



SPOTLIGHT

Intermittent orthodromic capture of the earliest activation site during atrial pacing in a case with reentrant atrial tachycardia originating from the atrioventricular node vicinity

Akira Ueoka MD, PhD¹ | Satoshi Nagase MD, PhD²  | Koji Nakagawa MD, PhD¹ | Hiroshi Morita MD, PhD³  | Hiroshi Ito MD, PhD¹

¹Department of Cardiovascular Medicine, Okayama University Graduate School of Medicine, Dentistry, and Pharmaceutical Sciences, Okayama, Japan

²Department of Advanced Arrhythmia and Translational Medical Science, National Cerebral and Cardiovascular Center, Suita, Japan

³Department of Cardiovascular Therapeutics, Okayama University Graduate School of Medicine, Dentistry, and Pharmaceutical Sciences, Okayama, Japan

Correspondence

Satoshi Nagase, Department of Advanced Arrhythmia and Translational Medical Science, National Cerebral and Cardiovascular Center, 6-1 Kishibe-shinmachi, Suita, Osaka 564-8565, Japan.

Email: snagase@ncvc.go.jp

Keywords: atrial tachycardia, entrainment, pacing, radiofrequency ablation, reentry

A 72-year-old man was referred to our institution due to frequent palpitations. Echocardiogram revealed normal left ventricular function without structural abnormalities. A 12-lead surface electrocardiogram (ECG) during sinus rhythm showed no delta waves (Figure 1A). During palpitation, the ECG exhibited regular narrow QRS complex tachycardia with a long RP interval (Figure 1B). The effect of adenosine and verapamil was not confirmed.

In an electrophysiological study, baseline AH and HV intervals were 82 ms and 38 ms, respectively. During ventricular burst stimulation and ventricular single extrastimulation, retrograde conduction was observed via a fast pathway, and the earliest atrial activation site (EAAS) was in the His bundle region. Programmed atrial extrastimulation exhibited an AH interval jump, demonstrating the presence of anterograde dual atrioventricular (AV) nodal pathways. The tachycardia was reproducibly induced by single atrial extrastimulation irrespective of AV block and A-A-V sequence was occasionally observed (Figure 1C). Because inverse correlation was found between the coupling interval of extrastimulation (S1-S2) and the return cycle of the first tachycardia beat (S2-Ae), the mechanism of the tachycardia was defined as reentry (Figure 1D). Single ventricular stimuli during the refractory period of the His bundle did not affect the tachycardia. Since differential atrial overdrive pacing from the posteroseptal right atrium (RA) and coronary sinus ostium showed that delta-VA interval was 25 ms, VA linking was considered negative.¹ The tachycardia was also induced with a V-A-A sequence

during ventricular stimulation (Figure 2A). Because of the A-A-V and V-A-A sequence and negative VA linking, this tachycardia was diagnosed as atrial tachycardia. Electro-anatomical mapping revealed that the EAAS was the His bundle region. Accordingly, entrainment pacing was attempted to investigate the entrance of the reentry circuit. During isoproterenol infusion, entrainment pacing was attempted at several sites in the RA. Constant and manifest entrainment with orthodromic capture of the EAAS was not demonstrated by the rapid atrial pacing with several cycle lengths delivered from several RA sites. During pacing at the high posteroseptal RA and coronary sinus ostium, all of the atrial electrograms were antidromically captured at the pacing cycle length. However, during pacing at the right atrial appendage (RAA), although electrograms at the free wall of the RA were constantly captured antidromically, electrogram at the EAAS was irregularly captured (Figure 2B). Careful observation revealed that the EAAS was orthodromically and antidromically captured at a ratio of 3:1, and the orthodromic conduction time from the RAA to the EAAS via slow conduction was gradually prolonged. Orthodromically captured electrograms at the EAAS showed morphologies identical to those during tachycardia. Then, we attempted entrainment pacing at the high anterolateral RA (ALRA), which is closer to the EAAS than the RAA. High ALRA pacing demonstrated 2:1 orthodromic capture of the EAAS (Figure 3A). After an entrainment study was attempted, we started to apply radiofrequency (RF) energy (20 W) at a site 25 mm remote from the EAAS, between the

This is an open access article under the terms of the [Creative Commons Attribution-NonCommercial-NoDerivs](https://creativecommons.org/licenses/by-nc-nd/4.0/) License, which permits use and distribution in any medium, provided the original work is properly cited, the use is non-commercial and no modifications or adaptations are made.

© 2022 The Authors. *Journal of Arrhythmia* published by John Wiley & Sons Australia, Ltd on behalf of the Japanese Heart Rhythm Society.

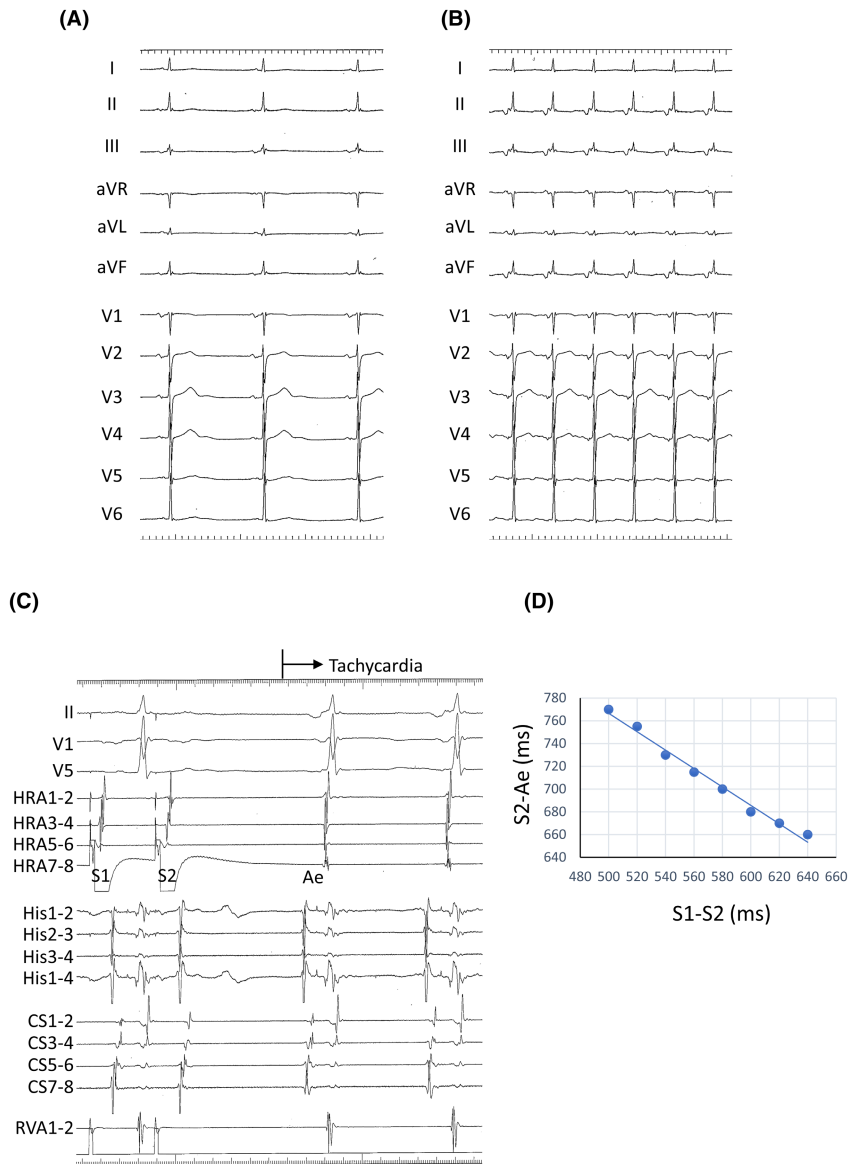


FIGURE 1 (A) ECG showing sinus rhythm at 42 beats/min. (B) ECG showing narrow QRS tachycardia at 97 beats/min. (C) Induction of tachycardia with atrial extrastimulation (S1-S1: 500 ms, S1-S2: 350 ms) irrespective of AV block. CS, coronary sinus; HRA, high right atrium; RVA, right ventricular apex; S, stimulation. (D) An inverse relationship was observed between the coupling interval of atrial extrastimulation (S1-S2) and the return cycle of the first tachycardia beat (S2-Ae). AV, atrioventricular; ECG, electrocardiogram

site at which the EAAS was orthodromically captured intermittently and the EAAS during tachycardia. When tachycardia was not terminated, the application site was moved by 2 mm toward the EAAS. After the first and second failed application, the third application at a site 21 mm remote from the EAAS immediately terminated the tachycardia (Figure 3B,C). After that, the tachycardia was no longer inducible despite isoproterenol infusion.

In the majority of cases with adenosine- and/or verapamil-sensitive reentrant atrial tachycardia originating from the AV node vicinity, the EAAS was located at or close to the His bundle region. Accordingly, RF application to the EAAS has the potential to cause AV conduction impairment. However, Yamabe et al. have reported that this atrial tachycardia could be entrained by constant atrial pacing at a site remote from the EAAS with manifest entrainment, and successfully eliminated at the site at which entrance of the slow conduction zone was suspected.² Several papers have also reported the effectiveness and safety of this manifest entrainment-guided ablation strategy.³⁻⁵ However, it is occasionally difficult to

demonstrate constant and manifest entrainment with the orthodromic capture of the EAAS by constant atrial pacing, even when delivered with several cycle lengths and from several atrial sites. The instability of slow conduction is considered to be one of the explanations of this difficulty. In fact, in this case, tachycardia cycle length was slightly unstable and fluctuated even with continuous administration of isoproterenol. When entrainment was attempted with a pacing cycle length very close to the tachycardia cycle length, tachycardia accelerated, which made evaluation difficult. Eventually, constant and manifest entrainment with the orthodromic capture of the EAAS was not demonstrated. However, the careful evaluation revealed that pacing from some atrial sites demonstrated 4:3 and 2:1 orthodromic capture of the EAAS. In accordance with previous reports, the RF application was delivered ≥ 20 mm remote from the EAAS between the intermittent capture site and the EAAS. Finally, RF application to the entrance of the slow conduction, 21 mm away from the EAAS, terminated the tachycardia immediately after the onset of energy delivery

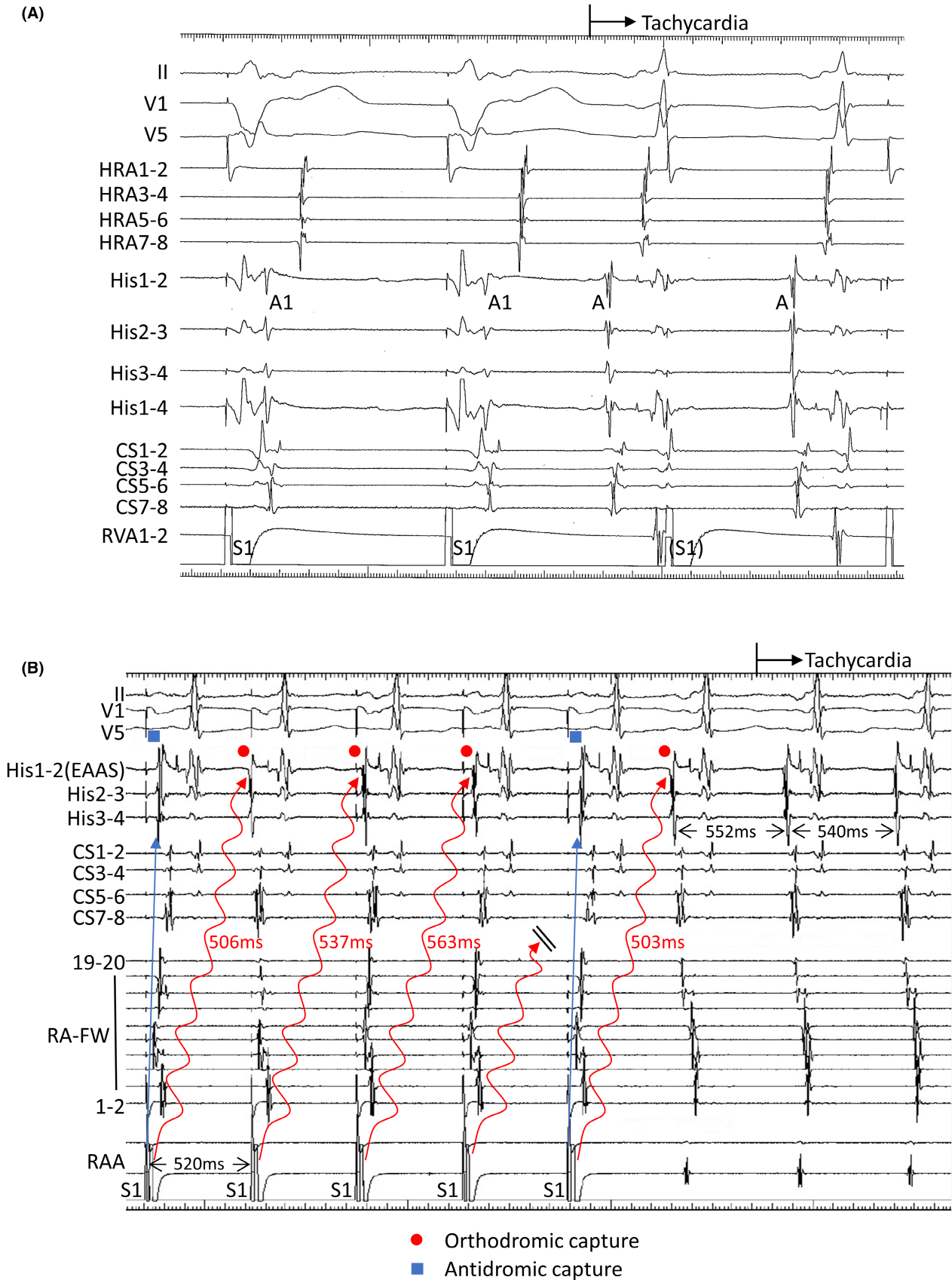


FIGURE 2 (A) Induction of tachycardia during constant ventricular pacing (S1-S1: 750 ms) with a V-A-A sequence. (B) Constant pacing (S1-S1: 520 ms) from the RAA during tachycardia. The tachycardia cycle length was 540 ms. RA-FW 1-2 was constantly captured antidromically. However, the earliest atrial activation site (EAAS = His1-2) was orthodromically (red circle) and antidromically (blue square) captured at a ratio of 3:1. The electrogram morphology at the EAAS was different between the orthodromically and antidromically captured electrograms during pacing. Moreover, the interval between the S1 and orthodromically captured electrogram (red numerical data) was gradually prolonged. RA-FW indicates free wall of the right atrium. EAAS, earliest atrial activation site; RAA, right atrial appendage

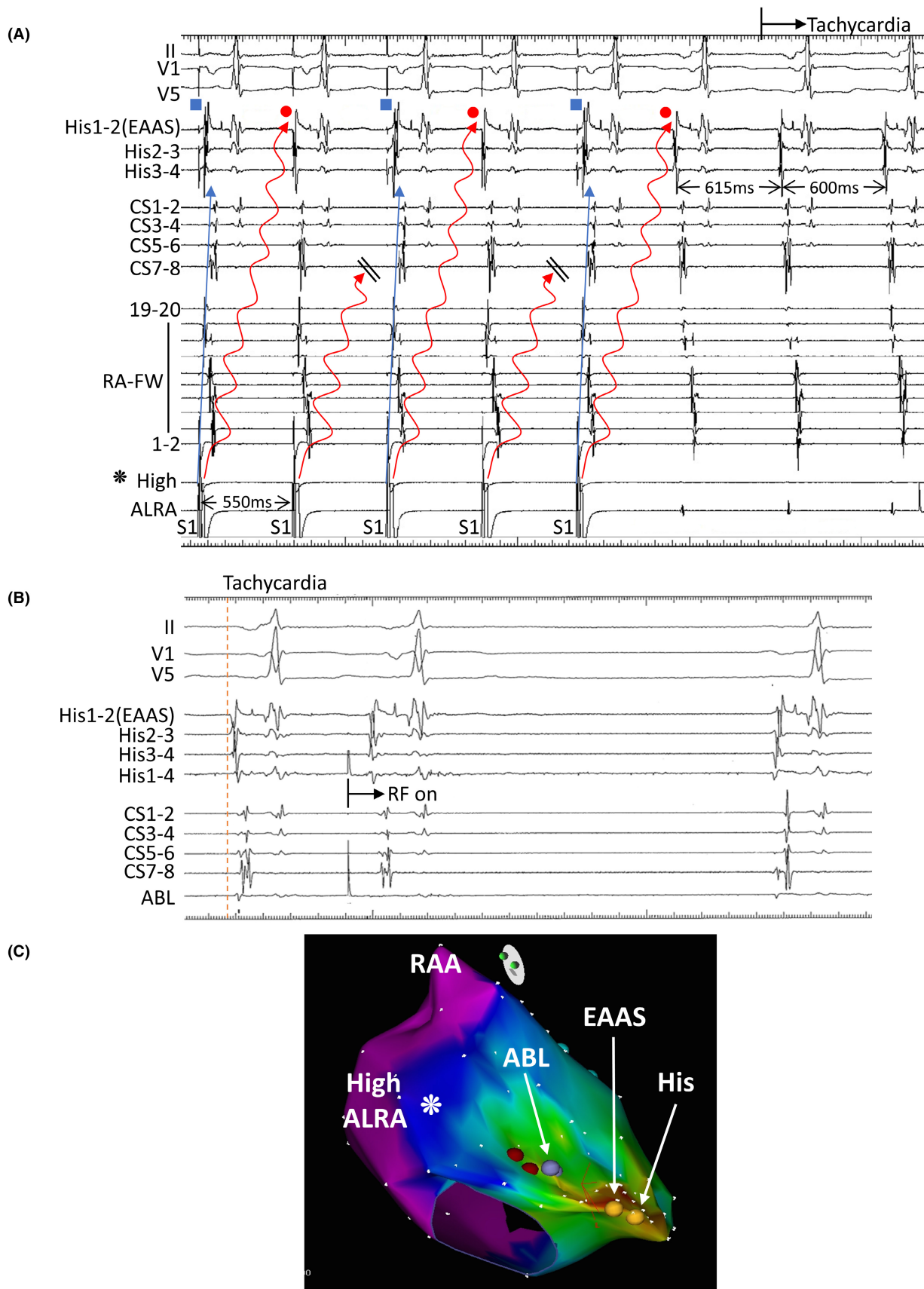


FIGURE 3 (A) Constant pacing (S1-S1: 550 ms) from the high ALRA during tachycardia. The tachycardia cycle length was 600 ms. RA-FW was constantly captured antidromically. However, the EAAS was orthodromically (red circle) and antidromically (blue square) captured alternately. The electrogram morphology at the EAAS also showed alternans. (B) RF application immediately terminated the tachycardia. ABL indicates ablation catheter. (C) Activation map during the tachycardia with the anatomical location. * indicates the location of the high ALRA. ALRA, anterolateral right atrium; EAAS, earliest atrial activation site; RF, radiofrequency

without AV conduction impairment. Hence, observation of intermittent orthodromic capture of the EAAS should be one of the important findings for secure ablation of reentrant atrial tachycardia originating from the AV node vicinity. Moreover, intermittent orthodromic capture of the EAAS accompanied by double atrial capture with single atrial pacing indicates a unidirectional block of the critical slow conduction. Importantly, the mechanism of the tachycardia must be reentry when the location of the RF application is determined in accordance with this observation. In the future, it will be necessary to verify whether this observation is always useful in other cases with adenosine- and/or verapamil-sensitive reentrant atrial tachycardia originating from the AV node vicinity.

CONFLICT OF INTEREST

Dr. Satoshi Nagase and Dr. Hiroshi Morita are affiliated with a department endowed by Japan Medtronic Inc. The remaining authors have nothing to disclose.

DISCLOSURES

There is no funding statement to declare. Since this is a case report, not a clinical trial, ethical approval has not been obtained. Informed consent was obtained from the patient. This patient has not been enrolled in a clinical trial.

ORCID

Satoshi Nagase  <https://orcid.org/0000-0001-7894-0914>

Hiroshi Morita  <https://orcid.org/0000-0001-6419-1246>

REFERENCES

1. Maruyama M, Kobayashi Y, Miyauchi Y, Ino T, Atarashi H, Katoh T, 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;18:1127–33.
2. Yamabe H, Okumura K, Morihisa K, Koyama J, Kanazawa H, Hoshiyama T, et al. Demonstration of anatomical reentrant tachycardia circuit in verapamil-sensitive atrial tachycardia originating from the vicinity of the atrioventricular node. *Heart Rhythm*. 2012;9:1475–83.
3. Yamabe H, Okumura K, Koyama J, Kanazawa H, Hoshiyama T, Ogawa H. Demonstration of anatomic reentrant circuit in verapamil-sensitive atrial tachycardia originating from the atrioventricular annulus other than the vicinity of the atrioventricular node. *Am J Cardiol*. 2014;113:1822–8.
4. Okumura K, Sasaki S, Kimura M, Horiuchi D, Sasaki K, Itoh T, et al. Usefulness of combined CARTO electroanatomical mapping and manifest entrainment in ablating adenosine triphosphate-sensitive atrial tachycardia originating from the atrioventricular node vicinity. *J Arrhythm*. 2016;32:133–40.
5. Inaba O, Nagata Y, Yamauchi Y, Miyamoto T, Goya M, Hirao K. Verapamil-sensitive atrial tachycardia with a slow conduction zone near the noncoronary aortic sinus and his bundle. *Clin Case Rep*. 2017;5:1623–7.

How to cite this article: Ueoka A, Nagase S, Nakagawa K, Morita H, Ito H. Intermittent orthodromic capture of the earliest activation site during atrial pacing in a case with reentrant atrial tachycardia originating from the atrioventricular node vicinity. *J Arrhythmia*. 2022;38:473–477. doi:[10.1002/joa3.12702](https://doi.org/10.1002/joa3.12702)