Demonstration of anatomical reentrant tachycardia circuit in verapamil-sensitive atrial tachycardia originating from the vicinity of the atrioventricular node

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BACKGROUND The anatomical location of the reentry circuit in verapamil-sensitive atrial tachycardia originating from the vicinity of atrioventricular node (V-AT) is not well clarified.

OBJECTIVE To define the reentry circuit of V-AT.

METHODS In 17 patients with V-AT, rapid atrial pacing at a rate 5 beats/min faster than the tachycardia rate was delivered from multiple sites of the right atrium (RA) during tachycardia to define the direction of the proximity of the slow conduction area of the reentry circuit. After identification of manifest entrainment and orthodromic capture of the earliest atrial activation site (EAAS), radiofrequency energy was delivered starting at a site 2 cm away from the EAAS in the direction of the pacing site. Radiofrequency energy application site was then gradually advanced toward EAAS until the termination of tachycardia to define the entrance of the slow conduction area.

RESULTS The EAAS was orthodromically captured by pacing delivered from one of the high anterolateral RA (n = 6), high posteroseptal RA (n = 9), and RA appendage (n = 2). Radiofrequency energy delivery to the site, 10.1 ± 2.8 mm away from the EAAS, terminated V-AT immediately after the onset of delivery (2.9 ± 1.0 seconds). The successful ablation site located outside the Koch's triangle, being more distant from the His bundle site than the EAAS (12.4 ± 2.9 vs 6.4 ± 1.9 mm; $P < .0001$).

CONCLUSION The reentry circuit of V-AT located outside the Koch's triangle. V-AT was eliminated by the radiofrequency energy delivered to the entrance of the reentry circuit, which was more distant from the His bundle site than the EAAS, under the navigation of entrainment.

KEYWORDS Atrioventricular node; Atrial tachycardia; Entrainment; Reentry; Slow conduction area

ABBREVIATIONS **AT** = atrial tachycardia; **AV** = atrioventricular; **CS** = coronary sinus; **EAAS** = earliest atrial activation site; **HB** = His bundle; **RA** = right atrium

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Introduction

It has been suggested that the underlying mechanism of focal atrial tachycardia (AT) arising from the apex of Koch's triangle, in close vicinity to the atrioventricular (AV) node, is reentry, which involves the calcium channel–dependent tissue in the circuit.[1–4] However, the exact boundaries of the reentry circuit have not been convincingly defined. To define the exact anatomical tachycardia circuit of this AT, we used the entrainment pacing technique. If manifest entrainment with the orthodromic capture of the earliest atrial activation site (EAAS) is demonstrated, then the pacing site is considered to be proximal to the slow conduction area.[5] We first identified such a site proximal to the slow conduction area and then delivered radiofrequency energy to a site located between the pacing site and the EAAS. Our study hypothesis is that if AT is terminated during the radiofrequency energy application, this ablation site is on the reentry circuit, most likely at the entrance of the slow conduction area. With the uses of these entrainment pacing and radiofrequency energy application techniques, we attempted to clarify the mechanism of verapamil-sensitive AT, focusing on the location of the slow conduction area.

Methods

Patients

The study subjects were 17 consecutive patients with verapamil-sensitive AT originating from the vicinity of the AV node (6 men and 11 women; mean age 69 years) (Table 1). The inclusion criterion for sustained AT was a stable tachycardia cycle length varying by no more than 10 ms over 20 consecutive beats. Written informed consent was obtained

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Table 1 Electrophysiologic characteristics of atrial tachycardia

Patient	Age (y)/sex	TCL (ms)	Location of EAAS	Manifest entrainment pacing site	Successful RF site relative to EAAS	Relation between EAAS and RF site		
						Distance between EAAS and successful RF site (mm)	Activation time between EAAS and successful RF site (ms)	Interval from RF onset to termination (ms)
1	57/F	380	Posterior to HB	High anterolateral RA	Lateral to EAAS	10	26	2.6
2	79/M	480	Posterior to HB	High posteroseptal RA	Posterior to EAAS	10	12	2.6
3	69/F	340	Superior to HB	High posteroseptal RA	Posterior to EAAS	8	16	1.8
4	65/F	470	Superior to HB	High posteroseptal RA	Posterior to EAAS	12	20	2.2
5	70/F	300	Septal to HB	High posteroseptal RA	Posterior to EAAS	9	10	1.6
6	80/F	430	Posterior to HB	High anterolateral RA	Lateral to EAAS	12	7	4.0
7	79/F	445	Posterior to HB	High anterolateral RA	Lateral to EAAS	7	10	3.6
8	65/F	350	Septal to HB	High posteroseptal RA	Posterior to EAAS	8	15	3.2
9	57/M	420	Posterior to HB	High posteroseptal RA	Posterior to EAAS	12	20	4.0
10	48/M	375	Lateral to HB	High posteroseptal RA	Posterior to EAAS	14	7	3.9
11	82/M	420	Posterior to HB	High anterolateral RA	Lateral to EAAS	10	25	3.5
12	67/M	525	Lateral to HB	Right atrial appendage	Posterolateral to EAAS	9	30	1.2
13	83/M	690	Posterior to HB	High anterolateral RA	Lateral to EAAS	19	20	1.9
14	61/F	385	Posterior to HB	Right atrial appendage	Posterolateral to EAAS	8	10	3.6
15	78/F	550	Lateral to HB	High anterolateral RA	Lateral to EAAS	10	20	2.9
16	66/F	350	Lateral to HB	High posteroseptal RA	Posterior to EAAS	8	12	3.2
17	71/F	420	Posterior to HB	High posteroseptal RA	Posterior to EAAS	8	14	4.0
	Mean ± SD	431 ± 94				10.1 ± 2.8	16.4 ± 6.5	2.9 ± 1.0

AT = atrial tachycardia; EAAS = earliest atrial activation site during AT; F = female; HB = His bundle region; M = male; RA = right atrium; RF = radiofrequency energy application; SD = standard deviation; TCL = tachycardia cycle length.

from each patient. The protocol was approved by the Hospital Human Research Committee.

Electrophysiological study

Two 6-F quadripolar electrode catheters (St Jude Medical, St Paul, MN) were positioned in the His bundle (HB) region and the right ventricular apex. A 6-F 20-pole or decapolar or quadripolar electrode catheter (St Jude Medical) was introduced into the coronary sinus (CS). Two 7-F 4-mm-tip, deflectable quadripolar electrode catheters with a 2-mm interelectrode distance (Biosense Webster, Inc, Diamond Bar, CA, or Japan Lifeline, Tokyo, Japan) were advanced into the right atrium (RA) for atrial mapping, pacing, and ablation. Bipolar electrograms were filtered between 50 Hz and 600 Hz and recorded along with the surface electrocardiogram by using a polygraph (RMC-3000; Nihon Kohden, Tokyo, Japan, or EP-workmate; EP Med. Systems, Inc, Mt Arlington, NJ). Atrial pacing and ventricular pacing were performed by using a cardiac stimulator (SEC-4103; Nihon Kohden). The AV nodal reentrant tachycardia was excluded by a combination of the following criteria: (1) Tachycardia induction was independent of the critical atrio-hisian interval prolongation; (2) induction and perpetuation of tachycardia was independent of AV block; (3) ventricular pacing delivered during tachycardia demonstrated AV dissociation without affecting the tachycardia cycle length; (4) advancement of His electrogram over 15 ms by the ventricular pacing during tachycardia with no change in the tachycardia