Published in final edited form as:

Heart Rhythm. 2012 October ; 9(10): 1637–1639. doi:10.1016/j.hrthm.2012.06.003.

Coronary Venous Ethanol Infusion for the Treatment of Refractory Ventricular Tachycardia

Alex Baher, MD*, Dipan J. Shah, MD†, and Miguel Valderrabano, MD FACC†

*Department of Medicine, The Methodist Hospital, Houston, Texas

†Methodist DeBakey Heart and Vascular Center, The Methodist Hospital, Houston, Texas

Introduction

Radiofrequency catheter ablation (RFCA) is used increasingly to treat drug-refractory ventricular tachycardias (VTs) (1). Although originally limited to endocardial VTs, access to the pericardial space via a nonsurgical subxyphoid technique has allowed RFCA to target epicardial circuits as well (2). Nevertheless, there still exists a subgroup of VTs that remain refractory to RFCA. This includes VTs with a deep intramural origin, either from reentrant or focal mechanisms. Additionally, there are cases where proximity to the coronary vessels limits attempts at aggressive ablation of the VT origin. Finally, the epicardial approach for RFCA may be difficult if not impossible in cases of prior cardiac surgery (3).

Transcoronary ethanol ablation (TCEA) has been used as an alternative strategy to effectively treat these difficult-to-ablate VTs (4). Although TCEA is reasonably successful in treating RFCA refractory VTs, there are technical difficulties and potential complications inherent to coronary artery instrumentation such as coronary arterial dissection, thrombosis, and myocardial infarction. Other complications are related to spillage of ethanol to non-targeted myocardium resulting into interventricular conduction blocks, and infarction of non-selected regions (5, 6).

Retrograde coronary venous ethanol ablation (RCVEA) can potentially avoid the aforementioned technical difficulties of TCEA. Catheter instrumentation in the coronary veins lacks the risks of arterial damage, and retrograde flow drives spilled ethanol towards the right atrium where it is diluted to non-damaging concentrations. This approach was first described by Wright *et al.* in dogs (7) but, to our knowledge, has not yet been tested on

© 2012 The Heart Rhythm Society. Published by Elsevier Inc. All rights reserved.

Corresponding author information: Miguel Valderrabano, MD, FACC Associate Professor of Medicine, Weill Cornell Medical College Adjunct Associate Professor of Medicine (Cardiology), Baylor College of Medicine Division Chief of Electrophysiology, Methodist DeBakey Cardiology Associates, The Methodist Hospital Address: Methodist DeBakey Cardiology Associates Smith Tower 6550 Fannin, Suite 1901 Houston, TX 77030 Telephone: (713) 441-1100 mvalderrabano@tmhs.org.

Publisher's Disclaimer: This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final citable form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

Conflict of Interest: AAB (none), DJS (none), MV (Consultant to St Jude Medical, Hansen Medical, Biosense-Webster; research support St Jude Medical, Hansen Medical, Medtronic).

human subjects. Here, we describe two cases in which RCVEA successfully terminated persistent VTs refractory to multiple RFCA procedures.

Case Reports

Patient # 1 is a 65 year-old man with hypertension, hyperlipidemia, type 2 diabetes mellitus, and recurrent syncope from drug-refractory sustained VTs, who was referred to us for VT ablation. He had a normal left heart catheterization and a cardiac magnetic resonance (CMR) showing a mildly depressed left ventricular (LV) ejection fraction (45%) and small mid-myocardial scarring of the basal anteroseptum and inferoseptum in a distribution consistent with nonischemic heart disease.

The patient had undergone 3 previous failed RFCA attempts where a focal VT had been documented. Activation maps (using NaxV, St Jude Medical, St Paul, MN) of the right ventricle (RV) and LV outflow tracts, epicardium and the anterior interventricular vein (AIV) had shown the earliest activation site in the AIV (Fig 1A). RCFA in the AIV and in the earliest activation sites of LV endocardium and epicardium had previously failed to terminate VT.

The coronary sinus was cannulated with an 8F sheath used for LV lead delivery (St Jude medical, St Paul, MN) advanced from the right internal jugular vein and a multipolar catheter was advanced in the AIV. Selective AIV venograms showed two septal branches (Fig 1B). One of these branches coincided with the site where the earliest activation times in the AIV had been mapped. A 12/12 pacemap match was obtained when pacing from this site. With the help of a 6F left internal mammary artery (LIMA) angioplasty guide, the septal vein was then cannulated with a 0.014-inch angioplasty wire and occluded with a 1.5 × 6-mm balloon. Ethanol (98%, 1 cc infused over 2 minutes) was then administered without complications, achieving acute elimination of VT. A repeat venogram was obtained following the infusion. The balloon remained inflated for about 2 minutes following the infusion.

The procedure had included mapping of the pericardial space via a subxyphoid puncture, during which transient entrance in the pleural space had occurred. Patient developed an inflammatory exudative left pleural effusion, 24 hours after the procedure requiring thoracentesis that did not recur. Post-procedure, the patient was recovered with a pericardial drain left after mapping the pericardial space, which drained sero-sanguinous fluid for 24 hours. CMR performed 48 hours after the procedure demonstrated significant expansion of basal anteroseptal hyperenhancement, consistent with ethanol-induced injury (Fig 1E). The patient subsequently underwent defibrillator implantation, given his prior syncope, and history of sustained VT. After 5 months of follow-up, only nonsustained VT (maximum 12 seconds) has been recorded.

Patient # 2 is a 69 year-old male with hypertension, hyperlipidemia, atrial fibrillation, non-ischemic cardiomyopathy, complete heart block, and history of aortic valve replacement who presented to us with a VT storm leading to multiple defibrillator shocks. Previously he

had undergone three RFCA procedures with VT recurrence despite extensive ablation in the basal inferior septal aspect of the LV.

In his fourth procedure, activation maps were obtained in the LV, and epicardially, via a subxiphoid puncture (2). The earliest activation site in the LV cavity was mapped to a large scar region within the inferobasal and mid-inferior LV (Fig 2A), where the patient had previously received extensive radiofrequency ablation, as well as areas with late potentials within the border zone. The epicardial map showed earlier activation times than the endocardial map. However, a large area in the epicardium had similar activation times (Fig 2B).

We decided to map the middle cardiac vein (MCV). The coronary sinus was cannulated from the right internal jugular vein with an 8F sheath, which was then selectively directed towards MCV. A coronary sinus venogram showed the presence of several septal branches within MCV. A 4F quadripolar mapping catheter was advanced into the MCV septal branches with the help of a LIMA angioplasty guide and a perfect pacemap was obtained in the third branch (Fig 2D-H). The septal branch was then cannulated with a 0.014-inch angioplasty wire and a 1.5 × 6-mm balloon was launched. Ethanol (98%, 2 cc) was infused over 2 minutes, followed by a saline flush, without complications and with acute termination of the VT. Subsequent attempts to induce a clinical VT failed. After 4 months of follow-up, no recurrence of VT has been recorded by his defibrillator.

Intravascular ethanol ablation was considered as within standard care. Both patients were informed about the unconventional nature of the venous delivery and gave informed consent. Data were recorded under an IRB-approved protocol.

Discussion

We present two cases in which RCVEA successfully terminated drug and RFCA refractory VTs. Although RFCA with epicardial and endocardial mapping can successfully treat most refractory VTs, there remains a subset of patients whose VT is not amenable to RFCA. These are mainly VTs with deep midmyocardial origin secondary to triggered activity or transmural reentry to which radiofrequency energy cannot reach with sufficient therapeutic effect. Another group is patients with epicardial circuits and history of previous heart surgery making epicardial access difficult if not impossible.

TCEA has been shown to successfully treat RFCA refractory VTs, however, TCEA poses some risks as well. Most notably, accessing the “tachycardia-associated” artery may be difficult in patients with coronary artery stenosis; Cannulation of coronary artery always carries risk of arterial perforation; Spillage of ethanol in non-targeted vascular beds can result in AV blocks and myocardial infarction of non-selected regions. A recent retrospective study reported a complete heart block in 38% patients with previously intact AV conduction who underwent TCEA (6).

RCVEA is an attractive alternative to TCEA. RCVEA has also been shown to successfully terminate VTs in dogs (7), however, it has never been tested in human subjects. To develop this technique, we built on our extensive experience in safely using ethanol infusion of the

vein of Marshall during the treatment of atrial fibrillation (8-10). Additionally, the success of cardiac resynchronization therapy for congestive heart failure has been coupled with extensive tool development for catheter instrumentation of the coronary venous system, specifically LV coronary veins. Thus, most electrophysiologists are now familiar with navigating the coronary venous system of the LV. Complications of such instrumentation include coronary sinus dissection that is rare and most commonly inconsequential.

RCVEA can be favored over TCEA for multiple reasons. With a venous approach, a relatively unobstructed access to the capillary bed is available even in patients with severe coronary artery disease. There is less risk associated with cannulation of the coronary veins than coronary arteries. There is also less risk of damaging collateral arteries. RCVEA also remains a feasible option in patients in which previous CABG may limit access to the pericardium and to the coronary arteries for arterial ethanol ablation. However, there remain uncertainties regarding the safety and utility of RCVEA that this initial report cannot address. Although the size of the ventricular vein selected for ethanol infusion can be expected to correlate with the extent of tissue reached and ablated by ethanol, there is no control as to the extent of myocardial tissue ablated by ethanol, which may be excessive. Additionally, the venous anatomy may not always provide access to the targeted myocardium. Further studies are needed to assess remaining safety and efficacy concerns of RCVEA.

References

1. Klein LS, Shih HT, Hackett FK, Zipes DP, Miles WM. Radiofrequency catheter ablation of ventricular tachycardia in patients without structural heart disease. *Circulation*. 1992; 85:1666–1674. [PubMed: 1572025]
2. Sosa E, Scanavacca M, D'Avila A, Pilleggi F. A new technique to perform epicardial mapping in the electrophysiology laboratory. *J Cardiovasc Electrophysiol*. 1996; 7:531–536. [PubMed: 8743758]
3. Soejima K, Couper G, Cooper JM, Sapp JL, Epstein LM, Stevenson WG. Subxiphoid surgical approach for epicardial catheter-based mapping and ablation in patients with prior cardiac surgery or difficult pericardial access. *Circulation*. 2004; 110:1197–1201. [PubMed: 15337702]
4. Brugada P, de Swart H, Smeets JL, Wellens HJ. Transcoronary chemical ablation of ventricular tachycardia. *Circulation*. 1989; 79:475–482. [PubMed: 2917386]
5. Sacher F, Sobieszczyk P, Tedrow U, et al. Transcoronary ethanol ventricular tachycardia ablation in the modern electrophysiology era. *Heart Rhythm*. 2008; 5(1):62–68. [PubMed: 18180021]
6. Tokuda M, Sobieszczyk P, Eisenhauer AC, et al. Transcoronary Ethanol Ablation for Recurrent Ventricular Tachycardia after Failed Catheter Ablation: An Update. *Circ Arrhythm Electrophysiol*. 2011; 4:889–896. [PubMed: 21984361]
7. Wright KN, Morley T, Bicknell J, Bishop SP, Walcott GP, Kay GN. Retrograde coronary venous infusion of ethanol for ablation of canine ventricular myocardium. *J Cardiovasc Electrophysiol*. 1998; 9:976–984. [PubMed: 9786078]
8. Valderrabano M, Chen HR, Sidhu J, Rao L, Ling Y, Khoury DS. Retrograde ethanol infusion in the vein of Marshall: regional left atrial ablation, vagal denervation and feasibility in humans. *Circ Arrhythm Electrophysiol*. 2009; 2(1):50–56. [PubMed: 19756206]
9. Báez-Escudero JL, Morales PF, Dave AS, Sasaridis CM, Kim YH, Okishige K, Valderrábano M. Ethanol Infusion the Vein of Marshall Facilitates Mitral Isthmus Ablation. *Heart Rhythm*. Mar 6.2012 [Epub ahead of print].
10. Dave AS, Báez-Escudero JL, Sasaridis C, Hong TE, Rami T, Valderrábano M. Role of the Vein of Marshall in Atrial Fibrillation Recurrences After Catheter Ablation: Therapeutic Effect of Ethanol

Infusion. J Cardiovasc Electrophysiol. Mar 19,2012 doi: 10.1111/j.1540-8167.2011.02268.x.
[Epub ahead of print].

Figure 1.

Coronary venous mapping and ethanol infusion in patient #1. A, Activation maps of VT in the AIV and in RV and LV cavities. The earliest activation site is shown coming from within the AIV. B, Selective venogram of the AIV showed two septal branches. C, The AIV was then cannulated with a decapolar catheter and the distal poles were inserted in the septal branch. Pacemaps were obtained that matched the QRS morphology of the clinical tachycardia as shown in F. D, The septal branch was cannulated with an angioplasty wire and occluded with a 1.5 × 6 mm balloon. Ethanol (98%, 1 cc) was then injected into the septal vein. E, CMR at baseline showed a preexisting mid myocardial septal area of delayed enhancement (Pre-ethanol), that was significantly expanded after ethanol injection. F, twelve-lead electrocardiogram of the spontaneous VT (left) and paced QRS from the septal branch of the AIV (right), along with automated calculation of percentage of similarity using Bard software (Bard Electrophysiology, Boston, MA).

Figure 2.

Coronary venous mapping and ethanol infusion in patient #2. A through C, activation maps of the LV cavity, MCV and epicardial surface. The earliest LV site activates later than the MCV. The earliest sites in the epicardial surface are similar in timing to the MCV, but are spread over a larger area. D, MCV venogram showing multiple septal branches. E, cannulation of a septal branch with a quadripolar catheter. We selected the septal branch from which pacemaps matched best the clinical VT. F, wire cannulation of the targeted septal branch. G, angioplasty balloon cannulation and selective contrast injection of the targeted septal branch, in which ethanol (98%, 2 cc) was injected. H, clinical VT (top) and pacemap from the targeted septal branch, with percentages of similarity included.