

Adenosine-sensitive atrial tachycardia originating from the para-Hisian region with the entrance of a slow conduction zone at the noncoronary aortic sinus



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Introduction

Adenosine-sensitive atrial tachycardias (ATs) originating from the atrioventricular (AV) node vicinity have been reported, and the mechanism responsible is considered to be reentry.¹ Previous reports have demonstrated manifest entrainment from the right atrium (RA)^{2–4} or mitral annulus (MA)^{4,5} during these ATs, and most of them could be ablated safely at the entrance of the slow conduction zone (SCZ), indicated by a manifest entrainment-guided strategy.^{2,3} We describe a case of adenosine-sensitive AT originating from the para-Hisian region that could be ablated at the entrance of the SCZ, indicated by the demonstration of manifest entrainment from the noncoronary aortic sinus (NCS).

Case report

A 68-year-old man with no history of heart disease developed symptoms of palpitations and was referred to our hospital. The electrocardiogram revealed a long-RP, narrow QRS, regular tachycardia with a heart rate of 153 beats per minute (Figure 1A), which incessantly persisted after hospitalization. After written informed consent was obtained from the patient, an electrophysiological study was performed. Multielectrode catheters were placed at the high right atrium (HRA), His bundle, coronary sinus (CS), and right ventricular apex (RVA). The supraventricular tachycardia (SVT) was easily and reproducibly induced by burst atrial pacing. The earliest atrial activation was recorded at the His-bundle electrodes (HBE) during the SVT. Any premature ventricular stimulation from RVA at various coupling intervals did not affect the SVT. After cessation of ventricular entrainment pacing during the SVT, the V-A-A-V sequence was observed (Figure 1B). VA linking was not observed on differential atrial pacing

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KEY TEACHING POINTS

- A mechanism responsible for adenosine-sensitive atrial tachycardias (ATs) originating from the atrioventricular node vicinity has been considered to be reentry, and they could be ablated safely at the entrance of the slow conduction zone (SCZ).
- In adenosine-sensitive AT cases, noncoronary aortic sinus (NCS) could be a target for ablation even if the NCS is not activated the earliest because there may be cases in which the NCS is located at the entrance of the SCZ.
- Precise entrainment mapping, including the NCS, may help to determine the optimal ablation site.

(Figure 1C)⁶ and the SVT was reproducibly terminated without an AV block by a 4-mg bolus injection of adenosine 5'-triphosphate (Figure 1D). Based on these findings, the SVT was diagnosed as an adenosine-sensitive AT.

The activation map in the RA during the AT revealed a centrifugal pattern. The earliest activation site (EAS) was located slightly posterior to the site where the His potentials were recorded (Figure 2A). A local atrial electrogram of the EAS preceded that of the HBE by 16 ms (Figure 2B). Radio-frequency (RF) application at the EAS had a risk of injuring the fast pathway or the AV node. Thus, to identify the entrance of the SCZ, entrainment pacing during the AT with a pacing cycle length of 15–20 ms shorter than the tachycardia cycle length was performed at various sites in the RA (RA appendage [RAA], high anterolateral RA, high posteroseptal RA, and cavotricuspid isthmus [CTI]) and CS electrodes (1 o'clock, 3 o'clock, 6 o'clock on the MA). However, manifest entrainment could not be observed.

Subsequently, the ablation catheter was inserted to the NCS via a retrograde transaortic approach and a local potential with a 16-ms delay compared to that of the HBE was recorded at site A. Entrainment pacing from site A revealed that the

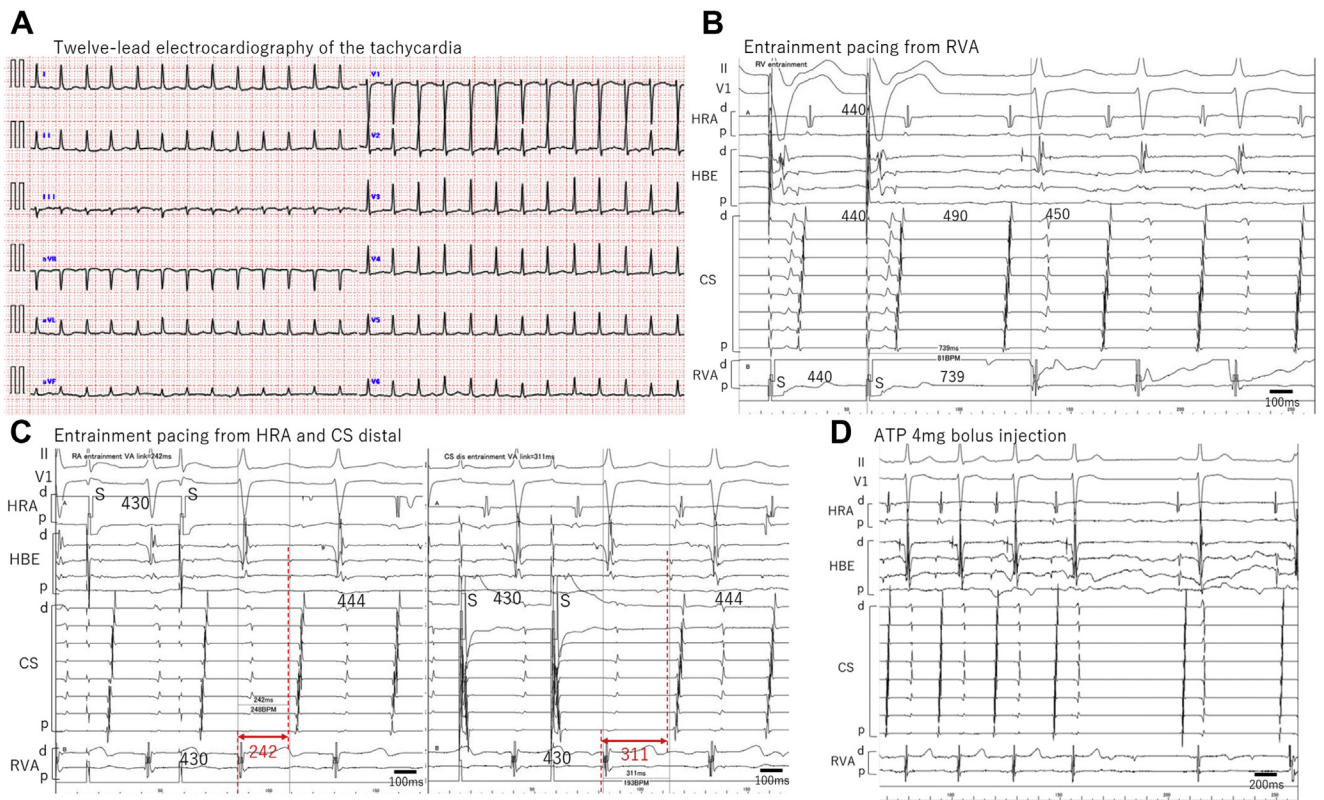


Figure 1 **A:** Twelve-lead electrocardiography revealed a long-RP, narrow QRS, regular tachycardia with a heart rate of 153 beats/min. The P-wave morphologies were positive in leads I, aVL, and V₁ and biphasic (positive/negative) in leads II, III, and aVF. **B:** After cessation of entrainment pacing from the right ventricular apex (RVA), the V-A-A-V sequence was observed. **C:** The difference in the postpacing ventriculoatrial interval between the high right atrium (HRA) and coronary sinus (CS) distal entrainment pacing was 69 ms > 14 ms, indicating the absence of VA linking.⁶ **D:** A 4-mg bolus injection of adenosine 5'-triphosphate (ATP) terminated the supraventricular tachycardia without atrioventricular block. d = distal; HBE = His-bundle electrode; p = proximal; S = stimulation.

postpacing interval was 30 ms longer than the tachycardia cycle length and demonstrated manifest entrainment (Figure 2C). Atrial electrograms recorded at the HRA were captured antidromically and those recorded at the CS and HBE were captured orthodromically with a long conduction interval. Ventricular electrograms recorded at the CS, HBE and RVA, and the His potential recorded at HBE were also orthodromically captured. Thus, this pacing site was considered to be proximal to the SCZ, and the entrance of the SCZ was presumed to be located between this pacing site and the EAS (Figure 2D).

RF application (25–30 W, 40°C, 30 seconds) was delivered at this site, following which the AT slowed down and terminated, but was easily induced by rapid atrial pacing after that. The RF application site was then gradually advanced in the NCS toward EAS. The last RF site (site B) was 14 mm away from the EAS and its local potential had a delay of 8 ms compared to that of the HBE. Entrainment pacing from site B terminated the AT, but the number of pacing stimuli needed to entrain was 3 (Figure 3A). After induction of the AT, RF application (25–30 W, 40°C, 30 seconds) at site B accelerated and terminated the AT in 4 seconds (Figure 3B). PR prolongation was not observed after RF

application. Thereafter, the AT could no longer be induced. The patient had no recurrence of tachycardia during the 12-month follow-up period without any antiarrhythmic drug.

Discussion

Iesaka and colleagues¹ first reported adenosine-sensitive AT originating from the vicinity of the AV node that was induced by premature atrial stimulation and was terminated with a small amount of adenosine. In the method of this AT ablation, RF application to the EAS close to the AV node has a potential risk of causing an inadvertent AV block. Yamabe and colleagues² and Okumura and colleagues³ clearly demonstrated manifest entrainment and successfully eliminated ATs by RF applications between the EAS and entrainment pacing site of the RA (RAA, high anterolateral RA, high posteroseptal RA, and CTI) without the occurrence of AV block. This indicates that the mechanism responsible for these ATs was reentry, and the optimal RF site was the entrance of the AT circuit. Recently, Inagaki and colleagues⁵ reported regarding adenosine-sensitive AT that originated from the anterior MA and demonstrated manifest entrainment from the CS electrode located at 1 o'clock on the MA.

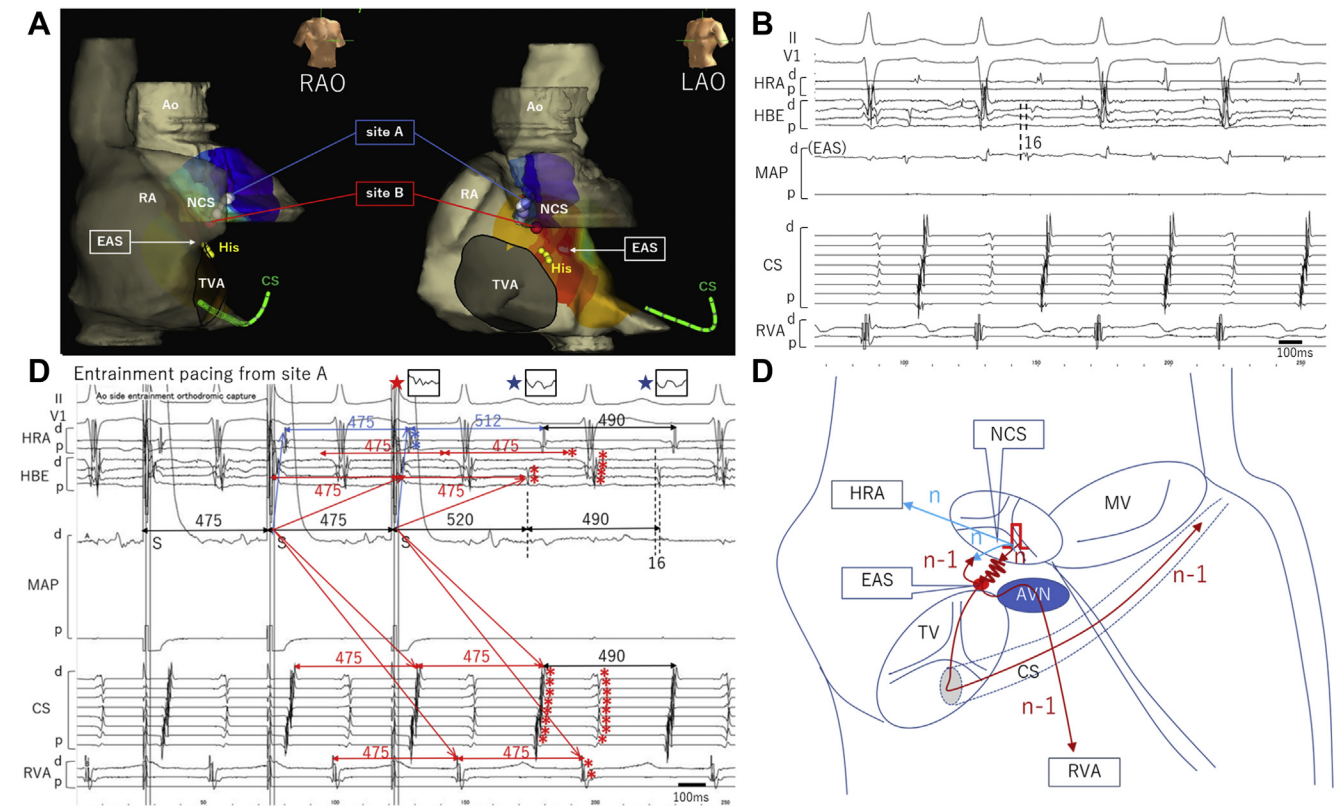


Figure 2 **A:** Activation maps during tachycardia. The blue tag (site A) shows the pacing site where manifest entrainment was observed. The white tags indicate the catheter ablation sites, where the atrial tachycardia (AT) was terminated transiently, but was subsequently induced by rapid atrial pacing. The red tag (site B) indicates the last successful catheter ablation site. The yellow tags indicate the sites where the His potentials were recorded. **B:** A local fragmented atrial electrogram of the earliest activation site (EAS) during the AT preceded that of the His-bundle electrode (HBE) by 16 ms. **C:** Entrainment pacing from site A. The red arrows indicate the orthodromic atrial activation, and red asterisks indicate the last orthodromic captured electrograms. The blue arrows indicate the antidromic atrial activation, and red asterisks indicate the last antidromic captured electrograms. Magnified surface P-wave morphologies in lead II shown in the square box during pacing (*red star*) were different from those during tachycardia (*blue star*), indicating the fusion of surface P wave during pacing except for the last captured beat. **D:** Schematic representation of manifest entrainment from the noncoronary aortic sinus (NCS). The antidromic wavefront (n) captures the high right atrium (HRA), and the orthodromic wavefront of the previous beat (n - 1) exits from the slow conduction zone of the reentrant circuit, enters the coronary sinus (CS) and the atrioventricular node (AVN), and proceeds to the ventricle. Ao = aorta; AVN = atrioventricular node; LAO = left anterior oblique; MAP = mapping catheter; MV = mitral valve; RA = right atrium; RAO = right anterior oblique; TV = tricuspid valve; TVA = tricuspid valve annulus. Other abbreviations are as in Figure 1.

Based on these reports, in the current case we performed entrainment pacing at various sites of the RA and CS electrodes; however, manifest entrainment could not be observed, and only was demonstrated from the NCS.

Ouyang and colleagues⁷ reported 9 cases of AT originating from the para-Hisian region in which successful ablation was achieved at the NCS, the majority of which had failed prior attempts at RF application from the RA or the left anteroseptal region. In this report and similar reports,^{8–10} the EAS was located at the NCS, which was recognized as the exit of the AT circuit.

To the best of our knowledge, the current case is the first report that demonstrates manifest entrainment from the NCS in a case with an adenosine-sensitive AT. This observation and successful ablation at the NCS (site B), which was not activated the earliest, showed that this AT was due to reentry, and the NCS was located at the entrance of the SCZ. The number of pacing stimuli needed to entrain with the entrainment pacing from site B was 3 (Figure 3A), which also sug-

gests involvement of the AT circuit near site B.¹¹ The reentrant circuit of this AT was presumed to be in the reverse direction of that of previous cases with EAS at the NCS. The reason why manifest entrainment could not be observed from RA could be explained by the location of the entrance of the SCZ. The high posteroseptal RA seemed to be located closest to the NCS among the pacing sites in the RA; however, the entrance of the SCZ at the NCS was located superior and “anterior” to the EAS. Therefore, the paced antidromic wavefront from a high posteroseptal RA might have captured EAS before the orthodromic wavefront of the previous beat exited from the SCZ.

Wang and colleagues¹² reported 35 cases of AT surrounding the anterior atrial septum that was successfully ablated at the NCS, 6 of which had a later atrial activation time of 5–10 ms at the NCS than that at the EAS. This report showed that NCS could be a target for ablation even if the NCS is not activated the earliest, and in some of these cases, the entrance of the SCZ could have

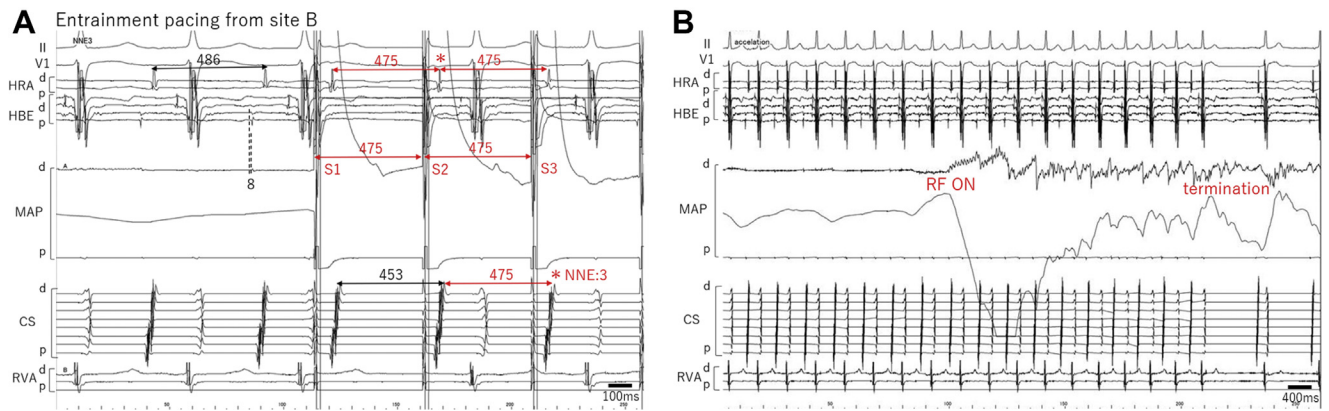


Figure 3 **A:** Entrainment pacing from site B. The cycle length of atrial electrograms at the high right atrium (HRA) and coronary sinus (CS) electrodes accelerated to the pacing cycle length (red asterisks), and the number of pacing stimuli needed to entrain (NNE) at the site was 3. **B:** Successful catheter ablation at site B. The atrial tachycardia accelerated just after delivery of radiofrequency (RF) energy and terminated. Other abbreviations are as in the previous figures.

been at the NCS, similar to the findings in our current case.

Bohora and colleagues¹³ showed the possible role of a retroaortic node in the circuit of adenosine-sensitive AT originating from the AV node vicinity that could have been ablated from the NCS, and in 3 patients thermal automaticity during ablation was seen. In our case, termination of the AT without an AV block by bolus injection of adenosine (Figure 1D), and acceleration before termination of the AT during RF application (Figure 3B), is supportive of our belief that the retroaortic node, which consists of AV node-like tissue, was involved in producing the AT.

In most previous reports,^{4,7–10} ablation at the NCS in case of AT arising adjacent to the AV node was effective and safe. However, Barkagan and colleagues¹⁴ reported the occurrence of a complete AV block requiring permanent pacemaker implantation by ablation at the NCS where the His potential was not recorded, and NCS was also reported as the target site of AV junction ablation.¹⁵ Thus, even with ablation at NCS, there is a potential risk of AV block, and RF application near the His bundle or fast pathway should be avoided as much as possible. Precise entrainment mapping, including from the NCS, may reveal the entrance of the SCZ and may help minimize the potential risk of AV block.

There were some limitations in this case. First, we performed entrainment pacing in the RA only from the RAA, high anterolateral RA, high posteroseptal RA, and CTI; however, there would have been a possible demonstration of the manifest entrainment if we had performed entrainment pacing from the high anteroseptal RA. Second, we could not show manifest or concealed entrainment from site B owing to termination of the AT by entrainment pacing, which therefore made it difficult to accurately understand the relationship between site B and AT circuit. The last successful ablation site might be not just at the entrance of the SCZ, but the SCZ near the entrance.

Conclusion

We describe a case of adenosine-sensitive AT originating from the para-Hisian region with the entrance of the SCZ at

the NCS. If manifest entrainment cannot be observed from the RA or CS, and the NCS is not activated the earliest, precise entrainment mapping in the NCS may help to determine the optimal RF site.

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References

1. Iesaka Y, Takahashi A, Goya M, et al. Adenosine-sensitive atrial reentrant tachycardia originating from the atrioventricular nodal transitional area. *J Cardiovasc Electrophysiol* 1997;8:854–864.
2. 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 atrio-ventricular node. *Heart Rhythm* 2012; 9:1475–1483.
3. Okumura K, Sasaki S, Kimura M, et al. Usefulness of combined CARTO electro-anatomical mapping and manifest entrainment in ablating adenosine triphosphate-sensitive atrial tachycardia originating from the atrioventricular node vicinity. *J Arrhythm* 2016;32:133–140.
4. Ueyama T, Shimizu A, Yoshiga Y, et al. Macroreentrant form of an adenosine 5'-triphosphate-sensitive atrial tachycardia arising from the vicinity of the atrioventricular node involving the tricuspid and mitral annuli as its reentrant circuit. *HeartRhythm Case Rep* 2017;3:289–293.
5. Inagaki D, Hojo R, Fukamizu S, et al. Adenosine-sensitive atrial tachycardia originating from the anterior mitral annulus. *HeartRhythm Case Rep* 2018; 4:542–544.
6. Maruyama M, Kobayashi Y, Miyauchi Y, et al. The VA relationship after differential atrial overdrive pacing: a novel tool for the diagnosis of atrial tachycardia in the electrophysiologic laboratory. *J Cardiovasc Electrophysiol* 2007; 18:1127–1133.
7. Ouyang F, Ma J, Ho SY, et al. Focal atrial tachycardia originating from the non-coronary aortic sinus: electrophysiological characteristics and catheter ablation. *J Am Coll Cardiol* 2006;48:122–131.
8. Kriatselis C, Roser M, Min T, et al. Ectopic atrial tachycardias with early activation at His site: radiofrequency ablation through a retrograde approach. *Europace* 2008;10:698–704.
9. Nakamura T, Hachiya H, Tanaka Y, et al. Distribution of the origin of adenosine triphosphate sensitive atrial tachycardias with the earliest activation recorded in the His bundle catheter: are they limited to the immediate vicinity of the His bundle? *Circ J* 2013;77:626–631.
10. Beukema RJ, Smit JJ, Adiyaman A, et al. Ablation of focal atrial tachycardia from the non-coronary aortic cusp: case series and review of the literature. *EP Europace* 2014;17:953–961.
11. Maruyama M, Yamamoto T, Abe J, et al. Number needed to entrain. A new criteria for entrainment mapping in patients with intra-atrial reentrant tachycardia. *Circ Arrhythm Electrophysiol* 2014;7:490–496.

12. Wang Z, Ouyang J, Liang Y, et al. Focal atrial tachycardia surrounding the anterior septum: strategy for mapping and catheter ablation. *Circ Arrhythm Electrophysiol* 2015;8:575–582.
13. Bohora S, Lokhandwala Y, Sternick EB, et al. Reappraisal and new observations on atrial tachycardia ablated from the non-coronary aortic sinus of Valsalva. *Europace* 2018;20:124–133.
14. Barkagan M, Michowitz Y, Glick A, et al. Atrial tachycardia originating in the vicinity of the noncoronary sinus of Valsalva: report of a series including the first case of ablation-related complete atrioventricular block. *Pacing Clin Electrophysiol* 2016;39:1165–1173.
15. Cuello C, Huang SK, Wagshal AB, et al. Radiofrequency catheter ablation of the atrioventricular junction by a supra-avalvular noncoronary aortic cusp approach. *Pacing Clin Electrophysiol* 1994;17:1182–1185.