

Reconsidering the utility of manifest entrainment pacing in macroreentrant ventricular tachycardia: A case report



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Introduction

Entrainment pacing is a useful maneuver not only for establishing reentry as a mechanism of tachycardia, but also for identifying a critical component of the circuit.^{1,2} Concealed entrainment with postpacing interval (PPI) equal to the tachycardia cycle length (TCL) strongly suggests the presence of the pacing site on the critical slow conduction zone (SCZ) within the circuit.^{1,2} On the other hand, manifest entrainment and orthodromic capture of the earliest activation site (EAS) can demonstrate that the SCZ is located between the pacing site and the EAS.³ Manifest entrainment-guided catheter ablation targeting the “entrance” to the SCZ is an effective method in the treatment of atrial tachycardia originating from the vicinity of the atrioventricular node⁴ and tricuspid annulus.⁵ However, limited information is available regarding the effectiveness of the manifest entrainment-guided strategy in the treatment of ventricular tachycardia (VT). We present a case of macroreentrant VT that was successfully ablated by targeting the midmyocardial “entrance” of critical SCZ identified using manifest entrainment pacing.

Case report

A 44-year-old man with unknown cardiomyopathy was referred to our hospital for an electrical storm with repetitive appropriate implantable cardioverter-defibrillator shocks. A hemodynamically stable and monomorphic wide QRS

KEY TEACHING POINTS

- Entrainment pacing can help identify critical components of macroreentrant tachycardias even without a complete delineation of the circuit.
- Localization of the slow conduction zone (SCZ) in the midmyocardium would be more convincingly elucidated with sandwiched entrainment from the endocardium and epicardium.
- Manifest entrainment-guided catheter ablation strategy targeting the entrance of the SCZ is an alternative method for ventricular tachycardia ablation.

tachycardia on the 12-lead electrocardiogram was incessantly observed in the emergency room (Supplemental Figure 1A). The VT rate was 145 beats per minute (TCL = 412 ms), and the QRS morphology exhibited a right bundle branch block configuration with left axis deviation, suggesting that the location of the VT origin was inferior-posterior in the left ventricle (LV). The physical examination, laboratory data, and echocardiographic data revealed no evidence of acute coronary syndrome or myocarditis. The hypokinetic motion and thinning (5 mm) of the LV posterior wall remained unchanged with an LV ejection fraction of 48%. Landilol and dexmedetomidine made the VT less frequent; however, treatment with medication could not completely prevent the VT occurrence, and a long-term therapy-free survival was strongly recommended, which made performing catheter ablation for the VT appropriate.

After informed consent was obtained, multipolar electrode catheters were inserted into the femoral vein and positioned in the great cardiac vein and left ventricle via a transeptal approach. Subsequently, heparin was administered to achieve an activated clotting time of 250–300 seconds. The surface electrocardiogram and bipolar intracardiac

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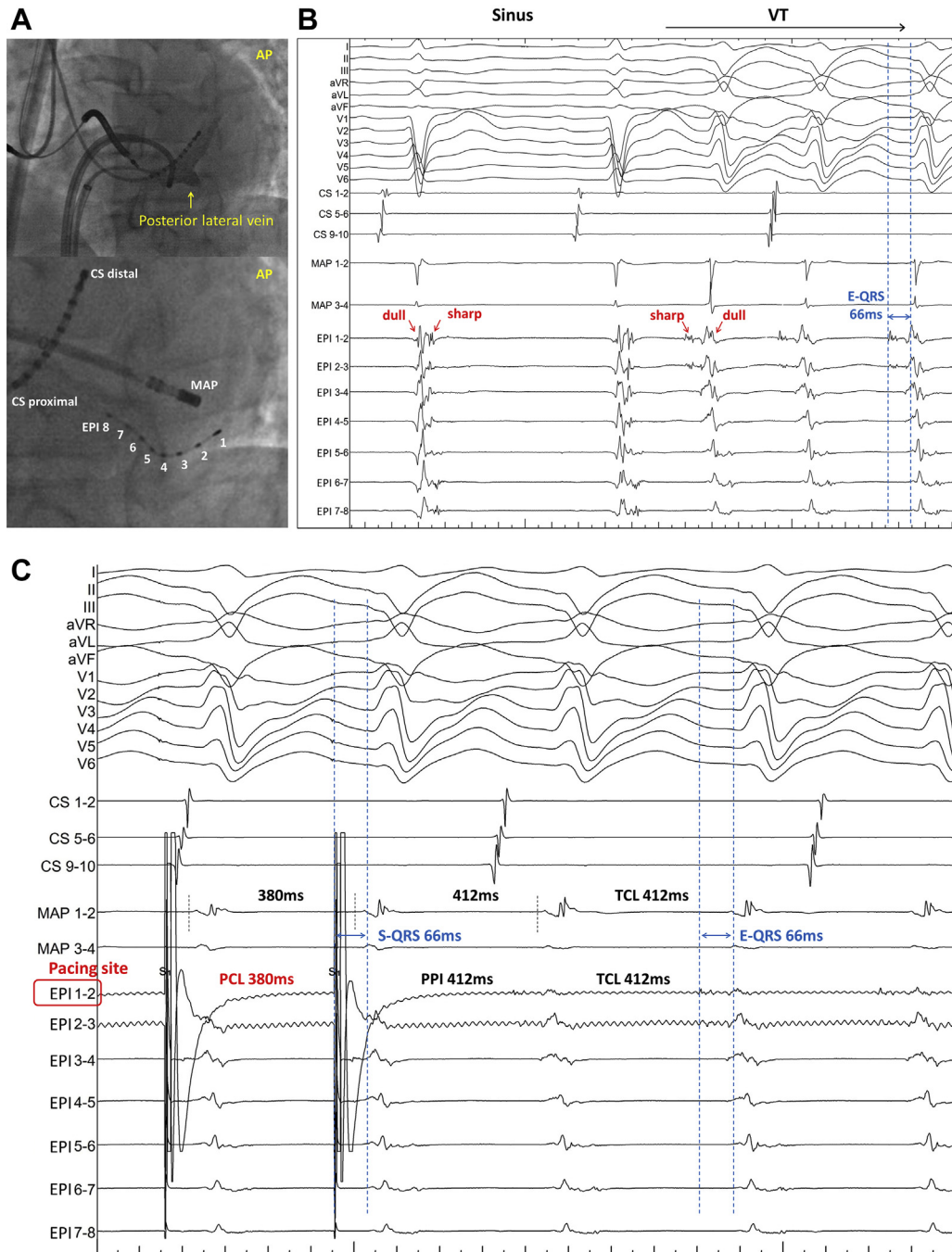


Figure 1 After insertion of a small electrode catheter into the posterior lateral vein and placement of the MAP catheter on the endocardial surface opposite to the vein (A), intracardiac electrograms were evaluated during both sinus rhythm and the ventricular tachycardia (VT) (B). After 2 sinus beats, the VT was spontaneously initiated. The sharp potential preceded the surface QRS complex by 66 ms during the VT, and the electrodes of the EPI 1-2 became the earliest activation sites of the entire electrodes. Entrainment pacing from the distal small electrodes (EPI 1-2) revealed entrainment with concealed fusion (C). I, II, III, aVR, aVL, aVF, V1, V2, V3, V4, and V5 represent the surface electrocardiogram leads; CS 1-2 to 19-20 represent the distal to proximal coronary sinus recordings; MAP 1-2 represents the electrograms of the ablation catheter; and EPI 1-2 to 7-8 represent the distal-to-proximal recordings of the small electrode catheter. CS = coronary sinus; E-QRS = electrograms-QRS; PCL = pacing cycle length; PPI = postpacing interval; S-QRS = stimulus-QRS; TCL = tachycardia cycle length.

electrograms were continuously monitored and stored on a computer-based digital recording system (LabSystem PRO; Bard Electrophysiology, Lowell, MA). The bipolar electrograms were filtered from 30 to 500 Hz. Electroanatomic mapping was performed using the CARTO3 mapping system (Biosense Webster, Diamond Bar, CA). First, voltage mapping of the LV was performed during sinus rhythm using a

multielectrode PentaRay catheter (Biosense Webster). LV scarring, defined as a bipolar electrogram amplitude of <1.5 mV, was patchy, with a predilection for the basal inferior septum (Supplemental Figure 1B). The endocardial unipolar voltage map showed additional remote low voltage area around the inferior posterior of the LV (Supplement Figure 1C).

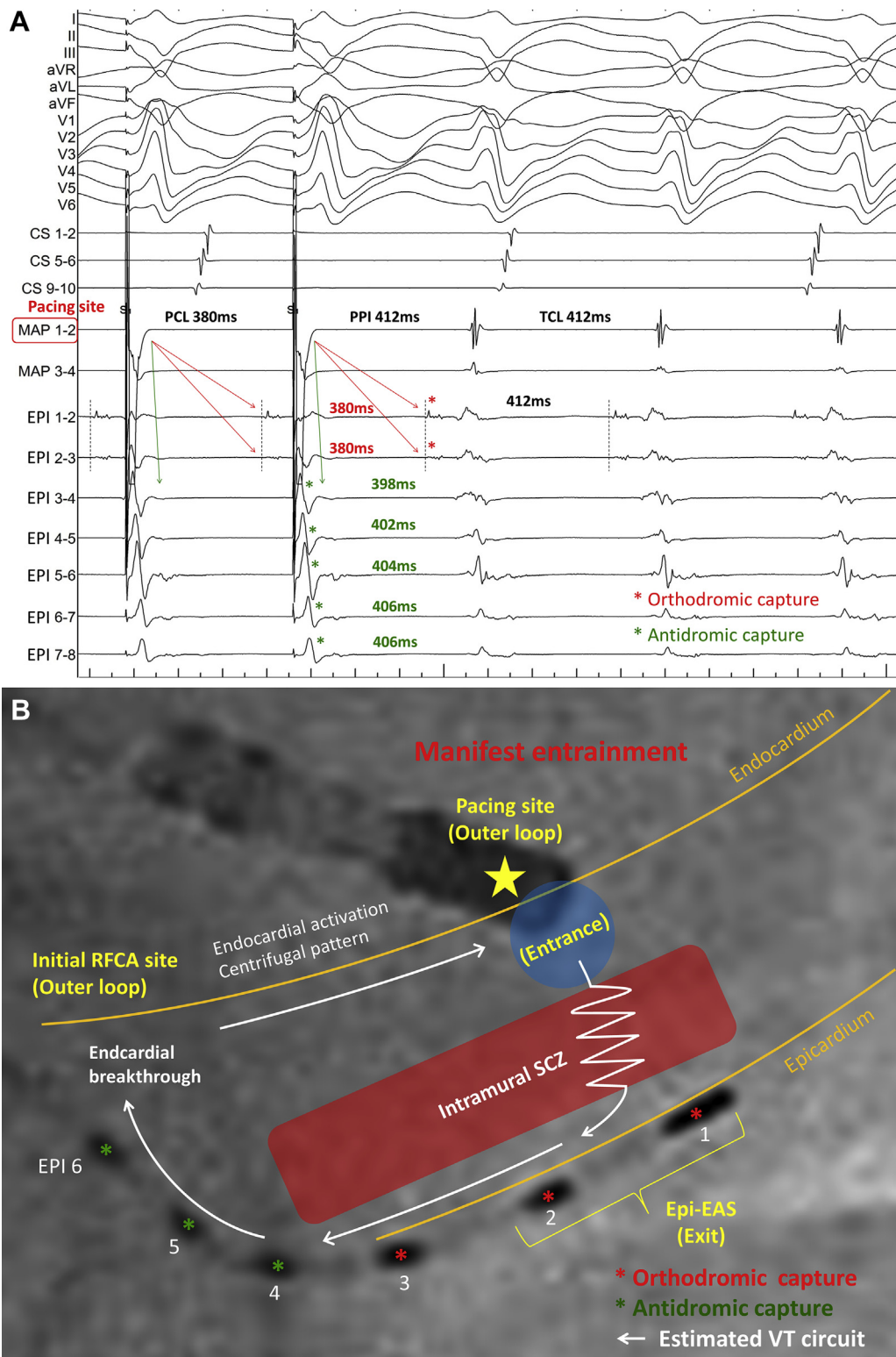


Figure 2 A: Tracing during manifest entrainment by pacing delivered from the MAP catheter placed on the opposite side of the epicardial earliest activation site (epi-EAS) is presented. The asterisks indicate the electrograms captured by the last pacing stimulus, occurring at the cycle length of 380 ms. The distal electrograms of the small microelectrode catheter (EPI 1-2 and 2-3) are orthodromically captured with long conduction intervals via the slow conduction zone (SCZ) (red asterisks), while the paroxysmal electrograms are antidromically captured (green asterisks). The presence of the SCZ between the pacing site and epi-EAS (orthodromic capture site) was suggested. B: Estimated ventricular tachycardia (VT) circuit is shown with a white arrow. Given the limited mapping and unknown captured area during entrainment pacing, it is also possible that the VT circuit is completely midmyocardium/epicardium. CS = coronary sinus; PCL = pacing cycle length; PPI = postpacing interval; RFCA = radiofrequency catheter ablation; TCL = tachycardia cycle length.

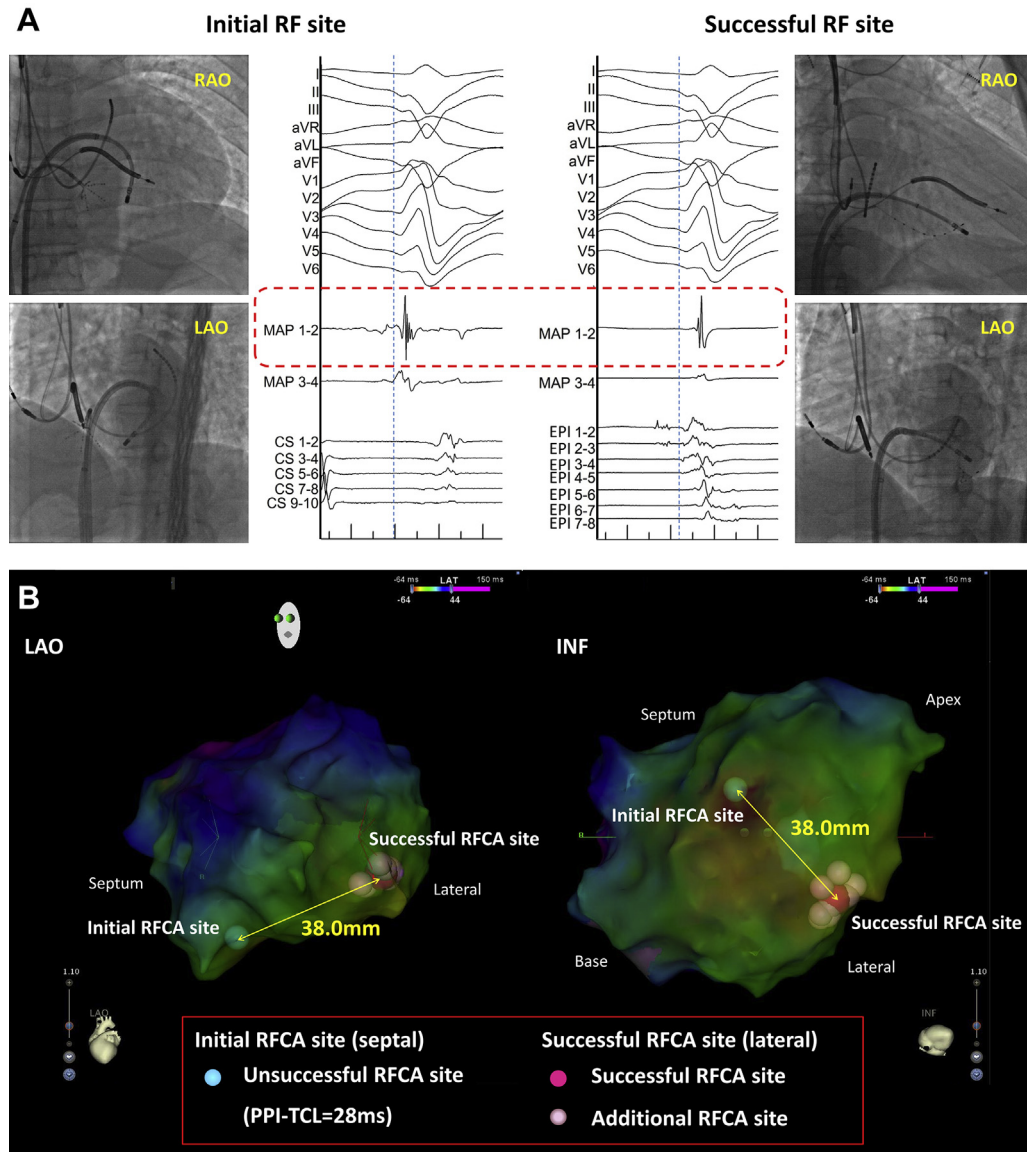


Figure 3 **A:** The fluoroscopic positions and local electrograms were compared between the initial radiofrequency catheter ablation (RFCA) site and successful RFCA site. Abnormal, low-amplitude, fractionated electrograms preceding the QRS were observed at the initial RFCA site. However, we did not record such abnormal potentials at the successful RFCA site. **B:** The anatomical position projected by the CARTO activation map (Biosense Webster, Diamond Bar, CA) showed that the successful RFCA site was inferior and lateral to the left ventricle and 38 mm remote from the initial RFCA site. INF = inferior; LAO = left anterior oblique; RAO = right anterior oblique; RFCA = radiofrequency catheter ablation.

Pacing maneuvers were conducted using bipolar electrodes with fixed output (5 mA at 1.0 ms pulse width) generated by the stimulator (SEC-5104; Nihon Kohden, Tokyo, Japan). Programmed ventricular stimulation at paced cycle lengths of 600 and 400 ms repeatedly induced the clinical VT. Constant fusion and progressive fusion during the overdrive pacing supported the tachycardia with a macroreentrant mechanism. Activation mapping of the LV endomyocardium demonstrated a centrifugal spread with the endocardial EAS (endo-EAS) located in the LV inferior septum. The total activation time within the LV endocardium was 108 ms, much shorter than the TCL of 412 ms. Radiofrequency (RF) energy in the range of 30–35 W was initially delivered to the endocardial EAS (endo-EAS) during VT using an irrigated-tip

catheter (ThermoCool ST SF; Biosense Webster). However, the ablation of the endo-EAS did not modify the VT. Ventricular overdrive pacing at that site exhibited manifest entrainment, a PPI – TCL (TCL subtracted from PPI) of 28 ms, and an electrograms-QRS equal to the paced QRS. These findings suggested that the site was in an “outer loop” of the VT. Involvement of the extra-endocardium in the reentrant circuit was suggested.

Before proceeding with the epicardial approach, we placed a microelectrode catheter (EPstar 2F Fix microcatheter; Japan Lifeline, Tokyo, Japan) in the posterior lateral vein to examine the epicardial potentials (Figure 1A). Discrete potentials (dull and sharp potentials) were recorded on the electrocardiogram (Figure 1B). The dull and sharp

potentials were considered to be the far-field LV endocardial potential and local epicardial potential, respectively. A sharp potential preceded the surface QRS complex during the VT by 66 ms and became the EAS on the epicardium (epi-EAS). Concealed entrainment, a $PPI - TCL = 0$ ms, and a $stimulus-QRS = electrograms-QRS = 66$ ms (16% of TCL) identified the epi-EAS as the “exit” of the VT circuit (Figure 1C). From the contralateral site, entrainment pacing with 5 mA at 1.0 ms pulse width was performed using distal bipoles, which produced manifest entrainment and orthodromic capture of the epi-EAS (Figure 2A). The result indicated that the “entrance” of the critical SCZ was located within the ventricular myocardium between the pacing site and epi-EAS (Figure 2B).

RF energy applications to the epi-EAS were difficult because of the small diameter of the vein and high tissue impedance. Therefore, we applied 30 W of RF energy from the endocardium targeting the SCZ between the epi-EAS (exit) and contralateral endocardial site (entrance side), which successfully terminated the VT in 7.5 seconds. Spatiotemporal dynamics of contact (10–40 g) governed by cardiac and respiratory motion increased the risk of the complications using high power. From the safety point of view, RF energy within the range of 30–35 W was repeatedly delivered until the ablation index reached 500 (estimated lesion depth = 5 mm equivalent to posterior wall thickness). No abnormal low-amplitude and fractionated electrograms, suggestive of slow conduction, were observed at the successful ablation site (Figure 3). The procedure was completed without any complications after confirming the noninducibility of the clinical VT even after an isoproterenol infusion. The patient was discharged 3 days after the CA with continuation of amiodarone (100 mg/d). During the 2-year observation period after the procedure, an appropriate implantable cardioverter-defibrillator shock for VT was experienced once in 15 months; however, no further VT storms have been observed following the ablation procedure at the time of writing this report.

Discussion

The role of entrainment in identifying the ablation target has become less important with the evolution of 3-dimensional (3D) electroanatomic mapping systems. However, increasing awareness of the limitations of a visual representation⁶ and recognition of the 3D nature of VT circuits⁷ remind us of the importance of entrainment pacing. Owing to this entrainment technique, we can identify the critical components of macroreentrant tachycardias even without the complete delineation of the circuit. In our case, involvement of the midmyocardium in the reentrant circuit prevented the visualization of the entire circuit on the endocardial 3D mapping system. However, we identified the “exit” and “entrance” of the SCZ by using concealed and manifest entrainment.

Precisely speaking, the site with manifest entrainment was an “outer loop” and was different from the “entrance” of the critical SCZ. However, in combination with orthodromic

capture of the EAS, we could identify the direction of the “entrance” from the pacing site. For a more accurate determination of the proximal site of the “entrance,” mapping during manifest entrainment is required to measure the time between the pacing and the EAS electrogram.³ The shorter the time, the more proximal is the pacing site to the “entrance.” Although the anatomical opposite of the epi-EAS does not always mean the electrically shortest endocardial site to the midmyocardial entrance, we applied RF energy from there because of the relatively short distance from the endocardial ablation site to the epi-EAS (5-mm wall thickness), which would enable the modification of the midmyocardial SCZ.

The other notable point in this case was that we could successfully approach the midmyocardial SCZ from the seemingly healthy endocardium. Conventionally, the localization of the SCZ in the midmyocardium is presumed by the discontinuity of the endocardial-epicardial activation⁷; however, manifest entrainment and orthodromic capture of the EAS by pacing from an opposite site of the EAS adds more direct electrophysiological evidence of a midmyocardial SCZ.

Discussion about the captured area is required for the accurate interpretation of entrainment pacing. Although changing pacing output can help differentiate between far-field or near-field capture, we did not examine it in the present case. However, the finding that PPI was equal to TCL (not $PPI < TCL$) at the successful ablation site indicated that the paced activation wavefront returned on time and did not advance by far-field downstream capture. This suggests that the captured area during entrainment did not include the midmyocardial SCZ. Although it is difficult to conclude how deep and how broad the endocardial pacing recruited myocardium, manifest entrainment and orthodromic capture of EAS suggests that SCZ is located between the pacing site and EAS.

Finally, although RF energy application from the epicardium is feasible, it is often limited by epicardial fat and involves potential risks, such as pericardial bleeding and collateral injury that includes the coronary vessels or phrenic nerve.^{1,2} Moreover, repetitive access to the pericardial space potentially produces pericardial adhesions, resulting in inaccessibility to the space for future treatment. A recent study has shown that RF energy applications from the endocardium can modify the epicardial substrate, especially in a region with wall thinning (<5 mm).⁸ Therefore, an initial endocardial ablation at the critical site identified using entrainment pacing would reduce the extent and in some cases the necessity of epicardial ablation.

Conclusions

This case highlighted the usefulness of entrainment pacing in the detection of a midmyocardial isthmus of the VT circuit. Although the exact boundaries of the reentry circuit were not convincingly defined, we elucidated the “exit” and “entrance” of the midmyocardial SCZ using epicardial and endocardial entrainment pacing. Manifest entrainment-guided catheter ablation strategy targeting the “entrance” of the SCZ can be an alternative method for VT ablation.

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Appendix Supplementary data

Supplementary data associated with this article can be found in the online version at <https://doi.org/10.1016/j.hrcr.2020.05.018>.

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