Successful ventricular fibrillation functional substrate ablation via a single vascular access site

Gordon Ho, MD,*† Kurt S. Hoffmayer, MD, PharmD, FHRS,*†
Christopher T. Villongco, PhD,‡ David Vidmar, MS,§ Wouter-Jan Rappel, PhD,§
David E. Krummen, MD, FHRS*†

*From the *Department of Medicine, University of California San Diego, La Jolla, California, †Veterans Affairs San Diego Healthcare System, San Diego, California, ‡Department of Bioengineering, University of California San Diego, La Jolla, California, and §Department of Physics, University of California San Diego, La Jolla, California.*

Introduction

Ventricular fibrillation (VF) is a common cause of sudden cardiac death worldwide.[1] Patients with clinical VF may receive frequent implantable cardioverter-defibrillator (ICD) shocks and suffer poor quality of life. In patients who fail antiarrhythmic medications and do not have identifiable triggers for VF, such as premature ventricular contractions (PVCs) or monomorphic ventricular tachycardia (VT), there are currently limited options.[2] However, recent work has demonstrated the importance of rotor substrate in maintaining VF,[3–5] and that suppression of VF may be achieved via substrate ablation using either endocardial basket catheter phase mapping[6,7] or electrocardiographic imaging[8] to localize VF sources. Prior reported cases were performed with multiple venous and atrial access sheaths to facilitate mapping and ablation. It is unclear, however, if similar efficacy can safely be achieved with limited vascular access and without access to the right ventricle (RV). Additionally, the presence and prevalence of late potentials (local abnormal ventricular activities, LAVA)[9] has not been reported for rotor-sustaining sites.

In this work, we report a patient with recurrent clinical VF refractory to medications, severe peripheral arterial disease, and extensive venous thrombosis who underwent VF rotor mapping and ablation of the left ventricle (LV) via a single arterial access site. Mapping revealed 3 rotor substrate sites, predominantly without LAVA, at which ablation rendered VF noninducible with no recurrence at 6 months follow-up.

Case report

A 68-year-old man with a history of ischemic cardiomyopathy (ejection fraction 26%), coronary artery bypass graft surgery, and deep venous thromboses status post inferior vena cava filter presented with progressive ICD shocks for VF despite escalating doses of sotalol. He had 9 episodes of VF requiring defibrillation in the 3 months preceding electrophysiology study and no episodes of VT. He underwent a coronary angiogram showing patent bypass grafts and no targets for revascularization. The patient was enrolled in a Veterans Affairs San Diego Institutional Review Board–approved protocol for VF mapping and ablation.

In the electrophysiology laboratory, bilateral femoral and jugular venograms and femoral arteriograms showed occlusion of all vascular access sites (Figure 1A–C) except a diseased but patent left common femoral artery. Using ultrasound and fluoroscopy guidance, left common femoral arterial access was obtained with a micropuncture kit with serial sheath dilations. Using a 12 French steerable sheath (Vado, Terumo, Somerset, NJ), a 3.5-mm force-sensing externally irrigated ablation catheter (Tacticath, Abbott, Abbott Park, IL) was inserted into the LV via a retrograde aortic approach. Bipolar electroanatomic mapping using Velocity (Abbott) demonstrated extensive anterior, apical, inferior, and posterior scar. The ablation catheter was exchanged with a 60-mm basket catheter (FIRMap, Abbott, Figure 1D) through the steerable sheath. Of note, 3-dimensional geometry

KEYWORDS Ventricular fibrillation; Rotors; Functional reentry; Phase mapping; Catheter ablation
(Heart Rhythm Case Reports 2018;4:173–176)

Dr Ho has received grant support from the UCSD Clinical Translational Research Institute: Galvanizing Engineering in Medicine Grant. Dr Villongco has received grant support from the NIH Training Grant (HL007444). Mr Vidmar has received grant support from the American Heart Association (16PRE30930015). Dr Krummen has received grant support from the American Heart Association (10 BGIA 3500045) and the UCSD Clinical Translational Research Institute: Galvanizing Engineering in Medicine Grant. Dr Ho has equity in Vektor Medical. Dr Rappel is a co-inventor on intellectual property owned by the University of California and licensed to Abbott EP. Dr Rappel and Mr Vidmar have filed patent applications related to the identification of fibrillation sources. Dr Krummen served as a consultant to Abbott, has equity in Vektor Medical, and has received fellowship program support from Abbott, Boston Scientific, Medtronic, Biosense-Webster, and Biotronik. **Address reprint requests and correspondence:** Dr David E. Krummen, Veterans Affairs San Diego Healthcare System, 3350 La Jolla Village Dr, Cardiology Section 111A, San Diego, CA 92161. E-mail address: dkrummen@ucsd.edu.

https://doi.org/10.1016/j.hrcr.2017.12.008

KEY TEACHING POINTS

- Targeted ablation of rotational functional substrate can successfully eliminate ventricular fibrillation at 6-month follow-up off of antiarrhythmic medications.
- In patients with ischemic cardiomyopathy, critical ventricular fibrillation (VF)-sustaining substrate may be located predominantly in the left ventricle and is not necessarily associated with local abnormal ventricular activities (LAVA) and fractionation.
- Rotor mapping and ablation of VF-sustaining substrate may be safely performed with only a single arterial access site.

and voltage data were obtained carefully using the ablation catheter with contact force. Following techniques described recently,[10] optimal basket catheter sizing and positioning were guided by cardiac computed tomography, and cinegram demonstrated good endocardial contact and stable positioning, allowing accurate registration within the electroanatomic map. Sustained VF episodes were easily induced with double extrastimuli at a cycle length of 500/280/260 ms (Figure 2A) and externally defibrillated after 10 seconds. VF functional substrate mapping identified 3 stable rotor sites (Figure 2B) located in the (1) LV mid posteroseptal wall, (2) LV mid anteroseptal wall, and (3) LV mid lateral wall (Figure 2C). All 3 rotor sites were identified in the same locations in both VF episodes. Notably, 1 of 3 (33%) rotor substrate sites exhibited late potentials (LAVA). Late potentials were also seen in other areas not identified as rotor substrate sites, and were not ablated as part of this protocol. After a total 34 minutes of ablation at sites of the 3 rotor cores (estimated 9.3% of total LV

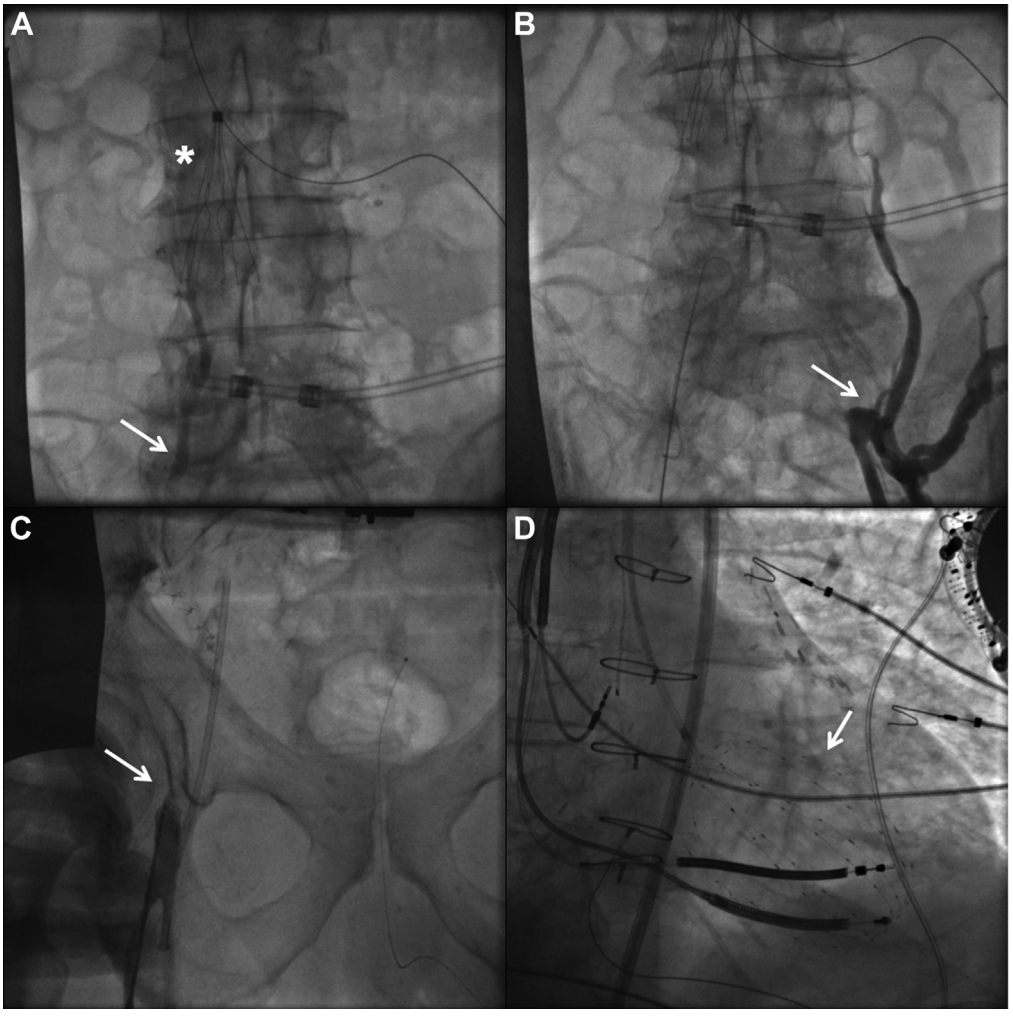

Figure 1 Limited vascular access owing to severe peripheral vascular disease and deep venous thromboses status post inferior vena cava filter (noted by the asterisk) depicted in the right (**A**) and left (**B**) femoral venograms. **C:** Right femoral arteriogram shows occlusion owing to severe peripheral arterial disease. Arrows depict the occluded vessels in A–C. An occluded superior vena cava with collateral veins was also observed (not shown for brevity). **D:** Mapping was accomplished via retrograde aortic insertion of a 60-mm basket catheter into the left ventricle via the left common femoral artery.

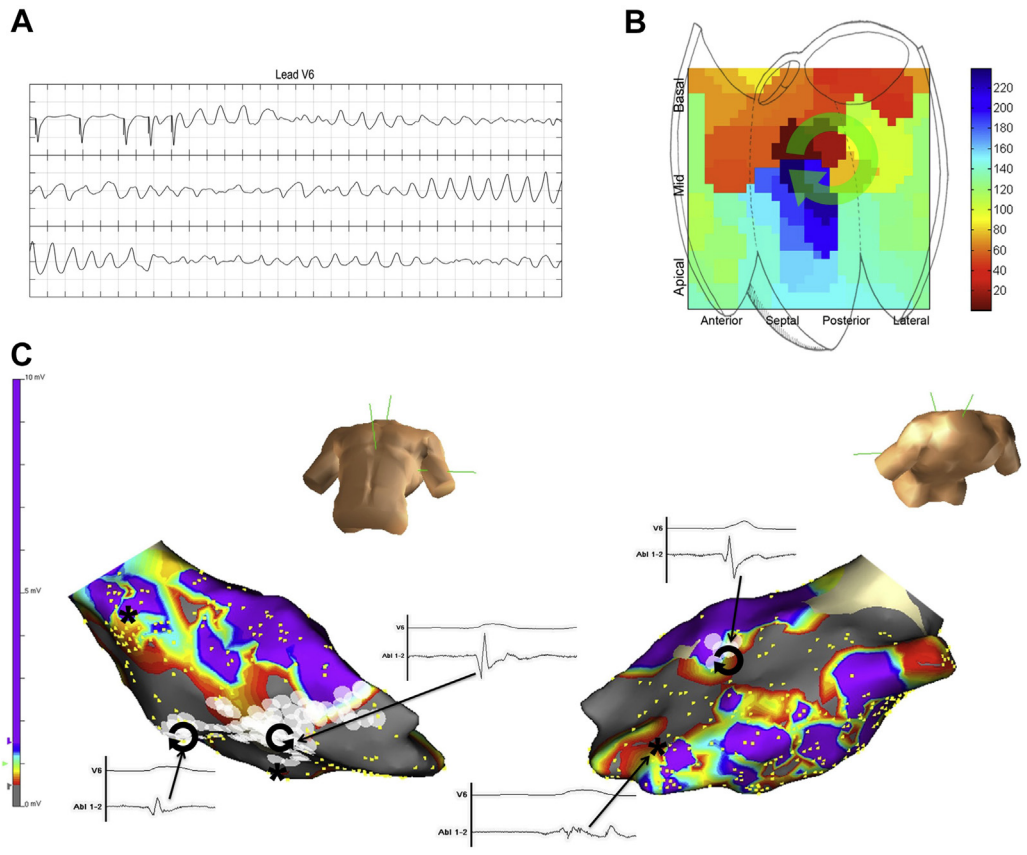

Figure 2 Ventricular fibrillation (VF) rotor substrate ablation in a 68-year-old man with ischemic cardiomyopathy. **A:** VF was easily inducible with double extrastimulus pacing at cycle length 500/280/260 ms, as shown on electrocardiography. VF substrate mapping with a single basket catheter in the left ventricle demonstrated a mid posteroseptal rotor, as represented by an isochronal map (**B**) with activation spanning the entire cycle length of 230 ms. **C:** A bipolar electroanatomical map shows significant scar burden, with arrows depicting the 3 identified rotor substrate sites with ablation lesions (*white dots*). Areas of late potentials were incidentally seen both next to ablated tissue at the rotor sites and in border zone tissue remote from the rotor sites (*black asterisks*).

endocardium), VF was noninducible despite single, double, and triple extrastimuli down to ventricular effective refractory period. The case was concluded and the patient was monitored overnight. He had no evidence of vascular complications.

After discharge, antiarrhythmic medications were not restarted. He was seen at 1, 3, and 6 months post-procedure. ICD interrogation was performed at each visit, and no sustained VT or VF events were seen through 6-month follow-up. Unfortunately, the patient was newly diagnosed with terminal stage IV lung cancer after the 6-month check-up, transitioned to hospice care, and died of respiratory failure.

Discussion

The management of isolated clinical VF refractory to antiarrhythmic drugs is challenging. For idiopathic VF, prior work has described targeting monomorphic PVCs originating in the Purkinje fibers, left ventricular septum, and anterior RV.[11,12] However, in patients without identifiable VF triggers, targeted ablation is difficult to perform. Patients with severe cardiomyopathy have extensive substrate for sustaining fibrillation. Without the ability to localize mechanistic sources, a substrate homogenization approach is possible. However, this is time-consuming, has unclear endpoints, and increases procedural duration and risks, and would require extensive ablation in many patients with advanced ischemic cardiomyopathy, such as this patient.

The mapping and targeted ablation of VF-sustaining sources is potentially an attractive therapeutic option. The patient described in this case had progressive VF refractory to medications and had a severely reduced LV function with 2 large areas of scar with extensive border zones. He did not have stereotypical PVC triggers or inducible VT.

Importantly, during voltage mapping a few sites of fractionation and late potentials were incidentally seen in ablated tissue near the periphery of the posteroseptal rotor sites as well as in areas that were remote from the rotor sites in border zone tissue that were not ablated. Conversely, there were large areas within the ablated rotor substrate tissue that did not contain late potentials. These observations support the idea that a nontargeted, substrate-only ablation of low voltage, fractionation, and late potentials may fail to identify tissue that is critical in promoting VF and may cause unnecessary ablation in areas of less importance. Unlike scar-based VT, in which late potentials may be markers of fixed, anatomical, slowly conducting isthmuses that sustain monomorphic VT, VF may be sustained not by fixed isthmuses but

rather by functional reentry caused by heterogeneities in conduction and repolarization characteristics that may not manifest as late potentials. Furthermore, a substrate-only approach in this patient would have been an extensive procedure, targeting a significant amount of bystander ventricular tissue.

This case demonstrates that targeted ablation of rotor substrate was sufficient to suppress clinical VF to 6 months follow-up off antiarrhythmic medications. If additional studies further validate the effectiveness of this approach, VF rotor substrate ablation may become a new therapeutic option in such patients.

Finally, this case demonstrates that VF substrate mapping can be accomplished using a single arterial access site. It is unknown whether RV substrate would have been identified in this patient, but ablation in LV border zone substrate alone was sufficient to suppress ventricular arrhythmias. While prior work identified VF-sustaining substrate in the RV, it was unclear if RV sites were central to sustaining VF, as VF induction was not attempted between the ablation of each site. Indeed, this case may thus serve as a hypothesis-generating observation that for ischemic cardiomyopathy, the sustaining mechanisms for VF may predominantly reside in left ventricular tissue, as is often the case in scar-based VT. In fact, it has been observed recently that rotors tended to migrate and stabilize at sites in the LV.[13] The etiology for this remains to be elucidated and may be multifactorial. Possible hypotheses include increased myocardial mass of the LV compared to the RV such that sustaining sources in the LV may dominate activation of the entire myocardium or that the LV may harbor more abnormalities in functional substrate. Future studies should evaluate the relative importance of LV and RV substrate to the maintenance of VF.

Conclusion

In this report, we describe VF substrate ablation in the LV alone via a singular vascular access site that safely and successfully rendered VF noninducible and suppressed VF at medium-term follow-up off antiarrhythmic medications. Rotor substrate sites predominantly did not exhibit LAVA, and other sites with late potentials were not ablated. These observations provide important insight into VF-sustaining substrate; larger studies of VF-sustaining substrate ablation are required to demonstrate the widespread utility of this approach.

References

1. Sasson C, Rogers MA, Dahl J, Kellermann AL. Predictors of survival from out-of-hospital cardiac arrest: a systematic review and meta-analysis. Circ Cardiovasc Qual Outcomes 2010;3:63–81.
2. Zipes DP, Camm AJ, Borggrefe M, et al. ACC/AHA/ESC 2006 Guidelines for Management of Patients With Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death: a report of the American College of Cardiology/American Heart Association Task Force and the European Society of Cardiology Committee for Practice Guidelines (writing committee to develop Guidelines for Management of Patients With Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death): developed in collaboration with the European Heart Rhythm Association and the Heart Rhythm Society. Circulation 2006;114:e385–484.
3. Gray RA, Jalife J, Panfilov AV, Baxter WT, Cabo C, Davidenko JM, Pertsov AM. Mechanisms of cardiac fibrillation. Science 1995;270:1222–1223. author reply 1224–1225.
4. Nash MP, Mourad A, Clayton RH, Sutton PM, Bradley CP, Hayward M, Paterson DJ, Taggart P. Evidence for multiple mechanisms in human ventricular fibrillation. Circulation 2006;114:536–542.
5. Krummen DE, Ho G, Villongco CT, Hayase J, Schricker AA. Ventricular fibrillation: triggers, mechanisms and therapies. Future Cardiol 2016;12:373–390.
6. Hayase J, Tung R, Narayan SM, Krummen DE. A case of a human ventricular fibrillation rotor localized to ablation sites for scar-mediated monomorphic ventricular tachycardia. Heart Rhythm 2013;10:1913–1916.
7. Krummen DE, Hayase J, Vampola SP, Ho G, Schricker AA, Lalani GG, Baykaner T, Coe TM, Clopton P, Rappel WJ, Omens JH, Narayan SM. Modifying ventricular fibrillation by targeted rotor substrate ablation: proof-of-concept from experimental studies to clinical VF. J Cardiovasc Electrophysiol 2015;26:1117–1126.
8. Cochet H, Dubois R, Sacher F, Derval N, Sermesant M, Hocini M, Montaudon M, Haissaguerre M, Laurent F, Jais P. Cardiac arrythmias: multimodal assessment integrating body surface ECG mapping into cardiac imaging. Radiology 2014;271:239–247.
9. Jais P, Maury P, Khairy P, et al. Elimination of local abnormal ventricular activities: a new end point for substrate modification in patients with scar-related ventricular tachycardia. Circulation 2012;125:2184–2196.
10. Narayan SM, Rodrigo M, Kowalewski CA, Shenasa F, Meckler GL, Vishwanathan MN, Baykaner T, Zaman JA, Wang PJ. Ablation of focal impulses and rotational sources: what can be learned from differing procedural outcomes? Curr Cardiovasc Risk Rep 2017;11:27.
11. Haissaguerre M, Shah DC, Jais P, et al. Role of Purkinje conducting system in triggering of idiopathic ventricular fibrillation. Lancet 2002;359:677–678.
12. Haissaguerre M, Shoda M, Jais P, et al. Mapping and ablation of idiopathic ventricular fibrillation. Circulation 2002;106:962–967.
13. Vidmar D, Krummen DE, Hayase J, Narayan SM, Ho G, Rappel W-J. Spatiotemporal progression of early human ventricular fibrillation. JACC Clin Electrophysiol 2017;3:1437–1446.