. Follow-up coronary angiography was not performed routinely. In addition, the long-term effects of RF delivery in proximity to coronary arteries were not systematically evaluated by the present study. The single case of late coronary stenosis, even if asymptomatic, suggests that a cautious approach to ablating in the CVS in proximity to the coronary arteries must be observed. We recommend considering ablation from adjacent anatomic structures without intervening coronary vasculature whenever possible.

Conclusion

VAs with earliest activation in the CVS comprise 9% of idiopathic VAs. Ablation inside the CVS is frequently limited by proximity of the site of origin to coronary arteries; therefore, coronary angiography should be performed before ablation. If ablation can be delivered at the site of earliest activation in the CVS, ablation is highly successful in eliminating these arrhythmias. In cases of inability to deliver RF energy at the site of origin, ablation can be attempted in adjacent sites, including slightly more proximal within the CVS, left aortic sinus of Valsalva, RVOT, and/or opposing endocardium when there is no intervening coronary anatomy. Direct epicardial mapping and ablation was not of significant benefit in this series; however, the incremental value of epicardial mapping in selected remains to be determined.

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CLINICAL PERSPECTIVES

Ventricular arrhythmias originating in proximity to the coronary venous system comprise 9% of idiopathic ventricular arrhythmias and should be considered when surface ECG is suggestive and mapping the endocardium fails to show a favorable site for ablation. More than 60% of the time, effective ablation is precluded within the venous system because of proximity to coronary arteries, inability to advance the ablation catheter to the distal site of origin, or inadequate energy delivery. Importantly, delivery of radiofrequency energy at adjacent anatomic sites, including left aortic sinus of Valsalva, RVOT, and/or opposing LV endocardium, yields a 70% overall success rate in eliminating these arrhythmias. Direct epicardial mapping and ablation appears to have limited value.