Utility of entrainment pacing to clarify the circuit of macroreentrant tachycardia with dual early sites on activation maps



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Introduction

Three-dimensional (3D) activation mapping is useful for identifying the substrate of macroreentrant tachycardias, such as atrial and ventricular tachycardias, that are caused by macroreentry. 1-3 To identify the reentrant circuit, the activation map should account for the entire tachycardia cycle length (TCL). 1,3 In some cases, the tachycardia cycle is longer than the total activation time (TAT) of the target chamber. In patients with a severely diseased heart and in those having undergone cardiac surgery or an ablation procedure, there can be bystander regions adjacent to the reentrant circuit that activate late, almost simultaneously with the next tachycardia beat. This phenomenon masks the actual reentrant circuit. We illustrate the utility of identifying orthodromic capture during entrainment pacing to distinguish the critical circuit of the macroreentrant tachycardia from the bystander region in a case of atrial tachycardia (AT) and a case of ventricular tachycardia (VT).

Case report A case of AT

The patient was a 65-year-old man who had undergone ablation for persistent atrial fibrillation and required radiofrequency (RF) ablation for AT. The previous ablation procedure included pulmonary vein isolation (PVI) and creation of a left atrial (LA) roof line. The electrocardiogram showed AT with a P-wave morphology that was positive/negative in the inferior limb leads and positive in V1. An activation map was obtained during AT (TCL: 205 ms) with a

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KEY TEACHING POINTS

- Three-dimensional (3D) activation mapping for macroreentrant tachycardia when the total activation time is greater than the tachycardia cycle length (TCL) is challenging because there can be bystander regions that activate late, simultaneously with the next tachycardia beat within the reentrant circuit.
- In such tachycardias, 3D mapping often shows 2 or more sites of earliest activation because the bystander region is detected as a region of early activation owing to the window of interest.
- From electrophysiological studies of an atrial macroreentrant tachycardia and a ventricular tachycardia, we propose a method in which mapping catheters are separately positioned at the 2 sites of earliest activation and entrainment pacing is performed from both sites.
- This allows us to identify the site at which the postpacing interval is identical to the TCL as the "true" site of earliest activation on the circuit and the site at which orthodromic capture of entrainment pacing from the "true" site occurs as the bystander site with "pseudo" earliest activation.

CARTO3 mapping system (Biosense Webster, Diamond Bar, CA) and use of a 20-pole, 5-spline catheter (4 mm interelectrode spacing; PentaRay NAV; Biosense Webster). The right atrial (RA) activation map accounted for only 53% (108 ms) of the TCL, and the site of earliest activation was a wide RA septal area. The LA activation map accounted for 93% (190 ms) of the TCL and revealed 2 sites of earliest activation: the roof of the LA and the posterior aspect of the left superior pulmonary vein (LSPV, Figure 1A). An ablation

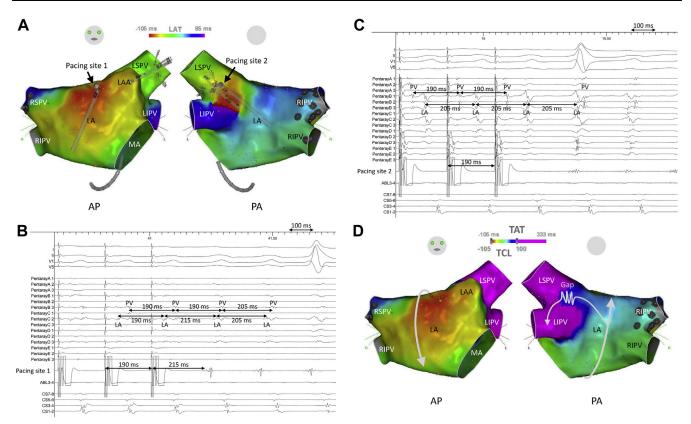


Figure 1 Electrocardiograms and 3-dimensional activation maps obtained in the case of atrial tachycardia (AT). A: Left atrial (LA) activation map of the AT. B: Entrainment pacing from the LA roof (pacing site 1, panel A). C: Entrainment from the other site of earliest activation in the left superior pulmonary vein (LSPV) (pacing site 2, panel A). D: The accurate activation map with the reannotation of the electrograms according to the tachycardia cycle length (TCL) and the results of entrainment pacing. The reentrant circuit is shown in red, yellow, green, and blue, and the bystander area is shown in pink. LA roof-dependent macroreentrant AT (black arrow) with a bystander LSPV region through the gap of the prior pulmonary vein isolation line (gray arrow) was diagnosed. ABL = ablation catheter; AP = anterior-posterior; CS = coronary sinus; LAA = left atrial appendage; LAT = local activation time; LIPV = left inferior pulmonary vein; MA = mitral annulus; PA = posterior-anterior; PV = pulmonary vein; RIPV = right inferior pulmonary vein; RSPV = right superior pulmonary vein; TAT, total activation time.

catheter and PentaRay catheter were positioned at the LA roof (Figure 1A, pacing site 1) and LSPV (Figure 1A, pacing site 2), respectively, and entrainment pacing from the LA roof (pacing site 1) revealed that the post-pacing interval (PPI) (215 ms) was almost identical to the TCL of 205 ms, suggesting that the LA roof was included in the circuit (Figure 1B). Furthermore, the last pacing stimulus from the LA roof also captured the LSPV 1 cycle after LA electrogram activation. In contrast, entrainment pacing from the LSPV (pacing site 2) failed to capture the LA potentials despite capture of the pulmonary vein potentials, suggesting unidirectional exit block at the prior PVI line (Figure 1C). LA roof-dependent macroreentrant AT was diagnosed, and delivery of RF energy to the LA roof eliminated the AT and was followed by repeat LSPV isolation. We created an accurate activation map by reannotation of the electrograms according to the TCL and the results of entrainment pacing. Correctly annotated, the LA TAT was 438 ms and greater than the TCL of 205 ms (Figure 1D).

A case of VT

The patient was a 79-year-old man in whom VT developed after he had undergone coronary stenting for treatment of

extensive anteroseptal myocardial infarction and then endoventricular circular patch plasty (Dor procedure) for a left ventricular (LV) apical aneurysm. The QRS complex morphology was that of left bundle branch block with an undetermined axis and a TCL of 350 ms. Right ventricular (RV) and LV activation mapping showed a centrifugal activation pattern with 2 sites of earliest activation: the RV apical septum and the LV apical septum. TAT of both ventricles on the 3D map accounted for 45% (156 ms) of the TCL, suggesting a focal VT mechanism rather than macroreentry (Figure 2A). However, entrainment pacing from the RV apex (site of earliest activation) showed concealed entrainment and a PPI-TCL of 5 ms with a short stimulus-QRS interval of 15 ms (Figure 2B). Concealed entrainment with a PPI identical to the TCL and a long stimulus-QRS interval of 320 ms was also obtained by pacing from the LV apex (the other site of earliest activation) opposite the RV site (Figure 2C). Furthermore, the last pacing stimulus also captured the basal aspect of the LV (LV potentials in the coronary sinus), as well as the QRS, until the next tachycardia beat at the LV apex, indicating that LV apical potentials were activated 1 cycle before the QRS and the basal LV. With this phenomenon and the bipolar voltage map indicating an extensive low-voltage LV apical scar, we presumed

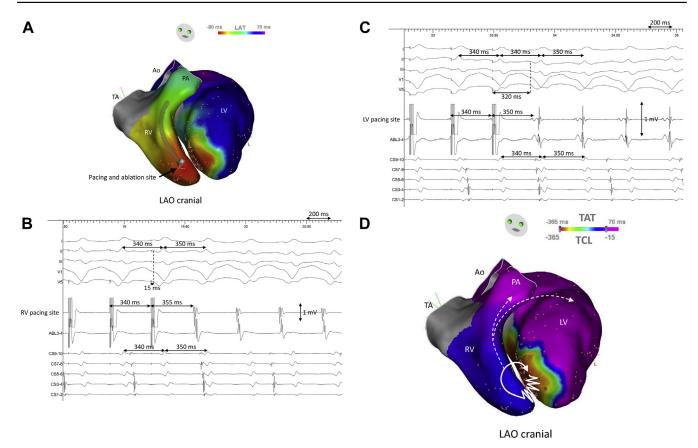


Figure 2 Electrocardiograms and 3-dimensional activation maps obtained in the case of ventricular tachycardia (VT). **A:** VT activation map of the right ventricle (RV) and left ventricle (LV). **B:** Entrainment pacing from the earliest activation site at the RV apex. **C:** Entrainment pacing from the other site of earliest activation at the LV apex opposite that of the RV apex. **D:** The accurate activation map with the reannotation of the electrograms according to the tachycardia cycle length (TCL) and the results of entrainment pacing. The reentrant circuit is shown in red, yellow, green, and blue, and the bystander area is shown in pink. Septal macroreentrant VT (*solid white arrow*) with a bystander area involving both ventricles (*dotted white arrows*) was diagnosed. ABL = ablation catheter; Ao = aorta; CS = coronary sinus; LAO = left anterior oblique; LAT = local activation time; PA = pulmonary artery; TA = tricuspid annulus; TAT = total activation time.

that the critical isthmus was located at the apical septum between the "LV entrance" site and the "RV exit" site. VT was terminated 7 seconds after delivery of RF energy to the site of earliest activation at the RV apex. The accurate activation maps by reannotation of the electrograms according to the TCL and the results of entrainment pacing indicated that the RV-LV TAT was 441 ms and greater than the TCL of 350 ms (Figure 2D).

Discussion

Three-dimensional activation mapping for macroreentrant tachycardia with a TAT that is longer than the tachycardia cycle is challenging because there can be bystander regions that activate late, simultaneously with the next tachycardia beat within the reentrant circuit. The difference between 1 electrogram within the circuit and a bystander site activating very slowly often exceeds the TCL. In the tachycardias presented here, the 3D mapping system often showed 2 or more sites of earliest activation because the bystander region was detected in the window of interest as a site of early activation. Therefore, we thought that positioning the mapping catheters at each of the sites of earliest activation and

performing entrainment pacing from each site to identify the "pseudo" site (that at which orthodromic capture occurs) and the other site as the "true" site of earliest activation would be useful to distinguish the critical circuit from the bystander region. Entrainment pacing from the "true" site of earliest activation on the circuit showed a PPI-TCL of ≤30 ms in both cases. Furthermore, as in the first case, the "pseudo" site of earliest activation in the bystander region was orthodromically captured by the entrainment pacing with a long stimulus-electrogram interval that exceeded the TCL. This was probably because of a substantial conduction delay at the gap of the prior PVI line. Therefore, we created accurate activation maps by reannotation of the electrograms according to the TCL and the results of entrainment pacing. Correctly annotated, the LA TAT was 438 ms and greater than the TCL of 205 ms in the first case (Figure 1D), and the RV-LV TAT was 441 ms and greater than the TCL of 350 ms in the second case (Figure 2D). In the first case, activation mapping of the LA roof and bystander area at the LSPV delineated the true tachycardia circuit. In the second case, the 2 sites of earliest activation were shown by entrainment pacing to be on the circuit. Further, the orthodromic QRS capture during LV entrainment indicated that the LV site of earliest activation was the "entrance site," ie, it was activated 1 cycle before the QRS, the earliest RV potential, and the basal portion of the LV. The accurate activation map revealed a huge bystander area involving the RV and LV (Figure 2D). However, parts of the area (yellow and green on Figure 2D) were not shown because the slow septal conduction of 300 ms was not mapped. The deep intramural septal area should be mapped to account in full for the TCL.

Identification on the 3D activation map of the "pseudo" site of earliest activation as the site with orthodromic capture during entrainment pacing from the "true" site of earliest activation can be used to clarify the actual reentrant tachycardia circuit. Development of a mapping algorithm that can distinguish regions that activate very late and simultaneously with the next tachycardia beat arising from the true region of earliest activation is warranted.

Conclusion

In patients with 2 or more sites of earliest activation, the identification of orthodromic capture at 1 site of earliest activation during entrainment pacing from another site of earliest activation, located by 3D activation mapping, is useful for detecting a reentrant tachycardia circuit in which the TAT is greater than the TCL.

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