Catheter ablation of ventricular tachycardia using intracoronary wire mapping and coil embolization: Description of a new technique

Venkatakrishna N. Tholakanahalli, MD, FHRS,[*] Stefan Bertog, MD, FACC, FSCAI,[*] Henri Roukoz, MD,[†] Kalyanam Shivkumar, MD, PhD, FHRS[‡]

*From the *Veterans Affairs Medical Center, Minneapolis, Minnesota, [†]University of Minnesota, Minneapolis, Minnesota and [‡]UCLA Cardiac Arrhythmia Center, UCLA Health System, Los Angeles, California.*

Introduction

Management of ventricular tachycardias (VTs) arising from within the intraventricular septum can be challenging. The focus is frequently not amenable to conventional endocardial mapping and ablation. We describe 2 cases of mapping using intracoronary guidewires and subsequent coil embolization to treat intraseptal VT.

Case report: patient 1

A 74-year-old man with a history of nonischemic cardiomyopathy who underwent previous cardiac resynchronization and intracardiac defibrillator device (ICD) implantation presented with multiple episodes of sustained monomorphic VT (Figure 1A) resulting in recurrent ICD shocks despite treatment with amiodarone 400 mg daily. Therefore, he was referred for catheter ablation.

The initial electrophysiology study (EPS) showed a VT arising from the basal high ventricular septum (Figure 1A). The VT could not be entrained from any location and showed overdrive suppression. Activation mapping (Figure 1B) demonstrated a large zone of early activation with small r waves on unipolar recordings on either side of the septum. Coronary venous mapping could not be performed owing to the presence of a coronary sinus lead. Radiofrequency (RF) ablation (Thermocool SF, Biosense Webster, Diamond Bar, CA) was unsuccessful from either side of the septum. Hence, the decision was made to perform an EPS with intraseptal mapping and potential ablation with either intraseptal alcohol injection or coil embolization. Bipolar RF ablation was not performed.

Right femoral venous access was obtained, diagnostic catheters were placed, and intracardiac electrograms were recorded from atrial, His, and ventricular locations. Right (RV) and left ventricular (LV) mapping was performed with a 3.5-mm Navistar Thermocool DF mapping ablation catheter (Biosense Webster).

Programmed extra stimulus testing induced sustained VT. Phenylephrine and vasopressin were used for hemodynamic support to allow VT mapping. After activation mapping was performed on either side of the high septum anteriorly, the earliest broad zone of activation was found on RV and LV sites opposite to each other at −62 and −43 ms from the surface electrocardiogram (ECG), respectively. It was noted that lead V_6 of the surface ECG was slightly different in RV and LV sites with subtle variation in cycle length. This was felt to be due to different exit sites with a common origin. Owing to previously failed open irrigated tip unipolar sequential ablation attempts at these sites, an intraseptal origin was suspected.

Coronary arteriography showed 2 septal perforators. There was a moderate hemodynamically insignificant (fractional flow reserve under maximal hyperemia 0.89) stenosis in the mid left anterior descending (LAD) coronary artery. Two coronary guidewires were placed into the first and second septal perforators (S-1 and S-2). These were subsequently exchanged over a microcatheter for two 0.014-in Vision (Biotronik SE & CO KG, Berlin, Germany) wires (insulated body with exposed 15-mm tip). Further, 1 Vision wire was placed in the proximal LAD coronary artery. Of note, the microcatheters were left in both septal perforators, allowing only the very distal tip of the septal Vision wires to be exposed.

During intracoronary wire mapping, the proximal LAD wire was used as the anode. The distal ends of the S-1 and S-2

KEYWORDS Ventricular tachycardia; Coil embolization; Intracoronary guidewire mapping

ABBREVIATIONS **ECG** = electrocardiogram; **EPS** = electrophysiology study; **HCM** = hypertrophic obstructive cardiomyopathy; **ICD** = intracardiac defibrillator device; **LV** = left ventricle/left ventricular; **RF** = radiofrequency; **RV** = right ventricle/right ventricular; **S-1** = septal perforator-1; **S-2** = septal perforator-2; **VT** = ventricular tachycardia (Heart Rhythm 2013;10:291–295)

This work for supported by National Heart, Lung, and Blood Institute grant RO1 HL084261 (to K.S.). **Address reprint requests and correspondence:** Dr Venkatakrishna N. Tholakanahalli, Department of Cardiology, Veterans Affairs Medical Center, University of Minnesota, 111-C, One Veterans Dr, Minneapolis, MN 55417. E-mail address: thola001@umn.edu.

1547-5271/$-see front matter Published by Elsevier Inc. on behalf of the Heart Rhythm Society. http://dx.doi.org/10.1016/j.hrthm.2012.10.031

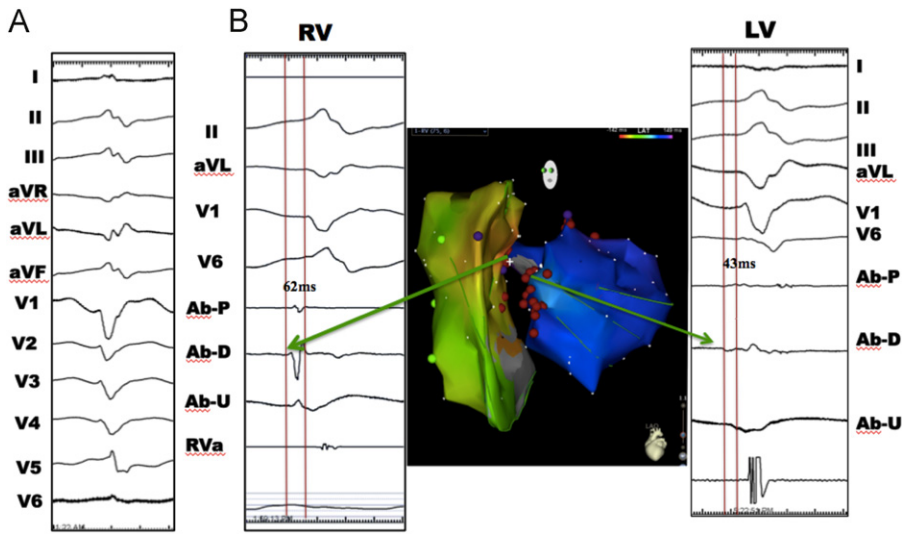

Figure 1 **A:** Twelve-lead electrocardiogram demonstrating ventricular tachycardia of patient 1. **B:** Activation mapping from the LV and RV. LV = left ventricle; RV = right ventricle.

wires were used as cathodes. Activation mapping performed by both endocardial and intraseptal approaches showed the earliest origin in the S-1 recording, 90 ms ahead of the surface ECG during VT (Figure 2A). This site was located between the earliest sites in the RV and LV, respectively. Pace mapping from S-1 showed 10 of 12 match with the clinical VT. These EPS findings narrowed the localization of the arrhythmic focus to the myocardial territory supplied by S-1. Therefore, the decision was made to proceed with intentional injury to the myocardium supplied by S-1.

Over a 0.014-in coronary wire, a (OTW) balloon catheter was positioned in S-1 without difficulty. The wire was removed and the balloon inflated at 8 atm. Cold saline was injected through the balloon into S-1, causing an increase in cycle length (Figure 2A) and termination within 30 seconds. Then, contrast was injected (1–2 cm^3) forcefully to assess for reflux into the LAD as well as for collateral flow and clearance. In addition, echo contrast diluted at 1:10 (1–2 cm^3) was injected via the balloon catheter while the balloon remained inflated to assess the area of injury by

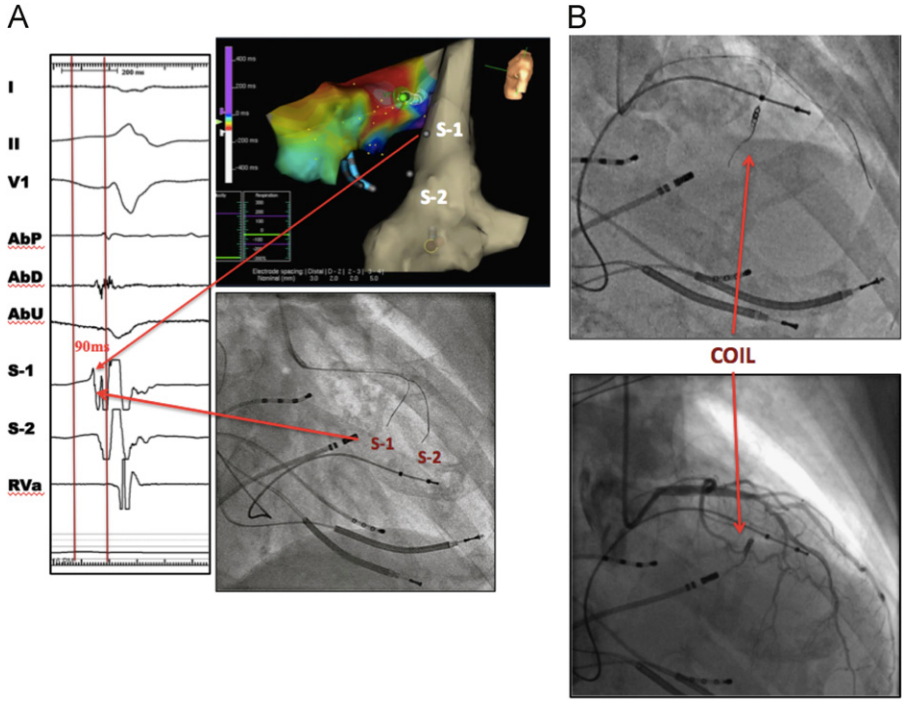

Figure 2 **A:** Three Vision wires are positioned in the proximal left anterior descending coronary artery and both septal perforators, respectively, in patient 1. Activation mapping and point-by-point timing mapping from the right ventricular septum and septal branches of the coronary arteries. **B:** Angiography demonstrating the septal perforators and coil delivery in patient 1. The final angiography confirms occlusion of the first septal perforator after coil deployment. S-1= septal perforator-1; S-2 = septal perforator-2.