Journal of Arrhythmia 29 (2013) 47–49

Contents lists available at SciVerse ScienceDirect

Journal of Arrhythmia

journal homepage: www.elsevier.com/locate/joa

EPS for Resident Physicians

Mechanism of alternans of diastolic potential cycles during overdrive pacing of ventricular tachycardia

Yoshiaki Kaneko, MD, PhD*, Tadashi Nakajima, MD, PhD, Tadanobu Irie, MD, Toshimitsu Kato, MD, Takafumi Iijima, MD, Masaki Ota, MD, Mio Tamura, MD, Takashi Iizuka, MD, Masahiko Kurabayashi, MD, PhD

Department of Medicine and Biological Science, Gunma University, Graduate School of Medicine, 3-39-22 Showa-machi, Maebashi, Gunma 371-8511, Japan

ARTICLE INFO

Article history:
Received 17 July 2012
Received in revised form
8 August 2012
Accepted 15 August 2012
Available online 23 October 2012

Keywords:
Diastolic potentials
Entrainment pacing

1. Case presentation

A 77-year-old man with a history of healed inferior myocardial infarction underwent radiofrequency catheter ablation for drug-refractory ventricular tachycardia (VT) with right bundle branch block and superior axis. VT was reproducibly induced by programmed ventricular stimulation. Pacing during VT at the basal edge of the low voltage zone, where a low-amplitude diastolic potential (DP) was recorded, showed concealed entrainment, in which the pacing stimulus-QRS was nearly equal to the DP-QRS interval, and the post-pacing interval (PPI) of the DP was equal to the tachycardia cycle length (TCL), consistent with pacing on the essential pathway of the reentrant circuit (Fig. 1). The delivery of radiofrequency energy at that site terminated and eliminated the induction of VT. Before ablation, overdrive pacing with identical output and a cycle length slightly shorter than that of the VT near the successful ablation site caused alternans of the DP to DP intervals (Fig. 2). What is the mechanism of this DP alternans?

2. Commentary

Understanding the electrophysiological phenomenon illustrated in Fig. 2 requires the accurate identification of the myocardial tissue captured by pacing in the reentry circuit. First, the morphology, amplitude, and timing of the local ventricular electrograms and DP relative to the QRS complex recorded from the ablation catheter during VT in Fig. 2 are slightly, though distinctly, different from those shown in Fig. 1. This suggests that the location of the tip of the ablation catheter, while near, was not strictly the same during both episodes of overdrive pacing. Second, the very short intervals between the DP and the following pacing stimuli suggest that, unlike in Fig. 1, pacing could not capture the essential pathway because it was refractory. Third, in contrast to Fig. 1, every even-numbered paced cycle in Fig. 2 shows (a) no latency of the pacing stimulus-QRS complex, (b) a slight change in the morphology of the QRS, and (c) a shortening of the pacing stimulus-ventricular electrogram, suggesting that the even-numbered cycles represent direct capture of the ventricular myocardium outside the zone of slow conduction. The wavefront of the 2:1 captures propagated antidromically to the ventricle and orthodromically advanced the DP and subsequent ventricular electrogram, while the interval between the DP preceding and that following the non-captured stimuli was similar to the TCL, causing 2:1 alternans of the DP cycles and ventricular electrograms. Furthermore, an oscillation in the intervals between the DPs preceding and those following captured stimuli was observed, whereas the interval between the DP preceding and that following non-captured stimuli was relatively constant. These phenomena were observed even during overdrive pacing at a shorter cycle length, and probably represented decremental conduction properties between captured ventricular myocardium and the recording site of the DP.

* Corresponding author. Fax: +81 27 220 8158.
E-mail address: kanekoy@gunma-u.ac.jp (Y. Kaneko).

1880-4276/$ - see front matter
http://dx.doi.org/10.1016/j.joa.2012.09.003

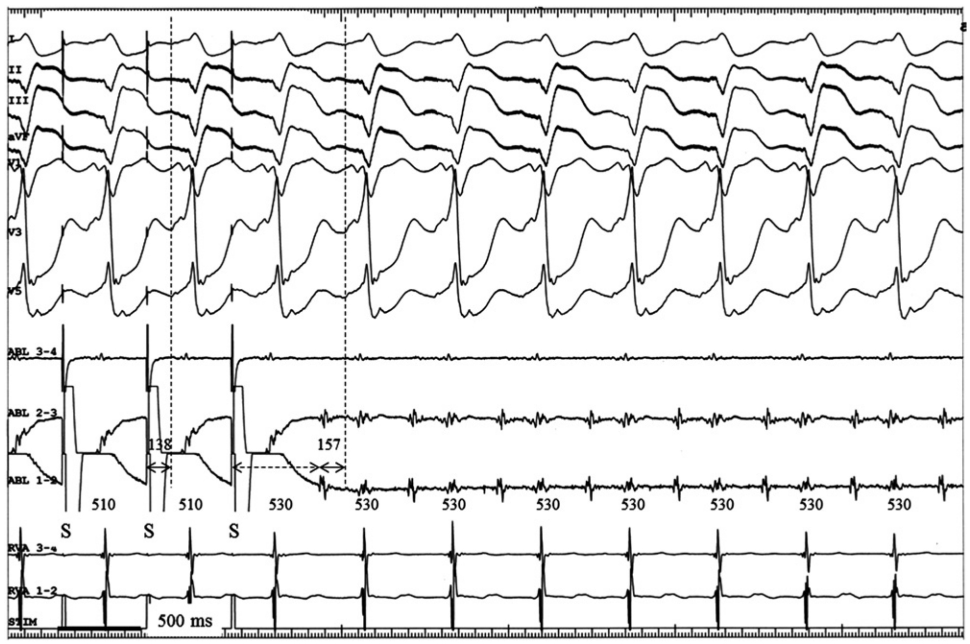

Fig. 1. Surface electrocardiograms and intracardiac recordings during entrainment pacing from electrode 1 to electrode 2 of the ablation catheter (ABL), at a cycle length of 510 ms. The QRS morphology during pacing and during VT was identical, consistent with concealed entrainment. The 138-ms interval (left bidirectional solid arrow) between the pacing stimuli (S) and QRS onset (left vertical dashed line) is nearly equal to the 157-ms interval (right solid bidirectional arrow) between the diastolic potential (DP) and the onset of the QRS (right vertical dashed line). The 530-ms post-pacing interval of the DP (dashed bidirectional arrow) is equal to the 530-ms TCL. The interval between S and the orthodromically captured ventricular electrogram recorded from the distal right ventricular apex (RVA) was 270 ms. See text for further explanations. I, II, III, aVF, V1, V3, and V5 = surface electrocardiogram; ABL 3 and 4–1 and 2 = proximal and distal bipoles of the ABL; RVA 3 and 4–1 and 2 = proximal and distal bipoles of the RVA.

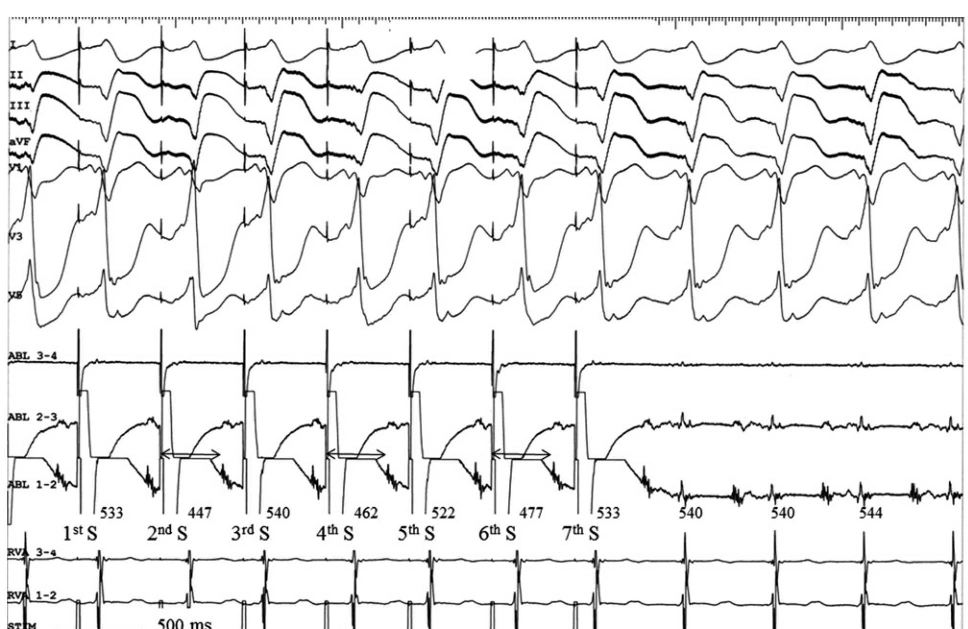

Fig. 2. Surface electrocardiograms and intracardiac recordings during entrainment pacing from electrode 1 and 2 of the ABL at a cycle length of 500 ms. Alternans of the DP cycles (shown in ms) is present. The 2nd, 4th, and 6th pacing stimuli (S) capture the QRS complexes, confirmed by the absence of an S-QRS delay and fused QRS morphology, and capture the next ventricular electrogram recorded from the distal RVA at an interval 120 ms shorter than in Fig. 1, suggesting antidromic captures of the ventricle. The post-pacing intervals (indicated by bidirectional horizontal solid arrows) between the 2nd, 4th, and 6th S and the next DP are all approximately 390 ms shorter than the tachycardia cycle length. The QRS morphology following the 1st, 3rd, 5th, and 7th S is identical to that during VT, consistent with no capture of the ventricular myocardium. See text for further explanations. Abbreviations as in Fig. 1.

The measurement of the PPI of DP following entrainment pacing usually consists of measuring the interval between the last capturing stimulus and the next DP. This technique is generally based on the assumption that, in the presence of 1:1 capture, pacing depolarizes the zone of slow conduction in the reentry circuit that generates the DP [1,2]. In the present case, the interval between the last stimulus and the following DP might be misidentified as the PPI, if one believes that entrainment pacing directly captured the zone of the slow conduction in a 1:1 manner despite the lack of capture of the ventricular myocardium by the last stimulus.

The 2:1 captures were comparable to single extrastimuli delivered at a fixed coupling interval in a bigeminal pattern during VT. When the extrastimulus penetrates the zone of slow conduction, its return cycle is identical to the PPI after entrainment pacing [3,4]. Because 2:1 pacing captured the ventricular myocardium only outside the zone of slow conduction, the interval between the pacing stimulus and the ventricular deflection following the subsequent DP should be measured as the return cycle. However, the ventricular deflections during entrainment pacing were superimposed upon the pacing artifact from the non-captured stimulus. While one might assume that the ventricular deflections in timing have the same interval between the DP and the ventricular deflection as that during VT, the true PPI measured between the captured spike and the presumed ventricular deflection would be longer than the TCL, suggesting a remote bystander as the captured ventricular myocardium outside the isthmus, which might be located beneath the sheet of “higher threshold scar tissue” [5].

In conclusion, 2:1 capture of the remote bystander advanced DP in a 2:1 manner and caused alternans of the DP interval during VT. This observation makes an important contribution to the identification of the PPI during atypical entrainment pacing of a scar-related VT.

Conflict of interest

The authors have no conflict of interest to disclose.

References

[1] Stevenson WG, Khan H, Sager P, et al. Identification of reentry circuit sites during catheter mapping and radiofrequency ablation of ventricular tachycardia late after myocardial infarction. Circulation 1993;88:1647–70.

[2] Stevenson WG, Khan H, Sager P, et al. Identification of reentry circuit sites during catheter mapping and radiofrequency ablation of ventricular tachycardia late after myocardial infarction. Circulation 1993;88(4 Pt 1):1647–70.

[3] Kay GN, Epstein AE, Plumb VJ. Resetting of ventricular tachycardia by single extrastimuli. Relation to slow conduction within the reentrant circuit. Circulation 1990;81:1507–19.

[4] Callans DJ, Hook BG, Josephson ME. Comparison of resetting and entrainment of uniform sustained ventricular tachycardia. Further insights into the characteristics of the excitable gap. Circulation 1993;87:1229–38.

[5] Kocovic DZ, Harada T, Friedman PL, et al. Characteristics of electrograms recorded at reentry circuit sites and bystanders during ventricular tachycardia after myocardial infarction. J Am Coll Cardiol 1999;34:381–8.