Endocardial ablation of postinfarction ventricular tachycardia with nonendocardial exit sites

Mohamad C. Sinno, MD, Miki Yokokawa, MD, Eric Good, DO, Hakan Oral, MD, Frank Pelosi, MD, Aman Chugh, MD, Krit Jongnarangsin, MD, Hamid Ghanbari, MD, Rakesh Latchamsetty, MD, Fred Morady, MD, Frank Bogun, MD

From the Division of Cardiology, University of Michigan Cardiovascular Center, Ann Arbor, Michigan.

BACKGROUND Most infarct-related ventricular tachycardias (VTs) have an exit site that can be targeted by endocardial ablation. However, some VT reentry circuits have an exit site that is intramural or epicardial. Even these circuits may have an endocardial component that can be endocardially ablated.

OBJECTIVE To assess the prevalence of postinfarction VTs with a nonendocardial exit site that can be successfully eliminated by endocardial ablation.

METHODS Twenty-eight consecutive patients with postinfarction VT (27 men, age 69 \pm 8 years, ejection fraction 0.25% \pm 0.15%) were referred for VT ablation. A total of 213 VTs were inducible (cycle length 378 \pm 100 ms). Pace mapping was performed throughout the scar, and critical sites were identified for 137 VTs (64.5%). Critical sites identified by entrainment mapping and/or pace mapping were divided into exit and nonexit sites depending on the stimulus-QRS/VT cycle length ratio (S-QRS/VT CL \leq 0.3 vs > 0.3).

RESULTS Endocardial exit sites (S-QRS/VTCL \leq 0.3) were identified for 100 of 137 VTs. Only critical nonexit sites were identified for 37 of 137 (27%) VTs. Nonexit sites were confined to a smaller area within the endocardium (1.81 ± 1.7 cm²) and were located within

Introduction

Most infarct-related ventricular tachycardias (VTs) have an exit site that can be targeted by endocardial ablation. However, some VT reentry circuits have an exit site that is intramural or epicardial. Even these circuits may have an endocardial component that can be endocardially ablated. The purpose of this study was to assess the prevalence of postinfarction VTs with a nonendocardial exit site that can be successfully eliminated by endocardial ablation.

Methods Patient characteristics

Twenty-eight consecutive patients (age 69 ± 8 years; 27 men; mean ejection fraction 0.25 ± 0.15) with prior

dense scar (0.28 \pm 0.24 mV) further away from the border zone (2.05 \pm 2.79 cm) than did the VT exit sites. Exit sites had a larger area of matching pace maps (3.86 \pm 1.9 cm²; P < .01) and were at a closer distance to the border zone (0.93 \pm 1.06 cm; P < .01). A total of 133 of 137 VTs were ablated. The success rate was similar for VTs in which exit sites were targeted (n = 90 of 100) and VTs in which only nonexit sites were targeted (n = 36 of 37) (P = .83).

CONCLUSIONS In about one-third of postinfarction VTs for which critical sites were identified, the exit site was not endocardial. Critical nonexit sites that are effective for ablation are often within dense scar at a distance from the border zone and can be missed if only the border zone is targeted.

KEYWORDS Ventricular tachycardia; Postinfarction; Mapping; Ablation

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infarction (anterior = 12; inferior = 16) and VT were referred for the catheter ablation of VT (Table 1). All patients had sustained episodes of ventricular tachycardia within the preceding 6 months and had failed to respond to antiarrhythmic drug therapy. Twenty-two of 28 patients were being treated with amiodarone, 5 with mexiletine, 2 with dofetilide, 2 with sotalol, 1 with disopyramide, and 21 with beta-blockers. Five patients were being treated with a combination of amiodarone and mexiletine. Antiarrhythmic medications except amiodarone were stopped at least 5 halftimes prior to the ablation procedure if at all possible. All patients had an implantable cardioverter-defibrillator (ICD), and 22 had experienced appropriate ICD discharges.

Electrophysiologic study and mapping

After informed consent was obtained, 2 multipolar electrode catheters were introduced into a femoral vein and positioned in the right ventricular apex and the His bundle position.

Address reprint requests and correspondence: Dr Frank Bogun, Division of Cardiology, University of Michigan Cardiovascular Center, 1500 East Medical Center Dr, SPC 5853, Ann Arbor, MI 48109-5853. E-mail address: fbogun@med.umich.edu.

Table 1	Characteristics	of patients	with	infarct-	related
ventricular	r tachycardia (n	= 28)			

Age (y)	69 ± 8
Sex: male/female	27/1
Left ventricular ejection fraction (%)	25 ± 15
Site of myocardial infarction	
Anterior	12
Inferior	16
Frequent ICD discharges	22
Antiarrhythmic drug	
Amiodarone	22
Mexiletine	5
Sotalol	2
Disopyramide	1
Dofetilide	2
Beta-blocker	21

Programmed stimulation was carried out with up to 4 extrastimuli and a minimum of 3 extrastimuli using basic drive cycle lengths of 400 and 600 ms. The extrastimulus coupling intervals were shortened in a stepwise fashion to a minimum of 200 ms. Sustained VT was defined as VT lasting for > 30 seconds or requiring termination secondary to hemodynamic compromise. Programmed stimulation was repeated postablation at 2 right ventricular sites. Conscious sedation was used, not general anesthesia.

Left ventricular mapping was performed using femoral artery access and a retrograde aortic approach. An electroanatomical mapping system (CARTO, Biosense Webster, Diamond Bar, CA) was used in all patients with a 7-F mapping/ablation catheter that had an irrigated 3.5-mm tip (Thermocool, Biosense Webster). Electrograms were filtered at 50–500 Hz. The intracardiac electrograms and leads V₁, I, II, and III were displayed on an oscilloscope and recorded at a speed of 100 mm/s. The recordings were stored on optical disk (EP Med Inc, New Berlin, NJ). Systemic anticoagulation was achieved with 5000 units of heparin that was administered intravenously, followed by 1000 units per hour to maintain an activated clotting time > 250 seconds.

A voltage map of the left ventricle was generated during baseline rhythm. Low voltage was defined as a bipolar voltage amplitude of ≤ 1.5 mV.¹ Dense scar was defined as a bipolar voltage of ≤ 0.5 mV.¹ The border zone was defined as a voltage of 0.5–1.5 mV. Sites with isolated potentials were defined as sites in which a distinct potential was separated from the ventricular electrogram by ≥ 20 ms of an isoelectric segment and/or a segment with low-amplitude noise (<0.05 mV). All patients had only endocardial ablation procedures. An epicardial procedure was performed in only 1 patient in whom endocardial ablation failed to eliminate all VTs.

Pace mapping was performed within the low-voltage areas ($\leq 1.5 \text{ mV}$) throughout the entire scar. The cycle length of pace mapping was that of the targeted VTs. Pace mapping was performed uniformly throughout the low-voltage area at sites where the local electrogram differed from the prior mapping site. A total of 390 ± 150 endocardial points were sampled. Pacing was performed at 62 ± 24 sites per patient. The pace maps were visually compared to the induced VTs by the investigator in real time. When comparing the pace maps with the VT morphologies, the following aspects of the

QRS complex were analyzed: bundle branch block morphology, axis, amplitude, and QRS notching. Off-line analysis was performed subsequently by a different investigator and confirmed matching pace maps in all instances.

The density of pace mapping was 2.04 ± 0.9 points/cm² for VTs with nonexit sites and 1.53 ± 1.33 for VTs with both exit and nonexit sites (P = .15). At least 2 consecutive captured beats were required to include the pace map in the analysis. Bipolar pacing was performed at an amplitude of 10 mA at a pulse width of 2 ms. If capture failed at 10 mA, the output was increased to 20 mA. If a VT was hemodynamically tolerated, entrainment mapping was performed to identify critical sites of the reentry circuit.²

Radiofrequency ablation

Radiofrequency ablation was performed at the critical isthmus of the VT reentry circuit. For hemodynamically tolerated VTs, an isthmus was defined as a site showing concealed entrainment with matching stimulus-QRS (S-QRS) and electrogram-QRS intervals³ or where VT terminated during pacing without global capture.⁴ If VT terminated after 20 seconds of radiofrequency energy delivery, the application of radiofrequency delivery was continued for another 60-120 s. Applications of radiofrequency energy were delivered at a power of 20-50 W titrated to achieve an impedance drop of 10 Ω . A VT was considered successfully ablated if the VT terminated during radiofrequency energy delivery and could not be induced subsequently. For nontolerated VTs, a critical area was defined as the presence of a matching pace map ($\geq 10/12$ leads) with the targeted VT and the inability to induce the targeted VT postablation. Radiofrequency energy was delivered at all sites with matching pace maps. In patients in whom a clinical VT was not inducible, ablation lesions were also delivered at sites with isolated and fragmented potentials.

A voltage map was performed initially, and this was followed by pace mapping throughout the scar. Radiofrequency energy was delivered at the sites of a matching pace map. After assessing for capture postablation, the catheter was moved to an alternate site within the low-voltage area where pacing was performed again.

Data analysis

Critical sites/areas were identified with entrainment mapping and pace mapping and categorized as exit sites or nonexit sites. An exit site was defined as a site where the S-QRS interval was $\leq 30\%$ of the VT cycle length (VT CL) when pacing was performed during sinus rhythm⁵ (Figure 1A). Sites with S-QRS/VT CL > 30% were defined as nonexit sites (Figure 1B). The spatial resolution of the exit and nonexit sites was assessed as previously reported.⁶ In brief, the spatial resolution of pace-mapping sites was defined as the endocardial area encompassing sites with matching pace maps. This area was measured on the electroanatomical map with the CARTO XP software (Figure 2). The spatial resolution was arbitrarily defined as 0 cm² if there was only 1 site with a matching pace map. The distance of each paceDownload English Version:

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