Ventricular Arrhythmias Near the Distal Great Cardiac Vein
Challenging Arrhythmia for Ablation

Koichi Nagashima, MD, PhD; Eue-Keun Choi, MD, PhD; Kaity Y. Lin, MD; Saurabh Kumar, MD, PhD; Usha B. Tedrow, MD, MSc; Bruce A. Koplan, MD, MPH; Gregory F. Michaud, MD; Roy M. John, MD, PhD; Laurence M. Epstein, MD; Michifumi Tokuda, MD, PhD; Keiichi Inada, MD, PhD; Gregory S. Couper, MD; William G. Stevenson, MD

Background—Catheter ablation for ventricular arrhythmia (VA) near the distal great cardiac vein (GCV) is often challenging, and data are limited.

Methods and Results—Analysis was performed in 30 patients (19 men; age, 52.8±15.5 years) who underwent catheter ablation for focal VA (11 ventricular tachycardia and 19 premature contractions) with early activation in the GCV (36.7±8.0 ms pre-QRS). Angiography in 27 patients showed earliest GCV site within 5 mm of a coronary artery in 20 (74%). Ablation was performed in the GCV in 15 patients and abolished VA in 8. Ablation was attempted at adjacent non-GCV sites in 19 patients and abolished VA in 5 patients (4 from the left ventricular endocardium and 1 from the left coronary cusp); all success had VA with an initial r wave in lead I and activation ≤7 ms after the GCV (GCV–non-GCV interval). In 13 patients, percutaneous epicardial mapping was performed, but because of adjacent coronaries only 2 received radiofrequency application with VA elimination in 1. Surgical cryoablation was performed in 3 patients and abolished VA in 2. Overall acute success was achieved in 16 (53%) patients. After a median of 2.8 months, 13 patients remained free of VA. Major complications occurred in 4 patients, including coronary injury requiring stenting.

Conclusions—Ablation for this arrhythmia is challenging and often limited by the adjacent coronary vessels. Success of anatomically guided endocardial ablation may be identified by a short GCV–non-GCV interval and r wave in lead I. (Circ Arrhythm Electrophysiol. 2014;7:906-912.)

Key Words: catheter ablation ■ premature ventricular contraction ■ ventricular tachycardia

Catheter ablation (CA) for ventricular arrhythmia (VA) is a widely accepted therapy for patients without structural heart disease, but the approach and outcomes depend on the site of arrhythmia origin. Most VAs originate from the right or left ventricular (LV) outflow regions, including myocardium around the aortic cusps and the summit of the LV. Approaches to ablation of VA from specific areas are being increasingly refined. Ablation of VA originating from near the distal great cardiac vein (GCV) is often difficult because of proximity to coronary arteries limited capability of radiofrequency energy application in the GCV itself. We have targeted these arrhythmias from various approaches, including the endocardial, epicardial via the GCV, and pericardial space, and also with open-chest surgery. The aim of this study was to evaluate the mapping features, outcomes, and risks for ablation of these VAs.

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From the Arrhythmia Unit, Cardiovascular Division, Brigham and Women’s Hospital, Boston, MA.
Guest Editor for this article was Gerhard Hindricks, MD.
Correspondence to William G. Stevenson, MD, Cardiovascular Division, Brigham and Women’s Hospital, 75 Francis St, Boston, MA 02115. E-mail wstevenson@partners.org
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with a history of sustained VT, programmed ventricular stimulation was performed with ≤3 extrastimuli scanned to a minimum coupling interval of 180 ms after basic drives of 600 and 400 ms from 2 RV sites and burst pacing. If sustained VT was not reliably inducible, nonsustained VT or PVCs felt likely to be originating from the same site were targeted. Intravenous infusion of isoproterenol and epinephrine was administered as needed for arrhythmia induction.

Mapping and ablation were performed using a 3.5-mm-tip catheter (NaviStar ThermoCool or ThermoCool SF; Biosense Webster, Diamond Bar, CA) in 25 patients. In 5 patients, a 4-mm-tip nonirrigated catheter (NaviStar; Biosense Webster) was used initially for mapping (5 patients) and attempted ablation (3 patients) before switching to an irrigated tip catheter. Activation mapping of VT or PVCs used an electroanatomic mapping system (CARTO 3 or XP; Biosense Webster) with bipolar electrograms high-pass filtered at 20 to 30 Hz and low-pass filtered at 400 Hz. Bipolar electrograms were also band pass filtered from 30 to 500 Hz and digitally recorded along with a 12-lead surface ECG (Cardiolab EP system; General Electric Healthcare, Buckinghamshire, United Kingdom). Pace mapping used unipolar stimuli at 10 mA and pulse width of 2 ms.

Epicardial mapping was considered if a subepicardial VT origin was suspected based on endocardial and GCV mapping, either at the same or a subsequent session. Percutaneous subxiphoid epicardial access was obtained as previously described either before administration of systemic anticoagulation or after anticoagulation was reversed.

Ablation
Coronary angiography was performed before initial ablation at sites with the possibility of an adjacent artery. If the distance of the catheter tip to a major coronary artery was ≤4 to 5 mm, radiofrequency delivery was avoided. Radiofrequency application was also not attempted if there was diaphragmatic capture during pacing from the ablation catheter. Irrigated radiofrequency energy was delivered at 25 to 50 W targeting an impedance drop of 10 to 20 Ω. At endocardial sites below the aortic valve, applications were usually repeated until unipolar pacing at 10 mA at 2 ms stimulus strength failed to capture. At target areas in the sinuses of Valsalva and GCV, power exceeding 35 W was avoided. At some sites where radiofrequency ablation was limited or coronary artery proximity was of concern, ablation with a 6-mm-tip cryocatheter (Cryocath Technologies, Montreal, Canada) was attempted.

At the end of the procedure, the same induction protocol was repeated. Acute success was defined as the absence of any target arrhythmia.

Data Collection and Follow-Up
Data were collected from a centralized system containing records of all patients treated and followed up at Brigham and Women’s Hospital and all associated Partners Healthcare sites. These records include emergency department visits, outpatient clinic visits, data recorded during inpatient care, and follow-up progress notes from referring physicians monitoring out-of-area patients. Referring physicians were contacted for inpatient care, and follow-up progress notes from referring physicians monitoring out-of-area patients. Referring physicians were contacted for clinical follow-up if necessary. Procedure success was defined as more than an 80% reduction in VA on 24-hour Holter monitoring or resolution of symptoms with no VA in all electrocardiograms during follow-up.

Statistical Analysis
Continuous variables were expressed as mean±SD values or median and interquartile ranges are shown in parentheses, as appropriate. Student t test or Mann–Whitney U test was used to compare continuous variables, depending on whether the values were normally distributed, and the χ2 test was used to compare dichotomous variables unless the expected values in any cells were ≤5, in which case Fisher exact test was used. P<0.05 was considered to be statistically significant. Statistical analyses were performed with JMP 9 software (SAS Institute, Cary, NC).

Results
Electrophysiological Characteristics
Patient and arrhythmia characteristics are shown in Table 1. Examples of VA morphology are shown in Figure I in the Data Supplement. Sustained VT was inducible in 10 of 11 patients who had a history of sustained VT. The lead V1 morphology was right bundle branch block like in 21 (70%) and left bundle branch block morphology with precordial transition before V3 in 8 (27%) and after V3 in 1 (3%). All VAs had an inferior axis (98.6°±10.0°). QRS duration was 161.9±18.8 ms (Table 1). The maximum deflection index defined as the interval from the earliest QRS onset to the earliest R-wave peak in the precordial leads divided by the QRS duration was >0.55, suggesting epicardial origin, in 10 (33%) patients, with a mean of 0.52±0.06. Previous ablation attempts had failed in

Table 1. Patient Characteristics With VA Close to the GCV

<table>
<thead>
<tr>
<th>Patients (n=30)</th>
<th></th>
</tr>
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<tbody>
<tr>
<td>Age, y</td>
<td>52.8±15.5</td>
</tr>
<tr>
<td>Men</td>
<td>19 (63)</td>
</tr>
<tr>
<td>LVEF, %</td>
<td>52.0±12.9</td>
</tr>
<tr>
<td>History of previous ablation</td>
<td>16 (53)</td>
</tr>
<tr>
<td>Clinical arrhythmias</td>
<td></td>
</tr>
<tr>
<td>Sustained VT</td>
<td>11 (37)</td>
</tr>
<tr>
<td>Nonsustained VT or PVCs</td>
<td>19 (63)</td>
</tr>
<tr>
<td>VA morphology during EPS</td>
<td></td>
</tr>
<tr>
<td>VT sustained</td>
<td>10 (33)</td>
</tr>
<tr>
<td>Cycle length of VTs, ms</td>
<td>396.2±81.8</td>
</tr>
<tr>
<td>QRS width, ms</td>
<td>161.9±18.8</td>
</tr>
<tr>
<td>MDI</td>
<td>0.52±0.06</td>
</tr>
<tr>
<td>GRS axis, °</td>
<td>98.6±10.0</td>
</tr>
<tr>
<td>Limb leads</td>
<td></td>
</tr>
<tr>
<td>QS pattern in lead I</td>
<td>16 (53)</td>
</tr>
<tr>
<td>R in lead I, mV</td>
<td>–0.23±0.35</td>
</tr>
<tr>
<td>R in lead II, mV</td>
<td>1.78±0.59</td>
</tr>
<tr>
<td>R in lead III, mV</td>
<td>1.97±0.61</td>
</tr>
<tr>
<td>Ratio in I/II</td>
<td>1.12±0.16</td>
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<tr>
<td>R in aVF, mV</td>
<td>1.87±0.59</td>
</tr>
<tr>
<td>Q in aVL, mV</td>
<td>1.12±0.38</td>
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<td>Q in aVR, mV</td>
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<td>Ratio in aVL/aVR</td>
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</tr>
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<td>Precordial leads</td>
<td></td>
</tr>
<tr>
<td>RBBB pattern</td>
<td>21 (70)</td>
</tr>
<tr>
<td>LBBB with R-wave transition in V3</td>
<td>8 (27)</td>
</tr>
<tr>
<td>Mapping</td>
<td></td>
</tr>
<tr>
<td>Activation time at GCV, ms</td>
<td>36.7±8.0</td>
</tr>
<tr>
<td>12/12 pace map at GCV</td>
<td>21/24 (88)</td>
</tr>
<tr>
<td>Ablation in GCV</td>
<td></td>
</tr>
<tr>
<td>Average power, W</td>
<td>23 (15, 27)</td>
</tr>
<tr>
<td>Average duration, s</td>
<td>30 (23, 39)</td>
</tr>
<tr>
<td>Initial impedance, Ω</td>
<td>244 (186, 266)</td>
</tr>
<tr>
<td>RFA+cryoablation</td>
<td>4 (13%)</td>
</tr>
</tbody>
</table>

Values are the mean±SD, median (25th, 75th interquartile range) or n (%). EPS indicates electrophysiological study; GCV, great cardiac vein; LBBB, left bundle branch block; LVEF, left ventricular ejection fraction; MDI, maximum deflection index; PVC, premature ventricular contraction; RBBB, right bundle branch block; RFA, radiofrequency ablation; VA, ventricular arrhythmia; and VT, ventricular tachycardia.
16 patients, 5 of whom had received radiofrequency applications at the earliest RV sites at our institution.

Mapping and Ablation in the GCV
Activation mapping revealed the earliest ventricular activation in the GCV preceded the QRS onset by 36.7±8.0 ms (Figures 1B and 2B; Figure IIB in the Data Supplement). At this site a perfect pace-map, matching the VA in 12 of 12 ECG leads was present in 21 of 24 (88%) patients (Figures 1A and 2A; Figure IIA in the Data Supplement). In the remaining 3 (12%) patients, an 11 of 12 lead pace-map match was observed. In 3 patients, pacing at the earliest GCV site failed to capture the ventricular myocardium even with the strength of 10 mA and pulse width of 9 ms.

A flow chart indicating procedure details is shown in Figure III in the Data Supplement. In 3 patients, an ablation catheter could not be advanced to the distal GCV. Coronary venography and angiography were performed in the remaining 27 patients.

Figure 1. An example of 1 patient with successful ablation of ventricular arrhythmia (VA) in the great cardiac vein (GCV). A, Twelve-lead ECG morphologies of sinus rhythm, VA, and pace-map morphologies recorded during electrophysiological study. A perfect pace-map (12/12 leads) is acquired in the GCV. B, The local electrogram of successful ablation site in the GCV. C, Corresponding fluoroscopic view of catheter positions during coronary venography from contrast injection thorough the irrigated ablation catheter. The ablation catheter tip is at the distal GCV. D, Corresponding fluoroscopic view of catheter positions during coronary angiography of the left coronary artery. The ablation catheter tip is ≥5 mm distant from any coronary artery. E, The surface ECG (top) and ablation catheter electrogram (bottom) during radiofrequency delivery. Cessation of the target VA is observed shortly after radiofrequency initiation. ABL–d indicates ablation catheter distal; ABL–p, ablation catheter proximal; AIV, anterior interventricular vein; EA, the earliest activation; LAD, left anterior descendant; LAO, left anterior oblique; LCX, left circumflex; PM, pace-map; PVC, premature ventricular contraction; RAO, right anterior oblique; RF, radiofrequency; SR, sinus rhythm; and V-QRS, the interval between ventricular electrogram and surface QRS.
In 7 patients (23%), the target site in the distal GCV was ≥5 mm distant from any coronary artery and ablation was performed, successfully abolishing the VA in 5 patients (Figure 1C–1E). In 8 of remaining 20 patients in whom the target site in the distal GCV was <5 mm from the coronary artery (Figure IIC and IID in the Data Supplement), ablation was attempted at a region 2 to 3 mm proximal to the target site with elimination of VA in 3 patients. For all GCV radiofrequency ablation applications, the average radiofrequency power was 23 (interquartile range, 15–27) W and duration was 30 (interquartile range, 23–39) s, limited by an impedance rise that typically began after 15 to 20 s starting from a relatively high initial impedance of 244 (interquartile range, 186–266) Ω. In 4 patients, cryoablation in GCV was attempted because of low power and high impedance with failure of radiofrequency ablation in 3 patients and for reinforcement of an acutely successful radiofrequency ablation in 1. Cryoapplication of −80°C freezes was performed for 4 to 8 minutes, successfully abolishing the VA in 2 patients.

Characteristics of patients for whom radiofrequency energy was delivered in the GCV are shown in the Table in the Data Supplement. The surface ECG of the VA morphology, including R-S in lead I, R ratio in lead III/II, and Q ratio in aVL/aVR, suggested that the VA origin was more leftward in the patients with successful ablation in the GCV when compared with patients in whom ablation in the GCV failed. The maximum deflection index was greater in those with successful ablation. However, there were no differences in the radiofrequency power and duration and initial impedance between the 2 groups.

Figure 2. Data from a patient in whom ventricular arrhythmia (VA) was successfully ablated from the endocardium below the aortomitral continuity (AMC) region, despite later activation at that site than in the great cardiac vein (GCV). A, Twelve-lead ECG morphologies of sinus rhythm, VA, and pace-map morphologies recorded during electrophysiological study. A perfect pace-map (12/12 leads) is acquired in the GCV compared with poor pace-map at AMC. B, The local electrograms in the GCV and AMC. The earliest ventricular activation is obtained in the GCV. C, Activation map and the successful ablation site at the AMC. D, The surface ECG (top) and ablation catheter electrogram (bottom) during radiofrequency (RF) delivery. Cessation of the target VA is observed shortly after radiofrequency initiation. ABL-d indicates ablation catheter distal; ABL-p, ablation catheter proximal; AV, aortic valve; CS, coronary sinus; LAO, left anterior oblique; LV, left ventricle; PM, pace-map; PVC, premature ventricular contraction; SR, sinus rhythm; and V-QRS, the interval between ventricular electrogram and surface QRS.
Endocardial Ablation Outside the GCV
In all patients additional non-GCV mapping was performed before successful GCV ablation or after GCV ablation failed; in the RV in all and in the LV cavity and the aortic sinus in 26 patients, respectively. Radiofrequency was applied at the earliest RV site in 4 patients and failed to abolish VA in all. In the LV and the aortic sinus, the earliest ventricular activation preceded the surface QRS onset by 20.9±6.6 ms and none had a perfect pace-map (Figure 2A and 2B; Figure II A and II B in the Data Supplement). In 4 of 12 (33%) patients who had radiofrequency application below the aortic-mitral continuity and 1 of 5 (20%) patients who had radiofrequency application in left coronary cusp (LCC), VA was successfully abolished, despite later activation at these sites that in the GCV (Figure 2 C and 2D). Ablation failed at endocardial LV sites or the aortic sinus in 10 patients.

As shown in Table 2, all patients with successful LV endocardial or LCC ablation had a VA morphology with an initial r wave in lead I when compared with 33% of patients with ablation failure at these sites (P=0.01). Furthermore, patients with successful LV endocardial or LCC ablation tended to have later activation times at the GCV (31.0±5.0 versus 38.1±8.7 ms preceding surface QRS; P=0.09) and the activation times at the earliest LV endocardial or LCC site tended to be earlier (25.8±4.0 versus 19.6±6.6 ms preceding surface QRS; P=0.06) when compared with patients with ablation failure at these sites. Thus, the interval between the GCV site and earliest LV endocardial or LCC site activation during the VA (GCV–non-GCV interval) was shorter for those with successful endocardial ablation when compared with those in whom ablation failed (5.8±1.6 versus 18.9±8.5 ms; P=0.003; Table 2) and was ≤7 ms in all. There were no differences in pace-map morphology for those with successful versus unsuccessful endocardial ablation.

Percutaneous Epicardial Ablation
Percutaneous epicardial mapping from the pericardial space was performed in 13 patients and revealed sites where the earliest ventricular activation preceded onset of surface QRS by 29.1±6.3 ms (Figure IIB in the Data Supplement); and 2 patients had sites that were slightly earlier than that of the GCV (by =2 ms in both). At the earliest epicardial site, a perfect pace-map was acquired in 2 of 10 patients, despite slightly later activation (by =2 ms) than the GCV (Figure IIA in the Data Supplement). Coronary angiography was repeated during epicardial mapping in 10 patients; no ablation was performed because of the proximity to a coronary artery in 8 (80%) patients (Figure IID in the Data Supplement). Radiofrequency ablation was performed in 2 patients (20%) with elimination of VA in 1 (10%). No cryoablation was performed.

Complications of CA
Major complication occurred in 3 patients. Coronary artery occlusion requiring stenting of a marginal branch of the circumflex artery occurred in 2 who received average radiofrequency energies of 23 and 27 W at ablation sites felt to be 2 mm and 5 to 7 mm distant from the vessel, respectively. GCV perforation without tamponade occurred in 1 patient.

Open-Chest Surgical Epicardial Ablation
In 3 patients who failed percutaneous CA, open-chest cryothermal surgical epicardial ablation with direct vision through a median sternotomy was subsequently performed. After dissection to reach the GCV area, freezes performed under direct visualization successfully abolished the VA in 2 patients. In the remaining patient, ablation was limited because of the vasospasm of the left anterior descendant and difficulty in fully dissecting the left anterior descendant.
Furthermore, angina developed several months after the procedure; angina and angiography revealed a stenosis in the left anterior descendant at the site of cryoablation that was treated with a drug-eluting stent.

**Acute Success and Long-Term Success During Follow-Up**

Overall acute success was achieved in 16 (53%) patients; 8 in GCV, 4 at aortic-mitral continuity, 1 in LCC, 1 in pericardium space, and 2 with surgical ablation. In these patients, 1 patient who had incessant VT before ablation procedure resumed low-dose amiodarone for 2 months.

After a median follow-up of 2.8 (0.9–26.5) months in these 16 patients, procedure success was evaluated by 24-hour Holter in 7 patients (2 with VT and 5 with PVCs) and clinical symptom and electrocardiography in 9 (7 with symptomatic VTs and 2 with symptomatic recurrent PVCs) patients. Thirteen patients (81%) were free of VA; VA recurred in 3 patients (19%), which had been ablated in the GCV in 1, from the endocardium at the aortic-mitral continuity in 1, and from the pericardial space in 1.

**Discussion**

CA of VA originating from near the GCV is often challenging. However, in 5 of 7 patients (71%), in whom radiofrequency application was effectively delivered in the GCV, VA was successfully eliminated. During follow-up, 88% of patients in whom VA was successfully ablated in the GCV were free from VA recurrence. Therefore, acute and long-term success can be favorable if radiofrequency application can successfully be performed.

Unfortunately, ablation is often not possible. In some cases, it is not possible to advance an ablation catheter to the distal GCV. Probably, the most important risk when delivering radiofrequency energy in the GCV is coronary artery injury. Coronary artery damage may be evident acutely or may not become manifest until several weeks after the procedure.

In all patients with successful endocardial ablation the GCV–non-GCV interval was ≤7 ms, suggesting closer proximity to the VA origin than at failed sites. A possible mechanism of the shorter GCV–non-GCV interval in the successful endocardial ablation group is shown in Figure IV in the Data Supplement. Furthermore, all VA successfully ablated from endocardial LV or LCC had an initial r wave in lead I. Hence, those 2 features suggest that VA origin might be located relatively closer to the endocardium and might be predictors for the successful ablation from those sites. Pace-mapping at the LV endocardium was not a reliable marker for successful ablation. These observations are similar to those of Jauregui Abularrach et al., who reported successful ablation from the LV endocardium from the left sinus of Valsalva, LV endocardium, or both sites in 9 of 16 in a similar group of patients.

Epicardial mapping was also limited in this patient population. Sedation and anesthesia as is generally used for epicardial procedures can suppress arrhythmias although we did not encounter this as a significant issue in the present small series. The GCV area is often covered by fat and the overlying anterior descendant at the site of cryoablation that was treated with deep lesions can be achieved with surgical application of cryoablation, which achieves temperatures substantially colder (≤−160°C) than those that can be achieved with present catheter cryoablation, and coronary arteries can be avoided under direct vision or occasionally mobilized for ablation of tissue beneath the vessel. However, surgical morbidity is a concern. Coronary injury is also possible. Mapping to identify the ablation target can be difficult in the operating room, and anesthesia can suppress the arrhythmia.

**Limitations**

This is a retrospective descriptive case series, with a relatively small number of patients. Patients were referred for ablation and 16 had failed before ablation attempts.
Conclusions
CA for arrhythmias originating in the region of the distal GCV is challenging with a favorable ablation outcome in only 53% in this referral population and likely a greater risk of complications than expected for idiopathic VA. Although epicardial ablation is often limited by the proximity of coronary vessels, anatomically guided endocardial ablation can be successful and predicted by a relatively short GCV–non-GCV interval and r wave in lead I. Surgical ablation can also be effective. Additional studies to identify optimal strategies are warranted.

Acknowledgments
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Disclosures
Dr Stevenson is co-holder of a patent for needle ablation that is consigned to Brigham and Women’s Hospital. The other authors report no conflicts.

References

CLINICAL PERSPECTIVE
Catheter ablation of ventricular arrhythmia originating from near the distal great cardiac vein (GCV) is often difficult because of proximity to coronary arteries and limited ability for radiofrequency application in the GCV. This report describes the efficacy and risk of various approaches including endocardial, epicardial via the GCV and pericardial space, and also with open-chest surgery. Ventricular arrhythmias were abolished in only 53% of patients in this referral population and with likely a greater risk of complications than expected for idiopathic ventricular arrhythmia. Ablation from adjacent non-GCV sites was successful in some patients, predicted by a relatively short GCV–non-GCV site interval and r wave in lead I. Thus, although epicardial ablation near the GCV is often limited by the proximity of coronary vessels, anatomically guided endocardial ablation can be successful and surgical ablation is an option.
Ventricular Arrhythmias Near the Distal Great Cardiac Vein: Challenging Arrhythmia for Ablation

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## SUPPLEMENT MATERIAL

Supplemental Table. Characteristics of patients for whom radiofrequency energy was delivered at the distal of great cardiac vein according to acute outcome

<table>
<thead>
<tr>
<th></th>
<th>Successful (n=8)</th>
<th>Failure (n=7)</th>
<th>P value</th>
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<tr>
<td>Age (y)</td>
<td>44.4±18.6</td>
<td>54.3±10.2</td>
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</tr>
<tr>
<td>Male</td>
<td>4 (50)</td>
<td>3 (43)</td>
<td>1.0</td>
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<tr>
<td>VA morphology during EPS</td>
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<tr>
<td>VT sustained</td>
<td>6 (75)</td>
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<td>Cycle length of VTs (ms)</td>
<td>423.2±102.8</td>
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<td>QRS width (ms)</td>
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<td>QRS axis (°)</td>
<td>102.7±6.1</td>
<td>94.5±12.1</td>
<td>0.14</td>
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<tr>
<td>Limb leads</td>
<td></td>
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<tr>
<td>Initial r wave in lead I</td>
<td>4 (50)</td>
<td>4 (57)</td>
<td>1.00</td>
</tr>
<tr>
<td>R-S in lead I (mV)</td>
<td>-0.35±0.22</td>
<td>0.02±0.39</td>
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<tr>
<td>R in lead II (mV)</td>
<td>1.60±0.60</td>
<td>1.85±0.64</td>
<td>0.46</td>
</tr>
<tr>
<td>R in lead III (mV)</td>
<td>1.87±0.72</td>
<td>1.84±0.53</td>
<td>0.92</td>
</tr>
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<td>Ratio in III/II</td>
<td>1.16±0.13</td>
<td>1.01±0.13</td>
<td>0.05</td>
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<tr>
<td>R in aVF (mV)</td>
<td>1.74±0.62</td>
<td>1.83±0.63</td>
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<td>Q in aVL (mV)</td>
<td>1.07±0.43</td>
<td>0.93±0.26</td>
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<td>0.73±0.26</td>
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<tr>
<td>Ratio in aVL/aVR</td>
<td>1.49±0.47</td>
<td>1.03±0.21</td>
<td>0.04</td>
</tr>
<tr>
<td>Precordial leads</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RBBB pattern</td>
<td>7 (88)</td>
<td>3 (43)</td>
<td>0.12</td>
</tr>
<tr>
<td>LBBB with R-wave transition ≤ V3</td>
<td>7 (88)</td>
<td>7 (100)</td>
<td>1.00</td>
</tr>
<tr>
<td>Mapping</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Activation time at GCV (ms)</td>
<td>37.5±11.0</td>
<td>36.0±7.33</td>
<td>0.76</td>
</tr>
<tr>
<td>12/12 pace map at GCV</td>
<td>7/7 (86)</td>
<td>7/7 (86)</td>
<td>1.00</td>
</tr>
<tr>
<td>Ablation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Irrigated catheter</td>
<td>7 (88)</td>
<td>5 (71)</td>
<td>0.57</td>
</tr>
<tr>
<td>Average power (Watts)</td>
<td>24 (21, 31)</td>
<td>17 (9, 27)</td>
<td>0.41</td>
</tr>
<tr>
<td>Average duration (seconds)</td>
<td>30 (25, 56)</td>
<td>30 (18, 51)</td>
<td>0.78</td>
</tr>
<tr>
<td>Initial impedance (ohms)</td>
<td>240 (181, 414)</td>
<td>248 (190, 279)</td>
<td>1.00</td>
</tr>
<tr>
<td>RFA + cryoablation</td>
<td>3 (38)</td>
<td>1 (14)</td>
<td>0.57</td>
</tr>
</tbody>
</table>
Values are the mean±SD or n (%). VA, ventricular arrhythmia; GCV, great cardiac vein; LVEF, left ventricular ejection fraction; VT, ventricular tachycardia; RBBB, right bundle branch block; LBBB, left bundle branch block; MDI, maximum deflection index; RFA, radiofrequency ablation.
Supplemental figure legends

Supplemental Figure 1.
Examples of 12-lead electrocardiogram morphologies of VA. (A) Patients who had successful ablation in the great cardiac vein (GCV). (B) Patients who failed ablation in the GCV. (C) Patients who had successful ablation at the aortic-mitral continuity (AMC).

Supplemental Figure 2.
An example from a patient in whom ablation of ventricular arrhythmia (VA) close to the great cardiac vein (GCV) failed.
(A) Twelve-lead electrocardiogram morphologies of sinus rhythm, VA and pace-map morphologies recorded during electrophysiological study. A perfect pace-map (12/12 leads) is acquired in the GCV and pericardium compared to poor pace-map at aortic-mitral continuity (AMC)
(B) The local electrograms in the GCV, AMC and pericardium. Earliest ventricular activation is obtained in the GCV.
(C) Corresponding fluoroscopic view of catheter positions during coronary venography from contrast injection thorough the irrigated ablation catheter. The ablation catheter tip is at the distal GCV.
(D) Corresponding fluoroscopic view of catheter positions during coronary angiography of the left coronary artery. The earliest site in the GCV is 2mm distant from the left anterior descending coronary artery, and the earliest site in the pericardium is adjacent to the diagonal branch of the left anterior descendant.
AMC, aortic-mitral continuity; other abbreviations are as in Figure 1.
Supplemental Figure 3.
A flow chart indicating the procedure details and follow-up data in patients with ventricular arrhythmias close to the distal great cardiac vein.
GCV, great cardiac vein; LCA, left coronary artery; RF, radiofrequency; RV, right ventricle; Ao, aorta; LV, left ventricle; AMC, aortic-mitral continuity; LCC, left coronary cusp.

Supplemental Figure 4.
A schematic diagram (A) and the ladder diagrams (B and C) explaining possible mechanism of the GCV-nonGCV interval is shown. The GCV-nonGCV interval is shorter in the successful endocardial ablation group (B) than the endocardial ablation failure group (C). In the successful endocardial ablation group, VA origin might locate relatively closer to the endocardium compared to failure group as the endocardial RF lesion reached the origin. Therefore, that location might result in a shorter activation time interval between the GCV and the earliest LV endocardial or LCC sites.
VA, ventricular arrhythmia; other abbreviations are as in Supplemental figure 3.
Supplemental figure 1

(A) Successful ablation in GCV

(B) Ablation failure in GCV

(C) Successful ablation at AMC
Supplemental figure 2

(A)
Pericardial space

V-QRS: 33ms

V-QRS: 15ms

V-QRS: 31ms
(C) EA site at pericardium (adjacent to Diagonal)

(D) EA site in GCV (2mm to LAD)
Supplemental figure 3

GCV mapping
n=30

- ≥5mm to LCA
n=7
  - GCV ablation
n=7
  - Acute success
n=5
  - Procedure success
n=4
    - RV mapping
n=30
      - RV ablation
n=4
        - Acute success
n=0
        - Procedure success
n=3

- <5mm to LCA
n=20
  - Proximal GCV ablation
n=8
  - Acute success
n=3
  - Procedure success
n=3
    - Ao cusp or LV mapping
n=26
      - AMC ablation
n=12
      - Acute success
n=4
      - Procedure success
n=1
      - RV mapping
n=30
      - Acute success
n=1

- No coronary angiography
n=3
  - Ablation catheter could not be advanced to the distal GCV.
n=3
  - Open-chest Surgical ablation
n=3
    - Procedure success
n=2
    - Acute success
n=2

- GCV ablation
n=8
  - Lack of acute success
n=0
  - Procedure success
n=1

- No coronary angiography
n=3
  - No coronary angiography
n=3
  - Procedure success
n=0

- Epicardial mapping
n=13
  - Acute success
n=1
  - Procedure success
n=1

- Pericardial ablation
n=2
  - Acute success
n=1
  - Procedure success
n=0
LV myocardium
Epicardium
Endocardium
GCV - nonGCV interval ≤ 7ms
GCV - nonGCV interval > 7ms
RF lesion
LV cavity

Supplemental figure 4

(A) LV myocardium
     GCV
     RF lesion
     LV cavity

(B) Epicardium
   VA origin
   VA origin *
   Endocardium
   GCV-nonGCV interval ≤ 7ms

(C) VA origin *
   GCV-nonGCV interval > 7ms

Successful endocardial ablation
Endocardial ablation failure