Successful catheter ablation of intraseptal ventricular tachycardia from the entrance side of the slow conduction zone

Masato Okada, MD, Koji Tanaka, MD, Akinobu Mizutani, Yusuke Ikada, BS, Nobuaki Tanaka, MD

From the Cardiovascular Center, Sakurabashi Watanabe Hospital, Osaka, Japan.

Introduction

Catheter ablation is an established treatment for ventricular tachycardia (VT). However, the intraseptal substrate remains a challenge for mapping and delivery of radiofrequency energy.[1–3] Recent advances in high-resolution electroanatomic mapping systems help us identify the reentrant VT circuit. However, endocardial activation mapping often exhibits a centrifugal spread during intraseptal VT, making it challenging to complete the delineation of the circuit. Additionally, the identification of intraseptal local electrograms of the critical slow conduction zone (SCZ) is difficult unless electrode catheters are inserted into septal perforators. Moreover, isolated septal substrates often require emergent technologies and experimental ablation approaches such as bipolar ablation, radiofrequency needle ablation, and chemical ablation.[4] These anatomical and technical difficulties limit the overall success of catheter ablation of intraseptal VTs, and exploring alternative techniques is demanding. Here we present a case of macroreentrant VT that was successfully ablated by targeting the intraseptal "entrance" of the critical SCZ identified by entrainment maneuvers.

KEY TEACHING POINTS

- Macroreentrant ventricular tachycardia (VT) with an intraseptal substrate exhibits a centrifugal spread, making it challenging to complete the delineation of the circuit.
- The localization of the slow conduction zone (SCZ) in the intraseptum would be more convincingly elucidated when the "entrance" and "exit" can be identified on the ventricular septum.
- Manifest entrainment and orthodromic capture of the earliest activation site indicate that the pacing site is proximal to the SCZ. When pacing from a limited area exhibits those findings, the pacing site would be located on the entrance side of the SCZ.
- Catheter ablation strategy targeting the entrance of the SCZ is an alternative method of intraseptal VT ablation, especially in cases with exit ablation failure.

Case report

A 69-year-old man with cardiac sarcoidosis was referred to our hospital for an electrical storm with repetitive appropriate implantable cardioverter defibrillator therapies. Hemodynamically stable and monomorphic wide QRS tachycardia on the 12-lead electrocardiography was incessantly observed in the emergency room. The VT rate was 108 beats per minute (tachycardia cycle length [TCL] = 560 ms), and the QRS morphology was a right bundle branch block configuration with left axis deviation (Figure 1B), suggesting that the exit of the VT was the inferior septum in the left ventricle. Physical examination, laboratory data, and echocardiographic data revealed no evidence of acute coronary syndrome or myocarditis. Hypokinetic motion and thinning (4 mm) of the left ventricular (LV) basal septum remained unchanged, with an LV ejection fraction of 42%. Treatment using medications (i.e., amiodarone 200 mg, carvedilol 20 mg, sacubitril/valsartan 200 mg, and prednisone 5mg) could not completely prevent the occurrence of the VT.

After obtaining informed consent, we performed catheter ablation of the VT. A multipolar electrode catheter (EP star Fix AIV; Japan Lifeline, Tokyo, Japan) was placed into a distal branch of the great cardiac vein. Another multielectrode catheter (PentaRay; Biosense Webster, Diamond Bar, CA) was inserted into the left ventricle via a transseptal approach.

KEYWORDS Entrainment; Catheter ablation; Ventricular tachycardia; Slow conduction zone; Postpacing interval
(Heart Rhythm Case Reports 2023;9:524–528)

Funding Sources: This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors. Disclosures: We thank Mr. John Martin for his linguistic assistance with the manuscript. **Address reprint requests and correspondence:** Dr Masato Okada, Cardiovascular Center, Sakurabashi Watanabe Hospital, 2-4-32 Umeda, Kita-ku, Osaka, 530-0001, Japan. E-mail address: masato.okada1105@gmail.com.

https://doi.org/10.1016/j.hrcr.2023.05.002

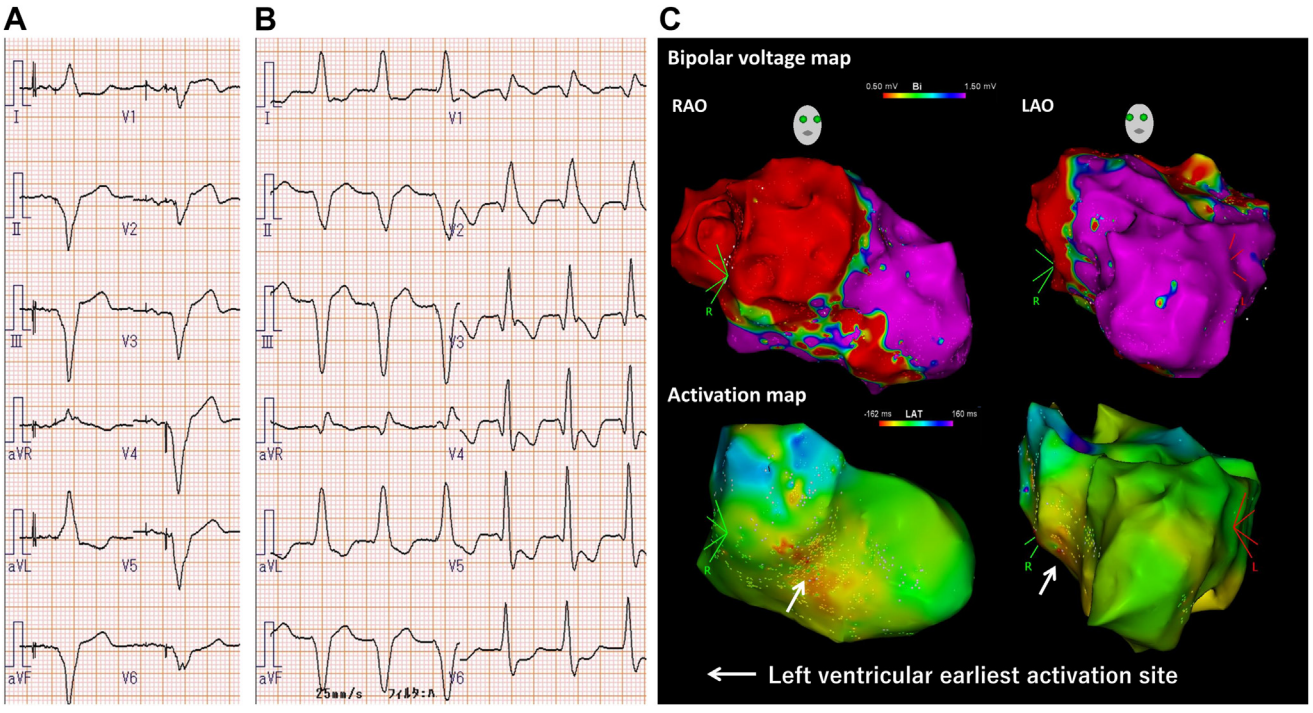


Figure 1 Surface 12-lead electrocardiograms: **A:** at baseline; **B:** during ventricular tachycardia. **C:** The electroanatomical maps recorded by the CARTO3 system (Biosense Webster, Diamond Bar, CA) are shown. The upper panel shows a bipolar voltage map (0.50–1.50 mV) during right ventricular pacing, and the lower panel shows an activation map during ventricular tachycardia. LAO = left anterior oblique, RAO = right anterior oblique.

Subsequently, heparin was administered to achieve an activated clotting time of 250–300 seconds. Electroanatomic mapping was performed using the CARTO3 mapping system (Biosense Webster, Diamond Bar, CA). First, voltage mapping of the left ventricle was performed during right ventricular (RV) pacing using a PentaRay catheter. Low-voltage areas, defined as a bipolar electrogram amplitude of <0.5 mV, were found in the ventricular septum, coinciding with thinning areas at the LV basal septum (Figure 1C).

Programmed ventricular stimulation at pacing cycle lengths of 600 and 400 ms repeatedly induced the clinical VT. Constant fusion and progressive fusion during overdrive pacing supported the tachycardia with a macroreentrant mechanism. Activation mapping of the left ventricle demonstrated a centrifugal spread with the earliest activation site (EAS) located in the LV inferior septum. The total activation time within the LV endocardium was 322 ms, which was much shorter than the TCL of 560 ms. Ventricular overdrive pacing on the LV-EAS exhibited near-concealed fusion, with a postpacing interval (PPI) equal to the TCL and an electrogram QRS equal to the stimulus QRS of 28 ms (Figure 2A). These findings suggested that the site was the "exit" of the VT. Radiofrequency energy in the range of 35–40 W was initially delivered to the LV-EAS during VT using an irrigated tip catheter (ThermoCool ST SF; Biosense Webster, Diamond Bar, CA). However, ablation of the LV-EAS did not modify the VT.

After placing a PentaRay catheter on the LV-EAS, we checked the local electrograms of the contralateral RV septum. Low-amplitude and fragmented local electrograms were observed at that site; however, the electrograms did not precede the QRS onset. Ventricular overdrive pacing exhibited entrainment with manifest fusion, a stimulus QRS that was not equal to the electrogram QRS, and a PPI–TCL = 102 ms, which indicated that the location was outside the VT circuit. However, the LV-EAS recorded by the PentaRay catheter was orthodromically captured by the pacing (Figure 2B), indicating that the pacing site was located proximal to the SCZ. These findings were repeatedly observed by pacing from an approximately 2 cm^2 area on the contralateral RV septum (Figure 3A). Pacing outside the 2 cm^2 area exhibited a PPI–TCL $\geq$100 ms and antidromic capture of the LV-EAS. Although the RV septum itself was outside the circuit, the critical SCZ was estimated to be located within the ventricular septum between the pacing site and LV-EAS.

We applied 40 W of radiofrequency energy from the RV septum targeting the entrance of the SCZ, which successfully terminated the VT in 7.6 seconds. However, the VT was still inducible and radiofrequency energy was repeatedly delivered from the entrance side until the ablation index reached 500. The procedure was completed without any complications after confirmation of the noninducibility of the clinical VT. The patient was discharged 3 days after the ablation with a continuation of amiodarone (100 mg/day). During 10 months post ablation, no VT occurred until this report was written.

Discussion

Successful ablation of scar-related VT relies on the identification and controlled modification of arrhythmogenic

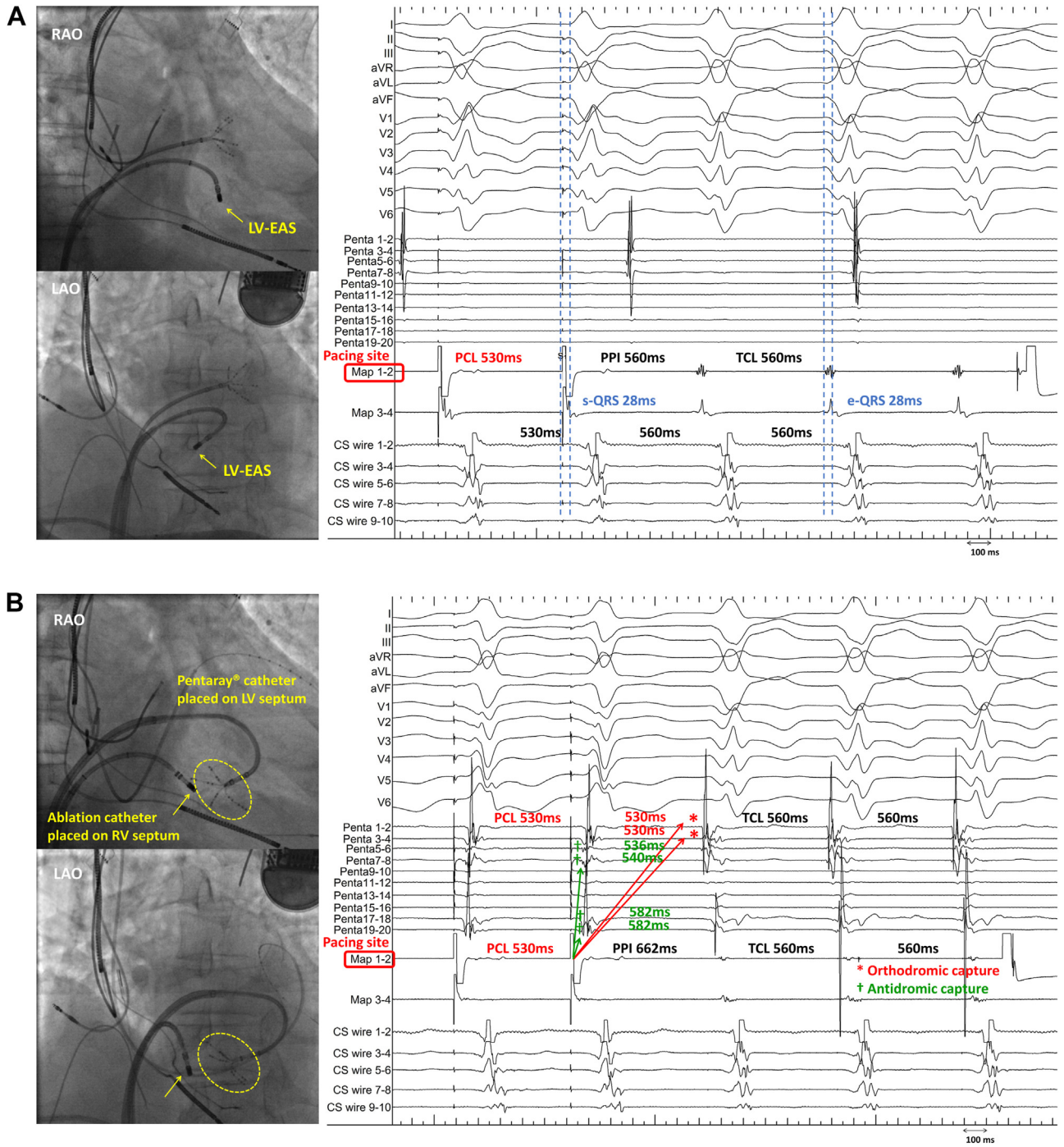
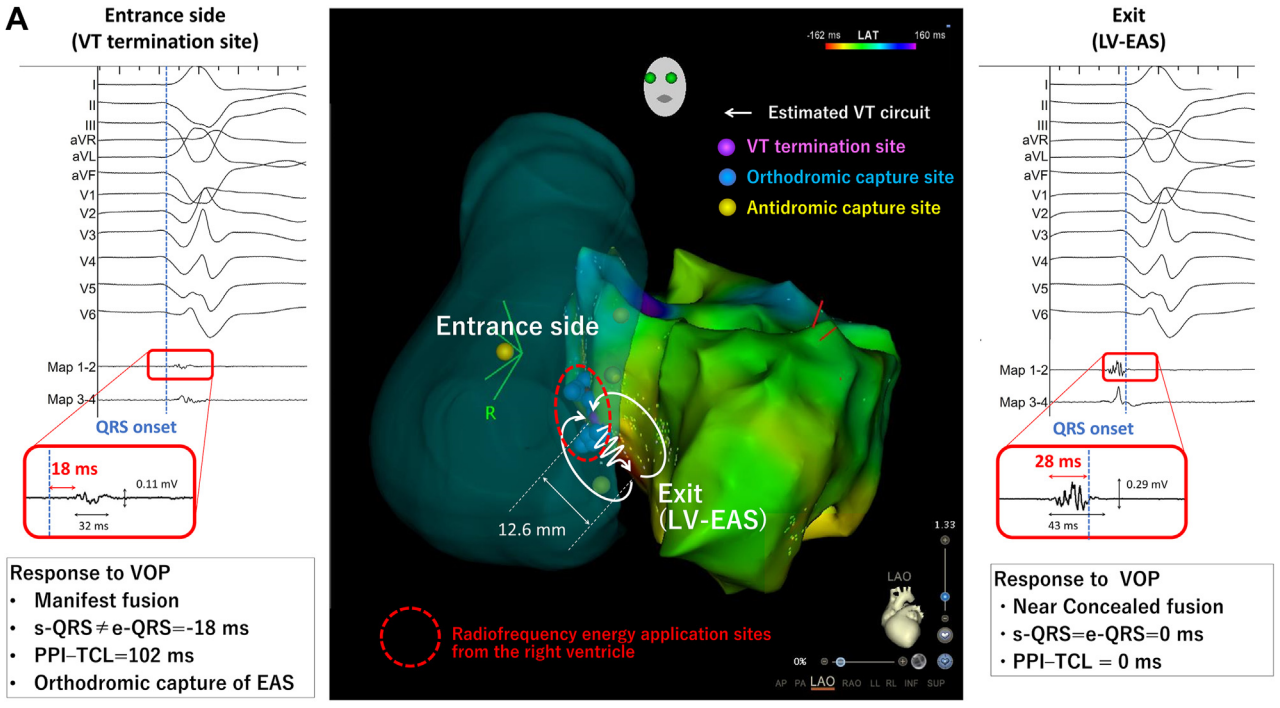


Figure 2 Fluoroscopic image and intracardiac electrograms during ventricular overdrive pacing (VOP) at the radiofrequency energy application sites. **A:** Placement of the MAP catheter on the left ventricular earliest activation site (LV-EAS) revealed fragmented potentials that preceded the surface QRS complex by 28 ms during ventricular tachycardia (VT). VOP from the LV-EAS exhibited entrainment with near-concealed fusion, the stimulus QRS equal to the electrogram QRS, and a PPI–TCL = 0 ms. The findings indicated that the LV-EAS was the "exit" of the VT circuit. **B:** After the PentaRay catheter (Biosense Webster, Diamond Bar, CA) was placed to cover the LV-EAS, the MAP catheter was placed on the opposite side of the LV-EAS on the right ventricular (RV) septum. A tracing during VOP delivered from the MAP catheter is presented. The red asterisks indicate the electrograms captured by the last pacing stimulus, occurring at a cycle length of 530 ms. The electrograms at the LV-EAS during VT (Penta 1–2 and 3–4) are orthodromically captured with long conduction intervals via the slow conduction zone (SCZ). The same polarity of the local electrograms during entrainment and tachycardia also supports the orthodromic conduction. Conversely, the other electrograms are antidromically captured with reversed polarity during entrainment and tachycardia (indicated by the green dagger). The presence of the SCZ between the pacing site and the LV-EAS (orthodromic capture site) is suggested. I, II, III, aVR, aVL, aVF, V_1, V_2, V_3, V_4, and V_5 represent the surface electrocardiography; CS 1–2 to 19–20 represent the distal-to-proximal CS recordings; MAP 1–2 represent the electrograms of the ablation catheter; and Penta 1–2 to 19–20 represent the electrograms of the PentaRay catheter. CS = coronary sinus; e-QRS = electrograms-QRS; LAO = left anterior oblique; PCL = pacing cycle length; PPI = postpacing interval; s-QRS = stimulus QRS; TCL = tachycardia cycle length.



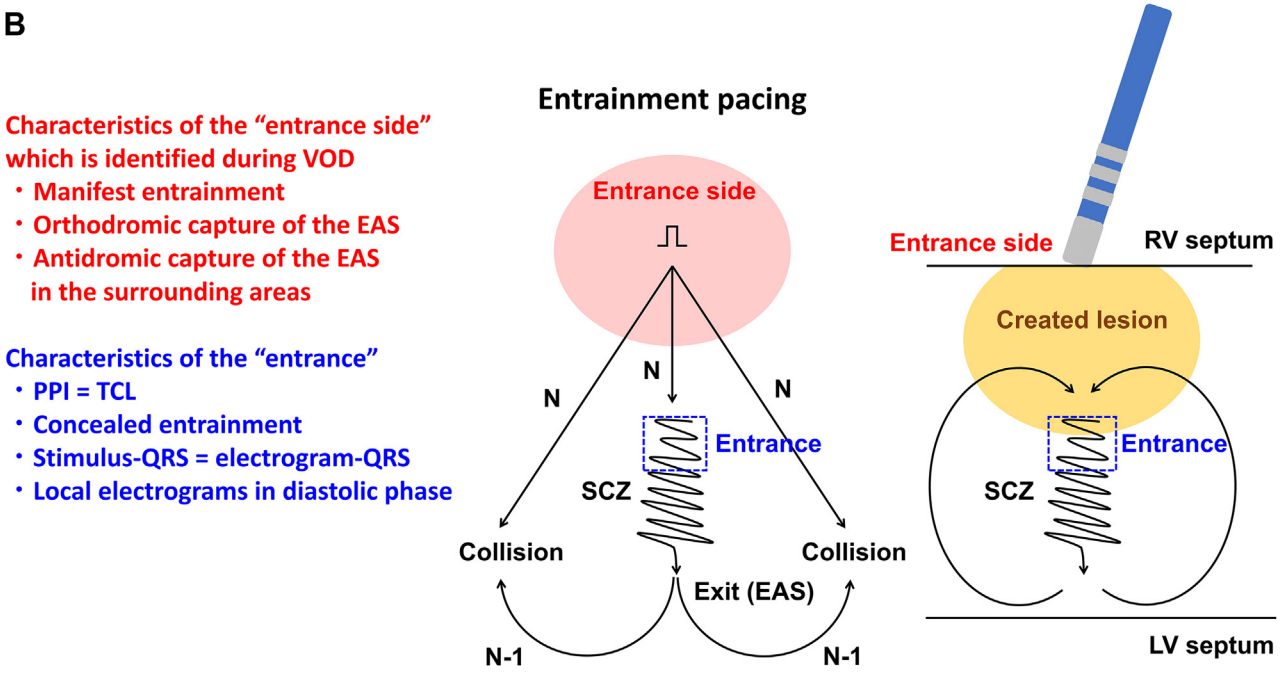


Figure 3 **A:** The local electrograms were compared between the initial ablation site (ie, LV-EAS) and the VT termination site (entrance side). Abnormal, low-amplitude, fractionated electrograms were observed at both sites. Although the first deflection of the local electrograms preceded the QRS onset by 28 ms in the LV-EAS, the electrograms at the VT termination site did not precede the QRS onset. The distance between the LV-EAS and VT termination site is 12.6 mm, and the anatomical distance of the isthmus is estimated to be small. **B:** Schematic representation of the conceptual differences between the "entrance" and "entrance side" of the SCZ. Because orthodromic activation of the EAS can theoretically occur at any site proximal to the isthmus, it is important to ensure that the orthodromic capture site is confined to a limited area and that antidromic activation of the EAS can be observed in the surrounding area to estimate the entrance side of the SCZ. The septal myocardium prevents the catheter from approaching the "entrance" of the SCZ. Regardless of the prolonged PPI–TCL (102 ms), radiofrequency energy applications from the entrance side would successfully affect the entrance of the tachycardia deep inside the septum. Abbreviations are as in Figure 2.

substrates within the critical SCZ. The interventricular septum is an area where the critical SCZ is commonly distributed but is difficult to access by the endocardial 3D mapping system. However, we identified the "exit" and "entrance side" of the SCZ using concealed and manifest entrainment, respectively. Radiofrequency energy applications from the entrance side successfully terminated the VT, probably owing to modifications of the entrance of the SCZ deep inside the septum (Figure 3B).

Entrainment pacing is still important with increasing awareness of the 3D nature of VT circuits. Concealed entrainment with a PPI equal to the TCL strongly suggests that the pacing site is located on the critical SCZ within the circuit.[5] Manifest entrainment and orthodromic capture of the EAS demonstrate that the pacing site is proximal to the SCZ.[6,7] Certainly, orthodromic capture of the EAS can theoretically occur at any site proximal to the SCZ. However, at a fixed pacing rate, orthodromic and antidromic capture of the EAS would help indicate the entrance side of the SCZ.

During entrainment with manifest fusion, the pacing stimuli continuously reset the tachycardia. The orthodromic wavefront advances the tachycardia, whereas the antidromic wavefront (N) collides with the preceding orthodromic wavefront (N-1). Antidromic capture occurs when the pacing impulse arrives at the EAS before the preceding wavefront and directly activates the EAS. Conversely, if the preceding wavefront reaches the EAS before the pacing impulse, the EAS is activated by the orthodromic conduction after passing through the SCZ. The location of the pacing site relative to the SCZ significantly impacts whether the EAS is activated orthodromically or antidromically during overdrive pacing at a fixed pacing rate. Orthodromic conduction occurs when the pacing site is close to the entrance of the SCZ, whereas antidromic conduction occurs when the pacing site is close to the exit of the SCZ (Supplemental Figure 1). If pacing from a limited area exhibits orthodromic capture of the EAS, the pacing site is believed to be proximal to the SCZ and would theoretically be closer to the entrance of the SCZ than to the adjacent antidromic capture sites. In this case, orthodromic activation of the LV-EAS was only observed in a limited area (2 cm^2) of the RV septum. Although not convincingly proven, manifest entrainment and orthodromic capture of the EAS would provide supportive information that the location is closer to the entrance of the SCZ than to the sites where pacing exhibits antidromic capture of the EAS.

Several limitations should be acknowledged. First, the pacing rate is an important modifier that would influence the result of entrainment pacing. A shorter pacing cycle length might have expanded the antidromic penetration, and the EAS might have been captured antidromically, even at the same site where orthodromic capture of the EAS is observed. Second, it is necessary to measure the time between the pacing site and EAS electrogram or PPI–TCL for a more accurate determination of the proximal site of the entrance. The shorter the time, the more proximal the pacing site is to the entrance. Although we aimed to ablate the entrance of the SCZ, the intraventricular septum did not allow the catheter to approach the entrance within the septum. Third, detailed activation mapping was not performed in the right ventricle owing to the risk of dislodging the implantable cardioverter-defibrillator lead. However, the absence of any diastolic potentials and the prolonged PPI–TCL on the RV septum indicated the minimal contribution of the right ventricle to the VT circuit. Finally, bipolar ablation and radiofrequency needle ablation would be useful for intraseptal VTs. However, those procedures are only available in limited hospitals. The correct identification of the "exit" and "entrance side" may minimize the tissue destruction created using radiofrequency energy.

Conclusion

This case highlights the usefulness of entrainment pacing in the detection of intraseptal substrates of the VT circuit. Although the exact boundaries of the reentry circuit have not been convincingly defined, we elucidated the "exit" and "entrance side" of the intraseptal SCZ using left and right entrainment pacing. Radiofrequency energy applications from the entrance side successfully terminated the VT. Ablation targeting the entrance of the circuit can be an alternative method in patients with intraseptal VT, especially when they are refractory to an exit ablation.

Appendix

Supplementary Data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrcr.2023.05.002.

References

1. Haqqani HM, Tschabrunn CM, Tzou WS, et al. Isolated septal substrate for ventricular tachycardia in nonischemic dilated cardiomyopathy: incidence, characterization, and implications. Heart Rhythm 2011;8:1169–1176.
2. Liang JJ, D'Souza BA, Betensky BP, et al. Importance of the interventricular septum as part of the ventricular tachycardia substrate in nonischemic cardiomyopathy. JACC Clin Electrophysiol 2018;4:1155–1162.
3. Yoshida K, Yokokawa M, Desjardins B, et al. Septal involvement in patients with post-infarction ventricular tachycardia: implications for mapping and radiofrequency ablation. J Am Coll Cardiol 2011;58:2491–2500.
4. Berte B, Derval N, Sacher F, Yamashita S, Haïssaguerre M, Jaïs P. A case of incessant VT from an intramural septal focus: ethanol or bipolar ablation? HeartRhythm Case Rep 2015;1:89–94.
5. Stevenson WG, Friedman PL, Sager PT, et al. Exploring postinfarction reentrant ventricular tachycardia with entrainment mapping. J Am Coll Cardiol 1997;29:1180–1189.
6. Okumura K, Olshansky B, Henthorn RW, Epstein AE, Plumb VJ, Waldo AL. Demonstration of the presence of slow conduction during sustained ventricular tachycardia in man: use of transient entrainment of the tachycardia. Circulation 1987;75:369–378.
7. Yamabe H, Okumura K, Morihisa K, et al. Demonstration of anatomical reentrant tachycardia circuit in verapamil-sensitive atrial tachycardia originating from the vicinity of the atrioventricular node. Heart Rhythm 2012;9:1475–1483.