

# Unusual response to atrial entrainment pacing dependent on pacing current strength: What is the mechanism?

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March 19, 2022

## Abstract

A 77-year-old woman with palpitations was referred for a second radiofrequency ablation for persistent atrial tachycardia (AT). She previously underwent pulmonary vein (PV) isolation for paroxysmal atrial fibrillation, linear ablation between the 3'o clock position of the mitral annulus (MA) and left PV from the endocardium, and ablation inside the coronary sinus (CS) for perimitral atrial tachycardia (PMAT) in the first procedure. A baseline 12-lead electrocardiogram in the second procedure showed stable AT with a cycle length (CL) of 250 ms. No PV reconnection was observed. The CS catheter was placed from 3:30 to 5:00 on the MA, and a proximal-to-distal pattern of CS activation during AT was observed. Activation mapping in the left atrial (LA) endocardium using a three-dimensional mapping system (CARTO3, Biosense Webster, Diamond Bar, CA, USA) revealed a sequence of counterclockwise rotations of the MA. Figures 1A and 1B show the intracardiac electrograms during high-output (20 V) and low-output (5 V) atrial entrainment pacing at a pacing CL of 240ms from CS 3,4, which corresponds to the 4'o clock position of the MA. Dose residual conduction occur across the mitral isthmus (MI) endocardium, epicardium, or both? What is the electrophysiological mechanism during high- and low-output entrainment pacing?

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The authors have no funding sources to disclose.

The authors have no conflicts of interest to disclose.

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## EP ROUNDS

### Case presentation

A 77-year-old woman with palpitations was referred for a second radiofrequency ablation for persistent atrial tachycardia (AT). She previously underwent pulmonary vein (PV) isolation for paroxysmal atrial fibrillation, linear ablation between the 3'o clock position of the mitral annulus (MA) and left PV from the endocardium, and ablation inside the coronary sinus (CS) for perimitral atrial tachycardia (PMAT) in the first procedure. A baseline 12-lead electrocardiogram in the second procedure showed stable AT with a cycle length (CL) of 250 ms. No PV reconnection was observed. The CS catheter was placed from 3:30 to 5:00 on the MA, and a proximal-to-distal pattern of CS activation during AT was observed. Activation mapping in

the left atrial (LA) endocardium using a three-dimensional mapping system (CARTO3, Biosense Webster, Diamond Bar, CA, USA) revealed a sequence of counterclockwise rotations of the MA. Figures 1A and 1B show the intracardiac electrograms during high-output (20 V) and low-output (5 V) atrial entrainment pacing at a pacing CL of 240ms from CS 3,4, which corresponds to the 4'o clock position of the MA. Does residual conduction occur across the mitral isthmus (MI) endocardium, epicardium, or both? What is the electrophysiological mechanism during high- and low-output entrainment pacing?

## Commentary

Creating a complete linear block is essential for preventing recurrent PMAT. Therefore, evaluating conduction across the MI linear lesion is important, and entrainment pacing and post-pacing interval (PPI) mapping during tachycardia are effective tools for identifying tachycardia circuits.<sup>1</sup> However, this is sometimes challenging because of the small or invisible potential after extensive ablation of the MI and inside the CS. Epicardial CS electrograms can be used as a surrogate for LA signals. However, CS activation can differ from contiguous LA activation. Careful signal analysis and discrimination of the local CS from the far-field LA electrograms are necessary.

In this case, the first ablation was performed at the 3'o clock line of the MI and inside the CS, but the second ablation was performed because the PMAT recurred. Figures 1A and 2A show intracardiac electrocardiograms taken during the low-output entrainment pacing at a pacing CL of 240ms from CS 3,4 corresponding to the 4'o clock position of the MA. The PPI was 291ms, longer than the tachycardia CL. Therefore, CS 3,4 are outside of the tachycardia circuit.

This case highlights the importance of the location of a multielectrode catheter (2 mm interspacing) in the LA close to the CS 3,4 to interpret the activation sequence during entrainment pacing. As shown in Figure 2A, the intervals between the CS 7,8 potentials during the first and second pacing were the same as the tachycardia CL (250 ms), while those after the third pacing coincided with the pacing CL (240 ms). Immediately after capturing the CS 7,8 potentials, the intervals between the LA potentials on LA 1,2 and 3,4 close to CS 3,4 coincided with the pacing CL. These findings indicate that low-output pacing from CS 3,4 captured only the CS musculature.

Figures 1B, 2B, and 2C show the intracardiac electrocardiograms taken during the high-output entrainment pacing at a pacing CL of 240ms from CS 3,4. In Figure 2B, only the terminal component of the CS 7,8 potential overlapped with the first pacing stimulus artifact at the same interval as the tachycardia CL. The CS 3,4 potential was observed immediately before the second pacing stimulus artifact, and the intervals between CS 3,4 potential after the second pacing were consistent with the pacing CL. In contrast, the LA potentials on LA 1,2 and 3,4 were captured by the first pacing stimulation. In Figure 2C, the PPI to LA 1,2 potential was 252ms, which was almost consistent with the tachycardia CL. Paradoxically, concealed fusion was demonstrated by selective LA capture only, although no clear far-field LA potential was observed at the site of pacing from CS 3,4 despite amplification. What mechanism underlies the unexpected inability to capture near-field CS potentials during high-output entrainment pacing from CS 3,4?

Entrainment pacing can be influenced by current strength. Different current strengths are helpful for distinguishing near-field from far-field component.<sup>2</sup> Theoretically, a relatively lower current strength probably results in selective capture of the CS musculature, while a relatively higher current strength results in non-selective capture of the LA and the CS owing to the capture of adjacent myocardium beyond the intracardiac electrocardiogram interest. In this case, the antidromic wavefront from the first pacing stimulation from CS 3,4 collided inside the CS with the orthodromic wavefront of the tachycardia. The orthodromic wavefront of the first pacing stimulation turns the MA counterclockwise and conducts into the CS, making the local CS musculature refractory to the second pacing stimulation. Therefore, subsequent entrainment pacing with concealed fusion was performed by direct LA capture; thus, only an orthodromic wavefront was observed and the CS potentials preceded the pacing stimulus artifacts. This mechanism is pseudo-selective LA capture by concealed remote capture from the CS 3,4.

Focal LA endocardial radiofrequency ablation at the gap site terminated the PMAT and caused a bidirec-

tional complete block of the MI.

## References

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## FIGURE LEGENDS

**Figure 1** Intracardiac electrograms during low-output (A) and high-output (B) atrial entrainment pacing at a pacing cycle length of 240ms from CS 3,4, which corresponds to the 4'o clock position of the mitral annulus. The numbers indicate the cycle lengths in ms. I, aV<sub>F</sub>, V1 = surface electrogram; CS = coronary sinus; HRA = high right atrium. **Figure 2** Intracardiac electrograms during low-output (A) and high-output (B and C) atrial entrainment pacing at a pacing cycle length of 240ms from CS 3,4 after the location of a multielectrode catheter in the left atrium close to the CS 3,4. Arrow = the terminal component of the CS 7,8 potential. The numbers indicate the intervals in ms. The other abbreviations are as in Figure 1.



