Successful catheter ablation of ventricular tachycardia at a site with manifest entrainment and a long postpacing interval: What is the mechanism?

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A R T I C L E  I N F O

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1. Case presentation

A 66-year-old man, with a long history of rheumatic heart disease, valvular cardiomyopathy, and congestive heart failure, underwent a mechanical mitral and aortic valve replacement in 1995. In 2005, an implantable cardioverter defibrillator (ICD) was implanted because of nonsustained ventricular tachycardia (VT) and a reduced left ventricular function (left ventricular ejection fraction, 30%). In 2010, the patient developed a VT storm refractory to amiodarone, sotalol, lidocaine and beta-blockers, and received multiple ICD shocks. There were at least 3 clinical VTs. VT1 exhibited a right bundle-branch block (RBBB) morphology, northwest axis, and a cycle length (CL) of 560 ms; VT2 exhibited an RBBB morphology, right-axis deviation, and CL of 490 ms; and VT3 had a left bundle-branch block (LBBB) morphology, right-axis deviation, and CL of 430 ms. During the electrophysiological study, the VTs were induced by programmed ventricular stimulation, hence bundle branch reentry was ruled out. Mapping in the coronary veins during VT suggested an epicardial circuit of the tachycardia. Electroanatomical mapping in the epicardium using a NaviStar catheter (Biosense-Webster, Diamond Bar, California) revealed the presence of a wide low-voltage area in the apical region. During the induced VT1 a presystolic potential (PP) was recorded from this area, which preceded the onset of the QRS by 60 ms (Fig. 1A). Entrainment pacing with a 9.9 V output (1.0 ms width) was performed from this site at a CL of 500 ms. The surface ECG during entrainment pacing showed some degree of fusion. The interval between the pacing stimulus and onset of the QRS complex (S-QRS) was 160 ms, whilst the interval from the electrogram recorded at the pacing site to the QRS onset during the VT (Eg-QRS) was just 60 ms. The post-pacing interval (PPI) was 680 ms (120 ms longer than the VT–CL). A radiofrequency (RF) energy application using open irrigation (30 ml/min, 35 W) was performed at this site. The VT was terminated 4 s after initiation of RF energy delivery (Fig. 1B). During the RF energy application the VT–CL, and interval between the QRS and next PP, gradually extended, and the VT terminated by block between the QRS and PP. The recording at the same site during sinus rhythm demonstrated an isolated delayed potential (IDP) after the QRS. After several additional RF energy deliveries at adjacent sites, both VT1 and VT2 became non-inducible. What is the mechanism underlying this successful ablation at this site with manifest entrainment and a long PPI?

2. Commentary

The concept of entrainment mapping for ablation of VT is that targeting the site will interrupt the reentrant VT circuit. This site must be located, not only within the circuit, but also in a protected and relatively narrow isthmus. During entrainment mapping, the QRS morphology, S-QRS interval, and PPI should be checked. If the site is within the VT circuit, and within a relatively protected zone of slow conduction, the following characteristics should be observed.

- The surface 12-lead ECG during entrainment pacing from this site should be an exact match for that recorded during the spontaneous VT (entrainment without fusion or concealed entrainment).
- The S-QRS interval during entrainment should be equal to the Eg-QRS + 20 ms during the VT. The relationships between the orthodromically captured electrogram components should remain identical during entrainment pacing and the spontaneous VT.
- The PPI at the pacing site should be equal to the VT–CL + 30 ms. After the pacing is stopped the electrogram at the pacing site should again be an activated one VT–CL after the last pacing stimulus. If this site is within the circuit, after the
last pacing stimulus, the stimulated wavefront will transverse the VT circuit to again reach this site.

Ablation at sites that demonstrated combinations of these favored characteristics resulted in VT termination in approximately 37% of applications [1]. In the present case, the successful ablation site exhibited manifest entrainment, a PPI longer than the VT–CL by 120 ms, and an S-QRS interval longer than the Eg-QRS by 100 ms. Why did ablation at this site successfully suppress the VT?

If you check the electrogram at the ablation site carefully you can detect a small fragmented electrogram before the PP (Fig. 1A). This diastolic potential (DP) preceded the onset of the QRS complex by 220 ms and the last stimulus to the DP was 560 ms, which was equal to the VT–CL. During the entrainment pacing with a 9.9 V output both the DP and PP might be simultaneously captured. To confirm that the DP was within the VT circuit, selective entrainment pacing of the DP was performed prior to the RF energy application. When the output was reduced to 8.0 V, the PP appeared after the stimulus and the DP was selectively captured (Fig. 2A). The surface ECG suddenly became identical to VT1, and the S-QRS interval increased to 220 ms. After stable entrainment pacing of the DP, the PPI was measured again (Fig. 2B). At that time the PPI was 600 ms and just 20 ms longer than the VT–CL. The Eg (DP)-QRS interval during the VT was 220 ms, was equal to the S-QRS interval during the entrainment pacing of the DP. Furthermore, the S-QRS interval was 38% of the VT–CL, suggesting it was located within the central isthmus of the circuit [1]. Therefore, we delivered RF energy at this site and successfully suppressed VT1. Why did both VT1 and VT2 become non-inducible after the RF energy applications in this area? A possible explanation is that those circuits had a common pathway. Before the ablation we performed pace mapping from this area (Fig. 3). Pacing was started at a high output and then the output was reduced until capture was lost. Initially the paced QRS configuration was similar to VT1, and then changed to a QRS configuration similar to VT2, and finally became a similar configuration to VT3. In fact, VT3 also became non-inducible following

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**Fig. 1.** (A) Entrainment pacing with a 9.9 V output. The surface ECG during entrainment pacing showed some degree of fusion (asterisk). The S-QRS was 160 ms; while the Eg-QRS was just 60 ms. The PPI was 680 ms (120 ms longer than VT–CL). The small artifacts are the atrial pacing stimuli from the implantable defibrillator. (B) VT termination by RF energy delivery. See text for further discussion. ABL=ablation catheter; IDP=isolated delayed potential; PP=presystolic potential; S=Stimulus.
Fig. 2. (A) Output reduction during entrainment pacing. When the output was reduced to 8.0 V, the PP appeared after the stimulus and the DP was selectively captured. The surface ECG suddenly became identical to VT1 (asterisk), and the S-QRS interval increased to 220 ms. (B) The PPI after entrainment pacing with an 8.0 V output. The PPI was 600 ms. The E-QRS interval was 220 ms, which was equal to the S-QRS interval. See text for further discussion.

Fig. 3. Pace mapping before the ablation. Pacing started with a high output and then the output was reduced until capture was lost. Initially the paced QRS configuration was similar to VT1, and then changed to a QRS configuration similar to VT2, and finally it became a similar configuration to VT3.
the additional RF energy applications to an opposite site of the low voltage area. During a 2-year follow-up, when the patient received low-dose amiodarone, no episodes of VT recurrence were detected.

Entrainment mapping is a powerful tool for VT ablation but must be used correctly to avoid confusion. Pacing at as low an output as possible, to consistently capture the myocardium, is important to avoid any far-field capture, and which electrogram has been captured should always be considered.

**Conflict of interest**

None.

**Reference**