Macroequent 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

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Introduction
Atrial tachycardias (ATs) with a unique pharmacologic response to verapamil or low-dose adenosine 5'-triphosphate (ATP) arising from the para-Hisian region have been reported, and their responsible mechanism is considered to be microreentry.1 Recently, some investigators successfully demonstrated manifest entrainment in the right atrium (RA), indicating that the mechanism of these ATs could be RA macroreentry.2,3 Here we describe a case with an ATP-sensitive AT that arose from the vicinity of the atrioventricular (AV) node and that demonstrated manifest entrainment from the right and left AV annuli including an earliest site in the RA.

Case report
A 72-year-old female patient was referred to our hospital for an electrophysiological study (EPS) and radiofrequency catheter ablation (RFCA) of a supraventricular tachycardia (SVT). The electrocardiogram (ECG) recorded previously at another hospital revealed a narrow QRS tachycardia with a heart rate of 154 beats per minute (bpm), which was terminated by 5 mg of verapamil. After written informed consent was obtained, an EPS was performed under deep sedation. All antiarrhythmic drugs were discontinued for 5 half-lives prior to the EPS. Three electrode catheters were placed in the high RA (HRA), His-bundle region (HBE), and right ventricular apex (RVA) via the femoral veins. A 7F deflectable quadripolar catheter with a 4-mm distal electrode (Celsius, Biosense Webster, Diamond Bar, CA) was introduced into the coronary sinus (CS) via the right subclavian vein. There was no retrograde conduction during ventricular pacing. The SVT was easily and reproducibly induced by atrial extrastimuli or rapid atrial pacing. The earliest local atrial activation was recorded by the HBE catheter’s distal electrodes during the SVT. Ventricular pacing during the SVT revealed ventriculoatrial (VA) dissociation. No VA linkage could be observed by differential atrial pacing and the SVT was reproducibly terminated without AV block by a low-dose (2-mg) bolus intravenous injection of ATP (Figure 1A). According to these findings, the SVT was diagnosed as an ATP-sensitive AT arising from near the His bundle. During the AT, electroanatomic mapping (Ensite NavX, St Jude Medical, St Paul, MN) using a 7F deflectable quadripolar catheter with a 4-mm distal electrode (Celsius, Biosense Webster, Diamond Bar, CA) was performed. The RA activation map revealed a centrifugal pattern and earliest atrial activation site on the AV node and that demonstrated manifest entrainment from the right and left AV annuli including an earliest site in the RA.

KEYWORDS Adenosine 5'-triphosphate; Atrial tachycardia; Catheter ablation; Entrainment; Macroreentry (Heart Rhythm Case Reports 2017;3:289–293)

Conflict of interest: The authors declare no conflict of interest associated with this article. Address reprint requests and correspondence: Dr Takeshi Ueyama, Division of Cardiology, Department of Clinical Science, Yamaguchi University Graduate School of Medicine, 1-1-1 Minami-Kogushi, Ube, 755-8505, Japan. E-mail address: tueyama@yamaguchi-u.ac.jp.

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On the other hand, pacing from the NCC, HRA, HBE, and CS captured antidromically (Figure 2C). Postpacing intervals (PPIs) 20 ms longer than the TCL were also documented on the anterior TA, septal MA, and NCC. The PPI on the RAAS was identical to the pacing cycle length. RFCA applications (20–30 W, 50°C–55°C, 30 s) were applied from the anterior TA and septal MA where manifest entrainment was documented with a PPI 20 ms longer than the TCL or the number of pacing stimuli needed to entrain (NNE) were ≤3 (Figures 3A, 3B). However, RFCA applications from the endocardial side (RF1 and RF2) were not effective. Finally, a single RFCA application (20 W, 50°C, 30 s) from the NCC (Figure 3C) terminated the AT 2.5 s after initiating the application. No junctional beats or PR prolongation were observed during the RFCA. AT could no longer be induced after the RFCA from the NCC. The patient was discharged without any medications and has been asymptomatic during a 12-month follow-up period.

Discussion
Iesaka et al first described ATP-sensitive AT arising from focal sites within the triangle of Koch and speculated that their mechanism was focal reentry and that the AV node or its transitional tissues were included in part of the reentrant circuit. Subsequently, a similar pharmacologic response to adenosine or low-dose ATP has been reported among ATs originating from non–para-Hisian regions.4–6 Thus, ATP-sensitive AT in which the earliest activation is recorded around the AV node should be considered to be among ATs arising from either the right or left AV annuli. In the current case, the AT was easily induced by burst atrial pacing or extrastimuli and was sensitive to verapamil and low-dose ATP, suggesting that its mechanism was either reentry or (Figure 2D). On the other hand, pacing from the NCC, HRA, HBE, and CS captured antidromically (Figure 2C). Postpacing intervals (PPIs) <20 ms longer than the TCL were also documented on the anterior TA, septal MA, and NCC. The PPI on the RAAS was identical to the pacing cycle length. RFCA applications (20–30 W, 50°C–55°C, 30 s) were applied from the anterior TA and septal MA where manifest entrainment was documented with a PPI ≤20 ms longer than the TCL or the number of pacing stimuli needed to entrain (NNE) were ≤3 (Figures 3A, 3B). However, RFCA applications from the endocardial side (RF1 and RF2) were not effective. Finally, a single RFCA application (20 W, 50°C, 30 s) from the NCC (Figure 3C) terminated the AT 2.5 s after initiating the application. No junctional beats or PR prolongation were observed during the RFCA. AT could no longer be induced after the RFCA from the NCC. The patient was discharged without any medications and has been asymptomatic during a 12-month follow-up period.

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triggered activity.\textsuperscript{6,7} The possible mechanism of this AT was reentry because of the successful demonstration of manifest entrainment during the AT; however, we were unable to show any progressive fusion due to tiny P waves (Figure 1B) during the AT. The location of the entrainment mapping revealed sites where the PPI was \(20\) ms longer than the TCL and distributed broadly from the anterior TA to the septal MA. Although the 3-dimensional map exhibited a focal activation pattern, this AT satisfied the definition of “macro”-reentry because the distance from the superior TA to the MA was \(2\) cm (Figure 1C), indicating its critical slow conduction area might have involved the region along the left-to-right atrioventricular annuli.\textsuperscript{8} To validate this idea, we estimated the NNE.\textsuperscript{9} According to the report, an NNE \(>3\) is highly predictive of pacing outside the tachycardia circuit.\textsuperscript{9} The NNEs on the anterior TA and septal MA, where manifest entrainment was observed, were \(<3\), suggesting involvement of a reentrant circuit and its entrance near this area (Figure 3A, 3B). Wit et al\textsuperscript{10,11} and McGuire et al\textsuperscript{12,13} reported the existence of periannular cells, which are histologically similar to the atrial myocardium and have nodal-like electrophysiological properties and which respond to adenosine and have a lack of connexin 43 expression. Liu et al\textsuperscript{14} reported anatomic insights into an AT arising adjacent to the NCC, which originated from paraseptal atrial myocardium, lying adjacent to the NCC on the epicardial side and interspersed with fibrofatty tissue on the endocardial side. These findings support the idea that the paraseptal endocardial region might provide a substrate and entrance for the reentry. Recently, Yamabe et al\textsuperscript{2} and Okumura et al\textsuperscript{3} clearly demonstrated manifest entrainment and successful elimination of ATs by radiofrequency applications between the earliest activation site and entrainment pacing site on the endocardial side of the RA, indicating the responsible mechanism of these ATs would be reentry with a slow conduction zone. Hence, they speculated the presence of a slow conduction zone near the area of the TA. In the present case, we demonstrated manifest entrainment with a long interval from the TA to the MA, including the earliest site of the RA, and that was the reason we could not abolish the AT from the endocardial side, probably because of multiple entrances on the endocardial side in the present AT (Figure 3D). To the best of our knowledge, this is the first observation of these findings, and it would be difficult to apply Yamabe’s and Okumura’s ablation strategy for such cases, and vice versa, these findings may account for the
effectiveness of the radiofrequency application from the NCC in this ATP-sensitive AT arising from near the AV node.\textsuperscript{15}

**Conclusion**

The mechanism of ATP-sensitive AT that we experienced could have been macroreentry, and the reentrant circuit might have involved the bilateral AV annuli. ATs with multiple entrances into the reentrant circuit are difficult to ablate from the endocardial side. When we are able to obtain manifest entrainment from both sides of the AV annulus in a case of a para-Hisian AT such as this case, ablation from the NCC may be considered a primary strategy to achieve a successful RFCA with avoidance of any potential risk of AV conduction block.

**Acknowledgments**

We thank Mr John Martin for his linguistic assistance with this article.

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